RECORD 1  
Coagulopathy Before and After Liver Transplantation: From the Hepatic to the   
Systemic Circulatory Systems  
Stine J.G. Northup P.G.  
Clinics in Liver Disease (2017) 21:2 (253-274). Date of Publication: 1 May   
2017  
  
The hemostatic environment in patients with cirrhosis is a delicate balance   
between prohemostatic and antihemostatic factors. There is a lack of   
effective laboratory measures of the hemostatic system in patients with   
cirrhosis. Many are predisposed to pulmonary embolus, deep vein thrombosis,   
and portal vein thrombosis in the pretransplantation setting. This   
pretransplantation hypercoagulable milieu seems to extend for at least   
several months post-transplantation. Patients with nonalcoholic fatty liver   
disease, inherited thrombophilia, portal hypertension in the absence of   
cirrhosis, and hepatocellular carcinoma often require individualized   
approach to anticoagulation. Early reports suggest a potential role for   
low-molecular-weight heparins and direct-acting anticoagulants.  
  
  
要求: 用红色标出有liver cancer ,PVT,portal vein thrombosis ,anticoagulation 的英文单词。  
不区分大小写，  
  
RECORD 1  
Coagulopathy Before and After Liver Transplantation: From the Hepatic to the   
Systemic Circulatory Systems  
Stine J.G. Northup P.G.  
Clinics in Liver Disease (2017) 21:2 (253-274). Date of Publication: 1 May   
2017  
  
The hemostatic environment in patients with cirrhosis is a delicate balance   
between prohemostatic and antihemostatic factors. There is a lack of   
effective laboratory measures of the hemostatic system in patients with   
cirrhosis. Many are predisposed to pulmonary embolus, deep vein thrombosis,   
and portal vein thrombosis in the pretransplantation setting. This   
pretransplantation hypercoagulable milieu seems to extend for at least   
several months post-transplantation. Patients with nonalcoholic fatty liver   
disease, inherited thrombophilia, portal hypertension in the absence of   
cirrhosis, and hepatocellular carcinoma often require individualized   
approach to anticoagulation. Early reports suggest a potential role for   
low-molecular-weight heparins and direct-acting anticoagulants.  
  
RECORD 7  
anticoagulation in cirrhosis: A new paradigm?  
Leonardi F. de Maria N. Villa E.  
Clinical and Molecular Hepatology (2017) 23:1 (13-21). Date of Publication:   
1 Mar 2017  
  
The liver plays a crucial role in coagulation cascade. Global hemostatic   
process is profoundly influenced by the presence of liver disease and its   
complications. Patients with cirrhosis have impaired synthesis of most of   
the factors involved in coagulation and fibrinolysis process due to a   
reduced liver function and altered platelet count secondary to portal   
hypertension. Altered routine tests and thrombocytopenia were considered in   
the past as associated with increased risk of bleeding. These concepts   
explain both the routine use of plasma and/or platelets transfusion in   
patients with liver cirrhosis, especially before invasive procedures, and   
why these patients were considered “auto-anticoagulated”. New recent   
evidences show that patients with liver cirrhosis have a more complex   
hemostatic alteration. Despite the presence of altered levels of factors   
involved in primary hemostasis, coagulation and fibrinolysis, patients with   
stable cirrhosis have a rebalanced hemostatic, which however can easily be   
altered by decompensation or infection, both in hemorrhagic or thrombotic   
direction. Patients with cirrhosis have an increased risk of venous   
thrombotic events (namely portal vein thrombosis) while bleeding seems to be   
related to the grade of portal hypertension rather than to a hemostatic   
imbalance. The use of anticoagulants both as treatment or prophylaxis is   
safe, reduces the rate of portal vein thrombosis and decompensation, and   
improves survival. Standard laboratory coagulation tests are unable to   
predict bleeding and are inadequate for the assessment of hemostatic status   
in these patients, hence more comprehensive tests are required to guide the   
management of thrombotic and bleeding complications.  
  
RECORD 8  
Effective Prevention for Portal Venous System Thrombosis after Splenectomy:   
A Meta-Analysis  
Zhang X. Wang Y. Yu M. Huang J. Deng D. Xue H.  
Journal of Laparoendoscopic and Advanced Surgical Techniques (2017) 27:3   
(247-252). Date of Publication: 1 Mar 2017  
  
Purpose: Portal venous system thrombosis (PVST) is a common and potentially   
life-threatening complication of splenectomy for portal hypertension due to   
cirrhosis. Methods: A meta-analysis was conducted to study the necessity of   
pharmacologic prophylaxis of PVST after splenectomy and how to select the   
feasible treatment method. Articles were searched through the PubMed,   
EMBASE, Cochrane Library databases, and CNKI. Results: Overall, 404 articles   
were initially identified, and 11 of them were eligible. Among these   
selected articles, 7 articles were associated with the necessity of   
anticoagulation for prevention of PVST, while 5 were about the drug   
selection. We first demonstrated that the incidence of PVST after   
splenectomy was significantly lower in patients who received the preventive   
measures than in those who did not (odds ratio [OR]: 0.22, 95% confidence   
interval [CI]: 0.13-0.39, P < .00001). Then, we compared the new-style   
treatment with the conventional treatment and found that patients with new   
therapy method had lower incidence of PVST than those who received   
conventional treatment (OR: 0.37, 95% CI: 0.27-0.51, P < .00001). Also, some   
studies (n = 4) reported that early and combination use of anticoagulation   
drugs can lead to better outcome for patients with splenectomy and   
devascularization. Conclusion: Preventative use of anticoagulant drugs might   
decrease the incidence of PVST after splenectomy in patients with portal   
hypertension, new anticoagulant drugs such as low-molecular-weight heparin   
should be used, and early or combination use of anticoagulation drugs might   
lead to lower PVST incidence for patients.  
  
RECORD 9  
Clinical impact of portal vein thrombosis prior to liver transplantation: A   
retrospective cohort study  
Karvellas C.J. Cardoso F.S. Senzolo M. Wells M. Alghanem M.G. Handou F.   
Kwapisz L. Kneteman N.M. Marotta P.J. Al-Judaibi B.  
Annals of Hepatology (2017) 16:2 (236-246). Date of Publication: 1 Mar 2017  
  
Introduction. To identify the impact of portal vein thrombosis (PVT) and   
associated medical and surgical factors on outcomes post liver transplant   
(LT). Material and methods. Two analyses were performed. Analysis One:   
cohort study of 505 consecutive patients who underwent LT (Alberta) between   
01/2002-12/2012. PVT was identified in 61 (14%) patients. Analysis Two:   
cohort study of 144 consecutive PVT patients from two sites (Alberta and   
London) during the same period. Cox multivariable survival analysis was used   
to identify independent associations with post-LT mortality. Results. In   
Analysis One (Alberta), PVT was not associated with post-LT mortality (log   
rank p = 0.99). On adjusted analysis, complete/occlusive PVT was associated   
with increased mortality (Hazard Ratio (HR) 8.4, p < 0.001). In Analysis Two   
(Alberta and London), complete/occlusive PVT was associated with increased   
mortality only on unadjusted analysis (HR 3.7, p = 0.02). On adjusted   
analysis, Hepatitis C (HR 2.1, p = 0.03) and post-LT portal vein   
re-occlusion (HR 3.2, p = 0.01) were independently associated with increased   
mortality. Conclusion: Well-selected LT patients who had PVT prior to LT had   
similar post-LT outcomes to non-PVT LT recipients. Subgroups of PVT patients   
who did worse post-LT (complete/occlusive thrombosis pre-LT, Hepatitis C or   
post-LT portal vein re-occlusion) warrant closer evaluation in listing and   
management post-LT.  
  
RECORD 10  
The influences by anticoagulation therapy on esophagogastric variceal   
hemorrhage to liver cirrhosis patients with portal vein thrombosis  
Jie C.Y. Yuan L. Jian W.  
Hepatology International (2017) 11:1 Supplement 1 (S324-S325). Date of   
Publication: 1 Feb 2017  
  
Background: portal vein thrombosis (PVT) is one of the common complications   
of decompensated liver cirrhosis (LC), now the most common method for   
thrombosis is anticoagulation therapy, the clinical use of anticoagulant   
treatment to portal vein thrombosis is very careful, even patients with   
portal vein thrombos are may not use anticoagulant therapy because of the   
risk of bleeding. But there are reports that anticoagulant therapy does not   
increase the incidence the upper gastrointestinal bleeding, and clinical   
tests show that patients giving anticoagulant therapy after endoscopic   
variceal sequential therapy does not increase the risk of upper   
gastrointestinal bleeding. Therefore, whether giving anticoagulation therapy   
for liver cirrhosis with portal vein thrombosis is still not unified.   
Methods: Review 239 cases of cirrhosis patients diagnosed in our hospital   
from 2012.1 to 2012.12, 33 cases of liver cirrhosis combined with PVT   
patients were thrombosis group. 10 patients giving anticoagulant therapy of   
33 cases were anticoagulation therapy group, and the other 23 cases were   
control group. In the 33 cases of cirrhosis patients with portal vein   
thrombosis, the 10 patients with esophagogastric variceal hemorrhage were   
hemorrhagic group, 23 patients without bleeding were not hemorrhage control   
group. Recording patients age, gender, etiology, whether there was a history   
of diabetes and splenectomy, spleen thickness and width of portal vein, the   
degree of esophageal and gastric varices, with or without portal   
hypertension and liver ulcer, ascites extent, Child-Pugh score, with or   
without endoscopic variceal sequential therapy and taking propranolol, as   
well as laboratory tests. Using anticoagulant drugs in the treatment of   
cirrhosis patients with PVT to observate whether upper gastrointestinal   
bleeding in nearly 1 years to analysis the influences by anticoagulation   
therapy on esophagogastric variceal hemorrhage, in order to elaborated the   
risk factors and preventive measures for liver cirrhosis patients with PVT   
with esophagogastric variceal hemorrhage. Result: 1. The degree of   
esophageal and gastric varices was a risk factor, P<0.05; endoscopic   
variceal sequential therapy was a protective factor, the bleeding rates   
compared with sequential therapy and no treated people was 30 and 73.9%   
respectively, a significant difference (P <0.05). 2. The bleeding rates   
between anticoagulant therapy group and control group in the use of   
anticoagulant drugs was 40 and 26.1% respectively, no significant difference   
(P >0.05). Conclusion: The esophagogastric variceal hemorrhage of cirrhosis   
patients with PVT was closely related to the degree of varicose vein. The   
endoscopic variceal sequential therapy can significantly reduce the risk of   
variceal bleeding of cirrhosis patients with PVT. Anticoagulant therapy for   
cirrhosis patients with PVT may not increase esophagogastric variceal   
hemorrhage incidence. (Table Presented).  
  
RECORD 11  
portal vein thrombosis: A Moroccan single center experience  
Zejly H. Benelbarhdadi I. Ajana F. Afifi R. Elfeyfi A.E.  
Hepatology International (2017) 11:1 Supplement 1 (S578). Date of   
Publication: 1 Feb 2017  
  
Background: portal vein thrombosis is a rare hepatic vascular disease. It is   
an important cause of noncirrhotic prehepatic portal hypertension. Over the   
last few years, it has been increasingly diagnosed by the widespread use of   
Doppler ultrasound. The aim of this study was to describe risk factors and   
etiologies, clinical presentation, complications, and treatment of portal   
vein thrombosis in a single center study. Methods: 120 patients were   
identified from 1991 to 2016 (25 years). All data were obtained from the   
patient records. Patients with cirrhosis were excluded. Result: The group   
included 76 women and 44 men. The mean age was 36.5 years (06-82 years).   
Common symptoms were hemorrhage in 58.3% of cases, abdominal pain in 55%,   
signs of portal hypertension were present in more than 75% of cases. The   
diagnosis was established by Doppler ultrasound that showed the portal   
thrombosis and its extension or the portal cavernous transformation in some   
cases. The endoscopy showed that 83% of patients had esophageal varices,   
associated with gastric varices in 16% of cases and to portal hypertensive   
gastropathy in 14% of cases. In our study, prothrombotic disorder was found   
in 32.5% of cases, especially protein C and S deficiency, the association of   
multiples deficiency was found in 22% of cases. Primary myeloproliferative   
syndromes were reported in 7% of all the patients. Other causes were found,   
like celiac disease by hyperhomocysteinemia, liver abscess, tuberculosis,   
hepatocellular carcinoma, systemic lupus, choledocolithiasis, abdominal   
trauma and pregnancy. Patients with varices were treated endoscopically with   
band ligation and/or sclerotherapy and pharmacological treatment by   
b-blockers with treatment of the cause if it was identified. anticoagulation   
therapy was proposed to 9 patients who had clinical manifestations of   
thrombosis. The extension of the thrombosis wasn't seen after the stopping   
of the anticoagulants and the patients who didn't receive any   
anticoagulation therapy didn't present any extension of thrombosis. 5 deaths   
were enregistered in our study with 1 case of intestinal infarction died   
after surgery. Conclusion: The portal vein thrombosis is a disorder with a   
good prognosis which should be rapidly diagnosed and which requires   
interdisciplinary collaboration in order to prevent or treat invariably   
ensuing complications. The outcome of some patients on our study is good   
even without anticoagulation therapy. It's indicated in acute portal vein   
thrombosis. The role of anticoagulation in chronic portal vein thrombosis   
needs to be further studied.  
  
  
  
  
  
  
  
  
RECORD 17  
Clinical outcome of 127 cases of splanchnic venous thrombosis: Benefit of   
anticoagulant therapy  
Canafoglia L. Rupoli S. Baroni G.S. Gironella M. Micucci G. Federici I.   
Offidani M. Fiorentini A. Riva A. Da Lio L. Scortechini A.R. Honorati E.   
Leoni P.  
Blood (2016) 128:22. Date of Publication: 2 Dec 2016  
  
Background: Splanchnic venous thrombosis (SVT) encompasses thrombosis in the   
mesenteric, splenic or portal veins (with or without hepatic veins   
involvement). Little is known about appropriate therapeutic interventions   
and long-term clinical outcome of SVT patients. Aim of this study was to   
identify the correct management of SVT and encourage a multidisciplinary   
approach by a team composed of hematologists, hepatologists, and   
infectivologists. Methods:We analyzed clinical, laboratory, therapeutic and   
outcome data of 127 patients with SVT that were recruited from 2000 to 2016.   
In patients with no active bleeding, anticoagulation treatment was started   
as soon as possible, according to platelet count. We administered   
intermediate or full therapeutic dose low-molecular-weight heparin (LMWH)   
and early initiation of vitamin-K antagonist (VKA; INR range 2-3 or 1.8-2.5   
in patients with high bleeding risk) for a platelet count >50.000/μl, only   
half or prophylactic dose of LMWH for a platelet count >30.000 and <   
50.000/μl and no treatment for a platelet count <30.000/μl. Indefinite   
duration treatment was used for patients with persistent or permanent risk   
(i.e. cirrhosis, active solid cancer and hematological cancer). Moreover, an   
appropriate prophylaxis with beta-blockers and endoscopic therapies were   
applied in cirrhotic SVT. The quality of VKA treatment was assessed by the   
time in therapeutic range (TTR). The number of vascular complications was   
expressed as incidence rate, calculated by the number of events per 100   
patients-year of observation. The Kaplan-Meier method was performed to   
estimate the time to reach vessel recanalization. Cox regression analysis   
was used to identify independent predictors of vascular events or   
recanalization. Results: Overall, 127 patients were included (median age 58   
years; 74% males). The median follow-up of all patients was 11 months   
(1-212). portal vein thrombosis was the most common site of thrombosis   
(50%), followed by multiple venous involvement (37%). liver cirrhosis and   
solid neoplasms were the common underlying disease (72% and 36%   
respectively) while myeloproliferative neoplasms were identified in 8   
patients (6.2%). Eighty-nine patients (70%) had esophageal varices (grade >2   
in 55 patients) and 81 (64%) had thrombocytopenia (mean 72.000/ μl range   
28.000/μl-148.000/μl). Ninety-nine patients (78%) were treated with   
anticoagulant therapy: 36% with intermediate or full dose of LMWH, 40% with   
half or prophylactic dose of LMWH and 24% with VKA (TTR 76%). During a   
median duration therapy of 7 months, the incidence rate of thrombotic events   
was 1.1 per 100 pt-y while the incidence rate of major bleeding was 1.6 per   
100 pt-y. At univariate analysis, esophageal varices (p=0.030), renal   
failure (p=0.001) and liver cirrhosis (p=0.05) significantly increased the   
risk of bleeding events. Moreover VKA exposure was associated with a   
significantly lower risk of bleeding events compared to LMWH (p=0.042).   
Fifty-six patients (44%) obtained vessel recanalization and the probability   
of recanalization of the occluded vessels was 50% at 18 months. At   
univariate analysis, factors associated with a lack of recanalization   
included liver cirrhosis (p=0.004) and solid tumor (p=0.010). Only one death   
was attributed to fatal bleeding whereas 31 patients died for causes not   
related to anticoagulation (cirrhosis, cancer). Conclusions: Our study   
suggests the effectiveness of anticoagulant therapy (especially VKA),   
leading to thrombus recanalization in 44% of patients with SVT. Notably, the   
anticoagulant treatment was associated with a very low bleeding incidence   
also in patients with major risk factors for bleeding (i.e. liver cirrhosis,   
cancer or esophageal varices). Treatment algorithm and therapeutic decisions   
were taken as a multidisciplinary team, able to adapt the individual   
approach and avoid fatal complications.  
  
RECORD 18  
portal vein thrombosis after laparoscopic sleeve gastrectomy: presentation   
and management  
Belnap L. Rodgers G.M. Cottam D. Zaveri H. Drury C. Surve A.  
Surgery for Obesity and Related Diseases (2016) 12:10 (1787-1794). Date of   
Publication: 1 Dec 2016  
  
Background portal vein thrombosis (PVT) is a serious problem with a high   
morbidity and mortality, often exceeding 40% of affected patients. Recently,   
PVT has been reported in patients after laparoscopic sleeve gastrectomy   
(LSG). The frequency is surprisingly high compared with other abdominal   
operations. Objective We present a series of 5 patients with PVT after LSG.   
The treatment was not restricted simply to anticoagulation alone, but was   
determined by the extent of disease. A distinction is made among   
nonocclusive, high-grade nonocclusive, and occlusive PVT. We present   
evidence that systemic anticoagulation is insufficient in occlusive   
thrombosis and may also be insufficient in high-grade nonocclusive disease.   
Setting Single private institution, United States. Methods We present a   
retrospective analysis of 646 patients who underwent LSG between 2012 and   
2015. In all patients, the diagnosis was established with an abdominal   
computed tomography (CT) scan as well as duplex ultrasound of the portal   
venous system. All patients received systemic anticoagulation. Depending on   
the extent of disease, thrombolytic therapy and portal vein thrombectomy   
were utilized. All patients received long-term anticoagulation. Results Four   
patients with PVT were identified. A fifth patient with PVT after LSG was   
referred from another center. The mean age of all patients was 49 years. One   
patient had a history of deep vein thrombosis (DVT). No complications were   
identified intraoperatively or during the hospital stay, and all patients   
were discharged by postoperative day 2. The patients presented with PVT at   
an average of 20 days (range: 10–35) post-LSG. The CT scan was positive for   
PVT in all patients. In stable noncirrhotic patients with nonocclusive   
disease, we administered therapeutic anticoagulation. One patient with   
high-grade, nonocclusive PVT received anticoagulation alone. Patients with   
occlusive disease were treated with operative thrombectomy including   
intraoperative and postoperative thrombolysis (tissue plasminogen activator)   
with subsequent therapeutic anticoagulation, followed by oral warfarin or a   
factor Xa inhibitor. There was 1 death from multisystem organ failure in the   
patient who was referred from another institution with occlusive disease,   
initially managed only with an anticoagulation infusion. Conclusions We   
maintain that portal vein patency is essential to normal gastrointestinal   
physiology and should be the treatment goal in all patients with PVT. In   
these patients, the therapeutic option should be guided by the extent of the   
thrombosis. In view of currently available approaches, we propose that   
operative portal vein thrombectomy, in conjunction with fibrinolysis and   
anticoagulation, offers the best long-term success in patients with   
occlusive PVT.  
  
RECORD 19  
Cavernous sinus thrombosis and meckel diverticular bleed associated with   
fusobacterium bacteremia  
Azadeh N. Wilson J. Karnatovskaia L.  
Critical Care Medicine (2016) 44:12 Supplement 1 (515). Date of Publication:   
1 Dec 2016  
  
Learning Objectives: Sinovenous thrombosis is a rare complication of   
Fusobacterium necrophorum infection, often associated with septic   
thrombophlebitis or Lemierre's syndrome. Meckel's diverticulum is uncommon   
and often clinically silent. We present a case of septic sinovenous   
thrombosis associated with Meckel's diverticular bleed. Methods: A 19 year   
old male with recurrent otitis media and chronic sinusitis was admitted to   
the intensive care unit with septic shock after a prodrome of sore throat,   
headache, and photophobia for 1 week. He was initiated on broad spectrum   
antibiotics for possible meningoencephalitis. Cerebrospinal fluid analysis   
revealed a neutrophilic pleocytosis, but cultures were negative. Within 21   
hours, blood cultures grew F. necrophorum. Computed tomography (CT) with   
contrast and magnetic resonance (MR) imaging of the head and neck were only   
significant for complete opacification of the right sphenoid sinus. He   
continued to have severe headaches. MR angiography and venography revealed   
cavernous sinus thrombosis. On hospital day 3, he developed hematochezia   
associated with a 3 g/dl drop in hemoglobin. Upper and lower endoscopies   
were unremarkable. A triple phase abdominal CT revealed Meckel's   
diverticulum. He underwent surgical resection (pathology showed focal   
ulceration and gastric heterotopia), was started on anticoagulation, and   
completed 6 weeks of antibiotics with a good clinical outcome. Results:   
Intracranial complications of F. necrophorum include sinovenous thrombosis,   
meningitis, and cerebral abscess. Sinovenous thrombosis is usually thought   
of in the setting of Lemierre's syndrome/ thrombophlebitis of the internal   
jugular veins, or more commonly in the setting of otogenic infection.   
Primary foci of F. necrophorum infection in other sites are uncommon but can   
occur in the urogenital or gastrointestinal (GI) tracts; portal vein   
thrombosis and liver abscess have been described. Oral ulcers are a reported   
complication of necrobacillosis; however, ulcers of the GI tract, namely   
Meckel's diverticulum, have not previously been reported in this setting.  
  
RECORD 20  
Progression of Thrombus in Portal Vein, Superior Mesenteric Vein, and   
Splenic Vein even on anticoagulation in a Patient with Ascending Colonic   
Malignancy with Liver Metastasis: portal vein thrombosis versus Portal Vein   
Tumor Thrombosis  
Sule A. Borja A. Chin T.J.  
International Journal of Angiology (2016) 25:5 (e97-e99). Date of   
Publication: 1 Dec 2016  
  
portal vein thrombosis (PVT) in a setting of liver metastasis is not easy to   
treat as it may be portal vein tumor thrombus (PVTT). A 77-year-old male   
patient was diagnosed as ascending colon carcinoma, underwent right   
hemicolectomy in 1991 with a recurrence in July 2009. In August 2009, he   
underwent computed tomography (CT) scan of the abdomen which showed evidence   
of superior mesenteric vein thrombosis with no liver metastasis. He was   
started with anticoagulation and decision was to treat long term. He was   
admitted with mesenteric artery ischemic symptoms in February 2012 on   
anticoagulation. CT scan abdomen and pelvis in February 2012 showed tumor   
thrombus involving the superior mesenteric vein, portal vein, and splenic   
vein with hepatic metastasis. His tumor marker chorioembryonic antigen was   
34 μg/L. He was continued on anticoagulation. A repeat CT scan abdomen after   
2 years (in January 2014) showed, increase in size of hepatic metastasis,   
extensive thrombus involving the superior mesenteric vein, portal vein, and   
splenic vein with collaterals. Mesentery was congested due to extensive   
superior mesenteric vein thrombus. He finally succumbed in June 2014. It is   
very important to differentiate PVT from PVTT as the prognosis is different.   
PVTT progresses despite of long-term anticoagulation with poor prognosis.  
  
RECORD 21  
Thrombus Resolution in Two Patients with portal vein thrombosis without   
anticoagulation: Do We Need to Anticoagulate Patients with Portal Vein   
Thrombosis?  
Borja A. Xing W. Lymen E. Azucena B. Sule A.A.  
International Journal of Angiology (2016) 25:5 (e93-e96). Date of   
Publication: 1 Dec 2016  
  
portal vein thrombosis (PVT) is a thrombosis that develops in the trunk of   
the portal vein which can extend to its branches. It results from a   
combination of local and systemic prothrombotic factors. anticoagulation is   
generally considered in PVT patients as long as the risk of bleeding is low.   
Limited data have been published regarding spontaneous resolution of PVT. We   
describe two cases of asymptomatic PVT who were not given anticoagulation in   
view of several factors, who, on repeat scans, showed resolution of their   
thrombus.  
  
RECORD 22  
Liver transplant recipients with portal vein thrombosis receiving an organ   
from a high-risk donor are at an increased risk for graft loss due to   
hepatic artery thrombosis  
Stine J.G. Argo C.K. Pelletier S.J. Maluf D.G. Northup P.G.  
Transplant International (2016) 29:12 (1286-1295). Date of Publication: 1   
Dec 2016  
  
We hypothesize that recipients with pretransplant portal vein thrombosis   
(PVT) receiving organs from high-risk donors (HRD) are at an increased risk   
of HAT. Data on all liver transplants in the United States from February   
2002 to March 2015 were analyzed. Recipients were sorted into two groups:   
those with PVT and those without. HRDs were defined by donor risk index   
(DRI) >1.7. Multivariable logistic regression models were constructed to   
assess the independent risk factors for HAT with the resultant graft loss   
≤90 days from transplantation. A total of 60 404 candidates underwent liver   
transplantation; of those recipients, 623 (1.0%) had HAT, of which 66.0% (n   
= 411) received organs from HRDs compared with 49.3% (n = 29 473) in   
recipients without HAT (P < 0.001); 2250 (3.7%) recipients had   
pretransplantation PVT and received organs from HRDs. On adjusted   
multivariable analysis, PVT with a HRD organ was the most significant   
independent risk factor (OR 3.56, 95% CI 2.52–5.02, P < 0.001) for the   
development of HAT. Candidates with pretransplant PVT who receive an organ   
from a HRD are at the highest risk for postoperative HAT independent of   
other measurable factors. Recipients with pretransplant PVT would benefit   
from careful donor selection and possibly anticoagulation perioperatively.  
  
RECORD 23  
Splanchnic vein thrombosis in myeloproliferative neoplasms: Risk factors for   
recurrences in a cohort of 181 patients  
De Stefano V. Vannucchi A.M. Ruggeri M. Cervantes F. Alvarez-Larrán A. Iurlo   
A. Randi M.L. Pieri L. Rossi E. Guglielmelli P. Betti S. Elli E. Finazzi   
M.C. Finazzi G. Zetterberg E. Vianelli N. Gaidano G. Nichele I. Cattaneo D.   
Palova M. Ellis M.H. Cacciola E. Tieghi A. Hernandez-Boluda J.C. Pungolino   
E. Specchia G. Rapezzi D. Forcina A. Musolino C. Carobbio A. Griesshammer M.   
Barbui T.  
Blood Cancer Journal (2016) 6:11 Article Number: e493. Date of Publication:   
4 Nov 2016  
  
We retrospectively studied 181 patients with polycythaemia vera (n=67),   
essential thrombocythaemia (n=67) or primary myelofibrosis (n=47), who   
presented a first episode of splanchnic vein thrombosis (SVT). Budd-Chiari   
syndrome (BCS) and portal vein thrombosis were diagnosed in 31 (17.1%) and   
109 (60.3%) patients, respectively; isolated thrombosis of the mesenteric or   
splenic veins was detected in 18 and 23 cases, respectively. After this   
index event, the patients were followed for 735 patient years (pt-years) and   
experienced 31 recurrences corresponding to an incidence rate of 4.2 per 100   
pt-years. Factors associated with a significantly higher risk of recurrence   
were BCS (hazard ratio (HR): 3.03), history of previous thrombosis (HR:   
3.62), splenomegaly (HR: 2.66) and leukocytosis (HR: 2.8). Vitamin   
K-antagonists (VKA) were prescribed in 85% of patients and the recurrence   
rate was 3.9 per 100 pt-years, whereas in the small fraction (15%) not   
receiving VKA more recurrences (7.2 per 100 pt-years) were reported.   
Intracranial and extracranial major bleeding was recorded mainly in patients   
on VKA and the corresponding rate was 2.0 per 100 pt-years. In conclusion,   
despite anticoagulation treatment, the recurrence rate after SVT in   
myeloproliferative neoplasms is high and suggests the exploration of new   
avenues of secondary prophylaxis with new antithrombotic drugs and JAK-2   
inhibitors.  
  
RECORD 24  
anticoagulation for portal vein thrombosis in cirrhosis  
Intagliata N.M. Ferreira C.N. Caldwell S.H.  
Clinical Liver Disease (2016) 8 Supplement 1 (S10-S15). Date of Publication:   
1 Nov 2016  
  
RECORD 25  
Splenic infarction and branch portal vein thrombosis secondary to   
PEG-asparaginase  
Kohorst M. Warad D. Rodriguez V. Nageswara Rao A.  
Pediatric Blood and Cancer (2016) 63 Supplement 3 (S114). Date of   
Publication: 1 Nov 2016  
  
Background/Objectives: PEG-asparaginase leads to plasma asparagine depletion   
and hepatotoxicity causing decreased synthesis of pro-coagulant and   
anti-coagulant proteins. Thrombotic complications have been reported in 3-5%   
of paediatric patients, with majority of the events related to either   
central nervous system or central venous catheters (CVC). Design/Methods:   
Case report of a rare thrombotic event following PEG-asparaginase   
administration and brief literature review. Results: An 18-year-old male   
with a poorly-differentiated lymphoblastic leukaemia (favoring T-cell) was   
treated with four drug induction regimen (prednisone, daunorubicin,   
vincristine and PEG-asparaginase). Five days following PEG-asparaginase,   
Doppler ultrasonography showed an acute occlusive superficial cephalic vein   
thrombus. Simultaneously, he also developed cramping epigastric/abdominal   
pain. Computed tomography imaging performed 12 days after PEG-asparaginase   
administration showed a moderate/large splenic infarct and portal vein   
branch thrombosis. At our institution, fibrinogen levels, antithrombin (AT)   
activity, prothrombin time (PT/INR) and activated partial thromboplastin   
time (aPTT) are monitored following PEG-asparaginase administration in   
adolescents and young adults. His evaluation two days prior to detection of   
the splenic infarct showed low fibrinogen (<50 mg/dl), elevated INR (2.8;   
range 0.8-1.2 sec) and aPTT levels (59 sec; range 28-38 sec). Following   
imaging studies, additional labs included low AT activity (44%; range   
80-130%). Management was supportive with blood products. Eleven days later,   
imaging studies revealed worsening splenic infarct and a new CVC related   
acute deep vein thrombosis. Low molecular weight heparin was started (target   
heparin level of 0.5-1.0 IU/ml). Antithrombin concentrates and   
cryoprecipitate were administered when AT activity <60% and fibrinogen <50   
mg/dl respectively, and PEG-asparaginase therapy was continued. No further   
thrombotic or bleeding complications were observed. Conclusion: Adolescents   
and young adults receiving PEG-asparaginase are at increased risk of   
thrombosis and bleeding. Monitoring of PT, aPTT, fibrinogen, and AT activity   
is recommended. In patients with abnormal laboratory evaluation and   
thrombosis, further PEG-asparaginase can be safely administered with   
appropriate anticoagulation in combination with AT and fibrinogen   
replacement therapy.  
  
RECORD 26  
A rare case of portal biliopathy and pylephlebitis following the injection   
of cyanoacrylate into the duodenal varix  
Rew J.S. Jun C.H. Cho E.  
Journal of Gastroenterology and Hepatology (Australia) (2016) 31 Supplement   
3 (295). Date of Publication: 1 Nov 2016  
  
There is no established standard therapy for duodenal variceal bleeding, and   
the treatment-related complications are not well known. We describe a case   
of symptomatic portal biliopathy and pylephlebitis after duodenal varix   
obliteration using an injection of cyanoacrylate. A 55-year-old man   
presented with melena. Esophagogastroscopy findings showed large duodenal   
varices with stigmata of recent bleeding; thus, cyanoacrylate was injected   
to achieve hemostasis. The patient was discharged from the hospital without   
additional signs of bleeding. Four months later, he developed a fever and   
abdominal pain. Results of abdominal-computed tomography and   
esophagogastroscopy showed that the duodenal varices disappeared, but portal   
biliopathy and pylephlebitis of the portal vein and superior mesenteric vein   
had developed. He was successfully treated with antibiotics and endoscopic   
biliary stenting. Our case suggests that once a patient presents with   
duodenal variceal bleeding, physicians should consider treatment options and   
their associated complications, especially when endoscopic sclerotherapy is   
planned. Additionally, when patients present with fever, jaundice, and   
abdominal pain after endoscopic sclerotherapy, septic thrombophlebitis and   
symptomatic portal biliopathy should be considered. Treatment with   
antibiotic therapy and endoscopic biliary decompression may relieve the   
patient's symptoms; however, anticoagulation therapy may not help in   
decreasing thrombosis in the portal vein and preventing collateral   
extension.  
  
RECORD 27  
Diagnosis and treatment of portal vein thrombosis after splenectomy and   
gastroesophageal devascularization  
Xue S. Zhang Q. Liu J. Wang P.-S. Chen G.  
World Chinese Journal of Digestology (2016) 24:29 (4063-4069). Date of   
Publication: 18 Oct 2016  
  
Splenectomy and gastroesophageal devascularisation is the most common   
clinical treatment for upper gastrointestinal bleeding in patients with   
portal hypertension. Its advantages include exact treatment and little   
impact on liver function. However, due to the postoperative high blood   
coagulation state and hemodynamic changes, it greatly increases the   
incidence of portal vein thrombosis (PVT), which causes serious   
complications. Ultrasound, CT and MRI are main methods for the diagnosis of   
PVT. After diagnosis, using anticoagulation, intervention and surgery can   
achieve effective control and treatment. PVT mostly occurs perioperatively   
and therapeutic effects are therefore limited, so perioperative PVT   
prevention is particularly important. It is recommended that anticoagulation   
drugs be given preoperatively to prevent PVT formation, injury to the   
vascular endothelium of the portal vein system be avoided intraoperatively   
to reduce the formation of spleen vein stump and stabilize postoperative   
portal vein blood flow dynamics, and ultrasound be performed postoperatively   
to achieve early diagnosis and treatment.  
  
RECORD 28  
Incidence and clinical presentation of portal vein thrombosis in cirrhotic   
patients  
Cagin Y.F. Atayan Y. Erdogan M.A. Dagtekin F. Colak C.  
Hepatobiliary and Pancreatic Diseases International (2016) 15:5 (499-503).   
Date of Publication: 15 Oct 2016  
  
Background portal vein thrombosis (PVT) is due to many risk factors, but its   
pathogenesis is still not clearly understood. To identify the risk factors   
for PVT, we analyzed the clinical characteristics and complications   
associated with PVT in cirrhotic patients. Methods We studied patients with   
liver cirrhosis who were admitted to our unit from April 2009 to December   
2014. The patients were divided into the PVT and non-PVT groups, and were   
compared by variables including gender, age, the etiology of cirrhosis,   
stage of cirrhosis, complications, imaging, and treatment. Results PVT was   
found in 45 (9.8%) of 461 cirrhotic patients admitted to our hospital. Most   
patients (45.9%) had hepatitis B virus (HBV)-related cirrhosis, with a   
similar distribution of etiologies between the groups. However, there was no   
positive relationship between PVT and etiologies of cirrhosis. Most patients   
(71.5%) were in the stage of hepatic decompensation. No statistically   
significant differences were found in complications including esophageal   
varices, ascites, and hepatic encephalopathy between the groups. However,   
there was a significant positive correlation between hepatocellular   
carcinoma (HCC) and PVT (P<0.01). In 30 patients with PVT, thrombosis   
occurred in the portal vein and/or portal branches, 37.8% were diagnosed on   
ultrasound. Conclusions The incidence of PVT was 9.8%, mainly in patients   
with HBV-related cirrhosis. The development of PVT was associated with the   
severity of liver disease and HCC.  
  
RECORD 29  
Evaluation of the anticoagulant effect and timing of the concomitant use of   
S-1 and warfarin  
Suzuki S. Ikegawa K. Yamamoto K. Saito S.  
Journal of International Medical Research (2016) 44:5 (1123-1130). Date of   
Publication: 1 Oct 2016  
  
Objectives: To evaluate the effects of the timing of warfarin (WF)   
administration in patients with gastric cancer who received S-1 oral   
chemotherapy. Methods: This retrospective chart review collected patient   
data including the prothrombin time international normalized ratio (PT-INR).   
Patients were categorized into three groups based on the timing of WF   
administration in relation to S-1 oral chemotherapy: group A patients   
received WF before S-1 chemotherapy; group B patients started WF during S-1   
chemotherapy; and group C patients started WF after completing S-1   
chemotherapy. Results: A total of 21 patients with gastric cancer were   
included in the study; group A (n = 8), group B (n = 10) and group C (n =   
3). Seven patients (88%) in group A, seven (70%) in group B and all of the   
patients (100%) in group C had >2.5 PT-INR. There was no significant   
difference in the time-to-exceed 2.5 PT-INR between groups A and B.   
Conclusions: These findings suggest that the timing of WF use in relation to   
S-1 chemotherapy might not be an important factor for PT-INR, although the   
low patient numbers included in the study should be taken into   
consideration.  
  
RECORD 30  
Early prophylactic anticoagulation for portal vein system thrombosis after   
splenectomy: A systematic review and meta-analysis  
Zhang N. Yao Y. Xue W. Wu S.  
Biomedical Reports (2016) 5:4 (483-490). Date of Publication: 1 Oct 2016  
  
A systematic review and meta-analysis were conducted to evaluate the   
efficacy and safety of early prophylactic anticoagulation for the prevention   
of portal vein system thrombosis (PVST) after splenectomy. A systematic   
search of the Pub Med, EMBASE, Springer and Cochrane Library databases was   
performed to identify studies comparing the outcomes in patients receiving   
or not receiving regular prophylactic anticoagulation after splenectomy. The   
quality of the included studies was assessed using the Jadad Score and the   
Newcastle-Ottawa Scale. Heterogeneity was evaluated using the χ(2) and I(2)   
tests. The parameters that were analyzed included the incidence of PVST and   
anticoagulation-associated complications. A total of seven studies qualified   
for the review, involving 383 and 283 patients receiving or not receiving   
regular prophylactic anticoagulation, respectively. The incidence of PVST   
was significantly reduced with an odds ratio (OR) of 0.31 [95% confidence   
interval (CI), 0.21-0.46; P<0.00001] in the regular prophylactic   
anticoagulation group compared with the control group. No difference in the   
incidence of anticoagulation-associated complications was identified between   
the two groups (OR=0.60, 95% CI, 0.23-1.56; P=0.30). Early prophylactic   
anticoagulation was associated with a reduced incidence of PVST, although it   
was not associated with the incidence of anticoagulation-associated   
complications. These results indicate that prophylactic anticoagulation   
could be safely administered after splenectomy, even to cirrhotic patients.  
  
RECORD 31  
Mesenteric thrombophlebitis: An unusual cause of abdominal pain  
Amjad W. Malik S. Sohail U.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S616-S617).   
Date of Publication: 1 Oct 2016  
  
Introduction: Thrombophlebitis in mesenteric and portal vein is a rare   
complication of intra-abdominal inflammatory process. The condition has high   
mortality and morbidity. We present an unusual case of a patient presenting   
with features of acute abdomen and was found to have acute mesenteric   
thrombophlebitis. Case Presentation: A 72 year old female with history of   
diabetes mellitus and hypertension presented to hospital with complains of   
left lower quadrant pain and fever for seven days. The pain was associated   
with anorexia and malaise. No symptoms of vomiting, hematochezia or bowel   
habit changes. No family history of clotting disorder. On presentation her   
vitals were unremarkable except low grade fever and left lower quadrant   
tenderness. Investigations showed WBC 10.7 thousand/ml. CT scan of abdomen/   
pelvis showed thrombosis of mesenteric vein extending from the proximal   
sigmoid colon to a tributary of the superior mesenteric vein along with mild   
inflammatory changes surrounding the thrombosed vein. There was colonic   
diverticulosis without diverticulitis. Blood cultures were negative. Patient   
was treated with flagyl and ciprofloxacin. She was started on   
anticoagulation with IV heparin and discharged on oral rivaroxaban.   
Discussion: Infected thrombosis of portal vein and its tributaries is called   
pylephlebitis. It is most commonly caused by diverticulitis, appendicitis,   
inflammatory bowel disease and pancreatitis. Most common symptoms include   
fever and abdominal pain. CT scan is the modality of choice to diagnose the   
condition. In our patient there were no signs of diverticulitis on CT scan   
but patient symptoms and presence of diverticuli in sigmoid region suggest   
patient recently developed diverticulitis which resolved by time of   
presentation to hospital. Most common organism causing pylephlebitis include   
bactroides fragalis, followed by E. coli, Strep viridans and klebsiella   
pneumonia. The patients should be treated with empiric broad spectrum   
antibiotics. anticoagulation improves long term outcome of pylephlebitis in   
terms of lower mortality and development of portal hypertension and septic   
embolization to liver. The duration of anticoagulation is variable but some   
studies suggest anticoagulation for 2 months. Conclusion: Mesenteric vein   
thrombophlebitis is rare but fatal complication of intra-abdominal   
inflammatory process and usually presents with nonspecific symptoms. Early   
diagnosis and prompt treatment can prevent long term complications.  
  
RECORD 32  
PNH: A rare hematological disease with profound implications for a   
gastroenterologist  
Sunkara T. Parvataneni S. Ajdir N. Vigoda I. Fulger I. Gaduputi V.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S908). Date of   
Publication: 1 Oct 2016  
  
Paroxysmal Nocturnal Hemoglobinuria (PNH) is a rare thrombophilic and   
hematopoietic stem cell disorder with an annual incidence rate of as low as   
1-2 cases per million. About 16% of these patients present with visceral   
vein thrombosis which is the most common cause of mortality. We here present   
an extremely rare case of a young man presenting with extensive thrombosis   
of multiple visceral veins from PNH. A 36-year-old Hispanic man with no   
significant medical history presented to the emergency department with   
diffuse abdominal pain of four days duration. The patient also reported   
weight loss of 5lbs over the preceding one month. Initial laboratory tests   
revealed transaminitis; and pancytopenia with reticulocytosis, increased   
serum lactate dehydrogenase (LDH) and decreased serum haptoglobin levels.   
The patient was found to have elevated urobilinogen in urine pointing   
towards intravascular hemolysis. A computerized tomography (CT) scan   
(Figure-1) of the abdomen demonstrated extensive portal vein thrombosis   
(PVT), superior mesenteric vein thrombosis and bilateral renal vein   
thrombosis. Flow cytometry and bone marrow biopsy (Figure-2) confirmed the   
diagnosis of PNH. The patient underwent esophagogastroduodenoscopy for   
screening esophageal varices. No varices were found. The patient was started   
on anticoagulation and Eculizumab. PNH caused by mutation of the PIG-A gene,   
is characterized by uncontrolled complement activity with decreased CD-55   
and 59 levels leading to- intravascular hemolysis, thrombosis, and bone   
marrow failure. Patients with PNH often present with hemoglobinuria,   
abdominal pain, fever, headache, and fatigue. While hepatic and cerebral   
veins are the most common sites of thrombosis, PVT is extremely rare with   
only about 12 cases reported in the literature. Sparing of hepatic vein with   
involvement of portal vein and mesenteric veins makes this case highly   
unusual. A gastroenterologist must be keenly aware and consider the   
diagnosis of PNH in any young patient presenting with abdominal pain and   
hemolysis with abdominal visceral vein thrombosis, especially in the absence   
of underlying cirrhosis. It is imperative that these patients are started on   
anticoagulation at the earliest given the high mortality. Eculizumab is a   
monoclonal antibody used in the secondary prevention of disease known to act   
by inhibiting the activation of complement cascade system. Allogeneic   
hemopoietic stem cell transplantation is considered for refractory cases.   
(Figure presented).  
  
RECORD 33  
Intrahepatic venous obstructions: A primary event in the development of   
hepatopulmonary syndrome ?  
Lejealle C. Paradis V. Bruno O. Francoz C. Soubrane O. Lebrec D. Valla D.C.   
Vilgrain V. Durand F. Rautou P.-E.  
Hepatology (2016) 63:1 Supplement 1 (49A). Date of Publication: 1 Oct 2016  
  
Background and aims: Hepatopulmonary syndrome (HPS) is characterized by   
hypoxemia and intrapulmonary vascular dilatations in patients with liver   
disease. The pathogenesis of HPS is poorly understood. Liver changes   
associated with HPS have not been studied. The aim of this study was to   
describe imaging and pathology changes associated with HPS. Patients and   
methods: We performed a monocentric retrospective case-control study. We   
included all patients having undergone a liver transplantation assessment   
between 1997 and 2015 with a pretransplantation diagnosis of alcoholic,   
viral or cryptogenetic cirrhosis associated with HPS, defined as PaO(4) ≤ 70   
mm Hg and a “positive” contrast-enhanced echocardiography. Each case was   
matched for age, cause and severity of the liver disease to 3 controls. All   
controls had PaO(4) ≥ 85 mm Hg on room air. Expert radiologist and   
pathologist, unaware of clinical and laboratory data, reviewed   
pretransplantation thoracic and abdominal imaging (contrast-enhanced MDCT   
and Doppler ultrasound) as well as explanted livers, according to predefined   
questionnaires. Results: 21 CT-scans and 19 explanted livers from patients   
with HPS were compared to 63 CT-scans and 57 livers from controls,   
respectively. Age, cause and severity of cirrhosis were similar between both   
groups. At imaging, compared with controls, patients with HPS more   
frequently had abnormal intrahepatic portal vein branches (i.e. reduced in   
caliber and/or not visible) (24% vs. 60%; p=0.03), stagnant or hepafugal   
portal blood flow (27% vs. 57%; p=0.05) and large portosystemic collaterals   
(25% vs. 65%; p=0.01), including larger paraumbilical vein diameter (2.0 vs.   
4.5 mm; p=0.01). Hepatic artery diameter was also larger (5.6 vs. 7.0 mm;   
p=0.001). At pathology, compared with controls, patients with HPS more   
frequently had liver parenchymal extinction (28% vs. 53%; p=0.05),   
incomplete septal cirrhosis (2% vs. 16%; p=0.046), intrahepatic portal vein   
thrombosis (12% vs. 47%; p=0.001), thickening or obstruction of   
centrilobular veins (40% vs. 63%; p=0.025), sinusoidal dilatation (44% vs.   
74%; p=0.048) and vascular proliferation in fibrous bands (72% vs. 95%;   
p=0.001). Conclusion: HPS is associated with imaging and pathology evidence   
of small portal vein obstructions, and with increased portosystemic shunting   
and larger hepatic artery. This results suggest that intrahepatic portal   
vein obstructions lead to the release by the ischemic liver of   
pro-angiogenic/vasodilatatory mediators responsible for the intrapulmonary   
vascular dilatations characteristic for HPS. anticoagulation might thus be   
useful in HPS patients.  
  
RECORD 34  
Recurrent hepatic artery and portal vein thrombosis leading to graft loss   
after liver transplantation in patient with antiphospholipid syndrome  
Sobotka L. Li F. Hanje A.J.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S869-S870).   
Date of Publication: 1 Oct 2016  
  
Case Description: A 60 year old male with a past medical history of   
decompensated cirrhosis secondary to alcohol abuse underwent successful   
orthotopic liver transplant (OLT). His past medical history was notable for   
deep venous thromboses (DVT) and positive lupus anticoagulant; however he   
was not on chronic anticoagulation due to history of variceal bleeding. On   
post-operative day 3, his transaminases and INR acutely increased: ALT   
increased from 114 to 1419, ALT increased from 182 to 2222, and INR   
increased from 1.4 to 4.7. A liver doppler showed a grossly patent portal   
vein (PV) with elevated flow velocity but no detectable flow in the main   
hepatic artery (HA). The patient was emergently taken to the operating room   
for an explorative laparotomy with thrombectomy of the HA and PV. Following   
thrombectomy, intraoperative ultrasound confirmed patent blood flow in both   
the HA and PV. Unfortunately, the patient's labs continued to worsen with   
ALT peaking at 3080, AST to 2066, total bilirubin 5.5, and INR 6.4. Repeat   
liver doppler was concerning for recurrent thrombosis. Figure 1 depicts the   
left HA doppler and Figure 2 shows the right HA doppler. The patient was   
emergently re-listed for transplant as status 1A and underwent repeat OLT.   
Notably, the main HA and PV in the initial graft were found to be   
re-thrombosed. Due to concern for antiphospholipid syndrome, the patient was   
continued on heparin drip but also started on aspirin post-operatively.   
Serologic testing for lupus anticoagulant was positive and anti-cardiolipin   
antibody IgG was weakly positive. The patient's remaining hospital course   
was uncomplicated and he was transitioned to warfarin with an INR goal of   
2.5 to 3.0 and continued on aspirin. At 1-year post-transplant follow-up,   
the patient was doing well and still maintained on aspirin and warfarin.   
Discussion: HA thrombosis (HAT) is a major cause of graft loss and mortality   
following OLT. This case illustrates a rare instance of recurrent HA and PV   
thrombosis following OLT. When recurrent HAT occurs, screening for an   
undergoing hypercoagulable condition is indicated. Based on the patient's   
clinical and laboratory evaluation, he does meet diagnostic criteria for   
antiphospholipid syndrome. He did have additional risk factor for HAT   
including negative recipient CMV status in the recipient. He was able to be   
managed with aspirin and warfarin for anticoagulation following his second   
OLT without evidence of recurrent thrombosis. (Figure presented).  
  
RECORD 35  
Acute liver failure as a first manifestation of polycythemia vera (PCV)  
Kaddourah O. Alba L. Ghanimeh M.A. Shobassy M. Doran S.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S911). Date of   
Publication: 1 Oct 2016  
  
This is a case of extensive vein thrombosis including Budd Chiari syndrome   
(BCS) that led to acute liver failure and diagnosis of Polycythemia Vera. It   
is important to recognize hypercoagulable conditions in patients presenting   
with BCS. Polycythemia Vera (PCV) is increasingly reported as a culprit in   
BCS patients. 71-year-old female with recent history of right Internal   
jugular (IJ) thrombosis, brachiocephalic thrombosis. She presented with   
two-week history of worsening abdominal swelling, peripheral edema. Patient   
was diagnosed previously with right IJ thrombus after she presented with   
neck pain one month ago. Hypercoagulable work-up was nonsignificant then.   
She was started on anticoagulation with Warfarin. CT scan of her abdomen in   
clinic follow-ups showed small ascites and multiple liver masses. MRI   
abdomen was suggestive of Budd Chiari syndrome. Patient presents now to our   
care with worsening ascites, impending acute liver failure.   
Multidisciplinary care initiated with Hepatology, Hematology/oncology and   
radiology. JAK2 V617F mutation was sent given thrombosis with high   
hematocrit and came back positive. Her course in hospital then included   
interventions to relieve obstruction via angioplasty by radiology,   
anticoagulation and management of acute liver failure. Phlebotomies were   
performed to keep hematocrit within limits set by hematologist. Patient   
deemed not a candidate for liver transplant. Management plan was to continue   
on anticoagulation with regular phlebotomies. Budd Chiari syndrome (BCS)   
characterized by thrombosis of hepatic vein and occasionally supra-hepatic   
part of Inferior Vena Cava (IVC). This syndrome often occurs in   
hypercoagulable states especially when oral contraceptives are on board.   
Polycythemia Vera, described as clonal proliferation of myeloid cells   
distinguished by elevated red cell mass, has been reported in literature as   
a cause of BCS. Venous thrombosis is not infrequent in PCV. Studies showing   
prevalence of JAK2 activation in BCS. While primary myeloproliferative   
diseases were leading causes of portal and hepatic vein thromboses in other   
studies. This case sheds the light on how drastic PCV can present. Acute   
liver failure caused by BCS might warrant work up for hypercoagulable status   
and PCV. Especially that interventions initiated further on can decrease the   
risk of recurrence once we know the cause.  
  
RECORD 36  
An uncommon cause of chronic portal vein thrombosis, large varices, and   
massive splenomegaly  
Bhalla R. Keaveny A. Harnois D.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S1356). Date   
of Publication: 1 Oct 2016  
  
A 68-year-old female presents to hepatology for further management of   
chronic portal vein thrombosis (PVT). She was diagnosed with mesenteric vein   
thrombosis in 1995 after she developed acute onset of severe abdominal pain.   
She required exploratory laparotomy at which time the diagnosis was made and   
was treated with warfarin for six months. She underwent hypercoagulable   
workup which was negative. In 2003, she was noted to have extension of the   
superior mesenteric vein (SMV) thrombus into the portal vein. She was   
restarted on warfarin which she remains on. She has a history of esophageal   
and fundal varices on propranolol 600 mg daily. The varices were never   
treated endoscopically and she denies history of gastrointestinal bleeding.   
She has not had prior liver biopsy. Family history is negative for venous   
thromboembolism. Social history is negative for alcohol use. She has two   
healthy children and no history of miscarriage. Review of systems is   
negative for jaundice, icterus, pruritus, encephalopathy, ascites, or   
peripheral edema. On exam, abdomen is distended but soft and nontender.   
Splenomegaly is present and there is no obvious ascites. There are no   
chronic liver disease stigmata. Labs are significant for a platelet count of   
80, normal liver function tests, normal albumin, and elevated INR in the   
setting of warfarin. MRI of liver with elastography reveals cavernous   
transformation of the intra- and extrahepatic portal veins with a prominent   
system of pericholecystic collaterals. Massive splenomegaly of 25 cm length   
is noted and the SMV is patent. There are no suspicious liver lesions. Stage   
III to IV liver fibrosis is noted. Transjugular liver biopsy shows   
extramedullary hematopoiesis without evidence of cirrhosis. Upper endoscopy   
with endoscopic ultrasound reveals grade III esophageal varices and type 1   
gastroesophageal varices without stigmata of recent bleeding. No further   
intervention is recommended by surgery or interventional radiology and her   
beta blockade and anticoagulation are continued. Patient is referred to   
hematology due to liver biopsy finding of extramedullary hematopoiesis. Bone   
marrow biopsy reveals hypocellular bone marrow with marked myelofibrosis and   
molecular analysis is positive for JAK2 V617F mutation, consistent with a   
diagnosis of primary myelofibrosis. This case illustrates the importance of   
keeping in mind myeloproliferative neoplasms as a potential etiology of PVT,   
noncirrhotic portal hypertension, and massive splenomegaly.  
  
RECORD 37  
Transjugular intra-hepatic porto-systemic shunts: A review of current   
practice and future avenues of application  
Karunasena S. Stephens M. Mott N.  
Journal of Medical Imaging and Radiation Oncology (2016) 60 Supplement 1   
(30-31). Date of Publication: 1 Oct 2016  
  
Learning objectives: To present the clinical indications and procedure   
details of transjugular intra-hepatic porto-systemic shunts (TIPS), and   
discuss technique challenges, complications, follow-up and future avenues.   
Background: The TIPS procedure has an established role in managing the   
sequelae of portal hypertension (1). The American Association for the Study   
of Liver Disease recommends TIPS for management of variceal bleeding when   
pharmacologic and endoscopic therapy fails, refractory ascites in patients   
intolerant of repeated drainage, hydrothorax refractory to salt-restriction   
and diuresis, and moderately severe Budd-Chiari syndrome not responsive to   
anticoagulation (2). TIPS has also been shown to improve renal function in   
hepatorenal syndrome, however its indication in this condition is still   
under investigation (3). Although TIPS is not a curative procedure, there is   
an emerging understanding that it can delay mortality and serve as a bridge   
to liver transplant (4). Another potential application is treatment and   
prevention of portal vein thrombosis (PVT), where TIPS functions to maintain   
portal vein flow (5). This is significant because the low-flow state in   
cirrhosis predisposes to PVT, and PVT complicates conventional end-to-end   
portal vein anastomosis in liver transplant (6). Procedure details: The   
procedure initially involves transjugular access of hepatic veins, passage   
of a guide-needle through liver parenchyma into a portal vein branch and   
measurement of the portosystemic gradient. Following balloon-dilatation of   
the tract created, a sheath is advanced over the guide-needle and the stent   
deployed. Serial dilatations are performed until satisfactory decompression   
is achieved and adjunct variceal embolization performed if required. (7) The   
most technically challenging step is portal vein access, which can be   
further complicated by anatomical variation (8). Prior evaluation of the   
vascular beds involved with cross-sectional imaging is helpful, and   
ultrasound and wedge CO(2) or contrast portography can aid portal vein   
targeting intra-procedure (9). Acute complications include intra-peritoneal   
haemorrhage, acute liver failure secondary to ischemia from portal flow   
diversion, cardiac failure from increased pre-load and contrast nephropathy   
(10). The most commonly discussed long-term complication is hepatic   
encephalopathy as portal blood is shunted into the systemic circulation (7).   
Since the introduction of poly-tetraflouroethylene stents, stenosis and   
thrombosis are rare (5). Doppler ultrasound is performed post-procedure to   
assess shunt flow, and three-monthly for surveillance. Venography is   
performed as indicated. (10) Conclusion: TIPS, whilst not a curative   
procedure, is a valued intervention in managing portal hypertension. It   
shows promise in becoming an accepted means of widening the window for liver   
transplant and management of PVT in transplant candidates.  
  
RECORD 38  
portal vein thrombosis as a complication of liver biopsy  
Levin N. Brown C. Zucker S.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S942). Date of   
Publication: 1 Oct 2016  
  
Liver biopsy and histologic examination remain the gold standard for   
evaluation of elevated liver tests. Reported complications of liver biopsy   
include pain, bleeding, infection, bile leak and rarely hepatic   
arteriovenous fistula. To the best of our knowledge, this is the first   
reported case of liver biopsy precipitating portal vein thrombosis. A 64   
year-old female with a history of remote cholecystectomy and chronic   
aminotransferase elevation [baseline AST 30-50 IU/L (reference 0-32 IU/L),   
ALT 30-50 IU/L (ref. 0-33 IU/L) and alkaline phosphatase 140-170 IU/L (ref.   
35-105 IU/L)] was admitted for complaints of worsening right upper quadrant   
abdominal pain occurring approximately one week following a percutaneous   
liver biopsy. The procedure involved three biopsies of the right hepatic   
lobe under ultrasound guidance with a 16-gauge instrument. Post-procedure,   
an ultrasound of the liver demonstrated no evidence of free fluid or hepatic   
hematoma. The biopsy revealed mild chronic hepatitis, mild portal chronic   
inflammation with mild interface hepatitis (grade 2/4), mild lobular   
inflammation with no hepatocellular death (grade 1/4) and mildly enlarged   
fibrotic portal tracts with no periportal or portal-portal septae,   
architectural distortion or cirrhosis (stage 1/4 per Scheuer). The patient   
denied jaundice, nausea, vomiting, fever, hematemesis or bloody stools.   
Work-up was notable for AST and ALT elevation to 70 IU/L and 89 IU/L   
respectively, alkaline phosphatase of 228 IU/L and normal bilirubin. Vital   
signs were within normal range; her abdominal exam was significant for mild   
tenderness to palpation of the right upper quadrant and a small hematoma   
noted at the anterior axillary line in the 9th rib space. Multiphasic   
abdominal CT was performed, demonstrating a tubular hypodensity within the   
posterior right liver consistent with thrombus in the posterior branch of   
the right portal vein with altered perfusion of the posterior right hepatic   
lobe [Figure 1]. Based on the acuity of pain and location of the portal vein   
thrombosis, we concluded the thrombosis was likely induced by injury to the   
posterior branch of the right portal vein following percutaneous liver   
biopsy. For this reason, the patient did not undergo any hypercoagulability   
testing; anticoagulation was not indicated. After brief observation, her   
labs improved to baseline and her pain resolved. This case demonstrates that   
portal vein thrombosis may be a complication of a percutaneous liver biopsy.   
(Figure Presented).  
  
RECORD 39  
It takes two to make a thing go right: A GI and hematology collaboration to   
diagnose an atypical cause of abdominal pain and GI bleeding  
Mendez V. Bade K.S. Moehlen M.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S978-S979).   
Date of Publication: 1 Oct 2016  
  
A 20-year-old African American man with history of aplastic anemia presented   
as a transfer for evaluation for a bone marrow transplant. GI was consulted   
to evaluate abdominal pain and bloody stool. He initially presented to an   
outside facility with worsening, nonradiating epigastric abdominal pain with   
associated “dark stool” and nausea with nonbloody emesis of one month   
duration. He denied diarrhea, bloody stool or similar episodes. Initial work   
up included a contrasted CT of abdomen and pelvis that showed bowel wall   
thickening in mid-distal duodenum and terminal ileum. A small bowel   
enteroscopy showed a hemorrhagic mass vs. necrotic ulcer with an adherent   
clot in the second/third portion of the duodenum. Biopsies revealed   
“features suggestive of ischemic mucosal injury, negative for dysplasia or   
malignancy.” He was treated supportively with narcotics, stool softeners, a   
proton pump inhibitor and sucralfate. He was transferred to our facility   
after failure to improve. Initial blood work included flow cytometry, which   
revealed a population of PNH clones. With worsening abdominal pain and new   
onset rectal bleeding, there was concern for an ischemic process. A repeat   
CT showed wall thickening in a 14cm segment of the distal ileum and a 5cm   
portion of the jejunum. There was concern for thrombosis in a peripheral   
segment of the right hepatic vein. He was started on eculizumab and a   
heparin drip, with clinical improvement and resolution of rectal bleeding.   
PNH is a rare condition occurring in 1-10 per million people. It typically   
presents with fatigue, jaundice, red urine and hemolysis, and can present   
with complications from thrombosis, such as abdominal pain and stroke. The   
leading cause of death in patients with PNH is thrombosis. Thromboses are   
more often seen in the hepatic, portal and mesenteric veins and may rarely   
cause ulcerations similar to those in our patient. Some patients with PNH   
have an overlap syndrome with bone marrow disorders, such as aplastic   
anemia, as well as other cytopenias and myelodysplastic syndrome. Clinicians   
must have high suspicion to diagnose mesenteric vein thrombosis, which is   
most commonly done with contrasted CT showing bowel wall thickening, with or   
without portal vein thrombus. Treatment is usually anticoagulation in acute   
and subacute cases. anticoagulation is not necessary in chronic thrombosis   
which have progressed to form collaterals with associated portal hypotension   
and varices. (Figure Presented).  
  
RECORD 40  
The seemingly benign abdomen with an underlying insidious pathology: A rare   
presentation of portal vein thrombosis  
Dulaney J. Saline L.C. Powers D.W. Sobrado J.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S1349-S1350).   
Date of Publication: 1 Oct 2016  
  
The portal vein is formed by the superior mesenteric vein and splenic vein.   
Thrombosis within this vessel may occur acutely or chronically over time due   
to hepatic cirrhosis or other pro-thrombotic disorders. While provoking   
agents and initial symptoms are variable, the common pathology involves   
general flow obstruction within the portal vasculature with possible new or   
worsening liver failure, portal hypertension, or intestinal ischemia. Our   
patient is a 61-year-old female with a history of ethanol abuse, who   
presented to the ER with progressive dyspnea and was admitted with   
community-acquired pneumonia. On admission, the patient denied any pain and   
had a benign abdominal exam. Due to an INR of 4.1, the GI service was   
consulted for suspected cirrhosis. An abdominal US was obtained and showed a   
near complete thrombosis of the portal and superior mesenteric veins. This   
was confirmed by an abdominal CT, which also revealed numerous ill-defined   
masses throughout the liver and at the head of the pancreas. An initial   
liver biopsy showed no evidence of malignancy but revealed acute and chronic   
inflammation, microabscess formation, and coagulation necrosis. A second   
liver biopsy corroborated an infectious etiology when 220 mL of perihepatic   
purulent fluid was collected. As the etiology of the hepatic and pancreatic   
lesions was investigated, the proposed mechanism of thrombus formation was   
attributed to the multiple pancreatic lesions compressing and potentiating   
venous stasis of the nearby vasculature. Treatment was geared toward   
anticoagulation initially with full dose lovenox and then with heparin.   
After 2 weeks of treatment, a repeat CT of the abdomen showed no further   
evidence of thrombus within the portal vein and anticoagulation was   
discontinued. This case demonstrates a unique pathologic process and   
etiology of portal vein thrombosis as attributed to venous stasis secondary   
from abscesses within the liver and pancreatic head. Although the patient   
presented with a known history of ethanol abuse, she had no abdominal   
ascites, GI upset, or pain on admission. Coagulopathy was the only aberrant   
factor that prompted investigation of hepatic pathology. While many factors   
are known to promote or exacerbate thrombus formation within the portal   
system, the infectious etiology in this case has been rarely observed within   
the literature and therefore underscores the variable symptomatology of this   
condition and the high clinical suspicion necessary for its detection.  
  
RECORD 41  
Antiphospholipid antibodies associated vascular events are an   
underrecognized cause of morbidity and mortality after liver   
transplantation: Benefit of plasmapheresis and anticoagulation in   
transplanted patients with high thrombotic risk  
Villamil A. Bandi J.C. Nunez F. Mullen E.G. Yamamoto L. De Santibanes E.   
Gadano A.  
Hepatology (2016) 63:1 Supplement 1 (494A). Date of Publication: 1 Oct 2016  
  
Antiphospholipid antibodies (aPL-ab) are frequently present in patients with   
end-stage liver disease and associated with morbidity and graft loss   
post-transplant as a result of vascular thrombosis. Risk is increased in   
patients with pre-OLT aPL-related thrombotic events or high titer   
circulating aPL-ab. Plasmapheresis and immunosuppression have been proposed   
as adequate therapy post-development of vascular complications. Aim: To   
evaluate the impact of pre-OLT plasmapheresis with post-OLT anticoagulation   
in patients transplanted for endstage liver disease with high risk for   
aPL-ab vascular complications. Patients and Methods: Between 2005 and 2015   
321 patients transplanted for end-stage liver disease were screened for   
aPL-ab and lupus anticoagulant activity. 86/321 patients (27%) had increased   
levels of aPL-ab (anticardiolipin IgM and/ or IgG isotypes, anti Beta-2   
glycoprotein) and/or lupus anticoagulant activity. 29/86 fulfilled high   
thrombotic risk criteria and were randomly divided in 2 groups: Group A   
(n=12): patients with standard low dose aspirin ± low weight heparin   
post-OLT. Group B (n=17): patients with 1-2 hours pre-OLT plasmapheresis   
with fresh frozen plasma followed by post OLT anticoagulation for at least 3   
months. Clinical and Doppler US evaluations were performed immediately post   
OLT and at different time-points for the first 6 months. Etiology, severity   
of cirrhosis and immunosuppression did not differ between groups.   
Immunosuppressive regimen included steroids + CyA (n=10) or tacrolimus   
(n=19) ± mycophenolate. Results: 11/12 patients in group A developed aPL-ab   
related complications (cerebrovascular ischemia n=3, humeral thrombosis n=2,   
hepatic artery thrombosis n=1, intestinal ischemia n=1, retinal artery   
thrombosis n=1, portal vein thrombosis, catastrophic antiphospholipid   
syndrome(CAPS) n=4) resulting in grafts loss (n=1), irreversible neurologic   
damage (n=1) and death (n=5). In Group B 3/17 patients developed an   
aPL-associated complication: 2 CAPS and 1 hepatic artery thrombosis   
resulting in 2 deaths. Thrombotic complication rate was 37.9 % vs 10.3 %,   
p<.0001. No differences were observed in the development of CAPS and renal   
microangiopathy. There was a tendency to higher aPL related deaths among   
patients with only standard therapy (17.2 % vs 6.9 %, p.06) Conclusion:   
aPL-ab are a significant under-recognized cause of thrombotic complications   
and mortality post-OLT. Pre-OLT plasmapheresis with post-OLT anticoagulation   
post-OLT may be an effective strategy to prevent aPL-ab associated vascular   
complications in high risk patients.  
  
RECORD 42  
A risk prediction model for portal vein thrombosis in patients waiting for   
liver transplantation developed using discovery and validation cohorts  
Haseeb A. Stevens S.M. Woller S.C. Horne B.D. Evans R.S. Lloyd J. Charlton   
M.R.  
Hepatology (2016) 63:1 Supplement 1 (126A). Date of Publication: 1 Oct 2016  
  
Purpose: portal vein thrombosis (PVT) in cirrhosis leads to worsening of   
liver disease, poorer clinical outcomes, and potential inoperability at   
liver transplantation (LT). Prevalence of PVT in patients undergoing   
transplantation or evaluation for transplantation is between 5% to 16%. The   
purpose of this study is to develop a PVT risk prediction model in cirrhotic   
patients awaiting LT. Methods: An analysis of patients waitlisted for liver   
transplantation and undergoing serial cross-sectional evaluation of portal   
and hepatic vessels from 12/1987 to 5/2014 was performed in the in a large,   
prospectively constructed electronic data warehouse. A total of 621 patients   
were identified with baseline assessment and subsequent imaging. Descriptive   
statistics were computed. Patients were divided randomly into derivation and   
validation populations using a 70% versus 30% split. Cox regression modeling   
was used in the derivation population to determine the association of risk   
factors with the outcome of portal vein thrombosis. Using the   
beta-coefficients for each variable from the final Cox regression model, a   
risk score for PVT was developed. Testing of the risk score was performed in   
the independent validation sample using the 30% of patients initially held   
aside. Results: A total of 63 patients developed PVT while waiting for LT.   
Hepatic encephalopathy (HR 2.74), bacterial peritonitis (HR 2.58),   
esophageal or gastric varices (HR 2.88) and a bilirubin >4.5 mg/dL (HR 3.87)   
at the time of listing were associated with subsequent development of PVT.   
We developed a PVT risk score using these four variables (Table 1). A PVT   
risk score >3 carried a hazard ratio of 15 for developing PVT. Survival   
curves were created for the proposed score. Conclusion: We have developed a   
simple, novel PVT risk score with a high predictivity for development of PVT   
in cirrhotic patients. PVT score can be used to categorize patients into   
high-risk and low-risk categories. Risk stratification and possible   
prophylactic therapy, e.g. anticoagulation, might be considered in high risk   
groups to improve outcomes of LT. (Table Presented).  
  
RECORD 43  
Pylephlebitis: A rare cause of abdominal pain with deadly consequences  
Sellers E. Mcvey M. Dantzler T.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S1372). Date   
of Publication: 1 Oct 2016  
  
Pylephlebitis is regarded as a septic thrombophlebitis of the portal vein or   
one of its tributaries and is commonly associated with intra-abdominal   
infection. Diagnosis can be difficult as its presentation is commonly   
associated with non-specific symptoms such as generalized abdominal pain and   
fever. Given its high morbidity and mortality rates and its low incidence, a   
high index of suspicion is needed to make the diagnosis and avoid a delay in   
treatment. A 39-year-old female with a medical history notable for an   
episode of pancreatitis complicated by pseudocyst and pancreatic duct leak   
requiring endoscopic transpapillary drainage initially presented to an   
outside hospital with progressively worsening right upper quadrant abdominal   
pain and malaise. A CT of the abdomen was performed and was notable for   
possible pancreatic cancer involving the head of the pancreas and metastasis   
to the liver. She was referred to our institution for endoscopic ultrasound   
with fine needle aspiration (FNA) for further evaluation. FNA of the lesions   
was notable for likely hepatic abscess, and after the procedure, the patient   
experienced a temperature of 103.1°, heart rate of 121 and rigors. She was   
subsequently admitted for further evaluation. MRI was performed for   
characterization of the pancreas and hepatic lesions and was notable for   
extensive portal vein thrombosis to the level of portal confluence with   
findings suggestive of superimposed thrombophlebitis. Percutaneous drainage   
of one the hepatic abscesses and blood cultures isolated peptostreptococcus   
micros. Ultimately, the patient was treated with a fourweek course of   
ertapenum. Due to the extensive clot burden, the patient was also placed on   
a heparin drip with transition over to warfarin by the time of discharge.   
The incidence of pylephlebitis has been reportedly as low as 0.6 % with a   
mortality upwards of 32 %. As its presentation is related to relatively   
nonspecific symptoms (abdominal pain, fevers, nausea), diagnosis can be   
difficult. The absence of a high index of suspicion can lead to a delay in   
diagnosis. Blood cultures with enteric organisms can assist with raising the   
index of suspicion and guiding antibiotic therapy, but CT and ultrasound   
should be used to appropriately visualize the portal vasculature.   
Ultimately, patients should be treated with a parenteral course of   
antibiotics and the role of anticoagulation should be discussed on a   
case-by-case basis as there is no clear consensus on its role. (Figure   
Presented).  
  
RECORD 44  
A rare case of liver abscess due to gemella species and portal vein   
thrombosis in a healthy individual  
Polavarapu A.D. Gumaste V. Mulrooney S. Tyagi P. Khalil A.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S928-S929).   
Date of Publication: 1 Oct 2016  
  
Introduction: In immunocompetent individuals, pyogenic liver abscess is   
often cryptogenic or develops secondary to ascending cholangitis,   
diverticulitis, appendicitis or systemic infections, and is usually   
polymicrobial. Risk factors include diabetes mellitus, hepatobiliary or   
pancreatic disease and liver transplant. Here we report a case of pyogenic   
liver abscess caused by Gemella species, which are facultative anaerobic   
gram-positive cocci primarily found in mucous membranes of humans. Gemella   
infrequently causes endocarditis, cerebral abscess and empyema but is not   
known to cause liver abscess. There are only 8 published cases. Case: A 59   
Year old female with no prior medical or travel history presented with 2   
weeks of generalized weakness, nausea, anorexia and cough with fevers and   
chills. On examination, she was febrile and ill-appearing with a soft, non   
tender abdomen. Lab results: WBC 27 K/mm3; liver enzymes: AST 49 U/L, ALT 54   
U/L, ALP 62 U/L; T. bili 0.3 mg/dl. Xray was unremarkable. Abdominal CT scan   
revealed an 11×6 cm multiloculated liver abscess with associated thrombosis   
of right portal vein and sigmoid diverticulosis. Initial therapy consisted   
of IV carbapenem and anticoagulation. HIV, viral hepatitis and amoebic   
antibodies were negative. Blood culture was negative and echocardiogram was   
normal. On CT-directed aspiration of the liver lesion, 31 cc of thick green   
fluid was evacuated which revealed acute inflammatory cells with necrosis   
and no malignant cell. Fluid culture grew Gemella species. Intravenous   
vancomycin was added. Although abscess decreased in size, high fevers   
persisted, prompting pigtail catheter placement. She improved clinically and   
was discharged with intravenous vancomycin and ertapenem for 4 weeks. Repeat   
CT scan after one month showed resolution of liver abscess but revealed a   
new 1.2x1.1 cm chronic intramural abscess in lateral wall of sigmoid colon.   
She is scheduled for colonoscopy later. Conclusion: This case illustrates   
the potential of Gemella species to cause liver abscess, including large   
abscesses which may require catheter drainage and prolonged antibiotics, in   
an immunocompetent noncirrhotic individual with diverticulosis. As Gemella   
is a commensal in the gut, it likely translocated from chronic sigmoid   
diverticulosis to the liver via portal vein, causing abscess formation. We   
successfully treated with vancomycin and carbapenem. Data is limited   
regarding management, given the rarity of this organism. (Figure Presented).  
  
RECORD 45  
A rare cause of diarrhea in a Crohn's patient: Pancreatic insufficiency due   
to portal vein thrombosis  
Nguyen A.A. Kesar V. Shamah S. Swaminath A.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S831). Date of   
Publication: 1 Oct 2016  
  
Inflammatory bowel disease (IBD) is an inflammatory state with an increased   
risk of venous thromboembolism (VTE). Patients with IBD have a three-fold   
higher risk of VTE compared to patients without IBD. This may occur due to   
disequilibrium between procoagulant and anticoagulant factors, bacterial   
translocation leading to portal pylephlebitis, or post-operative state.   
Herein, we present a case of Crohn's disease with portal vein thrombosis   
(PVT) causing exocrine pancreatic insufficiency (EPI). A 44 year-old man   
with fistulizing and fibrostenosing Crohn's Disease since age 12 presented   
with more than 10 bowel movements daily for 1 month. He was previously   
treated with adalimumab for 4 years without improvement in subjective   
symptoms, thus the patient self-discontinued medication at age 40. One year   
prior to presentation, the patient had severe abdominal pain and was found   
to have a peri-anal fistula and mid-transverse colonic stricture. He   
underwent an elective resection of 14.5 cm of his transverse colon with   
ileo-colonic anastomosis. On post-operative imaging, he was found to have   
PVT and completed 3 months of lovenox. Three months post-operative, he   
developed frequent non-bloody post-prandial diarrhea with early satiety and   
vague abdominal discomfort. He was tried on cholestyramine with minimal   
improvement. An EGD and colonoscopy to evaluate the diarrhea were   
non-diagnostic. He was sent for a CT scan to rule out small bowel to colonic   
fistula and was found to have extension of his portal vein thrombus from the   
left hepatic vein to the splenic vein. In addition, the CT scan showed   
atrophy of hepatic segments 6 and 7, but no evidence of pancreatitis. CBC   
and serum tryptase were unremarkable. He had an elevated AST, ALT, and   
alkaline phosphatase. He was started on pancrelipase and long-term   
anticoagulation with rivaroxaban with rapid improvement in symptoms   
consistent with EPI due to extension of the PVT into venous drainage of the   
pancreas. Active IBD is a risk factor for VTE. Here we describe a rare case   
of persistent diarrhea in a patient with Crohn's disease due to EPI   
associated with PVT. This has rarely been described in the literature, but   
the pathophysiology is thought to be due to PVT leading to obstruction of   
pancreatic venous drainage, causing pancreatic duct atrophy and exocrine   
insufficiency. Thus, in IBD patients with a history of PVT and persistent   
diarrhea it is important to consider EPI as an etiology.  
  
RECORD 46  
Streptococcal hepatic abscess: A rare complication of severe necrotizing   
pancreatitis  
Khurana S. Rahimi E. Nevah Rubin M.I. Kaila V. Guha S. Ashary N. Ertan A.   
Thosani N.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S591). Date of   
Publication: 1 Oct 2016  
  
A 60-year-old female was admitted with severe pancreatitis of unknown   
etiology, with symptoms of epigastric abdominal pain, and nausea with   
vomiting. Contrast enhanced computed tomography (CT) scan showed large areas   
of non-enhancement of the pancreatic body, and extensive peripancreatic free   
fluid and stranding in the regions of the pancreatic head and tail   
consistent with acute necrotizing pancreatitis. She was also noted to have   
non-occluding thrombi of the superior mesenteric vein that extended into the   
portal vein and was started on long-term anticoagulation treatment with oral   
warfarin. Her clinical course was complicated by the development of a large   
pancreatic pseudocyst (11 x 13 x 23 cm on CT) that was managed successfully   
by endoscopic cystogastrostomy [Figure 1]. Unfortunately, after initial good   
recovery, she was hospitalized 3 months later with ascites and failure to   
thrive. A repeat contrast enhanced CT scan showed a large multi-loculated   
7.4 x 7.8 x 9.2 cm mass in the liver involving caudate lobe, and right lobe   
of liver concerning for hepatocellular carcinoma [Figure 2]. On endoscopic   
ultrasound a large hypoechoic, heterogeneous mass was noted in the liver.   
Fine needle aspiration using a 25G needle was performed and on site   
evaluation by cytopathology showed extensive necrosis and acute inflammation   
concerning for hepatic abscess. Additional FNA was performed for culture and   
sensitivity that revealed gram-positive bacteria in clusters (Group F   
streptococcus). Patient was treated with longterm intravenous vancomycin.   
She had near complete resolution of liver abscess at 6-week follow-up.   
Pancreatic pseudocyst and splenic vein thrombosis are the known   
complications of severe pancreatitis. However, the development of a liver   
abscess possibly following pylephlebitis in the background setting of acute   
necrotizing pancreatitis is a rare entity with only a few cases reported in   
the literature. Our patient developed severe hepatic abscess and ascites   
mimicking hepatocellular cancer. Likely etiology of this abscess was   
superior mesenteric vein and portal vein thrombosis. This case highlights   
the rare complication of severe necrotizing pancreatitis and role of EUS   
guided tissue acquisition in diagnosis and management of hepatic abscess.   
(Figure Presented).  
  
RECORD 47  
Budd-chiari syndrome and portal vein thrombosis in crohn's disease  
Ghouri Y.A. Shenoy A.V. Stevenson H.L. Merwat S.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S815-S816).   
Date of Publication: 1 Oct 2016  
  
Introduction: Untreated Crohn's disease (CD) is a prothrombotic state   
associated with venous thromboembolism and can present with portal vein   
thrombosis, deep venous thrombosis and pulmonary embolism (PE). There are a   
handful reported cases of CD presenting as hepatic vein thrombosis or   
Budd-Chiari syndrome. Case description: A 27 year old male presented with   
hematochezia, abdominal pain, anasarca with ascites, fatigue and   
hepatomegaly. A year prior to presentation he developed hematochezia and was   
seen at a different hospital where he underwent colonoscopy that was   
suggestive of inflammatory bowel disease but was lost to follow up and   
remained untreated and continued to have intermittent hematochezia and   
abdominal pain. On admission his Hb was 5.7 g/dL with elevated ALT (83 U/L)   
and AST (81 U/L) and albumin of 2.1 g/dL. Stool studies were negative for C   
difficile toxins, ova & parasites, enteric cultures but positive for   
leukocytes. Colonoscopy was performed that showed terminal ileitis and   
pancolitis with rectal pseudopolyp formation. EGD demonstrated duodenal   
ulcerations and inflammation of pylorus which on biopsy showed   
non-necrotizing granuloma formation that was consistent with a diagnosis of   
CD (Figure 1). His hypoalbuminemia was suspected to be due to protein-losing   
enteropathy from active enterocolitis. Paracentesis of the ascitic fluid   
showed a serum:ascites albumin gradient of >1.4 which suggested portal   
hypertension. CT scan of abdomen demonstrated hepatomegaly with thrombi in   
the inferior vena cava, portal vein and right hepatic vein (Figure 2). Liver   
biopsy showed extensive perivenular sinusoidal dilation, areas of hepatocyte   
atrophy and drop-out, and mild centrizonal fibrosis (Budd-Chiari syndrome;   
Figure 3). He was then anti-coagulated with heparin drip and transitioned to   
apixiban. His anasarca improved with diuretics and CD was treated with   
prednisone and mesalamine at the time of discharge. Discussion: Active CD is   
a prothrombotic state that can lead to Budd-Chiari syndrome and subsequent   
hepatocyte injury as a result of venous congestion. Protein losing   
enteropathy can develop as a result of untreated CD leading to anasarca and   
loss of procoagulant factors which further increases the risk of venous   
thrombosis. Early recognition of thrombotic complications of CD and   
initiation of anticoagulation is recommended to prevent hepatocyte injury   
from venous congestion or fatal complications like development of PE.   
(Figure Presented).  
  
RECORD 48  
Suppurative thrombophlebitis of the portal vein (pylephelbitis) - An ominous   
complication of perforated appendicitis  
Morley G.L. Dilworth M. Bowley D.M.  
Colorectal Disease (2016) 18 Supplement 2 (69). Date of Publication: 1 Oct   
2016  
  
Background: Pylephelbitis is a rare consequence of infective intra-abdominal   
pathology with high mortality. Case report: A 22 year old man was admitted   
with a 10 day history of diarrhoea and vomiting. Examination revealed lower   
abdominal tenderness and septic shock (HR 128, BP 98/57, temperature   
40.1°C). In addition to raised inflammatory markers, admission blood tests   
revealed raised bilirubin 107umol/l. An initial diagnosis of gastroenteritis   
led to management of sepsis with resuscitation measures and ciprofloxacin.   
Surgical advice was sought on day 3 and CT indicated perforated   
appendicitis, plus portal vein thrombosis with air in the superior   
mesenteric vein. Laparoscopic appendicectomy was undertaken, broad spectrum   
antibiotics and therapeutic enoxaparin (1.5 mg/kg) were administered. The   
patient recovered after a stormy course and remained on IV antibiotics for 6   
weeks and warfarin for over 3 months. Discussion: Pylephelbitis develops   
when local sepsis creates a hyper-coagulable state with bacterial   
infiltration of vessels. Localised small vein thrombophlebitis drains to   
larger veins allowing extension of the septic inflammation into the superior   
mesenteric vein and portal vein. Mortality from pylephlebitis remains high,   
despite advances in imaging technology. The mainstay of treatment for   
pylephlebitis is broad spectrum antibiotics and controlling the source of   
infection; the use of anticoagulation is contentious. Conclusions:   
Pylephelbitis is a rare and deadly complication. In the absence of primary   
hepato-biliary disease, deranged liver function should raise suspicions and   
CT imaging is central to the diagnosis. Resuscitation, broad spectrum   
antibiotics and prompt source control are vital to achieve satisfactory   
outcomes.  
  
RECORD 49  
A rare cause of liver failure  
Jiang A. Musleh M.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S887). Date of   
Publication: 1 Oct 2016  
  
Primary hepatic lymphoma is defined as lymphoma that is either confined to   
the liver or has major liver involvement. This is a rare entity that   
constitutes less than 1% of extranodal lymphomas. Presentation can vary from   
being an incidental finding in otherwise asymptomatic patients to   
hepatocellular injury or fulminant hepatic failure. A 74 y.o. Caucasian male   
presented with right upper quadrant abdominal pain of 3 week duration,   
associated with excessive fatigue, and progressive jaundice of his eyes and   
skin. He did not have any past history of alcoholism or hepatitis. He did   
have a history of polycythemia vera and atrial fibrillation with   
anticoagulation on apixaban. On admission he was found to have AST 401 U/L,   
ALT 197 U/L, and alkaline phosphatase 903 U/L, albumin of 3.2 gm/dL and   
total bilirubin of 12.6 mg/dL, of which direct bilirubin was 8.9 mg/dL and   
indirect bilirubin was 3.7 gm/dL, as well as an international normalized   
ratio of 1.3. His Hepatitis A, B, and C, as well as EBV and CMV serologies   
were negative. Anti-mitochondrial antibodies, ceruloplasmin,   
alpha-1-antitrypsin, anti-neutrophil antibody, antismooth muscle antibody   
were all within normal limits. His LDH was elevated at 542 U/L though AFP   
and CEA were normal. His ferritin was also elevated at 554 ng/mL. A   
non-obstructing portal vein thrombosis was seen on duplex ultrasound despite   
the patient being on apixaban. Magnetic resonance imaging of the abdomen   
showed hepatomegaly with a macronodular appearance and multiple T2   
hyperintensities in the hepatic parenchyma. A live biopsy was obtained with   
the specimen showing Diffuse Large B-cell Lymphoma. Positron emission   
tomography scan did not show any lymph node, spleen or other organ   
involvement. He was started on chemotherapy with Cisplain, Etoposide, ArA-C,   
and Rituxan. This patient's clinical presentation closely mimics   
decompensated liver cirrhosis, from physical findings to laboratory values   
and imaging. portal vein thrombosis is likewise a common complication seen   
in cirrhotics. In patients who have additional findings of space-occupying   
liver lesions but normal levels of alpha-fetoprotein and CEA however,   
Primary Non-Hodgkin's Lymphoma of the liver should be considered. Case   
reports of hyperferritinemia have also been reported in conjunction with   
this disease. Liver biopsy is the gold standard for diagnosis, along with   
evidence that it is confined to the liver.  
  
RECORD 50  
An “obscure” presentation of early cirrhosis: Bleeding jejunal varices as   
initial presentation of cryptogenic cirrhosis  
Lewin S. El-Nachef N.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S953). Date of   
Publication: 1 Oct 2016  
  
Introduction: Isolated small intestinal varices are an uncommon   
manifestation of portal hypertension. Although uncommon, small intestinal   
variceal hemorrhage can be life-threatening. We report a case of isolated   
small bowel variceal hemorrhage in a man with previously undiagnosed   
cirrhosis. Case: An 82 year-old man presenting with 5 weeks of melena and   
anemia (hemoglobin 5.7 g/dL) was admitted to the hospital. Two months prior,   
he had undergone coronary artery bypass graft surgery. His post-operative   
course was complicated by new-onset atrial fibrillation; he was started on   
therapeutic anticoagulation with apixaban. His past medical history was   
otherwise notable for a history of chronic kidney disease secondary to   
hypertension requiring kidney transplant fourteen years prior. Inpatient EGD   
was normal. Colonoscopy with examination of ileum was normal except for   
colonic diverticulosis without active or stigmata of recent bleeding. A CT   
of the abdomen and pelvis without contrast revealed no abnormalities of the   
liver or spleen. The patient was instructed to stop apixaban and was   
discharged with stable hemoglobin level of 8.0 g/dL and platelet count   
158,000. He had no further episodes of melena while inpatient. An outpatient   
capsule endoscopy revealed the presence of blue tinged nodules in the distal   
jejunum. Double balloon enteroscopy confirmed the presence of jejunal   
varices without evidence of active bleeding or other high-risk stigmata   
(Figure 1). The patient underwent liver ultrasound, which showed liver   
contour nodularity and mildly enlarged splenic vein and spleen. There was   
normal flow through hepatic and portal veins and hepatic arteries.   
Discussion: We present a case of small intestinal varices in a patient with   
no previous diagnosis or manifestations of portal hypertension. Small   
intestinal varices are uncommon and are rarely reported to cause   
gastrointestinal hemorrhage, but can cause significant blood loss when   
hemorrhage does occur. Small intestinal variceal bleeding can present subtly   
with occult gastrointestinal hemorrhage or dramatically with hypovolemic   
shock, hematochezia and/or hematemesis. Small intestinal varices are most   
often caused by portal hypertension in the setting of liver cirrhosis or   
portal vein thrombosis, but have also been associated with adhesions from   
previous abdominal surgeries or other vascular anomalies. Prompt   
identification and treatment, if necessary, are essential in the management   
of small intestinal varices. (Figure Presented).  
  
RECORD 51  
Thrombophilia profile in pediatric patients with cirrhosis and liver failure   
from the pediatrics hospital at the Western National Medical Center  
Pérez M.M.R. De León Y.A.C. Cruz A.R.J. De León J.C.B. Covarrubias R.G.  
Journal of Pediatric Gastroenterology and Nutrition (2016) 63 Supplement 2   
(S54-S55). Date of Publication: 1 Oct 2016  
  
Introduction: The liver plays a central role in the hemostatic system. The   
coagulation system in patients with cirrhosis is in a state of rebalance   
between antihemostatic and prohemostatic factors. The observation of   
inherited thrombophilia (protein C deficiency, protein S deficiency,   
antithrombin III deficiency, mutation of factor V Leiden, gene mutation of   
prothrombin G20210A, polymorphism of methylenetetrahydrofolate reductase   
(MTHFR) C677T and A1298C, and polymorphism of angiotensin converting enzyme   
(ACE-1) increase the risk of thrombosis of the portal vein in patients with   
cirrhosis. It is suggested that hypercoagulability may play a role in   
thrombosis of the hepatic artery after liver transplantation. Objective: To   
characterize the profile of thrombophilia of pediatric patients with   
cirrhosis and liver failure at the Hospital of Pediatrics, Western National   
Medical Center. Material and Methods: A study was conducted in pediatric   
patients, carriers of cirrhosis and liver failure at the Hospital of   
Pediatrics. Anticoagulant activity protein (protein C, protein S and   
antithrombin III) and factor VIII were determined by clotting assay.   
Mutations of thrombophilia panel, including factor V Leiden mutation,   
prothrombin gene mutation G20210AA, MTHFR C677T and A1298C polymorphisms,   
and polymorphism of angiotensin converting enzyme ACE-1 were determined by   
the technique of polymerase chain reaction. Results: There were 25 children,   
13 males, 12 females. The average age was 50.76 ± 46.96 (4-189) months. The   
main cause of cirrhosis was biliary tract atresia (72%). Distribution based   
on the Child-Pugh stadium was the following: stage A 24%, stage B 48%, and   
stage C 28%. It was identified protein C deficiency in 14 patients (56%),   
protein S deficiency in 3 patients (12%), antithrombin III deficiency in 9   
patients (36%). Factor VIII elevated in 92% of the population was   
documented. The mutations were made only to 23 patients; the main identified   
mutation was polymorphism deletion ACE-1 in 8 patients (34.7%), the MTHFR   
C677T polymorphism was the second cause with 21.7%, MTHFR A1298C   
polymorphism in 8.6%, compound heterozygote of MTHFR C677T/A1298C in 17.3%.   
Conclusions: It is considered that the deficiency of anticoagulant proteins   
and elevation of factor VIII is acquired secondary to chronic liver disease   
itself. The highest frequency of submission of ACE-1 may be due to the   
association of ACE-1 in metabolic processes of the liver and liver   
fibrogenesis participation.  
  
RECORD 52  
Coagulation parameters in patients with cirrhosis and portal vein thrombosis   
treated sequentially with low molecular weight heparin and vitamin K   
antagonists  
Tripodi A. Primignani M. Braham S. Chantarangkul V. Clerici M. Moia M.   
Peyvandi F.  
Digestive and Liver Disease (2016) 48:10 (1208-1213). Date of Publication: 1   
Oct 2016  
  
Background/aims Information on coagulation for cirrhotics on anticoagulants   
is scanty. We investigated plasma from 23 cirrhotics treated with   
low-molecular-weight-heparin (LMWH) followed by vitamin K antagonists (VKA).   
Methods On days 1–4 patients received full-dose LMWH. On day-5 VKA was   
started and LMWH was terminated when INR therapeutic-interval was reached.   
Blood was collected at peak and trough during LMWH, LMWH + VKA and VKA.   
Non-cirrhotics on VKA were included as controls. Results Anti-factor Xa   
increased from baseline-to-peak during LMWH. During LMWH + VKA was high and   
reverted to zero during VKA. INR was slightly high at baseline, trough or   
peak during LMWH and increased to 2.2 during LMWH + VKA or VKA. Mean VKA   
weekly-doses for cirrhotics and controls were 28.5 mg and 28.6 mg. Protein C   
decreased upon VKA, but not to the expected extent.   
Endogenous-thrombin-potential (ETP) decreased from baseline (1436 nM min) to   
trough (1258 nM min) and peak (700 nM min) during LMWH and was further   
reduced during LMWH + VKA (395 nM min). Conclusions Target-INR for   
cirrhotics can be reached by VKA dosages similar to those for   
non-cirrhotics. ETP reduction parallels the effect of LMWH and/or VKA.   
Whether these parameters represent the antithrombotic action elicited by   
these drugs remains to be determined by clinical-trials and   
laboratory-measurements. ETP, being a global-test reflecting both pro- and   
anti-coagulants targeted by antithrombotic drugs, seems the candidate for   
these trials.  
  
RECORD 53  
Pylephlebitis: Infective suppurative thrombosis of the portal vein  
Houston J. Hazratjee N. Agrawal S.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S901). Date of   
Publication: 1 Oct 2016  
  
Introduction: Pylephlebitis, also known as infective suppurative thrombosis   
of the portal vein, is a serious condition with a high early mortality and   
morbidity. It occurs when there is a combination of bacteremia and   
thrombosis of the portal vein. We present a 60 year old male that had been   
having flu like symptoms for the past month. Case Description: 60 year old   
male with a history of type II diabetes mellitus was admitted for gram   
positive cocci bacteremia found on blood cultures in the clinic. Blood   
cultures were obtained as part of an evaluation for flu like symptoms of one   
month's duration. On arrival to the hospital, a CT scan of his abdomen   
revealed a thrombus in the right portal vein with impeded the portal venous   
return. There were also two hypodense lesions on the posterior segment of   
the right lobe of the liver that were thought to be abscesses. He was   
admitted and started on IV Vancomycin and Zosyn as well as anticoagulation   
with Coumadin. Blood cultures ended up growing B. fragilis and Strep   
viridans and his antibiotic therapy was switched to Unasyn. On discharge he   
was prescribed a course of Levaquin and Flagyl. On CT follow-up 1 month   
later the he was found to have resolution of his thrombus and no signs of   
the abscesses. Discussion: Any infection that occurs in the region of the   
body that is drained by the portal venous system can lead to pylephlebitis   
with diverticulitis being the most common. It has also been associated with   
contiguous infections such as choledocholithiasis, pancreatitis,   
intra-abdominal abscess and inflammatory bowel disease. The most common   
bacteria to be found are Bacteroides fragilis, Escherichia coli and   
Streptococcus spp but the bacteremia is commonly polymicrobial. Diagnosis   
can be made by abdominal ultrasound or CT scan of the abdomen showing a   
thrombus in the portal vein. Empiric antibiotics are the treatment of choice   
for pylephlebitis and should be based off of the suspected source of   
infection. Successful antibiotic regimens include ampicillin,   
fluoroquinolones, 3rd generation cephalosporins, metronidazole, clindamycin   
and gentamicin. Antibiotics should be given parenterally until there is a   
significant clinical improvement, and duration should be four to six weeks   
in total. anticoagulation therapy should also be considered, as   
anticoagulation therapy with antibiotics has a better outcome than   
antibiotics alone. Even with treatment, pylephlebitis has a mortality rate   
ranging from 11 to 32 percent.  
  
RECORD 54  
Portomesenteric vein thrombosis after gastric surgery  
Han J.-W. Kong S.-H. Shin C.-I. Min S.-K. Min S.-I. Kim T.H. Yang J.-Y. Oh   
S.-Y. Suh Y.-S. Lee H.-J. Yang H.-K.  
Gastric Cancer (2016) 19:4 (1135-1143). Date of Publication: 1 Oct 2016  
  
Background: Postoperative portomesenteric venous thrombosis (PMVT) is a rare   
but potentially serious complication of gastric surgery. This study analyzed   
the incidence, characteristics, risk factors, and outcomes of PMVT following   
gastric surgery. Methods: Medical records of patients who underwent gastric   
surgery between January 2007 and December 2012 were reviewed   
retrospectively. The risk factors of PMVT were analyzed by a logistic   
regression analysis with control group matched 1:4 by age, sex, and cancer   
stage and by a Poisson regression analysis with unmatched control group. The   
resolution rate of PMVT in 12 months was compared between the treatment   
group and the nontreatment group. Results: The total incidence of PMVT after   
gastric surgery was 0.67 % (31/4611). Most (54.84 %) PMVT cases were   
detected within 1 month postoperatively. No accompanying deep vein   
thrombosis (DVT) was noted. Multivariate comparison with 1:4 matched control   
showed that combined splenectomy, synchronous malignancy, and   
intra-abdominal complication were independent risk factors. Advanced stage,   
combined splenectomy, and synchronous malignancy were independent risk   
factors in Poisson regression analysis using unmatched controls. The   
resolution rate of PMVT was not different from patients treated with   
anticoagulation (n = 6) or antiplatelet therapy (n = 1) and were not   
significantly different with those of the untreated group [85.7 % (6/7) vs.   
82.3 % (14/17), p = 0.935] during 1-year follow up. Conclusions: PMVT after   
gastric surgery was associated with advanced cancer stage, combined   
splenectomy, and synchronous malignancy, but it was not related to   
laparoscopy or DVT. Significant differences in the natural course of PMVT   
were not found between the treatment group and observation group.  
  
RECORD 55  
Does venous thromboembolic events increase morbidity and mortality in   
hepatocellular carcinoma patients  
Wang Y. Attar B.M. Bedrose S. Hinami K. Krishnan J. Simons-Linares C.R.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S343). Date of   
Publication: 1 Oct 2016  
  
Introduction: Venous thromboembolic event (VTE) are frequently associated   
with malignancy and leads to increased mortality. Hepatocellular carcinoma   
(HCC) is often associated with concurrent cirrhosis which derange   
coagulation-anticoagulation balance, leads to higher risk of VTE. This study   
aim to characterize VTE in HCC, identify independent risk factors and assess   
effects of VTE on overall prognosis in HCC. Methods: We retrospectively   
analyzed patients with diagnosis of hepatocellular carcinoma (by ICD-9 code)   
at a large public hospital during 10 years (05/2006 through 05/2015). HCC   
was confirmed by characteristic radiologic features and/or histology from   
liver biopsy. VTE was further categorized into pulmonary embolism,   
peripheral deep vein thrombosis, and intra-abdominal thrombosis. We exclude   
portal vein thrombosis as tumor thrombus from direct invasion could be   
confounded with bland thrombus. We collected data of patient-related risk   
factors, tumor characters, laboratory at diagnosis, treatment- related risk   
factors. We constructed multivariable logistic regression model through   
STATA V.13. Results: 270 patients with complete dataset were included.   
Thromboembolism events were identified in 16(5.9%) patients at an average of   
6.2 months since diagnosis of HCC: 7(43.8%) pulmonary embolism, 4(25%)   
peripheral deep vein thrombosis, 6(37.5%) intra-abdominal thrombosis. VTE   
frequency by etiology of HCC: viral-HCC 2.60% (2/77), alcoholic-HCC 2.5%   
(1/40), viral-alcoholic-HCC 9.17% (11/120), nonviral-nonalcoholic-HCC 6.06%   
(2/33). VTE frequency by severity of cirrhosis: non-cirrhotic liver 4.88% (2   
cases of 41), Child A 1.03% (1 cases of 97), Child B 11.11% (10 cases of   
90), Child C 6.8% (3 cases of 44). Multivariable regression showed   
independent risk factors for VTE in HCC include: viralalcoholic- HCC (OR   
18.7, p=0.017; CI 1.69-207.3), age (OR 2.86, p=0.017; CI 1.21 -6.78),   
presence of extrahepatic metastasis (OR 7.27, p=0.025; CI 1.29-41.1), BMI   
(OR 1.15, p=0.020; CI 1.02-1.29). VTE is not an independent risk factor for   
preclusion from curative treatment, hospice or mortality. Conclusion: VTE   
occurs in approximately 5.9% of patients with HCC. Patients with   
viral-alcoholic HCC, elderly patients, patients with higher BMI or   
extrahepatic metastasis are at higher risk of developing VTE; cirrhosis   
severity by child score is not independent risk factor. VTE does not affect   
overall prognosis after HCC diagnosis.  
  
RECORD 56  
Lemierre's syndrome wears a new outfit: portal vein thrombosis associated   
with fusobacterium nucleatum liver abscess  
Khoudari G. Audi A. Alshoubaki N. Raheel K. Kothari T. El-Daher N. Matos   
M.M.  
American Journal of Gastroenterology (2016) 111 Supplement 1 (S1358). Date   
of Publication: 1 Oct 2016  
  
Majority of pyogenic liver abscesses (PLA) are caused by polymycrobial   
infections, PLA secondary to Fusobacterium nucleatum (F. nucleatum) has   
rarely been reported. We are describing a case of F. nucleatum liver abscess   
complicated with portal vein thrombosis (PVT). A 60 year-old male presented   
with right upper quadrant (RUQ) pain, fever, and chills for 2 months.   
Physical exam revealed hepatomegaly and RUQ tenderness. Laboratory data   
showed WBC 21.8 /μl, Lactic acid 24 mg/dL, ALT 75 u/l, AST 39 u/l, ALP 412   
u/l, and total bilirubin 2.2 mg/dL. Abdominal US revealed large liver   
lesions with decreased echogenicity. CT abdomen confirmed the large   
hypodense lesions in the liver (10.2 x 6.7 cm), along with colonic wall   
thickening and evidence of PVT. This raised suspicion for metastatic   
disease, so colonoscopy was done and revealed diverticular disease, but no   
diverticulitis. Subsequently, US guided liver aspiration and biopsy revealed   
purulent material and cultures grew F. nucleatum. He was started on   
intravenous Piperacillin/Tazobactam and Metronidazole along with Enoxaparin.   
Liver abscesses were drained which led to significant clinical improvement.   
Interval CT Scan 2 and 4 weeks after drainage showed near complete   
resolution of the abscesses and PVT. Liver is the most common site of   
visceral abscesses, likely due to its rich blood supply from the portal and   
systemic circulations. PLAs are often secondary to polymicrobial pathogens,   
however, mono microbial infections such as Escherichia coli, followed by   
Klebsiella pneumoniae, are still possible. F. nucleatum is a gram negative   
anaerobic bacterium which is considered normal flora of the oral cavity.   
Recent evidence indicated that it is also a normal resident of   
gastrointestinal tract. This pathogen has been rarely reported to cause PLA,   
typically in immunocompromised patients with periodontal infections.   
Fusobacterium is well know to cause pharyngitis with internal jugular venous   
thrombosis in Lemierre's syndrome. To our knowledge, this case is one of few   
cases linking F. nucleatum to PLA and PVT in an immunocompetent patient   
without identified periodontal or gastrointestinal infection. Early drainage   
and proper antibiotic are the definitive treatments that have shown to   
significantly reduce mortality. The role of anticoagulation therapy for PVT   
remains controversial. In conclusion, this case demonstrates F. nucleatum as   
a rare cause of liver abscess and highlights its ability to cause PVT.  
  
RECORD 57  
Neonatal Hemostatic Disorders: Issues and Challenges  
Hanmod S.S. Jesudas R. Kulkarni R. Chitlur M.  
Seminars in Thrombosis and Hemostasis (2016) 42:7 (741-751). Date of   
Publication: 1 Oct 2016  
  
Neonates form a unique cohort with distinct features associated with the   
hemostatic system compared with older children and adults. The development   
of the human hemostatic system begins around 10 weeks in utero and continues   
to evolve during childhood. This dynamic period termed developmental   
hemostasis should be taken into consideration when diagnosing a neonate with   
disorders of bleeding or thrombosis.  
  
RECORD 58  
Prehepatic portal hypertension  
Šembera S. Hulek P. Jirkovsky V. Fejfar T. Krajina A. Dulicek P. Lojik M.   
Raupach J. Chovanec V. Rene O. Subrt Z. Kopacova M.  
Gastroenterologie a Hepatologie (2016) 70:5 (432-437). Date of Publication:   
1 Oct 2016  
  
ntroduction: Prehepatic portal hypertension (PH) in the absence of cirrhosis   
and solid tumours is most commonly caused by thrombosis of the portal vein   
(PT). Thrombosis in the portal system manifests as either acute abdominal   
pain or occurs silently, and varices develop in response to increased portal   
blood pressure. In 2016, the European Association forthe Study of the Liver   
issued a new clinical practical guideline for the treatment of PT. To treat   
acute PT, it is advised that anticoagulation therapy is initiated   
immediately. It is recommended to treat patients with chronic PT by the same   
way as patients with PH caused by liver cirrhosis. Aim: The aim of this   
study was to describe a group of patients with portal thrombosis at the   
University Hospital in Hradec Kralove (FNHK) and to compare the therapeutic   
approaches used with those of the new guidelines. Method: Retrospective   
description of all patients treated forthe above-mentioned portal thrombosis   
in the FNHK that were identified in electronic records. Results: The cohort   
consisted of 52 patients (27 males and 25 females); 44 patients with chronic   
PT, six with acute PT, and two with subacute PT. All patients with acute or   
subacute PT had been receiving anticoagulant therapy. Up to that point, five   
patients had undergone transjugular intrahepatic portosystemic shunt (TIPS),   
four of whom had local thrombolysis. Patients with chronic PT were treated   
the same as patients with PH and liver cirrhosis. Up to that point, six   
spleno-renal shunt procedures, nine splenectomy procedures, seven   
azygo-portal disconnection procedures, four TIPS procedures, one   
mesentero-caval shunt procedure, and one splenic embolization procedure had   
been performed. Discussion and Conclusion: Treatment of patients in our   
study group meets the challenges of the new recommendations. In complicated   
cases, othertherapeutic approaches may be necessary.  
  
RECORD 59  
portal vein thrombosis in cirrhotic and non cirrhotic patients: from   
diagnosis to treatment  
Dell’Era A. Seijo S.  
Expert Opinion on Orphan Drugs (2016) 4:9 (927-940). Date of Publication: 1   
Sep 2016  
  
Introduction: portal vein thrombosis (PVT) may occur in non-cirrhotic and   
cirrhotic patients. It can be classified as acute (if a recent thrombus is   
present) and chronic (if portal cavernoma has developed). Patients can be   
symptomatic or may present signs and symptoms related to the development of   
portal hypertension. In rare cases bowel infarction may occur. Areas   
covered: This review provides an overview of the clinical presentation,   
complications, diagnostic challenges and available treatments for PVT in   
non-cirrhotic and cirrhotic patients (NCPVT). Expert opinion: Treatment of   
acute NCPVT aims at recanalizing the thrombosed veins and preventing   
intestinal infarction and portal hypertension. anticoagulation should be   
started promptly and maintained for at least 6 months. Long-term   
anticoagulation should be implemented in the presence of underlying   
persistent thrombotic state. In chronic NCPVT, treatment aims at managing   
portal hypertension and portal cavernoma cholangiopathy and preventing new   
thrombotic events. In this setting, the indication for anticoagulation   
should be individualized. No formal recommendations can be given for PVT in   
cirrhosis, since there are no randomized controlled trials, prospective   
studies, or ad hoc guidelines. High quality studies, including randomized   
controlled trials, will be needed to provide robust evidence on the best   
treatment strategy.  
  
RECORD 60  
anticoagulation for venous thromboembolism prophylaxis and treatment in   
children with severe traumatic brain injury  
Landisch R. Hanson S. Punzalan R. Braun K. Gourlay D.  
American Journal of Hematology (2016) 91:9 (E382). Date of Publication: 1   
Sep 2016  
  
Background: Children who suffer from traumatic brain injury (TBI) are at   
higher risk for developing venous thromboembolism (VTE) during their   
hospitalization. Adult studies have demonstrated that early institution of   
chemical prophylaxis protects against VTEs and has a low risk of expanding   
intracranial hemorrhage. The risks and benefits of chemical prophylaxis in   
pediatric TBI have not been defined, resulting in long delays or total   
avoidance of chemical prophylaxis. We sought to describe a series of   
patients with severe TBI who received anticoagulation without bleeding   
complications. Methods: Following IRB approval, a retrospective review of   
prospectively collected data of all injured children less than 18 years old   
admitted to Children's Hospital of Wisconsin Intensive Care Unit (ICU) from   
8/2010 to 8/2015 was performed. Patients with severe TBI were defined by an   
Abbreviated Injury Scale head ≥ 3 and Glasgow Coma Scale score <9 on   
admission. VTE incidence among patients with TBI was examined. Patient   
characteristics were compared between groups with vs. without VTE. Outcomes   
assessed were VTE incidence (deep venous thrombosis (DVT), pulmonary   
embolism (PE)) among severe TBI patients, use of mechanical (i.e.,   
sequential compression devices, SCDs) and chemical prophylaxis as well as   
bleeding complications secondary to anticoagulation. Independent T-tests   
(unequal variance) were used for numerical average data analysis and for   
comparison of proportions (alpha=0.05). Results: In our review of 4075   
hospitalized injured children, we found 141(3.5%) patients with severe TBI.   
Nine patients with severe TBI developed VTE (7 DVT, 1 PE, 1 portal vein   
thrombosis) resulting in a 6.4% incidence among this cohort. Age, gender,   
ventilation and ICU days were comparable between VTE and non-VTE cohorts.   
Within the TBI cohort, 33% (3/9) of the patients were diagnosed by   
ultrasound associated with symptoms of VTE at a median of 3 days (range   
1-9), 44% (4/9) by screening ultrasound at a median of 7 days (range 6-13),   
and two by computed tomography. Seven (78%) VTEs were CVL associated. Only   
one child received chemical prophylaxis prior to VTE diagnosis (11.1%) due   
to high bleeding risk, which compared to the 11.4% chemical prophylaxis in   
the non-VTE group. Both VTE and non-VTE groups had similar compliance with   
SCDs (44% vs. 39%, p=0.76). Six of the nine (67%) patients were   
anticoagulated, either therapeutic or partial, immediately upon discovery of   
the VTE, with a range of 6-19 days (mean 12.3, median 11) after admission.   
Three patients did not receive anticoagulation after diagnosed with VTE due   
to ongoing concerns for bleeding. There were no bleeding complications   
resulting from either prophylactic or treatment dose anticoagulation.   
Conclusions: This series is among the first to describe the higher VTE   
incidence among children with severe TBI. Thrombus formation likely occurs   
prior to symptom development, suggesting earlier surveillance could result   
more timely management of VTE. While this series demonstrated no clinically   
significant bleeding in the patients receiving anticoagulation after severe   
TBI, additional prospective studies aimed at defining the safety of early   
chemical prophylaxis should be pursued.  
  
RECORD 61  
portal vein thrombosis in a Preterm Newborn with Mutation of the MTHFR and   
PAI-1 Genes and Sepsis by Candida parapsilosis  
Giuffrè M. Verso C.L. Serra G. Moceri G. Cimador M. Corsello G.  
American Journal of Perinatology (2016) 33:11 (1099-1103). Date of   
Publication: 1 Sep 2016  
  
Objective This report discusses the role of both congenital and acquired   
risk factors in the pathogenesis of portal vein thrombosis (PVT). Study   
Design We describe the clinical management and treatment of PVT in a preterm   
newborn with a homozygous mutation of the methylenetetrahydrofolate   
reductase (MTHFR) and plasminogen activator inhibitor-1 (PAI-1) genes and   
sepsis by Candida parapsilosis. Results Although literature data suggest a   
minor role of genetic factors in thrombophilia in the case of only one   
mutation, we hypothesize that combined thrombophilic genetic defects may   
have a cumulative effect and significantly increase the thrombotic risk.   
Conclusion It could be appropriate to include more detailed analyses of   
procoagulant and fibrinolytic factors in the diagnostic workup of neonatal   
thrombosis, also through the investigation of genetic polymorphisms. The   
anticoagulant therapy and the removal of concurrent risk factors remain   
basic steps for the adequate management and prevention of complications.  
  
RECORD 62  
The ischemic liver cirrhosis theory and its clinical implications  
Mancuso A.  
Medical Hypotheses (2016) 94 (4-6). Date of Publication: 1 Sep 2016  
  
The canonical pathway theory of cirrhosis addresses inflammation as the main   
driver of hepatic fibrogenesis in hepatitis, so needing a further hypothesis   
for etiologies missing inflammation, for which parenchymal extinction is   
postulated. The present paper reports an alternative hypothesis suggesting a   
central role of micro-vascular ischemia in fibrogenesis and cirrhosis   
development, whatever is the aetiology of liver chronic injury. In fact,   
since chronic liver injury could finally result in endothelial damage and   
micro-vascular thrombosis, leading to a trigger of inappropriate hepatocyte   
proliferation and fibrosis, finally cirrhosis development could arise from   
chronic micro-vascular ischemia. Recently, some important confirmation of   
this hypothesis has been reported. In fact, in a murine experimental model   
of congestive hepatopathy, it was found that chronic hepatic congestion   
leads to sinusoidal thrombosis and strain, which in turn promote hepatic   
fibrosis. Furthermore, a study on a murine model of cirrhosis reported   
enoxaparin to reduce hepatic vascular resistance and portal pressure by   
having a protective role against fibrogenesis.In conclusion, the hypothesis   
giving a central role of micro-vascular ischemia in fibrogenesis and   
cirrhosis development could change the clinical scenario of chronic liver   
disease and have several main implications on management of various liver   
disease.  
  
RECORD 63  
First case with antithrombin deficiency, mesenteric vein thrombosis and   
pregnancy: Multidisciplinary diagnosis and successful management  
García-Botella A. Asenjo S. De La Morena-Barrio M.E. Corral J. Bolaños E.   
Carlin P.S. López E.S. García A.J.T.  
Thrombosis Research (2016) 144 (72-75). Date of Publication: 1 Aug 2016  
  
RECORD 64  
Clinical presentations, risk factors, treatment and outcomes in patients   
with splanchnic vein thrombosis: a single-center experience  
Klute K. DeFilippis E.M. Shillingford K. Chapin J. DeSancho M.T.  
Journal of Thrombosis and Thrombolysis (2016) 42:2 (267-271). Date of   
Publication: 1 Aug 2016  
  
Splanchnic vein thrombosis (SVT) is an uncommon form of venous thrombosis.   
Management can be challenging due to underlying conditions, increased   
bleeding risk, and lack of evidence from clinical trials. We sought to   
characterize the presentation and management of patients with SVT at a large   
tertiary hospital. A total of 43 patients’ electronic medical records were   
reviewed. Median age at diagnosis was 43 (18–71). Sixteen patients had   
isolated portal vein thrombosis (37.2 %), and 16 (37.2 %) had thrombosis   
involving multiple splanchnic veins. Abdominal pain was the most common   
clinical presentation (67.4 %). Thrombophilia was present in 18 patients   
(41.9 %), nine had underlying liver disease (20.9 %) and seven had   
inflammatory bowel disease (16.3 %). Thirty-nine (90.7 %) patients were   
treated with anticoagulation, and 11(25.6 %) of these patients underwent   
interventional procedures. Thirty (69.8 %) patients remained on indefinite   
anticoagulation. Results of follow-up imaging at least 1 month after   
diagnosis were available for 29 patients; imaging showed chronic, stable   
thrombosis in 14 patients (48.3 %), resolution of thrombosis in 13 patients   
(44.8 %) and asymptomatic progression in two patients (6.9 %). Recurrent   
thrombosis occurred in four patients (9.3 %). Major bleeding occurred in   
eight patients who received anticoagulation (18.6 %), including fatal   
subdural hematoma in one patient. In this cohort of patients managed by   
hematologists and gastroenterologists, the majority of patients were treated   
with anticoagulation. Interventional procedures were higher than in   
previously reported series. Our study strongly supports the   
interdisciplinary management of splanchnic venous thrombosis.  
  
RECORD 65  
Massive gastrointestinal bleeding due to isolated jejunal varices in a   
patient with extrahepatic portal hypertension: A case report  
Mansoor E. Singh A. Nizialek G. Veloso H.M. Katz J. Cooper G.S. Isenberg G.  
American Journal of Gastroenterology (2016) 111:8 (1209-1211). Date of   
Publication: 1 Aug 2016  
  
RECORD 66  
Management of portal vein thrombosis in cirrhosis: An update  
Mancuso A.  
European Journal of Gastroenterology and Hepatology (2016) 28:7 (739-743).   
Date of Publication: 1 Jul 2016  
  
Background portal vein thrombosis (PVT) is a complication of cirrhosis.   
However, whether PVT worsens cirrhosis outcome is a debated issue. Aim To   
report an update on the management of PVT. Methods A review was performed on   
the outcome, prevention, and treatment of PVT. Results Some studies suggest   
that PVT could worsen the rate of hepatic decompensation and survival of   
cirrhosis, whereas others report a non-negative impact of PVT in the outcome   
of cirrhosis. Therefore, the prognostic value of PVT in cirrhosis remains a   
gray zone. One single randomized-controlled trial reported that enoxaparin   
could prevent PVT, delay the occurrence of hepatic decompensation, and   
improve survival. However, no further study data confirmed this assumption   
and the issue is not actually generalizable. Numerous studies report that   
anticoagulation determines a relatively high rate of portal vein   
recanalization in cirrhotics PVT. However, further data are warranted to   
confirm the risk-to-benefit of anticoagulation, especially bleeding.   
Transjugular intrahepatic portosystemic shunt (TIPS) has been reported to be   
effective as a treatment of PVT in cirrhosis, with the advantage of avoiding   
the risk of bleeding linked to anticoagulation. However, there are no data   
comparing TIPS with anticoagulation as a treatment of PVT in cirrhosis.   
Furthermore, there is no evidence on whether both anticoagulation and TIPS   
improve survival. Conclusion It is uncertain whether PVT affects cirrhosis   
outcome. Further data are needed to weigh the risk/benefit ratio of   
enoxaparin for the prevention of PVT in cirrhosis. anticoagulation or TIPS   
should probably be indicated in liver transplantation candidates, but   
avoided in patients not suitable for liver transplantation and with an   
otherwise poor prognosis. Future studies should evaluate which subgroup of   
cirrhotics with PVT may benefit from treatment. Management of PVT in   
cirrhosis should be personalized.  
  
RECORD 67  
anticoagulation therapy with warfarin versus low-dose aspirin prevents   
portal vein thrombosis after laparoscopic splenectomy and azygoportal   
disconnection  
Jiang G.-Q. Xia B.-L. Chen P. Qian J.-J. Jin S.-J. Zuo S.-Q. Bai D.-S.  
Journal of Laparoendoscopic and Advanced Surgical Techniques (2016) 26:7   
(517-523). Date of Publication: 1 Jul 2016  
  
Background: Portal vein system thrombosis (PVST) is a frequent and   
potentially life-threatening complication after laparoscopic splenectomy and   
azygoportal disconnection (LSD) in patients with cirrhotic portal   
hypertension. The objective of this study was to investigate the safety and   
effectiveness of warfarin with a target international normalized ratio (INR)   
of 2.0-2.5 for the prevention of PVST after LSD. Hitherto, this is the first   
study to assess the use of warfarin in this field. Materials and Methods: We   
retrospectively analyzed a database of 73 consecutive patients who underwent   
LSD from January 2013 to September 2014. Patients were categorized into the   
warfarin group (34 patients) and the aspirin group (39 patients). The INR   
and incidence of PSVT were monitored for 90 days. Results: Compared with the   
aspirin group, the warfarin group had a lower incidence of PVST on   
postoperative day (POD) 30 [17/34 (50.0%) versus 29/39 (74.4%); P = .032]   
and POD 90 [8/34 (23.5%) versus 30/39 (76.9%); P < .0001] and main portal   
vein thrombosis (MPVT) on POD 90 [3 (8.8%) versus 13 (33.3%); P = .012].   
From POD 30 to 90, the warfarin group achieved more complete recanalization   
of PVST [9/17 (52.9%) versus 3/29 (10.3%), P = .005] and MPVT [9/12 (75.0%)   
versus 3/12 (25.0%), P = .039]. Multiple logistic regression analysis   
revealed that warfarin was an independent protective factor for PVST at POD   
90 (relative risk, 0.027; 95% confidence interval, 0.004-0.168; P < .001).   
No patients developed bleeding complications. Conclusions: anticoagulation   
therapy with warfarin is safe and effective for the prevention of PVST in   
cirrhotic patients with portal hypertension after LSD.  
  
RECORD 68  
Hematological Issues in Liver Disease  
Allison M.G. Shanholtz C.B. Sachdeva A.  
Critical Care Clinics (2016) 32:3 (385-396). Date of Publication: 1 Jul 2016  
  
Acute and chronic liver failure are associated with numerous alterations in   
different features of the coagulation system. Consequently, there is   
widespread confusion regarding the potential for both bleeding and   
thrombosis in patients with liver disease. The risk of bleeding is related   
to the hemodynamic changes in portal pressures and venous congestion whereas   
the thrombotic risk stems from changes in the coagulation system.   
Antithrombotic prophylaxis and treatment of patients with hemorrhage and   
thrombosis requires careful assessment, interpretation of laboratory workup,   
and attention to coexistent morbidities. A framework for the management of   
these conditions is presented for clinicians.  
  
RECORD 69  
Evidence-based clinical practice guidelines for liver cirrhosis 2015  
Fukui H. Saito H. Ueno Y. Uto H. Obara K. Sakaida I. Shibuya A. Seike M.   
Nagoshi S. Segawa M. Tsubouchi H. Moriwaki H. Kato A. Hashimoto E. Michitaka   
K. Murawaki T. Sugano K. Watanabe M. Shimosegawa T.  
Journal of Gastroenterology (2016) 51:7 (629-650). Date of Publication: 1   
Jul 2016  
  
The Japanese Society of Gastroenterology revised the evidence-based clinical   
practice guidelines for liver cirrhosis in 2015. Eighty-three clinical   
questions were selected, and a literature search was performed for the   
clinical questions with use of the MEDLINE, Cochrane, and Igaku Chuo Zasshi   
databases for the period between 1983 and June 2012. Manual searching of the   
latest important literature was added until August 2015. The guidelines were   
developed with use of the Grading of Recommendations Assessment,   
Development, and Evaluation (GRADE) system. This digest version in English   
introduces selected clinical questions and statements related to the   
management of liver cirrhosis and its complications. Branched-chain amino   
acids relieve hypoalbuminemia and hepatic encephalopathy and improve quality   
of life. Nucleoside analogues and peginterferon plus ribavirin combination   
therapy improve the prognosis of patients with hepatitis B virus related   
liver cirrhosis and hepatitis C related compensated liver cirrhosis,   
respectively, although the latter therapy may be replaced by direct-acting   
antivirals. For liver cirrhosis caused by primary biliary cirrhosis and   
active autoimmune hepatitis, urosodeoxycholic acid and steroid are   
recommended, respectively. The most adequate modalities for the management   
of variceal bleeding are the endoscopic injection sclerotherapy for   
esophageal varices and the balloon-occluded retrograde transvenous   
obliteration following endoscopic obturation with cyanoacrylate for gastric   
varices. Beta-blockers are useful for primary prophylaxis of esophageal   
variceal bleeding. The V(2) receptor antagonist tolvaptan is a useful add-on   
therapy in careful diuretic therapy for ascites. Albumin infusion is useful   
for the prevention of paracentesis-induced circulatory disturbance and renal   
failure. In addition to disaccharides, the nonabsorbable antibiotic   
rifaximin is useful for the management of encephalopathy. anticoagulation   
therapy is proposed for patients with acute-onset or progressive portal vein   
thrombosis.  
  
RECORD 70  
portal vein thrombosis in cirrhosis: Controversies and latest developments  
Harding D.J. Perera M.T.P.R. Chen F. Olliff S. Tripathi D.  
World Journal of Gastroenterology (2016) 22:22 (6769-6784). Date of   
Publication: 14 Jun 2016  
  
portal vein thrombosis (PVT) is encountered in liver cirrhosis, particularly   
in advanced disease. It has been a feared complication of cirrhosis,   
attributed to significant worsening of liver disease, poorer clinical   
outcomes and potential inoperability at liver transplantation; also   
catastrophic events such as acute intestinal ischaemia. Optimal management   
of PVT has not yet been addressed in any consensus publication. We review   
current literature on PVT in cirrhosis; its prevalence, pathophysiology,   
diagnosis, impact on the natural history of cirrhosis and liver   
transplantation, and management. Studies were identified by a search   
strategy using MEDLINE and Google Scholar. The incidence of PVT increases   
with increasing severity of liver disease: less than 1% in well-compensated   
cirrhosis, 7.4%-16% in advanced cirrhosis. Prevalence in patients undergoing   
liver transplantation is 5%-16%. PVT frequently regresses instead of uniform   
thrombus progression. PVT is not associated with increased risk of   
mortality. Optimal management has not been addressed in any consensus   
publication. We propose areas for future research to address unresolved   
clinical questions.  
  
RECORD 71  
Retrospective review on isolated distal deep vein thrombosis (IDDVT) - A   
benign entity or not?  
Ho P. Lim H.Y. Chua C.C. Sleeman M. Tacey M. Donnan G. Nandurkar H.  
Thrombosis Research (2016) 142 (11-16). Date of Publication: 1 Jun 2016  
  
Introduction Isolated distal deep venous thrombosis (IDDVT) is traditionally   
associated with less severe clinical sequelae, with ongoing debate on   
multiple aspects of its management. Despite numerous studies evaluating its   
acute management, there remains a paucity of data evaluating long-term   
complications such as recurrence and subsequent malignancy. We aim to   
evaluate the characteristics of IDDVT in institutions that routinely perform   
whole leg ultrasonography, and the risks of recurrence and complications in   
comparison to major venous thromboembolism (major VTE; defined as above-knee   
or proximal DVT and pulmonary embolism (PE)). Methods Retrospective   
evaluation of consecutive IDDVT and major VTE from July 2011 to December   
2012 in a hospital network in Melbourne, Australia. Patients were followed   
up for a minimum of 24 months. Patients with active malignancy were   
excluded. Results Of 1024 VTE cases, there were 164 non-cancer patients (92   
males, 72 females, median age of 61 years) with IDDVT. Compared to major   
VTE, IDDVT was more likely to be provoked (73% vs 59%, p < 0.01), has   
shorter duration of anticoagulation (median 3.5 months vs 6.0 months, p <   
0.01) and less clinically significant bleeding (2.4% vs 6.7%, p = 0.05),   
independent of duration of therapy. Recurrence was non-inferior compared to   
major VTE (10% vs 7%, p = 0.36) and 60% recurred with major VTE. Three   
(1.8%) were subsequently diagnosed with cancer (vs 1.9% in major VTE, p =   
0.97). Conclusions IDDVT has non-inferior rates of recurrence and subsequent   
cancer detection compared to major VTE and hence, its clinical significance   
should not differ from major VTE. Further studies are required to determine   
the adequate length of anticoagulation.  
  
RECORD 72  
Incidental splanchnic vein thrombosis: preliminary registry data  
Ames P.R.J. Margaglione M.  
The Lancet Haematology (2016) 3:6 (e256-e257). Date of Publication: 1 Jun   
2016  
  
RECORD 73  
Negative and positive predictors of portal vein system thrombosis after   
laparoscopic splenectomy and azygoportal disconnection: A 3-month follow-up  
Jiang G.-Q. Bai D.-S. Chen P. Xia B.-L. Qian J.-J. Jin S.-J.  
International Journal of Surgery (2016) 30 (143-149). Date of Publication: 1   
Jun 2016  
  
Introduction: Portal vein system thrombosis (PVST) is an alarming and   
potentially life-threatening complication of laparoscopic splenectomy and   
azygoportal disconnection (LSD). The objective of this study was to   
investigate negative and positive predictors of PVST after LSD in patients   
receiving anticoagulant regimens with aspirin or warfarin. Methods:   
Seventy-five consecutive patients who underwent LSD from 2013 to 2014 were   
retrospectively reviewed. Patients received anticoagulant regimen with   
warfarin (n = 35) or aspirin (n = 40) according to individual preference.   
International normalized ratio (INR) and the incidence of PSVT were compared   
in patients received anticoagulant regimen with warfarin or aspirin on   
postoperative days (POD) 7, 30, and 90, and factors associated with PVST at   
these time points were determined by univariate and logistic multivariable   
regression analyses. Results: Portal vein diameter was an independent   
negative predictor of PVST on PODs 7, 30, and 90. anticoagulation with   
warfarin was an independent positive predictor of PVST on PODs 30 and 90,   
and INR was an independent positive predictor of PVST on POD 90. Dynamic   
changes in the incidence of PVST on the day of admission and on PODs 7, 30,   
and 90 differed significantly between the warfarin and aspirin groups (P =   
0.002). No patient experienced perioperative bleeding. Conclusions: Portal   
vein diameter was an independent negative predictor, while anticoagulation   
therapy with warfarin and INR were independent positive predictors, of PVST   
after LSD. Early anticoagulation with warfarin is safe and effective for the   
prevention of PVST after LSD.  
  
RECORD 74  
Risk factors of portal vein thrombosis in acute pancreatitis  
Chooklin S. Pidhirnyy B. Osmilovska I. Usach O.  
Pancreatology (2016) 16:3 SUPPL. 1 (S91-S92). Date of Publication: June 2016  
  
Introduction: portal vein thrombosis (PVT) is a one local complication of   
acute pancreatitis. PVT does not cause any additional symptom in most cases   
and is usually detected incidentally on ultrasonography and computed   
tomography performed to evaluate the severity of pancreatitis. Aims: The aim   
of this study was to investigate to determine the risk factors of PVT in   
severe acute pancreatitis. Patients & methods: 276 patients with severe   
acute pancreatitis were studied. PVT was identified in 15 patients.   
Univariate and multivariate regression analyses were applied to explore   
potential risk factors for the development of PVT in acute necrotizing   
pancreatitis patients. Mortality, organ failure and length of hospital stay   
were also compared between patients with or without PVT. Results: Leukocytes   
(>10 × 10(9)/L), hyperglycemia (≥ 10 mmol/L), alcoholic etiology high   
intra-abdominal pressure, infected pancreatic necrosis were risk factors for   
PVT. Patients with PVT showed higher mortality, longer hospital duration,   
higher rates of a variety of complications and more utilization of invasive   
interventions. Conclusion: We identified the risk factors of portal vein   
thrombosis in severe acute pancreatitis. In these cases the physicians   
should consider therapeutic anticoagulation to prevent PVT.  
  
RECORD 75  
Outcomes of anticoagulation use for portal vein thrombosis in University of   
Wisconsin liver transplant patients  
Falls M. Said A. Fernandez L. Foley D. Frith K. Hager D. Hoy H. Leverson G.   
Mezrich J. Prickette T. Wakefield M. D'Alessandro A.  
American Journal of Transplantation (2016) 16 Supplement 3 (673-674). Date   
of Publication: 1 Jun 2016  
  
Background: The prevalence of portal vein thrombosis (PVT) in cirrhotic   
liver transplant patients increases the risk of morbidity and mortality   
post-liver transplantation. Current research suggests that PVT should be   
managed with anticoagulation in those with cirrhosis that are waiting for   
liver transplant. anticoagulation therapy and management is not uniformly   
practiced in pre-liver transplant patients with cirrhosis and PVT, nor are   
there established clinical guidelines. The purpose of this study was to   
compare the outcomes in mortality following liver transplant in patients   
from the University of Wisconsin who had PVT prior to transplantation and   
were, or were not treated with anticoagulation. Methods: This was a   
single-institution, retrospective review from a prospectively collected   
database to evaluate the utility of anticoagulation therapy in patients with   
PVT that underwent liver transplantation between January 1, 2006 and June   
30, 2014. The primary outcome of interest was patient mortality. Patients   
were excluded as a result of additional diagnoses including atrial   
fibrillation, cerebral vascular accident, pulmonary emboli and deep vein   
thrombosis. Results: From a total of 755 liver transplants performed between   
January 1, 2006 and June 30, 2014, 56 patients with PVT were evaluated in   
this study(Table 1). There was no statistically significant difference in   
mortality between the 4 groups in table 1 (p = 0.67). Conclusion: This study   
suggests the use of anticoagulation and thrombectomy at liver transplant may   
improve the mortality outcomes in this population (Table 1). The results are   
not statistically significant, likely due to the small sample size. A   
multi-center study of the same design would be of benefit to improve the   
power of the data and better evaluate the use of anticoagulation therapy in   
the liver transplant patient with PVT. Review of this data in 2 years would   
allow us to continue to further evaluate morbidity and mortality in this   
patient cohort. (Table Presented).  
  
RECORD 76  
anticoagulation for portal vein thrombosis in cirrhosis  
Intagliata N.M. Ferreira C.N. Caldwell S.H.  
Clinical Liver Disease (2016) 7:6 (126-131). Date of Publication: 1 Jun 2016  
  
RECORD 77  
Clinical history and antithrombotic treatment of incidentally detected   
splanchnic vein thrombosis: a multicentre, international prospective   
registry  
Riva N. Ageno W. Schulman S. Beyer-Westendorf J. Duce R. Malato A. Santoro   
R. Poli D. Verhamme P. Martinelli I. Kamphuisen P. Dentali F.  
The Lancet Haematology (2016) 3:6 (e267-e275). Date of Publication: 1 Jun   
2016  
  
Background Little information is available about the clinical history of   
patients with incidentally detected splanchnic vein thrombosis and its   
therapeutic management remains controversial. The aim of this study was to   
assess the risk factors, therapeutic strategies, and long-term outcomes of   
incidentally detected splanchnic vein thrombosis. Methods We analysed data   
from patients with incidentally detected splanchnic vein thrombosis who were   
enrolled in an international, multicentre, prospective cohort study of   
splanchnic vein thrombosis between 2008 and 2012. The study was done at 31   
centres in 11 countries (Italy, South Korea, Germany, Canada, Belgium, the   
Netherlands, Brazil, USA, France, Israel, UK). Information about demographic   
characteristics, risk factors, and treatment was collected. The study   
outcomes during the 2-year follow-up were major bleeding (International   
Society on Thrombosis and Haemostasis definition plus the need for hospital   
admission), thrombotic events (venous or arterial thromboses), and   
mortality. The primary analysis period was from the diagnosis of   
incidentally detected splanchnic vein thrombosis until the first adjudicated   
clinical outcome or the end of follow-up. Findings Between May 2, 2008, and   
Jan 30, 2012, we enrolled 177 patients with incidentally detected splanchnic   
vein thrombosis (median age 57 years [IQR 49–66], 118 [67%] men, 138 [78%]   
patients with portal vein thrombosis). The most common underlying diseases   
were liver cirrhosis (82 [46%] patients) and solid cancer (62 [35%]   
patients). Anticoagulant treatment was prescribed to 109 (62%) patients.   
Median duration of anticoagulation was 6 months (IQR 5–12) for patients who   
received parenteral anticoagulants alone and 24 months (IQR 12–24) for   
patients treated with vitamin K antagonists. During a median follow-up of 2   
years (IQR 1–2), the incidence of major bleeding was 3·3 events (95% CI   
1·7–6·3) per 100 patient-years and the incidence of thrombotic events was   
8·0 events (95% CI 5·2–12·1) per 100 patient-years. On-treatment incidence   
was 3·2 events (95% CI 1·2–8·4) per 100 patient-years for major bleeding and   
3·9 events (95% CI 1·6–9·5) per 100 patient-years for thrombotic events. In   
multivariate analysis, anticoagulant treatment as a time-dependent variable   
reduced the incidence of thrombotic events (hazard ratio 0·85, 95% CI   
0·76–0·96) without increasing the risk of major bleeding (p>0·05). In   
patients with clinically suspected splanchnic vein thrombosis, the incidence   
of major bleeding was 3·9 events (95% CI 2·6–5·7) per 100 patient-years and   
the incidence of thrombotic events was 7·0 events (95% CI 5·2–9·3) per 100   
patient-years. Interpretation Our results show that the prognosis of   
incidentally detected splanchnic vein thrombosis is similar to that of   
clinically suspected splanchnic vein thrombosis and suggest that similar   
treatment strategies should be applied. Funding Pfizer Canada research   
grant.  
  
RECORD 78  
Hepatic arterial and portal venous complications after adult and pediatric   
living donor liver transplantation, risk factors, management and outcome (A   
retrospective cohort study)  
Gad E.H. Abdelsamee M.A. Kamel Y.  
Annals of Medicine and Surgery (2016) 8 (28-39). Date of Publication: 1 Jun   
2016  
  
Objectives: Hepatic arterial (HA) and portal venous (PV) complications of   
recipients after living donor liver transplantation(LDLT) result in patient   
loss. The aim of this study was to analyze these complications. Methods: We   
retrospectively analyzed HA and/or PV complications in 213 of 222 recipients   
underwent LDLT in our centre. The overall male/female and adult/pediatric   
ratios were 183/30 and 186/27 respectively. Results: The overall incidence   
of HA and/or PV complications was 19.7% (n = 42), while adult and pediatric   
complications were 18.3% (n = 39) and 1.4% (n = 3) respectively. However   
early (<1month) and late (>1month) complications were 9.4% (n = 20) and   
10.3% (n = 22) respectively. Individually HA problems (HA stenosis, HA   
thrombosis, injury and arterial steal syndrome) 15% (n = 32), PV problems   
(PV thrombosis and PV stenosis) 2.8% (n = 6) and simultaneous HA and PV   
problems 1.9% (n = 4). 40/42 of complications were managed by angiography (n   
= 18), surgery (n = 10) or medically (Anticoagulant and/or thrombolytic) (n   
= 12) where successful treatment occurred in 18 patients. 13/42 (31%) of   
patients died as a direct result of these complications. Preoperative PVT   
was significant predictor of these complications in univariate analysis. The   
6-month, 1-, 3-, 5- 7- and 10-year survival rates in patients were 65.3%,   
61.5%, 55.9%, 55.4%, 54.5% and 54.5% respectively. Conclusion: HA and/or PV   
complications specially early ones lead to significant poor outcome after   
LDLT, so proper dealing with the risk factors like pre LT PVT (I.e. More   
intensive anticoagulation therapy) and the effective management of these   
complications are mandatory for improving outcome.  
  
RECORD 79  
Is Post-TIPS anticoagulation Therapy Necessary in Patients with cirrhosis   
and portal vein thrombosis? A Randomized Controlled Trial  
Wang Z. Jiang M.-S. Zhang H.-L. Weng N.-N. Luo X.-F. Li X. Yang L.  
Radiology (2016) 279:3 (943-951). Date of Publication: 1 Jun 2016  
  
Purpose To determine whether posttransjugular intrahepatic portosystemic   
shunt (TIPS) placement anticoagulation therapy could benefit patients with   
cirrhosis and portal vein thrombosis (PVT) from the perspective of a change   
in portal vein patency status and clinical outcomes. Materials and Methods   
The study was approved by the institutional review board, and informed   
consent was obtained from each patient. From October 2012 to February 2014,   
patients with cirrhosis and PVT who underwent TIPS placement were randomly   
assigned to the anticoagulation therapy or control group. All patients were   
followed at 1, 3, 6, and 12 months after the TIPS procedure. Outcome   
measures were a change of portal vein patency status and clinical measures   
including gastrointestinal rebleeding, shunt dysfunction, hepatic   
encephalopathy, and survival. Student t test, χ(2) test, Fisher exact test,   
Mann-Whitney U test, and logistical regression were applied where   
appropriate. Results A total of 64 patients were enrolled in the study, with   
31 allocated to the anticoagulation group and 33 allocated to the control   
group. Overall, thrombi were improved in 61 patients (96.8%) after the   
procedure. PVT recanalization (ie, complete disappearance; reconstruction of   
cavernous transformation) was achieved in 26 patients (83.9%) in the   
anticoagulation therapy group and in 23 (71.8%) patients in tthe control   
group (P = .252). The presence of a superior mesenteric vein thrombus may   
help predict recanalization failure (unadjusted relative risk = 0.243; 95%   
confidence interval: 0.070, 0.843; P = .026). Clinical outcomes were also   
similar between the two groups. Conclusion anticoagulation therapy may not   
be necessary in certain patients with PVT because TIPS placement alone can   
achieve a high persistent recanalization rate. (©) RSNA, 2015.  
  
RECORD 80  
Direct Oral Anticoagulants in cirrhosis Patients Pose Similar Risks of   
Bleeding When Compared to Traditional anticoagulation  
Intagliata N.M. Henry Z.H. Maitland H. Shah N.L. Argo C.K. Northup P.G.   
Caldwell S.H.  
Digestive Diseases and Sciences (2016) 61:6 (1721-1727). Date of   
Publication: 1 Jun 2016  
  
Background and Aims: Direct oral anticoagulants (DOAC) are important new   
anticoagulant therapies that are not well studied in patients with chronic   
liver disease. The aim of this study was to compare rates of bleeding in   
cirrhosis patients treated with DOAC (factor Xa inhibitors: rivaroxaban and   
apixaban) to those in cirrhosis patients treated with traditional   
anticoagulation (warfarin and low molecular weight heparin). Methods: We   
identified a total of 39 patients with cirrhosis who received   
anticoagulation therapy over a 3-year period (20 DOAC and 19 traditional   
anticoagulation) from a research database. Medical records were reviewed to   
obtain clinical data to compare between the groups. Results: Clinical   
characteristics between the two groups were similar. There were three   
documented bleeding events in the traditional anticoagulation group and four   
bleeding events in the DOAC group (p = 0.9). There were two major bleeding   
events in the traditional anticoagulation group and one major bleeding event   
in the DOAC group. There were no documented reports of drug-induced liver   
injury during this study period. Among all patients, no significant   
predictors of bleeding were identified using univariate regression and Cox   
proportional hazard modeling. Conclusions: This is the first clinical study   
evaluating the use of DOAC in patients with cirrhosis. DOAC display similar   
safety characteristics when compared to traditional anticoagulation in   
patients with cirrhosis and are potentially attractive agents for   
anticoagulation therapy. Larger studies are now needed to better understand   
the safety and efficacy of these agents in cirrhosis.  
  
RECORD 81  
Should anticoagulation be offered in patients with PVT in the setting of   
HCC?  
Mahmoudi T. Kayal A. Carvalho R. Weiss A.  
Canadian Journal of Gastroenterology and Hepatology (2016) 2016. Date of   
Publication: 2016  
  
Background. portal vein thrombosis (PVT) is a seen in about 20-44% of   
patients with hepatocellular carcinoma (HCC). To our knowledge, no other   
study has looked at the need for anticoagulation in patients with HCC and   
PVT. Aims. The aim of this study is to investigate the natural history and   
progression of portal vein thrombosis in patients with hepatocellular   
carcinoma with or without anticoagulation therapy. Methods. Using the   
British Columbia Cancer Agency database, a cohort of 54 patients who were   
diagnosed with both conditions were evaluated retrospectively. Nine patients   
were excluded secondary to lack of follow up. HCC and PVT diagnosis and   
followup was made with contrast enhanced CT or MRI. Most patients received a   
single or a combination of the following treatments: transarterial   
chemoembolization, radiofrequency ablation or surgical resection. Thirty   
five (78%) patients received systemic therapy with Sorafenib. Results.   
Thirty eight patients were males and mean age was 62.8. Liver disease   
etiology was HCV in 19 (42%), HBV in 18 (40%), ETOH in 5 (11%) and   
hemochromatosis in 1 (2%). Results: Average survival after HCC diagnosis was   
28 months and 15 months after PVT diagnosis. Among the 45 patients   
evaluated, 8 patients received anticoagulation while 39 did not. PVT   
progression occurred in 19 (49%) of the non anticoagulated group, and 4   
(67%) of the anticoagulated group. Right portal vein involvement was seen in   
18 (40%) patients with progression in 67% of the time, Left PVT in 13 (28%)   
with a progression in 7(54%), and main PVT 6 (13%) with a progression in   
(67%). In 1 case, PVT progressed from the main PVT to Superior mesenteric   
vein (SMV) and from the LPV to SMV in 2 other cases. No symptoms directly   
related to PVT development were reported. Conclusions. The possible   
anticoagulation related complications need to be considered before   
attempting therapy in patients with HCC and PVT. Despite the small number of   
patients included in this study, this review shows that PVT progression in   
patients with HCC and the absence of clinical complications is similar in   
both anticoagulated and non anticoagulated groups. Thus, the usefulness of   
anticoagulation in this patient population needs to be further studied.   
(Table presented).  
  
RECORD 82  
Length of anti-coagulation in splanchnic venous thrombosis  
Hasan M. Rashid A. Moiz B. Sarwar S.  
Journal of Thrombosis and Haemostasis (2016) 14 SUPPL. 1 (34). Date of   
Publication: May 2016  
  
Background: anticoagulation therapy of SVT is a clinical challenge. Patients   
are at risk of developing certain complications and may experience   
recurrence. Anticoagulant therapy remains the cornerstone of treatment and   
should be started as soon as possible to prevent recurrence. Many patients   
are left untreated because the risks associated with anticoagulant therapy   
are calculated to exceed its benefits. However, the majority of patients   
receive anticoagulation with heterogeneous timing of initiation, drugs, and   
doses. Aims: To observe the length of anti-coagulation in splanchnic venous   
thrombosis. Methods: Retrospective, observational study of case charts of   
hospitalized patients diagnosed with SVT at Aga Khan Hospital Karachi during   
January to June 2015. Patients suffering SVT were identified by using ICD 9   
coding. Details were obtained from electronic medical record system.   
Results: SVT was found in 17 patients. Presenting compliant was abdominal   
pain in 9 patients. anticoagulation was not started in 8 patients because of   
either risk of bleeding or chronic nature of portal vein thrombosis. 5   
patients were started on Enoxaparin 60 mg twice daily and Warfarin (5-10 mg   
daily). Enoxaparin was stopped after achieving therapeutic INR and warfarin   
was continued. 4 patients were started only on warfarin (5-10 mg daily).   
Only 1 patient had bleeding due to warfarin after 20 days of initiation so   
it was stopped and he received enoxaparin for around 12 months. Out of other   
8 patients only one had unstable INR (i.e. time in therapeutic range< 60%).   
Warfarin was continued for more than 4 months in these patients with median   
time 5.3 months. No episode of recurrence was reported in any of these   
patients till to date. Conclusions: anticoagulation was started in patients   
having SVT immediately after the diagnosis with warfarin with or without   
enoxaparin. Median length of anticoagulation with warfarin was 5.3 months.   
This is an ongoing study so results may vary in final set of data.  
  
RECORD 83  
A rare pain in a common scenario  
Seth A. Shah M.A.  
Journal of General Internal Medicine (2016) 31:2 SUPPL. 1 (S524-S525). Date   
of Publication: May 2016  
  
LEARNING OBJECTIVE #1: Recognize pylephlebitis as a rare etiology of   
abdominal pain LEARNING OBJECTIVE #2: Identify the indications for   
anticoagulation CASE: A 62 year old Caucasian male with history of   
hypertension and remote cholecystectomy presented with 1 week ofmalaise,   
night sweats, fevers, and anorexia after consuming fast food. Three days   
prior to admission, he developed non- bloody, non-bilious emesis and   
non-radiating, cramping, epigastric pain not associated with meals. He   
denied preceding weight loss, changes in stool frequency, color, or caliber,   
dysuria, joint pains, or skin rashes. He had no recent sick contacts,   
travel, or family history of malignancy. Upon arrival, vital signs   
demonstrated temperature 101°Fahrenheit, blood pressure 74/53 mmHg, heart   
rate 105 beats/min, and respiratory rate 18 breaths/min. The patient had   
pallor, dry mucus membranes, diffuse abdominal tenderness with no rebound or   
guarding, splenomegaly without hepatomegaly, and cool extremities. Pertinent   
labs included leukocytosis of 14.2 K/ul, platelets 51 K/ul, creatinine 3.59   
mg/dL, bicarbonate 20 mmol/L, total bilirubin 6.9 mg/dL, direct bilirubin   
5.3 mg/dL, alkaline phosphatase 562 U/L, alanine transaminase 128 U/L,   
aspartate transaminase 152 U/L, venous lactate 46 mmol/L, and normal   
coagulation factors. Blood cultures grew Escherichia coli and Klebsiella   
pneumoniae. Abdominal computed tomography demonstrated a thrombus in the   
portal vein confluence and adjacent superior mesenteric vein with small   
bowel wall thickening. The presence of a thrombus prompted evaluation for   
malignancy and hypercoagulability disorder, respectively. No malignancy was   
found; however, he tested positive for antiphospholipid antibody syndrome   
(APLS). Ultimately, he was diagnosed with pylephlebitis secondary to small   
bowel enteritis and underlying APLS. Despite antibiotics and heparin, the   
patient developed recurrent abdominal pain and fever requiring partial   
resection of ischemic small bowel. DISCUSSION: Septic thrombophlebitis of   
the portal vein, or pylephlebitis, is a rare clinical entity. In the past,   
pylephlebitis was frequently associated with appendicitis; however, common   
etiologies today are biliary infections, colonic infections and   
hypercoagulable conditions. Symptoms are non-specific including fevers,   
chills, malaise, and abdominal pain. Bacteremia often occurs, particularly   
due to Escherichia coli, Klebsiella pneumoniae, Peptostreptococcus, and   
Bacteroides fragilis. Splenomegaly in the absence of hepatomegaly or chronic   
liver disease should prompt consideration of portal vein thrombosis.   
Splenomegaly is present in 10-25 % of patients with pylephlebitis. The   
cornerstone of treatment is antibiotics. Some patients may require surgical   
intervention if complications of pylephlebitis occur, which include hepatic   
abscess formation and small bowel ischemia. Patients with pylephlebitis   
should be evaluated for underlying malignancy, hypercoagulability disorders,   
inflammatory bowel disease, and human immunodeficiency virus. The   
indications for anticoagulation are controversial but include   
hypercoagulable state (i.e. APLS), persistent fever despite antibiotics, and   
thrombus involving the superior mesenteric vein due to risk of small bowel   
ischemia. Internists should have a high index of suspicion for pylephlebitis   
in patients presenting with abdominal pain and septic shock given the   
potential morbidity and mortality associated with this under-recognized   
condition. Additionally, physicians should consider the use of   
anticoagulation in patients with thrombus involving the superior mesenteric   
vein as this can lead to small bowel ischemia, as was the case in our   
patient. All cases of pylephlebitis should warrant work up for an underlying   
hypercoagulable state.  
  
RECORD 84  
Our clinical experience in the evaluation of mesenteric vein thrombosis  
Ilhan M. Bademler S. Azamat I.F. Baysal A. Kaan Gök A.F. Guloglu R. Kurtoglu   
M.  
Thrombosis Research (2016) 141 Supplement 1 (S43). Date of Publication: 1   
May 2016  
  
Background: Mesenteric vein thrombosis occurs rarely and is responsible for   
approximately 5-15% of all cases of acute mesenteric ischemia. The aim of   
this report was to discuss the management of mesenteric vein thrombosis   
based on our experience with 59 patients. Methods: In the present study, 59   
patients who were admitted to our emergency surgery department between   
January 2010 and July 2015 with a diagnosis of acute mesenteric ischemia   
were assessed retrospectively. Patients with peritoneal signs first   
underwent diagnostic laparoscopy to rule out perforation or bowel necrosis.   
All patients were administered 100 mg/kg of the anticoagulant enoxaparin   
twice daily. Results: CT angiography revealed superior mesenteric vein   
thrombosis in 14 (23%) patients, portal vein thrombosis in 6 (10%) patients,   
and splenic vein thrombosis in 2 (3%) patients. Four patients with   
peritoneal signs underwent diagnostic laparoscopy; two of the patients   
performed small bowel resection, anastomosis, and trocar insertion. In a   
patient reactional fluid and edema was seen in 60 cm small intestine and   
another patient 20 cm segmental edema seen and second look laparoscopy was   
made. Conclusions: Early diagnosis with CT angiography, conservative   
treatment with proper anticoagulation and laparoscopic second look detecting   
with supportive intensive care are the cornerstones of successful treatment   
of mesenteric vein thrombosis.  
  
RECORD 85  
Re-exploration after liver transplant-does it affect outcomes?  
Mehrotra S. Lalwani S. Mangla V. Nundy S. Mehta N.  
Transplantation (2016) 100:5 Supplement 1 (S241). Date of Publication: 1 May   
2016  
  
Introduction Re-exploration after any surgical procedure is technically more   
difficult and associated with higher morbidity and mortality. Few studies   
have shown outcomes of re-exploration after liver transplant which is   
expected to be worse than other surgical procedures Aim To assess the short   
term and long term outcomes in patients undergoing reexploration after liver   
transplant. Patients and Methods We analysed our prospectively collected   
data from January 2011 to July 2015 for patients undergoing re-exploration   
for various indications after liver transplant. Routine post-operative   
anticoagulation was not used in all the patients. The study group was   
compared with other patients who underwent liver transplant during the same   
period Results Re-exploration after liver transplant was done in 20 of   
210(9.5%). Bleeding was the most common indication for re-exploration in   
13(65%) patients and vascular complications were the cause in 5(25%)   
patients, while wound dehiscence and early bile leak in 1 patient each.   
Diaphragmatic surface was the most common site of bleeding in 7 patients   
while bile duct cut surface in 2 patients and hepatic artery surface in 1   
patient while no site was identified in 3 patients. Of the 5 patients with   
vascular complications 3 patients had portal vein thrombosis (1 of 3 had   
preoperative portal vein thrombosis) and 2 had arterial complications.   
Re-exploration was done after a mean period of 3.8 days for the whole group   
but patients who underwent re-exploration for bleeding were explored after   
mean of 1.1 days. Mean postoperative stay was 18 days. 4 of 20 (20%)   
patients expired within the study group and 3 of these patients were   
explored for bleeding and one for Portal venous thrombosis. 3 patients   
expired within 30 days of transplant while 1 patient expired after 2 months.   
On long term follow up 1 patient expired after 2 years of liver transplant.   
We compared our study group with other patients undergoing liver transplant   
during the same study period in terms of age, sex, MELD score, GRWR, blood   
transfusion, portal vein flow and postoperative morbidity, length of stay &   
mortality and found no statistical significant difference between the two   
groups apart from mortality which was higher in group undergoing   
re-exploration (20% vs 11%). Conclusions Re-exploration after liver   
transplant affects the short term outcomes without significant effect on   
long term results.  
  
RECORD 86  
Transient impact of treatment exposures and one-year incidence of thrombosis   
in multiple myeloma: A casetime-control analysis  
Brown J. Moga D. Adams V.  
Journal of Thrombosis and Haemostasis (2016) 14 SUPPL. 1 (122). Date of   
Publication: May 2016  
  
Background: Multiple myeloma (MM) has an inherent high risk of thrombosis of   
nearly exacerbated by specific treatment modalities. Aims: This study sought   
to assess the acute, transient impact of treatment- related exposures on the   
risk of thrombosis in MM. Methods: A case-time-control (CTC) analysis was   
conducted within a larger cohort of patients with MM. Individuals were   
identified by the first inpatient primary diagnosis of MM (ICD-9-CM 203.xx)   
from administrative claims during 2008-2013. Individuals included were over   
the age of 18 with continuous enrolment for 6 months preceding the index   
date. Subjects were followed until loss to follow-up, death, or a thrombotic   
event occurred (deep vein thrombosis, pulmonary embolism, arterial   
thrombosis, portal vein thrombosis). Cases included 502 subjects with at   
least 90 days of look-back preceding the thrombosis event. Cases were   
matched 1:4 with controls based by the year of MM diagnosis and controls   
were assigned the same event date as the case. Exposures were assessed in   
hazard (1-30 days) and comparison (61-90 days) periods preceding the event   
for cases and controls. Conditional logistic regression was used to compute   
adjusted odds ratios (aOR) for the transient effect of exposures on   
thrombosis. Exposures of interest included thalidomide/lenalidomide (IMIDs),   
protease inhibitors (PIs), steroids, cytotoxic agents, stem cell transplant,   
hospitalizations, and anticoagulation. Results: The cohort included 13,700   
individuals with 1,050 thrombotic events - a rate of 107.2 (100.9-113.9) per   
1,000 person-years. The CTC analysis showed transient risk associated with   
IMIDs used alone (aOR=1.5 [1.1-2.1]) or with PIs (aOR=1.6 [1.0-2.6]). Stem   
cell transplant had the highest transient impact on thrombosis (aOR=3.7   
[3.3- 4.2]). PIs alone had a lower impact on thrombosis (aOR=0.8 [0.5-1.4]).   
Conclusions: CTC results identify exposures with increased transient risk   
where surveillance and prophylaxis may be most useful.  
  
RECORD 87  
Endoscopic ultrasound guided injection of 2-octylcyanoacrylate for treatment   
of refractory bleeding from peristomal varices  
DeWitt J.M.  
Gastrointestinal Endoscopy (2016) 83:5 SUPPL. 1 (AB475-AB476). Date of   
Publication: May 2016  
  
Background: Bleeding peristomal varices are typically treated with topical   
therapy, octreotide, percutaneous parastomal embolization, TIPS or   
transplant. The role of EUS-guided injection of cyanoacrylate is limited to   
a single case report. Aim: To report to a prospective, single center case   
series of EUS-guided injection of 2-octylcyanoacrylate for treatment of   
refractory bleeding from peristomal varices. Methods: An endoscopic database   
of EUS-guided vascular therapy was queried for patients undergoing injection   
of peristomal varices. All patients had ongoing bleeding despite failed   
previous treatments or significant comorbidities precluding surgical,   
radiologic or other treatments. During EUS, potential varices for treatment   
were identified by doppler exam and traced distally to the stoma. Varix   
injection was performed with a 19g or 22g needle under EUS guidance. No   
coils were used for any patients. Follow up was performed by phone call and   
review of hospital records. Results: From 4/2013-10/2015, seven patients   
(mean age: 56 ± 11 yrs; 4F) were evaluated, including three hospitalized for   
peristomal bleeding. Bleeding was daily in three and at least once weekly in   
four. Six patients had cirrhosis (median MELD 10.5, range 9-38), whereas one   
had portal vein thrombosis requiring anticoagulation. Previous TIPS and   
liver transplant were performed in one patient each. Four patients had   
recently failed: endoscopic cautery in two, IV octreotide in one and   
surgical ligation in one. Prior to endoscopy, additional therapy was refused   
by surgery in all and radiology in two. CT in four showed peristomal varices   
in two but none in two. White light endoscopy into a previous ileostomy   
(nZ6) or colostomy (nZ1) performed a median 12 years (range 4-33) prior to   
referral was normal in all. Linear EUS located one (nZ4) or two (nZ2)   
varices (median diameter 4mm, range 4-8) in six, but no varix was found in   
one. Using a 19g (nZ5) or 22g (nZ1) needle, a median 1.5mL (range: 1-2) of   
2-octylcyanoacrylate (Dermabond) was injected in 0.75-1mL aliquots. Median   
follow up was 4.5 months (range 2-20). In 4/6 patients injected, bleeding   
resolved completely and in one permitted liver transplant 3 weeks later. In   
2/6, bleeding decreased significantly but did not resolve. In one of these   
two, repeat hosptialization for peristomal bleeding occurred 4 months after   
EUS followed by transplant one month later. No blood transfusions after EUS   
were required in any patient. Treatment in three hospitalized patients   
permitted eventual discharge. Adverse events included peristomal pain for   
less than one week in three and minor bleeding in one after injection which   
was treated with tamponade and application of bipolar probe to the stoma.   
Conclusion: EUS-guided injection of 2- octylcyanoacrylate to peristomal   
varices appears to be a promising technique to treat refractory bleeding in   
this population.  
  
RECORD 88  
Thrombin generation assay and its application in the clinical laboratory  
Tripodi A.  
Clinical Chemistry (2016) 62:5 (699-707). Date of Publication: 1 May 2016  
  
Background: A gap exists between in vivo and ex vivo coagulation when   
investigated by use of the coagulation tests prothrombin time (PT) and   
activated partial thromboplastin time (APTT). The thrombin generation assay   
(TGA) has been developed to fill this gap. CONTENT: TGA evaluates thrombin   
generation (resulting from the action of the procoagulant driver) and decay   
(resulting from the action of the anticoagulant driver), thus assessing the   
balance between the two. Coagulation of the test plasma (platelet poor or   
platelet rich) is activated by small amounts of tissue factor and   
phospholipids, and the reaction of thrombin generation is continuously   
monitored by means of a thrombin-specific fluorogenic substrate. Among the   
parameters derived from the thrombin-generation curve, the most important is   
the endogenous thrombin potential, defined as the net amount of thrombin   
that test plasmas can generate on the basis of the relative strength of the   
pro-and anticoagulant drivers. TGA is therefore the candidate assay to   
investigate hypo-or hypercoagulability. SUMMARY: From my analysis of the   
literature, I draw the following conclusions. There is strong evidence that   
TGA is helpful to elucidate coagulation mechanisms in various clinical   
conditions that until recently were poorly understood (chronic liver   
disease; diabetes; inflammatory bowel disease, myeloproliferative neoplasms,   
nonalcoholic fatty liver disease). TGA is a promising laboratory tool for   
investigating hemorrhagic coagulopathies and monitoring replacement therapy   
in hemophiliacs, predicting the risk of recurrent venous thromboembolism   
after a first event, and monitoring patients on parenteral or oral   
anticoagulants. These applications require clinical trials in which TGA   
results are combined with specific clinical end points.  
  
RECORD 89  
Hemostatic balance in patients with liver cirrhosis: Report of a consensus   
conference  
Andriulli A. Tripodi A. Angeli P. Senzolo M. Primignani M. Giannini E.G.   
Riggio O. Colli A. Prati D. Sacerdoti D. Merkel C. Basili S. Ferro D. Villa   
E. Di Minno G. Caraceni P. Marzioni M. Mannucci P.M. Violi F. Piscaglia F.   
Calvaruso V. De Pietri L. Falcone M. Feltracco P. Grandone E. La Mura V.   
Licata A. Lucidi C. Maimone S. Marietta M. Morisco F. Napoleone L. Piano S.   
Raparelli V. Rebulla P. Ribero D. Sartori M.T. Scalera A. Schepis F.   
Sicilianom M. Baroni G.S. Tufano A. Vitale A. Zuin M.  
Digestive and Liver Disease (2016) 48:5 (455-467). Date of Publication: 1   
May 2016  
  
Patients with cirrhosis present with hemostatic alterations secondary to   
reduced availability of pro-coagulant and anti-coagulant factors. The net   
effect of these changes is a rebalanced hemostatic system. The Italian   
Association of the Study of the Liver (AISF) and the Italian Society of   
Internal Medicine (SIMI) promoted a consensus conference on the hemostatic   
balance in patients with cirrhosis. The consensus process started with the   
review of the literature by a scientific board of experts and ended with a   
formal consensus meeting in Rome in December 2014. The statements were   
graded according to quality of evidence and strength of recommendations, and   
approved by an independent jury. The statements presented here highlight   
strengths and weaknesses of current laboratory tests to assess bleeding and   
thrombotic risk in cirrhotic patients, the pathophysiology of hemostatic   
perturbations in this condition, and outline the optimal management of   
bleeding and thrombosis in patients with liver cirrhosis.  
  
RECORD 90  
RUNX1 amplification increases the risk for thrombosis in children with   
B-cell acute lymphoblastic Leukemia  
Boucher M.O. Smitherman A.B. Pahl K.S. Rao K.W. Deal A.M. Blatt J.  
Journal of Pediatric Hematology/Oncology (2016) 38:3 (e125-e128). Date of   
Publication: 13 Apr 2016  
  
Background: RUNX1 (AML1) amplification in patients with B-cell acute   
lymphoblastic leukemia (B-ALL) has been associated with poor survival for   
unclear reasons. Our anecdotal experience suggests that children with B-ALL   
and RUNX1 amplification might be predisposed to thrombosis. Procedure: We   
performed a retrospective cohort study of children with B-ALL treated from   
2008 to 2014 at the North Carolina Children's Hospital. Patient   
demographics, cytogenetics, and diagnosis of thrombosis were extracted by   
blinded chart review. Analysis was performed examining the relationship   
between RUNX1 amplification and thrombosis. Results: We identified 119   
patients with B-ALL and a median age of 4.9 years (interquartile range, 2.9   
to 8.6 y) at diagnosis. Four patients (3%) had RUNX1 amplification. The   
average number of RUNX1 copies among those with amplification was 5 (SD 0.81   
[range, 4 to 6]). Eighteen thromboses were diagnosed within 6 months of   
starting treatment. These events were more likely among patients with RUNX1   
amplification than in patients without amplification (75% vs. 13%; RR 5.75,   
95% confidence interval, 2.75-12.01). Conclusions: RUNX1 amplification may   
predispose to early thrombotic events in children with B-ALL which could, in   
part, contribute to their poorer outcomes. Treatment implications, including   
possible prophylactic anticoagulation of patients with of RUNX1   
amplification, justify larger studies to confirm these findings.  
  
RECORD 91  
Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy: 3   
case reports and a literature review  
Muneer M. Abdelrahman H. El-Menyar A. Zarour A. Awad A. Al Dhaheri M.   
Al-Thani H.  
American Journal of Case Reports (2016) 17 (241-247). Date of Publication:   
12 Apr 2016  
  
Objective: Rare co-existance of disease or pathology Background:   
Porto-mesenteric venous thrombosis (PMVT) is an infrequent but severe   
surgical complication developing in patients who underwent laparoscopic   
bariatric surgery (sleeve gastrectomy). Herein, we describe the clinical   
presentation, management, and outcome of 3 rare cases of PMVT after   
laparoscopic sleeve gastrectomy (LSG), successfully treated at our center.   
Case Report: All patients developed PMVT post-LSG and presented with   
diffused abdominal pain, nausea, and vomiting. Computed tomography (CT) of   
the abdomen confirmed the diagnosis of portal vein thrombosis. Two patients   
were treated conservatively with anticoagulation and thrombolytic therapy   
and the third patient required operative intervention with bowel resection.   
Conclusions: PMVT is a rare presentation after LSG, which requires early   
diagnosis and management. Conservative management through anticoagulants and   
thrombolytic therapy is quite effective and, if indicated, should always be   
considered as the primary treatment option.  
  
RECORD 92  
The incidence of rethrombosis in patients with non-cirrhotic, non-tumoral   
chronic portal vein thrombosis-a prospective observational study  
Procopet B. Silva Jr. G. Llop E. Darnell A. Garcia-Criado M.A. Turon F.   
Baiges A. Calleja J.L. Bosch J. Hernandez-Gea V. Garcia-Pagan J.C.  
Journal of Hepatology (2016) 64:2 SUPPL. 1 (S432-S433). Date of Publication:   
April 2016  
  
Background and Aims: Non-cirrhotic, non-tumoral chronic portal vein   
thrombosis (NCPVT) is caused by a prothrombotic condition in 1/3 of cases, a   
local factor in other 1/3 of cases and is considered idiopathic in the   
remaining 1/3 patients (pts). In NCPVT, indefinite anticoagulation is   
recommended if a prothrombotic condition is found, if thrombosis is   
manifested by severe intestinal ischemia or if familial thrombotic history   
is present. However, the effectiveness of this attitude and/or the risk of   
rethrombosis in pts not anticoagulated and in relation with the underlying   
etiological conditions is scarce. The aim of the present study was to   
evaluate the rate of rethrombosis in the splanchnic venous system and the   
occurrence of any extrasplanchnic thrombotic events (EVE) in patients with   
chronic NCPVT. Methods: Patients prospectively included the REVASC registry   
and followed routinely with repeated angio-CT-scan or angio-MRI in order to   
assess the patency of the portal venous system were considered for   
inclusion. For splanchnic rethrombosis the patients were censored at the   
moment of rethrombosis or at the moment of the last imaging study. For EVE   
pts were censored at last day of follow-up. Results: 108 pts were included,   
39 with systemic prothombotic conditions (29 myeloproliferative diseases   
(MPD) and 10 with prothombotic abnormalities), 35 with an identifiable local   
factor and 34 were idiopathic, summing 652.75 person-years. During the   
follow-up 14 (13%) pts had rethrombosis (asymptomatic in 7). The actuarial   
rate of rethrombosis was 2, 6 and 12% at 1, 2 and 5 years, respectively. The   
incidence of rethrombosis was 2.15 (95% CI: 1.17-3.59) per 100 person-years.   
None of the 12 pts with NCPVT idiopathic/local with anticoagulation   
developed rethrombosis while this occurred in 11 out of 57 (19.2%) not   
receiving anticoagulation. Three patients of those with MPD (10%) and none   
of those with thrombophilia developed rethrombosis. During follow-up 11 pts   
had extrasplachnic thrombotic events, 5 of them with idiopathic/local NCPVT   
without anticoagulation. The remaining patients were under anticoagulation   
because had MPD (n = 4), thrombophilia (n = 1) and local factor with initial   
intestinal ischemia (n = 1). Conclusions: Rethrombosis in patients with   
local or idiopathic NCPVT not receiving anticoagulation is not a rare   
phenomenon. Efforts should be made to identify risk factors for rethrombosis   
in these patients.  
  
RECORD 93  
Portomesenteric thrombosis after laparoscopic sleeve gastrectomy  
Talishinskiy T. Eid S. Mazpule G. Novack R. Trivedi A. Ewing D. Schmidt H.  
Gastroenterology (2016) 150:4 SUPPL. 1 (S1245-S1246). Date of Publication:   
April 2016  
  
INTRODUCTION Laparoscopic sleeve gastrectomy is currently the most common   
bariatric surgery at our institution. Portomesenteric vein thrombosis is a   
relatively uncommon surgical complication with an insidious presentation and   
a high risk of bowel compromise. The purpose of this study was to present a   
series of patients who developed postoperative portomesenteric vein   
thrombosis after sleeve gastrectomy, and to identify the associated risk   
factors, overall incidence, clinical presentation and management. This is   
the largest case series presented from North America. METHODS This is a   
retrospective analysis of patients who underwent sleeve gastrectomy and   
developed portomesenteric vein thrombosis. Demographic data, personal risk   
factors, clinical presentation, and postoperative results of hypercoagulable   
work up were analyzed in this study. RESULTS A total of 2185 laparoscopic   
sleeve gastrectomies were performed from August 2011 till August 2015.   
Twelve patients (0.55%) developed portal vein thrombosis after surgery. Out   
of these patients seven were women and only two had a remote history of   
smoking. Mean BMI was 42.7 (range 37 - 49), mean age was 43.1 (range 18 - 53   
years). Mean operative time was 67 minutes (range 44 - 90 minutes).   
Abdominal pain was the most common symptom, presenting at a median of 14   
days (range 7 - 178 days) after sleeve gastrectomy. The most common   
laboratory abnormalities were elevated alkaline phosphatase and lipase. CT   
with IV contrast was performed in 11 patients, and was diagnostic in all.   
Due to a severe contrast allergy the twelfth patient had a non-contrast CT   
which was not diagnostic. Abdominal ultrasound identified portomesenteric   
thrombosis in this patient, but failed to demonstrate it in three of the   
other patients. Four patients in the study required additional surgical   
intervention, including 2 small bowel resections. All patients were treated   
with anti-coagulation with eventual resolution of symptoms. Six patients had   
repeat interval imaging, but only two of them were found to have   
radiographic resolution. Seven patients underwent hypercoagulable work up,   
revealing an abnormality in five. CONCLUSIONS Portal vein and mesenteric   
thrombosis is a relatively rare but morbid complication in patients   
undergoing bariatric surgery. Immediate diagnosis and a high index of   
suspicion is essential for the appropriate care of these patients.  
  
RECORD 94  
Inflammatory bowel disease is associated with an increased risk of arterial   
and venous thrombosis in a tertiary hospital-based patient cohort  
Gutta A. Redd M.K. Shah R. Jeepalyam S. Yousef O. Clarkston W.K.  
Gastroenterology (2016) 150:4 SUPPL. 1 (S562). Date of Publication: April   
2016  
  
BACKGROUND: Patients with inflammatory bowel disease are at an increased   
risk of thromboembolic events when compared to the general population. AIM:   
The aim of our study is to quantify the risk of arterial and venous   
thrombosis in hospitalized patients with Crohns disease and Ulcerative   
Colitis. METHODS: A retrospective analysis was conducted to evaluate all   
adult patients at Truman Medical Center, a primary teaching hospital for the   
University of Missouri-Kansas City from January 1st, 2010 to December 31st,   
2014 and 233,218 patients were identified. Using ICD-9 codes, a database   
search was undertaken to identify patients with inflammatory bowel disease   
as well as all patients diagnosed with arterial or venous thrombosis on   
admission or during their hospitalization. Odds ratio was calculated to   
assess the risk and was further adjusted using logistic regression. Patients   
with acute diverticulitis, liver cirrhosis, pancreatitis, pancreatic cancer   
and colon cancer were excluded from the study. RESULT: A total of 224,769   
patients admitted over a four-year period met the inclusion criteria.   
Analysis of these patients showed that 2,056 (0.9%) patients had   
thromboembolic events. 2,408 patients had IBD with 77 (3.2%) having arterial   
or venous thrombosis. The distribution of embolic events included; DVT (45),   
PE (27), unspecified arterial thrombosis (19), mesenteric arterial   
thrombosis (3), mesenteric venous thrombosis (3), cerebral venous thrombosis   
(1), cerebral artery thrombosis (1), and portal vein thrombosis (2).   
Patients with inflammatory bowel disease are 3.68 times (CI 2.92, 4.63) more   
likely to develop a thrombosis when compared to a hospital-based patient   
population without IBD. The risk decreased marginally to 2.88 (CI 2.23,   
3.73) when adjusted for age, race, sex, smoking and BMI. The risk of   
thrombosis in Crohns Disease was 2.64 (CI 1.49, 4.69) and Ulcerative Colitis   
was 4.35 (CI 2.38, 7.96). The patients were also more likely to have a PE   
with a risk of 3.32 (CI 2.26, 4.89) and a DVT with a risk of 3.53 (CI 2.62,   
4.77) compared to other types of thrombosis. There was also a statistically   
significant increased risk of arterial thrombosis at 7.8 (CI 5.1, 11.98)   
over venous thrombosis at 3.44 (CI 2.68, 4.42). CONCLUSION: Inflammatory   
bowel disease is associated with an increased risk of arterial and venous   
thrombosis. The risk is higher in Ulcerative Colitis compared with Crohns   
Disease, and there is a higher likelihood of DVT and PE, when compared to   
other types of thrombosis. Close attention to prophylaxis of thromboembolism   
in this patient population is critical. Further investigation must be done   
to determine specific risk factors for thromboembolic disease in the IBD   
patient population, and whether a high-risk subset of these patients would   
benefit from long-term anticoagulation.  
  
RECORD 95  
Tips versus endoscopy plus propranolol and anticoagulation for variceal   
rebleeding in cirrhotic patients with portal vein thrombosis: Results of a   
randomized controlled trial  
Qi X. He C. Yin Z. Wang Z. Zhang H. Xie H. Yao L. Wang J. Xia J. Cai H. Yang   
Z. Bai M. Guo W. Niu J. Wu K. Fan D. Han G.  
Journal of Hepatology (2016) 64:2 SUPPL. 1 (S167-S168). Date of Publication:   
April 2016  
  
Background and Aims: No consensus regarding the prevention of variceal   
rebleeding in cirrhotic patients with portal vein thrombosis (PVT) has been   
established. A randomized controlled trial was conducted to prospectively   
compare the efficacy of transjugular intrahepatic portosystemic shunt (TIPS)   
versus conventional treatment (i.e., endoscopic treatment, propranolol, and   
anticoagulants) in such patients. Methods: Cirrhotic patients with a history   
of variceal bleeding and PVT were randomized into TIPS with covered stents   
group and conventional treatment group. Primary endpoint was variceal   
rebleeding. Secondary endpoints included survival, complications, and portal   
vein recanalization. Results: In TIPS group, 23 of 24 patients successfully   
underwent TIPS procedures. In conventional treatment group, 5 of 25 patients   
were transferred to TIPS procedures. During a median follow-up period of   
20.9 months (range: 0.1-44.7), the 6- and 12-month cumulative rates of free   
of variceal rebleeding were significantly higher in TIPS group than in   
conventional treatment group (95% and 85% vs. 63% and 55%, p = 0.025). The   
12- and 24-month cumulative rates of overall survival were 83% and 73% in   
TIPS group and 88% and 84% in conventional treatment group (p = 0.228).   
Complete disappearance of main portal vein thrombosis was more frequently   
observed in TIPS group than in conventional treatment group (85% vs. 35%, p   
= 0.001). The 6- and 12-month cumulative rates free of hepatic   
encephalopathy were statistically similar between the two groups (77% and   
77% vs. 82% and 78%, p = 0.888). Two patients developed shunt dysfunction in   
TIPS group. Conclusions: Compared with conventional treatment, TIPS could   
prevent from variceal rebleeding and improve the rate of portal vein   
recanalization with a similar incidence of hepatic encephalopathy in   
cirrhotic patients with PVT.  
  
RECORD 96  
Diagnosis of epigastric pain in the puerperium: A rare case of portal system   
thrombosis with bilateral renal infarcts  
Howell C. Belchita A. Chaudry M. Ciantar E.  
BJOG: An International Journal of Obstetrics and Gynaecology (2016) 123   
SUPPL. 1 (33-34). Date of Publication: April 2016  
  
Case At 4 weeks post emergency caesarean section, a 25-year-old woman was   
admitted to hospital with epigastric pain and bilateral renal angle   
tenderness. Investigation with ultrasound was unremarkable and the pain   
settled. She was then readmitted with the same pain, requiring opiate   
analgesia, and raised inflammatory markers. A computed tomography (CT) scan   
of abdomen and pelvis revealed a portal vein thrombosis extending to the   
superior mesenteric vein and splenic vein, and bilateral renal infarcts. Her   
past medical and family history was unremarkable for venous thromboembolism,   
thrombophilias or portal system pathology. Paroxysmal nocturnal   
haemoglobulinuria and myeloproliferative disorders were excluded. She was   
commenced on anticoagulation under guidance of a multidisciplinary team.   
Discussion The hypercoagulable physiological state of pregnancy is   
well-known and predisposes women to venous thromboembolism in pregnancy and   
up to 6 weeks postpartum. Portal system thrombosis is a particularly rare   
event during pregnancy and postpartum. Episodes of portal system thrombosis   
in the puerperium are very few, and nearly all reports are case reports.   
Diagnosis is difficult because the clinical signs are nonspecific. The main   
symptom was upper abdominal pain, which may be wrongly interpreted as a   
gastric problem. Imaging with Doppler ultrasound and/or CT scan can lead to   
an early diagnosis and treatment. Most cases of portal vein thrombosis   
reported in the literature during pregnancy or postpartum occurred against a   
background of thrombophilia. In this case the patient had no personal or   
family history of thrombophilia. The mortality rate of portal system   
thrombosis in the puerperium is not higher than that of the general cases.  
  
RECORD 97  
Should anticoagulation be offered in patients with PVT in the setting of   
HCC?  
Mahmoudi T.M. Kayal A. Carvalho R. Azalgara V.M. Weiss A.  
Gastroenterology (2016) 150:4 SUPPL. 1 (S518-S519). Date of Publication:   
April 2016  
  
portal vein thrombosis (PVT) is a seen in about 20-44% of patients with   
hepatocellular carcinoma (HCC). To our knowledge, no other study has looked   
at the need for anticoagulation (AC) in patients with HCC and PVT. The aim   
of this study is to investigate the natural history and progression of PVT   
in patients with HCC with or without anticoagulation. Patients and Methods:   
A cohort of 60 patients, 54 from the British Columbia Cancer Agency database   
and 6 from the Vancouver general hospital thrombosis clinic, diagnosed with   
both conditions, were evaluated retrospectively. 9 patients were excluded   
secondary to lack of follow up. HCC and PVT diagnosis and follow up was made   
with contrast enhanced CT or MRI. Most patients received a single or a   
combination of the following treatments: Transarterial chemoembolization,   
radiofrequency ablation or surgical resection. 35(69%) of the patients   
received systemic therapy with Sorafenib. Among the 51 patients evaluated,   
12 patients received AC while 39 did not. Cox proportional hazards analysis   
was used to determine the survival benefit of AC use and Pearson chi squared   
analysis to access the impact of AC on PVT extension in this patient   
population. The date of November 26, 2015 was selected for administrative   
censoring since 6 patients are still alive. Results: 42 patients were males   
and mean age was 60.3. Liver disease etiology was HCV in 19(37%), HBV in   
23(45%), ETOH in 8(15%) and hemochromatosis in 1(2%). Average survival after   
HCC diagnosis was 32.9 months and 18.4 months after PVT diagnosis. After   
adjusting for age, HCC type at presentation (single VS multicentric) and   
Child's Pugh score (a marker of liver function), AC was associated with an   
improved survival after HCC diagnosis, adjusted hazard ratio was 0.37 (95%   
confidence interval CI 0.14 to 0.99) and after PVT diagnosis, 0.34 (95% CI   
0.13-0.88). PVT progression happened in 19(49%) of the non-AC group, and   
6(50%) of the AC group. AC did not impact PVT progression after adjusting   
for HCC type at presentation and Child's Pugh score. Odds ratio was 1.32(95   
% CI 0.41-4.19). No symptoms directly related to PVT development were   
reported. Dalteparin was used in 7 patients and warfarin in 5 patients for   
AC. Average length of AC was 7.8 months. Reasons for AC discontinuation   
were: 2 patients had an UGI bleed, 1 patient had an intracranial bleed   
secondary to brain metastasis, precautionary in high risk for bleed/fall in   
4 patients, recanalization in 2 patients, 1 death from liver disease   
decompensation. No reason was documented in 2 patients. Conclusion: This   
study demonstrates that PVT progression in patients with HCC is similar in   
both anticoagulated and non-anticoagulated groups. There were no symptoms   
attributable to PVT in these patients. The possible anticoagulation related   
complications need to be considered before attempting therapy in patients   
with HCC and PVT.  
  
RECORD 98  
High-risk non-alcoholic steatohepatitis liver transplant candidates are at   
the greatest risk for pre-transplantation portal vein thrombosis  
Stine J.G. Argo C.K. Pelletier S.J. Northup P.  
Gastroenterology (2016) 150:4 SUPPL. 1 (S1118). Date of Publication: April   
2016  
  
Purpose: Given that liver transplant recipients who receive an organ for   
high-risk nonalcoholic steatohepatitis (HR-NASH) have lower survival and   
that NASH is associated with increased portal vein thrombosis (PVT) risk, we   
hypothesize that liver transplant candidates with HR-NASH are at increased   
risk for PVT. Methods: Data on all transplants in the United States during   
the MELD era through September 2014 were obtained with permission from the   
United Network for Organ Sharing. Status 1a, multi-visceral, living donor,   
re-transplants, pediatric recipients, donation after cardiac death,   
recipients with pre-transplantation transjugular intrahepatic portosystemic   
shunts and hepatocellular carcinoma were excluded. Recipients were sorted   
into three distinct groups: those with HR-NASH, low-risk NASH (LRNASH) and   
non-NASH (all other etiologies except cryptogenic cirrhosis, which was   
excluded due to the potential for misclassification of NASH). HR-NASH was   
defined as the presence of the following: age >65, BMI >30 kg/m(2) and   
diabetes. Multivariable logistic regression models were constructed to   
assess independent risk factors for pre-transplant PVT. Findings: 35,959   
candidates underwent liver transplantation and of those organ recipients,   
505 were transplanted for HR-NASH and 2,796 for LR-NASH. 2,626 (7.5%) of   
recipients had pretransplant PVT, of which 68 (13.7%) were high-risk NASH   
versus 326 (11.9%) low-risk NASH (p<0.001). In general, NASH recipients were   
less likely to be male (p<0.001) or African American (p<0.001). While   
severity of clinical liver disease (moderate-severe ascites and   
encephalopathy) was similar amongst the groups, MELD scores both at listing   
(HRNASH 19.4, 95% CI 18.7-20.1, 20.1 95%CI 19.8-20.5, non-NASH 19.6, 95%CI   
19.5-19.7, p<0.001 when comparing HR to LR but no difference between HR and   
non-NASH) and at allocation (HR-NASH 22.5, 95% CI 21.8-23.4, LR-NASH 23.7,   
95% CI 23.4-24.0, non- NASH 22.8, 95% CI 22.7-22.9, p<0.001 when comparing   
HR-NASH to LR-NASH but no difference between HR and non-NASH) were greatest   
in the LR-NASH. BMI varied as well: HR-NASH 35.0 kg/m(2), 95% CI 34.7-35.4,   
LR-NASH 31.7 kg/m(2), 95% CI 31.5-32.0, non- NASH 27.8 kg/m(2), 95% CI   
27.7-27.9, p<0.001 for each within group comparison. In adjusted   
multivariable regression analysis, recipients with HR-NASH had the greatest   
risk of pre-transplant PVT with OR 2.05 (95% CI 1.57-2.67, p=0.001) when   
referenced to the non-NASH group and 33% greater risk when compared to   
LR-NASH (OR 1.72, 95% CI 1.49-1.97, p=0.044). Conclusions: Liver transplant   
candidates with HR-NASH are at the highest risk for PVT development. HR-NASH   
patients may benefit from prophylactic anticoagulation to decrease their   
likelihood of PVT formation and resultant downstream hepatic decompensating   
events. Prospective study investigating this seems warranted.  
  
RECORD 99  
Liver transplant recipients with pre-transplant portal vein thrombosis   
receiving an organ from a high-risk donor are at the highest risk for graft   
loss due to hepatic artery thrombosis  
Stine J.G. Argo C.K. Pelletier S.J. Northup P.  
Gastroenterology (2016) 150:4 SUPPL. 1 (S1034-S1035). Date of Publication:   
April 2016  
  
Purpose: To examine hepatic artery thrombosis (HAT) risk factors in liver   
transplant recipients. We hypothesize that recipients with pre-transplant   
portal vein thrombosis (PVT) who receive organs from high-risk donors are at   
increased risk of HAT. Methods: Data on all transplants in the United States   
during the MELD era through September 2014 were obtained from UNOS. Status   
1a, multi-visceral, living donor, re-transplants, pediatric recipients,   
donation after cardiac death and recipients with pre-transplantation   
transjugular intrahepatic portosystemic shunts (TIPS) were excluded.   
Recipients were sorted into two groups: those with HAT and those without   
HAT. Univariate comparisons were performed. Univariate comparisons were   
performed. High-risk donors were defined as a Donor Risk Index (DRI) >1.7.   
Incomplete HAT data were excluded. Multivariable logistic regression models   
were constructed to assess independent risk factors for HAT with resultant   
graft loss within 90 days of transplantation. Findings: 57,791 candidates   
underwent liver transplantation and of those organ recipients, 5,700 had   
complete data regarding HAT. 612 recipients had HAT and 65.7% (n=402) had   
received organs from high-risk donors compared to 60.9% (n= 3,097) in   
recipients without HAT (p=0.021). Pre-transplant PVT was found in 13.2% (n=   
81) of recipients with post-transplantation HAT versus 6.7% (n=339) in those   
without HAT (p<0.001). 2,079 (3.6%) recipients had pre-transplantation PVT   
and received organs from high-risk donors. Recipients with HAT had similar   
body mass index (28.4 m/kg2 95% CI 27.9-28.9 vs. 28.3 m/kg2, 95% CI   
28.1-28.5, p=0.678), cold ischemia time (7.32 hours, 95% CI 6.96-7.69 vs.   
7.43, 95% CI 7.32-7.55, p=0.530) and donor liver hepatic steatosis (8.5%,   
95% CI 6.6-10.3 vs. 8.9, 9% CI 8.3-9.6, p=0.187) when compared to recipients   
without HAT. On multivariable (adjusted) analysis, PVT and high risk donors   
were the most statistically significant independent risk factors for HAT (OR   
2.09 95% CI 1.41-3.07, p= 0.002) and this risk was 39% higher than in those   
recipients with PVT and a low risk donor (OR 1.80 95% CI 1.12-2.91,   
p=0.007). Recipients without PVT with a high risk donor did not have an   
increased risk of HAT. (OR 1.05 95% CI 0.82-1.35, p=0.169). Other   
significant covariates included male donors (OR 0.51 95% CI 0.41-0.62, p   
<0.001), hepatocellular carcinoma (OR 0.65 95% CI 0.48-0.90, p=0.008),   
heparin use at cross-clamp (OR 0.68, 95% CI 0.51-0.90, p=0.008), and   
international normalized ratio (INR) at transplantation (OR 0.86 95% CI   
0.75-0.99, p=0.033). Conclusions: Candidates with pre-transplant PVT who   
receive an organ from a high-risk donor are at the highest risk for   
post-operative HAT independent of other measurable factors. Recipients with   
pre-transplant PVT would benefit from careful donor selection and possibly   
anticoagulation perioperatively.  
  
RECORD 100  
Non-tumoral portalvein thrombosis and end-stage liver disease in patients   
with cirrhosis: A longitudinal retrospective cohort  
Roux J. Sultanik P. Bouam S. Vallet-Pichard A. Fontaine H. Corouge M. Mallet   
V. Pol S. Sogni P.  
Journal of Hepatology (2016) 64:2 SUPPL. 1 (S267-S268). Date of Publication:   
April 2016  
  
Background and Aims: The relationship between non-tumoral portal vein   
thrombosis (NTPVT) and occurrence of liver-related events in patients with   
cirrhosis is uncertain. We evaluated the relation between NTPVT and   
end-stage liver disease in a cohort of cirrhotic patients. Methods: We   
selected from the 2006 to 2015-hospital discharge database of our unit all   
patients with cirrhosis. We excluded patients who developed hepatocellular   
carcinoma during the study period, those who developed a NTPVT after ESLD   
and those with a follow-up below 6 months. Outcome measure was the   
occurrence of non-cancerous liver-related complication (End-Stage Liver   
Disease [ESLD]), including ascites, jaundice, hepatic encephalopathy, upper   
digestive bleeding or liver failure. We evaluated the relation between   
NTPVTand ESLD in a Cox proportional model adjusted for age, gender, HCV or   
HBV or HIV infections, alcohol use disorders, obesity, diabetes with time of   
follow-up as the time-scale. Charlson index was also determined at entry and   
tested in a separated model. Patients were censored at time of liver-related   
event or at the last consultation. Results: 623 patients (median age: 54   
years (IQR: 47-63), 395 (63%) men) with compensated cirrhosis were available   
at baseline. Median follow-up was 38 (IQR: 18-72) months. Positive anti-HCV   
Ab, HBsAg and anti-HIV Ab were present in 360 (58%), 53 (8%) and 44 (7%)   
patients, respectively. Alcohol use disorders, obesity and diabetes were   
present in 258 (41%), 38 (6%) and 90 (14%) patients, respectively. The   
occurrence of NTPVT was recorded in 20 (3.2%) patients and 80 (12.8%)   
patients developed ESLD. Patients with or without NTPVT were not different   
concerning age, sex or comorbidities. Alcohol use disorders (aHR: 1.93, p =   
0.004), HBsAg positivity (aHR: 3.26, p < 0.0001), obesity (aHR: 2.38, p =   
0.01) and NTPVT (aHR: 3.21, p = 0.001) were independent risk factors of ESLD   
(Figure 1). Adjusted on Charlson index, NTPVT remained independently   
associated with ESLD (aHR: 2.27, p = 0.002). Conclusions: NTPVT affects   
prognosis of patients with cirrhosis, regardless of comorbidities. This may   
suggest a benefit of preemptive anticoagulation in cirrhotic patients.   
(Figure Presented).  
  
RECORD 101  
Post-hepatectomy thrombosis: Evaluation of risk factors and clinical   
outcomes  
Han J.H. Kim D.-S. Yu Y.D. Jung S.W.  
HPB (2016) 18 SUPPL. 1 (e231). Date of Publication: April 2016  
  
Introduction: There have been a lot of reports for complications after   
hepatectomy. However, studies for hepatectomy related thrombosis including   
portal vein thrombosis have rarely reported. Authors evaluated risk factors   
for post-hepatectomy thrombosis and clinical outcomes. Methods: From   
February 2009 to December 2014, we analyzed retrospectively 534 patients who   
had undergone hepatectomy at our hospital. The post-hepatectomy thrombosis   
was defined as thrombosis which was seen in portal vein, hepatic vein and   
inferior vena cava on postoperative imaging study. The patients with   
pre-operatively confirmed thrombosis and tumor recurrence related thrombosis   
were excluded. Results: Of the 534 patients, 22 (4.1%) developed thrombosis   
after hepatectomy. Among them, portal vein thrombosis was 19 (86.4%) and   
other site (hepatic vein and inferior vena cava) was 3 (13.6%). Proportion   
of the patients who resected more than two sections such as lobectomy was   
higher in post-hepatectomy thrombosis group (54.5 vs 35.2%). Patients with   
thrombosis had a significantly longer operation time (p = 0.001) and it   
occurred more commonly in cholangiocarcinoma patients in this study (p =   
0.022). Although, there was no statistically significance, the mean duration   
of Pringle's maneuver was longer in post-hepatectomy thrombosis group (24.3   
vs 17.7 minutes). 13 (59.1%) were received anticoagulation therapy and   
almost of them (12 patients) were improved. Conclusion: Large resection   
volume, longer operative time and duration of Pringle's maneuver are seemed   
to be related with higher incidence of post-hepatectomy thrombosis.   
anticoagulation therapy is recommended for almost patients without   
contraindications, especially for main portal vein thrombosis or possibility   
of main portal flow disturbance.  
  
RECORD 102  
Enoxaparin reduces hepatic vascular resistance and portal pressure in   
cirrhotic rats  
Cerini F. Vilaseca M. Lafoz E. García-Irigoyen O. García-Calderó H. Tripathi   
D.M. Avila M. Reverter J.C. Bosch J. Gracia-Sancho J. García-Pagán J.C.  
Journal of Hepatology (2016) 64:4 (834-842). Date of Publication: 1 Apr 2016  
  
Background & Aims Increased hepatic vascular resistance due to fibrosis and   
elevated hepatic vascular tone is the primary factor in the development of   
portal hypertension. Heparin may decrease fibrosis by inhibiting   
intrahepatic microthrombosis and thrombin-mediated hepatic stellate cell   
activation. In addition, heparin enhances eNOS activity, which may reduce   
hepatic vascular tone. Our study aimed at evaluating the effects of acute,   
short-, long-term and preventive enoxaparin administration on hepatic and   
systemic hemodynamics, liver fibrosis and nitric oxide availability in   
cirrhotic rats. Methods Enoxaparin (1.8 mg/kg subcutaneously), or its   
vehicle, was administered to CCl(4)-cirrhotic rats 24 h and 1 h before the   
study (acute), daily for 1 week (short-term) or daily for 3 weeks   
(long-term) and to thioacetamide-cirrhotic rats daily for 3 weeks   
with/without thioacetamide (preventive/long-term, respectively). Mean   
arterial pressure, portal pressure, portal blood flow, hepatic vascular   
resistance and molecular/cellular mechanisms were evaluated. Results No   
significant changes in hemodynamic parameters were observed in acute   
administration. However, one-week, three-week and preventive treatments   
significantly decreased portal pressure mainly due to a decrease in hepatic   
vascular resistance without significant changes in mean arterial pressure.   
These findings were associated with significant reductions in liver   
fibrosis, hepatic stellate cell activation, and desmin expression. Moreover,   
a reduction in fibrin deposition was observed in enoxaparin-treated rats,   
suggesting reduced intrahepatic microthrombosis. Conclusion Enoxaparin   
reduces portal pressure in cirrhotic rats by improving the structural   
component of increased liver resistance. These findings describe the   
potentially beneficial effects of enoxaparin beyond the treatment/prevention   
of portal vein thrombosis in cirrhosis, which deserve further investigation.  
  
RECORD 103  
Nontumoral portal vein thrombosis in patients awaiting liver transplantation  
Chen H. Turon F. Hernández-Gea V. Fuster J. Garcia-Criado A. Barrufet M.   
Darnell A. Fondevila C. Garcia-Valdecasas J.C. Garcia-Pagán J.C.  
Liver Transplantation (2016) 22:3 (352-365). Date of Publication: 1 Mar 2016  
  
portal vein thrombosis (PVT) occurs in approximately 2%-26% of the patients   
awaiting liver transplantation (LT) and is no longer an absolute   
contraindication for LT. Nearly half of PVT cases are accidentally found   
during the LT procedure. The most important risk factor for PVT development   
in cirrhosis may be the severity of liver disease and reduced portal blood   
flow. Whether other inherited or acquired coagulation disorders also play a   
role is not yet clear. The development of PVT may have no effect on the   
liver disease progression, especially when it is nonocclusive. PVT may not   
increase the risk of wait-list mortality, but it is a risk factor for poor   
early post-LT mortality. anticoagulation and transjugular intrahepatic   
portosystemic shunt (TIPS) are 2 major treatment strategies for patients   
with PVT on the waiting list. The complete recanalization rate after   
anticoagulation is approximately 40%. The role of TIPS to maintain PV   
patency for LT as the primary indication has been reported, but the safety   
and efficacy should be further evaluated. PVT extension and degree may   
determine the surgical technique to be used during LT. If a "conventional"   
end-to-end portal anastomotic technique is used, there is not a major impact   
on post-LT survival. Post-LT PVT can significantly reduce both graft and   
patient survival after LT and can preclude future options for re-LT. Liver   
Transpl 22:352-365, 2016.  
  
RECORD 104  
Institutional review of therapeutic enoxaparin hemorrhagic complications in   
morbid obesity  
Carraro E.A. Mikami D.J. Needleman B.J. Noria S.F.  
Surgical Endoscopy and Other Interventional Techniques (2016) 30 SUPPL. 1   
(S481). Date of Publication: March 2016  
  
Introduction: Enoxaparin, a low molecular weight heparin, is often used   
prophylactically to reduce the risk of thromboembolic events, and   
therapeutically to bridge to full oral anticoagulation postoperatively. In   
individuals with obesity (BMI >30 kg/m(2)), there is concern regarding the   
optimal dosing as drug distribution and pharmacokinetics may be altered.   
While laboratory evaluation with anti-Xa levels has been proposed, risk of   
thrombosis and hemorrhage have not been shown to correlate well with anti-Xa   
levels. Overall risk of major hemorrhagic complications on low molecular   
weight heparin is 1.1 %, however we noted several bleeding complications in   
our patient population and decided to evaluate our experience and identify   
risk factors that may be contribute to hemorrhagic complications. Methods: A   
retrospective chart review was performed on postoperative patients   
discharged, from a single surgical service, on therapeutic enoxaparin as a   
bridge to full anticoagulation from “year start”to “year finish”.   
Demographic informations, surgical intervention and surgical complications   
were reviewed to assess risks related to anticoagulation. Results: A total   
of 41 patients met the inclusion criteria. The mean age was 49.8 years with   
68 % females and a mean BMI of 47.0 kg/m(2). Surgical interventions   
included, sleeve gastrectomy (36.6 %), abdominal wall hernia repair (26.8   
%), Roux en Y gastric bypass (22 %), exploratory laparotomy (9.7 %) and   
others (2.4 %). The indication for anticoagulation included a history of   
deep vein thrombosis/pulmonary embolism (75.6 %), atrial fibrillation (14.6   
%), portal vein thrombosis (4.9 %), and other (4.9 %). Fifteen (36.6 %)   
patients were readmitted for complications directly related to their   
surgical intervention. Of these, 3 (7.6 %) were admitted secondary to   
hemorrhagic complications and specific interventions included, (1) medical   
management with blood transfusion and reversal of supratherapeutic   
anticoagulation, (2) transfusion and stenting of subsequent, possible   
resultant, gastric sleeve leak, and (3) transfusion and empiric   
embolization. Conclusions: Post-operative bridging of morbidly obese   
patients with therapeutic enoxaparin should be approached with caution as   
the incidence of hemorrhagic complications may be greater than expected.   
However, further studies are needed to identify those at increased risk of   
complications including more consistent evaluation of anti-Xa levels, both   
at initial administration and at readmission, in order to adjust dosing or   
pursue alternative options for anticoagulation.  
  
RECORD 105  
portal vein thrombosis after laparoscopic bariatric surgery it's a rare   
complication but should be considered: Description of three cases with   
literature review  
Ghasoup A. Qurashi T.A. Widinly M. Sadieh O.  
Surgical Endoscopy and Other Interventional Techniques (2016) 30 SUPPL. 1   
(S454). Date of Publication: March 2016  
  
Background: portal vein thrombosis (PVT) refers to an obstruction in the   
trunk of the portal vein it's an uncommon complication after Laparoscopic   
Bariatric Surgery (LBS) However it is a potentially lifethreatening   
condition reported after laparoscopic bariatric surgery. Clinical symptoms   
may be insidious, and progression can lead to intestinal infarction and   
portal hypertension. Main Outcome Measures: Systematic review of the   
literature on PVT after LBS and report three cases encountered at our   
institution. Patients and Methods: We reviewed the literature between   
January 1990, and January 2015, using the search terms portal vein   
thrombosis, mesenteric venous thrombosis, laparoscopic surgery and bariatric   
surgery. The inclusion criteria were documented PVT by imaging studies such   
as angiography, ultrasonography, computed tomography [CT], or magnetic   
resonance imaging (MRI) or surgery following LBS. We include three cases   
after laparoscopic sleeve gastrectomy from our institution. Results: One   
developed a chronic cavernoma with extension of the thrombus to the superior   
mesenteric vein and splenic vein, the other two cases recovered using   
anticoagulation therapy. Conclusions: PVT is a rare complication after LBS,   
however Laparoscopic surgeons should be aware of the risk of PVT, and it   
should be suspected in cases with an atypical outcome after LBS. Once PVT is   
diagnosed, prompt anticoagulation therapy may resolve the thrombotic event.  
  
RECORD 106  
Trousseau’s syndrome in cholangiocarcinoma: The risk of making the diagnosis  
Blum M.F. Ma V.Y. Betbadal A.M. Bonomo R.A. Raju R.R. Packer C.D.  
Clinical Medicine and Research (2016) 14:1 (53-59). Date of Publication: 1   
Mar 2016  
  
We report a case of Trousseau’s syndrome with cholangiocarcinoma complicated   
by a fatal pulmonary embolism after liver biopsy. A 69-year-old man who   
presented with right upper quadrant pain was found to have portal vein   
thrombosis and nonspecific liver hypodensities after imaging by   
comput­erized tomography. Following four days of anticoagulation, heparin   
was held for percutaneous liver biopsy. After the biopsy, he developed acute   
hepatic failure, acute kidney injury, lactic acidemia, and expired. Autopsy   
revealed intrahepatic cholangiocarcinoma and a pulmonary embolism.   
Trousseau’s syndrome with cholangiocarcinoma is rarely reported and has a   
poor prognosis. This case highlights a fundamental challenge in the   
diagnosis and early management of intrahepatic cholangiocarcinoma with   
hypercoagulability. Diagnostic biopsy creates an imperative to reduce   
post-operative bleeding risk, but this conflicts with the need to reduce   
thrombotic risk in a hypercoagulable state. Considering the risk of   
withholding anticoagulation in patients with proven or suspected   
cholangiocarcinoma complicated by portal vein thrombosis, physicians should   
consider biopsy procedures with lesser bleeding risks, such as transjugular   
liver biopsy or plugged percutaneous liver biopsy, to minimize interruption   
of anticoagulation.  
  
RECORD 107  
Occult portal venous system thrombosis complicating acute pancreatitis:   
Three case reports and a literature review  
Li S. Shang D. Varghese H.J. Liu M. Li X. Tong M.  
International Journal of Clinical and Experimental Medicine (2016) 9:2   
(3621-3627). Date of Publication: 29 Feb 2016  
  
Portal venous system thrombosis (PVT) is a relative rare complication of   
acute pancreatitis (AP), especially in China, and the incidence thereof in   
published studies may be overestimated. The management of PVT complicat­ing   
AP by the use of anticoagulation therapy remains controversial due to the   
lack of standardized treatment. We herein report three cases of occult PVT   
complicating AP. Referring to the literatures and our clinical experiences,   
if the thrombosis detected recently and lack of evidence of bleeding   
tendencies, anticoagulation therapy is safe and is not associated with an   
increase in major complication. Since the study was done only in three   
cases, the necessity of implementing anticoagulation therapy in PVT   
complicating AP will require more supportive data in future as more   
evidence-based data emerges.  
  
RECORD 108  
anticoagulation therapy for non malignant portal vein thrombosis in   
cirrhotic patients: A safe treatment?  
Sbrancia M. Antonelli E. Bassotti G. Clerici C. Morelli O.  
Digestive and Liver Disease (2016) 48 SUPPL. 2 (e82). Date of Publication:   
24 Feb 2016  
  
Background and aim: Non-neoplastic portal vein thrombosis (PVT) is a   
frequent event in cirrhotic patients but its natural history is poorly   
understood. It can be treated with anticoagulants, however the safety and   
efficacy of this therapeutic approach are still unknown. We performed a   
retrospective study evaluating the effect of anticoagulants in a series of   
cirrhotic patients with nonneoplastic PVT. Material and methods: A   
retrospective ultrasound chart review of cirrhotic patients seen in our   
Liver Unit between February 2008 and March 2015 was performed. Subjects with   
non-neoplastic PVT (defined as the absence of invasion or infiltration of   
the portal vein by neoplasia) were identified by reviewing US and TC   
reports. Partial vs complete PVT was considered as the absence or presence   
of power- Doppler signal at the ultrasound. Demographic, clinical,   
laboratory, endoscopic parameters and thrombophilia screening were analyzed.   
Dose, duration, efficacy and side effects of anticoagulant therapy were also   
evaluated. Results: Charts of 375 cirrhotic patients of any etiology were   
evaluated. Non-neoplastic PVT was identified in 28 cases (7,5%) and it was   
mostly partial. Low platelet count, high MELD score (13±4), Child-Pugh class   
B or C and esophageal varices were the most frequent characteristics of   
these patients. Trombophilic disorders (antithrombin deficiency, protein C   
deficiency, protein S deficiency, presence of Lupus Anticoagulant   
antibodies) were observed in 9 patients; 16 patients received   
anticoagulation therapy (low-weight heparin or warfarin) for 3-6 months and   
12 patients received no treatment. Partial or complete recanalization was   
achieved in 12 anticoagulated patients (75%), while in 3 patients (25%)   
spontaneous improvement of PVT (p=0,025) was observed. The recurrence of   
thrombosis was seen in 43% patients after stopping anticoagulation therapy.   
Five anticoagulated patients developed bleeding complications but no deaths   
were observed. Ten patients without treatment developed liver-related events   
(portal hypertension-related bleeding, ascites, hepatic encephalopathy) and   
4 patients died. Conclusions: In our study, anticoagulation therapy is a   
safe treatment for PVT, leading to recanalization of the portal vein in 75%   
of patients. It seems to be reasonable to maintain indefinitely the   
anticoagulation therapy to prevent thrombosis recurrence.  
  
RECORD 109  
Abstracts 22nd National Congress of Digestive Diseases, Italian Federation   
of Societies of Digestive Diseases - FISMAD 2016  
Digestive and Liver Disease (2016) 48 SUPPL. 2. Date of Publication: 24 Feb   
2016  
  
The proceedings contain 105 papers. The topics discussed include:   
anticoagulation therapy for non malignant portal vein thrombosis in   
cirrhotic patients: a safe treatment?; drug-eluting beads versus   
conventional chemoembolization for the treatment of hepatocellular   
carcinoma: a meta-analysis; risk factors for the occurrence of sporadic   
pancreatic neuroendocrine tumours: a multicenter European study (EPINET);   
European colonoscopy quality investigation group: improving standards in   
colonoscopy through a practice level audit tool; serum determination of   
squamous cellular carcinoma antigen as a biomarker of Barrett's esophagus   
and esophageal cancer: a phase III study; laryngopharyngeal symptoms in   
primary care: usefulness of salivary pepsin measurement in predicting gerd;   
oxidative stress and thromboxane-dependent platelet activation in   
inflammatory bowel disease (IBD): effects of anti-TNF-ALFA treatment; and   
knockdown of SMAD7 with mongersen attenuates colitis and colitis-driven   
fibrosis in mice.  
  
RECORD 110  
Efficacy and safety of treatment of acute nonmalignant portal vein   
thrombosis with subcutaneous fondaparinux in patients with cirrhosis and   
marked thrombocytopenia  
Tonon M. Piano S. Sacerdoti D. Dalla Valle F. Spiezia L. Bolognesi M.   
Simioni P. Angeli P.  
Digestive and Liver Disease (2016) 48 SUPPL. 1 (e25-e26). Date of   
Publication: 10 Feb 2016  
  
Introduction and aims: Fondaparinux (FPX), a factor Xa inhibitor, has been   
recommended for anticoagulation therapy in patients deep vein thrombosis. It   
rarely induces thrombocytopenia since anti-PF4/heparin antibodies which are   
rarely generated during FPX treatment, are not able to bind PF4/FPX   
complexes. Despite these potential advantages, there are no data regarding   
the use of FPX as anticoagulant treatment of acute nonmalignant portal vein   
thrombosis (PVT) in patients with liver cirrhosis. The aim of this   
prospective pilot study was to evaluate the safety, and efficacy of   
subcutaneous FPX therapy in patients with cirrhosis and PVT. Methods:   
Forty-two patients with liver cirrhosis and acute nonmalignant PVT were   
included in the study between 2010 and 2014. The extension of PVT at   
baseline and its evolution on treatment were evaluated by both Doppler   
ultrasound and CT. The dose of FPX was adjusted to body weight, and on   
platelet count, ranging from 2.5 to 7.5 mg/die. Results: At baseline, 31   
patients (74%) had a platelet count lower than 70×10(3)/mm(3), while 19   
(45%) had a platelet count < 50×10(3)/mm(3). After a mean period of 16±14   
months of treatment, 18 patients (43%) showed a complete resolution of PVT.   
Ten patients (24%) showed a partial resolution. Fourteen patients (33%)   
showed no response or a progression of thrombosis. One patient developed a   
non-lethal major bleeding event (haemoperitoneum three day after   
paracentesis) while 5 patients developed a non-major bleeding events during   
the study (14%). In all these patients but one FPX was discontinued. No   
significant change was observed in the platelet count before and during   
treatment (67×10(3)/mm(3) vs 68×10(3)/mm(3), p = N.S.). Conclusions: In   
patients with cirrhosis and marked thrombocytopenia, FPX seems to be   
effective and safe in the treatment of acute nonmalignant PVT.  
  
RECORD 111  
Treatment of portal vein thrombosis in cirrhosis: A multicenter real life   
study  
Samonakis D.N. Triantos C.K. Gatselis N. Thalheimer U. Leandro G. Mantaka A.   
Zachou K. Konstantakis C. Saitis A.I. Thomopoulos K. Dalekos G.N.   
Kouroumalis E.A.  
Hepatology International (2016) 10:1 SUPPL. 1 (S374). Date of Publication:   
February 2016  
  
Aims: portal vein thrombosis (PVT) is common in cirrhosis and can be cause   
or consequence of disease progression. Small studies have shown benefit of   
anticoagulation. We assessed anticoagulation on this population for safety,   
efficacy and survival. Method: Cirrhotics with PVT, the majority   
decompensated, were included in a data base retrospectively (before 2013)   
and prospectively (2013-10/2014). Demographics: 76 patients (61 male),   
median age 67 (36-88) and BMI 26.8 (17.9-32.4), etiologies: alcoholic 40 %,   
HBV 25 % and HCV 16 %, HCC in 47.4 %. Median MELD-score was 12 (6-25),   
Child-Pugh 7 (5-12).79.5 % of patients were decompensated at PVT diagnosis,   
89.6 % had varices (62.5 % large), 33 % high-risk signs. Main trunk   
involvement in 77 %, cavernoma existed in 17 %. 51 patients anticoagulated   
(65 % LMW-Heparin, 25 % warfarin). Pretreatment varices eradicated in 30 %,   
while a 75 % of patients were on beta-blockers. Survival was inferior for   
treated (median 15 months) albeit not statistically (ns)-significant (p =   
0.311); HCC patients had n.s. trend for inferior survival as for alcoholics   
(p = 0.06). PV patency 28.5 % of treated (n.s). Portal hypertension (PHT)   
bleeding identified in 24 patients (31.6 %), only in 6 after PVT diagnosis,   
2 under treatment (fatal). Two patients experienced non- PHT   
gastrointestinal bleeding (1 fatal). Majority (75 %) of deaths were due to   
liver failure and HCC-related causes. Conclusion: Treatment of PVT in   
cirrhotics is feasible with acceptable side-effects. Alcoholic etiology and   
HCC have negative impact on survival. In our cohort there was no clear   
benefit of treating PVT in cirrhotics, mainly decompensated. A bias,   
commencing anticoagulation in patients with more advanced disease, cannot be   
excluded.  
  
RECORD 112  
Successful percutaneous thrombectomy for portal vein thrombosis following   
liver transplant  
Rodriguez-Payan N. Zaragoza-Organista R. Zaragoza-Solis S.I. Chavez-Perez R.   
Chavez-Appendini R. Garcia-Gallegos V. Rodriguez-Sancho L.C. Garcia-Moreno   
A.S. Moreno-Luna L.E.  
Hepatology International (2016) 10:1 SUPPL. 1 (S431). Date of Publication:   
February 2016  
  
This is the case of a 48-year-old woman with type 2 Diabetes, chronic   
hypertension, and HCV cirrhosis presented a Child Pugh Score of B. Patient   
received an orthotopic liver transplant (OLT), during the surgery a chronic   
portal vein thrombosis was found in the receiver, performing thrombectomy of   
the thrombus without complete success. During the first 48 h the patient   
presented elevated hepatic enzymes, refractory ascites, with an excess drain   
of more than 5 liters per day, and general deterioration. Due to the   
extension of the clot and the torpid evolution of the patient, surgical   
thrombectomy is performed. Nevertheless, due to the morphological   
characteristics and the chronicity of the thrombus, the complete extraction   
is not achieved. The day after the second intervention, deterioration of the   
renal and hepatic function with hyperamylasemia and metabolic acidosis is   
found. A magnetic resonance imaging shows an increase of the thrombus   
extension. Procoagulant factors were analyzed and an antithrombin III   
deficiency was found. Percutaneous thrombectomy was realized by the   
interventional radiologists, with no immediate complications post-procedure   
observed. At the end of the procedure permeable flows were corroborated with   
doppler ultrasound. After the procedure, anticoagulation with low molecular   
weight heparin is administered. After 3 weeks the patient is discharged with   
renal and hepatic function improvement. Actually, the patient is alive 10   
months post OLT, with normal renal and hepatic function. She is being   
treated with tacrolimus, mofetilic acid, and oral anticoagulants. Control   
ultrasounds and hepatic function tests are normal.  
  
RECORD 113  
Danaparoid sodium thrombolytic therapy followed by warfarin in cirrhotic   
portal vein thrombosis  
Kawamura E. Enomoto M. Kotani K. Motoyama H. Kozuka R. Hagihara A. Yamamoto   
A. Uchida-Kobayashi S. Morikawa H. Kawabe J. Murakami Y. Tamori A. Shiomi S.   
Kawada N.  
Hepatology International (2016) 10:1 SUPPL. 1 (S374). Date of Publication:   
February 2016  
  
Background: portal vein thrombosis (PVT) is a complication of cirrhosis that   
reduces the hepatic reserve and causes variceal bleeding. The therapeutic   
efficacy of danaparoid sodium (DS), a heparinoid anti-coagulation factor Xa,   
for PVT has been reported. Methods: We retrospectively analyzed 41   
hospitalized cirrhotic patients: 16 hepatitis C virus, 5 hepatitis B virus,   
20 others; the model for end-stage liver disease (MELD) score 8.6 ± 4.7;   
platelets 80 ± 40 × 10(3)/μL; 3 esophageal varices F0, 16 F1, 5 F2, 0 F3,   
and 17 unknown. DS 2500 units were administered daily (n = 41, mean   
duration: 9.5 days), followed by oral warfarin (prothrombin   
time-international normalized ratio: 1.5 ± 0.3) in outpatient clinic (n =   
16, 25.8 weeks). The volume of PVT (PVTV) measured with a threedimensional-   
image analyzer (n = 28), serum D-dimer (n = 29), and scintigraphic portal   
shunt indices (normal, <10 %; n = 6) were monitored. Results: Thrombi formed   
at one site in 25 patients (18 portal, 4 superior mesenteric, and 3 splenic   
veins) and at two or more sites in 16. At the end of DS therapy, the PVTV   
decreased to 55.1 ± 40.2 % of baseline (8.6 ± 10.3 cm(3), p<0.0001), D-dimer   
decreased from 11.8 ± 12.6 μg/mL to 7.0 ± 7.4 μg/mL (p = 0.007), and the   
shunt indices decreased from 62.4 ± 10.5 % to 56.9 ± 7.1 % (p = 0.250).   
During DS therapy, Grade 2 intraperitoneal bleeding occurred in one patient   
(2.4 %). During follow-up, PVTV increased in 33.3 % of the patients, MELD   
score increased in 37.5 %, platelets decreased in 50.0 %, and varices grade   
increased in 18.2 %. Conclusions: PVT could be resolved with DS with safety.   
Warfarin did not always maintain the effects of DS.  
  
RECORD 114  
portal vein thrombosis: When to treat and how?  
Sharma A.M. Zhu D. Henry Z.  
Vascular Medicine (United Kingdom) (2016) 21:1 (61-69). Date of Publication:   
1 Feb 2016  
  
portal vein thrombosis is an unusual thrombotic condition not frequently   
seen in the general population; however, it has a higher prevalence in   
special circumstances such as in liver cirrhosis and hepatic or pancreatic   
malignancy. It also can be associated with significant morbidity and   
mortality. In this review, we discuss the current data available to guide   
therapy in the setting of different associated co-morbidities,   
hypercoagulable states, and associated thrombosis of the remaining   
splanchnic circulation. We discuss indications for anticoagulation,   
including the choice of anticoagulants, as well as the role of conservative   
'wait and watch' and invasive therapies, such as thrombolysis, thrombectomy,   
and transjugular intrahepatic portosystemic shunt.  
  
RECORD 115  
Thromboembolism and anticoagulation management in the preterm infant  
Rajagopal R. Cheah F.-C. Monagle P.  
Seminars in Fetal and Neonatal Medicine (2016) 21:1 (50-56). Date of   
Publication: 1 Feb 2016  
  
The incidence of preterm thromboembolism has been increasing due to advances   
in diagnostic imaging which allow better detection of thrombi in sick   
preterm infants. At the same time, improvement in neonatal intensive care   
unit supportive care has increased the number of surviving and living   
preterm infants with thromboembolic risk factors. Disruption in the fine   
balance of hemostasis with potential risk factors, specifically septicemia   
and indwelling catheters, increase the occurrence of thromboembolic events.   
Treatment strategies in preterm infants are challenging due to limited data.  
  
RECORD 116  
anticoagulation in Patients With cirrhosis: Caught Between a Rock-Liver and   
a Hard Place  
Ha N.B. Regal R.E.  
Annals of Pharmacotherapy (2016) 50:5 (402-409). Date of Publication: 2016  
  
Objective: To review current literature for anticoagulation in patients with   
cirrhosis and provide a summary of the effects of cirrhosis on the   
coagulation cascade, therapeutic monitoring through interpretation of   
antifactor Xa (anti-Xa), activated partial thromboplastin time (aPTT), and   
international normalized ratio (INR) as well as current prophylaxis and   
treatment recommendations in cirrhotic patients. Methods: A systematic   
electronic literature search was conducted in PubMed using the key terms   
anticoagulation, warfarin, low-molecular-weight heparin (LMWH),   
unfractionated heparin (UFH), target-specific oral anticoagulants, deep-vein   
thrombosis (DVT), pulmonary embolism (PE), portal vein thrombosis (PVT),   
venous thromboembolism, anti-Xa, activated partial thromboplastin time,   
anticoagulation therapeutic monitoring, coagulopathy, coagulation cascade,   
chronic liver disease, cirrhosis, and decompensated liver disease. Study   
Selection: Studies written in the English language from January 2000 to   
December 2015 were considered for this review article. All search results   
were reviewed, and the relevance of each article was determined by authors   
independently. Conclusions: Patients with cirrhosis are at higher risk for   
both bleeding and thrombosis-related complications. cirrhosis affects   
production of both procoagulant and anticoagulant factors, thus resulting in   
increased INR and aPTT levels and decreased anti-Xa levels. LMWH is the   
treatment of choice for the prevention and treatment of DVT/PE/PVT in   
patients with cirrhosis, and monitoring with anti-Xa levels for dose   
adjustment is not recommended. UFH is an alternative in cirrhotic patients   
for shorter-term use and in cases of severe renal dysfunction and/or   
hemodynamic instability. Cirrhotic patients on anticoagulation therapy   
should be monitored closely for signs and symptoms of bleeding and   
thrombosis.  
  
RECORD 117  
TIPS in portal and hepatic vein thrombosis  
Punamiya S.  
CardioVascular and Interventional Radiology (2016) 39:3 Supplement 1   
(S128-S130). Date of Publication: 2016  
  
Learning Objectives 1. To review current indications for TIPS in hepatic   
and/or portal vein thrombosis 2. To learn about additional techniques in   
these settings 3. To review results of TIPS in patients with the hepatic   
vein thrombosis, and acute or chronic thrombosis of the portal vein Portal   
vein thrombosis (PVT) and Budd-Chiari syndrome (BCS) are caused by   
thrombotic obstruction of the extrahepatic portal veins and the hepatic   
venous outflow, respectively, usually producing significant symptoms of   
portal hypertension. Several heterogenous prothrombotic disorders in   
combination with local triggering factors have been implicated in causing   
this thrombosis. Medical management, including anticoagulation, forms the   
backbone in treating both disorders; radiological and surgical intervention   
being reserved for refractory and severely symptomatic cases. Amongst these,   
TIPS has traditionally been considered a relative contraindication, as   
technical challenges produced by the occluded veins often resulted in   
procedural failure. However, the past decade has witnessed better procedural   
and clinical success rates, and consequently, TIPS is being increasingly   
offered to treat complications of portal hypertension in this group of   
patients. A. portal vein thrombosis The aim of treatment in PVT is to   
reverse or prevent progression of PVT and to treat complications of portal   
hypertension. anticoagulation results in recanalisation of acute PVT in   
majority of patients and minimises serious complications like bowel ischemia   
and development of varices, provided it is initiated early. Most often,   
however, patients with PVT manifest at a chronic stage where anticoagulation   
cannot reverse complications like variceal bleeding, symptomatic portal   
biliopathy and hypersplenism. Variceal bleeding in such cases is managed in   
standard fashion, using vasoconstrictors, antibiotics and endoscopic   
treatment. TIPS can be offered in these patients if the bleeding is not   
controlled or if it recurs despite conventional therapy. PVT occurs in up to   
26% of patients with liver cirrhosis, and in this setting it has been   
proposed that an occlusive PVT potentially changes the natural history of   
liver cirrhosis as it increases the incidence of variceal bleeding and   
decreases the patients' survival. Conceptually, TIPS would benefit these   
patients by not only resolving the portal hypertension, but also improving   
transplant outcomes as it allows for a more physiological and durable   
end-to-end anastomosis. Technique of TIPS in PVT TIPS is challenging in the   
presence of PVT due to difficulty encountered during portal vein access. The   
procedure is essentially done in 2 steps. In the first step, the portal vein   
is recanalised using a transjugular, transhepatic, transplenic or   
transmesenteric approach. Once the portal vein is recanalised, the TIPS is   
completed in routine fashion from jugular venous access For initial portal   
vein recanalisation, the portal vein can be approached from various routes:   
Transjugular access: The technique is similar to TIPS, wherein a liver   
access needle is advanced across the liver parenchyma into a patent   
peripheral portal venous branch from the jugular puncture. Once in the   
peripheral branch, a curved angiographic catheter and hydrophilic wire are   
then advanced and manipulated across the portal vein occlusion. Transhepatic   
access: Here, a peripheral portal venous radicle is accessed percutaneously   
using US or fluoroscopy, following which an angiographic catheter and   
hydrophilic wire is manipulated across the occluded portal vein.   
Transsplenic access: In this method, a splenic hilar vein is accessed   
percutaneously and catheter advanced to reach the portal vein occlusion and   
cross it retrogradely. Transmesenteric access: A mini-laparotomy is   
performed in the angiography suite to expose an ileal loop. A sheath is then   
placed within the ileal vein, through which the angiographic catheter and   
wire are advanced through the occluded portal vein. Once access into the   
portal vein is gained, the occluded segment can be recanalised using a   
variety of techniques, depending on the age of the thrombus. An acute portal   
vein thrombus can be effectively removed by thrombolysis, thromboaspiration,   
and/or mechanical thrombectomy. Alternatively, the thrombus can be trawled   
into the intrahepatic portal venous radicles using a Fogarty thrombectomy   
catheter. Any residual flow limiting thrombus that is refractory to these   
therapies is generally dilated or stented. A chronic portal vein occlusion   
is treated with angioplasty and/or stenting with either bare or covered   
stents. TIPS is generally inserted after the portal vein is recanalised.   
This is fairly straightforward if the initial access to the portal vein is   
transjugular, as the recanalisation and TIPS creation would be over the same   
wire access. However, if the initial access is from any approach other than   
jugular, the conversion to TIPS requires a portal vein target for the TIPS   
needle. This can be achieved by positioning a snare or an inflated balloon   
in the recanalised portal vein or by guiding the needle toward the top of   
end the portal vein stent. Once the portal vein entry is successful, the   
TIPS is placed in standard fashion. Results of TIPS in PVT TIPS can be   
successfully inserted in portal vein thrombosis in almost 99.5% of patients   
when thrombosis is partial. The success rates drop to 79% when the portal   
vein is completely occluded, and dip further to 63% when the occlusion is   
chronic, suggested by presence of a portal cavernoma. A successful TIPS   
reduces the incidence of variceal rebleeding significantly. A 1- and 5-year   
cumulative variceal rebleeding rate of 10% and 28% is noted in patients of   
PVT that had a TIPS inserted, versus 43% and 100% for patients that did not   
succeed in getting a TIPS. Also, the short-term survival with TIPS is   
excellent (the 1- and 2-year cumulative survival rates are 80-89% and   
72-81%), and the longterm prognosis in these patients appears to be higher   
than general patients with decompensated cirrhosis. B. Budd-Chiari syndrome   
Hepatic venous outflow obstruction causes an increase in hepatic sinusoidal   
pressure that leads to a cascade of events, beginning with hepatocellular   
congestion, necrosis and finally cirrhosis. Depending on extent of venous   
involvement, speed of occlusion, and degree of venous collateralisation,   
manifestation can vary markedly, ranging from asymptomatic disease to   
fulminant liver failure. Majority of patients present with abdominal pain,   
ascites, hepatosplenomegaly, dilated abdominal wall veins, leg oedema and   
near normal liver function despite overt portal hypertension.   
anticoagulation and, if possible, treatment of underlying disorders (e.g.   
myeloproliferative disease, paroxysmal nocturnal hemoglobinuria) form the   
cornerstone of therapy in BCS, and should be initiated as early as possible   
in the disease. anticoagulation alone will succeed in controlling liver   
disease in 10% of patients. Next, whenever possible, recanalisation of the   
hepatic venous outflow by angioplasty and stenting should be attempted, as   
it is a low risk procedure that decongests the liver while maintaining   
physiological blood flow. TIPS is recommended in symptomatic patients with   
BCS when (a) the hepatic vein occlusive segment is long, (b) there is   
failure to recanalise the hepatic veins, or (c) there is no clinical benefit   
from hepatic vein recanalisation. Technique of TIPS in BCS The procedure of   
TIPS requires few technical modifications. Since the hepatic veins are   
occluded, parenchymal puncture is initiated either from a stump of the   
hepatic vein or directly from the retrohepatic IVC, usually about 2-6 cm   
distance from the right atrium. To aid penetration through the IVC wall, a   
left sided jugular approach is preferred by some, as is the use of a coaxial   
21G fine needle. Either maneuver embeds the needle in the caval wall and   
prevents it from sliding down the IVC. Once the caval wall is penetrated,   
the needle is advanced through the liver parenchyma toward the hepatic   
hilum. With each throw of the needle into the liver parenchyma, entry into   
the portal vein is best confirmed by injection of contrast (PTC-style)   
rather than aspiration of blood, as blood is invariably aspirated from the   
congested liver or from small intra-hepatic venous collaterals. Longer and   
more frequent throws of the needle should be anticipated, as the liver is   
enlarged; most parenchymal tracts from the IVC to the portal vein extend   
over 7-10 cm in length. The liver is also much softer and congested. This   
feature, along with the longer tracts and frequent needle passes,   
potentially increases the risk of intraperitoneal hemorrhage, intrahepatic   
hematomas or pseudoaneurysms. Utilisation of a fine needle and aids to   
target the portal vein can reduce this risk. Results of TIPS in BCS TIPS has   
become the preferred form of treatment when medical therapy has failed, as   
it provides improvement in clinical symptoms and liver function and arrests   
progression of liver fibrosis. One of the largest multi-centre study on TIPS   
in BCS revealed technical success in over 90%, and a 1- and 10-year   
transplant-free survival of 88% and 69%, respectively. Although TIPS-related   
complications are not infrequent, procedural mortality is rare. Patients   
with BCS are known to have a high incidence of TIPS dysfunction from intimal   
hyperplasia and thrombotic occlusion, requiring frequent re-interventions to   
maintain its patency. Covered stents have improved the patency rates   
significantly, with 6- and 12-month patency rates of 100% and 85.7%,   
respectively, compared to 16.7% and 0% for bare stents; hence, its use is   
strongly recommended in BCS.  
  
RECORD 118  
EASL Clinical Practice Guidelines: Vascular diseases of the liver  
Garcia-Pagán J.C.  
Journal of Hepatology (2016) 64:1 (179-202). Date of Publication: 2016  
  
RECORD 119  
anticoagulation for portal vein thrombosis in cirrhosis: Response to   
Naeshiro and collaborators  
Rodriguez-Castro K.I.  
Hepatology Research (2015) 45:12 (1256-1257). Date of Publication: 1 Dec   
2015  
  
RECORD 120  
Antithrombotic treatment with direct-acting oral anticoagulants in patients   
with splanchnic vein thrombosis and cirrhosis  
De Gottardi A. Trebicka J. Klinger C. Plessier A. Seijo S. Terziroli B.   
Magenta L. Semela D. Buscarini E. Langlet P. Görtzen J. Puente A. Müllhaupt   
B. Navascuès C. Nery F. Deltenre P. Turon F. Engelmann C. Arya R. Caca K.   
Peck-Radosavljevic M. Leebeek F.W.G. Valla D. Garcia-Pagan J.C.  
Liver International (2016). Date of Publication: 2016  
  
Background: Direct-acting oral anticoagulants (DOACs) are used in patients   
with splanchnic vein thrombosis (SVT) and cirrhosis, but evidence for safety   
and efficacy in this setting is limited. Our aim was to identify indications   
and reasons for starting or switching to DOACs and to report adverse   
effects, complications and short-term outcome. Methods: Data collection   
including demographic information, laboratory values, treatment and   
complications through the Vascular Liver Disease Interest Group Consortium.   
Results: Forty-five centres (90%) of the consortium completed the initial   
eCRF. We report here a series of 94 patients from 17 centres. Thirty-six   
patients (38%) had cirrhosis. Child-Pugh score was 6 (range 5-8), and MELD   
score 10.2 (range 6-19). Indications for anticoagulation were splanchnic   
vein thrombosis (75%), deep vein thrombosis (5%), atrial fibrillation (14%)   
and others (6%). DOACs used were rivaroxaban (83%), dabigatran (11%) and   
apixaban (6%). Patients were followed up for a median duration of 15 months   
(cirrhotic) and 26.5 months (non-cirrhotic). Adverse events occurred in 17%   
of patients and included one case of recurrent portal vein thrombosis and   
five cases of bleeding. Treatment with DOACs was stopped in three cases. The   
major reasons for choosing DOACs were no need for monitoring or inadequacy   
of INR to guide anticoagulation in cirrhotic patients. Renal and liver   
function did not change during treatment. Conclusions: A consistent number   
of patients with SVT and/or cirrhosis are currently treated with DOACs,   
which seem to be effective and safe. These data provide a basis for   
performing randomized clinical trials of DOACs vs. low molecular weight   
heparin or vitamin K antagonists.  
  
RECORD 121  
Too much cortisol may make you clot: portal vein thrombosis as an unusual   
complication of cushing's syndrome  
Gurung A. McDow A. Poola R. Fratianni C.M. Garfinkel M. Jakoby M.G.  
Endocrine Reviews (2016) 37:2 Supplement 1. Date of Publication: 2016  
  
Background: The hallmark manifestations of Cushing's syndrome (CS) are well   
known, but hypercoagulability is perhaps least recognized. Patients with   
Cushing's syndrome are at increased risk of both postoperative and   
spontaneous thromboembolic events. Expression of factors that favor   
clotting, chiefly factors VIII, IX, and von Willebrand factor, are   
increased, and fibrinolytic capacity is reduced by increased expression of   
plasminogen activator inhibitor-1. We report an unusual patient whose CS was   
diagnosed during evaluation of unprovoked portal vein thrombosis. Case: A 61   
year-old female with history of hypertension and rheumatoid arthritis   
presented to her physician for evaluation of abrupt onset upper abdominal   
pain, nausea, fevers, and chills. Computed tomography of the abdomen   
revealed complete thrombosis of the left intrahepatic portal vein and a 3.5   
cm, well circumscribed, and low attenuation left adrenal nodule. Examination   
was notable for mild hypertension, moon facies, plethora, hirsutism,   
dorsocervical fat pad hypertrophy, and scattered bruises. Midnight plasma   
cortisol (10.5 mg/dL, expected < 7.5), midnight salivary cortisol (550   
ng/dL, expected < 100), and 24 hr urine free cortisol (146 mg, ref 3.5-45)   
were all unequivocally elevated, and 8 AM ACTH level was suppressed (7.2   
pg/mL, ref 10-60) consistent with adrenal hypercortisolemia. Evaluation for   
other potential etiologies of thrombosis, including procoagulant antibodies   
(e.g. antiphospholipid antibodies), deficiencies of protein C, protein S,   
and antithrombin III, prothrombin G2021A mutation, and activated protein C   
resistance was unremarkable. Acute symptoms resolved on treatment with   
heparin and parenteral antibiotics. After three months of anticoagulation   
with warfarin, the patient underwent an uneventful laparoscopic left   
adrenalectomy. Postsurgical pathology confirmed an adrenal adenoma.   
Conclusions: Approximately 8-10% of patients with CS experience deep vein   
thrombosis or pulmonary embolism, with slightly more than half of events   
related to surgery and the rest occurring spontaneously. Most cases of   
venous thrombosis occur in the lower extremities. A PubMed search with the   
terms “portal vein thrombosis” and “Cushing's syndrome” yields only one   
brief French language case report; to the best of our knowledge, this is   
only the second reported case of CS complicated by spontaneous portal vein   
thrombosis. Hypercoagulability is an important complication of   
hypercortisolemia, and CS should be considered in the differential diagnosis   
of spontaneous deep vein thrombosis. CS patients undergoing surgery require   
close observation and perioperative thromboprophylaxis.  
  
RECORD 122  
Guidance for the management of venous thrombosis in unusual sites  
Ageno W. Beyer-Westendorf J. Garcia D.A. Lazo-Langner A. McBane R.D.   
Paciaroni M.  
Journal of Thrombosis and Thrombolysis (2016) 41:1 (129-143). Date of   
Publication: 1 Jan 2016  
  
Venous thromboembolism (VTE) is a serious and often fatal medical condition   
with an increasing incidence. The treatment of VTE is undergoing tremendous   
changes with the introduction of the new direct oral anticoagulants and   
clinicians need to understand new treatment paradigms. This manuscript,   
initiated by the anticoagulation Forum, provides clinical guidance based on   
existing guidelines and consensus expert opinion where guidelines are   
lacking. In this chapter, we address the management of patients presenting   
with venous thrombosis in unusual sites, such as cerebral vein thrombosis,   
splanchnic vein thrombosis, and retinal vein occlusion. These events are   
less common than venous thrombosis of the lower limbs or pulmonary embolism,   
but are often more challenging, both for the severity of clinical   
presentations and outcomes and for the substantial lack of adequate evidence   
from clinical trials. Based on the available data, we suggest anticoagulant   
treatment for all patients with cerebral vein thrombosis and splanchnic vein   
thrombosis. However, in both groups a non-negligible proportion of patients   
may present with concomitant bleeding at the time of diagnosis. This should   
not contraindicate immediate anticoagulation in patients with cerebral vein   
thrombosis, whereas for patients with splanchnic vein thrombosis   
anticoagulant treatment should be considered only after the bleeding source   
has been successfully treated and after a careful assessment of the risk of   
recurrence. Finally, there is no sufficient evidence to support the routine   
use of antithrombotic drugs in patients with retinal vein occlusion. Future   
studies need to assess the safety and efficacy of the direct oral   
anticoagulants in these settings.  
  
RECORD 123  
portal vein thrombosis  
Malik A. Yeoman A. Allison M. Czajkowski M.  
Gut (2016) 65 Supplement 1 (A264). Date of Publication: 2016  
  
Introduction portal vein thrombosis (PVT) is defined as the presence of   
thrombus in the trunk of PV and/or its right and left intra-hepatic   
branches. PVT can be classified as acute or chronic, intra or extra-hepatic   
and occlusive or non-occlusive. Patients may be asymptomatic or present with   
upper GI bleeding or abdominal pain. PVT in cirrhotic patients can present   
with acute decompensation such as ascites or variceal bleeding. Methods A   
retrospective review of all radiological diagnoses of PVT was done with a   
view to understanding the aetiology, clinical spectrum, treatment and   
prognosis of patients managed under a large district hospital. Results A   
total of 115 patients, median age 62 years (range 25 to 90) were diagnosed   
with PVT between 2010 and 2015, of whom 71 (62%) were male. Usual   
indications for radiological investigations were abdominal pain, weight loss   
and decompensation or routine surveillance in cirrhotic patients. PVT was   
intra-hepatic alone in 29 patients and extra-hepatic with or without   
intra-hepatic extension in the rest. Cavernous transformation was reported   
in 11 patients. PVT was most commonly seen in association with abdominal   
malignancy (55 cases - 48%) being due to HCC in 21 cases and other local or   
metastatic abdominal malignancy in 34. PVT was observed to be due to   
pancreatitis in 21 cases, liver cirrhosis without HCC in 15, acute   
diverticulitis/cholecystitis in 6, post surgical in 4 with no clear cause   
identified in just 14 cases (12%). Thrombophilia screening was performed in   
11/14 patients with unclear aetiology and was positive in 3 (1 JAK-2   
positive, 1 elevated anti-b2GP1 antibodies, 1 low in both protein C and S,   
rest negative), 2/15 patients with liver cirrhosis (both negative) and 4/86   
(1 positive for lupus anti-coagulant) of remaining patients. In total 24   
patients were anticoagulated whilst 3 patients were already on warfarin for   
atrial fibrillation. Of these, 10 were patients of unclear aetiology, 4 with   
cirrhosis without HCC, 3 had diverticulitis, 3 local or metastatic   
malignancy, 2 pancreatitis, 1 cholecystitis and 1 post surgical. Eleven of   
the 15 patients with cirrhosis and PVT died, typically from hepatic   
decompensation with a median life expectancy of 8 months (range 1-48   
months). Patients who were anticoagulated survived for 12 months as opposed   
to 4 months for those not anticoagulated. Conclusion PVT has a wide   
aetiological spectrum and management strategies are highly variable   
reflecting the diversity of causes. anticoagulation was most likely to be   
commenced in those with no clear cause even in the absence of thrombophilia.   
This study confirms that PVT in the context of cirrhosis is an adverse   
prognostic indicator even in the absence of HCC.  
  
RECORD 124  
Thrombotic risk factors in nonmalignant and noncirrhotic patients with   
portal vein thrombosis: Need for extensive investigation  
Kurtcehajic A. Zerem E. Hujdurovic A. Fejzic J.A.  
European Journal of Gastroenterology and Hepatology (2016) 28:1 (116-118).   
Date of Publication: 2016  
  
RECORD 125  
Feasibility and outcomes of laparoscopic sleeve gastrectomy after solid   
organ transplantation  
Khoraki J. Katz M.G. Funk L.M. Greenberg J.A. Fernandez L.A. Campos G.M.  
Surgery for Obesity and Related Diseases (2016) 12:1 (75-83). Date of   
Publication: 1 Jan 2016  
  
Background: Obesity is common after solid organ transplantation and is   
associated with worse transplantation-related outcomes. Laparoscopic sleeve   
gastrectomy (LSG) may be the preferred bariatric operation in   
transplantation patients over other techniques, such as gastric bypass,   
given the concerns about medication absorption. However, little is known   
about LSG outcomes in posttransplantation patients. Objectives: We report   
the outcomes in 10 consecutive patients who underwent solid organ   
transplantation followed by LSG. Setting: An academic medical center.   
Methods: Primary outcomes studied were weight loss, perioperative   
complications, resolution or improvement of obesity-related co-morbidities,   
and markers of graft function following LSG. Results: The types of   
transplantation before LSG were as follows: liver = 5, kidney = 4, and heart   
= 1. Mean body mass index (BMI) at LSG was 44.7 ±1.7 kg/m . All patients had   
hypertension, and 6 had type 2 diabetes. Perioperative complications   
occurred in 2 patients, and there were no deaths. Excess weight loss at 12   
and 24 months after LSG was 45.7% and 42.5%, respectively. At 1 year after   
LSG, there was a significant reduction in the number of antihypertensive   
medications (2.4 to 1.5; P = .02). Three patients achieved complete   
remission of type 2 diabetes, and the other 3 significantly reduced their   
dosages of insulin. Graft function remained preserved in liver   
transplantation patients; left ventricular ejection fraction (LVEF)   
increased by 10% in the heart transplantation subject, and the estimated   
glomerular filtration rate (eGFR) increased significantly in kidney   
transplantation patients (53 ± 3 to 82 ± 3 mL/min; P = .03). Conclusions: We   
concluded that LSG, in selected patients with severe obesity after solid   
organ transplantation, results in significant weight loss, improvement or   
resolution of obesity-related conditions, and preservation or improvement of   
graft function. Larger studies are needed to determine tolerability   
standards.  
  
RECORD 126  
Efficacy and safety of anticoagulation in more advanced portal vein   
thrombosis in patients with liver cirrhosis  
Chen H. Liu L. Qi X. He C. Wu F. Fan D. Han G.  
European Journal of Gastroenterology and Hepatology (2016) 28:1 (82-89).   
Date of Publication: 2016  
  
Background and aim portal vein thrombosis (PVT) is a frequent event in   
patients with cirrhosis. The effects of anticoagulation on these patients   
were still unclear, especially for more advanced PVT. The aim of this study   
was to retrospectively assess the resolution of PVT and liver disease   
progression in a large cohort of cirrhotic patients with PVT with or without   
anticoagulation therapy. Methods We analyzed data from 66 cirrhotic patients   
with PVT from January 2002 to June 2014. Thirty patients were anticoagulated   
with warfarin and 36 patients were untreated. PVT and hepatic decompensation   
were evaluated. Results For anticoagulated patients, the thrombosis had   
improved in 15 (68.2%) patients, was stable in four patients (18.2%), and   
progressed in three patients (13.6%). For untreated patients, the thrombosis   
had improved in four patients (25%), was stable in six patients (37.5%), and   
progressed in six patients (37.5%). The anticoagulation group had   
significantly better recanalization rates than the untreated group   
(P=0.011). Degree of superior mesenteric vein (P=0.032, hazard ratio: 15.4;   
95% confidence interval: 1.3-200) was a significant predictor. In addition,   
anticoagulation can effectively improve PVT with a degree less than 75% in   
the main portal vein compared with untreated patients (6/6 vs. 2/6, P=   
0.030). The probability of hepatic decompensation at 1 year was 15.6 and   
17.9% between the anticoagulation and the untreated groups (P= 0.847).   
Albumin (P= 0.06, hazard ratio: 0.860; 95% confidence interval: 0.772-0.959)   
was a significant predictor. Conclusion anticoagulation with warfarin might   
result in the resolution of more advanced PVT effectively and safely in   
patients with liver cirrhosis. In addition, we did not demonstrate the   
benefit of anticoagulation for the decompensation or death. Eur J   
Gastroenterol Hepatol 28:82-89.  
  
RECORD 127  
Recurrent acute portal vein thrombosis in liver cirrhosis treated by   
rivaroxaban  
Yang H. Kim S.R. Song M.J.  
Clinical and Molecular Hepatology (2016) 22:4 (499-502). Date of   
Publication: 2016  
  
cirrhosis can occur with the development of portal vein thrombosis (PVT).   
PVT may aggravate portal hypertension, and it can lead to hepatic   
decompensation. The international guideline recommends for anticoagulation   
treatment to be maintained for at least 3 months in all patients with acute   
PVT. Low-molecular-weight-heparin and changing to warfarin is the usual   
anticoagulation treatment. However, warfarin therapy is problematic due to a   
narrow therapeutic window and the requirement for frequent dose adjustment,   
which has prompted the development of novel oral anticoagulants for   
overcoming these problems. We report a 63-year-old female who experienced   
complete resolution of recurrent acute PVT in liver cirrhosis after   
treatment with rivaroxaban.  
  
RECORD 128  
Neonatal portal vein thrombosis: A single institutional experience of short   
and longterm outcomes  
Patel V. Bhatt M. Paes B. Chan A.  
European Journal of Pediatrics (2016) 175:11 (1765-1766). Date of   
Publication: 2016  
  
Background and aims The reported rate of incidence of neonatal portal vein   
thrombosis (PVT) is 36 per 1000 neonatal intensive care admissions, may in   
fact be higher. There is paucity of literature describing outcomes of   
neonatal PVT. The aim of our study was to describe the outcomes of neonatal   
PVT in order to facilitate clinical decisions regarding the need for   
aggressive potential treatment strategies. Methods Retrospective chart   
review of neonates diagnosed with PVT between January, 2008 and December,   
2015 in a tertiary care neonatal unit. Results Eighteen premature (mean   
gestational age (GA): 31.6 weeks) and 19 term (mean GA: 39.1 weeks) neonates   
were diagnosed with PVT. 34 involved the left portal vein and 3 involved   
more than one vein. PVT was catheter-associated in 27 (73%) neonates; none   
of the 5 neonates tested had a pro-thrombotic condition. Of the 37 neonates,   
12 received anticoagulation therapy and 25 were untreated. The mean   
follow-up duration was 15.5 months; 19% were followed for >2 years. On last   
diagnostic imaging, thrombus resolution was documented in treated (n=12) and   
non-treated (n=25) neonates: 5 (42%) and 14 (56%) complete, 0 and 2 (8%)   
partial, and 7 (58%) and 9 (36%) stable, respectively. No complications were   
detected in 32 (86%) neonates, while 2 had hepatomegaly, 2 had abnormal   
liver enzymes and 1 had splenomegaly and abnormal liver enzymes. (Table   
presented) Conclusions In our cohort, the PVT resolution rate was similar to   
previously reported studies. Although a low complication rate was detected,   
longer follow-up is necessary to determine the exact incidence of outcomes   
such as portal hypertension.  
  
RECORD 129  
Direct intrahepatic portocaval shunt for treatment of portal thrombosis and   
Budd-Chiari syndrome  
Pedersen M.R. Molloy P. Wood D. Seetharam A.  
Annals of Hepatology (2016) 15:1 (127-130). Date of Publication: 1 Jan 2016  
  
Budd-Chiari syndrome (BCS) refers to hepatic venous outflow obstruction that   
in severe cases can lead to acute liver failure prompting consideration of   
revascularization or transplantation. Here, a 22 year old female with   
angiographically proven BCS secondary to JAK2/V617F positive Polycythemia   
vera on therapeutic warfarin presented with acute liver failure (ALF).   
Imaging revealed a new, near complete thrombotic occlusion of the main   
portal vein with extension into the superior mesenteric vein. An emergent   
direct intrahepatic portocaval shunt (DIPS) was created and liver function   
promptly normalized. She has been maintained on rivaroxaban since that time.   
Serial assessment over 1 year demonstrated continued shunt patency and   
improved flow in the mesenteric vasculature on ultrasound as well as normal   
liver function. DIPS is a viable alternative in the treatment of ALF from   
BCS when standard recanalization is not feasible. Improved blood flow may   
also improve portal/mesenteric clot burden. While further investigation is   
needed, new targeted anticoagulants may be viable as a long term   
anticoagulation strategy.  
  
RECORD 130  
Portal hypertensive complications and clinical outcomes in paediatric and   
adolescent patients presenting with portal vein thrombosis  
Direkze S. Bancil A. Dawan A. Samyn M. Heaton N. Velez-Mendes H. Davenport   
M. Kane P. Karani J. Joshi D.  
Gut (2016) 65 Supplement 1 (A98). Date of Publication: 2016  
  
Introduction portal vein thrombosis (PVT) has multiple aetiologies which can   
lead to the development of portal hypertension and variceal bleeding. Data   
on the long term sequelae of PVT in paediatric and adolescent patients is   
limited. Methods Patients included had a diagnosis of PVT from Jan 2000- Dec   
2014. Data collection included patient demographics, aetiologies,   
presentation and initial treatment of PVT. Data was also collected on   
further variceal bleeds, shunt surgery, liver transplantation and long term   
mortality. Results 123 patients (63 male) were identified. Median age at   
first presentation was 5years 9 months (range 2 days to 25 years). Overall   
survival was 95.9, 94.3 and 93.5% at 1, 10 and >20 years from PVT diagnosis,   
respectively. Median age at follow up was 13years 3 months (range 6 months   
to 33 years 2 months). In the majority of cases (52%) no cause for PVT was   
identified. 78% of patients had extrahepatic PVT (EHPVT), with no extension   
in to the superior mesenteric vein. 19 patients (15.4%) were anticoagulated   
or had received a course of anticoagulation therapy. Initial presentation   
was usually due to oesophageal variceal bleeding (52.8%) of which 24   
patients had further variceal bleeds (18.7%, oesophageal/ duodenal/rectal).   
At follow up, 26% of patients (N = 32) were on a beta blocker. Of these, 10   
patients (31.3%) had further variceal bleeds versus 14/77 patients, not on a   
beta blocker (18.2%, p = 0.07). Portal biliopathy was also present in 17.9%   
(N = 22) as was splenomegaly (82.9%). Ascites and hepatic encephalopathy   
were uncommon (<7%). 18 patients had shunt surgery, majority of which were   
meso-caval Rex, of which 7/18 (38.8%) had further variceal bleeds. Two   
patients underwent transplantation, one of which was for variceal bleeding.   
Overall mortality in this group of patient with PVT was low (6.5% N = 8)   
though only 2 of these patients had a previous history of recurrent variceal   
bleeds. Conclusion Oesophageal variceal bleeding is a common index   
presentation of PVT in paediatric and adolescent patients. Approximately 20%   
of patients will go on to have further variceal bleeds despite medical   
intervention. Beta blocker use is associated with recurrent variceal   
bleeding which may suggest that it is ineffective in preventing further   
variceal bleeding in patients with established portal cavernomas. Overall,   
long term survival is good.  
  
RECORD 131  
Portal or splenic vein thrombosis after splenectomy for immune cytopenia: A   
retrospective cohort study  
Morbieu C. Brunetti F. Baranès L. Languille L. Limal N. Loustau V. Bierling   
P. Michel M. Godeau B. Mahévas M.  
Blood (2015) 126:23 (3483). Date of Publication: 3 Dec 2015  
  
Introduction Portal and/or splenic vein thrombosis (PVST), stemming from   
immune thrombocytopenia (ITP), warm autoimmune hemolytic anemia (wAIHA) or   
other splenic diseases, is highly associated with splenectomy complications.   
Although symptomatic PVST is a rare event (incidence rate 1-3%) reportedly   
associated to mortality, it has been shown through systematic computed   
tomography (CT) scans that PSVT events exist in up to 50% of splenectomized   
patients. The clinical significance of this finding remains unclear.   
Indications of anticoagulation depend on the site and extent of PSVT. The   
relevance of postoperative CT scanning is discussed, specifically in the   
context of immune cytopenia, where PSVT seems to be less frequent and less   
extended. We conducted a retrospective study to assess the incidence, the   
sites and the outcome of PSVT in ITP and wAIHA patients. Patients and   
methods The study was conducted between 2009 and April 2015 in a   
tertiary-care center for immune cytopenias (Henri Mondor Hospital, France)   
where postoperative CT scans are systematically performed. We analyzed   
splenectomized ITP or wAIHA (without underlying malignancy) adult patients   
undergoing a postoperative enhanced abdominal CT scan. Clinical and   
biological data were collected from medical records. All CT scans were   
reviewed by a senior radiologist using a standardized form. Results Thirty   
nine patients (19 men, 20 women), with a mean age of 45 years (range,   
19-83), were splenectomized mostly by laparoscopic surgery (n=38), for ITP   
(n=30) or wAIHA (n=9). Thirty six patients (92%) had received a preventive   
anticoagulation treatment for a median duration of 24 post-operative days   
(range, 7-36). Among ITP patients, the mean preoperative platelet count was   
95.6 G/L (SD: 97). ITP patients had received corticosteroids (75%),   
intravenous immunoglobins (43%), or thrombopoietin receptor agonists (41%)   
in preparation for splenectomy). The overall initial response rate at 2   
months from splenectomy was 69% (ITP: 63%, wAIHA: 89 %). The CT scan was   
performed within 5 days on average (range: 3-12) of splenectomy. The   
incidence rate of PSVT was 74% (29/39), without discrimination between ITP   
(22/30, 73%) and wAIHA (7/9, 78%, p = 0.79) (Table 1). There were mostly   
distal splenic vein (dSVT) (n=14), or dSVT associated with intrahepatic   
portal vein thrombosis (iPVT) (n=12). There were no mesenteric vein   
thromboses, 1 extrahepatic portal vein (ePVT) and 1 proximal splenic vein   
thrombosis (pSVT). Most of the patients were asymptomatic (n=18). No   
significant association between PSVT and any clinical or biological   
characteristics such as platelet count, preoperative treatments,   
thrombocytosis, or efficacy of splenectomy was observed. However, PSVT   
patients tended to have higher spleen weights (206 g vs 102 g, p = 0.14).   
Thirteen of the 29 patients with a PVST were treated with a curative   
anticoagulation treatment for a mean duration of 11 weeks (range, 4-37).   
Among the 26 patients evaluable in follow-up, the rate of complete   
thrombosis resolution was 84% (21/26); specifically, 62% (8/13) in   
anticoagulated patients and 100% in the 13 non anticoagulated patients (9   
dSVT, 4 iPVT+dSVT). A portal cavernoma occurred after iPVT+dSVT despite   
anticoagulation in the context of wAIHA. anticoagulation complications   
included abdominal wall hematoma requiring transfusions (n=1) and iron   
deficiency anemia (n=1). Conclusion In our study, post-splenectomy PSVT was   
frequent and often asymptomatic, involving mostly intrahepatic portal or   
distal splenic veins and was resolved in half of the cases without any   
curative anticoagulative therapy. Our results suggest that a systematic   
screening of PSVT by CT scan should not be performed in absence of risk   
factors for thrombosis. (Table Presented).  
  
RECORD 132  
Real-world use of therapeutic anticoagulation in patients with paroxysmal   
nocturnal hemoglobinuria. Results of a survey of physicians in Australia  
Szer J. Forsyth C.J. Giese A.  
Blood (2015) 126:23 (4537). Date of Publication: 3 Dec 2015  
  
Paroxysmal nocturnal hemoglobinuria (PNH) is a rare and life-threatening   
hematopoietic stem cell disorder characterized by uncontrolled   
complement-mediated hemolysis. Patients with PNH are at increased risk of   
thromboembolism and premature death. This risk is predominantly due to the   
effects of chronic hemolysis and platelet activation. Eculizumab, a   
monoclonal antibody that inhibits terminal complement activation, has been   
shown to reduce hemolysis and dramatically reduce the rate of   
thromboembolism. A previous publication (Kelly et al, 2011) suggested that   
cessation of therapeutic anticoagulation (TAC) in PNH patients on eculizumab   
with no prior history of thrombosis is safe. There are very few reports on   
the outcomes of cessation of TAC in PNH patients on eculizumab who have a   
prior history of thrombosis or on the use of non-vitamin K antagonist oral   
anticoagulant (NOAC) agents in PNH patients with a history of thrombosis. In   
Australia, patients with PNH are predominantly managed by individual   
hematologists rather than at a single centre and hence anticoagulation   
practices following the introduction of eculizumab therapy are variable. We   
surveyed Australian hematologists managing eculizumab-treated patients with   
PNH to obtain the details of anticoagulation management and incidence of   
thrombotic events in their patients. We received responses from 30   
hematologists caring for a total of 58 patients with PNH on eculizumab (1-17   
patients per hematologist) and the table summarises the results. TAC as   
primary prophylaxis had been ceased in 10 patients with no recurrent   
thrombotic events. One (1) patient remains on primary prophylaxis due to   
persistently high D-dimer and factor VIII levels. TAC for secondary   
prophylaxis had been ceased in 2 patients due to bleeding (1 patient with   
subdural hematoma, 1 patient with gastrointestinal bleeding) and neither of   
these patients had a further thrombotic event. One patient, with a prior   
history of thrombosis, requested cessation of TAC and subsequently developed   
a provoked thrombosis. Three patients not receiving TAC when eculizumab was   
commenced developed thrombosis; two (2) patients had provoked deep venous   
thromboses and one patient developed a splanchnic vein thrombosis following   
a cholecystectomy in association with severe sepsis. One patient had a   
portal vein thrombosis immediately prior to commencing eculizumab therapy   
but has never received TAC due to severe coexistent thrombocytopenia from   
myelodysplasia. This patient has not had a recurrent thrombosis. Three (3)   
patients with thrombotic events prior to eculizumab therapy (1 patient with   
pulmonary emboli, 1 patient with cerebral venous sinus thrombosis and 1   
patient with inferior vena cava thrombosis) had anticoagulant therapy   
changed from warfarin to rivaroxaban. At a follow-up of at least twelve   
months for all 3 patients there have been no recurrent thrombotic events and   
no bleeding complications. In conclusion, these Australian data are   
consistent with those reported by Kelly suggesting that cessation of primary   
prophylaxis in PNH patients on eculizumab is safe. Cessation of TAC in PNH   
patients on eculizumab with a prior thrombosis can be considered if there   
are clear contraindications to anticoagulation. Thromboprophylaxis in   
situations of increased risk of venous thromboembolism remains essential for   
all PNH patients not on TAC, even when they are on eculizumab therapy. The   
three patients on rivaroxaban as secondary prophylaxis are, to our   
knowledge, the first reported patients with PNH treated on a NOAC. (Table   
Presented).  
  
RECORD 133  
Continued use of tinzaparin at therapeutic doses for prophylaxis of venous   
thromboembolism in patients with intolerance to antivitamins K  
Ruiz M.A.G. Martinez F.J.R. Constantin E.M. Morales M.G. Jurado M.  
Blood (2015) 126:23 (4735). Date of Publication: 3 Dec 2015  
  
OBJECTIVES The low molecular weight heparins (LMWH) are typically   
administered at fixed doses like thromboprophylaxis or at doses adjusted to   
the weight of the patient in order to obtain a therapeutic effect. Generally   
they do not require laboratory monitoring, although it could be considered   
in special situations (renal failure, extreme weights, pregnant women). The   
LMWH do not affect the APTT, so it has been proposed to determine the   
anti-factor Xa activity when it is necessary to monitor its effect. The   
anti-factor Xa activity should be determined approximately 4 hours after sc   
administration of the LMWH that it is employed, concurring with the peak of   
activity. The therapeutic range of the anti-factor Xa activity is between   
0.6 and 1 IU / mL when LMWH is administered every 12 hours. At single daily   
dose is less clear, although it seems that lies above 1 IU / mL. Nowadays,   
LMWH are the anticoagulant of choice during pregnancy. Numerous in vitro and   
in vivo studies have shown the existence of an antineoplastic effect of   
heparin. LMWH is commonly used for prolonged treatment of thrombosis   
associated with cancer. METHODS The main aim of our study is to evaluate the   
efficacy of tinzaparin sodium at therapeutic doses in preventing VTE in   
renal failure, active cancer and/or patients with contraindications to oral   
anticoagulation. The dose has been therapeutic and adjusting it has been   
made in terms of anti-factor Xa levels obtained monthly. Hemorrhagic or   
thrombotic complications and other possible side effects have been assessed.   
Until now, a total of 70 patients, 42 men and 28 women aged between 30 and   
95 years old, have received tinzaparin sodium treatment. The main reason of   
anticoagulation are: atrial fibrillation and atrial flutter (with or without   
valve disease), VTE (with or without thrombophilia), stroke and transient   
ischemic attacks and mechanical prosthetic aortic and mitral valves (some of   
the patients carrying a double metal prosthesis). There was 1 resistance and   
1 allergic reaction to anti-vitamin K. 4 of the patients were pregnant and   
14 had renal failure. Prior to initiation of therapy, analytical   
determinations were performed, including: blood count, blood coagulation and   
biochemistry to assess renal function (urea and creatinine). 20 patients (14   
were anticoagulated by atrial fibrillation, 2 for bearing a mechanical   
aortic prosthesis and 4 because of DVT, 1 of which had also a TEP) had   
active cancer or were in remission from their neoplasia (3 multiple myeloma,   
1 LAM, 1 CMML, 4 renal tumors, 1 lung cancer, 5 prostate cancers, 1   
hepatocellular carcinoma, 2 colon cancer, 1 endometrial adenocarcinoma and 1   
retroperitoneal leiomyosarcoma). 1 with MDS was treated with LMWH because he   
had intra- and extrahepatic portal vein thrombosis. RESULTS Some of the   
patients had received prior treatment with anti-vitamin K (INR objective   
depending on pathology) but, in other cases, the low molecular weight   
heparin was the only treatment since the beginning of their anticoagulation.   
All the patients had received 175 IU / Kg of Tinzaparin Sodium once a day as   
initial dose, then the dose was adjusted according to the anti-factor Xa   
levels. They were controlled until 31/07/2015. In terms of side effects, 8   
patients presented complications: 3 mucosal bleeding, 2 episodes of stroke   
in a patient, hemoptysis, deep vein thrombosis and 2 bleeding at the   
puncture site of heparin, which have not required discontinuation of   
therapy. When these complications occurred, we proceeded to the   
corresponding heparin dose adjustment based on new determinations of   
anti-factor Xa. CONCLUSIONS Although only in 70 cases, the results obtained   
confirm the efficacy, safety and cost-effectiveness of the continuous use of   
LMWH. Determination of anti-factor Xa levels are considered very useful for   
dose adjustment parameter. In our study, tinzaparin sodium has proved to be   
very useful in preventing venous thromboembolism associated or not with   
cancer, in patients with conditions requiring anticoagulation and presenting   
contraindications to the use of anti-vitamin K. The results obtained have   
demonstrated that tinzaparin is safe and, most likely, further studies will   
provide valuable confirmation data to support the use of low molecular   
weight heparins in the prolonged treatment of patients who require oral   
anticoagulation and can not receive it.  
  
RECORD 134  
Ten years of cerebral venous thrombosis (CVT) in melbourne, australia: Male   
gender and presence of myeloproliferative neoplasm is associated with   
thrombotic recurrence in unprovoked events  
Lim H.Y. Ng C. Smith C.L. Donnan G. Nandurkar H. Ho P.  
Blood (2015) 126:23 (4468). Date of Publication: 3 Dec 2015  
  
Aim Cerebral venous thrombosis (CVT) accounts for 0.5-1.0% of all strokes   
and is a common cause of stroke in young people. The presentations are often   
heterogeneous and can be associated with significant morbidity and   
mortality. This review aims to evaluate our local experience in CVT compared   
to other venous thromboembolism (VTE) with a focus on risk factors for   
thrombotic recurrence. Methods Retrospective evaluation of consecutive CVT   
presentations from January 2005 to June 2015, at two major tertiary   
hospitals in Melbourne, Australia. Data collected included demographics,   
risk factors, management, complications, modified Rankin score (mRS) and   
mortality. Results 52 patients (31 female, 21 male) with median age 9.5   
(18-83) years, including 4 with cancer, presented with 53 episodes of CVT.   
Females were younger (32 vs 41 years, p=0.06). Typical presenting symptoms   
were headache (87%), nausea/vomiting (43%), visual disturbances (38%), focal   
neurological deficits (28%) and seizures (17%). All but one case was   
symptomatic, with 53% reporting symptoms in the preceding week. 18 (34%)   
failed to be diagnosed on initial presentation while 35% (13/37) of CT brain   
yielded false negative for thrombosis; all of which were subsequently   
diagnosed on magnetic resonance imaging (MRI) or CT angiography/venography.   
Commonly thrombosed sinuses included transverse/sigmoid (40%), superior   
sagittal (11%) or both (43%), with no location-dependent outcome   
differences. Nine (17%) had CVT-related haemorrhagic transformation and was   
associated with CVT-related death (2/9 vs 0/44; p=0.04). 28 episodes were   
provoked - twice more common in female (p=0.02) with 45% attributed to oral   
contraceptive pill(OCP). 44 patients (85%) had thrombophilia screen   
performed with 21% positivity. Median duration of anticoagulation was 6.5   
months (8 remained on long-term); 78% treated with warfarin. Eight (15%)   
required intensive care support, while 2 patients required decompressive   
surgery. 12 (23%) were not followed up in our institutions. At last   
follow-up of the remaining 40, 2 (5%) had worsening mRS of 3 2 compared to   
premorbid, 2 had CVT-related deaths and 2 succumbed to malignancy. 30%   
reported ongoing symptoms such as headaches, residual neurological deficits,   
seizures and memory impairment. There were three clot recurrences (1 CVT, 2   
portal vein thrombosis) - all male with initial unprovoked events and were   
subsequently diagnosed with myeloproliferative neoplasm (MPN). Of the 3, one   
was positive for JAK2V617F mutation. Men with unprovoked CVT had a 20% risk   
of recurrence, significantly higher compared to women with unprovoked events   
(3/15 vs 0/10; p=0.02). Clot progression, defined as increased clot burden   
on repeat imaging, occurred in 2 patients - one was associated with MPN   
while another progressed in the setting of subtherapeutic anticoagulation   
post partum. There was one episode of Grade III bleeding (following a   
procedure) in addition to the 2 (4%) clot-related deaths discussed prior.   
Conclusions CVT is rare and may be missed on initial presentation (34%)-with   
a high degree of clinical suspicion required to improve detection rate.   
Given there was 35% of CT brain had false negative, MRI or CT angiography is   
the preferred modality of investigation. It is more common in young people,   
particularly females on OCP. The presence of haemorrhagic transformation was   
associated with higher mortality. All thrombotic recurrences in this audit   
occurred in men with unprovoked events, who were subsequently diagnosed with   
MPN. This suggests the need for further evaluation, particularly for MPN in   
males with unprovoked events. (Table Presented).  
  
RECORD 135  
Predictive factors of splanchnic vein thrombosis in acute pancreatitis: A   
6-year single-center experience  
Toqué L. Hamy A. Hamel J.-F. Cesbron E. Hulo P. Robert S. Aube C. Lermite E.   
Venara A.  
Journal of Digestive Diseases (2015) 16:12 (734-740). Date of Publication: 1   
Dec 2015  
  
Objective: Splanchnic vein thrombosis (SVT) is a potentially severe   
complication of pancreatitis. The aim of this single-center, retrospective   
cohort study was to investigate the incidence of SVT and to determine the   
connected risk factors. Methods: All consecutive patients with acute   
pancreatitis (AP) managed in our hospital were included. The primary outcome   
was the occurrence of SVT and data was collected in accordance with Ranson's   
criteria. Results: A total of 318 patients were included, of whom 124   
(39.0%) were women. Biliary lithiasis was the main cause of pancreatitis   
(n=156, 49.1%). A total of 19 (6.0%) SVT were identified. In univariate   
analysis, alcohol intake, smoking and male gender were associated with SVT   
(P = 0.005, 0.003 and 0.007, respectively). Biological parameters   
significantly associated with thrombosis were lactate dehydrogenase   
(LDH)<500 U/L and hyperglycemia (≥10 mmol/L) (P=0.009 and 0.016,   
respectively). In multivariate analysis, prothrombin time>75% was a   
protective factor against thrombosis (OR 0.148, P=0.019). Leukocytes   
>10×10(9)/L (OR 6.397, P=0.034), hyperglycemia (≥10mmol/L) (OR 6.845,   
P=0.023), LDH<500 U/L ((OR 22.61, P=0.001) and alcoholic etiology (OR 8.960,   
P =0.041) were risk factors for SVT. Conclusions: Alcohol intake, male   
gender and smoking should focus the physician's attention on the risk of   
SVT. When further associated with certain biological parameters, the   
physicians should consider therapeutic anticoagulation to prevent SVT.  
  
RECORD 136  
Outcome of anticoagulation in patients with cirrhosis and portal vein   
thrombosis  
Srijith K. Joseph D. Ramu M. Chethan G. Prasanth T.S. Suraj N. Gopu Sreejaya   
S. Das K.  
Indian Journal of Gastroenterology (2015) 34:1 SUPPL. 1 (A36). Date of   
Publication: November 2015  
  
Background and Aims: portal vein thrombosis is a frequent event seen in   
patients with cirrhosis and can be treated with anticoagulants. There are   
limited data regarding its safety in patients with cirrhosis. We   
retrospectively analyzed this treatment approach in patients with cirrhosis   
and portal vein thrombosis. Methods: We analyzed data of 45 patients with   
cirrhosis and portal vein thrombosis diagnosed in between the period January   
2011 to December 2014 who presented to our Department of Medical   
Gastroenterology, Govt. Medical College, Thiruvananthapuram. Portal vein   
thrombosis was diagnosed and recanalization was evaluated by using Doppler   
ultrasound and CECT abdomen (hepatic protocol). Results: Partial or complete   
recanalization was achieved in 22 patients (48.88 %). The factor   
significantly associated with recanalization was early initiation of   
anticoagulation. Patients with recanalization had less frequent   
complications such as portal hypertension related bleeding, ascites and   
hepatic encephalopathy during the follow up period. Four patients developed   
bleeding complications. The patients who developed bleeding complications   
were Child C and had had a platelet count less than 50,000. Two deaths were   
reported related to bleeding complications related to anticoagulation   
therapy. Conclusions: anticoagulation is a relatively safe treatment that   
leads to partial or complete recanalization of the portal vein in about 49 %   
of patients with cirrhosis and PVT. Bleeding complications though infrequent   
may be related to low platelet count.  
  
RECORD 137  
Cutaneous thrombosis as the presenting finding of paroxysmal nocturnal   
haemoglobinuria  
Salim O. Yücel O.K. Karatas G. Alan S. Bassorgun C.I. Undar L.  
British Journal of Haematology (2015) 171:3 (296-296). Date of Publication:   
1 Nov 2015  
  
RECORD 138  
Family history of venous thromboembolism is a risk factor for venous   
thromboembolism in combined oral contraceptive users: A nationwide   
case-control study  
Zöller B. Ohlsson H. Sundquist J. Sundquist K.  
Thrombosis Journal (2015) 13:1 Article Number: 34. Date of Publication: 21   
Oct 2015  
  
Background: The aim was to assess the risk of venous thromboembolism (VTE)   
associated with use of combined oral contraceptives (COCs) in women with a   
family history of VTE. Methods: The study is a Swedish nationwide   
case-control study based on the Multigeneration register, the Swedish   
Hospital Discharge Register, the Outpatient Care Register, and the Swedish   
Prescribed Drug Register. Cases (n = 2,311) were non-pregnant Swedish women   
aged 15-49 with first VTE diagnoses between January 2006 and December 2010.   
Five controls without VTE were matched to each case on age and education   
level. Conditional logistic regression examined the associations with VTE   
with determination of odds ratio (OR) for first VTE diagnosis. Effect   
modification was assessed by interaction testing. Results: Both among   
controls (14.6 % vs. 4.5 %; p < 0.0001) and cases (27.2 % vs. 8.8 %; p <   
0.0001) COC use was more common in women without a family history of VTE   
compared with women with a family history of VTE. In a multivariate   
conditional logistic regression model the OR for VTE was 2.53 (95 % CI   
2.23-2.87) for COC users and 2.38 (2.09-2.71) for individuals with a family   
history of VTE. The OR for VTE for COC users with a family history of VTE   
was 6.02 (5.02-7.22). There was no significant interaction between family   
history of VTE and COC use (OR 0.92, 0.57-1.46). Conclusions: Family history   
of VTE is a risk factor for VTE in women using COCs. The low prevalence of   
COC use among women with a family history of VTE suggests that family   
history of VTE is considered when COCs are prescribed in Sweden. The present   
study may therefore even underestimate the importance of family history of   
VTE. The lack of interaction indicates that the risk of COC use in women   
with family history of VTE is determined by the product of the ORs for   
family history and COC use.  
  
RECORD 139  
Impact of regional vein thrombosis in patients with Klebsiella pneumoniae   
liver abscess  
Molton J.S. Chee Y.L. Hennedige T.P. Venkatesh S.K. Archuleta S.  
PLoS ONE (2015) 10:10 Article Number: e0140129. Date of Publication: 7 Oct   
2015  
  
Klebsiella liver abscess (KLA) is an emerging infection in Asia caused by   
hypermucoviscous strains of Klebsiella pneumoniae. It is associated with   
thrombophlebitis of portal and hepatic veins. The natural history and role   
of anticoagulation for this regional thrombophlebitis is unclear. In a   
retrospective study of 169 subjects with KLA over 7 years, thrombophlebitis   
was identified in 53/169 (31.4%). Only 1 received therapeutic   
anticoagulation. Despite this 30/49 (73.2%) of those with follow up scan   
available showed improvement or recanalization (mean duration between scans   
44 days). Abscess resolution was associated with improvement in   
thrombophlebitis. Copyright:  
  
RECORD 140  
anticoagulation in patients with cirrhosis and portal vein thrombosis is   
associated with increased portal vein recanalization and better prognosis  
Ferreira C.N. Rodrigues T. Pedro A.J. Ferreira P. Dias M.S. Gonc¸alves A.   
Xavier Brito L. Serejo F. Marinho R.T. B. Costa C. Fatela N. Cortez-Pinto H.   
Ramalho F. Alexandrino P. Velosa J.F.  
Hepatology (2015) 62 SUPPL. 1 (285A). Date of Publication: October 2015  
  
Introduction: cirrhosis is recognized as a prothrombotic state. A recent   
study showed that prophylactic anticoagulation prevented portal vein   
thrombosis (PVT) and decreased episodes of decompensation of cirrhosis.   
Aims: To analyze the effect of anticoagulation on recanalization of   
non-tumoral PVT in patients with cirrhosis and its effect on prognosis.   
Methods: 69 consecutive patients with cirrhosis diagnosed with non-tumoral   
PVT were studied. The clinical features at diagnosis of PVT and factors   
associated with anticoagulation use were studied. Decision to start   
anticoagulation was taken at the discretion of the clinician managing the   
patient. The effect of anticoagulation on PVT recanalization and mortality   
was analyzed. Results: The average age was 58.6±11.8 years and 44(64%) were   
males. Severity of cirrhosis: Median(Range) Child-Pugh(CP) score: 8(5-15),   
MELD score:13(6-35). CP class: A-15(22%), B-32(46%), C-22(32%). At diagnosis   
of PVT, 55(80%) were symptomatic. Variceal bleeding(VB) in 30(46%) and   
abdominal pain in 19(29%) were the main clinical presentations.   
anticoagulation (LMWH-9, warfarin-16) was administered in 25(36%) patients   
one of whom with cavernoma. Patients with VB were less likely to be given   
anticoagulation (p=0.037). There were no differences in age, gender,   
etiology, severity of cirrhosis and extent of PVT in patients receiving, or   
not, anticoagulation. Recanalization of PVT was assessed by at least one   
imaging study in 60 patients and recanalization (Total-13, partial - 9) of   
the portal vein was documented in 22(37%) patients. Median (Range) follow-up   
was 21(0-376) months. At the end of follow-up, 29(42%) patients died, of   
which sixteen deaths were related to infectious complications with no deaths   
due to anticoagulation related bleeding. By Cox regression analysis, factors   
associated with mortality at the end of follow-up were: Age (HR 1.040, 95%   
C.I. 1.002-1.078, p=0.037), CP score (HR 1.35, 95% C.I. 1.18-1.55,   
p<0.001),MELD score (HR 1.14, 95% C.I. 1.08-1.21, p<0.001), creatinine   
(HR1.52, 95% C.I. 1.06-2.16, p=0.021). anticoagulation significantly   
decreased mortality at the end of follow-up even after adjusting for VB at   
diagnosis of PVT (HR 0.30 95% C.I.0.11-0.82, p=0.019). KM survival analysis   
confirmed that patients with cirrhosis and PVT given anticoagulation had   
better outcome compared to those not given anticoagulation(p=0.025) Portal   
vein recanalization was more frequent in patients on anticoagulation than no   
anticoagulation (61% vs 22%) (p=0.005). Conclusions: anticoagulation in   
patients with cirrhosis and PVT seems to be safe and associated with higher   
portal vein recanalization rates and significantly lower mortality.  
  
RECORD 141  
Pylephlebitis: A case of suppurative thrombophlebitis of the portal system  
Achdjian H.S. Scherback D. Young M.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S315-S316). Date   
of Publication: October 2015  
  
Pylephlebitis is a suppurative thrombophlebitis of the portal system, a rare   
complication of an intraabdominal infection with severe and life-threatening   
illness. Nonspecific clinical findings make the diagnosis of pylephlebitis   
challenging. Broad-spectrum antibiotics should be started immediately.   
anticoagulation is considered case-by-case basis. A 67-year-old Caucasian   
male with minimal past medical history presented with a two week duration of   
diffuse abdominal pain associated with fevers, chills and generalized   
fatigue. On admission, he was febrile to 103oF, abdomen was soft, mildly   
tender to palpation in the right upper quadrant and epigastrium, and bowel   
sounds were normal. WBC 10.8, AST 42, ALT 43. Hypercoagulable workup was   
negative. CT abdomen/pelvis with contrast showed thrombosis of the left   
portal, superior mesenteric and the inferior mesenteric veins. PET scan   
demonstrated metabolic activity in the left lobe of the liver, corresponding   
to the course of thrombosed left portal vein. Colonoscopy revealed terminal   
ileum with mucosal changes with blue discoloration, representing vascular   
congestion from thrombosis. Patient was started on pipercillin/tazobactam   
upon admission. Blood cultures subsequently grew Bacteroides fragilis.   
Patient was discharged home on enoxaparin as a bridge to warfarin for   
anticoagulation, completed a twenty-six day course of moxifloxacin and was   
doing well on follow-up three months later. Pylephlebitis occurs secondary   
to intra-abdominal infection; most commonly from acute colonic   
diverticulitis, appendicitis, inflammatory bowel disease, suppurative   
pancreatitis or bowel perforation. It is a polymicrobial infection with   
Bacteroides fragilis being the most common isolate. The clinical   
presentation is often vague and nonspecific with symptoms of non-localizing   
abdominal pain, fever, fatigue, nausea and vomiting. Laboratory findings   
include leukocytosis, elevation of AST and/or ALT, an increase of ALP and/or   
GGT. Positive blood cultures are found in 55-88% of patients. The diagnosis   
is ultimately based on an abdominal source of infection along with imaging   
demonstrating portal vein thrombus. An abdominal CT with oral and IV   
contrast is the modality most widely used. When suspected, broadspectrum   
antibiotics should be started immediately. Parenteral antibiotics are   
administered for the first 1-3 weeks until clinical improvement is noted and   
subsequently transitioned to oral antibiotics. The role of anticoagulation   
in pylephlebitis has not been well established and remains controversial,   
yet useful in mesenteric vein thrombosis, in patients with hypercoagulable   
states and in infection with Bacteroides species. Pylephlebitis mortality   
rate is 25% and is more likely secondary to severe sepsis leading to bowel   
infarction. (Figure Presented).  
  
RECORD 142  
A case of persistent JP drain output and ileus after cholecystectomy  
Mittal V. Sao R. Gollapudi L.A. Jodorkovsky D.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S448-S449). Date   
of Publication: October 2015  
  
Introduction: portal vein thrombosis (PVT) can cause complications such as   
ascites, diarrhea, ileus, transient moderate elevation in serum   
aminotransferases and even intestinal ischemia from clot extension into   
mesenteric veins. Here, we describe a case of persistent ileus resulting   
from PVT after cholecystectomy. Case presentation: 72 year-old male with   
DM-2 was diagnosed with choledocholithiasis and biliary pancreatitis two   
weeks prior for which he underwent endoscopic retrograde   
cholangiopancreatography (ERCP) with stone extraction. Subsequently, he was   
taken for laparoscopic cholecystectomy which was converted into open   
cholecystectomy due to portal vein damage which was immediately repaired. He   
then developed small bowel ileus, persistent nasogastric tube drainage and   
non-bloody diarrhea. The JP drain had persistent output as high as 1   
liter/day. A HIDA scan ruled out bile leak and an ultrasound with doppler   
ruled out thrombosis. He was subsequently transferred to our medical center.   
On examination, abdomen was soft , moderately distended, diffusely tender   
and an indwelling JP drain in RUQ was draining serous fluid. The fluid   
analysis was not suggestive of bile leak (fluid total bilirubin = 0.4 mg/dL,   
serum bilirubin = 1 mg/dL) and SAAG ratio was 2.2 (fluid albumin = 0.7 g/dL,   
serum albumin = 2.9 g/dL), consistent with portal hypertension. An   
ultrasound with doppler study showed patent portal veins with slow flow. A   
CT scan with contrast showed a non-occlusive thrombus involving the   
extrahepatic main portal vein, obstructing >75% lumen, superior and inferior   
mesenteric venous thrombosis, and ascites. Over course of next few days   
after patient was started on anticoagulation, the ileus resolved and JP   
drain output decreased gradually. Patient was able to advance his diet and   
was discharged home. Discussion: The major risk factors for PVT include   
cancer of any abdominal organ, focal inflammatory lesions (e.g.   
pancreatitis, cholecystitis, duodenal ulcer), portal vein injury and   
cirrhosis. Several factors particular to laparoscopic procedures such as   
venous stasis, alteration in coagulation parameters as a result of   
pneumoperitoneum and damage of splanchnic endothelium by surgical   
manipulation also play a role. The portal vein is much less vulnerable to   
injury in cholecystectomy than the right hepatic artery, so a high index of   
suspicion is required for diagnosis. Although ultrasound with doppler is   
fairly sensitive (89-93%) and specific (92-93%), CT or MR angiography have a   
higher sensitivity for diagnosing PVT. The goal of treatment is to   
recanalize the obstructed veins, which will prevent hepatic and intestinal   
infarction and portal hypertension. (Figure Presented).  
  
RECORD 143  
portal vein thrombosis as initial manifestation of ulcerative colitis  
Vega K.J. Kanagala R. Smith Z.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S305-S306). Date   
of Publication: October 2015  
  
Introduction: Although uncommon, inflammatory bowel disease (IBD) has been   
found to be an independent risk factor for acquired thrombosis, even rarer   
is portal vein thrombosis (PVT) as the presenting illness. Case Report: A 28   
y/o nonsmoking female presented with 10 days of worsening abdominal pain. In   
addition, she reported nausea, vomiting, and bloody stools. On PE, she was   
alert, oriented, afebrile, mild tachycardia, normal blood pressure and   
generalized abdominal tenderness on deep palpation only. Routine labs   
indicated mild anemia, normal WBC and platelets, INR of 2.3 and elevated   
transaminases. Infectious stool evaluation did not reveal C. difficile,   
Salmonella, Shigella, Campylobacter, E. coli, or Ova/ parasitic infection.   
However, stool showed increased Lactoferrin. Abdominal imaging revealed   
acute PVT on RUQ ultrasound with Doppler and CT abdomen/pelvis suggested   
colitis. Hypercoagulable state assessment did not confirm Lupus   
anticoagulant, Factor V Leiden or prothrombin abnormalities. Colonoscopy   
revealed hemorrhagic, inflamed and ulcerated mucosa from the rectum to   
transverse colon; biopsies displayed an interstitial   
neutrophilic/lymphoplasmocytic infiltrate with surface ulceration, cryptitis   
and crypt abscesses. She was treated simultanously with intravenous   
methylprednisolone q8 hours and enoxaparin BID for colitis and PVT,   
respectively. Patient responded well to treatment, eventually transitioning   
to oral prednisone and mesalamine as well as warfarin with resolution of all   
presenting symptoms. Discussion: Fewer than 10% of IBD patients have an   
extra intestinal manifestation at initial presentation. These are most   
commonly sacroilitis, peripheral arthritis, ocular, mucocutaneous and   
vascular. In one study, thromboembolic complications occurred in 1.3% of IBD   
patients with majority having deep vein thrombosis or pulmonary embolism. In   
IBD patients, inpatient status and steroid therapy as well as post bowel   
resection are risk factors for development of porto-mesenteric venous   
thrombosis. However, acute PVT has not been reported as part of an IBD   
initial presentation previously. Conclusion: PVT is very uncommon in   
patients with IBD, especially at presentation. If present, complete   
evaluation (including hypercoagulable state assessment, medication history   
review, smoking status and imaging) should occur. Therapeutic options   
including anticoagulation or thrombolysis can be used while simultaneously   
treating the IBD episode. (Figure Presented).  
  
RECORD 144  
Infective portal vein thrombosis: A rare complication of pancreatitis  
Al-Hamid H. Manatsathit W. Johnson L. Barawi M.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S101). Date of   
Publication: October 2015  
  
Introduction: “Pylephlebitis” or infective suppurative thrombosis of the   
portal vein is a rare condition with a 30-80% mortality rate. Most cases are   
associated with intra-abdominal sepsis. Diverticulitis and appendicitis are   
the primary foci in most reported cases. Only 5% of cases are associated   
with pancreatitis. Case: A 38-year-old African American female with recent   
history of alcoholic pancreatitis complicated by pseudocyst status post   
endoscopic retrograde cholangiopancreatography (ERCP) with pseudocyst   
drainage presented with acute abdominal pain, fevers, nausea and vomiting.   
Physical examination revealed normal heart rate and blood pressure.   
Abdominal exam was significant for severe epigastric tenderness and palpable   
liver margin. Laboratory values included WBC 10.5, hematocrit 23.6, lipase   
31, alkaline phosphatase 144 and normal hepatic transaminases. Contrast   
enhanced computerized tomography scan of the abdomen showed acute portal   
vein thrombosis with multiple low-density lesions within the liver   
suggestive of abscesses. Blood cultures grew the anaerobic bacteria   
Eubacterium aerofaciens. The diagnosis of infective suppurative thrombosis   
of the portal vein was rendered. Parenteral antibiotics and anticoagulation   
were initiated. CT-guided drainage of the largest liver abscess was   
performed and cultures grew Streptococcus viridans. She showed progressive   
clinical improvement and she was successfully transitioned to outpatient   
care. Discussion: Portal vein pylephlebitis is very rare and usually   
presents with nonspecific clinical and laboratory findings. Pancreatitis is   
an uncommon etiology. Hepatic abscesses can complicate severe cases. The   
prothrombotic effect of the underlying infectious or inflammatory process is   
the main proposed mechanism. The early recognition and management of   
pylephlebitis with antibiotics and anticoagulation play significant role in   
outcome.  
  
RECORD 145  
Intestinal ischemia after thrombosis of a mesocaval shunt  
Sao R. Mehta D. Sharma N. Agarwal A. Haq K.F. Kassab M. Sule S. Wolf D.C.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S460). Date of   
Publication: October 2015  
  
Introduction: portal vein thrombosis (PVT) is a known common complication of   
umbilical vein catheterization. However, most cases resolve spontaneously.   
We report a case of a patient with chronic PVT secondary to umbilical vein   
catheterization - managed with mesocaval shunt surgery - who subsequently   
presented with shunt thrombosis and intestinal ischemia. Case Summary: A 34   
year old woman with a history of a mesocaval shunt presented with severe   
abdominal pain that was attributed to intestinal ischemia. The patient was   
born prematurely at 25 weeks of gestation. She underwent umbilical vein   
catheterization as a neonate. At age 1, she presented with PVT and variceal   
bleeding. This was managed with splenic artery embolization. At age 16, she   
again presented with variceal bleeding, this time managed by the creation of   
a mesocaval shunt. At age 34, she experienced the acute onset of severe   
diffuse abdominal pain and melena. There was severe diffuse abdominal   
tenderness with no rebound. Labs showed Hgb 8.7 gm/dL, platelets 52000/ mcL,   
normal liver function, INR 1.13. CT angiogram demonstrated extensive   
intraabdominal varices and occlusion of the mesocaval shunt. Venography   
demonstrated that the mesocaval shunt was completely thrombosed. Mesocaval   
shunt dilation was performed with an angioplasty balloon. After balloon   
deflation, venography showed that both the portal and caval anastomoses were   
narrowed. A Wallstent prosthesis was placed across both anastomoses,   
resulting in a functional shunt and decreased hepatopetal flow. Abdominal   
pain gradually resolved after stent placement. A work-up to rule out a   
hypercoagulable state was unremarkable. Pan-endoscopy post shunt revision   
showed no active bleeding, moderate esophageal varices, small gastric and   
rectal varices and severe portal hypertensive gastropathy. The specific GI   
bleeding site was not identified. Given patient's history of falls,   
anticoagulation was not begun and the patient was discharged on aspirin and   
pantoprazole. Plans were made for periodic reassessment of shunt patency.   
Discussion: Mesocaval shunts undergo thrombosis in about 10% of cases. When   
they thrombose, subsequent intestinal ischemia is rarely reported.   
anticoagulation has not been shown to alter the outcome of umbilical vein   
catheterization-related PVT. However, anticoagulation is an important tool   
for the prevention of recurrent mesenteric venous thrombosis. It should be   
employed whenever possible.  
  
RECORD 146  
Efficacy and safety of treatment of acute nonmalignant portal vein   
thrombosis with subcutaneous fondaparinux in patients with cirrhosis and   
marked thrombocytopenia  
Tonon M. Piano S. Sacerdoti D. Dalla Valle F. Grbec M. Spiezia L. Bolognesi   
M. Simioni P. Angeli P.  
Hepatology (2015) 62 SUPPL. 1 (591A). Date of Publication: October 2015  
  
Fondaparinux (FPX), a factor Xa inhibitor, has been recommended for   
anticoagulation therapy in patients at high risk of bleeding. In addition,   
it rarely induces thrombocytopenia since anti-PF4/heparin antibodies which   
are rarely generated during FPX treatment, are not able to bind PF4/FPX   
complexes. In spite of these potential advantages, there are no data   
regarding the use of FPX as anticoagulant treatment of acute nonmalignant   
portal vein thrombosis (PVT) in patients with liver cirrhosis. The aim of   
this prospective pilot study was to evaluate the safety, and efficacy of   
subcutaneous FPX therapy as anticoagulation therapy in a cohort of patients   
with cirrhosis and acute non malignant PVT. Methods Forty-two patients with   
liver cirrhosis and acute nonmalignant PVT were included into the study   
between 2010 and 2014. Patients with malignant PVT, Budd-Chiari syndrome,   
underlying primary hematologic disorders, were excluded from the analysis.   
The extension of PVT at baseline as well as its evolution on treatment were   
evaluated by both Doppler ultrasound and CT. At baseline, 31 out of 42   
patients (73.8.%) had a platelet count lower than 70.000/ mm3, while 19   
(45.23%) had a platelet count < 50.000/ mm3. For anticoagulation, the dose   
of FPX was adjusted on the body weight (BW), and on the trend of platelet   
count ranging from 2.5 to 7.5 mg/die. Results After a mean period of 16,36 ±   
13,86 months of treatment, 18 patients (42,86%) showed a complete resolution   
of PVT, defined as disappearance of all evidence of thrombosis. Ten patients   
(23,81%) showed a partial resolution, defined by a decrease ≥ 30% reduction   
in the main diameter of the main thrombus and/or a decrease ≥ 50% in   
cross-sectional area without evidence of the appearance of new thrombi. 14   
patients (33,33%) showed no response, defined as a decrease in thrombus size   
that did not qualify for partial resolution, or as a progression of   
thrombosis or the appearance of new thrombus. One patient developed a   
non-lethal major bleeding event (haemoperitoneum three day after   
paracentesis) while 5 patients developed a non-major bleeding events during   
the study (14.3%). In all these patients but one FPX was discontinued. No   
significant change was observed in the platelet count during treatment   
(67.318,18/mm3 ± 36.830,08 mm3 versus 68.255,81/mm3 ± 36.358,72, P = N.S.).   
Nevertheless, in 6 patients FPX was withdrawn for a worsening of   
thrombocytopenia (last value during treatment <15.000/mm3). Conclusions In   
patients with cirrhosis and marked thrombocytopenia, FPX seems to be   
effective and safe in the treatment of acute nonmalignant PVT.  
  
RECORD 147  
Use of betablockers, previous hepatic encephalopathy and low albumin levels   
as risk factors of portal vein thrombosis in a cohort of cirrhotic patients  
Gomez M.L. Llop E. Puente A. De La Revilla J. Fernández-Carrillo C. Pons F.   
Martinez J.L. Fernández N. Trapero M. Crespo J. Calleja J.L.  
Hepatology (2015) 62 SUPPL. 1 (947A-948A). Date of Publication: October 2015  
  
portal vein thrombosis(PVT) is a complication of liver cirrosis( LC). The   
aim of our study was to evaluate anual incidence of PVT and related risk   
factors.Methods: We retrospectively reviewed clinical and radiological data   
collected prospectively of consecutive cirrhotic patients included in the   
database of two Universitary Hospitals. Patients out of Milan criteria HCC,   
known PVT, TIPS and pregnancy were excluded. All patients with ultrasound   
diagnosis of PVT underwent MR or CTangiography.Results: From September 2013   
to September 2014, 747 cirrhotic patients were reviewed, 179 had exclusion   
criteria. Baseline characteristics are described in Table 1. 23(4%) patients   
presented PVT during the inclusion period. Significant differences between   
patients with/without PVT were observed in: albumin   
(3.4SD0.8vs4.0SD0.5;p<0.001), AST(41.5 SD23.2vs62SD47;p=0.04), hemoglobin   
(12.6SD2.3vs13.8SD2.2;p=0.01) and prothrombin activity (0.64   
SD0.16vs0.77SD0.18;p=0.01). The presence of ascites( 60.9%vs29.7;p 0.02),EV   
(77.3%vs39.1%;p<0,001),previous history of HE(39.1%vs9.9%;p<0,01),SBP(13%vs   
1,7%;p<0,01),VB(56.5%vs20.8%;p<0.01), and use of BB(65.2%vs26.6%;p<0.01)   
were also significantly associated. In the mutlivariate analysis   
BB(OR4.3IC1.4-12.6;p=0.01) and HE(OR3.2 IC 1.1-8.; p0.03) were risk factors   
and high albumin levels(OR0.3IC0.2-0.8p=0.01) was as a protective factor.   
Besides, significant differences were observed in PVD(12.2SD-   
5vs10.7SD2;p=0.02) and SD(15SD3vs13SD2.6;p<0.001). Although,PVS was not   
significantly lower in patients with PVT, patients with BB had significantly   
lower PVS(15SD4.2vs16.4 SD3.9;p <0.003). 20(87%) patients received   
anticoagulation a median time of 9 months(1-12), 5 achieved   
repermeabilization and 1 presented decompensation. Conclusions: PVT had a 4%   
incidence. Risk factors were the use of BB and HE. High albumin levels were   
a protective factor. (Table Presented).  
  
RECORD 148  
Cerebral venous sinus thrombosis associated with weight loss pills  
Elkouzi A. Karroum E.G. Kale S.  
Annals of Neurology (2015) 78 SUPPL. 19 (S25-S26). Date of Publication:   
October 2015  
  
Objective: To associate cerebral venous Sinus thrombosis (CVST) in a young   
male with intake of weight loss pills. Background: 13% of CVST remain   
idiopathic. Weight loss pills were not reported before as causative agents   
for CVST. Method: Case report. Description: 34 year old man with focal   
seizures was diagnosed to have extensive CVST. He took weight loss pills for   
1 year prior to event. He progressed to status epilepticus despite   
treatment. MRV brain and angiogram shows the extent of his CVST.   
Echocardiogram, Venous Duplex of the extremities, hypercoagulable profile   
were normal. Factor VIII level was elevated. There was no mutation of Factor   
V or prothrombin gene. He underwent endovascular suction thrombectomy with   
improvement in his clinical status. He was discharged home stable on oral   
anticoagulation. “Cassia angustifolia-(Senna Sennoside)” was reported to   
cause portal vein thrombosis with chronic use. There was a temporal   
association between intake of weight loss pills and development of CVST in   
this young man. Whether ingredients like “Cassia Angustifolia” induced CVST   
through Elevation of factor VIII levels or by an alternative mechanism   
potentiating the prothrombotic effect of elevated factor VIII needs further   
studies. Conclusion: To our knowledge this is the first reported case of   
CVST associated with weight loss pills.  
  
RECORD 149  
Benefit Stratification of Prophylactic anticoagulation in liver cirrhosis:   
More Questions Than Answers  
Qi X. Guo X. Fan D.  
Clinical Gastroenterology and Hepatology (2015) 13:10 (1856-1857) Article   
Number: 54351. Date of Publication: 1 Oct 2015  
  
RECORD 150  
Usefulness of balloon-occluded retrograde obliteration (B-RTO) as a   
consolidation procedure after anticoagulation therapy in cirrhotic patients   
with portal vein thrombosis  
Inao M. Hirahara K. Sugawara K. Nakayama N. Imai Y. Mochida S.  
Hepatology (2015) 62 SUPPL. 1 (935A). Date of Publication: October 2015  
  
Aim: Although anticoagulation therapies with Xa inhibitors and antithrombin   
concentrates were shown to be effective for attenuation of portal vein   
thrombosis in cirrhotic patients, aggravation or recurrence of the lesions   
may occur following the therapies leading to derangement of liver function.   
Decrease of blood flow in the portal vein as a consequence of porto-systemic   
shunts may responsible for thrombosis development. Thus, the usefulness of   
B-RTO as a consolidation procedure after anticoagulation therapies was   
evaluated. Methods: The subjects were 43 patients (23 men and 20 women, aged   
from 40 to 76 years old) with liver cirrhosis complicating portal vein   
thrombosis. Both danaparoid Na (2,500 units/day) and antithrombin   
concentrates (1,500 units/day) were intravenously administrated for 3 days   
followed by danaparoid Na injections for further 11 days. Patients seen in   
April 2013 and later received B-RTO procedures after anticoagulation   
therapies, when porto-systemic shunts were observed on CT and/or MRI   
imaging. A balloon catheter was inserted into the shunts followed by   
injection of 5% ethanolamine oleate through the catheter under balloon   
inflation. The balloon was kept inflation for 6 to 48 hours depending on   
sizes of the shunts. Results: Immediately after anticoagulation therapies,   
portal vein thrombosis was completely disappeared in 11 patients (25%) and   
the sizes of thrombosis were attenuated in 15 patients (35%), while the   
lesions did not change in 17 patients (40%). B-RTO was additionally done in   
4 patients; 2 patients showing complete thrombosis disappearance and 2   
patients failing to achieve thrombosis attenuation. Following B-RTO   
procedures, thrombosis did not recur in both of the former patients and the   
lesions disappeared in both of the latter patients despite that   
anticoagulation therapies were ineffective. In contrast, in 39 patients   
without additional B-RTO procedures, thrombosis recurred in 4 among 9   
patients after thrombosis disappearance and was aggravated in 6 among 15   
patients achieving thrombosis attenuation. Conclusion: B-RTO was effective   
as a consolidation procedure after anticoagulation therapies for patients   
with portal vein thrombosis even in those failing to achieve attenuation of   
the lesions when porto-systemic shunts responsible for decrease of blood   
flows in the portal vein were observed.  
  
RECORD 151  
portal vein thrombosis as a cause of massive ascites in a non-cirrhotic   
patient  
Ramirez C.B. Preeshagul I. Sanchez J.G. Shrensel J.A. Kutner M. Favila K.  
American Journal of Gastroenterology (2015) 110 SUPPL. 1 (S388). Date of   
Publication: October 2015  
  
The prevalence of portal vein thrombosis (PVT) in the general population is   
< 1%. Predisposing conditions associated PVT include cirrhosis, most   
commonly (up to 35%), hepatobiliary malignancies, abdominal infectious or   
inflammatory processes, abdominal trauma or myeloproliferative disorders.   
PVT in a non-cirrhotic patient is rare and is hypothesized to be secondary   
to an underlying hypercoagulable state. A 65-year-old male presented with   
progressive abdominal pain and distention over the past 3 months. He denied   
any fevers, melena or hematemesis. He consumed 1 pint of vodka daily over   
the last 5 years. Exam was remarkable for abdominal distention, however,   
there was no stigmata suggestive of cirrhosis. Labs were significant for   
hemoglobin 8.2 g/dL, total bilirubin 0.6mg/dl, direct bilirubin 0.4mg/dl,   
alkaline phosphase 88 U/L, AST 123 U/L and ALT 64 U/L. Hepatitis C antibody   
was positive with an undetectable viral load. Otherwise, white blood cell   
count (WBC), platelet count, albumin, BUN/creatinine, coagulation profile,   
and remaining hepatitis serologies were normal. An abdominal CT was   
consistent with a large amount of ascites with a normal liver and spleen   
size and without morphologic changes suggestive of cirrohsis. Hypodensities   
in the lumen of the main and right portal veins were consistant with thrombi   
(fig1). Paracentesis was negative for subacute bacterial perotonitis with a   
calculated SAAG of 2.2 g/dL. Cytology was negative for malignant cells.   
Hypercoagulable work-up and additional imaging were planned however not   
completed as that patient left against medical advice. Ascites is a common   
symptom in patients with portal hypertension secondary to cirrhosis, however   
in this case, our patient was found to have ascites secondary to PVT in the   
absence of cirrhosis. PVT complications include bowel ischemia, portal   
cholangiopathy, septic portal vein thrombosis, and portal hypertension.   
Management is controversial and typically depends on the acuity of the   
thrombi. anticoagulation therapy is generally warranted. However, mortality   
rates in PVT range from 1-20% with a majority of deaths caused by variceal   
bleeding. Ascites, in the setting of PVT is a significant and independent   
prognostic factor and associated with a decreased long-term survival. It is   
important to recognize portal vein thrombosis as an alternative etiology of   
ascites in patients without evidence of cirrhosis, as this can guide further   
management decisions. (Figure presented).  
  
RECORD 152  
Occult diffuse cholangiocarcinoma of the liver presenting as portal vein   
thrombosis  
Vakil A. Reddy D. Guru P.K. Iyer V.N.  
American Journal of Respiratory and Critical Care Medicine (2015) 191   
MeetingAbstracts. Date of Publication: 2015  
  
Introduction: portal vein thrombosis (PVT) is an extremely rare   
entity/condition with exact incidence being unknown in patients without   
cirrhosis. Some of the commonly known causes include cirrhosis, primary or   
secondary hepatobiliary malignancies, infectious or inflammatory abdominal   
processes and myeloproliferative disorders. We report the case of an elderly   
man who presented with idiopathic PVT. He rapidly developed encephalopathy   
requiring admission to the intensive care unit (ICU) followed by multiorgan   
failure leading to his death. Autopsy revealed diffuse intrahepatic   
cholangiocarcinoma almost entirely replacing his liver parenchyma. Case   
Description: A 64 year-old previously healthy male, presented with 4-week   
history of vague right upper quadrant abdominal pain. Laboratory values   
showed an elevated alkaline phosphatase (340 IU/L) with other markers of   
liver function, complete blood count and pancreatic enzymes in the normal   
range. Imaging studies revealed extensive portal, splenic, superior and   
inferior mesenteric vein thrombosis with mosaic perfusion and wedge shaped   
areas of liver infarction (Figure 1a). Extensive work-up failed to reveal   
any obvious cause for PVT. Surgical exploration and catheter directed   
thrombolysis options were not felt to be feasible given the extensive clot   
burden. The patient was started on anticoagulation therapy. Over the course   
of next 10 days he continued to deteriorate and developed progressive   
hepatic encephalopathy with worsening liver function requiring ICU   
admission. The patient continued to decline and eventually died of   
multi-organ failure. Autopsy revealed extensive, diffuse intrahepatic   
cholangiocarcinoma that had almost entirely replaced his normal liver   
parenchyma (Figure 1b). Figure 1(a): Computed tomograhy of abdomen showing   
mosaic perfusion and and wedge shaped areas of liver infarction, 1(b):   
Autopsy showing extensive, diffuse intrahepatic cholangiocarcinoma almost   
entirely replacing normal liver parenchyma. Conclusion: Although known to be   
one of the common causes of PVT, underlying hepatobiliary malignancies may   
sometimes remain undiagnosed therefore needing a high index of suspicion.   
The imaging findings can mimic liver infarction or be non-diagnostic   
especially if the underlying malignancy replaces the entire liver   
parenchyma. (Figure Presented).  
  
RECORD 153  
Expanding consensus in portal hypertension Report of the Baveno VI Consensus   
Workshop: Stratifying risk and individualizing care for portal hypertension  
De Franchis R. Abraldes J.G. Bajaj J. Berzigotti A. Bosch J. Burroughs A.K.   
D'Amico G. Dell'Era A. Garcia-Pagàn J.C. Garcia-Tsao G. Grace N. Groszmann   
R. Krag A. Laleman W. La Mura V. Lebrec D. Lo G.H. Merkel C. O'Beirne J.   
Peck M. Primignani M. Salerno F. Sarin S.K. Thabut D. Trebicka J. Zipprich   
A. Aabakken L. Albillos A. Augustin S. Bañares R. Boyer T. Bureau C. Castera   
L. De Gottardi A. Escorsell A. Genesca J. Gralnek I. Hernandez-Gea V.   
Leebeek F. Merli M. Moreau R. Nevens F. Pinzani M. Reiberger T. Ripoll C.   
Rudler M. Seijo S. Tandon P. Tsochatzis E. Valla D. Villanueva C. Vorobioff   
J. Shneider B. Talwalkar J. Wiest R.  
Journal of Hepatology (2015) 63:3 (743-752) Article Number: 5694. Date of   
Publication: 1 Sep 2015  
  
RECORD 154  
Treatment algorithm for portal and mesenteric vein thrombosis in cirrhosis  
Rössle M.  
Journal of Viral Hepatitis (2015) 22 SUPPL. 3 (12-13). Date of Publication:   
September 2015  
  
Portal and/or mesenteric vein thrombosis is detected in up to 28% of   
cirrhotic patients, with a cumulative incidence of 12.8, 20, and 38.7% at 1,   
5, and 8-10 years of followup, respectively (1, 2). In contrast to   
non-cirrhotic portal vein thrombosis (PVT) where coagulation or haematologic   
disorders play the dominant role, haemodynamic factors, i.e. decelerated   
blood flow, are of major importance. PVT in cirrhosis has a negative effect   
on outcome and transplantation (3), a fact which underlines the importance   
of a treatment algorithm. anticoagulation using low-molecularweight heparin   
(LMWH) or vitamin K antagonists are effective in the treatment of patients   
with limited and recent PVT, resulting in a recanalization in up to 50%   
irrespective whether low-molecular-weight heparin (LMWH) or warfarin was   
given. The efficacy of anticoagulation correlated negatively with delayed   
initiation of treatment (thrombus age) and extension of the thrombus (4). In   
10% of the patients receiving anticoagulation, the thrombus increased during   
treatment into mesenteric veins (5) with a respective clinical   
deterioration. Discontinuation of anticoagulation results in an early   
recurrence of 38% (4). Additional issues showing the limitations of   
anticoagulation are the probably limited compliance of long-term LMWH and   
the problem with monitoring of vitamin K antagonists (6). In contrast to   
anticoagulation, TIPS corrects the pathophysiology of PVT formation by   
considerably increasing the portal vein flow velocity. It results in a   
recanalization of 67-100% and reduces the rebleeding rate considerably in   
patients with chronic PVT, including cavernoma (1). The largest study   
including 70 consecutive patients with PVT in cirrhosis found an overall   
response of 87% with almost complete (>75%) or complete recanalization in   
81% (7). The rebleeding rates were very low after successful TIPS and   
differed significantly when compared to patients with unsuccessful   
intervention (8). Considering the pros and cons regarding anticoagulation   
and TIPS, the algorithm shown in Figure 1 is suggested. Patients with recent   
or incomplete PVT and limited extension and without symptoms of portal   
hypertension should receive anticoagulation as primary treatment. TIPS may   
also be a second-line treatment if patients do not respond to   
anticoagulation within 3-6 months. In contrast, TIPS may be the first-line   
treatment when thrombosis is complete, extended, or chronic, when patients   
have symptomatic portal hypertension (ascites, bleedings) or are candidates   
for liver transplantation (1). It should be kept in mind that   
recommendations are preliminary as long as randomized studies are lacking.   
(figure present).  
  
RECORD 155  
Percutaneous treatment options in portal vein thrombosis  
Krajina A. Hulek P. Chovanec V. Raupach J. Lojik M. Cabelkova P. Fejfar T.  
CardioVascular and Interventional Radiology (2015) 38:3 SUPPL. 1   
(S148-S149). Date of Publication: September 2015  
  
Learning Objectives 1. To describe the indications for acute and chronic   
portal and mesenteric vein occlusion treatment 2. To outline techniques and   
devices for recanalising portal and mesenteric vein occlusion 3. To describe   
the results of thrombectomy, thrombolysis, and mechanical recanalisation The   
portal vein (PV) is a closed system between two low-pressure capillary   
networks (1). Its thrombosis mainly occurs not only in patients with liver   
cirrhosis (2-4) but also in patients without liver disease (5, 6) as a   
complication of hypercoagulable syndromes, latent or overt   
myeloproliferalive disorder (7), and inflammatory processes in the abdominal   
cavity and as a result of iatrogenic injury (8, 9). PV obstruction causes   
portal hypertension. There are several terms describing various clinical   
settings in relationship with PV obstruction in the current literature. 1.   
PV thrombosis in liver cirrhosis could be caused by impaired blood flow due   
to intrahepatic sinusoidal block, and it is observed in up to 17% of   
patients with liver cirrhosis, especially in more advanced stages of   
disease. PV invasion frequently occurs in hepatocellular carcinoma, and it   
has become one of the most important prognostic factors for this disease   
(10). 2. Extrahepatic PV obstruction (EHPVO) is a vascular disorder of the   
liver. It is defined by the obstruction of the extra-hepatic PV with or   
without the involvement of intra-hepatic PV branches or splenic or superior   
mesenteric veins. 3. Isolated occlusion of the splenic vein caused by   
pancreatitis and/or external compression or infiltration by pancreatic   
tumorous expansion or tumors. 4. Acute PV thrombosis nonspecifically   
presents with abdominal pain, fever, and nausea. Majority of patients have   
splenomegaly. In contrast to the Budd-Chiari syndrome, ascites are rarely   
present. The most significant complication is venous bowel ischemia due to   
the extension of thrombosis to mesenteric veins (11). 5. Chronic PV   
thrombosis has a variety of clinical presentations. Majority of patients   
could be asymptomatic, and PV chronic thrombosis is an incidental finding.   
This can be explained by two compensatory mechanisms. There is compensatory   
increase of arterial blood flow in the hepatic artery and fast development   
of the collateral venous network bypassing the obstruction. Due to this   
compensatory arterial and venous blood flow, there is no or minimal   
reduction of blood inflow to the liver. However, portal hypertension   
develops with bleeding from gastroesophageal varices and portal gastropathy.   
There is a 12% risk of bleeding per year; higher risk is observed in   
patients with larger varices and previous history of bleeding. Portal   
biliopathy is another possible complication of chronic PV obstruction. It   
results from the obstruction of bile ducts by ectatic venous collaterals in   
their wall. There is a risk of extension of thrombosis to mesenteric veins   
with bowel ischemia (6). Percutaneous recanalization of acute PV thrombosis   
significantly differs from chronic PV occlusion in indications, technique,   
technical results, clinical outcome, and complications. Partial or complete   
acute PV thrombosis, which arises frequently as an urgent indication for   
TIPS because of endoscopically uncontrolled variceal bleeding, does not   
change the usual technique of TIPS. Released thrombi in the PV can cause   
obstruction of the new shunt and have to be mechanically removed. As soon as   
sufficient flow is established, remaining thrombi in the PV will dissolve   
with time. TIPS is technically difficult in chronic EHPVO, and its   
indication depends on the patency of some intrahepatic PV branches and on   
the extension of chronic thrombosis towards splenic and/or mesenteric veins.   
Technical success depends on the possibility to cross chronic vein   
obstruction with hydrophilic guidewire (12-15). Isolated splenic vein   
occlusion is usually indicated to splenectomy and/or surgical porto-systemic   
bypass. However, endovascular recanalization via transjugular or   
trans-splenic approach is feasible (16). Percutaneous endovascular   
procedures are used as an alternative to sclerotherapy or surgical shunting   
in order to improve clinical symptoms. Their main role is to debulk the   
thrombus by means of mechanical thrombectomy or pharmacological thrombolysis   
alone or by blood flow facilitation using TIPS (1,17,18). Techniques of   
portal vein recanalization The crucial imaging modality is contrast-enhanced   
CT, which demonstrates patency of intrahepatic portal branches, splenic,   
mesenteric, and hepatic veins, and the inferior vena cava, and extension of   
the thrombus towards feeders of the PV. Our primary approach is a   
transjugular one for the portal vein access using Rosch-Uchida set (Cook   
Inc., USA) and 180-cm angled tip hydrophilic guidewire (Terumo, Japan). As   
soon as the guidewire is safely in the PV, TIPS is performed using bare   
stent. Through this approach, we utilize various mechanical devices to   
fragment and aspirate the thrombus (Arrow-Trerotola Over-The-Wire PTD Kit;   
Arrow International, Inc.). As soon as the blood flow is reestablished, we   
wait for at least 10 minutes for any sign of recurrent thrombosis or flow   
impairment. Acute and subacute thrombus is soft and easy to cross with   
hydrophilic guidewire. In case blood flow is not established, a 5-F catheter   
is left wedged in the thrombus for overnight local thrombolysis infusion.   
Thrombolysis is allowed to proceed only if there are no contraindications   
such as recent variceal bleeding or multiple errant punctures made during   
the PV access (19). Recanalization of chronic PV occlusion is difficult and   
should be performed as an elective procedure by an experienced   
interventional radiologist. In this procedure, we use transjugular access as   
a primary approach and transhepatic or trans-splenic as auxiliary accesses   
if transjugular approach fails. In some cases, combined approach is   
necessary as the initial one (20, 21). The crucial step is crossing the   
occluded segment of the vein by hydrophilic guidewire. Balloon angioplasty   
is performed with a 4-5-mm balloon catheter. Portogram should follow   
immediately after dilatation to exclude extravasation. Recanalized segment   
is definitively dilated with a bare stent, including intrahepatic channel.   
Usually two overlapped stents are required to cover the whole tract.   
Embolization of portosystemic collaterals can facilitate blood flow through   
the shunt. Stent implantation should be performed always with respect to   
future liver transplant (22-24). Besides complications of TIPS or   
transhepatic access, there is a higher risk of intraperitoneal bleeding in   
the recanalization of chronic PV occlusion. This increased risk is because   
of more complex procedures lasting usually twice as long as regular TIPS.   
Acute rethrombosis of relatively long shunt can occur early. This can be   
facilitated by low flow through the shunt and possible hypercoagulation   
syndrome presented in patients with myeloproliferative disease. These   
patients require strict anticoagulation, and the longterm patency of their   
shunts is always worse than that in patients with regular liver cirrhosis.   
Technical success rate in acute PV thrombosis does not differ from the usual   
TIPS. Good long-term patency in patients with thrombophilia has to be   
maintained by anticoagulation therapy, and more frequent ultrasonographic   
controls are required to reveal asymptomatic stenosis of the shunt. Use of   
dedicated ePTFE stent-grafts is recommended in these patients because these   
stent-grafts proved to be less thrombogenic than bare stents. Technical   
success rate of recanalization procedures performed for chronic PV occlusion   
varies among centers. It has been reported from 35% to 100%. Investigated   
series included 12-57 patients (13-15,17).  
  
RECORD 156  
Individualized care for portal hypertension: Not quite yet  
Kamath P.S. Mookerjee R.P.  
Journal of Hepatology (2015) 63:3 (543-545) Article Number: 5736. Date of   
Publication: 1 Sep 2015  
  
RECORD 157  
The incidence and risk factors of portal vein system thrombosis after   
splenectomy and pericardial devascularization  
Wu S. Wu Z. Zhang X. Wang R. Bai J.  
Turkish Journal of Gastroenterology (2015) 26:5 (423-428). Date of   
Publication: 1 Sep 2015  
  
Background/Aims: This study aimed to investigate the incidence and risk   
factors of portal vein system thrombosis (PVST) in patients with liver   
cirrhosis after splenectomy and pericardial devascularization. Materials and   
Methods: We retrospectively analyzed 71 patients who underwent splenectomy   
with pericardial devascularization for portal hypertension due to cirrhosis.   
Patients were categorized into Group A (n=23): early prophylactic   
anticoagulants therapy; Group B (n=29): late prophylactic anticoagulants   
therapy; and Group C (n=19): no anticoagulation therapy. Univariate and   
multivariate analyses of the risk factors of PVST were performed. The   
incidence of PVST and the effect of thrombolytic therapy were evaluated.   
Results: Multivariate analysis revealed a wider preoperative splenic vein   
diameter (≥8 mm), and lower preoperative platelet counts (<50∼109/L) were   
significantly correlated with PVST development. The incidence of PVST in   
Groups A, B, and C was 26.1% (6/23), 44.8% (13/29), and 52.6% (10/19),   
respectively (all p>0.05). The complete resolution rate of portal, superior   
mesenteric, and splenic vein thrombosis was 75%, 62.5%, and 23.8%,   
respectively. Conclusion: A wider preoperative splenic vein diameter and   
lower preoperative platelet counts are independent risk factors of PVST.   
Early anticoagulation therapy had a tendency towards a reduced incidence of   
PVST, but it was not statistically significant. The complete resolution rate   
of splenic vein thrombosis was lower than that of portal and superior   
mesenteric vein thrombosis.  
  
RECORD 158  
Thrombosis in the Neonatal Intensive Care Unit  
Saxonhouse M.A.  
Clinics in Perinatology (2015) 42:3 (651-673). Date of Publication: 1 Sep   
2015  
  
RECORD 159  
Supporting the use of a coagulometric method for rivaroxaban control: A   
hypothesis-generating study to define the safety cut-offs  
Altman R. Gonzalez C.D.  
Thrombosis Journal (2015) 13:1 Article Number: 26. Date of Publication: 6   
Aug 2015  
  
Aims: Although quantitative anti-FXa assays can be used to measure   
rivaroxaban plasma levels, they are not widely performed or available. We   
aimed to tentatively determine the cut-off for thromboembolism and bleeding   
prevention based on the clotting effect of non-rivaroxaban   
conjugate-activated FX plasma levels in patients with rivaroxaban using a   
coagulometric method. Methods and results: Rivaroxaban was added in vitro to   
normal plasma at a range of 0 to 241 μg/L to cover expected peak and trough   
levels. Rivaroxaban chromogenic (μg/L) and RVV-confirm as a ratio were   
determined. Patient plasma samples were assayed with the RVV-confirm   
reagent. The appropriate rivaroxaban plasma concentration to inhibit   
clotting mechanisms was based on the remaining FXa in plasma, which was   
expressed as the ratio of patients/normal, R-C. There is a high correlation   
between R-C in vitro and spiked normal plasma rivaroxaban concentration   
(R-Square 0.910, linear equation; 0.971 quadratic equation, p < 0.0001 for   
both) but not with plasma rivaroxaban chromogenic assays. We propose a   
cut-off R-C value of 1.65 and 4.5 for safety. Based on the proposed   
therapeutic range, in 158 assays performed in 58 patients, 6.3 % assays were   
above the level of bleeding tendency at the peak (R-C 5.39 ± 1.01, median   
5.13) and 42 % assays were below the prevention cut-off at the trough (R-C   
1.31 ± 0.18, median 1.35). Conclusions: RVVconfirm® is fast and sensitive to   
measure the effect of rivaroxaban. Clinical studies are needed to establish   
whether this cut-off is useful for identifying patients at increased risk of   
hemorrhage or those who exhibit a low level of anticoagulation.  
  
RECORD 160  
A rare complication after gastric bypass: Thrombosis of a branch of the   
portal vein  
Elias B. Hanna P. Beche C. Coupez L. Saint-Eve P.  
Obesity Surgery (2015) 25:1 SUPPL. 1 (S342). Date of Publication: August   
2015  
  
Introduction: Thrombosis of the portal vein or one of its branches has been   
documented after laparoscopic procedures; however it is very rare after   
bariatric surgery. Several etiologies have been suggested (infection,   
inflammation, trauma, malignancy...) Objectives: Early diagnosis and   
treatment of this entity may avoid its progression. Methods: A 61-year-old   
diabetic woman, with previous history of deep venous thrombosis, underwent   
gastric bypass for morbid obesity. The operation was performed in a standard   
technique (Lonroth). Operative time was 120 min. The left lobe of the liver   
was retracted with a liver retractor held by an articulated arm.   
Postoperatively the patient received preventive dose of low molecular weight   
heparin twice daily. On the third postoperative day, the patient started to   
have abdominal pain, fever and leukocytosis (23000/mm(3)). An enhanced CT   
scan of the abdomen and pelvis showed thrombosis of left branch of the   
portal vein with signs of liver ischemia. There were no signs of anastomotic   
leak. Results: Therapeutic dose of low molecular weight heparin was started.   
The patient had progressive significant improvement over few days. CT scan 4   
weeks later showed complete resolution of the portal vein thrombosis and   
anticoagulation was stopped at 8 weeks postoperatively. Conclusion: Our   
patient presents multiple risk factors of venous thrombosis. However this   
isolated thrombosis of a branch of the left portal vein suggests a traumatic   
factor due to the liver retractor held by an articulated arm for more than   
an hour. Therefore, while operating patients with multiple risk factors, we   
recommend a gentle and intermittent retraction of the left lobe of the liver   
whenever possible.  
  
RECORD 161  
Extracorporeal Elimination of Piperacillin/Tazobactam during Molecular   
Adsorbent Recirculating System Therapy  
Personett H.A. Larson S.L. Frazee E.N. Nyberg S.L. El-Zoghby Z.M.  
Pharmacotherapy (2015) 35:8 (e136-e139). Date of Publication: 1 Aug 2015  
  
Use of the Molecular Adsorbent Recirculating System (MARS) as a liver   
support device continues to grow worldwide. Various components of the MARS   
circuit remove both protein-bound and water-soluble molecules. Little is   
known about the extent of the enhanced clearance mechanisms used in MARS   
therapy on drug elimination. Of particular interest to acute care   
practitioners is the impact of MARS on antibiotic clearance, as suboptimal   
concentrations of such drugs can negatively impact patient outcomes. The   
properties of piperacillin/tazobactam suggest that elimination may be   
enhanced in the setting of MARS therapy. We describe two cases in which this   
was studied. Piperacillin concentrations were determined at various points   
within the MARS circuit, and patient serum concentrations were reported   
throughout the dosing interval while receiving MARS therapy. Piperacillin   
concentrations in both cases were in excess of the desired goal minimum   
inhibitory concentrations for treatment of gram-negative infections. Use of   
an extended-infusion strategy of piperacillin/tazobactam 3.375 or 4.5 g   
given every 8 hours maintained desired serum levels throughout the dosing   
interval. To our knowledge, this is the second published report on the use   
of piperacillin/tazobactam during MARS therapy. These case reports reveal   
successful dosing strategies for patients requiring piperacillin/tazobactam   
while receiving MARS therapy, as well as quantify the influence of   
individual MARS elements on drug extraction.  
  
RECORD 162  
portal vein thrombosis after laparoscopic bariatric surgery it's a rare   
complication but should be considered. Description of three cases with   
literature review  
Al Qurashi T. Ghasoup A. Ahmad S. Widnly M.  
Obesity Surgery (2015) 25:1 SUPPL. 1 (S224). Date of Publication: August   
2015  
  
Background: portal vein thrombosis (PVT) refers to an obstruction in the   
trunk of the portal vein it's an uncommon complication after Laparoscopic   
Bariatric Surgery (LBS) However it is a potentially life-threatening   
condition reported after laparoscopic bariatric surgery. Clinical symptoms   
may be insidious, and progression can lead to intestinal infarction and   
portal hypertension. Main Outcome Measures: Systematic review of the   
literature on PVT after LBS and report three cases encountered at our   
institution. Patients and Methods: We reviewed the literature between   
January 1990, and January 2015, using the search terms portal vein   
thrombosis, mesenteric venous thrombosis, laparoscopic surgery and bariatric   
surgery. The inclusion criteria were documented PVT by imaging studies such   
as angiography, ultrasonography, computed tomography [CT], or magnetic   
resonance imaging (MRI) or surgery following LBS. We include three cases   
after laparoscopic sleeve gastrectomy from our institution. Results: One   
developed a chronic cavernoma with extension of the thrombus to the superior   
mesenteric vein and splenic vein, the other two cases recovered using   
anticoagulation therapy. Conclusions: PVT is a rare complication after LBS,   
however Laparoscopic surgeons should be aware of the risk of PVT, and it   
should be suspected in cases with an atypical outcome after LBS. Once PVT is   
diagnosed, prompt anticoagulation therapy may resolve the thrombotic event.  
  
RECORD 163  
Optimal management of portal vein thrombosis in patients with liver   
cirrhosis: A review  
Huard G. Bissonnette J. Bilodeau M.  
Current Hepatitis Reports (2015) 14:3 (203-211). Date of Publication: 22 Jul   
2015  
  
portal vein thrombosis (PVT) is a fairly common complication of cirrhosis,   
especially in patients with advanced liver disease and reduced portal vein   
flow velocity. Prospective studies have shown that its occurrence parallels   
rather than causes the progression of liver disease and that spontaneous   
regression is a frequent finding. PVT occurrence is associated with an   
increased mortality after liver transplantation. Treatment options include   
anticoagulation and insertion of a transjugular intrahepatic portosystemic   
shunt. anticoagulation with vitamin K antagonists or low-molecular-weight   
heparins achieves partial or complete portal vein recanalization in most   
patients. anticoagulation does not seem to increase the bleeding risk when   
proper prophylaxis of variceal bleeding is applied. Monitoring of   
anticoagulation difficulties exist, inherent to the coagulopathy of chronic   
liver disease. Placement of a transjugular intrahepat i c portosystemic   
shunt is an alternative in selected patients. Trials are underway to   
evaluate the potential benefit of PVT prophylaxis with low-molecular-weight   
heparins.  
  
RECORD 164  
Efficacy and safety of anticoagulation therapy with different doses of   
enoxaparin for portal vein thrombosis in cirrhotic patients with hepatitis B  
Cui S.-B. Shu R.-H. Yan S.-P. Wu H. Chen Y. Wang L. Zhu Q.  
European Journal of Gastroenterology and Hepatology (2015) 27:8 (914-919).   
Date of Publication: 11 Jul 2015  
  
Background Patients with cirrhosis have a high incidence of portal vein   
thrombosis (PVT), and optimal management of PVT in cirrhotic patients   
remains unclear. Currently, there is no paper on optimal doses of enoxaparin   
for the management of PVT with cirrhosis. Aims To evaluate the efficacy and   
safety of anticoagulation therapy with different doses of enoxaparin for PVT   
in cirrhotic patients with hepatitis B. Materials and methods Sixty-five   
patients with hepatitis B-related cirrhosis and acute PVT were treated by   
different doses of enoxaparin. All the patients were assigned randomly to   
two groups: one group received enoxaparin 1 mg/kg subcutaneously every 12 h   
and the other group received enoxaparin 1.5 mg/kg subcutaneously every 24 h.   
Clinical, biochemical evaluation, Doppler ultrasound, and contrast-enhanced   
computed tomography were performed during the anticoagulation treatment.   
Results Of the 65 patients, 51 patients (78.5%) achieved complete/partial   
recanalization of PVT after 6 months of anticoagulation therapy. Child-Pugh   
scores were lower in the 51 patients who achieved complete/partial   
recanalization than those of the 14 nonresponders (P<0.01). No patients   
showed variceal bleeding during anticoagulation therapy in the two groups.   
The rates of nonvariceal bleeding with the use of 1.5 mg/kg every 24 h   
(23.5%) were higher than those with the use of 1 mg/kg every 12 h (6.4%).   
Conclusion anticoagulation therapy with different doses of enoxaparin for   
PVT in hepatitis B patients with cirrhosis is efficient and safe, and 1   
mg/kg enoxaparin subcutaneously every 12 h is a better anticoagulation   
regimen in the treatment of PVT in cirrhotic patients.  
  
RECORD 165  
5 years' experience in a pediatric liver transplant program in Chile  
Pattillo J.-C. Guerra J.-F. Jarufe N. Gana J.-C. Soriano H. Concha M.   
González A. Castillo A. Carrasco J.-A. Tobar A. Dellepiane P. Martínez J.  
Transplantation (2015) 99:7 SUPPL. 1 (244). Date of Publication: July 2015  
  
Introduction: Pediatric liver transplant is the treatment of choice in   
several end stage liver diseases in children, with reported one-year   
survival rates of 90%. We started a new pediatric liver transplant program   
in 2009. Our aim is to communicate the results of the program during the   
first 5 years. Methods: Retrospective review of clinical database of   
pediatric liver transplants (LT). Results: Since May 2009, 18 LT in 16   
patients were performed, 9 boys and 7 girls. The average age was 2y 6m old   
(from 6m to 12y), with an average weight of 12.8±8 kg (from 5.5kg to 38kg).   
Median follow up was 43.8 months. Indications for LT were: Biliary atresia   
(BA) (8), acute liver failure (4), hepatoblastoma (2), Alagille's syndrome   
(1) and OTC deficit (1). Over this period we performed 10 Living donor LT,   
six deceased donor LT and two Split LT. The hepatic artery reconstruction   
was performed under microscope in 11 patients (9 living donors and 2   
splits). Immunosupresion regimen included steroids and tacrolimus. Three   
patients died during follow up: two patients with BA died in the early post   
op period, one due to portal vein thrombosis and another due to hemorrhage   
secondary to graft outflow obstruction in a large for size graft; the third   
patient died 9 months after LT due to doxorubicin related heart failure. The   
one year survival rate of the program was 81,6%. Other complications   
included: another patient with portal vein thrombosis, successfully   
corrected with surgery, two patients with significant portal vein stenosis   
treated with surgery and percutaneous dilation, one patient with   
suprahepatic vein thrombosis treated with anticoagulation. We had no hepatic   
artery thrombosis in this series. Four patients developed biliary   
complications, 3 of them required surgery. Two patients had mild biopsy   
proven rejection reverted with steroids, and one patient has chronic   
rejection. All of our patients have returned to a normal life after LT.   
Conclusions: The creation of a pediatric LT program is a challenging   
opportunity for a transplant center. Our perioperative, short and long-term   
results are comparable to those reported in the literature in the field.  
  
RECORD 166  
Early prophylactic anticoagulation via transjugular intrahepatic route for   
portal vein thrombosis after splenectomy in cirrhotic portal hypertension  
Yang S. He C. Fan X. Ding W. Wu X. Li J.  
Journal of Vascular and Interventional Radiology (2015) 26:7 (1009-1017).   
Date of Publication: 1 Jul 2015  
  
Purpose To evaluate early transcatheter anticoagulation via the transjugular   
intrahepatic route to prevent portal vein thrombosis (PVT) after splenectomy   
in cirrhotic patients with portal hypertension. Materials and Methods This   
retrospective study included 98 cirrhotic patients with portal hypertension   
who underwent open splenectomy (48 men and 50 women; age, 45.4 y ± 13.6).   
Systemic anticoagulation was given to 52 patients in group I, and   
transcatheter anticoagulation was performed in 46 patients in group II.   
Results The technical success rate of catheterization by the transjugular   
intrahepatic route was 93.5% in group II. The 30-day (6.52% vs 23.1%, P   
<.05) and 6-month (8.70% vs 26.9%, P <.05) incidences of PVT were   
significantly lower in group II than in group I. The postoperative bleeding   
rate was 6.52% in group II and 25% in group I (P <.05). There was no   
significant difference between groups in 30-day (5.77% vs 2.17%) and 6-month   
(1.92% vs 6.52%) mortality. After splenectomy, the portal trunk vessel   
diameter was 16.0 mm ± 3.5 in group I and 14.5 mm ± 2.5 in group II (P   
<.05). The portal flow velocity was 25.9 cm/s ± 7.1 in group I and 28.2 cm/s   
± 5.3 in group II (P >.05). During the first week after splenectomy, notable   
hypercoagulability was detected within the portal vein compared with   
peripheral blood. Decreased portal flow velocity was considered an   
independent risk factor for PVT by univariate and multivariate analysis.   
Conclusions Transcatheter anticoagulation via the transjugular intrahepatic   
route can decrease the incidence of PVT and postoperative bleeding after   
open splenectomy in cirrhotic patients with portal hypertension.  
  
RECORD 167  
Hypercoagulability in cirrhotic patients with hepatocellular carcinoma (HCC)   
and portal vein thrombosis (PVT)  
Zanetto A. Ferrarese A. Rodriguez-Kastro K.-I. Fadin M. Gavasso S. Radu C.   
Zerbinati P. Vitale A. Cillo U. Farinati F. Russo F.P. Germani G. Simioni P.   
Burra P. Senzolo M.  
Transplantation (2015) 99:7 SUPPL. 1 (228). Date of Publication: July 2015  
  
Background and aim: studies which explores the hypercoagulable induced by   
HCC in cirrhosis are lacking. The aim of the present study was to evaluate   
the thrombophilic role of HCC as risk factor for development of PVT.   
Methods: cirrhotic patients with and without HCC were prospectively enrolled   
in the study and underwent: thromboelastometry (ROTEM), platelet count,   
determination of prothrombin time and of levels of pro and anticoagulation   
factors. During follow-up, PVT onset in both patients with and without HCC   
was recorded. Results: 76 cirrhotics, 41 with HCC, were included. Volume of   
active HCC was >5 cm(2) in 18 patients. Levels of pro and anticoagulation   
factors were similar between patients with and without HCC, but fibrinogen   
was increased in HCC patients with active volume [>5cm(2) HCC compared to   
those with <5cm(2)HCC bulk (348,72mg/dL±124,06mg/dL vs 237,64mg/   
dL±99,18mg/dL) and to cirrhotics without HCC (260,57mg/dL±126,07mg/ dL)   
(p=0,006). Platelet count was significantly increased in HCC compared to   
non-HCC patients, and this was especially true in Child A group. ROTEM   
demonstrated a significantly lower clotting time and maximum clot formation   
in HCC patients compared to controls and non-HCC cirrhotics, especially in   
Child A group. The incidence of PVT was 24,4% (10/41) and 11.4% (4/35) in   
HCC and non-HCC patients, respectively (OR: 2,5; 95%, CI 0,70-8,83). In the   
HCC group, 5/10 portal vein thromboses occurred in patients in Child Class   
A. Fibrinogen test of ROTEM, MCF and AUC were statistically greater in HCC   
patients who later developed PVT. Conclusions: cirrhotics with HCC   
demonstrate a prothrombotic hemostatic balance resulting in an increased   
risk of PVT development. This prothrombotic state seems to be detectable by   
ROTEM and thus possibly suggest those who could benefit from   
thromboprophylaxis.  
  
RECORD 168  
Successful pregnancy on basiliximab in a liver transplant (LT) recipient   
with recurrent acute rejection and difficult-to-control recurrent autoimmune   
hepatitis (AIH)  
Te H.S. Renz J. Aronsohn A. Pote L. Dasgupta K.A. Millis J.M.  
Transplantation (2015) 99:7 SUPPL. 1 (276-277). Date of Publication: July   
2015  
  
AIH recurrence following LT is typically managed with azathioprine (AZA) or   
mycophenolic acid (MPA). However, MPA is a teratogen and cannot be used   
during pregnancy. AIM: This is a case report of a LT recipient who had a   
successful pregnancy on basiliximab, cyclosporine (CSA), azathioprine (AZA),   
and prednisone. CASE: A 29 year old Hispanic female received a living-donor   
LT from her sister for AIH related-cirrhosis. Initial immunosuppression (IS)   
were tacrolimus (TC) and steroids, but elevation in her liver enzymes   
prompted the addition of MPA and prevented weaning of steroids. At month 6,   
she developed a portal vein thrombus and had a percutaneous thrombectomy.   
She was diagnosed with hypercoagulability secondary to antiphospholipid   
syndrome and was started on anticoagulation. She also developed posterior   
reversible encephalopathy syndrome that prompted a change from TC to CSA.   
While on the combination of CSA, MPA, and steroids, she had multiple   
episodes of biopsy-proven acute rejection at month 8, year 2 and year 4   
following LT, and she developed recurrent AIH at year 2. Her graft function   
finally stabilized on higher trough CSA levels of 200-250, MPA 720 mg bid,   
and prednisone 10 mg daily. Despite high risks to her own health, she   
expressed her desire to become pregnant. Stabilization of graft function for   
a full year after the last acute rejection episode was established, then she   
was switched from MPA to AZA 50 mg daily and basiliximab at 40 mg IV   
infusions monthly, with continuation of CSA and prednisone 10 mg daily.   
Stable graft function was confirmed for three months on this novel regimen   
before she was allowed to conceive. She became pregnant and successfully   
carried her pregnancy to full term on this regimen with stable graft   
function throughout, and delivered a healthy baby boy. Her post-partum   
course was complicated by a small subdural hematoma attributed to her   
anticoagulation, which resolved with no permanent neurologic sequelae. She   
has resumed her pre-pregnancy IS without any graft compromise. CONCLUSION:   
Monthly basiliximab infusions can be an acceptable alternative maintenance   
IS during pregnancy, particularly in patients whose graft function require   
potent IS agents that cannot be continued during pregnancy. This approach   
merits further study in a prospective manner in a larger population.  
  
RECORD 169  
Diffuse cholangiocarcinoma presenting with hepatic failure and extensive   
portal and mesenteric vein thrombosis  
Vakil A. Guru P. Reddy D.R. Iyer V.  
BMJ Case Reports (2015) 2015. Date of Publication: 29 Jun 2015  
  
A 64-year-old previously healthy man presented with a 4-week history of   
vague right upper quadrant abdominal pain. Imaging studies revealed   
extensive portal, splenic, superior and inferior mesenteric vein thrombosis   
with mosaic perfusion and wedge-shaped areas of liver perfusion   
abnormalities. An extensive thrombophilia workup including tests for factor   
V Leiden, prothrombin G20210A, lupus anticoagulant, paroxysmal nocturnal   
haemoglobinuria, protein C and S, homocysteine and antinuclear antibody   
titres were all negative. Other laboratory testing revealed an elevated   
alkaline phosphatase (340 IU/L). Surgical exploration and catheter-directed   
thrombolysis were not felt to be feasible given the extensive clot burden.   
He was started on anticoagulation therapy. Over the next 10 days, he   
required intensive care unit admission due to progressive hepatic   
encephalopathy and fulminant liver failure. He continued to decline and   
eventually died of multiorgan failure. Autopsy revealed extensive, diffuse   
intrahepatic cholangiocarcinoma that had almost entirely replaced his normal   
liver parenchyma.  
  
RECORD 170  
Splanchnic Vein Thrombosis  
Valla D.  
Seminars in Thrombosis and Hemostasis (2015) 41:5 (494-502) Article Number:   
02214. Date of Publication: 16 Jun 2015  
  
Splanchnic vein thrombosis includes thrombosis of the hepatic venous system   
(Budd-Chiari syndrome) and thrombosis of the portal venous system. Both   
conditions share uncommon prothrombotic disorders as causal factors, among   
which myeloproliferative neoplasms rank first. Budd-Chiari syndrome presents   
with acute or chronic, asymptomatic or severe liver disease. Diagnosis   
depends on noninvasive imaging of the obstructed hepatic venous outflow   
tract. A spontaneously fatal course can be prevented by a stepwise approach:   
(1) anticoagulation therapy, specific therapy for underlying disease, and   
medical or endoscopic management of liver-related complications, (2)   
angioplasty/stenting in a second step, and (3) eventually the insertion of   
transjugular intrahepatic stent shunt or liver transplantation. Recent   
portal vein thrombosis mostly jeopardizes the gut. Early anticoagulation   
prevents thrombus extension but is incompletely successful in achieving   
recanalization. Chronic portal vein thrombosis is complicated by bleeding   
related to portal hypertension, which can be prevented by usual   
pharmacological and endoscopic means. The prevention of recurrent thrombosis   
is achieved by anticoagulation therapy the impact of which on the risk of   
bleeding remains unclear. portal vein thrombosis in patients with cirrhosis   
is likely neither a direct consequence of nor a direct cause for liver   
disease progression. Therefore, the indications and effects of   
anticoagulation therapy for portal vein thrombosis in patients with   
cirrhosis remain uncertain.  
  
RECORD 171  
Risk Factors, Diagnosis, Management, and Outcome of Splanchnic Vein   
Thrombosis: A Retrospective Analysis  
Derman B.A. Kwaan H.C.  
Seminars in Thrombosis and Hemostasis (2015) 41:5 (503-513) Article Number:   
02191. Date of Publication: 16 Jun 2015  
  
Objectives This study aims to determine the risk factors, diagnostic methods   
employed, treatment modalities, and outcome in patients with splanchnic vein   
thrombosis (SVT). Methods A retrospective chart review of patients, age 18   
to 90 years, diagnosed with SVT at a single institution from January 1, 2010   
to November 10, 2012. They were grouped as portal vein thrombosis   
(PVT)-including those combined with splenic vein thrombosis (SPVT) or   
mesenteric vein thrombosis (MVT)--and Budd-Chiari syndrome (BCS). Results   
Overall 246 SVT patients were identified, including 225 PVT and 21 BCS. Risk   
factors were liver disease, upper abdominal (regional) cancer and surgery,   
pancreatitis, and hereditary thrombophilia. The most common symptom was   
abdominal pain and most patients had abnormal liver function. Among those   
tested, the JAK2 V617F mutation was present in only 20% of the patients with   
PVT and 14% of the patients with BCS. Most patients were diagnosed by   
computed tomography. Anticoagulants were given to 30% of the patients with   
PVT and to 60% of the patients with BCS, with recurrence of SVT in 15% of   
the patients with PVT and 24% of the patients with BCS, regardless of   
anticoagulation. Conclusion As compared with published literature on SVT, we   
found a higher incidence of regional cancer and surgery and a lower   
incidence of the JAK2 V617F mutation.  
  
RECORD 172  
Anticoagulant Therapy in Patients with cirrhosis  
Intagliata N.M. Northup P.G.  
Seminars in Thrombosis and Hemostasis (2015) 41:5 (514-519). Date of   
Publication: 6 Jun 2015  
  
Recent studies have greatly expanded our understanding of the coagulopathy   
of cirrhosis. It is clear that cirrhosis patients are at a risk of both   
bleeding and thrombosis. While prediction of these events remains   
challenging, cirrhosis patients are not protected from the development of   
venous and arterial thrombosis. In fact, studies show that   
hypercoagulability may promote hepatic decompensation and development of   
fibrosis. anticoagulation for thrombosis is now becoming a common prospect   
in many clinical situations. Our understanding of the efficacy and safety of   
commonly used therapeutics is only beginning to emerge and the risks and   
benefits remain unclear in this unique population. In this review, we   
discuss the role of anticoagulation in the treatment and prevention   
peripheral and splanchnic thrombosis in patients with cirrhosis, as well as   
examine the potential role of anticoagulants in altering the progression of   
chronic liver disease.  
  
RECORD 173  
Pharmacist supported anticoagulation management clinic-improving patient   
care in an emerging country  
Najmi N. Moiz B. Khan S.  
Journal of Thrombosis and Haemostasis (2015) 13 SUPPL. 2 (401). Date of   
Publication: June 2015  
  
Background: Joint Commission International Accreditation (JCIA) outlined the   
National Patient Safety Goal related to anticoagulation therapy (03.05.01).   
We observed 10% of drug overdosage was due to warfarin toxicity. Aims: The   
objective of this study is to observe improvement in patient care during   
anticoagulation therapy. Methods: We studied our patients enroled in   
Antocoagulation Clinic from March 2013 to December 2014. Quality indicators   
were monitered as bleeding and thrombotic events. Food consultation and   
patient education was done to 100% patients. 15 patients migrating to other   
cities were communicated through e-mails and telephonic messages for INR   
monitoring and dose adjustments. Results: A total of 105 patients (49 males/   
56 females) were registered in anticoagulation Clinic. Diagnosis included   
deep venous thrombosis (n = 23), pulmonary embolism (n = 18), portal vein   
thrombosis (n = 7), superior mesenteric vein thrombosis (n = 5), cerebral   
venous thrombosis (n = 5), others (n = 47). During the study of 105 patients   
2 events were reported. These events included incidents of minor (n = 1) and   
major bleeding (n = 0) and thrombotic event (n = 1) despite anticoagulation.   
Conclusion: The results showed improved patient care for bleeding and   
thrombotic events.  
  
RECORD 174  
Under australian sand  
Pizzini A.M. Galimberti D. Muratore F. Casali A. Jordana Sanchez M.D.M.   
Salvarani C. Iori I.  
Italian Journal of Medicine (2015) 9 SUPPL. 2 (86-87). Date of Publication:   
2015  
  
Introduction: Abernethy malformation (AM) is a rare congenital disease with   
portal blood diversion from the liver: it includes congenital absence of the   
portal vein, portosystemic shunt, liver nodes, congenital heart disorders   
such as atrial/ventricular septal defect and patent doctus arteriosus. In   
type 1 AM blood is diverted from portal system to inferior vena cava (IVC)   
and intrahepatic portal flood is absent. In type 2 AM there is a partial   
diversion and the portal system is twisting. Etiology is unknown   
(intrauterine infection). Venous stasis, even without other prothrombotic   
factors, may induce portal vein thrombosis and severe bleeding   
complications. Case report: A 33 year old man was admitted for recurrent   
oral aphta. He had a history of intrauterine toxoplasmosis. In 2003 he had   
had an incidental echographic diagnosis of spleno-portomesenteric vein   
dilatation with perigastric porto-caval shunts (type 2 AM); it was   
associated with hepatic focal nodal hyperplasia but not with portal   
hypertension. In 2012, back from a tour in Australia, he complained of a   
severe epigastric pain and hematemesis: blood transfusions were needed.   
Gastroscopic examination showed F3 varices that were ligated. At CT a large   
portal cavernoma was found with splenic and mesenteric vein thrombosis. He   
was treated with enoxaparin with gradual recovery. Trombophylia and Bechet   
syndrome were excluded. Conclusions: AM causes venous stasis with possible   
severe throm-botic complications. Portal cavernoma could benefit from   
chronic anticoagulation (INR 2-2,5), with haemoglobin and varices   
monitoring.  
  
RECORD 175  
Antithrombotic treatment and outcomes of cirrhotic patients with splanchnic   
vein thrombosis: A sub-study from the ISTH registry  
Riva N. Ageno W. Senzolo M. Schulman S. Beyer-Westendorf J. Duce R. Santoro   
R. Kamphuisen P. Oh D. Becattini C. Rodriguez K. Barillari G. Passamonti   
S.M. Guardascione M.A. Vidili G. Vaccarino A. Dentali F.  
Journal of Thrombosis and Haemostasis (2015) 13 SUPPL. 2 (69-70). Date of   
Publication: June 2015  
  
Background: Cirrhotic patients have often been excluded from studies   
addressing the anticoagulant treatment of splanchnic vein thrombosis (SVT),   
although liver cirrhosis is associated with an increased risk of SVT through   
hypercoagulability. Aims: To assess the outcomes of cirrhotic patients from   
an unselected cohort of SVT patients. Methods: International prospective   
registry of consecutive SVT patients, enrolled from 2008 to 2012.   
Therapeutic strategies and outcomes were analysed separately for cirrhotic   
patients. A Central Adjudication Committee reviewed and classified clinical   
outcomes as vascular events (venous or arterial thrombosis) and major   
bleeding (MB; ISTH definition plus the need for hospitalization). Results: A   
toal of 167 (28%) of 604 patients had liver cirrhosis (median age 59 years,   
70.7% males, 79.6% portal vein thrombosis, 49.4% inci- dentally detected   
SVT). Sixty-six patients (39.5%) received no anticoagulation; 62 received   
parenteral anticoagulants alone (median duration 6 months, IQR 3-15) and 39   
were started on vitamin K antagonists (median 10 months, IQR 4-24). Median   
follow-up duration was 2 years (IQR 0.5-2); 5 patients (3.0%) were lost to   
follow-up. The overall incidence of recurrent thrombotic events was 11.3/100   
patient-years (pt-y) (95% CI 7.7-16.8) and the incidence of MB was 10.0/100   
pt-y (95% CI 6.6-15.1). The incidence of these two outcomes in never-treated   
cirrhotic patients was 14.1/100 pt-y and 11.3/100 pt-y, respectively. In   
multivariate analysis, anticoagulant treatment was associated with lower   
rates of both vascular events (HR 0.86, 95% CI 0.77-0.96) and MB (HR 0.83,   
95% CI 0.69-0.99). Conclusion: Our real-life data suggest that more than a   
half of cirrhotic patients with SVT receive anticoagulant treatment. The   
incidence of thrombotic and bleeding complications was not negligible in   
this subgroup of patients. In selected cirrhotic patients, the anticoagulant   
treatment, usually administered at adjusted doses according to the   
individual risk of bleeding, appeared to be beneficial.  
  
RECORD 176  
Portal vein reconstruction in adult living donor liver transplantation for   
patients with portal vein thrombosis in single center experience  
Mori A. Iida T. Iwasaki J. Ogawa K. Fujimoto Y. Uemura T. Hatano E. Okajima   
H. Kaido T. Uemoto S.  
Journal of Hepato-Biliary-Pancreatic Sciences (2015) 22:6 (467-474). Date of   
Publication: 1 Jun 2015  
  
Background Liver transplantation (LT) used to be contraindicated in patients   
with portal vein thrombosis (PVT). In comparison to deceased donor LT,   
living donor LT (LDLT) still presents additional difficulties in determining   
appropriate vein grafts and overcoming small-for-size syndrome. Here, we   
introduce our LDLT strategies and assess their outcomes in adult patients   
with pre-existing PVT. Methods We performed 282 consecutive adult LDLTs   
between April 2006 and December 2011. Forty-eight patients (17%) had   
pre-existing PVT (grade I; 15, II; 20, III; 12, IV; 1). Results Our   
preferred treatments for PVT were thrombectomies/thromboendovenectomies in   
30 patients, replaced grafts in seven, jump grafts in seven, renoportal   
anastomosis in one and no surgical intervention owing to minimal thrombosis   
in three. Post-transplant portal vein complications occurred in eight of 48   
(17%) cases, which were treated by surgery, anticoagulation therapy, and/or   
interventional radiology. Post-transplant survival rates of patients with   
preexisting PVT at 1 year and 5 years were comparable to a PVT-free cohort   
(1 year; 81% vs. 77%, 5 years; 81% vs. 73%). Conclusions The excellent   
survival rates in patients with PVT who underwent LDLT could be attributed   
to our strategies, which included surgical techniques and timely treatment   
of postoperative complications.  
  
RECORD 177  
portal vein thrombosis in patients with cirrhosis: Outcome of   
anticoagulation  
Cheruvathur P. Peter G. Mashhood V. Vinayakumar K. Sunil P.  
Journal of Clinical and Experimental Hepatology (2015) 5 Supplement 2   
(S34-S35). Date of Publication: 1 Jun 2015  
  
Introduction: The development of portal vein thrombosis (PVT) in patients   
with end stage liver disease (ESLD) is a multifactorial process, primarily   
from reduction of portal flow, damage to the vessel wall and   
hypercoagulability. Development of PVT is often accompanied by increased   
rate of morbidity and mortality. There is limited data regarding the role of   
anticoagulation therapy in patients with PVT and liver cirrhosis. Aim: To   
assess the outcome of anticoagulation in patients with liver cirrhosis and   
PVT. Methods: This was a retrospective observational study conducted in the   
Department of Medical Gastroenterology, Govt. Medical College Trivandrum.   
All patients with cirrhosis and PVT who received anticoagulant treatment,   
admitted in our department from January 2010 to December 2014 were included   
in the study. Data was collected from electronic medical records using a   
self developed proforma. Patients were excluded if they had incomplete   
medical records or had not completed 6 months of anticoagulation.   
Statistical analysis was done using SPSS-17. Results: There was a total of   
46 cases of cirrhosis with PVT who underwent anticoagulation during the   
study period. anticoagulation was initiated using Enoxaparin or Fondaparinux   
and continued for a period of 6 months with oral warfarin. Enoxaparin and   
Fondaparinux was used in 29 and 17 cases, respectively. PVT resolved in 18   
(39%) patients, no change in 8 (18%) patients, and 20 (43%) patients showed   
partial resolution of thrombus on ultrasound Doppler evaluation.   
Complications noted were 4 cases of GI bleed (melena), 1 case of bleeding PV   
and 3 case of skin bleed. All bleeding episodes were controlled with   
conservative management. No deaths were reported as a result of   
anticoagulation treatment during the study period. Conclusions:   
anticoagulation using warfarin in patients with cirrhosis and PVT is   
relatively safe and effective.  
  
RECORD 178  
Idiopathic non-cirrhotic portal hypertension: A review  
Schouten J.N.L. Verheij J. Seijo S.  
Orphanet Journal of Rare Diseases (2015) 10:1 Article Number: 67. Date of   
Publication: 30 May 2015  
  
Idiopathic non-cirrhotic portal hypertension (INCPH) is a rare disease   
characterized of intrahepatic portal hypertension in the absence of   
cirrhosis or other causes of liver disease and splanchnic venous thrombosis.   
The etiology of INCPH can be classified in five categories: 1) immunological   
disorders (i.e. association with common variable immunodeficiency syndrome,   
connective tissue diseases, Crohn's disease, etc.), 2) chronic infections,   
3) exposure to medications or toxins (e.g. azathioprine, 6-thioguanine,   
arsenic), 4) genetic predisposition (i.e. familial aggregation and   
association with Adams-Oliver syndrome and Turner disease) and 5)   
prothrombotic conditions (e.g. inherited thrombophilias myeloproliferative   
neoplasm antiphospholipid syndrome). Roughly, INCPH diagnosis is based on   
clinical criteria and the formal exclusion of any other causes of portal   
hypertension. A formal diagnosis is based on the following criteria: 1)   
presence of unequivocal signs of portal hypertension, 2) absence of   
cirrhosis, advanced fibrosis or other causes of chronic liver diseases, and   
3) absence of thrombosis of the hepatic veins or of the portal vein at   
imaging. Patients with INCPH usually present with signs or symptoms of   
portal hypertension such as gastro-esophageal varices, variceal bleeding or   
splenomegaly. Ascites and/or liver failure can occur in the context of   
precipitating factors. The development of portal vein thrombosis is common.   
Survival is manly limited by concomitant disorders. Currently, treatment of   
INCPH relies on the prevention of complications related to portal   
hypertension, following current guidelines of cirrhotic portal hypertension.   
No treatment has been studied aimed to modify the natural history of the   
disease. anticoagulation therapy can be considered in patients who develop   
portal vein thrombosis.  
  
RECORD 179  
Hepatic portal venous gas and portal venous thrombosis following colonoscopy   
in a patient with terminal ileal Crohn's disease  
Ma A.S.C. Ewing I. Murray C.D. Hamilton M.I.  
BMJ Case Reports (2015) 2015 Article Number: 206854. Date of Publication: 4   
May 2015  
  
A 27-year-old man developed extensive hepatic portal venous gas (HPVG)   
shortly after staging colonoscopy for active, ulcerating, terminal ileal   
Crohn's disease. Nonoperative management was instigated with broadspectrum   
antibiotics and thromboprophylaxis. Radiology at 72 h demonstrated   
resolution of HPVG but revealed fresh non-occlusive left portal vein   
thrombus. anticoagulation with warfarin was continued for 1 year, during   
which the thrombus initially progressed and then organised with   
recanalisation of the portal vein. There were no long-term clinical   
consequences. HPVG has previously been documented as a rare complication of   
inflammatory bowel disease and endoscopic intervention. We hypothesise that   
the barotrauma sustained during endoscopy, in association with active   
ulceration and mucosal friability, predisposes to the influx of gas and   
bacteria into the portal system. We describe successful non-operative   
management of HPVG in this setting and draw attention to an additional   
complication of portal venous thrombosis, highlighting the importance of   
thromboprophylaxis and serial radiological examination.  
  
RECORD 180  
Pre-transplant portal vein recanalization-transjugular intrahepatic   
portosystemic shunt in patients with chronic portal vein thrombosis  
Thornburg B. Desai K. Baker T. Fryer J. Abecassis M. Caicedo J. Kulik L.   
Salem R.  
American Journal of Transplantation (2015) 15 SUPPL. 3. Date of Publication:   
May 2015  
  
Background: Chronic, occlusive portal vein thrombosis (PVT) associated with   
cirrhosis represents a relative contraindication to liver transplantation   
(LT) in some centers. From a surgical perspective, portal vein   
recanalization-transjugular intrahepatic portosystemic shunt (PVR-TIPS) may   
facilitate LT and enhance transplant eligibility. Our objective was to   
evaluate the effect of PVR-TIPS on liver function, transplant eligibility   
and long-term outcomes following liver transplantation (LT). Methods: 44   
patients with chronic main PVT were identified during our institutional LT   
selection committee and, following joint imaging review by transplant   
surgery/radiology, were referred to interventional radiology for PVR-TIPS to   
enhance transplant eligibility. Following PVR-TIPS, patients were followed   
by hepatology/transplant until LT, and in posttransplant clinic. Baseline   
characteristics were recorded, TIPS venography and serial ultrasound/MRI   
were used subsequently to document PV patency. Results: The main portal vein   
(MPV) was completely thrombosed in 17/44 (39%) patients; near complete   
(>95%) occlusion was noted in 27/44 (61%). Direct transhepatic and   
trans-splenic punctures were required in 11/43 (26%) and 3/43 cases (7%),   
respectively. Technical success was 43/44 (98%). At PVR-TIPS completion,   
persistence of MPV thrombus was noted in 33/43 (77%). One-month TIPS   
venography demonstrated complete resolution of MPV thrombosis in 22/29 (76%)   
without anticoagulation. 36 patients were listed for transplantation; 18   
(50%) have been transplanted. 89% MPV patency rate and 82% survival was   
achieved at 5 years. Conclusion: PVR-TIPS may be considered for patients   
with PVT being considered for LT. The high rate of MPV patency post-TIPS   
placement suggests flow re-establishment as the dominant mechanism of   
thrombus resolution.  
  
RECORD 181  
Prevention and management of vascular complications in pediatric liver   
transplantation: A global peri-operative strategy  
Grimaldi C. Pietrobattista A. Chiusolo F. Di Francesco F. Basso M. Rollo M.   
Monti L. Candusso M. Picardo S. Torre G. De Ville De Goyet J.  
Pediatric Transplantation (2015) 19 SUPPL. 1 (115). Date of Publication: May   
2015  
  
Purpose: Vascular complications are a well-known cause of graft loss and   
eventually of patient death after liver transplantation. We evaluate the   
effect of a perioperative strategy of prevention and early diagnosis on the   
incidence of vascular complications and outcome. Methods: from December 2008   
to July 2014, 107 liver transplantations were performed in 106 children. The   
strategy included: -high flow vascular reconstructions, use of microsurgical   
technique, repeated intraoperative Doppler ultrasound (US) -regular   
post-operative Doppler US follow up (peak velocities and arterial resistance   
index) -anticoagulation and anti-platelet prophylaxis as per protocol -high   
level of suspicion of thrombosis with preemptive and immediate management   
Results: Intraoperative period: immediate good flows were observed at   
Doppler US in 95 cases (88.7%). In 9 cases the flow was absent either at   
declamping or after abdomen closure: revision with graft repositioning, redo   
of anastomosis, prosthetic abdominal closure allowed to correct the problem.   
In 3 cases the flow was non-optimal in hepatic artery/portal vein (1/2)   
despite revision, however it improved after few days under anticoagulation.   
Early postoperative complications: 5 patients with signs of splenic steal   
syndrome were managed either by interventional radiology (N=3) or surgery   
(N=2). One early hepatic artery thrombosis underwent to successful surgical   
redo. Late complications (> 30 days): 2 late hepatic artery (HA) stenosis   
were treated by radiological balloon dilatation while 1 thrombosis was   
managed conservatively. Late portal vein stricture was diagnosed in 5   
patients: all underwent to successful trans-hepatic angioplasty (1 stent).   
Two patients with portal vein (PV) thrombosis were treated by meso-Rex   
bypass. There were no hepatic vein or inferior vena cava complications in   
the series. Overall outcome: although 28 patients (25%) presented at some   
point with signs of vascular complications, 90% was successfully treated.   
Among 3 patients with vascular thrombosis (2 PV and 1 HA), 2 of them were   
cured by meso-Rex bypass. Overall Patient and Graft survival are 96% and 95   
% respectively, with no loss due to vascular complications (mean follow-up:   
32 months). Conclusion: perioperative aggressive strategy and protocol are   
effective for prevention or preemptive management of vascular complications.  
  
RECORD 182  
Big spleens and hypersplenism: Fix it or forget it?  
Boyer T.D. Habib S.  
Liver International (2015) 35:5 (1492-1498). Date of Publication: 1 May 2015  
  
Hypersplenism is a common manifestation of portal hypertension in the   
cirrhotic. More than half of cirrhotics will have low platelet counts, but   
neutropenia is much less common. Despite being common in the cirrhotic   
population, the presence of hypersplenism is of little clinical consequence.   
The presence of hypersplenism suggests more advanced liver disease and an   
increase in risk of complications, but there is no data showing that   
correcting the hypersplenism improves patient survival. In most series, the   
most common indications for treating the hypersplenism is to increase   
platelet and white blood cell counts to allow for use of drugs that suppress   
the bone marrow such as interferon alpha and chemotherapeutic agents. There   
are several approaches used to treat hypersplenism. Portosystemic shunts are   
of questionable benefit. Splenectomy, either open or laparoscopically, is   
the most effective but is associated with a significant risk of portal vein   
thrombosis. Partial splenic artery embolization and radiofrequency ablation   
are effective methods for treating hypersplenism, but counts tend to fall   
back to baseline long-term. Pharmacological agents are also effective in   
increasing platelet counts. Development of direct acting antivirals against   
hepatitis C will eliminate the most common indication for treatment. We lack   
controlled trials designed to determine if treating the hypersplenism has   
benefits other than raising the platelet and white blood cell counts. In the   
absence of such studies, hypersplenism in most patients should be considered   
a laboratory abnormality and not treated, in other words forget it.  
  
RECORD 183  
The role of anticoagulation for portal vein thrombosis prior to orthotopic   
liver transplantation  
Bozanich N.K. Ghabril M. Agrawal S. Lacerda M.A. Tector J. Fridell J.A.   
Mangus R.S. Kubal C.A. Kwo P.Y.  
Gastroenterology (2015) 148:4 SUPPL. 1 (S1040). Date of Publication: April   
2015  
  
portal vein thrombosis (PVT) is common complication in the setting of end   
stage liver disease. The presence of PVT in the setting of orthotopic liver   
transplantation (OLT) can be associated with the need for additional   
anastomoses and potentially reduced survival. The goal of anticoagulation is   
to achieve partial recanalization to allow end-to-end portal vein   
anastomosis. Our AIM was to determine the impact of anticoagulation for PVT   
when indicated on recanalization of the portal vein at the time of OLT and   
on post OLT outcomes. Methods : This is a single center retrospective study   
of all patients who underwent OLT who were previously diagnosed with PVT   
between March 2011 and July 2014. The study included all patients over age   
18 with PVT diagnosed by CT or MRI and who subsequently underwent OLT. Data   
abstracted included demographic data, anatomic extent of PVT, presence/type   
of anticoagulation, effect on PVT, complications of anticoagulation, and   
outcomes after transplant including use of jump graft and survival. PVT was   
classified as occlusive or non-occlusive involving portal vein with or   
without extension. The decision to anticoagulate was made by a   
multidisciplinary team at selection conference. Results: 43/333 (13%)   
patients were diagnosed with PVT before OLT by axial imaging. Median age was   
59 years (IQR= 52-63), 27/43 male, median BMI 28 (IQR= 25.7-33), median MELD   
score 20 (IQR= 17-25). PVT was diagnosed at median of 338 days prior to OLT.   
In 30/43 patients anticoagulation was initiated (27 warfarin, 3 enoxaparin)   
prior to OLT for median duration of 9 months (IQR 5- 17). The median time to   
demonstrated improvement or resolution of PVT was 5 months (IQR 3- 7.2).   
19/30(63%) of anticoagulated patients achieved partial to full PVT   
resolution at time of OLT compared to 8/14 patients (57%) in whom no   
anticoagulation was initiated. 3 patients with partial or full resolution of   
PVT had recurrent thrombosis post OLT. 3 patients required jump grafts due   
to thrombosis. In the entire PVT cohort, there were 5 deaths post OLT (2 no   
anticoagulation, 1 warfarin, 2 enoxaparin). 1 month survival (no   
anti-coagulation 86% ;warfarin 100%;enoxaparin 67%), 6 month survival (no   
anti-coagulation 86% ;warfarin 96%%;enoxaparin 33%)and 1 year survival (no   
anti-coagulation 85% ;warfarin 94%;enoxaparin 0%) were superior in the   
warfarin treated group compared to enoxaparin or no anticoagulation   
(p<0.05). Bleeding complications were rare with no difference noted between   
the anticoagulated and non-anticoagulated groups. Conclusion:   
anticoagulation for PVT prior to OLT is safe, and lead to partial or   
complete resolution in 19/30 patients. Improved survival was noted in the   
PVT cohort who received anticoagulation with warfarin prior to OLT. Data   
collection is ongoing to better refine which PVT patients derive benefit   
with this strategy.  
  
RECORD 184  
A differential to remember: Pylephlebitis in a patient with HIV presenting   
with abdominal pain and fever  
Sliwa D.F. Ryzewicz S.  
Journal of General Internal Medicine (2015) 30 SUPPL. 2 (S336). Date of   
Publication: April 2015  
  
LEARNING OBJECTIVE #1: Recognize pylephlebitis as part of the differential   
diagnosis in a patient with abdominal pain and fever, especially in patients   
with HIV/AIDS. LEARNING OBJECTIVE #2: Recognize the challenge of deciding   
whether to initiate anticoagulation given the underlying etiology of   
pylephlebitis. CASE: A 46-year-old male with a background history of HIV on   
HAART, Hepatitis C in remission, hypertension, hyperlipidemia, GERD and   
depression presented to the emergency room with epigastric pain, associated   
with mild nausea and loose, black stools. He took ibuprofen, pepto bismol   
and Alka-Seltzer at home without relief. In the emergency room, the patient   
developed a fever of 102.7, rigors and vomiting. Two weeks prior the patient   
had similar symptoms, which resolved. He denied sick contacts, recent travel   
or new food exposure. The remainder of the physical exam was significant for   
a heart rate of 120 and a stable blood pressure and respiratory rate. He had   
mild epigastric and right upper quadrant tenderness. There was no   
organomegaly or icterus. Initial laboratory studies demonstrated   
leukocytosis and elevated transaminases and total bilirubin. Lactate and   
lipase were normal. His most recent CD4 count was >1000 with a viral load of   
<50. Chest x-ray and abdominal CT with IV contrast were negative for acute   
processes. An abdominal ultrasound revealed biliary sludge without acute   
cholecystitis. EKG showed sinus tachycardia. The patient was started on IV   
fluids and antibiotics and admitted for further evaluation of a probable   
intraabdominal infection. Due to a continuing rise in the patient's   
transaminases and bilirubin, an MRI of the abdomen was completed and   
revealed thrombosis of the left portal vein, enlarged porta hepatis and   
reactive peripancreatic lymph nodes. Blood cultures grew Klebsiella   
pneumoniae, however stool and urine cultures were negative for growth. A   
diagnosis of pylephlebitis was made based on the presence of leukocytosis,   
fever and portal vein thrombosis. The patient was treated for 4 weeks with   
Levofloxacin. An outpatient work-up for thrombophilia was negative and due   
to the bacteremia being the likely cause of the portal vein thrombosis,   
anticoagulation was not initiated. DISCUSSION: Pylephlebitis is an uncommon   
but critical diagnosis to make in patients presenting with abdominal pain   
and fever, especially those with HIV/AIDS. Diagnostic criteria include   
portal vein thrombosis, fever, and often, bacteremia. Although pylephlebitis   
was universally fatal in the preantibiotic era, the morbidity and mortality   
have decreased with early identification and antibiotic management. There is   
data suggesting that people living with HIV and AIDS are at a 2 to 10 fold   
greater risk of venous thromboembolic disease compared to age-matched   
controls. Therefore, it is important to consider pylephlebitis in the   
differential diagnosis when evaluating HIV/AIDS patients presenting with   
these symptoms. Furthermore, anticoagulation in pylephlebitis is not well   
described in the literature, making the decision to anticoagulate these   
patients a difficult one. In the case of our patient, the etiology of the   
portal vein thrombosis was the pylephlebitis, making anticoagulation   
unnecessary with adequate antibiotic treatment. However, it is prudent to   
rule out underlying hypercoagulable states and to ensure complete response   
to antibiotics before deciding against anticoagulation.  
  
RECORD 185  
Should patients with hepatocellelar carcinoma complicated by portal vein   
thrombosis be treated with anticoagulation?  
Mahmoudi T.M. Kayal A. Carvalho R. Weiss A.  
Gastroenterology (2015) 148:4 SUPPL. 1 (S650-S651). Date of Publication:   
April 2015  
  
portal vein thrombosis (PVT) is a seen in about 14.3% of patients with   
hepatocellular carcinoma (HCC). There is presently no evidence based   
guideline on the need for anticoagulation in this particular group of   
patients. The aim of this retrospective study was to investigate the   
clinical outcome of patients with HCC complicated by portal vein thrombosis.   
Patients and methods: 54 patients who were diagnosed with HCC and PVT from   
July 21st 2001 to September 131st, 2014 were retrospectively evaluated. Nine   
patients were excluded secondary to lack of follow up. HCC and PVT diagnosis   
and follow up was determined with contrast enhanced CT or MRI. Most of the   
patients were initially treated with a single or a combination of the   
following treatments: transarterial chemoembolization, radiofrequency   
ablation, surgical resection, systemic therapy with Sorafenib.   
Characteristics and results are shown in table 1. 38 patients were males and   
mean age was 62.8. Liver disease etiology was HCV in 42%, HBV in 40%, ETOH   
in 11% and hemochromatosis in 2%. Results: Average survival after HCC   
diagnosis was 28 months and 15 months after PVT diagnosis. Among the 45   
patients evaluated, 6 patients received anticoagulation while 39 did not.   
Progression happened in 19 (49%) of the non anticoagulated group, and 4   
(67%) of the anticoagulated group. Right portal vein involvement was seen in   
18 (40%) patients with progression in 67% of the time, Left PVT in 13 (28%)   
with a progression in 54%, and Main PVT 6 (13%) with a progression in (67%).   
In 1 case, PVT progressed from the main PVT to Superior mesenteric vein   
(SMV) and in 2 other cases from the Left portal vein to SMV. Ascites was   
present in 2 patients, at the time PVT diagnosis but no symptoms or adverse   
clinical sequalae directly related to PVT development were reported in the   
other 43 patients. Conclusion, in our review of 45 patients with HCC   
complicated by chronic PVT, there was no adverse clinical consequence. The   
rate of progression of PVT determined by contrast enhanced CT or MRI imaging   
was similar in the group treated with anticoagulation and the non   
anticoagulated group of patients. Thus, the need for anticoagulation,   
considering its risks in patients with HCC and PVT, needs to be carefully   
assessed. The usefulness of anticoagulation in this patient population needs   
to be further studied. (Table Presented).  
  
RECORD 186  
Non-malignant portal vein thrombosis in patients with cirrhosis. Response to   
treatment  
Artaza T. Lopes M. Muñoz D. Romero M. González C. De La Cruz G. Sánchez J.J.   
Gómez R.  
Journal of Hepatology (2015) 62 SUPPL. 2 (S364). Date of Publication: April   
2015  
  
Background and Aims: The prevalence of nonmalignant portal vein thrombosis   
(PVT) ranges from 10 to 25% in patients with liver cirrhosis and it is   
associated with a worsening of its natural course. Optimal management of PVT   
in cirrhosis is not available in any consensus publication. Nevertheless, it   
seems that anticoagulation may constitute the initial treatment. The aim of   
our study was to evaluate the results of anticoagulation therapy in a group   
of cirrhotic patients with non-malignant PVT. Methods: 27 cirrhotic patients   
with non-malignant PVT were studied retrospectively in our hospital between   
March 2009 and March 2014. Both diagnosis and follow-up of patients were   
performed by Doppler and contrast-enhanced ultrasound and by computed   
tomography. Results: 27 patients (14 women, mean age: 59±11.8 years) were   
evaluated. All cases were incidental findings during routine   
ultrasonography. The mean MELD score was 10 (range: 6-22). 11% were on   
active liver transplant list. 26 patients received anticoagulation: 23   
low-molecular-weight heparin and three oral anticoagulation. The median time   
from diagnosis to the initiation of treatment was 2 weeks. The outcome in   
18/26 patients was recanalization, 15 complete (57.6%). The median time   
until achieving this complete response was 10 months (95% CI: 3-17).   
Rethrombosis occurred in five of the patients who had discontinued treatment   
after complete recanalization (35.7%). Patients with no response to   
treatment, did not show progression of thrombosis. Only two patients, one of   
them with 30,000 platelets, presented a bleeding complication (mild in both   
cases). No significant differences regarding the appearance of portal   
hypertensionrelated complications were observed. Patients with MELD score   
below 8 achieved recanalization in a significantly shorter time compared to   
the other patients (p = 0.04). Six patients died, four from complications of   
liver disease, but not related with anticoagulation. Thrombophilia testing   
was performed in 22 patients and five of them had a positive result: three   
with Factor V Leiden mutation, one with JAK2 gene mutation and another with   
hyperhomocysteinemia. Conclusions: In cirrhotic patients with nonmalignant   
PVT, anticoagulation therapy led to recanalization in over half of cases,   
with a broad safety profile. Best outcomes seem to be achieved in a less   
advanced stage of liver disease. Due to the existing rethrombosis rate,   
long-term anticoagulation should be considered.  
  
RECORD 187  
Acute portal vein thrombosis: Clinical features, diagnosis and outcomes   
after 5 years of follow-up  
Peixoto A. Silva M. Pereira P. Macedo G.  
Journal of Hepatology (2015) 62 SUPPL. 2 (S836). Date of Publication: April   
2015  
  
Background and Aims: Acute portal vein thrombosis (APVT) is a rare   
thrombotic obstruction of extrahepatic/intrahepatic portal venous system,   
associated with local and systemic risk factors. The clinical features APVT   
are poorly defined in the literature. The proportion that progress to   
chronic PVT and the influences of various treatments are unknown. The aim   
was to summarize the clinical features of APVT in a Portuguese reference   
center as well as their evolution over five years of follow-up. Methods: A   
total of 5 APVT patients admitted in our hospital from 2008 to 2009 were   
analyzed retrospectively. Results: APVT was diagnosed in 1 female and 4   
males, with a mean age of 47 years old. Most patients (n = 4) presented with   
abdominal pain and tenderness. Only one patient had cirrhosis by the time of   
diagnosis. A hereditary thrombofilia was detected in 2 cases, an   
intra-abdominal infection in 1, a mieloproliferative disease in 1, and   
nocturnal paroxistic hemoglobinuria in another one. Diagnosis was confirmed   
by angio-CT in 3 patients (60%) and MRI in another 3 (40%). None of the   
patients underwent thrombolysis nor thrombectomy. Four patients (80%) were   
anticoagulated after diagnosis (warfarin). The one patient with cirrhosis   
was not anticoagulated because of previous hemorrhagic events, and died soon   
after. The mean duration time of anticoagulation was 39 months, and was   
effective in half of the patients (n = 4). Intestinal infarction was the   
solo complication reported in one patient. No other patient died within the   
period of follow-up. Conclusions: Our data reveals to be similar to that   
found in the literature, showing the value of following the available   
recommendations regarding diagnosis and therapeutics. It also confirms that   
anticoagulation therapy is the treatment of choice in this rare disease,   
with real impact on long time survival.  
  
RECORD 188  
A case of elusive portal vein thrombus: The devil is in the detail  
Duran J.M. Brailovsky Y. Baang J.  
Journal of General Internal Medicine (2015) 30 SUPPL. 2 (S308). Date of   
Publication: April 2015  
  
LEARNING OBJECTIVE #1: Recognize idiopathic portal vein thrombosis and know   
the clinical and radiological findings that may mimic a gastrointestinal   
malignancy. LEARNING OBJECTIVE #2: Recognize the importance of discussing   
radiologic studies with a radiologist, especially when confronted with a   
diagnostic dilemma. CASE: A 57-year-old Caucasian woman presented with   
abdominal pain, and 1 week of diarrhea. The patient was found to have a   
fever of 102, mild abdominal distention and tenderness but an otherwise   
normal exam. She was mildly anemic with significant leukocytosis. She had a   
recent history of recurrent Clostridium difficile colitis after taking   
ciprofloxacin for a urinary tract infection. Upon admission she was found to   
have a urinary tract infection and a relapse of her clostridium difficile   
colitis. She was treated with appropriate antibiotics but her abdominal pain   
persisted. A CT abdomen with contrast was performed which revealed a   
mass-like lesion in the colon “concerning for metastatic colon cancer”,   
hepatic lesions, moderate amount of ascites, thrombus in the portal and   
intrahepatic vein and a “heterogenous mass” in the pancreatic head.   
Suspicion for malignancy was high and the thrombus was thought to be   
secondary to this malignancy. An aggressive GI work up including a   
colonoscopy, diagnostic paracentesis, upper GI endoscopy with biopsy of a   
nodular lesion in the duodenum and endoscopic ultrasound were performed, and   
all were negative for any significant findings. The pathology of the   
biopsied tissue was benign in nature. The patient's confusing picture   
prompted the primary team to discuss the CT findings with a   
hepatobiliary-imaging specialist that was not involved in the original   
radiologic readings. It was noted that the pancreatic duct was still intact,   
making malignancy very unlikely since this would cause destruction of the   
pancreatic duct. It was concluded that the stranding and “heterogenous mass”   
noted on the CT scan were secondary changes originating from the portal vein   
thrombosis. The patient was started on anticoagulation and was discharged   
with significant resolution of her symptoms. DISCUSSION: Acute portal vein   
thrombosis is a condition not infrequently encountered in the hospital. It   
is being diagnosed more frequently mainly due to the wide availability of   
ultrasound doppler devices. The clinical presentation of acute portal vein   
thrombosis is broad and can be easily confused with other conditions that   
may have similar presentations. Radiologic findings can mimic   
cholangiocarcinoma or a pancreatic head mass. In our case there were many   
“red herrings” that guided the team and consultants towards a diagnosis of a   
GI malignancy. The key element in coming up with the diagnosis was an   
additional review of our imaging study with a radiologist, also known as   
radiology rounds. Concerns have been raised regarding the disappearance of   
traditional radiology rounds, mainly driven by wide spread implementation of   
PACS and teleradiology, and the impact it may have on diagnostic errors. Our   
case exemplifies the importance of recognizing the signs and symptoms of   
portal vein thrombosis, which canmimic GI malignancies, and the significance   
of traditional radiology rounds, especially when confronted with a   
diagnostic dilemma.  
  
RECORD 189  
anticoagulation does not increase portal hypertension related bleeding, but   
exposes patients with cirrhosis to a high risk of minor hemorrhages. Results   
from a comparative cohort study  
La Mura V. Braham S. Branchi F. Moia M. Fracanzani A.L. Colombo M. Tripodi   
A. Primignani M.  
Journal of Hepatology (2015) 62 SUPPL. 2 (S365). Date of Publication: April   
2015  
  
Background and Aims: anticoagulation with vitamin K antagonists (VKAs) is an   
effective and relatively safe therapy for patients with portal vein   
thrombosis (PVT). However, the haemorrhagic risk of VKAs in relation with   
the presence of cirrhosis, has poorly been investigated Methods: We compared   
the VKAs-related bleeding risk in cirrhotic patients with de novo PVT   
(PVT-cohort, n = 62) vs non-cirrhotic patients with a thromboembolic event   
(TE-cohort, n = 160). Any bleeding during four years of follow-up or up to   
withdrawal of anticoagulation therapy, was recorded. The quality of   
anticoagulation control was measured by the time in therapeutic range (TTR)   
of the INR. Bleeding risk due to portal hypertension (PHT) in the PVT-cohort   
was compared with an independent series of cirrhotics with PHT unexposed to   
VKAs during follow-up (CHcohort, n = 53). Major bleeding episodes under   
anticoagulation were intracranial or retroperitoneal events, fatal bleeding   
events, need of hospitalization or transfusion, otherwise they were   
considered minor bleedings. All patients with cirrhosis were under   
prophylaxis for PHT-related bleeding according to current guidelines.   
Results: TE-cohort and PVT-cohort were comparable for age, sex. The mean of   
TTR was 67.7±20.9% for the former, 70.5±19.1% for the latter (p = 0.379) but   
treatment with VKAs was longer for the TE-cohort (31.1±16.9 vs 23.0±16.2   
months, p = 0.001). Overall, 41 patients under anticoagulation experienced a   
bleeding episode (14 major/27 minor). The actuarial probability of   
major/minor bleedings was higher in PVT-cohort (23%/30%) than in the   
TE-cohort (6%/20%) (p < 0.001). However, the risk of upper-gastro-intestinal   
bleeding in PVT-cohort (15%) was the same as in the CH-cohort (13%) also   
adjusting for potential confounders, confirming the lack of impact of VKAs   
on the risk of bleeding due to PHT. Finally, the exclusion of the   
upper-gastrointestinal bleeding in the PVT-cohort led to a significant   
reduction of major bleedings accountable for VKAs, leaving a significant   
residual risk only for minor bleeding episodes (p < 0.05). Conclusions: VKAs   
expose patients with cirrhosis and PVT to an additional risk of minor   
bleedings. This should be taken into account in future clinical studies to   
ameliorate the benefit/risk ratio of anticoagulation in this clinical   
setting.  
  
RECORD 190  
Risk factors associated with overall and bleeding-related mortality in   
patients with portal vein thrombosis on the waiting list for liver   
transplantation  
Iacob S. Iacob R. Ester C. Popescu I. Gheorghe C. Gheorghe L.  
Gastroenterology (2015) 148:4 SUPPL. 1 (S648). Date of Publication: April   
2015  
  
Background: The reported prevalence of PVT is increasing in patients with   
end stage liver disease (ESLD) awaiting LT. It significantly affects waiting   
list survival, complicates the liver transplant operation and impacts   
post-transplant survival and morbidity. anticoagulation is a challenging   
therapy in patients with ESLD because of the well-recognized coagulation   
abnormalities in cirrhotics, the increased risk of bleeding, and the lack of   
evidence of a real clinical benefit from the therapy. Aim: To investigate   
the risk factors for overall and hemorrhage related death in a cohort of 104   
cirrhotic patients with PVT included on the waiting list for LT. Methods: We   
tested separately the association between different parameters and overall   
death while on the waiting list using Cox regression model. Results: There   
were 68.3% men with a mean age of 53.0 ± 9.8 years, 30.8% of patients had   
HCV and 36.5% had HBV-related cirrhosis. Overall death was encountered in   
31.7% of patients, out of whom 14 (13.5%) were hemorrhage-related. As   
independent risk factors for overall death were identified the following:   
associated superior mesenteric vein thrombosis (p=0.04), refractory ascites   
with frequent paracentesis (p=0.01), shorter time from liver cirrhosis to   
PVT diagnosis (p<0.0001). Hemorrhage-related death was associated only with   
the administration of anticoagulation therapy (p=0.002). Conclusion:   
Anticoagulant therapy is associated with higher risk of hemorrhage-related   
death, but not with overall death on the waiting list.  
  
RECORD 191  
Risk factors associated with overall and bleeding-related mortality in   
patients with portal vein thrombosis on the waiting list for liver   
transplantation  
Iacob S. Ester C. Iacob R. Gheorghe C. Popescu I. Gheorghe L.  
Journal of Hepatology (2015) 62 SUPPL. 2 (S361). Date of Publication: April   
2015  
  
Background and Aims: The reported prevalence of PVT is increasing in   
patients with ESLD awaiting LT. It significantly affects waiting list   
survival, complicates the liver transplant operation and impacts   
post-transplant survival and morbidity. anticoagulation is a challenging   
therapy in patients with ESLD because of the wellrecognized coagulation   
abnormalities in cirrhotics, the increased risk of bleeding, and the lack of   
evidence of a real clinical benefit from the therapy. The aim of the study   
was to investigate the risk factors for overall and hemorrhage related death   
in a cohort of 104 cirrhotic patients with PVT included on the waiting list   
for LT. Methods: We tested separately the association between different   
parameters and overall death while on the waiting list using Cox regression   
model. Results: There were 68.3% men with a mean age of 53.0±9.8 years,   
30.8% of patients had HCV and 36.5% had HBV-related cirrhosis. Overall death   
was encountered in 31.7% of patients, out of whom 14 (13.5%) were   
hemorrhage-related. As independent risk factors for overall death were   
identified the following: associated superior mesenteric vein thrombosis (p   
= 0.04), refractory ascites with frequent paracentesis (p = 0.01), shorter   
time from liver cirrhosis to PVT diagnosis (p < 0.0001). Hemorrhage-related   
death was associated only with the administration of anticoagulation therapy   
(p = 0.002). Conclusions: Anticoagulant therapy is associated with higher   
risk of hemorrhage-related death, but not with overall death on the waiting   
list.  
  
RECORD 192  
An outcome survey of 43 patients with Budd-Chiari syndrome due to Behçet's   
syndrome followed up at a single, dedicated center  
Seyahi E. Caglar E. Ugurlu S. Kantarci F. Hamuryudan V. Sonsuz A. Melikoglu   
M. Yurdakul S. Yazici H.  
Seminars in Arthritis and Rheumatism (2015) 44:5 (602-609). Date of   
Publication: 1 Apr 2015  
  
Background: Behçet's syndrome (BS) is a well-recognized cause of Budd-Chiari   
syndrome (BCS); however, information about its clinical characteristics and   
outcome is limited. Methods: We reviewed the records of about 9000 patients   
with BS registered at the multidisciplinary Behçet's syndrome outpatient   
clinic at Cerrahpasa Medical Faculty between July 1977 and October 2013. We   
identified 43 (40 M/3 F) patients who were diagnosed as having BCS. Their   
outcome was evaluated between September 2012 and October 2013. Results: In   
total, 33 patients (77%) had presented with liver-related symptoms (Group   
I), while 10 (23%) were asymptomatic for liver disease (Group II). This   
latter group had presented with symptoms related to the presence of major   
vessel disease such as fever, leg swelling, or dyspnea. The site of venous   
obstruction determined in 41 patients was inferior vena cava (IVC) and   
hepatic veins combined in 25 (61%), IVC alone in 12 (29%), and only hepatic   
veins in 4 patients (10%). The number of patients with concurrent   
obstruction in the hepatic veins and the IVC was less in Group II than in   
Group I (3/10 vs 22/31, p = 0.06). A total of 20 (19 M/1 F) patients (47%)   
had died at a median of 10 months after diagnosis. Mortality was   
significantly lower in Group II (10%) than in Group I (58%), (p = 0.011). By   
the end of the survey, 23 patients were alive, of whom 21 could be   
re-evaluated at the clinic. Conclusions: BCS associated with BS is usually   
due to IVC thrombosis with or without hepatic vein thrombosis. Silent cases   
exist and have a better prognosis. The mortality rate among the patients   
symptomatic for liver disease remains high.  
  
RECORD 193  
Use of warfarin for the treatment of portal vein thrombosis in cirrhotic   
patients awaiting liver transplantation  
Butt W. Agrawal V. Rezk A. Komar M. Smith R. Khara H.S.  
Gastroenterology (2015) 148:4 SUPPL. 1 (S1041). Date of Publication: April   
2015  
  
Background: Untreated portal vein thrombosis (PVT) in cirrhotic patients   
leads to poor pre and post liver transplant outcomes. PVT prior to liver   
transplantation is an independent prognostic factor for post-transplant   
survival and prior studies have shown that complete or partial portal vein   
recanalization is associated with better survival after liver   
transplantation. Few studies have shown the safety and efficacy of low   
molecular weight heparin in the treatment of PVT, however, available data   
for the use of warfarin in this clinical setting is sparse and there are no   
clear guidelines for target INR for this patient population. Aim: The aim of   
our study was to evaluate the safety and efficacy of warfarin for the   
treatment of PVT in cirrhotic patients who underwent liver transplantation.   
Methods: We conducted a retrospective chart review of all cirrhotic patients   
listed for liver transplantation from Jan 2006 to Nov 2014 at our tertiary   
academic medical center with diagnosis of PVT pretransplantation. These   
patients were treated with warfarin therapy with a therapeutic INR target of   
one point higher than their baseline at the time of diagnosis of PVT.   
Results: A total of 86 patients underwent liver transplantation during the   
study period. Nine patients were diagnosed with thrombosis of pre-hepatic   
venous system, of which seven patients with portal vein thrombosis met our   
inclusion criteria. None of these patients had any contraindication for   
anti-coagulation. All study patients were males, mean age 52 years (range   
47-59), mean BMI 29.7 (range 26.7-36.7), with underlying etiology of chronic   
hepatitis C (n=3), or alcohol induced cirrhosis (n=1) or both (n=3). All   
patients treated with warfarin had a mean baseline INR of 1.43 (range   
1.1-1.92) and therapeutic goal INR was set at one point higher than the   
baseline at the time of diagnosis of PVT. All patients were treated until   
transplant with a mean duration of treatment of 11 months (range 2-20).   
Average MELD score at the time of PVT diagnosis was 24 (range 21-29). Six   
patients achieved re-canalization prior to liver transplantation within a   
mean time of 6 months (range 1-13). Only one patient had extension of   
thrombus while on warfarin treatment. No significant differences were noted   
in the occurrence of hepatic decompensation before or after the treatment.   
There were no adverse events related to the warfarin use. There was no   
posttransplant mortality at 1 year follow up. Conclusions: Treatment of PVT   
in cirrhotic patients awaiting liver transplantation with warfarin using   
therapeutic goal INR of one point higher than their baseline was safe and   
effective in our small pilot study. This data gives insight into determining   
the appropriate therapeutic goal INR for treatment of PVT with warfarin in   
these patients. Larger, multi-center studies should be conducted to further   
validate these findings.  
  
RECORD 194  
Complications of laparoscopic sleeve gastrectomy  
Fortounis K. Delivorias P. Paralikoudi D. Anastasiadou A. Makridis C.  
Surgical Endoscopy and Other Interventional Techniques (2015) 29 SUPPL. 1   
(S215). Date of Publication: April 2015  
  
Aim: The aim of our study was to examine the morbidity and mortality arising   
from laparoscopic sleeve gastrectomy (LSG) as a single-stage bariatric   
procedure. Method: 204 patients (F/M: 165/39) who underwent LSG as a   
single-stage bariatric procedure in our Department from 2009 to 2013 were   
retrospectively reviewed. Postoperative course, clinical presentation and   
treatment of complications were recorded. Results: Mortality rate was zero.   
No conversions to open operation occurred. A total complication rate of 7.3   
% was recorded. Staple line leak was the most frequent postoperative   
complication, observed in 5 patients (2.4 %). Conservative treatment with   
total parenteral nutrition and antibiotics was successful in 2 cases.   
However, 3 patients required a combination of percutaneous drainage under CT   
guidance, stenting and reoperation including abdominal washout, drainage and   
establishment of a feeding jejunostomy. There were also 3 cases (1.5 %) of   
hemorrhage (2 from the staple line and 1 from a port site) that needed   
transfusion, 4 cases (2 %) of nutrient deficiencies (2 of vitamin B12, 1 of   
vitamin B1 and 1 of folic acid) presented with peripheral neuropathy   
symptoms and managed with proper supplementation, 1 case (0.5 %) of sleeve   
stricture presented 6 months after LSG with dysphagia and vomiting that   
required open repair with Roux-en-Y gastrojejunostomy, 1 case (0.5 %) of   
superior mesenteric and portal vein thrombosis managed with therapeutic   
anticoagulation and 1 case (0.5 %) of intraabdominal abscess, in a patient   
under immunosuppressive therapy for rheumatoid arthritis, conservatively   
managed with antibiotics. Conclusion: LSG is a safe surgical option as a   
single-stage bariatric procedure, with relatively low complication rates.   
Early diagnosis and adequate multidisciplinary management of its major   
complications is the key for further reduction of LSG-related morbidity and   
mortality.  
  
RECORD 195  
Incidence and risk factors for perioperative thromboembolic events among   
patients with renal cell carcinoma and inferior vena cava tumor thrombus  
Viers B. Boorjian S. Lohse C. Psutka S. Morrisson G. Leibovich B. Thompson   
R.H.  
Journal of Urology (2015) 193:4 SUPPL. 1 (e615). Date of Publication: April   
2015  
  
INTRODUCTION AND OBJECTIVES: There is a high rate of mortality among   
patients who develop a perioperative thromboembolic (TE) event. While renal   
cell carcinoma (RCC) will present with inferior vena cava tumor thrombus   
(IVC-TT) in up to 10% of patients, the incidence of perioperative TE event   
in these patients remains understudied. As such, we evaluated our experience   
with the treatment of RCC IVC-TT to investigate the incidence and to   
evaluate factors associated with TE diagnosis. METHODS: We reviewed 183   
patients with RCC IVC-TT who underwent radical nephrectomy and IVC tumor   
thrombectomy between 2000-2010. A TE event was defined as the perioperative   
development of a bland thrombus or embolism not related to the IVCTT.   
TE-free, cancer-specific and overall survival were estimated using the   
Kaplan-Meier method. anticoagulation use and TE event were analyzed as a   
time-dependent covariate. Associations of clinicopathologic features with   
time to TE event, cancer-specific and all-cause mortality were evaluated   
using Cox proportional hazard regression models. RESULTS: A total of 23   
(13%) patients presented with a TE event at RCC diagnosis. Postoperatively,   
55 (30%) patients developed a TE event, at a median 23 (IQR 5-146) days   
following surgery, including 24 (13%) pulmonary emboli, 17 (9%) deep venous,   
13 (7%) bland IVC thrombi and 1 (0.5%) portal vein thrombosis. At a median   
follow-up of 1.2 (IQR 0.3-4.0) years, the cumulative incidence of TE event   
at 30, 90 and 365 days following surgery was 17%, 21% and 25%, respectively.   
An Eastern Cooperative Oncology Group performance status ≥1 (HR 2.03;   
p=0.01), hypercoagulability disorder (HR 4.66; p=0.001) and IVC ligation at   
the time of surgery (HR 2.33; p=0.02) were associated with an increased risk   
of TE event. Meanwhile, the development of a postoperative TE was   
significantly associated with an increased risk of RCC-related (HR 1.74;   
p=0.004) and all-cause mortality (HR 1.65; p=0.006). CONCLUSIONS: A TE event   
was identified postoperatively in 30% of patients with surgically treated   
RCC IVC-TT. While the majority of these events occur within 90 days after   
surgery, poor functional status, hypercoagulability disorder and IVC   
ligation were associated with higher risks of TE event.  
  
RECORD 196  
Safety and efficacy of anticoagulation therapy for portal/splanchnic vein   
thrombosis in patients with liver cirrhosis on the waiting list for liver   
transplantation  
Gheorghe L. Iacob S. Ester C. Iacob R. Gheorghe C. Popescu I.  
Journal of Hepatology (2015) 62 SUPPL. 2 (S312). Date of Publication: April   
2015  
  
Background and Aims: In patients with advanced cirrhosis, the presence of   
portal vein thrombosis (PVT) represents a cause of increased morbidity and   
mortality. Despite the high frequency of PVT in patients with end stage   
liver disease on the waiting list (WL) for liver transplantation (LT), there   
are few data on the efficacy and safety of anticoagulation therapy in this   
setting. Therefore neither clear recommendations, nor consensus regarding   
the optimal regimen and duration of anticoagulation therapy have been   
addressed in recent consensus publications on this specific issue. Aim: To   
investigate the safety and efficacy of anticoagulation therapy for   
portal/splanchnic vein thrombosis in patients with liver cirrhosis on the WL   
for LT. Methods: We included 121 patients with liver cirrhosis included on   
the waiting list for LT. Results: The prevalence of PVT on the National WL   
for LT is 19.1%. Out of 121 patients, 44.6% received anticoagulant therapy.   
35.1% received low weight heparine and 64.9% received acenocumarol for a   
mean time of 9.4±1.4 months. 39.6% of them had recanalization of the PVT,   
while 31.4% remained stable after 3 months of anticoagulant therapy.   
Complete and partial repermeabilization was acheived in 14.5% (7/48) and   
85.5% (41/48) respectively. 13.2% of patients were transplanted. Overall   
death was encountered in 28.1% (34/121) of patients while on the WL and 4.9%   
(6/121) died after LT. Severe hemmorhagic events (variceal   
bleeding/hemoperitoneum/ hemorrhagic stroke) occured in 9.2% of patients   
receiving anticoagulant therapy. Conclusions: PVT prevalence on WL for LT is   
high. Anticoagulant therapy is administred in 44.6% of patients, is safe and   
rather efficacious in recanalization of PVT before LT.  
  
RECORD 197  
Safety and efficacy of anticoagulation therapy for splanhnic vein thrombosis   
in patients with liver cirrhosis on the waiting list for liver   
transplantation  
Gheorghe L. Iacob S. Ester C. Iacob R. Popescu I. Gheorghe C.  
Gastroenterology (2015) 148:4 SUPPL. 1 (S1063). Date of Publication: April   
2015  
  
Background: In patients with advanced cirrhosis, the presence of portal vein   
thrombosis (PVT) represents a cause of increased morbidity and mortality.   
Despite the high frequency of PVT in patients with end stage liver disease   
on the waiting list (WL) for liver transplantation (LT), there are few data   
on the efficacy and safety of anticoagulation therapy in this setting.   
Therefore neither clear recommendations, nor consensus regarding the optimal   
regimen and duration of anticoagulation therapy have been addressed in   
recent consensus publications on this specific issue. Aim: To investigate   
the safety and efficacy of anticoagulation therapy for portal/splanhnic vein   
thrombosis in patients with liver cirrhosis on the WL for LT. Results: The   
prevalence of PVT on the National WL for LT is 19.1%. Out of 121 patients,   
44.6% received anticoagulant therapy. 35.1% received low weight heparine and   
64.9% received acenocumarol for a mean time of 9.4±1.4 months. 39.6% of them   
had recanalization of the PVT, while 31.4% remained stable after 3 months of   
anticoagulant therapy. Complete and partial repermeabilization was acheived   
in 14.5% (7/48) and 85.5% (41/48) respectively. 13.2% of patients were   
transplanted. Overall death was encountered in 28.1% (34/121) of patients   
while on the WL and 4.9% (6/121) died after LT. Severe hemmorhagic events   
(variceal bleeding/hemoperitoneum/ hemorrhagic stroke) occured in 9.2% of   
patients receiving anticoagulant therapy. Conclusions: PVT prevalence on WL   
for LT is high. Anticoagulant therapy is administred in 44.6% and is safe   
and rather efficacious in recanalization of PVT before LT.  
  
RECORD 198  
Results of pancreatic resection associated with portal vein resection in an   
Australian tertiary care centre  
Iorgulescu D.G. Ling S. Nikfarjam M. Fink M.A. Jones R. Muralidharan V.   
Starkey G. Christophi C.  
ANZ journal of surgery (2015) 85:4 (270-273). Date of Publication: 1 Apr   
2015  
  
BACKGROUND: Portal vein resection (PVR) with pancreatectomy is now accepted   
practice in cases with involvement by tumour. We present our experience of   
this procedure with particular emphasis on morbidity and survival.METHODS: A   
retrospective case-control analysis of a prospectively maintained database   
between 2004 and 2012 was undertaken. A total of 17 patients had pancreatic   
resections with PVR for cancer and were compared with 17 patients with   
identical tumour type and stage who underwent pancreatic resection without   
PVR next in chronological order. Information obtained included patient   
demographics, radiological and histological evidence of major vein   
involvement and post-operative morbidity. Disease- and recurrence-free   
survival were calculated using Kaplan-Meier curves.RESULTS: Procedures   
associated with PVR included pancreatico-duodenectomy in 11 and total   
pancreatectomy in six. Three patients underwent pancreatic resection as a   
re-operation. Pathological staging showed 2× T2N0, 5× T3N0, 1× T1N1, 2× T2N1   
and 7× T3N1 tumours. Seven PVR patients (41%) had post-operative morbidity   
Clavien 3 and 4, compared with none in no-PVR group, but rates of Clavien 1   
and 2 complications were similar. Six PVR patients developed PV thrombosis   
(35%), all with significant clinical consequences. Comparing the PVR group   
with the no-PVR group, there was significantly reduced median overall   
survival in (13.8 versus 43.1 months; P = 0.028) and recurrence-free   
survival (7.5 months versus 39.7; P = 0.004).CONCLUSIONS: Survival of   
patients after pancreatectomy with PVR was reduced and morbidity was high   
compared with no-PVR. Delayed portal vein thrombosis due to recurrence was   
common. Routine post-operative anticoagulation may be indicated in this   
group.  
  
RECORD 199  
Imbalance of pro- Vs. anti-coagulation factors in chinese patients with   
budd-chiari syndrome and non-cirrhotic portal vein thrombosis  
Chen H. Liu L. Qi X. He C. Yin Z. Wu F. Fan D. Han G.  
PLoS ONE (2015) 10:3 Article Number: e0119909. Date of Publication: 30 Mar   
2015  
  
Background and Aim: The coagulation abnormalities in non-cirrhotic   
Budd-Chiari syndrome (NC-BCS) and non-cirrhotic portal vein thrombosis   
(NC-PVT) are unclear. We conducted this case-control study to investigate   
the coagulation profile of NC-BCS and NC-PVT in Chinese patients. Methods:   
We measured the levels of factors II, V, VII, VIII, IX, X, XI, XII, protein   
C (PC), protein S (PS) and antithrombin (AT) in blood samples from 37 NC-BCS   
patients, 74 NC-PVT patients, and 100 healthy controls. The levels and   
ratios of pro- and anti-coagulation factors were compared between patients   
with NC-BCS and healthy controls, between different types of NC-BCS and   
between NC-PVT and healthy controls. Results: In patients with NC-BCS,   
factor VIII (P<0.001) was significantly elevated; factor V (P<0.001), VII (P   
<0.001), IX (P = 0.003), X (P<0.001), XI (P<0.001), XII (P<0.001), PC   
(P<0.001) and AT (P <0.001) were significantly decreased; and no difference   
was observed for factor II (P = 0.088) and PS (P = 0.199) compared with   
healthy controls. Factor VIII-to-PC (P = 0.008), factor VIII-to-PS (P =   
0.037) and factor VIII-to-AT (P = 0.001) were significantly increased; other   
ratios were significantly reduced or did not show any difference. No   
differences were observed between different types of NC-BCS for individual   
pro- and anticoagulation factors or the ratios between them. Among patients   
with NC-PVT, factor VIII (P<0.001) was significantly elevated and other   
factors were significantly decreased. Factor II-to-PC (P<0.001), factor   
VIII-to-PC (P<0.001), factor IX-to-PC (P<0.001), factor VIII-to-PS (P<   
0.001), factor II-to-AT (P<0.001), factor VIII-to-AT (P<0.001) and factor   
IX-to-AT (P<0.001) were significantly increased; all other ratios for NC-PVT   
were significantly reduced or did not show any significant difference.   
Conclusions: NC-BCS and NC-PVT are associated with elevated levels of factor   
VIII and the decreased levels of PC and AT were probably the most   
significant features of coagulation imbalance. Additionally, NC-PVT was   
associated with decreased levels of PS.  
  
RECORD 200  
portal vein thrombosis and arterioportal shunting due to chronic   
cholangitis: A rare complication of living donor liver transplantation  
Hsieh C. Chou C. Lin K. Lin C. Chen Y.  
HPB (2015) 17 SUPPL. 2 (91-92). Date of Publication: March 2015  
  
Objectives: The incidence of late onset portal vein thrombosis after living   
donor liver transplantation (LDLT) is approximately 6% in adults and 8% in   
children. To the best of our knowledge, portal vein thrombosis and   
arterioportal shunting due to chronic cholangitis after LDLT has never been   
reported. Methods: We present a patient with portal vein thrombosis due to   
chronic cholangitis after liver donor liver transplantation (LDLT). Results:   
A 52-year-old woman with a history of hepatitis B virus-related liver   
cirrhosis underwent LDLT. After the surgery, the patient had recurrent   
episodes of cholangitis due to common bile duct and intrahepatic bile duct   
stricture. Biliary stricture due to cholangitis eventually resulted in acute   
portal vein thrombosis. A stent was inserted via percutaneous transluminal   
portography. Blood flow through the portal vein progressively improved from   
the third through the 10th day after stent placement. The anticoagulation   
regimen was change to acetylsalicylic acid and clopidogrel hydrogen sulfate   
(Plavix®). On post-stenting day 10, follow-up CT scan showed good patency of   
the main portal vein and no evidence of arterioportal shunting. Conclusion:   
Cholangitis after liver transplantation is a rare cause of portal vein   
thrombosis. Regular follow-up examinations with color Doppler ultrasound are   
needed to monitor portal vein flow in patients with biliary complications   
after LDLT.  
  
RECORD 201  
Cases of portal vein thrombosis in hepatocellular carcinoma and liver   
cirrhosis treated with anticoagulation  
Lee H.Y. Ahn B.M. Lee E.S. Kim S.H. Lee B.S.  
Hepatology International (2015) 9:1 SUPPL. 1 (S337). Date of Publication:   
March 2015  
  
The prevalence of portal vein thrombosis (PVT) with cirrhosis has been   
reported more frequently in recent years. The reported prevalence of PVT is   
in the range of 0.6 ∼ 15.8 % in patient with liver cirrhosis or portal   
hypertension. If the patient has hepatocellular carcinoma(HCC), thrombus is   
likely to be malignant thrombus. Malignancy, frequently of hepatic origin,   
are responsible for 21 ∼ 24 % of overall cases. The overall mortality rate   
of PVT has been reported to be less than 10 %, but is increased to 26 % when   
associated with HCC and cirrhosis. Because actually it is not easy to   
distinguish between malignant thrombus and benign thrombus in clinical   
aspect, PVT in HCC are still debatable whether or not treatment when it   
diagnosed. Many studies have been made to distinguish malignant PVT and   
benign PVT. Fine needle biopsy of the thrombus has the potential of   
clarifying the nature of PVT. Tarantino et al. noted a sensitivity of only   
76 % for portal vein sampling in determining malignancy. Clinically, benign   
PVT was imaging documentation of at least 12 months of stability, and rapid   
progressive thrombi (within 3 months) despite adequate anticoagulation   
therapy were considered malignant. In recent years, the possibility of using   
color Doppler sonography, contrast-enhanced color Doppler sonography, CT,   
MRI and 18FDG-PET scan to determine the benign or malignant nature of PVT   
has been reported. We experienced 3 cases of PVT(54 year-old male, 73   
year-old female and 50 year-old male) with LC and HCC and treated with   
anticoagulation. After treatment, PVT has been improved and the patients   
receives a maintenance anticoagulation therapy without complication.  
  
RECORD 202  
portal vein thrombosis after total pancreatectomy and autologous islet cell   
transplantation  
Lancaster W.P. Adams D.B. Morgan K.A.  
HPB (2015) 17 SUPPL. 1 (63). Date of Publication: March 2015  
  
Introduction: portal vein thrombosis (PVT) is a rare complication of total   
pancreatectomy with autologous islet transplantation (TPIAT). Little is   
reported about the risk factors, consequences, or treatment for this   
complication. Methods: A retrospective review and analysis of a   
prospectively-collected database of patients undergoing TPIAT from March   
2009 to August 2014 was conducted. Two-tailed t-tests were used comparing   
continuous data and Fisher's exact test comparing categorical data. Results:   
135 patients (102 women,76%) underwent TPIAT; Nine(7%) had PVT. All patients   
with PVT were women. There were no differences in age or islet equivalents   
transplanted in patients with and without PVT. Mean BMI of patients with PVT   
was lower than those without (21.8 vs 26.5 kg/m2,p = 0.03).Mean portal   
pressure post-islet infusion was higher in patients with PVT (25.2 vs 16.0,p   
= 0.0007), with 4/9 having pressures over 30 mmHg. The median time to   
diagnosis of PVT was 10.5 days postoperative(range 7 to 210),with 7/9 having   
negative duplex POD1. Eight of 9 patients with PVT were treated with   
systemic anticoagulation and 7/8 had resolution on repeat imaging. One   
patient died from complications of anticoagulation. Two patients developed   
cavernous transformation(CTPV), one untreated and one diagnosed after CTPV.   
All patients with PVT were insulin-requiring at latest follow-up versus   
72/94 patients(77%) without PVT with at least 1-year follow-up(p = 0.035).   
Conclusions: PVT following TPIAT is an uncommon but serious complication. It   
occurs late in the postoperative period in women with a low BMI. A   
standardized follow-up imaging protocol is suggested. The treatment for PVT   
is anticoagulation. Patients with PVT can expect to be insulindependent.  
  
RECORD 203  
Irreversible electroporation (NanoKnife) for pancreatic cancer: A single   
institution series of 50 consecutive patients  
Mahendraraj K. Epelboym I. Schrope B. Chabot J.A. Kluger M.D.  
HPB (2015) 17 SUPPL. 1 (5). Date of Publication: March 2015  
  
Introduction: The NanoKnife® irreversible electroporation system (IRE) uses   
electrical energy to destroy neoplastic tissue invading surrounding   
neurovascular structures. Large scale IRE for pancreatic cancer has yet to   
be reported. This study examines a large cohort of IRE-treated pancreatic   
cancer patients to evaluate the safety of this novel surgical approach.   
Methods: Data was abstracted on all T3 and T4 pancreatic cancer patients who   
underwent IRE at a tertiary hepatobiliary unit from 2012-2014. Standard   
statistical methodology was used. Results: 50 consecutive patients were   
treated with IRE by 3 pancreatic surgeons, with 36(72%) cases performed by a   
single surgeon. Mean patient age was 65.8 ± 7.8 years, with 31(62%) male   
patients. There were 45(90%) adenocarcinoma cases, most commonly involving   
the pancreatic head (n = 16;32%) or body (n = 16;32%). IRE was used for   
primary local control in 25(50%) cases and margin ablation in 21(42%).   
Median survival was 11.8 ± 6.2 months. Median follow-up was 7.8 ± 9.6   
months, with length of stay 7.34 ± 5.6 days and readmission rate of 20%(n =   
10). 30- and 90-day complication rates were 36%(n = 18) and 6%(n = 3), most   
commonly portal vein thrombosis(n = 4;8%), intraabdominal collection(n =   
3;6%), and anemia requiring transfusion(n = 3;6%). Overall mortality   
attributable to IRE was 6%(n = 3). 3 additional mortalities were related to   
disease progression. Conclusions: IRE offers a feasible technique to manage   
advanced pancreatic cancer. To reduce morbidity and mortality,   
anticoagulation should be considered when performing IRE near the portal   
vein, and plastic stenting should be considered when performing IRE near the   
common bile duct. IRE is a potentially crucial tool in the arsenal of   
surgeons treating otherwise inoperable pancreatic cancer. (Table presented).  
  
RECORD 204  
Danaparoid sodium thrombolytic therapy followed by warfarin in cirrhotic   
patients with portal vein thrombosis  
Kawamura E. Enomoto M. Jogo A. Kotani K. Motoyama H. Kozuka R. Hagihara A.   
Yamamoto A. Fujii H. Uchidakobayashi S. Iwai S. Nishida N. Morikawa H.   
Kawabe J. Murakami Y. Tamori A. Shiomi S. Kawada N.  
Hepatology International (2015) 9:1 SUPPL. 1 (S338). Date of Publication:   
March 2015  
  
Background: portal vein thrombosis (PVT) is a complication of cirrhosis that   
reduces the hepatic reserve and causes variceal bleeding. In Japan, the   
efficacy of danaparoid sodium (Orgaran®), a hepa-rinoid anti-coagulation   
factor Xa, therapy (DS) for PVT has been reported. Methods: We   
retrospectively analyzed 41 hospitalized cirrhotic patients: 16 hepatitis C   
virus, 5 hepatitis B virus, 20 others; the model for end-stage liver disease   
(MELD) score 8.6 ± 4.7; platelets 80 ± 40 9 10[SUP]3[/SUP]/μL; 3 esophageal   
varices F0, 16 F1, 5 F2, 0 F3, and 17 unknown. DS 2500 units were   
administered daily (n = 41, mean duration: 9.5 days), followed by oral   
warfarin (prothrombin time-international normalized ratio: 1.5 ± 0.3) in   
outpatient clinic (n = 16, 25.8 weeks). The volume of PVT (PVTV) measured   
with a three-dimensional-image analyzer (SYNAPSE VINCENT®, n = 28), serum   
D-dimer (n = 29), and scinti-graphic portal shunt indices (normal, ≤ 10 %; n   
= 6) were monitored. Results: Thrombi formed at one site in 25 patients (18   
portal, 4 superior mesenteric, and 3 splenic veins) and at two or more sites   
in 16. At the end of DS, the PVTV decreased to 55.1 ± 40.2 % of baseline   
(8.6 ± 10.3 cm[SUP]3[/SUP], P<0.0001), D-dimer decreased from 11.8 ± 12.6   
μg/mL to 7.0 ± 7.4 μg/mL (P = 0.007), and the shunt indices decreased from   
62.4 ± 10.5 % to 56.9 ± 7.1 % (P = 0.250). During DS, Grade 2   
intraperitoneal bleeding occurred in one patient (2.4 %). During follow-up,   
PVTV increased in 33.3 % of the patients, MELD score in-creased in 37.5 %,   
platelets decreased in 50.0 %, and varices grade increased in 18.2 %.   
Conclusions: PVT could be resolved with DS with relative safety. Warfarin   
did not always maintain the effects of DS.  
  
RECORD 205  
Efficacy of transjugular intrahepatic portosystemic shunt (TIPS) for   
flow-enabled dissolution of spleno-mesenterico-portal venous thrombosis  
Lakhoo J. Bui J.T. Knuttinen M. Minocha J. Ray Jr. C.E. Gaba R.C.  
Journal of Vascular and Interventional Radiology (2015) 26:2 SUPPL. 1 (S96).   
Date of Publication: February 2015  
  
Purpose: portal vein thrombosis (PVT)-with or without splenic vein (SVT) or   
superior mesenteric vein (MVT) thrombosis- is a liver cirrhosis complication   
with potentially devastating implications, including intestinal   
ischemia/infarction, portal hypertensive variceal hemorrhage, and   
technically challenging liver transplantation. By improving portal venous   
flow dynamics, TIPS can enable PVT, SVT, and MVT clearance and may prevent   
deleterious outcomes. This study aimed to evaluate the efficacy of TIPS in   
clearing PVT and associated thrombosis. Materials and Methods: In this   
single-center, IRB-approved retrospective study, 16 patients underwent TIPS   
from 2008- 2014 for PVT as a primary (n=9) or secondary (n=7) indication.   
TIPS were not accompanied by pharmacomechanical clot disruption; rather,   
shunts served to increase portal blood flow to allow flow-mediated   
physiologic clot dissolution. Four patients with inadequate follow-up were   
excluded. Preand post-TIPS cross-sectional imaging were used to assess clot   
location, size, and degree of occlusion, with attention to resolution   
(vessel patency with no clot in previously occluded veins), reduction   
(decrease in clot size), stability (no change in clot size), or extension   
(increase in clot size). Results: The cohort included 5 men and 7 women   
(mean age 61 years). Thrombus was non-occlusive and asymptomatic in all   
cases, and spanned main PVT (n=9), intrahepatic PVT (n=5), SVT (n=6), and   
MVT (n=8). TIPS were created with 10 mm covered stents; mean final   
portosystemic pressure gradient was 8 mm Hg. At mean 190 days post-TIPS, 67%   
(n=8) had clot resolution, 25% (n=3) had clot reduction, and 8% (n=1) had   
stable clot; there were no cases of clot extension. Resolution rate was 67%   
for PVT and SVT, and 75% for MVT. Of note, 3/12 (25%) patients underwent   
anticoagulation during the post-TIPS period (warfarin for deep vein   
thrombosis in 2, dalteparin for cardiac thrombus in 1); all 3 patients had   
clot resolution. Two of 12 (17%) patients underwent successful liver   
transplant post-TIPS. Conclusion: TIPS effectively dissolves or decreases   
PVT, SVT, and MVT in cirrhotic patients. This may be a useful approach   
notwithstanding omission of pharmacomechanical methods.  
  
RECORD 206  
portal vein thrombosis: An emerging indication for transjugular intrahepatic   
portosystemic shunt creation?  
Hur M.J. Jajko R. Zivin S.P. Lakhoo J. Minocha J. Bui J.T. Ray Jr. C.E.   
Knuttinen M. Gaba R.C.  
Journal of Vascular and Interventional Radiology (2015) 26:2 SUPPL. 1   
(S215-S216). Date of Publication: February 2015  
  
Learning Objectives: 1. To describe portal vein thrombosis (PVT)   
epidemiology and classification 2. To define clinical presentation,   
diagnosis, and sequela of PVT 3. To review conventional medical and surgical   
therapies for PVT 4. To illustrate the application of transjugular   
intrahepatic portosystemic shunt (TIPS) creation for treatment of PVT 5. To   
summarize technical and clinical outcomes of TIPS for treatment of PVT   
Background: PVT is a relatively common occurrence in patients with liver   
cirrhosis, and may result in significant morbidity. Not only can this   
condition lead to complications such as mesenteric ischemia, portal   
hypertension with variceal bleeding, and portal cholangiopathy, but this   
entity can also negatively impact liver transplantation by both increasing   
operative complexity and diminishing post-surgical survival. Systemic   
anticoagulation is the current mainstay of therapy for PVT, but has limited   
capacity to spur portal venous recanalization, and its use may be limited in   
patients with variceal bleeding risk. Emerging data suggests that TIPS, by   
enhancing portal venous blood flow velocity and providing portal venous   
access for clot disruption and/or thrombolysis, may effectively clear portal   
venous clot and thereby potentially benefit patients with portal vein   
thrombosis. Clinical Findings/Procedure Details: This exhibit will review   
relevant aspects of patient selection as well as TIPS procedure technique   
and approaches (including flow-enhanced clot dispersal, thrombolytic agent   
assisted dissolution, direct mechanical disruption, maceration, and/or   
aspiration, and stent muralization or recanalization), with depiction using   
case examples. This poster will also summarize the available data on TIPS   
for treatment of portal vein thrombosis, present a case for portal vein   
thrombosis as an emerging TIPS procedure indication, and identify areas of   
research need to confirm the utility of TIPS for this application.   
Conclusion and/or Teaching Points: TIPS constitutes an emerging means to   
manage PVT. An up-to-date knowledge of patient selection, technical   
approaches, and procedure outcomes will assist practicing Interventional   
Radiologists involved in the care of liver cirrhotic patients to utilize   
this expanding application of TIPS.  
  
RECORD 207  
Portal vein recanalization-transjugular intrahepatic portosystemic shunt   
using the trans-splenic approach to achieve transplant candidacy in patients   
with chronic portal vein thrombosis: Proof of concept  
Thornburg B. Desai K.R. Hickey R.M. Sato K.T. Lewandowski R.J. Salem R.  
Journal of Vascular and Interventional Radiology (2015) 26:2 SUPPL. 1   
(S100). Date of Publication: February 2015  
  
Purpose: The aim of this study is to test our hypothesis that the   
trans-splenic (TS) route can be employed as an alternate approach for portal   
vein recanalization-transjugular portosystemic shunts (PVR-TIPS) for chronic   
main portal vein thrombosis (mPVT) in potential transplant candidates.   
Materials and Methods: With IRB approval, 11 consecutive patients with   
cirrhosis-induced chronic mPVT underwent transsplenic PVR-TIPS in 2013-2014.   
All patients were denied listing by our transplant team due to the presence   
of mPVT, a relative contraindication at our center. Patients were followed   
for adverse events. PV patency at follow-up was assessed by 1-month   
splenoportography and subsequently by ultrasound/MRI every 3 months.   
Following PVR-TIPS, patients were reviewed (and subsequently listed) at   
weekly multidisciplinary conference. Results: PVR-TIPS using the TS approach   
was successful in all 11 patients with no major complications. Median age   
was 61 years (range: 33-67), 9/11 (82%) were male, and nonalcoholic   
steatohepatitis / hepatitis C were the leading causes of liver disease   
(8/11, 4 each). Complete mPVT was found in 8/ 11 (73%) patients. Four out of   
11 patients (36%) had a MELD >18 and 8/11 (73%) had a baseline Child-Pugh   
score 7-10. Minor adverse events occurred in 2/11 (fever, encephalopathy).   
Five out of eleven patients (45%) exhibited some minor remaining thrombus in   
the PV at the end of the procedure; of these patients, 3/5 had complete   
thrombus resolution at one month, with the remaining 2/5 resolving at 3   
months (no anticoagulation). Three patients underwent successful   
transplantation with end-to-end anastomoses. Conclusion: Trans-splenic   
PVR-TIPS is a potentially safe and effective method to treat PVT and improve   
transplant candidacy.  
  
RECORD 208  
portal vein thrombosis in patient with gastric diffuse B large cell lymphoma  
Antic D. Djurasinovic V. Vukovic V. Mihaljevic B.  
Thrombosis Research (2015) 135 SUPPL. 1 (S78-S79). Date of Publication:   
February 2015  
  
A 57-year-old woman was admitted in hospital with a 4-month-history of   
epigastric pain and melena. On presentation hematologic values were:   
hemoglobin 104 g/L, white blood cell count 8×10(9)/L, platelets 614×10(9)/L   
and d dimer was 1.42 (reference range below 0.5). Abdominal MSCT scan   
revealed thickening of the stomach wall (up to 2 cm), enlarged spleen with   
wedge shaped low-attenuation defect described as infarction as well as   
portal and splenic vein thrombosis. Total gastrectomy was performed and   
pathohistological analisys confirmed diagnosis of diffuse large B cell   
lymphoma. Tests for thrombophilia: antithrombin, protein S and C level,   
lupus anticoagulant, anticardiolipin antibodies, factor V Leiden and factor   
II were normal while patient is MTHFR heterozigot and has PAI 4G/5G   
polymorphismus. Low molecular weight heparin were administered for long-term   
use and imunochemotherapy (adriablastin, cyclophosphamide, oncovin,   
prednisone, rituximab) was initiated. After 8 cycles of therapy control   
abdominal MSCT showed resolution of spleen changes and there were no signs   
of portal and splenic vein thrombosis. Pathogenesis of thromboembolic events   
in the setting of hematological malignancies is mulifactorial. In our case   
we can consider malignancy, thrombocytosis as well as MTHFR and PAI status.   
Very limited experience on treatment is currently available in the   
literature and generally derives from guidelines of solid cancer patients.   
Low molecular-weight heparins are being considered of interest for long-term   
anticoagulation rather than vitamin K antagonists, because of their short   
half time life having in mind potential development of thrombocytopenia   
caused by chemotherapy.  
  
RECORD 209  
portal vein thrombosis  
Chawla Y.K. Bodh V.  
Journal of Clinical and Experimental Hepatology (2015) 5:1 (22-40). Date of   
Publication: 1 Mar 2015  
  
portal vein thrombosis is an important cause of portal hypertension. PVT   
occurs in association with cirrhosis or as a result of malignant invasion by   
hepatocellular carcinoma or even in the absence of associated liver disease.   
With the current research into its genesis, majority now have an underlying   
prothrombotic state detectable. Endothelial activation and stagnant portal   
blood flow also contribute to formation of the thrombus. Acute non-cirrhotic   
PVT, chronic PVT (EHPVO), and portal vein thrombosis in cirrhosis are the   
three main variants of portal vein thrombosis with varying etiological   
factors and variability in presentation and management. Procoagulant state   
should be actively investigated. anticoagulation is the mainstay of therapy   
for acute non-cirrhotic PVT, with supporting evidence for its use in   
cirrhotic population as well. Chronic PVT (EHPVO) on the other hand requires   
the management of portal hypertension as such and with role for   
anticoagulation in the setting of underlying prothrombotic state, however   
data is awaited in those with no underlying prothrombotic states. TIPS and   
liver transplant may be feasible even in the setting of PVT however proper   
selection of candidates and type of surgery is warranted. Thrombolysis and   
thrombectomy have some role. TARE is a new modality for management of HCC   
with portal vein invasion.  
  
RECORD 210  
Review article: Portal vein obstruction - Epidemiology, pathogenesis,   
natural history, prognosis and treatment  
Kumar A. Sharma P. Arora A.  
Alimentary Pharmacology and Therapeutics (2015) 41:3 (276-292). Date of   
Publication: 1 Feb 2015  
  
Summary Background Portal vein obstruction may be due to portal vein   
thrombosis (PVT) or its sequale, the portal cavernoma. PVT is a common   
complication in liver cirrhosis, however, it may also occur as a primary   
vascular disorder, in absence of any liver disease. Aim To review the   
current knowledge on nomenclature, etiology, pathophysiology, clinical   
presentation, diagnostic workup and management of adult patients with   
obstruction in the portal vein, either as a primary vascular disease in   
adults, or as a complication of liver cirrhosis. Methods A structured search   
in PubMed was performed using defined keywords (portal vein obstruction,   
extra-hepatic portal vein obstruction, PVT and portal cavernoma), including   
full text articles and abstracts in English language. Results Several   
causes, operating both at local and systemic level, might play an important   
role in the pathogenesis of PVT. Frequently, more than one risk factor could   
be identified; however, occasionally no single factor is discernible.   
Diagnosis of portal vein obstruction depends on clinical presentation,   
imaging and laboratory investigations. Prompt treatment greatly affects the   
patient's outcome. Conclusions Portal vein obstruction occurring either due   
to thrombosis in the portal vein or due to the portal cavernoma, can   
contribute to significant morbidity and mortality in patients with or   
without cirrhosis. In recent years our understanding of etio-pathogenesis of   
portal vein obstruction has evolved tremendously, which has led to   
significant improvement in treatment outcomes. There are still areas where   
more studies are needed to better clarify the management issues of portal   
vein obstruction.  
  
RECORD 211  
Early joint application of anticoagulant drugs to prevent portal vein   
thrombosis after splenectomy and devascularisation  
Zheng C.-L. Zhao Y.-F. Tang Z. Wu Y. Qiao S.-S. Zhang S.-J.  
World Chinese Journal of Digestology (2015) 23:1 (129-133). Date of   
Publication: 2015  
  
AIM: To assess the preventive effects of early joint application of   
anticoagulant drugs on portal vein thrombosis after splenectomy and   
devascularization and to explore the possible mechanism. METHODS: One hundr   
ed and twent y - eight patients with portal hypertension who underwent   
splenectomy and devascularization were included, including 28 patients with   
early application of low molecular heparin calcium (heparin group), 42 with   
joint application of low molecular heparin calcium and low molecular dextran   
(joint group), and 58 without the use of anticoagulant drugs (control   
group). The rate of postoperative thrombosis, postoperative platelet count,   
and prothrombin time (PT) were compared among the three groups. The indexes   
of portal vein blood flow were also recorded. RESULTS: One month after   
surgery, portal vein thrombosis developed in 5 (8.57%) cases in the heparin   
group, in 1 (2.38%) case in the joint group, and in 14 (24.13%) cases in the   
control group. The rate of postoperative portal vein thrombosis was   
significantly higher in the control group than in the heparin group (P <   
0.05), and in the heparin group than in the joint group (P < 0.05).   
CONCLUSION: Early anticoagulation use can effectively prevent the formation   
of portal vein thrombosis after splenectomy, and combined use of low   
molecular heparin calcium and low molecular dextran has better effects.  
  
RECORD 212  
Reply  
Martinez M. Tandra A. Vuppalanchi R.  
Hepatology (2015) 61:4 (1436-1437). Date of Publication: 1 Apr 2015  
  
RECORD 213  
Management of portal hypertension, Budd-Chiari syndrome and portal vein   
thrombosis  
Robertson M. Hayes P.  
Medicine (United Kingdom) (2015) 43:11 (669-673). Date of Publication: 1 Nov   
2015  
  
Portal hypertension is associated with many of the known complications of   
cirrhosis and has an enormous impact on a patient's prognosis. Ascites and   
hepatic encephalopathy represent the most common complications of cirrhosis;   
both are associated with a significantly worse prognosis, with 50% survival   
over the next 1-2 years. Acute variceal bleeding is a life-threatening   
complication and represents a leading cause of death in patients with   
cirrhosis. With advances in care, such as prophylactic antibiotics,   
vasoactive drugs and early transjugular intrahepatic portosystemic shunt   
(TIPSS) in patients with bleeding refractory to early endoscopic management,   
the mortality rate has significantly improved but remains 15-20%. Secondary   
prophylaxis of variceal bleeding with non-selective β-adrenoceptor blockers   
and/or endoscopic variceal ligation has also improved survival. Budd-Chiari   
syndrome (BCS) is a life-threatening disorder resulting from hepatic venous   
outflow obstruction. Myeloproliferative neoplasms (MPN) represent the most   
common cause of BCS, although a significant proportion of patients have more   
than one risk factor. Therapeutic anticoagulation remains the first-line   
treatment for both BCS and symptomatic portal vein thrombosis. TIPSS is   
increasingly used in the management of BCS and can reduce the need for liver   
transplantation.  
  
RECORD 214  
Cytomegalovirus-associated portal vein thrombosis in an immunocompetent   
patient: An underestimated complication  
Wang T. Kuttikat A. Pulsalkar P. Nanguzgambo A. Bhalara S.  
Oxford Medical Case Reports (2015) 2015:5 (294-296). Date of Publication:   
2015  
  
We describe an immunocompetent adult with acute cytomegalovirus (CMV)   
infection complicated by extensive portal vein thrombosis. A literature   
review on the incidence, presentation, pathophysiology and management of   
CMV-associated thrombosis is included. Previously thought to be a rare   
complication, recent large case series and the present case reconfirm the   
increasing prevalence of CMV-associated thromboembolism in the   
immunocompetent adult.  
  
RECORD 215  
Issues with monitoring of unfractionated heparin in cirrhosis  
Potze W. Lisman T.  
Therapeutic Drug Monitoring (2015) 37:2 (279-280). Date of Publication: 25   
Apr 2015  
  
RECORD 216  
The risks of thromboembolism vs. recurrent gastrointestinal bleeding after   
interruption of systemic anticoagulation in hospitalized inpatients with   
gastrointestinal bleeding: A prospective study  
Sengupta N. Feuerstein J.D. Patwardhan V.R. Tapper E.B. Ketwaroo G.A. Thaker   
A.M. Leffler D.A.  
American Journal of Gastroenterology (2015) 110:2 (328-335). Date of   
Publication: 5 Feb 2015  
  
OBJECTIVES: Anticoagulants carry a significant risk of gastrointestinal   
bleeding (GIB). Data regarding the safety of anticoagulation   
continuation/cessation after GIB are limited. We sought to determine the   
safety and risk of continuation of anticoagulation after GIB. METHODS: We   
conducted a prospective observational cohort study on consecutive patients   
admitted to the hospital who had GIB while on systemic anticoagulation.   
Patients were classified into two groups at hospital discharge after GIB:   
those who resumed anticoagulation and those who had anticoagulation   
discontinued. Patients in both groups were contacted by phone 90 days after   
discharge to determine the following outcomes: (i) thromboembolic events,   
(ii) hospital readmissions related to GIB, and (iii) mortality. Univariate   
and multivariate Cox proportional hazards were used to determine factors   
associated with thrombotic events, rebleeding, and death. RESULTS: We   
identified 197 patients who developed GIB while on systemic anticoagulation   
(n=145, 74% on warfarin). Following index GIB, anticoagulation was   
discontinued in 76 patients (39%) at discharge. In-hospital transfusion   
requirements, need for intensive care unit care, and etiology of GIB were   
similar between the two groups. During the follow-up period, 7 (4%) patients   
suffered a thrombotic event and 27 (14%) patients were readmitted for GIB.   
anticoagulation continuation was independently associated on multivariate   
regression with a lower risk of major thrombotic episodes within 90 days   
(hazard ratio (HR)=0.121, 95% confidence interval (CI)=0.006-0.812, P=0.03).   
Patients with any malignancy at time of GIB had an increased risk of   
thromboembolism in follow-up (HR=6.1, 95% CI=1.18-28.3, P=0.03).   
anticoagulation continuation at discharge was not significantly associated   
with an increased risk of recurrent GIB at 90 days (HR=2.17, 95%   
CI=0.861-6.67, P=0.10) or death within 90 days (HR=0.632, 95% CI=0.216-1.89,   
P=0.40). CONCLUSIONS: Restarting anticoagulation at discharge after GIB was   
associated with fewer thromboembolic events without a significantly   
increased risk of recurrent GIB at 90 days. The benefits of continuing   
anticoagulation at discharge may outweigh the risks of recurrent GIB.  
  
RECORD 217  
Impact of anticoagulation on upper-gastrointestinal bleeding in cirrhosis. A   
retrospective multicenter study  
Cerini F. Gonzalez J.M. Torres F. Puente A. Casas M. Vinaixa C. Berenguer M.   
Ardevol A. Augustin S. Llop E. Senosiaín M. Villanueva C. de la Peña J.   
Bañares R. Genescá J. Sopeña J. Albillos A. Bosch J. Hernández-Gea V.   
Garcia-Pagán J.C.  
Hepatology (2015) 62:2 (575-583). Date of Publication: 1 Aug 2015  
  
Recent studies have shown that liver cirrhosis (LC) behaves as an acquired   
hypercoagulable state with increased thrombotic risk. This is why   
anticoagulation therapy (AT) is now frequently used in these patients.   
Variceal bleeding is a severe complication of LC. It is unknown whether AT   
may impact the outcome of bleeding in these patients. Fifty-two patients on   
AT with upper gastrointestinal bleeding (UGIB) were evaluated. Portal vein   
thrombosis (PVT) and different cardiovascular disorders (CVDs) were the   
indication for AT in 14 and 38 patients, respectively. Overall, 104 patients   
with LC and UGIB not under AT matched for severity of LC, age, sex, source   
of bleeding, and Sequential Organ Failure Assessment (SOFA) score served as   
controls. UGIB was attributed to portal hypertension (PH) in 99 (63%)   
patients and peptic/vascular lesions in 57 (37%). Twenty-six (17%) patients   
experienced 5-day failure; SOFA, source of UGIB, and PVT, but not AT, were   
independent predictors of 5-day failure. In addition, independent predictors   
of 6-week mortality, which was observed in 26 (11%) patients, were SOFA,   
Charlson Comorbidity index, and use of AT for a CVD. There were no   
differences between patients with/without AT in needs for rescue therapies,   
intensive care unit admission, transfusions, and hospital stay. Conclusions:   
Factors that impact the outcome of UGIB in patients under AT are degree of   
multiorgan failure and comorbidity, but not AT itself.  
  
RECORD 218  
Complete pathological response to transcatheter arterial infusion despite a   
rapidly progressing recurrent hepatocellular carcinoma with portal vein   
tumor thrombus: A case report  
Taguchi M. Sakuma Y. Sasanuma H. Sata N. Lefor A.K. Sasaki T. Tanaka A.   
Yasuda Y.  
International Journal of Surgery Case Reports (2015) 10 (20-24) Article   
Number: 1282. Date of Publication: 2015  
  
Abstract Introduction We report a patient with a rapidly progressing   
recurrence of hepatocellular carcinoma (HCC) with a portal vein tumor   
thrombus after radiofrequency ablation of the original lesion, then treated   
with transcatheter arterial infusion. Radical hepatic resection demonstrated   
a complete pathological response. Presentation of case A 60-year old male   
with alcoholic cirrhosis and gastric varices was diagnosed with HCC   
measuring 12 mm in segment 8. He underwent laparoscopic radiofrequency   
ablation, but recurred three months later. The lesion progressed rapidly and   
the right portal vein was occluded. He then underwent transcatheter arterial   
infusion with miriplatin and iodized oil, which was effective in reducing   
the size of the main lesion and portal vein tumor thrombus. Right anterior   
sectionectomy was then performed. Pathologically, there were no viable HCC   
cells in either the main lesion or the portal vein thrombus. He is alive two   
years and nine months after surgery without recurrence. Discussion A rapidly   
progressing HCC recurrence with portal vein tumor thrombus is usually   
associated with a poor prognosis. No effective treatments have been reported   
in this situation except hepatic resection. In this patient the tumor was   
effectively reduced after three courses of transarterial miriplatin and   
subsequent radical hepatic resection. This is the first report to achieve a   
complete pathological response for such an aggressive recurrence after   
initial radiofrequency ablation. Conclusion This strategy may result in   
long-term survival of patients with rapidly progressing recurrent HCC with   
portal vein thrombus, and further study is warranted.  
  
RECORD 219  
portal vein thrombosis in Patients with cirrhosis—Etiology, Diagnosis, and   
Management  
Intagliata N.M. Henry Z.H. Northup P.G.  
Current Hepatitis Reports (2015) 14:1. Date of Publication: 1 Jan 2015  
  
Non-neoplastic portal vein thrombosis is common in cirrhosis. As our   
understanding of coagulopathy in cirrhosis evolves, clinicians are now   
recognizing that cirrhosis patients are not protected from thrombosis. In   
particular, factors innate to cirrhosis and portal hypertension promote a   
local environment conducive to portal vein thrombosis. Improvement in   
current diagnostic imaging has made diagnosis of portal vein thrombosis   
accurate, and we are now beginning to understand the incidence and   
prevalence. Development of occlusive portal vein thrombosis portends a worse   
outcome after transplant. Medical therapy for portal vein thrombosis in   
cirrhosis patients is effective and safe in certain circumstances.   
Furthermore, evidence is now emerging that prevention of portal vein   
thrombosis may reduce hepatic decompensation and progression of liver   
disease. Identifying patients that will benefit from therapy and improvement   
of diagnosis and prognostication should be the focus of future   
investigation.  
  
RECORD 220  
portal vein thrombosis: What is new?  
Manzano-Robleda M.C. Barranco-Fragoso B. Uribe M. Méndez-Sánchez N.  
Annals of Hepatology (2015) 14:1 (20-27). Date of Publication: 2015  
  
portal vein thrombosis (PVT) is one of the most common vascular disorders of   
the liver with significant morbidity and mortality. Large cohort studies   
have reported a global prevalence of 1%, but in some risk groups it can be   
up to 26%. Causes of PVT are cirrhosis, hepatobiliary malignancy, abdominal   
infectious or inflammatory diseases, and myeloproliferative disorders. Most   
patients with PVT have a general risk factor. The natural history of PVT   
results in portal hypertension leading to splenomegaly and the formation of   
portosystemic collateral blood vessels and esophageal, gastric, duodenal,   
and jejunal varices. Diagnosis of PVT is made by imaging, mainly Doppler   
ultrasonography. According to its time of development, localization,   
pathophysiology, and evolution, PVT should be classified in every patient.   
Some clinical features such as cirrhosis, hepatocellular carcinoma, and   
hepatic transplantation are areas of special interest and are discussed in   
this review. The goal of treatment of acute PVT is to reconstruct the   
blocked veins. Endoscopic variceal ligation is safe and highly effective in   
patients with variceal bleeding caused by chronic PVT. In conclusion, PVT is   
the most common cause of vascular disease of the liver and its prevalence   
has being increasing, especially among patients with an underlying liver   
disease. All patients should be investigated for thrombophilic conditions,   
and in those with cirrhosis, anticoagulation prophylaxis should be   
considered.  
  
RECORD 221  
Acute portal vein thrombosis, No Longer a Contraindication for Transjugular   
Intrahepatic Porto-Systemic Shunt (TIPS) Insertion  
Mammen S. Keshava S.N. Kattiparambil S.  
Journal of Clinical and Experimental Hepatology (2015) 5:3 (259-261). Date   
of Publication: 1 Sep 2015  
  
portal vein thrombosis, once considered as a contraindication to   
transjugular intrahepatic porto-systemic shunt (TIPS) is now considered as   
an indication. We report a case with clinical and technical success in a   
patient with Budd Chiari syndrome and acute portal venous thrombosis. Though   
it is a well-established option, with the best of our knowledge, we could   
not find a report from India.  
  
RECORD 222  
anticoagulation for the treatment of portal vein thrombosis in liver   
cirrhosis: A systematic review and meta-analysis of observational studies  
Qi X. De Stefano V. Li H. Dai J. Guo X. Fan D.  
European Journal of Internal Medicine (2015) 26:1 (23-29). Date of   
Publication: 1 Jan 2015  
  
Background & aims Systematic review and meta-analysis were performed to   
evaluate the safety and efficacy of anticoagulation for the treatment of   
portal vein thrombosis (PVT) in cirrhotic patients. Methods The PubMed,   
EMBASE, Cochrane Library, and ScienceDirect databases were searched. The   
rates of bleeding complications and portal vein recanalization in patients   
who received anticoagulant therapy were pooled. The odds ratio (OR) with 95%   
confidence interval (CI) was calculated to express the difference in the   
rate of portal vein recanalization between anticoagulation and   
non-anticoagulation groups. All meta-analyses were conducted by using a   
random-effects model. Results Sixteen of 960 initially identified papers   
were included. Two studies reported a low incidence of major   
anticoagulation-related complications (4% [2/55] and 3% [1/33]), but no   
lethal complications occurred. The rate of anticoagulation-related bleeding   
ranged from 0% to 18% with a pooled rate of 3.3% (95% CI = 1.1%-6.7%). The   
heterogeneity was not significant in the meta-analysis. The total rate of   
portal vein recanalization ranged from 37% to 93% with a pooled rate of   
66.6% (95% CI = 54.7%-77.6%). The rate of complete portal vein   
recanalization ranged from 0% to 75% with a pooled rate of 41.5% (95% CI =   
29.2%-54.5%). However, the heterogeneity was significant in the 2   
meta-analyses. The rate of complete portal vein recanalization was   
significantly higher in anticoagulation group than in non-anticoagulation   
group (OR = 4.16, 95% CI = 1.88-9.20, P = 0.0004). The heterogeneity was not   
significant in the meta-analysis. Conclusion anticoagulation could achieve a   
relatively high rate of portal vein recanalization in cirrhotic patients   
with PVT. Given that only a small number of non-randomized comparative   
studies are reported, randomized controlled trials are warranted to confirm   
the risk-to-benefit of anticoagulation in such patients, especially   
anticoagulation-related bleeding.  
  
RECORD 223  
Pregnancy and vascular liver disease  
Bissonnette J. Durand F. de Raucourt E. Ceccaldi P.-F. Plessier A. Valla D.   
Rautou P.-E.  
Journal of Clinical and Experimental Hepatology (2015) 5:1 (41-50). Date of   
Publication: 1 Mar 2015  
  
Vascular disorders of the liver frequently affect women of childbearing age.   
Pregnancy and the postpartum are prothrombotic states. Pregnancy seems to be   
a trigger for Budd-Chiari syndrome in patients with an underlying   
prothrombotic disorder. Whether pregnancy is a risk factor for other   
vascular liver disorders is unknown.In women with a known vascular liver   
disorder and a desire for pregnancy, stabilisation of the liver disease,   
including the use of a portal decompressive procedure when indicated, should   
be reached prior to conception. The presence of esophageal varices should be   
screened and adequate prophylaxis of bleeding applied in a manner similar to   
what is recommended for patients with cirrhosis. Most women likely benefit   
from anticoagulation during pregnancy and the postpartum. Labor and delivery   
are best managed by a multidisciplinary team with experience in this   
situation. Assisted vaginal delivery is the preferred mode of delivery.   
Although the risk of miscarriage and premature birth is heightened, current   
management of these diseases makes it very likely to see the birth of a live   
baby when pregnancy reaches 20 weeks of gestation.  
  
RECORD 224  
portal vein thrombosis in cirrhosis: Controversies and latest developments  
Harding D.J. Perera M.T.P.R. Chen F. Olliff S. Tripathi D.  
World Journal of Gastroenterology (2015) 21:22 (6769-6784). Date of   
Publication: 14 Jun 2015  
  
portal vein thrombosis (PVT) is encountered in livercirrhosis, particularly   
in advanced disease. It has been a feared complication of cirrhosis,   
attributed to significant worsening of liver disease, poorer clinical   
outcomes and potential inoperability at liver transplantation; also   
catastrophic events such as acute intestinal ischaemia. Optimal management   
of PVT has not yet been addressed in any consensus publication. We review   
current literature on PVT in cirrhosis; its prevalence, pathophysiology,   
diagnosis, impact on the natural history of cirrhosis and liver   
transplantation, and management. Studies were identified by a search   
strategy using MEDLINE and Google Scholar. The incidence of PVT increases   
with increasing severity of liver disease: less than 1% in well-compensated   
cirrhosis, 7.4%-16% in advanced cirrhosis. Prevalence in patients undergoing   
liver transplantation is 5%-16%. PVT frequently regresses instead of uniform   
thrombus progression. PVT is not associated with increased risk of   
mortality. Optimal management has not been addressed in any consensus   
publication. We propose areas for future research to address unresolved   
clinical questions.  
  
RECORD 225  
Acute infective portal vein thrombosis secondary to acute sigmoid   
diverticulitis  
Cheesman A. Gremida A. Burton M.  
Journal of Gastroenterology and Hepatology Research (2015) 4:4 (1582-1584).   
Date of Publication: 2015  
  
Infective thrombosis of the portal vein may complicate any infectious   
process of the abdominal portion of the gastrointestinal tract. The   
diagnosis requires a high index of suspicion, and the most common findings   
include fever, abdominal pain and abnormal liver function tests. Abdominal   
CT scan is the preferred diagnostic modality. Treatment should be initiated   
immediately, including empiric use of antibiotics and concomitant   
anticoagulation in view of the high mortality rate.  
  
RECORD 226  
Decreased in vitro anticoagulant potency of Rivaroxaban and Apixaban in   
plasma from patients with cirrhosis  
Potze W. Adelmeijer J. Lisman T.  
Hepatology (2015) 61:4 (1435-1436). Date of Publication: 1 Apr 2015  
  
RECORD 227  
portal vein thrombosis of a newborn with corrected total anomalous pulmonary   
venous return  
Çakır U. Kahvecioğlu D. Alan S. Erdeve Ö. Atasay B. Uçar T. Arsan S.   
Çakmaklı H. Ertem M. Atalay S.  
Turkish Journal of Hematology (2015) 32:3 (267-270). Date of Publication: 4   
Aug 2015  
  
Total anomalous pulmonary venous return (TAPVR) is a rare and frequently   
isolated defect identified in 1% to 3% of all congenital heart diseases. To   
the best of our knowledge, portal vein thrombosis (PVT) associated with   
TAPVR has not been reported in the literature. We report a successfully   
managed PVT in a newborn with infracardiac-type TAPVR and review the   
literature. anticoagulation therapies were used during the neonatal period   
to prevent thrombus progression. PVT should be kept in mind in TAPVR   
patients who have open heart repair with total correction. The treatment in   
each neonate should be individualized with consideration of the risk/benefit   
ratio.  
  
RECORD 228  
Surgical Ligation of Portosystemic Shunt to Resolve Severe Hematuria and   
Hemafecia Caused by Type II Abernethy Malformation  
Jiang C. Ye W. Liu C. Wu W. Li Y.  
Annals of Vascular Surgery (2015) 29:5 (1020.e11-1020.e16). Date of   
Publication: 1 Jul 2015  
  
The purpose of this study was to report the use of venous pressure   
measurement during surgery for Abernethy malformation (AF). This is a case   
report of a 19-year-old man who suffered from hematuria and hemafecia for 3   
months with worsening symptoms a week before being sent to the emergency   
room. He was diagnosed with type II AF based on portal phlebography. We   
performed an open surgery; measured portal vein, inferior mesenteric vein   
(IMV), and inferior vena cava pressure; and decided to completely suture the   
IMV. anticoagulation therapy was used during follow-up, and CTV showed   
increased portal vein diameter at 12 months after the procedure. For type II   
AF, measuring extrahepatic portal venous pressure changes before and after   
shunt blockage during surgery can help determine whether it is feasible to   
block the shunt, and anticoagulation therapy can improve patient prognosis.  
  
RECORD 229  
Treatment of non-cirrhotic, non-tumoural portal vein thrombosis  
Llop E. Seijo S.  
Gastroenterologia y Hepatologia (2015) 39:6 (403-410). Date of Publication:   
2015  
  
Thrombosis of the splenoportal axis not associated with liver cirrhosis or   
neoplasms is a rare disease whose prevalence ranges from 0.7 to 3.7 per   
100,000 inhabitants. However, this entity is the second most common cause of   
portal hypertension. Prothrombotic factors are present as an underlying   
cause in up to 70% of patients and local factors in 10-50%. The coexistence   
of several etiological factors is frequent. Clinical presentation may be   
acute or chronic (portal cavernomatosis). The acute phase can present as   
abdominal pain, nausea, vomiting, fever, rectorrhagia, intestinal   
congestion, and ischemia. In this phase, early initiation of anticoagulation   
is essential to achieve portal vein recanalization and thus improve patient   
prognosis. In the chronic phase, symptoms are due to portal hypertension   
syndrome. In this phase, the aim of treatment is to treat or prevent the   
complications of portal hypertension. anticoagulation is reserved to   
patients with a proven underlying thrombophilic factor.  
  
RECORD 230  
Imaging Diagnosis of Splanchnic Venous Thrombosis  
Rajesh S. Mukund A. Arora A.  
Gastroenterology Research and Practice (2015) 2015 Article Number: 101029.   
Date of Publication: 2015  
  
Splanchnic vein thrombosis (SVT) is a broad term that includes Budd-Chiari   
syndrome and occlusion of veins that constitute the portal venous system.   
Due to the common risk factors involved in the pathogenesis of these   
clinically distinct disorders, concurrent involvement of two different   
regions is quite common. In acute and subacute SVT, the symptoms may overlap   
with a variety of other abdominal emergencies while in chronic SVT, the   
extent of portal hypertension and its attendant complications determine the   
clinical course. As a result, clinical diagnosis is often difficult and is   
frequently reliant on imaging. Tremendous improvements in vascular imaging   
in recent years have ensured that this once rare entity is being   
increasingly detected. Treatment of acute SVT requires immediate   
anticoagulation. Transcatheter thrombolysis or transjugular intrahepatic   
portosystemic shunt is used in the event of clinical deterioration. In cases   
with peritonitis, immediate laparotomy and bowel resection may be required   
for irreversible bowel ischemia. In chronic SVT, the underlying cause should   
be identified and treated. The imaging manifestations of the clinical   
syndromes resulting from SVT are comprehensively discussed here along with a   
brief review of the relevant clinical features and therapeutic approach.  
  
RECORD 231  
Long-term clinical outcomes of splanchnic vein thrombosis results of an   
international registry  
Ageno W. Riva N. Schulman S. Beyer-Westendorf J. Bang S.M. Senzolo M.   
Grandone E. Pasca S. Di Minno M.N.D. Duce R. Malato A. Santoro R. Poli D.   
Verhamme P. Martinelli I. Kamphuisen P. Oh D. D'Amico E. Becattini C. De   
Stefano V. Vidili G. Vaccarino A. Nardo B. Di Nisio M. Dentali F.  
JAMA Internal Medicine (2015) 175:9 (1474-1480). Date of Publication: 1 Sep   
2015  
  
IMPORTANCE: Little information is available on the long-term clinical   
outcome of patients with splanchnic vein thrombosis (SVT). OBJECTIVE: To   
assess the incidence rates of bleeding, thrombotic events, and mortality in   
a large international cohort of patients with SVT. DESIGN, SETTING, AND   
PARTICIPANTS: A prospective cohort study was conducted beginning May 2,2008,   
and completed January 30,2014, at hospital-based centers specialized in the   
management of thromboembolic disorders; a 2-year follow-up period was   
completed January 30, 2014, and data analysis was conducted from July 1,   
2014, to February 28, 2015. Participants included 604 consecutive patients   
with objectively diagnosed SVT; there were no exclusion critieria.   
Information was gathered on baseline characteristics, risk factors, and   
antithrombotic treatment. Clinical outcomes during the follow-up period were   
documented and reviewed by a central adjudication committee. MAIN OUTCOMES   
AND MEASURES: Major bleeding, defined according to the International Society   
on Thrombosis and Hemostasis; bleeding requiring hospitalization; thrombotic   
events, including venous and arterial thrombosis; and all-cause mortality.   
RESULTS: Of the 604 patients (median age, 54 years; 62.6% males), 21 (3.5%)   
did not complete follow-up. The most common risk factors for SVT were liver   
cirrhosis (167 of 600 patients [27.8%]) and solid cancer (136 of 600   
[22.7%]); the most common sites of thrombosis were the portal vein (465 of   
604 [77.0%]) and the mesenteric veins (266 of 604 [44.0%]). anticoagulation   
was administered to 465 patients in the entire cohort (77.0%) with a mean   
duration of 13.9 months; 175 of the anticoagulant group (37.6%) received   
parenteral treatment only, and 290 patients (62.4%) were receiving vitamin K   
antagonists. The incidence rates (reported with 95% CIs) were 3.8 per 100   
patient-years (2.7-5.2) for major bleeding, 7.3 per 100 patient-years   
(5.8-9.3) for thrombotic events, and 10.3 per 100 patient-years (8.5-12.5)   
for all-cause mortality. During anticoagulant treatment, these rates were   
3.9 per 100 patient-years (2.6-6.0) for major bleeding and 5.6 per 100   
patient-years (3.9-8.0) for thrombotic events. After treatment   
discontinuation, rates were 1.0 per 100 patient-years (0.3-4.2) and 10.5 per   
100 patient-years (6.8-16.3), respectively. The highest rates of major   
bleeding and thrombotic events during the whole study period were observed   
in patients with cirrhosis (10.0 per 100 patient-years [6.6-15.1] and 11.3   
per 100 patient-years [7.7-16.8], respectively); the lowest rates were in   
patients with SVT secondary to transient risk factors (0.5 per 100   
patient-years [0.1-3.7] and 3.2 per 100 patient-years [1.4-7.0],   
respectively). CONCLUSIONS AND RELEVANCE: Most patients with SVT have a   
substantial long-term risk of thrombotic events. In patients with cirrhosis,   
this risk must be balanced against a similarly high risk of major bleeding.   
Anticoagulant treatment appears to be safe and effective in most patients   
with SVT.  
  
RECORD 232  
Pretransplant portal vein recanalization-transjugular intrahepatic   
portosystemic shunt in patients with complete obliterative portal vein   
thrombosis  
Salem R. Vouche M. Baker T. Herrero J.I. Caicedo J.C. Fryer J. Hickey R.   
Habib A. Abecassis M. Koller F. Vogelzang R. Desai K. Thornburg B. Hohlastos   
E. Resnick S. Lewandowski R.J. Sato K. Ryu R.K. Ganger D. Kulik L.  
Transplantation (2015) 99:11 (2347-2355). Date of Publication: 23 Oct 2015  
  
Background.Chronic, obliterative portal vein (PV) thrombosis (PVT)   
represents a relative contraindication to liver transplantation (LT) in some   
centers. When PV thromboembolectomy is not feasible, alternative techniques   
(portacaval hemitransposition, portal arterialization, multivisceral   
transplantation) are associated with suboptimal outcomes. In cases where a   
chronically thrombosed PV has become obliterated, we developed PV   
recanalization (PVR)-transjugular intrahepatic portosystemic shunt (TIPS) to   
potentiate LT.We evaluated the impact of PVR-TIPS on liver function,   
transplant eligibility, and long-termoutcomes after LT. Methods. Forty-four   
patients with chronic obliterative main PVTwere identified during our   
institutional LTselection committee. After joint imaging review by   
transplant surgery/radiology, these patients underwent PVR-TIPS to   
potentiate transplant eligibility. Patients were followed by   
hepatology/transplant until LT, and ultimately in posttransplant clinic. The   
TIPS venography and serial ultrasound/MRI were used subsequently to document   
PV patency. Results. The main PV (MPV) was completely thrombosed in 17 of 44   
(39%) patients; near complete (>95%) occlusion was noted in 27 of 44 (61%)   
patients. Direct transhepatic and transsplenic punctures were required in 11   
of 43 (26%) and 3 of 43 (7%) cases, respectively. Technical success was 43   
of 44 (98%) cases. At PVRTIPS completion, persistence of MPV thrombus was   
noted in 33 of 43 (77%) cases. One-month TIPS venography demonstrated   
complete resolution of MPVThrombosis in 22 of 29 (76%)without   
anticoagulation. Thirty-six patients were listed for transplantation; 18   
(50%) have been transplanted. Eighty-nine percentMPVpatency rate   
and82%survival were achievedat 5 years. Conclusions.The PVR-TIPS may be   
considered for patients with obliterative PVTwho are otherwise appropriate   
candidates for LT. The high rate ofMPV patency post-TIPS placement suggests   
flow reestablishment as the dominantmechanism of thrombus resolution.  
  
RECORD 233  
portal vein thrombosis  
Clinics in Liver Disease (2015) 19:1 (199-221). Date of Publication: 2015  
  
portal vein thrombosis (PVT) is a rare event in the general medical setting   
that commonly complicates cirrhosis with portal hypertension, and can also   
occur with liver tumors. The diagnosis is often incidental when a thrombus   
is found in the portal vein on imaging tests. However, PVT may also present   
with clinical symptoms and can progress to life-threatening complications of   
ischemic hepatitis, liver failure, and/or small intestinal infarction. This   
article reviews the pathophysiology of this disorder, with a major focus on   
PVT in patients with cirrhosis, and presents detailed guidelines on optimal   
diagnostic and therapeutic strategies.  
  
RECORD 234  
The coagulation system in patients with end-stage liver disease  
Valla D.-C. Rautou P.-E.  
Liver International (2015) 35:s1 (139-144). Date of Publication: 1 Jan 2015  
  
In patients with cirrhosis, routine laboratory tests for primary hemostasis   
and coagulation usually show anomalies that are associated with excess   
bleeding in other settings, in particular low platelet counts and prolonged   
prothrombin time. However, under conditions similar to those in vivo,   
primary hemostasis and thrombin production do not appear to be decreased in   
patients with cirrhosis, particularly when the platelet count is above 75   
000/μl. Furthermore, there is laboratory and epidemiological evidence of a   
mild procoagulant and prothrombotic state in patients with cirrhosis.   
Bleeding is mainly because of portal hypertension rather than defective   
hemostasis. There is some evidence that anticoagulation therapy is not   
associated with an excess of severe bleeding and that it could improve the   
outcome in patients without portal vein thrombosis. At present, there is no   
clear evidence that portal vein thrombosis is responsible for the   
progression of liver disease and that anticoagulation therapy would improve   
the outcome of patients with portal vein thrombosis.  
  
RECORD 235  
Recurrent Thrombotic Events after Discontinuation of Vitamin K Antagonist   
Treatment for Splanchnic Vein Thrombosis: A Multicenter Retrospective Cohort   
Study  
Riva N. Ageno W. Poli D. Testa S. Rupoli S. Santoro R. Lerede T. Piana A.   
Carpenedo M. Nicolini A. Ferrini P.M. Martini G. Mangione C. Contino L.   
Bonfanti C. Gresele P. Tosetto A.  
Gastroenterology Research and Practice (2015) 2015 Article Number: 620217.   
Date of Publication: 2015  
  
It is generally recommended that patients with splanchnic vein thrombosis   
(SVT) should receive a minimum of 3 months of anticoagulant treatment.   
However, little information is available on the long-term risk of recurrent   
thrombotic events. The aim of this study was to evaluate the risk of venous   
and arterial thrombosis after discontinuation of vitamin K antagonist (VKA)   
in SVT patients. Retrospective information from a cohort of SVT patients   
treated with VKA and followed by 37 Italian anticoagulation Clinics, up to   
June 2013, was collected. Only patients who discontinued VKA and did not   
receive any other anticoagulant drug were enrolled in this study. Thrombotic   
events during follow-up were centrally adjudicated. Ninety patients were   
included: 33 unprovoked SVT, 27 SVT secondary to transient risk factors, and   
30 with permanent risk factors. During a median follow-up of 1.6 years, 6   
venous and 1 arterial thrombosis were documented, for an incidence of   
3.3/100 patient-years (pt-y). The recurrence rate was highest in the first   
year after VKA discontinuation (8.2/100'pt-y) and in patients with permanent   
risk factors (10.2/100'pt-y). liver cirrhosis significantly increased the   
risk of recurrence. In conclusion, the rate of recurrent vascular   
complications after SVT is not negligible, at least in some patient   
subgroups.  
  
RECORD 236  
Etiology and management of portal vein thrombosis: Recent progress in   
research  
Ma J.-Q. Yan Z.-P.  
Journal of Interventional Radiology (China) (2015) 24:4 (362-368). Date of   
Publication: 1 Apr 2015  
  
With the progress of imaging techniques, the diagnosis rate for portal vein   
thrombosis (PVT), that is used to be considered as a rare disease, has been   
rapidly increasing. PVT can be caused by systemic reasons such as various   
thrombophilie risk factors as well as a lot of local reasons such as   
cirrhosis, abdominal trauma and infection, malignant tumor, etc. At present,   
PVT is classified into acute and chronic entities based on the duration of   
clinical symptoms as well as on the presence or absence of portal cavernous   
transformation. The clinical manifestations and the treatment principles of   
the acute and chronic PVT are quite different. For acute PVT, the principle   
of treatment is to reopen the obstructed portal vein and to prevent the   
thrombus from entering into the superior mesenteric vein, while for chronic   
PVT the principle of treatment is focused on the management of the   
complications due to portal hypertension. The interventional management of   
portal thrombus plays an important role in reopening portal vein, reducing   
complications caused by portal hypertension, and restoring portal blood   
flow, etc. This paper aims to make a comprehensive review about the etiology   
and management of portal vein thrombosis.  
  
RECORD 237  
Progress in treatment of nontumoral portal vein thrombosis in liver   
cirrhosis  
Zhou J. Yang J.-H.  
World Chinese Journal of Digestology (2015) 23:5 (735-740). Date of   
Publication: 2015  
  
portal vein thrombosis (PVT) is not uncommon in patients with liver   
cirrhosis, and it increases the risk of gastroesophageal hemorrhage. At   
present, pharmacological t reatment is the preferred select ion of   
management of PVT. Studies have shown that anticoagulation therapy does not   
increase the risk of gastrointestinal bleeding. Therefore, pat ient s having   
indi cat ions should be given anticoagulation therapy as early as possible.   
When patients fail to respond to anticoagulation therapy, interventional   
therapy or surgery may be considered. This article reviews the recent   
knowledge about the treatment of PVT and discusses the progress in treatment   
of nontumoral PVT in liver cirrhosis.  
  
RECORD 238  
Is there a rationale for treatment of chronic liver disease with   
antithrombotic therapy?  
Hugenholtz G.C.G. Northup P.G. Porte R.J. Lisman T.  
Blood Reviews (2015) 29:2 (127-136). Date of Publication: 2015  
  
Recent advances in the understanding of the coagulopathy in chronic liver   
disease have provided a strong support for anticoagulation as a new   
therapeutic paradigm for patients with cirrhosis. Laboratory studies   
indicate that the net effect of changes in hemostasis in many patients with   
chronic liver disease is a hypercoagulable status. In turn, clinical   
thrombosis is increasingly recognized as a complication of liver disease.   
When occurring within the liver, thrombosis may even progress the disease   
course. Exciting preliminary data regarding the potential of   
low-molecular-weight heparin to slow down the progression of liver disease   
indicate that this class of drugs may improve outcome without a major   
increase in bleeding risk. However, this new era for antithrombotic therapy   
in chronic liver disease is still hindered by a persistent false notion that   
patients with cirrhosis are "auto-anticoagulated" by their underlying liver   
disease. In addition, there is insufficient clinical evidence on safety and   
efficacy of anticoagulant therapy in cirrhosis and the studies conducted so   
far are limited by small sample sizes, uncontrolled treatment arms, or by   
their retrospective nature. Finally, a lack of knowledge on how or when to   
monitor antithrombotic treatment to optimize the risk-benefit ratio has   
restricted a widespread application of anticoagulant treatment in clinical   
management algorithms. Nonetheless, by systematically covering possibilities   
and pitfalls, this review highlights the potential of antithrombotic therapy   
to improve the quality of life and the clinical outcome of patients with   
chronic liver disease.  
  
RECORD 239  
Effects of restoring portal flow with anticoagulation and partial   
splenorenal shunt embolization  
Intagliata N.M. Saad W.E. Caldwell S.H.  
Hepatology (2015) 61:3 (1088-1090). Date of Publication: 1 Mar 2015  
  
RECORD 240  
Portal vein recanalization-transjugular intrahepatic portosystemic shunt   
using the transsplenic approach to achieve transplant candidacy in patients   
with chronic portal vein thrombosis  
Habib A. Desai K. Hickey R. Thornburg B. Vouche M. Vogelzang R.L. Salem R.  
Journal of Vascular and Interventional Radiology (2015) 26:4 (499-506). Date   
of Publication: 1 Apr 2015  
  
Purpose To present the transsplenic route as an alternative approach for   
portal vein recanalization-transjugular portosystemic shunt (PVR-TIPS) for   
chronic main portal vein thrombosis (PVT) in potential transplant   
candidates. Materials and Methods In 2013-2014, 11 consecutive patients with   
cirrhosis-induced chronic main PVT underwent transsplenic PVR-TIPS. All   
patients had been denied listing for transplant because of the presence of   
main PVT, a relative contraindication in this center. The patients were   
followed for adverse events. Portal vein patency was assessed at 1 month by   
splenoportography and every 3 months subsequently by ultrasound or magnetic   
resonance imaging. After PVR-TIPS, patients were reviewed (and subsequently   
listed for transplant) at a weekly multidisciplinary conference. Results   
PVR-TIPS using the transsplenic approach was successful in all 11 patients   
with no major complications. Median age was 61 years (range, 33-67 y) and 9   
of 11 patients (82%) were men. Nonalcoholic steatohepatitis was the leading   
cause of liver disease in 4 of 11 patients (36%), and hepatitis C was   
present in 4 of 11 patients (36%). Complete main PVT was found in 8 of 11   
patients (73%). Of 11 patients, 4 (36%) had a Model for End-Stage Liver   
Disease score > 18, and 8 (73%) had a baseline Child-Pugh score of 7-10.   
Minor adverse events occurred in 2 of 11 patients (fever, encephalopathy).   
At the end of the procedure, 5 of 11 patients (45%) exhibited some minor   
remaining thrombus in the portal vein; 3 of the 5 patients (60%) had   
complete thrombus resolution at 1 month, with the remaining 2 patients   
having resolution at 3 months (no anticoagulation was needed). Three   
patients underwent successful liver transplant with end-to-end anastomoses.   
Conclusions Transsplenic PVR-TIPS is a potentially safe and effective method   
to treat PVT and improve transplant candidacy.  
  
RECORD 241  
Portal venous system thrombosis complicating acute pancreatitis  
Li S. Zhang G.-X. Shang D.  
World Chinese Journal of Digestology (2015) 23:28 (4529-4535). Date of   
Publication: 8 Oct 2015  
  
Acute pancreatitis (AP) is an acute inflammatory disease of the pancreas   
characterized by local or systemic complications. Portal venous system   
thrombosis (PVT) is a relatively rare complication, which is often an   
incidental finding on contrast-enhanced computed tomography (CECT) performed   
to assess symptoms or local complications. If clinicians focus on AP while   
ignoring PVT, it may have serious clinical consequences. Previous studies on   
PVT complicating pancreatitis focused principally on chronic pancreatitis   
(CP) patients. Only a few single-center studies have been reported in   
foreign countries, and there are rare studies in China. For PVT complicating   
AP, there is still controversy over whether to implement anticoagulation   
therapy or not, suggesting the lack of a standardized treatment. This paper   
aims to explore the characteristics and treatment of PVT complicating AP and   
reviews the literature with an aim to raise awareness of this complication.  
  
RECORD 242  
Antiphospholipid syndrome in Sarawak: real world experience in a developing   
country  
Teh C.L. Leong T.S.  
Clinical Rheumatology (2015) 34:1 (175-178). Date of Publication: 1 Jan 2015  
  
We performed a cross-sectional study of all antiphospholipid syndrome (APS)   
patients during an 8-year period (2006–2013) to describe the clinical   
features, serology profiles, treatment regimes, and outcomes in our center.   
There were a total of 59 patients in our study with the female to male ratio   
of 9:1. They have a mean age of 41.6 ± 12.1 years and a mean duration of   
illness of 38.4 ± 68.5 months. The majority of patients presented with   
vascular thrombosis (69.5 %) with equal arterial and venous involvements.   
Twenty-six patients (44.1 %) presented with obstetric complications with   
recurrent abortions (32.2 %) as the main manifestation. Most patients were   
on daily warfarin doses of 2–6 mg (91.0 %) with target INR of 2–3. There was   
neither recurrent thrombosis nor bleeding complications documented. There   
were 80 % live births following treatment in our patients.  
  
RECORD 243  
Parietal peritoneum as an autologous substitute for venous reconstruction in   
hepatopancreatobiliary surgery  
Dokmak S. Aussilhou B. Sauvanet A. Nagarajan G. Farges O. Belghiti J.  
Annals of Surgery (2015) 262:2 (366-371). Date of Publication: 30 Aug 2015  
  
Objective: To evaluate the parietal peritoneum (PP) as an autologous   
substitute for venous reconstruction during hepatopancreatobiliary (HPB)   
surgery. Background: Venous resection during liver or pancreatic resection   
may require a rapidly available substitute especially when the need for   
venous resection is unforeseen. Methods: The PP was used as an autologous   
substitute during complex liver and pancreatic resections. Postoperative   
anticoagulation was standard and venous patency was assessed by routine   
computed tomographic scans. Results: Thirty patients underwent vascular   
resection during pancreatic (n = 18) or liver (n = 12) resection, mainly for   
malignant tumors (n = 29). Venous resection was an emergency procedure in 4   
patients due to prolonged vascular occlusion. The PP, with a mean length of   
22 mm (15-70), was quickly harvested and used as a lateral (n = 28) or a   
tubular (n = 2) substitute for reconstruction of the mesentericoportal vein   
(n = 24), vena cava (n = 3), or hepatic vein (n = 3). Severe morbidity   
included Clavien grade-III complications in 4 (13%) patients but there was   
no PP-related or hemorrhagic complications. Histological vascular invasion   
was present in 18 (62%) patients, and all had an R0 resection (100%). After   
a mean follow-up of 14 (7-33) months, all venous reconstructions were patent   
except for 1 tubular graft (97%). Conclusions: A PP can be safely used as a   
lateral patch for venous reconstruction during HPB surgery; this could help   
reduce reluctance to perform vascular resection when oncologically required.   
Clinical trials identification: NCT02121886.  
  
RECORD 244  
Operative interventions for extrahepatic portomesenteric venous aneurysms   
and long-term outcomes  
Fleming M.D. Lall P. Nagorney D.M. Gloviczki P. Kalra M. Duncan A. Oderich   
G. Toomey B. Bower T.C.  
Annals of Vascular Surgery (2015) 29:4 (654-660). Date of Publication: 1 May   
2015  
  
Background Extrahepatic portal venous aneurysms (PVAs) are rare, and the   
pathogenesis is not fully understood. The optimum management of these   
patients is unknown. Methods Consecutive patients with PVA were identified   
over an 18-year period (1992-2010). A retrospective review was conducted.   
Clinical presentation, modality of diagnosis, surgical treatment, 30-day   
morbidity and mortality, and follow-up are reported. Results Four patients   
were identified who underwent surgical management of an extrahepatic PVA.   
Operative technique using left renal vein, femoral vein panel graft,   
polytetrafluoroethylene (ePTFE) graft, and segmental aneurysm wall resected   
with aneurysmorrhaphy is described. Early complications occurred in 1   
patient with an ePTFE graft. The patient returned to the operating room for   
bleeding. In addition, the same patient had a late graft thrombosis 6 years   
postoperatively when the anticoagulation was discontinued for pregnancy. The   
remainder of the patients recovered without complication, and their repairs   
are still patent with a mean follow-up of 78 months (17-144 months). There   
were no mortalities in the series. Conclusions Operative intervention for   
portomesenteric venous aneurysm can be done safely in select patients and   
should be considered in those with symptoms, rapid growth, mural thrombus,   
or aneurysms ≥4 cm in diameter. Repair with an autogenous interposition   
graft affords good long-term patency. Aneurysmorrhaphy may be performed if   
the remaining venous wall is of good quality.  
  
RECORD 245  
Risk factors for portal venous thrombosis under anticoagulation therapy   
after operation of portal hypertension  
Zhang B.-H. Wang G.-F. Chi P.  
Journal of Xi'an Jiaotong University (Medical Sciences) (2015) 36:4   
(565-567). Date of Publication: 5 Jul 2015  
  
Objective: To investigate the risk factors for portal vein thrombosis (PVT)   
under anticoagulation therapy after surgery in patients with portal   
hypertension. Methods: We made a retrospective analysis of clinical data of   
96 portal hypertension patients for surgical treatment at our hospital. All   
the patients with postoperative PVT or without were divided into two groups.   
Risk factors that may predict PVT were analyzed. Results: PVT developed in   
41 (40.08%) of 96 patients after surgery. Risk factors such as sex, age,   
Child-Pugh classification, type of operation, portal pressure and the   
pressure difference before and after surgery, preoperative prothrombin time,   
preoperative platelet count, spleen index, and portal vein diameter were not   
predictors of PVT. However, splenic vein diameter was an independent risk   
factor for PVT (P= 0.036); postoperative PVT tended to develop when the   
splenic vein diameter was larger than 11 mm. Conclusion: Preoperative color   
Doppler testing of splenic vein diameter can predict PVT after surgery in   
patients with portal hypertension.  
  
RECORD 246  
portal vein thrombosis associated with psoriasis: a case report  
Yudhishdran J.M. Navinan R. Jeyalakshmy S. Ratnatilaka A.  
BMC research notes (2015) 8 (87). Date of Publication: 2015  
  
BACKGROUND: Psoriasis is no longer viewed as an isolated dermatological   
ailment and instead is considered a systemic disease. The extension of this   
spectrum has heightened the known risk of morbidity and mortality due to the   
involvement of cardiovascular system and the risk of venous thrombosis. A   
number of cases have reported the increased occurrence of deep vein   
thrombosis and pulmonary embolism in the background of psoriasis, however   
portal vein thrombosis has not been reported to date. We report an index   
case of chronic portal vein thrombosis in a diagnosed patient with   
psoriasis.CASE PRESENTATION: A 67-year-old South-Asian female previously   
diagnosed and treated for psoriasis presented with a four month history of   
abdominal pain associated with abdominal distension. Clinical examination   
revealed an enlarged spleen and free fluid in the abdomen. Imaging with   
ultrasonography and computed tomography of the abdomen revealed features   
compatible with chronic portal vein thrombosis with cavernous   
transformation.CONCLUSION: This case highlights the importance of having   
clinical awareness of occurrence of thrombosis in patients with psoriasis.   
Typical symptoms favoring thrombosis should prompt thorough investigation to   
exclude this rare yet possible complication in patients with psoriasis,   
including that of portal vein thrombosis. Prophylaxis with anticoagulation   
still lacks strength of evidence to be justified in psoriasis. The exact   
pathogenesis of venous thromboembolism in psoriasis is still unexplained and   
further studies are needed to clarify the causal association.  
  
RECORD 247  
Managing unusual presentations of venous thromboembolism  
Ageno W.  
Journal of Thrombosis and Thrombolysis (2015) 39:3 (304-310). Date of   
Publication: 2015  
  
Venous thromboembolism that occurs in unusual sites is challenging because   
of the potential severity of presentation, the presence of some major   
provoking risk factors, the high prevalence of potential contraindications   
to antithrombotic therapies, the lack of solid evidence to guide therapeutic   
decisions, and because of the severity of long-term consequences. For   
example, venous thrombosis in the splanchnic veins frequently occurs in   
patients with liver cirrhosis. Not uncommonly, these patients present with   
concomitant active gastrointestinal bleeding, and/or low platelet count or   
oesophageal varices. If inadequately treated, splanchnic vein thrombosis   
(SVT) may further worsen portal hypertension and, thus, increase the   
long-term risk of bleeding. Up to 40 % of patients with cerebral vein   
thrombosis (CVT) have signs of intracranial bleeding at the time of the   
diagnosis. This finding is associated with worst prognosis in terms of death   
or severe disability. Despite the apparent presence of a major   
contraindication to anticoagulation, only a timely administration of   
parenteral anticoagulant drugs may improve this unfavourable outcome. The   
available evidence on the management of these two challenging disorders, SVT   
and CVT, will be reviewed in this article.  
  
RECORD 248  
Splanchnic vein thrombosis associated with myeloproliferative neoplasms: A   
study of the agimm & IWG-MRT groups in 519 subjects  
Pieri L. Guglielmelli P. Primignani M. Betti S. Randi M.L. Rumi E. Pascutto   
C. Cervantes F. Ellis M. Chen F. Delaini F. Harrison C.N. Specchia G.   
Gisslinger H. Vianelli N. Ruggeri M. Girodon F. Bosi A. Santarossa C.   
Carobbio A. Koren-Michowitz M. Lavi N. Tripathi D. Rajoriya N. Gupta R.   
Rossi E. Garcia N.C. Ricco A. Gisslinger B. Polverelli N. Cazzola M. De   
Stefano V. Barbui T. Tefferi A. Vannucchi A.M.  
Blood (2014) 124:21. Date of Publication: 6 Dec 2014  
  
Philadelphia-negative Myeloproliferative Neoplasms (MPN), including   
Polycythemia Vera (PV), Essential Thrombocythemia (ET), Myelofibrosis   
(Primary [PMF] and secondary to PV and ET [PPV-, PET-MF] and unclassified   
MPN (U-MPN), are associated with an increased risk of venous thrombosis in   
unusual sites, such as splanchnic vessels (SVT). SVT can lead to   
complications such as portal hypertension, esophageal and gastric varices,   
ascites,hepatic failure and biliopathy. According to a meta-analysis MPN is   
the underlying cause of portal vein thrombosis (PVT) in 31.5% and Budd   
Chiari syndrome (BCS) in 40.9% of cases (Smalberg, 2012); a more in-depth   
analysis of clinical characteristics and evolution of MPN-associated SVT has   
been hampered by heterogeneity of cohorts comprising small number of cases.   
We conducted a retrospective multicenter study in patients (pts) with SVT   
associated with WHO2008-diagnosed MPN, with the aim to describe patient   
characteristics, disease course and prognostic factors with potential   
implications for clinical practice. Data were collected from 16   
international hematologic centers in the framework of the Italian AGIMM and   
the IWG-MRT groups. We collected 519 cases of pts with PVT, splenic or   
mesenteric vein thrombosis (75.1%) and BCS (24.9%) associated with MPN. We   
used as comparator a cohort of 1686 controls (Ctr) represented by MPN   
without (w/o) SVT: 741 ET (43.9%), 684 PV (39.7%), 261 PMF (15.5%).   
Frequency of MPN associated with SVT was 37.8% ET (n=196), 36.8% PV (n=191),   
15.4% MF (n=80), 10% U-MPN (n=52). Median follow-up was 89.9 months (mo)   
(range 0.5-430). For SVT vs Ctr group females were 54.5% vs 44.4% in PV   
(P=0.001), 68.4 vs 63.5% (p=0.13) in ET, 63.7% vs 29.1% in PMF (p<0.0001);   
median age at MPN diagnosis (dg) was 43.5 yr (range 12-90) vs 60.6 yr (range   
12-93) (p<0.0001). Age at SVT dg was 44 yr (range 15-85). In 240 cases   
(46.7%) MPN and SVT dg were coincident, in 121 (23.6%) SVT occurred before   
MPN dg (median 26 mo, range 4-307) and in 153 (29.8%) during MPN follow up   
(median 68 mo, range 4-362). JAK2V617F mutation was found in 94% PV vs 94%   
in Ctr, 84% vs 61% ET (p<0.0001), 88.1% vs 68% PMF (p=0.006) and in 93%   
U-MPN. Erythropoietin-independent colonies (EEC) were evaluated in 111 SVT   
pts and found in 80 (72%), accounting for 38/48 PV (79%), 31/44 ET (70.5%),   
9/12 PMF (75%) and 2/7 U-MPN (28.6%). At dg, SVT PV pts had lower hemoglobin   
levels than Ctr: median was 17.4 g/dL vs 18.5 g/dL (p<0.0001) in male, 16.9   
g/dL vs 17.7 g/dL (p=0.0006) in female. A co-existing thrombophilic status   
was found in 38.5% SVT vs 11.8% of Ctr (p<0.0001). Recurrent SVT occurred in   
12.2% of pts with a rate of 1.6% person/year (CI 1.2-2.1); risk of venous   
thrombosis other than SVT was increased in SVT group vs Ctr (p=0.02), with   
no difference for arterial thrombosis. Hemorrhage was more frequent in SVT   
group (32%) vs Ctr (7.2%)(p<0.0001), mainly related to esophageal varices,   
which were present in 66.9% of SVT pts. There was no difference in evolution   
to MF and acute leukemia (AL) for PV and ET pts with and w/o SVT, while risk   
of AL was lower in MF with SVT (p<0.00001). Overall survival was shorter in   
ET pts with SVT vs Ctr (p<0.0001). In PMF survival was better in SVT group   
(p<0.00001) and was associated with a higher proportion of SVT pts in lowest   
risk categories: IPSS low 65%, intermediate-1 20%, intermediate-2 10% and   
high 5% compared with 15%, 34%, 25% and 26% in Ctr group. At last FU, 79/519   
pts (15.2%) had died; causes of death were evolution to AL (15.4%), other   
cancers (13.8%), disease progression without AL (10.8%), SVT (10.8%),   
hepatic failure and venous thrombosis other than SVT (7.7% each), heart   
failure and arterial thrombosis (6.2% each), hemorrhage (5.5%), renal   
failure and infection (4.6% each). Therapy after SVT included   
anticoagulation in 77%, antiaggregant in 21.2% and combination in 1.8%; 70%   
received cytotoxic drugs; 12.4% were treated with transjugular   
porto-systemic shunt. Beta blocker therapy was used in 48.5% of pts and   
correlated with improved survival (p=0.041) MPN associated with SVT   
correlated with younger age and female sex and might antedate the clinical   
phenotype in a quarter of the patients. MPN-associated SVT equally affected   
PV and ET, was more likely to occur in the presence of JAK2V617F or   
underlying thrombophilia and predicted recurrent venous but not arterial   
thrombosis. The apparent association of SVT with better or worse prognosis   
in PMF and ET, respectively, requires further investigation.  
  
RECORD 249  
Long-term complications after splenectomy in adult chronic immune   
thrombocytopenia with a minimum follow up of 10 years. First results from a   
single-center case-control study in 140 patients with primary ITP  
Thai L.-H. Mahevas M. Roudot-Thoraval F. Languille L. Dumas G. Khellaf M.   
Bierling P. Michel M. Godeau B.  
Blood (2014) 124:21. Date of Publication: 6 Dec 2014  
  
Introduction: Splenectomy was historically regarded as the gold standard for   
treatment in chronic adult immune thrombocytopenic purpura (ITP). However,   
the recent emergence of new drugs has deeply modified ITP management and   
splenectomy is no longer viewed as an unavoidable step in adult chronic ITP   
in many countries. The estimation of the risk over benefit of this potential   
curative treatment remains challenging both for patients and physicians. A   
retrospective Italian study focused on long-term outcome of patients   
splenectomized for ITP gave reassuring data concerning safety. A recent   
study from a large cohort of American veterans showed an increased risk of   
death due to septicemia, pulmonary embolism, coronary artery disease and   
cancer more than 10 years after splenectomy. We reported here the results of   
the first single center case-control study evaluating the long-term   
incidence of splenectomy complications with a minimum follow-up of 10 years.   
Methods: We retrospectively selected in a clinical computer database all   
primary ITP patients splenectomized more than 10 years ago in our unit. We   
matched 1 by 1 to non-splenectomized ITP patients based on date and age at   
ITP diagnosis and sex criteria. Clinical data were then completed from   
medical charts. All patients were interviewed by phone and a standardized   
questionnaire was used. Medical records from general practitioner or from   
Medical care center have been systematically obtained if necessary,   
especially for deceased patients. Comparison between groups were made using   
Fisher's test for qualitative variables, Kaplan-Meier method to estimate   
incidence and Rank test for comparison of cumulative incidence, with p<0.05   
defining significance. Results: Seventy splenectomized ITP patients were   
included (19men/51women) with a median age at ITP diagnosis of 37 years   
(range: 3-92). Sixty one (87%) initially responded to splenectomy but only   
34(48.5%) maintained a sustained response after a median follow-up of 189   
months (range:120-528). Matched non-splenectomized ITP patients had a median   
age at diagnosis of 40 years (range: 3-93) and a median follow-up since ITP   
diagnosis of 197 months (range: 96-504).Cumulative incidence of   
thromboembolic events was higher in the splenectomized group (p=0.029)   
(Figure1). Four (6%) episodes of post-operative portal vein thrombosis were   
observed, 3 were complicated by portal cavernoma requiring long-term   
anticoagulation. They tended to present with more thromboembolic events on a   
long-term (n=7) than non-splenectomized ITP patients (n=3, p=0.113). Two   
splenectomized (2.8%) and 1 non-splenectomized (1.4%) patients were   
diagnosed with post-embolic pulmonary arterial hypertension. The incidence   
of cardiovascular events was significantly higher in splenectomized group   
(9(13%) versus 2(2.8%), p=0.005) (Figure 2) with 6 transient and/or ischemic   
strokes in splenectomized patients (none in non-splenectomized).Infectious   
events were similar in the two groups (splenectomized: 12 (17%) vs 10 (14%))   
but infections were more frequent and severe in splenectomized patients.   
Indeed, 12 splenectomized patients presented 20 infectious events requiring   
hospitalization, 13 of them were pneumonia (Streptococcus Pneumoniae: n=4,   
Haemophilus Influenzae: n=1, undocumented: n=9). Five complicated   
septic-shocks leading to 3 deaths. In non-splenectomized group, 10 patients   
had 10 infectious events (Pneumonia n=4, Streptococcus Pneumoniae n=1), 7   
were hospitalized, none had septic-hock. Incidence of cancer was similar in   
the 2 groups (splenectomized: 11 (16%), non-splenectomized: 10   
(14%).Finally, the mortality rate was not different between two groups   
(splenectomized: n=14 (20%), non-splenectomized n=9, 13%). Ten (38%) of the   
36 non-responders patients deceased, 7 from hemorrhage and/or septic shock.   
Other splenectomized and non-splenectomized patients died from malignant   
cancer/hemopathy (n=5), coronary artery disease (n=2), other (n=6).   
Conclusion: Based on this case control single center study, we observed that   
long-term splenectomized patients have not only an increase risk of   
life-threatening infections, but also an increased risk of thromboembolic,   
and cardiovascular events. A long-term follow-up is therefore recommended in   
this patient population regardless the status of ITP in order to better   
prevent and manage such complications.  
  
RECORD 250  
Management of incidental splanchnic vein thrombosis in cancer patients  
Kreuziger L.B. Ageno W. Lee A.  
Hematology / the Education Program of the American Society of Hematology.   
American Society of Hematology. Education Program (2014) 2014:1 (318-320).   
Date of Publication: 5 Dec 2014  
  
A 75-year-old male with metastatic pancreatic cancer is undergoing   
chemotherapy with gemcitabine. A portal vein thrombosis was incidentally   
found on surveillance CT scan. He does not report any new abdominal pain or   
ascites. Should anticoagulation be used to treat asymptomatic portal vein   
thrombosis?  
  
RECORD 251  
Impact of hepatitis B on human immunodeficiency virus patients in Malaysia:   
A retrospective study  
Khan A.H. Syed Sulaiman S.A. Akhtar A. Adnan A.S. Aftab R.A.  
Value in Health (2014) 17:7 (A803). Date of Publication: November 2014  
  
Objectives: To assess the prevalence and clinical outcomes of Hepatitis B   
(HBV) patients co-infected with Human Immunodeficiency Syndrome (HIV) in a   
tertiary care hospital. Methods: A retrospective cross-sectional study was   
performed, of HBV positive HIV infected patients following HAART therapy   
from 2007 to 2012 in Infectious disease Unit, Hospital Palau Pinang (HPP),   
Malaysia. The demographic and clinical data of the patients was collected   
retrospectively. The collected data was analyzed with SPSS software (Version   
20) to measure the correlation of variables and their infection rates.   
Results: A total of 664 HIV infected patients including 495 (74.5%) males   
and 169 (25.5%) females with mean age of 40 ± 10.35 years were included in   
present study. Of these, 86 (13%) were co-infected with HBV. The main race   
involved in current study was Chinese 455 (68.5%) followed by Indians 88   
(13.3%), Malay 83 (12.5%) and minorities 38 (5.7%). The route of   
transmission was mainly male heterosexual contact 464 (69.9%) followed by   
homosexual 47(7.1%) and Intra- Venous Drug Users (IVDU) 48 (7.2%). The mean   
CD4 count, ALT and AST levels in HBV-HIV co-infected patients were 385 ±   
148.55, 51.48 ± 39.42, 105.581 ± 38.37 respectively. The co-infection is   
significantly associated with gender (p = 0.05), and IVDU (p = 0.01). The   
co-morbidities seen in the present study were Pulmonary Tuberculosis   
(17.9%), Pneumocystis pneumonia (15.4%), Hyperlipidemia (4.1%), Dyslipidemia   
(4.1%), Anemia (5.1%), Ischemic Heart Disease (1.8%), Diabetes Mellitus   
(8.7%), Hypertension (6.9%), Asthma (1.5%), Oral Candiasis (5.6%), Syphillus   
(4.2%), Liver Cirrohsis (0.6%), Cerebral Toxoplasmosis (1.8%), Virological   
Failure (0.6%). Conclusions: The overall prevalence of HBV among HIV   
patients were about 13% in which 74.5% was males while 25.5 % females.   
Raised levels of liver enzymes and lowered CD4 counts were seen in the   
co-infected patients. There was a significant correlation between   
co-infection with HBV among HIV patients depending on different variables.  
  
RECORD 252  
Chronic hepatitis C prevalence and its correlation with CD4 cells and liver   
enzymes among HIV positive patients: A Malaysian scenario  
Khan A.H. Sulaiman S.A. Soo C.T. Akhtar A. Hamzah D.A.B.A. Khan K.  
Value in Health (2014) 17:7 (A803). Date of Publication: November 2014  
  
Objectives: To evaluate the occurrence and clinical outcomes of Hepatitis C   
(HCV) patients co-infected with Human Immunodeficiency Syndrome (HIV) in a   
tertiary care hospital. Methods: A retrospective study of the patients with   
clinical histories of HIV co-infection with HCV following HAART therapy in   
Infectious disease Unit at Hospital Palau Pinang (HPP), Malaysia from the   
year 2007 to 2012. The clinical and demographic data was collected from   
patient's records. In present study we analyzed the collected data by using   
SPSS software (Version 20) to determine the correlation of variables and   
measure their infection rates in a particular population. Results: The study   
involves a total of 708 HIV infected patients with the mean age of 40 ±   
10.17 years together with 541(76.4%) males and 167(23.6%) females. There   
were 130(18.4%) patients co-infected with HCV. The assigned population   
involve in current study was Chinese 427(60.3%) followed by Indians   
96(13.6%), Malay 151(21.3%) and minorities 34 (4.8%). There were three main   
modes of transmission including male heterosexual contact 506(71.5%),   
homosexual contact 47(6.6%) and intravenous drug users (IVDU) 114(16.1%).   
The mean CD4 count, ALT and AST levels in HBV-HIV co-infected patients were   
374 ± 150.65, 64 ± 76.15, 129 ± 61.06 respectively. The calculated result   
shows the significant association of several factors like sex (p = < 0.001),   
IVDU (p = < 0.001) with co-infection of HIV-HCV. The co-morbidities observed   
in the current study were Pulmonary Tuberculosis (23.6%), Pneumocystis   
pneumonia (14.4%), Hyperlipidemia (4.4%), Dyslipidemia (3.2%), Anemia   
(4.5%), Ischemic Heart Disease (2.5%), Diabetes Mellitus (8.2%),   
Hypertension (6.5%), Asthma (1.4%), Oral Candiasis (5.2%), Syphillus (3.1%),   
Liver Cirrohsis (1.1%), Cerebral Toxoplasmosis (2.3%), Virological Failure   
(1.1%). Conclusions: The incidence rate of HCV among HIV individuals were   
about 18.4% including 76.4% males and 23.6% females. There was a significant   
correlation between HCV among HIV-positive patients depending on various   
variables like gender, age, exposure to risk factors. (p< 0.001).  
  
RECORD 253  
portal vein thrombosis in cirrhosis: Predictors of successful   
anticoagulation therapy  
Rodriguez-Castro K.I. Simioni P. Rossetto V. Ferrarese A. Zanetto A. Fadin   
M. Zerbinati P. Vitale A. Burra P. Senzolo M.  
Digestive and Liver Disease (2014) 46 SUPPL. 4 (e132). Date of Publication:   
10 Oct 2014  
  
Introduction: Predictors of successful anticoagulation therapy for the   
treatment of portal vein thrombosis (PVT) in cirrhosis are yet unknown. Aim:   
To assess the hemostatic status, as well as patient and thrombus   
characteristics, as predictors of therapeutic efficacy. Materials and   
methods: We evaluated 57 cirrhotics with PVT treated with LMWH for 1 year or   
until portal vein (PV) recanalization. The interval between PVT onset and   
start of anticoagulation was estimated. All cases were characterized in   
terms of severity of liver disease, extension ofPVTto other splanchnic   
vessels, occlusion grade, platelet number, and dosing of pro- and   
anti-coagulation factors, with calculation of factor VIII/Protein C ratio.   
PV recanalization was evaluated every two months using abdominal ultrasound,   
and every 3 months by CT scan, or every 2 months when ultrasound was not   
diagnostic. Results: Median age was 59 years (range 30-83), males 41/57,   
median MELD score was 12 (range 6-31). Etiology of cirrhosis was viral 44%   
and alcohol-related in 38%. PVT was partial in 44/57 patients.   
anticoagulation was started within 6 months of estimated thrombus onset in   
43/57 patients (75.4%). At 1 year, the recanalization rate was 38/57 (66.7%)   
patients (25 complete) after a median of 4.0 months (range 1-12 months). At   
multivariate analysis, Child Classes B/C vs A (OR 0.09; 95% CI 0.01-0.61, p   
< .01), interval between thrombus onset and start of therapy ≥6 months (OR   
0.03; 95% CI 0.1-0.28, p < .01), and total vs partial occlusion (OR 0.22;   
95% CI 0.04-1.14, p < .01) correlated negatively with the probability of   
response to anticoagulation therapy. Using these 3 variables, we developed a   
score with a high ability (AUC = 0.84) to predict PVT recanalization (range   
0-11 points). Conclusions: Both PV thrombus and patient characteristics, but   
not hemostatic status, correlate with the efficacy of anticoagulation. When   
approaching a cirrhosis patient with PVT, the clinician may consider this   
prognostic score in analyzing the net risk-benefit balance before initiating   
anticoagulation therapy.  
  
RECORD 254  
Hypercoagulability in cirrhotic patients with hepatocellular carcinoma (HCC)   
and portal vein thrombosis (PVT)  
Zanetto A. Ferrarese A. Rodriguez K. Fadin M. Gavasso S. Radu C. Zerbinati   
P. Vitale A. Cillo U. Farinati F. Russo F.P. Germani G. Simioni P. Burra P.   
Senzolo M.  
Digestive and Liver Disease (2014) 46 SUPPL. 4 (e138). Date of Publication:   
10 Oct 2014  
  
Background and aim: Studies which explores the hypercoagulable induced by   
HCC in cirrhosis are lacking. The aim of the present study was to evaluate   
the thrombophilic role of HCC as risk factor for development of PVT.   
Methods: Cirrhotic patients with and without HCC were prospectively enrolled   
in the study and underwent: thromboelastometry (ROTEM), platelet count,   
determination of prothrombin time and of levels of pro and anticoagulation   
factors. During followup, PVT onset in both patients with and without HCC   
was recorded. Results: 76 cirrhotics, 41 with HCC, were included. Volume of   
active HCC was >5cm3 in 18 patients. Levels of pro and anticoagulation   
factors were similar between patients with and without HCC, but fibrinogen   
was increased in HCC patients with active volume >5cm3 HCC compared to those   
with <5cm3 HCC bulk (348.72 mg/dL±124.06 mg/dL vs 237.64 mg/dL±99.18 mg/dL)   
and to cirrhotics without HCC (260.57 mg/dL±126.07 mg/dL) (p = 0.006).   
Platelet count was significantly increased in HCC compared to non-HCC   
patients, and this was especially true in Child A group. ROTEM demonstrated   
a significantly lower clotting time and maximum clot formation in HCC   
patients compared to controls and non-HCC cirrhotics, especially in Child A   
group. The incidence of PVT was 24.4% (10/41) and 11.4% (4/35) in HCC and   
non-HCC patients, respectively. At Cox multivariate analysis HCC and   
fibrinogen test of ROTEM were independently associated with risk of   
developing PVT. In the HCC group, 5/10 portal vein thromboses occurred in   
patients in Child Class A. At FIBTEM test of ROTEM, MCF and AUC were   
statistically greater in HCC patients who later developed PVT. Conclusions:   
Cirrhotics with HCC demonstrate a prothrombotic hemostatic balance resulting   
in an increased risk of PVT development. This prothrombotic state seems to   
be detectable by ROTEM and thus possibly suggest those who could benefit   
from thromboprophylaxis.  
  
RECORD 255  
Characteristics of splanchnic veins thrombosis: A multicenter community   
hospitals study  
Sherid M. Sifuentes H. Parikh M. Spurr C. Sridhar S.  
American Journal of Gastroenterology (2014) 109 SUPPL. 2 (S431). Date of   
Publication: October 2014  
  
Introduction: Splanchnic veins thrombosis (SVT) (mesenteric, portal &   
hepatic veins) is rare in general population, but is common in certain   
conditions (cirrhosis, pancreatitis and IBD). The clinical features,   
etiology, and outcomes are Different among these conditions. The aim of the   
study was to provide 7-year outcomes of SVT in our institutions. Medical   
records of patients with SVT from Jan. 2007-May 2014 reviewed. The study was   
conducted in 2 community hospitals after IRB approval with collaboration   
with GRU. ICD-9 codes were used to identify cases. The demographic details,   
clinical features, imaging findings, comorbidities, treatment and mortality   
were collected. The initial search identified 85 cases of these 3 entities;   
however after careful reviewing, most were excluded due to less specific   
ICD-9. Finally, only 5 cases were identified. We found 4 with acute   
mesenteric vein thrombosis (3 female). The precipitating factors were   
antiphospholipid antibody syndrome, unresectable pancreatic cancer, recent   
colectomy for colon cancer and end-stage liver disease. All 4 had abdominal   
pain of 1-3 days before admission & nausea in 3 patients. The patient with   
colectomy also had rectal bleeding & peritoneal signs. All cases were   
identified on CT scan showing thrombosis in superior mesenteric vein (SMV)   
in 3 cases and SMV occlusion by pancreatic mass in the 4th case. In the case   
of colectomy, the CT scan showed infarction of the ileum requiring emergent   
surgery. Two cases were treated with therapeutic dose of heparin. Long term   
anticoagulation with warfarin & aspirin was instituted only in the   
antiphospholipid patient. Both cases of unresectable pancreatic cancer and   
endstage cirrhosis died during follow up period of 42 and 254 days after the   
thrombotic events. The other 2 were followed for 2,214 and 1,893 days   
without adverse events (until the day of entering the data) and they had   
patent SMV on repeated CT scan. Additionally, we had an 86-year-old female   
with portal vein thrombosis in association with essential thrombocytosis.   
She presented 1 week after surgery for small bowel obstruction. CT scan   
showed thrombosis of left portal vein & superior mesenteric artery (SMA),   
infarction in left hepatic lobe and spleen, small bowel inflammatory changes   
and perforation, amd changes of acalculus cholecystitis requiring emergent   
surgery. She was placed on therapeutic dose of heparin followed by warfarin   
& aspirin. The patient recovered uneventfully. A repeat CT scan after 11   
days showed patent portal vein and SMA. She was followed for 949 days and   
had no adverse events. There were no cases of hepatic vein thrombosis during   
the study period. In this study, SVT occurred rarely (only 5 cases) during   
our 7-year study period. Precipitating factors & underlying disorders were   
usually identifiable.  
  
RECORD 256  
Thinking outside the bowels: Splanchnic venous thrombosis presenting as   
subacute abdominal pain  
Davis J. Houry M. Lattimer L. Kumar A. Borum M.  
American Journal of Gastroenterology (2014) 109 SUPPL. 2 (S343). Date of   
Publication: October 2014  
  
Introduction: Patients that present with abdominal pain and normal initial   
laboratory evaluation and imaging studies can be challenging to diagnose.   
portal vein thrombosis (PVT) is a rare cause of abdominal pain, particularly   
in non-cirrhotic patients. One autopsy study showed PVT in 1% of a Swedish   
population. Of that 1%, only 14% were considered “idiopathic,” with the   
majority related to cirrhosis or malignancy. We present a case of subacute   
abdominal pain that was found to be due to a splanchnic venous thrombus in a   
non-cirrhotic patient. A 67-year-old female with past medical history   
significant for hypertension presented with 2 weeks of epigastric pain that   
worsened post-prandially and radiated to her back. There was no associated   
nausea, vomiting, or change in bowel habits. On exam, she was afebrile with   
a pulse of 85 and mildly hypertensive at 151/77. Her abdomen was soft , but   
had decreased bowel sounds and tenderness over the epigastrium. Her initial   
laboratory tests were notable for a leukocytosis of 22,000, normal chemistry   
panel, normal liver enzymes (AST 37, ALT 28), lactate 1.8, and lipase 101.   
An abdominal ultrasound showed no abnormalities. Abdominal CT demonstrated a   
thrombus of her portal vein extending to the splenic vein and down into her   
superior mesenteric vein. She was started on therapeutic anticoagulation   
with unfractionated heparin, but had no improvement in her pain or clot   
burden after 3 days of therapy. She underwent thrombectomy and thrombolysis   
with resolution of her symptoms. She was discharged on therapeutic low   
molecular weight heparin and was subsequently diagnosed with   
antiphospholipid syndrome, requiring life-long anticoagulation. This is an   
unusual case of a patient presenting with mesenteric ischemic related to   
venous thrombus from antiphospholipid syndrome. Our patient had subacute,   
moderately severe abdominal discomfort related to mesenteric ischemia from   
venous thrombosis. Venous thrombosis accounts for only 5% of all cases of   
mesenteric ischemia. Notably, splanchnic thrombosis involving the mesenteric   
venous system is significantly more likely to present with pain and/or   
infarction than thrombus involving the portal vein alone. All patients with   
splanchnic thrombosis must undergo a thrombophilia evaluation, including an   
assessment for antiphospoholipid syndrome. Our case underscores the need to   
consider extraluminal etiologies of abdominal pain, particularly those that   
require urgent intervention.  
  
RECORD 257  
Pylephlebitis with pyogenic liver abscesses: A rare complication of   
pancreatitis  
Al-Hamid H. Manatsathit W. Johnson L. Barawi M.  
American Journal of Gastroenterology (2014) 109 SUPPL. 2 (S166). Date of   
Publication: October 2014  
  
Introduction: Pylephlebitis, or infective suppurative thrombosis of the   
portal vein, is a rare condition with a 30-80% mortality rate. Most cases   
are associated with intra-abdominal sepsis. Diverticulitis and appendicitis   
are the primary foci in most reported cases. Only 5% of cases are associated   
with pancreatitis. Case Report: A 38-year-old African American female with   
recent history of alcoholic pancreatitis complicated by pseudocyst status   
post endoscopic retrograde cholangiopancreatography (ERCP) with pseudocyst   
drainage presented with acute abdominal pain, fevers, nausea, and vomiting.   
Physical examination revealed normal heart rate and blood pressure.   
Abdominal exam was significant for severe epigastric tenderness and palpable   
liver margin. Laboratory values included WBC 10.5, hematocrit 23.6, lipase   
31, alkaline phosphatase 144, and normal hepatic transaminases.   
Contrast-enhanced computerized tomography scan of the abdomen showed acute   
portal vein thrombosis with multiple low-density lesions within the liver,   
suggestive of abscesses. Blood cultures grew the anaerobic bacteria   
Eubacterium aerofaciens. The diagnosis of infective suppurative thrombosis   
of the portal vein was rendered. Parenteral antibiotics and anticoagulation   
were initiated. CT-guided drainage of the largest liver abscess was   
performed, and cultures grew Streptococcus viridans. She showed progressive   
clinical improvement and she was successfully transitioned to outpatient   
care. Discussion: Portal vein pylephlebitis is very rare and usually   
presents with nonspecific clinical and laboratory findings. Pancreatitis is   
an uncommon etiology. Hepatic abscesses can complicate severe cases. The   
prothrombotic effect of the underlying infectious or inflammatory process is   
the main proposed mechanism. The early recognition and management of   
pylephlebitis with antibiotics and anticoagulation play a significant role   
in outcome.  
  
RECORD 258  
portal vein thrombosis significantly increases mortality in advanced   
cirrhosis with improved prognosis being associated with portal vein   
recanalization  
Ferreira C.N. Rodrigues T. Sousa P. Ramalho F. Alexandrino P. Velosa J.F.  
Hepatology (2014) 60 SUPPL. 1 (398A-399A). Date of Publication: October 2014  
  
Clinical significance of portal vein thrombosis(PVT) in cirrhosis not   
associated with hepatocellular carcinoma(HCC) is unclear. Aims 1.Analyse   
clinical features and factors associated with mortality in cirrhotics with   
PVT. 2.Study effect of anticoagulation(ACO) on portal vein   
recanalization(PVR) and influence on outcome. Methods: The study included 65   
consecutive cirrhotics with PVT without HCC. We analysed effect of severity   
of cirrhosis, clinical features and PVT on mortality at end of   
follow-up(FU). Mortality in study sample patients given ACO and those with   
PVR was compared to controls-175 patients without PVT with similar severity   
of cirrhosis (Child-Pugh(CP),MELD scores). Statistical analysis-SPSS 21.   
Results: 63%(41)males, age:58.7±12y. cirrhosis etiology: Alcohol-62%(40);   
viral-11%(7); alcohol+viral-12%(8); others-15%(10). cirrhosis   
severity:CP-8(2-15),MELD-13(6-35). CP class:A-19%(12),B-49(32),C-32%(21).   
Type of PVT: Acute-88%(57),chronic-12%(8). Extent of PVT: Trunk-80%(52);left   
branch-35%(23);right branch-57%(37);trunk+branches-31%(20);superior   
mesenteric vein-28(18);splenic vein-19%(12). Symptoms at PVT   
diagnosis:82%(53). Main features:Variceal bleed-45%(29),abd   
pain-30%(19),fever-16%(10). ACO after PVT diagnosis given in 19 patients   
(varfarin-15,LMWH-4). In 50 patients with FU imaging, PVR noted in   
50%(25)(Partial-13,total-12). Spontaneous PVR noted in 22%(7/32) patients.   
Median follow-up:10(0-376) m. Mortality: End of FU:25/65(39%); 1   
year:37%(18/49) 3 years63%(22/35) Cirrhotics with PVT who died had higher   
CP(p=0.004) and MELD(p=0.016 scores. cirrhosis etiology type and extent of   
PVT and clinical features did not influence mortality. CP class C cirrhotics   
with PVT had higher mortality at end of FU compared to class A+B (OR   
6,95%CI1.9-18.7,p=0.002). Overall, cirrhotics with PVT had similar mortality   
compared to controls. ACO improved PVR rates compared to no   
ACO(95%(18/19)vs22%(7/32), p<0.001) (OR 0.019,95%CI0.002-0.161,p<0.001), but   
did not reduce mortality compared to no ACO/controls. Patients with PVR had   
lower mortality (OR 0.14,95%CI0.04-0.49,p=0.002). Benefit of PVR on   
mortality reduction was observed only in CP class C patients(p=0.028).   
Conclusions: PVT is associated with higher mortality in CP class C   
cirrhosis. Spontaneous and ACO induced PVR signficantly reduced mortality in   
patients with cirrhosis and PVT. (Table presented).  
  
RECORD 259  
Variceal bleeding at diagnosis of portal vein thrombosis does not increase   
mortality in patients with cirrhosis  
Ferreira C.N. Rodrigues T. Sousa P. Ramalho F. Alexandrino P. Velosa J.F.  
Hepatology (2014) 60 SUPPL. 1 (1192A). Date of Publication: October 2014  
  
Introduction: portal vein thrombosis (PVT) in cirrhosis may aggravate portal   
hypertension with higher risk of failure to control variceal bleeding(VB)   
and early rebleeding. Aims: In patients with cirrhosis and PVT without   
hepatocellular carcinoma( HCC) 1. Analyze the clinical significance of VB at   
PVT diagnosis. 2. Evaluate influence of VB on mortality at 1 and 3 years.   
Methods: The study included 65 consecutive cirrhotics with PVT without HCC   
classified into two groups according to presentation at diagnosis of PVT:   
variceal bleed(VB) or no variceal bleed(NVB). We compared patients with VB   
with NVB and controls-74 patients with cirrhosis without PVT with VB at   
admission and similar Child-Pugh(CP) and MELD scores. Statistical   
analysis-SPSS 21. Results:Gender: 63%(41)males, age: 58.7±12years. cirrhosis   
etiology: Alcohol-62%(40); viral-11%(7); alcohol+viral-12%(8); others-   
15%(10). Severity of cirrhosis: CP class:A-19%(12), B-49%(32), C-32%(21).   
Scores:CP-8(2-15) and MELD-13(6-35). Type of PVT: Acute- 88%(57) and   
chronic-12%(8). Extent of PVT: Main trunk- 80%(52); left branch-35%(23);   
right branch-57%(37); main trunk+branches-31%(20); SMV-28%(18); splenic   
vein- 19%(12). anticoagulation after PVT diagnosis was given in 19 patients   
(varfarin-15, LMWH-4). In 50 patients with follow-up imaging tests, portal   
vein recanalization(PVR) was noted in 50%(25)(Partial-13, total-12). Median   
follow-up(FU) 10(0- 376) months. Mortality at end FU 25/65(39%). VB at   
diagnosis of PVT was noted in 45%(29) patients. Patients with VB were   
significantly older (63±9.3 vs 54±12.1, p=0.003) and had lower Hb levels   
(9.3±2.3 vs 10.8±2.2g/dL, p=0.01) compared to NVB. VB was more frequent in   
women than in men (65% vs 34%, OR 3.6, 95% CI1.24-10.5, p=0.02) There were   
no significant differences in etiology and severity of cirrhosis, type and   
extent of PVT in VB and NVB patients. Patients with VB were less likely to   
receive anticoagulant therapy (OR 0.24 95%CI 0.069-0.84, p=0.03). A trend   
for lower PVR rates was observed in patients with VB at diagnosis of PVT   
compared to NVB (25% vs 50%, p=0,069) By Cox and logistic regression   
analysis, there were no differences in mortality at end of FU (p=0.24) and   
at 1 year (p=0.42) between VB and NVB. Interestingly, mortality in patients   
with VB was lower at 3 years compared to NVB (0R 0.17, 95% CI 0.04-0.75,   
p=0.03). Kaplan Meier survival analysis showed that mortality in patients   
with VB at PVT diagnosis did not differ significantly from that in NVB or   
controls without PVT. Conclusion: Variceal bleeding at diagnosis of PVT in   
patients with cirrhosis does not increase mortality and is significantly   
more frequent in older and female patients.  
  
RECORD 260  
Incidence and outcome of newly diagnosed portal vein thrombosis in patients   
with cirrhosis awaiting liver transplantation  
Law C.S. Chatterji M. Chacko K. Schiano T.D. Chang C.Y.  
Hepatology (2014) 60 SUPPL. 1 (381A). Date of Publication: October 2014  
  
BACKGROUND: The incidence and natural history of acute portal vein   
thrombosis (PVT) in cirrhotic patients is poorly understood. We performed a   
case control study of cirrhotic patients listed for liver transplantation   
(LT) at Mount Sinai Medical Center to determine the: 1) incidence of new PVT   
in cirrhotics awaiting LT, 2) natural history of PVT, and 3) risk factors   
for poor clinical outcome in cirrhotics who develop PVT. METHODS: A   
retrospective chart review of patients listed for LT between Jan 1, 2002 and   
Dec 31, 2011 was performed. Subjects with new PVT (defined as PVT in a   
patient whose prior imaging showed patent PV) were identified via review of   
radiology, operative and explant pathology reports. Diagnosis, partial vs   
complete PVT, segmental (sPV) vs main (MPV) PVT and radiologic outcome   
(resolution, progression) of cases were confirmed through blinded review by   
a radiologist. Cirrhotic controls without PVT were matched (2:1) for age and   
time on the waitlist. Imaging and clinical outcomes (death on the waitlist,   
transplantation) were assessed. RESULTS: 1,761 patients were listed for LT   
between 2002-2011. 1,148 cases were excluded for reasons including HCC   
(n=739), chronic PVT (n=26), PVT prior to listing (n=77), and prior TIPS   
(n=59). We identified 20 cases of new PVT on imaging and 8 cases of   
incidental PVT found at the time of LT. Incidence of PVT was 4.6% over 10   
years. No patients received anticoagulation. Most of the imaging cases   
involved MPV only (n=10) or MPV + sPV (n=7) and 3 involved sPV only (n=3).   
14 had follow-up imaging, of which there were 4 (28%) cases of progression,   
7 (50%) cases of no change, and 3 (21%) cases of improvement, recanalization   
or cavernous transformation. There were 11 deaths on the waitlist (55%   
mortality rate) among PVT cases. Median time between PVT diagnosis and death   
was 133 days. Development of PVT while on the waitlist was associated with   
an increased risk for death (OR 3.44, p=0.03) compared to controls. A   
significantly increased risk for death while waiting was observed in   
patients with any involvement of the main PV (OR 6.73, p=0.002) or complete   
PVT (OR 10.33, p=0.003), but not in those with only sPVT (OR 2.36, p=0.49)   
or partial PVT (OR 3.37, p=0.08) compared to controls. CONCLUSIONS: The   
development of PVT in cirrhotic patients awaiting LT is associated with a   
high mortality rate. Spontaneous improvement of PVT is uncommon. Patients   
with cirrhosis who develop main PVT or complete PVT warrant consideration   
for intervention trials using anticoagulation. Prioritization on the waiting   
list with a variance may mitigate the burden of high mortality without   
transplant seen in this population.  
  
RECORD 261  
Impact of portal vein thrombosis prior to liver transplantation: A   
multi-center retrospective cohort study  
Karvellas C.J. Cardoso F.S. Wells M.M. Handoo F.A. Kwapisz L. Alghanem M.G.   
Kneteman N. Marotta P. Al-Judaibi B.  
Hepatology (2014) 60 SUPPL. 1 (455A). Date of Publication: October 2014  
  
Background/Aims: To identify the impact of portal vein thrombosis (PVT) on   
post liver transplant (LT) outcomes along with other covariates and assess   
factors associated with complications amongst PVT patients. Methods:   
Retrospective cohort study of 621 adult LT recipients (University of   
Alberta, London Health Sciences Centre) between 01/2002-12/2012. PVT was   
identified in 147 (24%) patients and 474 (76%) non PVT patients served as   
controls. Cox survival analysis was performed to determine independent   
associations with overall mortality. Results: Demographic factors (mean age   
53, 69% male) were similar between groups. There were also no differences in   
mean MELD (PVT 19 vs. controls 19, p=0.9) and Child Pugh scores (10 vs. 10,   
p=0.9) on the day of LT. Donor factors (mean DRI:1.6 vs. 1.5, p=0.2) were   
similar. Using Cox multivariable survival analysis, covariates independently   
associated with overall mortality included Age (adjusted Hazard ratio ∼ aHR   
1.02, p=0.015) and requiring ICU support pre-LT (aHR 2.17, p=0.006), but not   
PVT (p=0.67). 5-year survival was similar between PVT and controls (75%,p=   
0.8). In comparing PVT patients who did not survive (n=32) with PVT   
survivors (n=115), non-survivors (n=32) were more likely to have complete   
thrombus occlusion (38% vs. 13%, p=0.027) and hepatofugal flow (31% vs. 13%,   
p=0.08). Non-survivors were more likely require thrombectomy (69 vs. 31%,   
p=0.08) and develop reocclusion post-LT (16% vs. 3%, p=0.024).   
anticoagulation rates were similar between groups. Conclusion: Well-selected   
LT patients who had PVT prior to LT have similar post-LT outcomes with   
controls when adjusting for donor and recipient factors. Subgroups of PVT LT   
patients who did worse post-LT (complete thrombosis pre-LT, thrombectomy at   
LT and reocclusion post-LT) warrant closer evaluation in listing and   
management post-LT. Adjusted survival (Cox) for PVT LT recipients vs.   
controls (p=0.67). (Figure Presented).  
  
RECORD 262  
Venous diseases a case of fatal cerebral venous thrombosis in familial   
mediterranean fever  
Rha J. Park H. Yoon H. Choi J. Ahn J. Kwon J. Kim S.  
International Journal of Stroke (2014) 9 SUPPL. 3 (329). Date of   
Publication: October 2014  
  
Introduction and aims: Familial Mediterranean fever is a rare genetic   
autoinflammatory disease with recurrent fever and inflammation.Various   
neurological manifestations have been reported in the literature, but   
cerebral venous thrombosis has never been described. Methods and results   
(case description): A 23-year-old male patient visited our emergency   
department on August 27, 2013, with the chief complaint of right arm   
weakness. He had many previous episodes of recurrent fever and arthralgia   
since childhood, and had been diagnosed as familial Mediterranean fever with   
MEFV gene identification. One year ago, he experienced portal vein   
thrombosis, which was improved with anticoagulation. Initial brain CT   
revealed bilateral multiple intracranial hemorrhages along the superior   
sagittal sinus, more severe in left. Severe thrombocytopenia was also   
noticed, probably due to the splenomegaly which was previously diagnosed.   
Under the impression of superior sagittal sinus thrombosis, MR and catheter   
venography was performed, which showed complete obliteration of superior   
sagittal sinus. Intravenous anticoagulation with high dose heparin soon   
started, but activated partial thromboplastin time was not promptly   
prolonged, and his status rapidly progressed with brain swelling despite   
intracranial pressure lowering treatment. Seizure followed and more   
intracranial hemorrhages developed in follow-up brain CT. He was sentenced   
to brain death after 3 days, and expired 1 week later. Conclusions: This   
familial Mediterranean fever patient was characterized with recurrent   
thrombotic spell, one of which resulted in fatal cerebral venous thrombosis,   
which we describe first time.  
  
RECORD 263  
Hypercoagulability in cirrhotic patients with hepatocellular carcinoma (HCC)   
and portal vein thrombosis (PVT)  
Zanetto A. Ferrarese A. Rodriguez K.I. Pepe V. Fadin M. Radu C.M. Gavasso S.   
Vitale A. Cillo U. Farinati F. Russo F.P. Germani G. Nadal E. Simioni P.   
Burra P. Senzolo M.  
Hepatology (2014) 60 SUPPL. 1 (861A). Date of Publication: October 2014  
  
Background and aim: studies which explore the hypercoagulable state   
associated with neoplastic disease and its correlation with the risk of   
developing PVT in patients with HCC are lacking. The aim of the present   
study was to evaluate the thrombophilic role of HCC in cirrhotics with and   
without HCC and in controls and to correlate the presence of HCC and the   
coagulation profile with the incidence of PVT. Methods: cirrhotic patients   
with and without HCC were prospectively enrolled in the study. Age- and   
sex-matched healthy individuals constituted the control group for   
thromboelastometry (ROTEM). All cirrhotic patients with and without HCC   
underwent: ROTEM, platelet count, determination of prothrombin time and of   
levels of pro and anticoagulation factors. During follow-up, PVT onset in   
both patients with and without HCC was recorded. Results: 76 cirrhotics, 41   
with HCC and 35 without HCC, were included. Forty-eight healthy volunteers   
were included as the control group. Volume of active HCC was >5 cm3 in 18   
patients. Levels of pro and anticoagulation factors were similar between   
patients with and without HCC, but fibrinogen was increased in HCC patients   
with active volume >5cm3 HCC compared to those with <5cm3HCC bulk   
(348,72mg/dL±124,06mg/ dL vs 237,64mg/dL±99,18mg/dL) and to cirrhotics   
without HCC (260,57mg/dL±126,07mg/dL) (p=0,006). Platelet count was   
significantly increased in HCC patients compared to non- HCC patients, and   
this was especially true in Child Class A subjects. Patients with HCC showed   
significantly lower clotting formation time (CFT) and maximum clot formation   
(MCF) at ROTEM compared to healthy controls. The hypercoagulable state was   
present even when HCC patients were compared to cirrhotics without HCC, and   
was more evident when performing a subgroup analysis of Child Class A   
patients, with statistically significant differences in MCF EXTEM, MCF NATEM   
e CFT NATEM. During the 12 months follow-up there were 14 PVT episodes (10   
in HCC and 4 in non HCC group). At Cox multivariate analysis HCC and   
fibrinogen test of ROTEM were independently associated with risk of   
developing PVT. In the HCC group, 5/10 PVT occurred in patients in Child   
Class A. At FIBTEM test of ROTEM, MCF and AUC were statistically greater in   
HCC patients who later developed PVT. Conclusions: cirrhotics with HCC   
demonstrate a prothrombotic hemostatic balance resulting in an increased   
risk of PVT development. ROTEM seems to be a sensitive method to identify   
hypercoagulability, that would otherwise be undetected by routine laboratory   
testing. This prothrombotic state seems to be detectable by ROTEM and thus   
possibly suggest those who could benefit from thromboprophylaxis.  
  
RECORD 264  
Imbalance of pro-vs. Anti-coagulation factors in Chinese patients with   
Budd-Chiari syndrome and non-cirrhotic portal vein thrombosis  
Chen H. Liu L. Qi X.S. He C.Y. Yin Z.X. Wu F.F. Fan D.M. Han G.H.  
Journal of Digestive Diseases (2014) 15 SUPPL. 1 (81). Date of Publication:   
October 2014  
  
Background and aims The coagulation abnormalities of non-cirrhotic   
Budd-Chiari syndrome (NC-BCS) and portal vein thrombosis (NC-PVT) have not   
been extensively investigated in Chinese patients. To explore these   
coagulation imbalances and other associated influential factors, we   
conducted a case-control study. Methods We measured the levels of factors   
II, V, VII, VIII, IX, X, XI, XII, protein C (PC), protein S (PS) and   
antithrombin (AT) in blood samples from 37 NC-BCS patients, 98 NC-PVT   
patients, and 100 healthy controls. Results Compared with healthy controls,   
factor VIII was significantly elevated; factor II, V, VII, X, XI, XII, PC   
and AT were significantly decreased for both NC-BCS and NC-PVT; no   
differences were observed for PS of NC-BCS and for factor IX and PS of   
NC-PVT. Factor VIII-to-PC and factor VIII-to-AT were significantly increased   
for both NC-BCS and NC-PVT; factor VIII-to-PS was only significantly   
increased for NC-PVT; other ratios either significantly reduced or did not   
show any difference. No differences were observed for pro- and   
anti-coagulation factors or the ratios between them for different types of   
NC-BCS. Almost no coagulation factors had a moderate correlation with liver   
function for NC-BCS and NC-PVT. For correlations between coagulation factors   
and spleen size or platelet count, nearly no associations were observed for   
NC-BCS, and there were some correlations for NC-PVT. Conclusions The present   
study demonstrated the spectrum of coagulation imbalance for NC-BCS and   
NC-PVT in Chinese patients. The development of NC-BCS and NC-PVT might be   
associated the hypercoagulability resulting from increased factor VIII and   
decreased PC and AT.  
  
RECORD 265  
Balloon-occluded retrograde transvenous obliteration (BRTO) for the   
treatment of refractory hepatic encephalopathy  
Waller L. Jafri S.-M. Prushani A. Schwartz S. Moonka D.  
American Journal of Gastroenterology (2014) 109 SUPPL. 2 (S360). Date of   
Publication: October 2014  
  
Introduction: Hepatic encephalopathy (HE) develops in up to 50% of patients   
with decompensated cirrhosis. Balloon-occluded retrograde transvenous   
obliteration (BRTO) has been shown to be effective in controlling gastric   
variceal bleeding. We describe a case of BRTO for treatment of refractory   
hepatic encephalopathy (HE). Case Report: We describe here a 71-year-old   
woman with past medical history of cryptogenic cirrhosis, likely secondary   
to NASH, COPD on home oxygen, and coronary artery disease, who had recurrent   
admissions for HE despite standard of care treatment. The patient was not a   
transplant candidate because of her cardiac and pulmonary comorbidities.   
From June 2012 to June 2013, she had 12 admissions for hepatic   
encephalopathy. No precipitating factors were initially identified. A   
non-occlusive portal vein thrombosis was found in July 2012. anticoagulation   
was initiated without change. The patient continued to suffer from repeated   
bouts of HE despite medical therapy. Abdominal cross-sectional imaging   
identified a large splenorenal shunt. She underwent a balloon retrograde   
transvenous obliteration of a splenorenal shunt in June 2013, and had marked   
reversal of encephalopathy. At 12 months' follow-up, she had no recurrence   
of her HE. MELD score decreased slightly in spite of intervention.   
Discussion: HE can be precipitated by noncompliance, infection,   
gastrointestinal bleeding, medications, over diuresis, or other inciting   
factors. All patients should be evaluated for secondary triggers of HE.   
Treatment should be initiated with a non-absorbable disaccharide (ie,   
lactulose). Rifaximin can be added in patients not responding to lactulose.   
Most patients improve after correction of precipitants and medical therapy.   
Large portosystemic shunts may be embolized in patients with medically   
refractory, recurrent, or severe HE. The BRTO procedure is performed often   
in Asia for the management of gastric varices. BRTO has advantages over TIPS   
in that it is less invasive and can be performed on patients with poor   
hepatic reserve and those with encephalopathy. We describe an unusual case   
of the use of this therapeutic modality for the successful management of   
hepatic encephalopathy due to a splenorenal shunt. Conclusion: Refractory   
hepatic encephalopathy is difficult to manage, particularly in patients who   
are not transplant candidates. BRTO is a technique that can be used to   
effectively treat HE by occluding large mesenteric-systemic shunts without   
sacrificing hepatic function. We describe a patient in whom BRTO was an   
effective technique to treat her refractory hepatic encephalopathy caused by   
a splenorenal shunt.  
  
RECORD 266  
Successful treatment of partial portal vein thrombosis (PVT) with low dose   
rivaroxaban  
Lenz K. Dieplinger B. Buder R. Piringer P. Rauch M. Voglmayr M.  
Zeitschrift fur Gastroenterologie (2014) 52:10 (1175-1177). Date of   
Publication: 1 Oct 2014  
  
Abstract In a 63-year-old cirrhotic patient, recanalisation of a partial   
portal vein thrombosis was achieved by a low dose of rivaroxaban (10 mg   
daily). After anticoagulant therapy was stopped, partial vein thrombosis   
recurred. Restarting rivaroxaban at a dose of 10 mg led to recanalisation.   
The patient did not suffer any complications; in particular no bleeding   
occurred during 8 months of treatment.  
  
RECORD 267  
A rare case of hepatocellular carcinoma with self-embolization and   
regression of tumor  
Agrawal K. Al Mardini N. Agrawal K.  
American Journal of Gastroenterology (2014) 109 SUPPL. 2 (S176). Date of   
Publication: October 2014  
  
Case Report: This is a 70-year-old female who presented with right upper   
quadrant abdominal pain. On computerized tomography, a large right hepatic   
lobe mass extending directly into the portal vein was identified. MRI   
abdomen confirmed the extension of thrombosis into the right portal vein.   
Alphafetoprotein was elevated at 62,148 ng/mL. Liver biopsy was done later   
and showed findings consistent with hepatocellular carcinoma. Although she   
had Child-Pugh class A, she declined radioembolization and systemic   
treatment, so she enrolled in hospice. The patient didn't receive   
anticoagulation.During the following 4 months, the patient improved   
clinically with alleviation of her abdominal pain. She revoked hospice and   
had CT scan abdomen done, which showed spontaneous regression of her liver   
mass. Her Alpha-fetoprotein decreased significantly to 2448.24 ng/mL. Two   
months later, the patient continues to do well. The patient did not receive   
any forms of liver cancer treatments. This is a likely case of   
selfembolization of the hepatocellular carcinoma leading to spontaneous   
regression. Discussion: Advanced stages of hepatocellular carcinoma carry   
very poor prognosis. portal vein thrombosis in hepatocellular carcinoma is   
associated with poor prognosis as it limits the blood supply to normal liver   
parenchyma. However, we postulate that in some rare cases like ours, tumor   
thrombosis can cause self-embolization of the tumor itself. The other   
alternative explanation for the spontaneous regression of the tumor could be   
that the immune system was able to control the tumor growth. We present this   
rare case of self-embolization of hepatocellular carcinoma to highlight the   
importance of follow-up patients while they are on hospice to detect   
potential regression in the tumor burden, differentiating between bland and   
tumor thrombosis to guide the anticoagulation therapy and the need for   
further studies in this subject to develop further treatment options and   
predict the prognosis of similar hepatocellular carcinomas.  
  
RECORD 268  
A multicenter survey of the efficacy and safety of danaparoid sodium   
treatment for portal vein thrombosis  
Ohtake T. Tsuji K. Kawanishi T. Machida T. Takagi H. Mezawa S. Yazaki Y.   
Shinomura Y. Kohgo Y.  
Hepatology (2014) 60 SUPPL. 1 (399A-400A). Date of Publication: October 2014  
  
[Background and aim] As a complication of cirrhosis, portal vein thrombosis   
(PVT) is a critical condition that worsens hepatic reserve function. The   
standard treatment is anticoagulation therapy with unfractionated heparin,   
low-molecular-weight heparin, or warfarin. Danaparoid sodium (DS) is a   
heparinoid anticoagulant. Here we retrospectively report the efficacy and   
safety of DS in the treatment of PVT. [Methods] This is a retrospective   
epidemiological study analyzing integrated clinical data of patients treated   
with DS for PVT. Six facilities in Hokkaido, Japan participated in this   
study. Patients with firsttime treatment from register data were included.   
Patient personal information was protected by the anonymizing method.   
[Results] Eighty-five patients [51 males, 34 females; median age, 66 years   
(35-85)] were analyzed. Thrombosis sites were the following: portal trunk   
only, 28 cases; portal trunk with intrahepatic branches, 17; mainly   
intrahepatic branches 36; and principal tributaries only (superior   
mesenteric vein or splenic vein), 4. The complications observed were liver   
cirrhosis in 65% cases. The etiology was HBV-associated in 17 cases,   
HCV-associated in 21, alcoholic liver disease in 17 both viral and alcohol   
in 4, autoimmune in 7, NASH-related in 4, and others in 15. Complication   
rate of hepatocellular carcinoma was 39%; furthermore, 47% patients were   
treated for esophageal varices. Child-Pugh class of patients was A in 41 and   
B + C in 44 cases. The duration of DS therapy was median 14 days (4-150).   
Total dose of DS was median 37,500 units (5,000-255,000). Therapeutic   
efficacy was complete resolution of thrombosis, 39%; residual thrombosis   
<50%, 33%; residual thrombosis ≥50%, 6%; unchanged 19%; and unknown, 3%.   
Univariate analysis revealed higher serum ammonia levels as a predictive   
factor of therapeutic efficacy of DS in clinical background and blood test   
before treatment. In addition, higher dosage of DS tended to have   
therapeutic efficacy. Two of 85 patients had adverse events: one had   
bleeding from esophageal ulcer after endoscopic variceal ligation and the   
other had thrombocytopenia. In the mean observation period of 747 days, 54   
patients survived and 31 died. The efficacy of DS therapy and no   
complication of HCC contributed to the cumulative survival by Kaplan-Meier   
curve (p = 0.036 and 0.007, respectively). [Conclusions] In all, 72%   
patients with PVT treated with DS had complete resolution of thrombosis or   
<50% residual thrombosis. No serious adverse events were observed. Efficacy   
of DS therapy contributed to the cumulative survival. These results support   
the efficacy and safety of DS in the treatment of PVT.  
  
RECORD 269  
Inherited Thrombophilia and the Risk of portal vein thrombosis: Progress   
Toward Individualized anticoagulation in cirrhosis?  
Fallon M.B. Batra S.  
Clinical Gastroenterology and Hepatology (2014)  
  
RECORD 270  
Efficacy and safety of the anticoagulant drug, danaparoid sodium, in the   
treatment of portal vein thrombosis in patients with liver cirrhosis  
Naeshiro N. Aikata H. Hyogo H. Kan H. Fujino H. Kobayashi T. Fukuhara T.   
Honda Y. Nakahara T. Ohno A. Miyaki D. Murakami E. Kawaoka T. Tsuge M.   
Hiraga N. Hiramatsu A. Imamura M. Kawakami Y. Ochi H. Chayama K.  
Hepatology Research (2014). Date of Publication: 2014  
  
Aim: To assess the efficacy and safety of the anticoagulant drug, danaparoid   
sodium, in the treatment of portal vein thrombosis (PVT) in patients with   
liver cirrhosis. Methods: A consecutive 26 cirrhotic patients with PVT were   
enrolled in this retrospective cohort study. The etiologies of cirrhosis   
were hepatitis B virus-related, hepatitis C virus-related, alcoholic and   
cryptogenic in five, 14, three and four patients, respectively. Child-Pugh   
grade A, B and C was noted in 13, eight and five patients, respectively.   
Patients were treated with 2 weeks' administration of danaparoid sodium   
followed by the evaluation of PVT reduction and adverse events. Results: All   
patients experienced reduction of PVT through the treatment. The median   
volume of PVT before and after treatment was 2.40cm(3) (range, 0.18-16.63)   
and 0.37cm(3) (range, 0-5.74), respectively. The median reduction rate of   
PVT volume was 77.3% (range, 18-100%). According to the reduction rate,   
complete reduction (CR), partial reduction (PR, ≥50%) and stable disease   
(SD, <50%) were observed in four (15%), 16 (62%) and six patients (23%),   
respectively. The median volume of PVT before treatment was significantly   
different between CR+PR and SD (2.09 vs 4.35cm(3), P=0.045). No severe   
adverse events such as bleeding symptoms (e.g. gastrointestinal bleeding and   
cerebral hemorrhage) and thrombocytopenia were encountered. Conclusion:   
Danaparoid sodium for the treatment of PVT in patients with liver cirrhosis   
was safe and effective. Therefore, anticoagulation therapy with danaparoid   
sodium could have potential as one of the treatment options in PVT   
accompanied by cirrhosis. © 2014 The Japan Society of Hepatology.  
  
RECORD 271  
Treatment of portal vein obstruction  
Keussen I.  
CardioVascular and Interventional Radiology (2014) 37:2 SUPPL. 1   
(S109-S110). Date of Publication: September 2014  
  
Learning Objectives 1. To review the aetiology of portal vein thrombosis 2.   
To describe the methods in chronic portomesenteric vein thrombosis   
recanalisation 3. To compare the results of medical and endovascular therapy   
in acute portal vein thrombosis Portal vein (PV) obstruction (PVO) is a   
relatively rare condition, which may have serious consequences (1,2). The   
most common cause is PV thrombosis. PV thrombosis in children is frequently   
caused by umbilical vein catheterization with secondary infection, and less   
frequently parasitosis. In adults, PV thrombosis is most frequent in   
patients with cirrhosis, but may be secondary to coagulation disorders,   
malignancy, infection, inflammatory diseases, external compression, or a   
combination of these entities. Esophageal varices may appear as soon as 1   
month after the first symptoms of PV thrombosis (3). Idiopathic PVO may also   
be present (4). PVO may be either prehepatic or intrahepatic, include both   
localizations, or extend to more peripheral branches. Prehepatic occlusion   
is usually secondary to acute or chronic thrombosis or to malignant disease.   
Diagnosis is usually established using ultrasonography, computed tomography,   
or magnetic resonance tomography. Multiple collaterals as in cavernous   
transformation of PV or gastrointestinal, mesenteric, or subcutaneous   
varices may be detected on these examinations. If the intrahepatic PV   
branches are patent, the pressure gradient between these branches and the   
systemic circulation is low. In chronic PVO, pressure gradient between   
intrahepatic and open part of PV may be relatively low if the patient has   
welldeveloped collaterals. If only the splenic vein is obstructed with   
elevated pressure gradient, the condition is called “left-sided portal   
hypertension.” In case of acute PVO, the patient presents with symptoms of   
abdominal disorder with or without gastrointestinal bleeding. In case of   
chronic PVO, symptoms may be more diffuse and often include ascites,   
splenomegaly, chronic abdominal pain, and/or intermittent gastrointestinal   
bleeding. Asymptomatic PVO may be found accidentally and in most cases, does   
not require further attention. Depending on the severity of symptoms,   
anticoagulant therapy, endoscopic sclerotherapy, or tapping of ascites may   
be necessary. Surgical treatment methods include splenectomy, mesosystemic   
shunts, bowel resection, and liver transplant. Percutaneous interventional   
radiological (IR) options include the following (5): • Intraarterial   
thrombolytic therapy • Intraportal thrombolysis • Removal and fragmentation   
of the thrombus • Stent recanalization • Additional TIPS following the   
aforementioned methods • Partial splenic embolization • Variceal   
embolization The IR treatment is usually performed under general anesthesia,   
but in some cases, it may be performed under local anesthesia with systemic   
sedation. The patient's heart rate, blood pressure, and oxygen saturation   
should be monitored continuously. Smallest possible instruments should be   
used. Planning of the IR treatment should be based on the location and   
extent of PVO. In case of extrahepatic PVO, percutaneous transhepatic or   
transjugular transhepatic, similar to the TIPS technique, should be used   
(6). If the intrahepatic PV branches are occluded, transhepatic access may   
be difficult or impossible. In these cases, trans-splenic access may be   
preferred. For a percutaneous transhepatic or trans-splenic access, a   
micropuncture technique is recommended. When access to PV has been   
established, venography is performed using ionic contrast or CO(2). The   
pressure gradient is assessed, and recanalization of the obstructed segment   
is attempted. The occluded segment can usually be traversed using   
hydrophilic guidewire and standard angiographic catheters. Thrombolysis with   
or without thrombus fragmentation/aspiration may be attempted in cases of   
acute/subacute thrombosis. Use of different tools intended for the treatment   
of arterial or venous thrombosis may be beneficial. Stent or stent-grafts   
are used to stabilize the recanalized segment if necessary (7-9). Additional   
embolization of varices may be performed with an aim to increase the flow to   
the recanalized segment. After the procedures, the transhepatic or   
trans-splenic tract should be embolized with coils, plugs, and/or gelatine   
sponge. Intraarterial thrombolysis with an infusion of thrombolytics in SMA   
may be tried in cases of acute PV thrombosis. Another type of treatment is   
partial splenic embolization, which may also be performed in order to   
decrease the inflow of blood to the portal system and/or decrease symptoms   
(5). The results depend on the origin of PVO. Recanalization of the occluded   
segments has a relatively good outcome if intrahepatic PV branches are   
patent. Otherwise, additional TIPS may be necessary (10,11). In patients   
with malignant PV invasion, the outcome after stent placement is less   
favorable if the splanchnic veins are involved and/or if severe hepatic   
dysfunction is present (8). The treatment in children should be adapted to   
the age and size of the patient (12,13). Due to the relative rarity of PVO,   
comparison of results of different treatment methods is difficult, as is   
planning of randomized studies. It was reported that the recanalization rate   
was higher in patients receiving anticoagulation compared to no treatment at   
all (3). In reports describing endovascular treatment, the results were   
generally promising (5). PVO is a serious condition, which may cause   
life-threatening bleeding or bowel ischemia. Imaging can define the extent   
and localization of PVO. The interventions provided by IR should be based on   
the decision of a multidisciplinary team. Possibility of future liver   
transplant should be discussed, if stents or stent-grafts are used. The IR   
treatment usually has a good outcome, but should be planned carefully   
according to the extent and localization of PVO, hemodynamic flow pattern,   
causality, and vascular anatomy.  
  
RECORD 272  
A case report: Venous infarction of the spleen. A rare and unexpected   
sequelae of portal vein thrombosis. An incidental finding and treatment   
dilemma  
Sokolowsky A. Tan B.  
Journal of Medical Imaging and Radiation Oncology (2014) 58 SUPPL. 1 (321).   
Date of Publication: September 2014  
  
We present a case of a 75-year-old female who presented to a rural emergency   
department following a four day history of left upper quadrant abdominal   
pain. There was no history of recent trauma, only a background of   
diverticulosis and bronchiectasis. Computed tomography (CT) initially showed   
multiple hypodense wedge shaped splenic lesions consistent with a mixed age   
splenic infarct and a peri-splenic collection. There was no evidence of   
splenic arterial disease or aneurysm. A thrombus within the splenic vein was   
incidentally seen. The patient was extensively investigated for underlying   
haematological conditions, collagen vascular disease, occult malignancy and   
infective endocarditis. None could be found. A diagnosis of venous   
infarction of the spleen was therefore made, an exceedingly rare, but not   
unheard of entity. The patient was treated with therapeutic anticoagulation,   
but subsequently had a turbulent clinical course, necessitating multiple   
admissions and further radiological investigations. The patient is currently   
on ongoing outpatient management.  
  
RECORD 273  
Mesenteric vein thrombosis; not going with the flow  
Lee L.Y.W. Aubrey-Jones H. Lacey R. De Silva A.  
BMJ Case Reports (2014). Date of Publication: 21 Aug 2014  
  
A 71-year-old woman presented with a 2-week history of epigastric pain,   
nausea and vomiting; on examination she demonstrated signs of peritonism. CT   
imaging was performed and this demonstrated extensive thrombosis of the   
superior mesenteric, omental and portal veins with infarction of the distal   
small bowel. A non-operative approach was initiated and anticoagulation   
rapidly started. Within 48 h the patient demonstrated significant clinical   
improvement and she subsequently made a full recovery. Copyright 2014 BMJ   
Publishing Group. All rights reserved.  
  
RECORD 274  
Efficacy of postoperative anticoagulation therapy with enoxaparin for portal   
vein thrombosis after hepatic resection in patients with liver cancer  
Yamashita Y.-i. Bekki Y. Imai D. Ikegami T. Yoshizumi T. Ikeda T. Kawanaka   
H. Nishie A. Shirabe K. Maehara Y.  
Thrombosis Research (2014)  
  
Backgrounds: Enoxaparin, low-molecular-weight heparin, has become a routine   
thromboprophylaxis in general surgery. Study design: A retrospective cohort   
study was performed in 281 patients who underwent hepatic resections for   
liver cancers from 2011 to 2013. These patients were divided into two   
groups; an enoxaparin (-) group (n = 228) and an enoxaparin (+) group (n =   
53). Short-term surgical results including venous thromboembolism (VTE) and   
portal vein thrombosis (PVT) were compared. Results: In the enoxaparin (+)   
group, the patients' age (65 vs. 69 years; p = 0.01) and BMI (22.9 vs. 24.4;   
p < 0.01) were significantly higher. According to the symptomatic VTE,   
symptomatic pulmonary embolism occurred in one patient (0.4%) in the   
enoxaparin (-) group, but the complication rate was not significantly   
different (p = 0.63). The complication rate of PVT was significantly lower   
in the enoxaparin (+) group (10 vs. 2%; p = 0.04). The independent risk   
factors for PVT were an operation time ≥ 300 minutes (Odds ratio 6.66) and   
non-treatment with enoxaparin (Odds ratio 2.49). Conclusions: Postoperative   
anticoagulant therapy with enoxaparin could prevent PVT in patients who   
underwent hepatic resection for liver cancers. © 2014 Elsevier Ltd. All   
rights reserved.  
  
RECORD 275  
Risk factors for death on the waiting list for liver transplantation in   
patients with non-malignant portal vein thrombosis  
Iacob S. Ester C. Popescu I. Gheorghe L.  
Transplantation (2014) 98 SUPPL. 1 (707). Date of Publication: 15 Jul 2014  
  
Background: portal vein thrombosis (PVT) is relatively common in candidates   
for liver transplantation (LT) and long-term outcome of patients with PVT   
who undergo LT is not well defined. anticoagulation is a challenging therapy   
in individuals with decompensated liver cirrhosis because of the   
well-recognized coagulation abnormalities and of the increased risk of   
bleeding. Aim: To investigate the risk factors for overall and hemorrhage   
related death in a cohort of 88 cirrhotic patients with PVT included on the   
waiting list for LT. Methods: We tested separately the association between   
different parameters and overall death while on the waiting list using   
logistic regression. Results: There were 63.6% men with a mean age of   
52.4±10.5 years, 30.7% of patients had HCV and 35.2% had HBV-related   
cirrhosis. 39.8% of patients received anticoagulation and 38.2% of them had   
recanalization of the PVT, while 42.9% remained stable. Overall death was   
encountered in 26 patients (29.5%), out of whom 12 (46.2%) were   
hemorrhage-related. 13.6% of patients were transplanted. As independent risk   
factors for overall death were identified the following: MELD score at PVT   
diagnosis (p=0.01), associated superior mesenteric vein thrombosis   
(p=0.0001), refractory ascites with frequent paracentesis (p=0.003),   
complete occlusion of the portal vein (p=0.0001), lack of recanalization   
after 3 months of anticoagulation therapy (p=0.03), multiple band ligations   
(p=0.04). Hemorrhagerelated death was associated with the following risk   
factors: administration of anticoagulation therapy (p=0.004) and patient age   
(p=0.04). Conclusion: anticoagulation therapy is efficacious in   
recanalization or stabilization of the thrombotic process in patients with   
liver cirrhosis awaiting LT, but is associated with hemorrhage-related   
death.  
  
RECORD 276  
Successful treatment of diffuse portal vein thrombosis after splenectomy   
following living donor liver transplantation patient  
Kang S. Hwang S. Lee S. Shin M. Yoon Y. Choi E. Kwon J. Kim W. Song G. Park   
G.  
Transplantation (2014) 98 SUPPL. 1 (797-798). Date of Publication: 15 Jul   
2014  
  
Splenectomy is performed after living donor liver transplantation(LDLT) for   
various reasons.portal vein thrombosis(PVT) is rare but dreaded complication   
after splenectomy in LDLT recipients that can compromise patient and graft   
survival. We recently experienced a case of acute and diffuse PVT after   
splenectomy in LDLT recipient who was successfully treated with thrombectomy   
and anticoagulation therapy.The patient was a 56-year-old female who   
underwent LDLT using modified right lobe graft on June 2, 2006. Recently she   
developed thrombocytopenia and splenomegaly. We performed splenectomy to   
resolve thrombocytopenia. On postoperative fifth day, she complained pain on   
her left shoulder. A CT scan showed diffuse portal vein thrombosis. (Figure   
Presented) The patient was taken immediately to the operating room. We   
opened splenic vein stump and Fogarty thrombectomy was attempted under   
intra-operative ultrasound guiding. After thrombectomy, portogram revealed   
recanalization of the splenic vein and main portal vein but still remained   
intra-hepatic PVT. An interventional radiologist put the McNamara   
thrombectomy catheter into intra-hepatic portal vein via inferior mesenteric   
vein. After several times of aspiration thrombectomy, portogram showed   
completed recanalization of intra-hepatic portal vein. We put the stent into   
spleno-mesenteric junction to prevent recurrent PVT. (Figure Presented)   
Systemic heparinization was started immediately after operation and was   
converted warfarin and antiaggregation therapy. A postoperative Doppler   
ultrasound and CT scan showed patent portal vein.This case showed that PVT   
after splenectomy can be treated with surgical thrombectomy, intra-operative   
interventional procedure and anticoagulation therapy.Routine.  
  
RECORD 277  
Early pancreas thrombosis (Within 90 Days) after solitary pancreas   
transplants: A comprehensive study of the incidence, outcomes and risk   
factors  
Patil V. Welsch B. Leverson G. Sollinger H. Kaufman D. Odorico J.  
Transplantation (2014) 98 SUPPL. 1 (861). Date of Publication: 15 Jul 2014  
  
Background: Graft thrombosis is a dreaded complication of pancreas   
transplantation directly impacting graft survival. As the majority of   
published studies focuses on simultaneous pancreas transplants and use   
varied defi nitions, it is diffi cult to draw inferences about the true   
incidence and the factors impacting early graft thrombosis (within 90 days)   
in solitary pancreas transplants (SPTx) .Aim: The aim of the study was to   
identify the clinically relevant incidence of early graft thrombosis in SPTx   
and summarize the risk factors. Methods: Between 1997 and 2012, 192 SPTx   
were performed at our center. Patients were diagnosed as either partial   
thrombosis (nonocclusive thrombus in the main iliac artery Y graft or portal   
vein, thrombosis of the main splenic vein,splenic artery), or complete   
thrombosis (complete occlusion of the iliac Y graft, portal vein) or no   
thrombosis (no evidence of thrombosis, small distal splenic vein   
thrombus)Results:11.5% of patients demonstrated either partial or complete   
pancreas graft thrombosis within 90 days of transplant.64% of which were   
patients with complete graft thrombosis (portal vein 51%, iliac artery Y   
graft 36%); 36% had partial graft thrombosis (portal vein 87%, iliac artery   
Y graft 13%). The median time to detection of thrombosis was 4 days and 15   
days for complete and partial early graft thrombosis, respectively. 59% of   
early graft thrombosis required pancreatectomy for complete graft   
thrombosis. 23% of the early graft thrombosis received anticoagulation (80%   
partial thrombosis, 20% complete thrombosis) 18%received No treatment .The   
median graft survival for patients with early complete graft thrombosis was   
5 days and for early partial thrombosis was 72 days. (Graph presented) 50%   
of the patients with early partial thrombosis were treated with   
anticoagulation and the rest of the 50% received no treatment.Conclusion:   
This study identifi es the clinically relevant incidence of early thrombosis   
in SPTx and helps to gain insight into the various factors that contribute   
to it and reports on the outcome of different treatments.  
  
RECORD 278  
Splanchnic and extrasplanchnic thrombosis in cirrhosis: Prophylaxis vs   
treatment  
Nery F. Valla D.  
Current Hepatitis Reports (2014) 13:3 (224-234). Date of Publication: 1 Jul   
2014  
  
Venous thromboembolism (deep vein thrombosis and pulmonary embolism) and   
portal vein thrombosis (PVT) occur in up to 6.3 % and 15.9 % of patients   
with cirrhosis, respectively. There is recent evidence that a procoagulable   
prothrombotic state is related to cirrhosis despite the reduced levels of   
many coagulation factors, and decreased platelet counts. Indeed, (i) the   
combination of high levels of factor VIII, with low levels of protein C and   
antithrombin induces a procoagulant state in vitro; while (ii) increased   
levels of von Willebrand factor and decreased ADAMTS 13 activity can   
compensate for decreased platelet counts. PVT is partial in a majority of   
patients in whom it develops and may spontaneously resolve in some of them.   
Although PVT is associated with features of more severe liver disease, it is   
uncertain whether it plays a causal role in the decompensation of cirrhosis.   
In patients listed for liver transplantation, PVT may make the procedure   
difficult or impossible. Pretransplant PVT is associated with increased   
post-transplant mortality rates. Studies evaluating clinical outcome of   
anticoagulation therapy for splanchnic or extrasplanchnic venous thrombosis   
are scarce. anticoagulation therapy, given to patients with cirrhosis of   
intermediate severity before PVT occurrence, in prophylactic doses, appears   
to decrease decompensation and mortality rate. Interestingly, this   
improvement is out of proportion of the prophylaxis of extrahepatic portal   
vein thrombosis. The risk of bleeding does not seem to be increased in   
patients with cirrhosis receiving anticoagulation therapy, once prophylaxis   
for bleeding related to portal hypertension has been implemented. Overall,   
the room for anticoagulation therapy is probably larger than previously   
recognized, and may be of particular benefit in patients without portal vein   
thrombosis. However, clinical trials remain to be done before the benefit   
risk ratio of anticoagulation therapy is properly evaluated.  
  
RECORD 279  
Successful treatment of diffuse portal vein thrombosis after splenectomy   
following living donor liver transplantation patient  
Kang S.-H. Hwang S. Lee S.-G. Choi E. Kwon J.-H.  
Liver Transplantation (2014) 20 SUPPL. 1 (S216-S217). Date of Publication:   
June 2014  
  
Splenectomy is performed after living donor liver transplantation(LDLT) for   
various reasons.portal vein thrombosis(PVT) is rare but dreaded complication   
after splenectomy in LDLT recipients that can compromise patient and graft   
survival. We recently experienced a case of acute and diffuse PVT after   
splenectomy in LDLT recipient who was successfully treated with thrombectomy   
and anticoagulation therapy.The patient was a 56-year-old female who   
underwent LDLT using modified right lobe graft on June 2, 2006. Recently she   
developed thrombocytopenia and splenomegaly. We performed splenectomy to   
resolve thrombocytopenia. On postoperative fifth day, she complained pain on   
her left shoulder. A CT scan showed diffuse portal vein thrombosis. (Figure   
presented) The patient was taken immediately to the operating room. We   
opened splenic vein stump and Fogarty thrombectomy was attempted under   
intra-operative ultrasound guiding. After thrombectomy, portogram revealed   
recanalization of the splenic vein and main portal vein but still remained   
intra-hepatic PVT. An interventional radiologist put the McNamara   
thrombectomy catheter into intra-hepatic portal vein via inferior mesenteric   
vein. After several times of aspiration thrombectomy, portogram showed   
completed recanalization of intra-hepatic portal vein. We put the stent into   
spleno-mesenteric junction to prevent recurrent PVT. (Figure presented)   
Systemic heparinization was started immediately after operation and was   
converted warfarin and antiaggregation therapy. A postoperative Doppler   
ultrasound and CT scan showed patent portal vein.This case showed that PVT   
after splenectomy can be treated with surgical thrombectomy, intra-operative   
interventional procedure and anticoagulation therapy.  
  
RECORD 280  
Clinical presentations, risk factors, treatment and outcomes in patients   
with splanchnic vein thrombosis  
De Sancho M. Shillinford K. Chapin J.  
American Journal of Hematology (2014) 89:6 (E34). Date of Publication: June   
2014  
  
Background: Splanchnic vein thrombosis (SVT) is an unusual form of venous   
thrombosis that affects the hepatic, portal, mesenteric and splenic veins.   
Risk factors for SVT include liver cirrhosis, inflammatory and autoimmune   
diseases, post-operative state (mainly after abdominal surgery), congenital   
anatomical abnormalities, JAK2 positive myeloproliferative neoplasms (MPN),   
paroxysmal nocturnal hemoglobinuria (PNH), malignancies, inherited and   
acquired thrombophilia, female hormonal therapy, pregnancy and puerperium.   
The management of SVT is challenging and depends on the underlying risk   
factor, manner or presentation and risk factors for bleeding. Objective: To   
evaluate the clinical presentations, risk factors, treatment modalities and   
outcomes in patients with SVT referred to our hematology clinic at a   
tertiary care center. Methods: Electronic medical records were reviewed from   
29 consecutive patients referred to our hematology clinic for management of   
SVT from January 2006 to December 2013. Data collected included age at   
presentation, gender, ethnicity, and location of thrombosis (hepatic,   
portal, mesenteric, splenic or combined). Splanchnic vein thrombosis risk   
factors were evaluated. Treatment modalities including anticoagulation,   
thrombolytic therapy, thrombectomy, and trans jugular portosystemic shunt   
(TIPS) and spleno-renal shunt were reviewed. Clinical outcomes of interest   
were improvement or resolution of thrombosis, recurrent thrombosis,   
bleeding, and mortality. Results: We identified 29 patients (15 females and   
14 males). The mean age was 44 years (range: 18 -71). There were 13 patients   
with portal vein thrombosis, one of whom had extension to the inferior vena   
cava; eight combined thrombosis, four hepatic vein thrombosis, three   
mesenteric vein thrombosis and one splenic vein thrombosis. Of these 29   
patients, four had liver cirrhosis, five had inflammatory/autoimmune   
conditions, six had JAK2-positive MPNs, five were using female hormones, one   
was post-partum and one had a congenital anatomical vascular abnormality. In   
terms of thrombophilia, four were heterozygote carriers of prothrombin gene   
mutation G20210A (PGM) and one was a carrier of Factor V Leiden (FVL). Four   
had antiphospholipid antibodies (aPLs). Twenty-five patients were placed on   
anticoagulation, one patient had a TIPS, and one patient had a splenorenal   
shunt. Two patients had recurrent thrombosis; six patients had major   
bleeding events, including one fatal subdural hematoma. Two patients died   
one after complications of subdural hematoma and the other after a bone   
marrow transplant performed after he developed acute leukemia. Conclusions:   
A wide variety of thrombotic risk factors contribute to splanchnic vein   
thrombosis. The most common associated finding with SVTs was JAK2 mutations.   
Bleeding is a major complication in the setting of SVT. Multidisciplinary   
approaches are needed to optimize the care of these patients.  
  
RECORD 281  
Clinical presentations, risk factors, treatment and outcomes in patients   
with splanchnic vein thrombosis  
De Sancho M. Shellingford K. Chapin J.  
American Journal of Hematology (2014) 89:6 (E60-E61). Date of Publication:   
June 2014  
  
Background: Splanchnic vein thrombosis (SVT) is an unusual form of venous   
thrombosis that affects the hepatic, portal, mesenteric and splenic veins.   
Risk factors for SVT include liver cirrhosis, inflammatory diseases,   
post-operative state (mainly after abdominal surgery), JAK2 positive   
myeloproliferative neoplasms (MPN), paroxysmal nocturnal hemoglobinuria   
(PNH), malignancies, thrombophilias such as factor V leiden (FVL),   
prothrombin gene mutation (PGM) and antiphospholipid syndrome (APLs), female   
hormonal therapy, pregnancy and puerperium. The management of SVT is   
challenging and depends on the underlying risk factor, manner or   
presentation and risk factors for bleeding. Objective: To evaluate the   
clinical presentations, risk factors, treatment modalities and outcomes in   
patients with SVT referred to our hematology clinic at a tertiary care   
center. Methods: Electronic medical records were reviewed from 29   
consecutive patients referred to our hematology clinic for management of SVT   
from January 2006 to December 2013. Data collected included age at   
presentation, gender, ethnicity, location of thrombosis (hepatic, portal,   
mesenteric, splenic or combined), risk factors and treatment. Treatment   
modalities including anticoagulation, thrombolytic therapy, thrombectomy,   
and trans jugular porto-systemic shunt (TIPS) were reviewed. Clinical   
outcomes of interest were recurrent thrombosis, bleeding, and resolution.   
Results: We identified 29 patients (15 females and 14 males). The mean age   
was 44 years (range: 18 -71). There were 13 patients with portal vein   
thrombosis, one of whom had extension to the inferior vena cava, eight   
combined thrombosis, four hepatic vein thrombosis, three mesenteric vein   
thrombosis and one splenic vein thrombosis. Of these 29 patients, four had   
liver cirrhosis, five had inflammatory/ autoimmune conditions, six had   
JAK2-positive MPNs, five were using female hormones and one was post-partum.   
In terms of thrombophilia, four were heterozygote carriers of PGM and one   
was a carrier of FVL. Four had APLs. Twenty-five patients were placed on   
anticoagulation, One patient had a TIPS, one patient had a splenorenal   
shunt. Two patients had recurrent thrombosis, six patients had major   
bleeding events, including one fatal subdural hematoma. Two patients had   
recurrence of thrombosis. Conclusions: A wide variety of thrombotic risk   
factors contribute to splanchnic vein thrombosis. The most common associated   
finding with SVTs were JAK2 mutations. Bleeding is a major complication in   
the setting of SVT. Multidisciplinary approaches are needed to optimize the   
care of these patients.  
  
RECORD 282  
Budd chiari syndrome: Transplantation and beyond  
Pareek S. Gupta S. Goyal N. Wadhawan M. Vohra S.  
Liver Transplantation (2014) 20 SUPPL. 1 (S366-S367). Date of Publication:   
June 2014  
  
Introduction: Budd Chiari syndrome presents as a spectrum of vascular   
disease requiring liver transplantation as well as innovative techniques to   
establish inflow and outflow. With a vast experience in Living Related Liver   
Transplantation we looked into our data to share our experience in managing   
these patient. Materials and Method: At our centre we follow the stepwise   
protocol with anticoagulation, vascular intervention and finally liver   
transplantation for cirrhotic liver. All the patient undergo work-up for   
pro-coagulant state ( protein C, S; Leiden V, antithrombin III,   
anticardiolipin, antiphospholipid, Homocysteine assessment) along with   
screening for myeloproliferative disease (JAK-2 mutation )and CT liver angio   
gram. Results : Pre-operatively out of 12 patients we transplanted 4 had   
protein C and S deficiency. Indication of transplantation was   
Hepato-pulmonary syndrome, blocked MHV stent and cirrhosis. All patient   
underwent surgery by abdominal approach with porta dissection and ligation   
first technique. The operative time as well as the blood loss was higher as   
compared to controls requiring meticulous control of the collaterals. Five   
patient had portal vein thrombosis in addition to outflow obstruction   
requiring portal vein thrombectomy. Liver was explanted in all cases with   
individually clamping the hepatic veins and dividing them without the need   
for caval clamping. Associated thrombus in the inferior venacava was dealt   
with Thrombectomy, Thrombectomy and dilatation of the venacava, Cavatomy and   
interposition onlay graft to restore the lumen (7cm long and 1.5 cm wide   
circumferential Gortex graft around 180 degrees ) In one case spontaneous   
recannalisation of the Inferior venacava was observed. Post-operatively all   
the patients receive heparin for 2 weeks and are overlapped with Warfarin   
anticoagulation and all are doing well with median survival of 23 months   
Patients with protein C/S deficiency are off anticoagulation after 6 months   
of anticoagulation once the normal level of the deficient protein are   
ascertained rest of the patient are on Warfarin anticoagulation. Two patient   
required balloon dilatation of outflow tract due to non compliance of   
anticoagulation Conclusion: Liver transplantation for Budd chiari syndrome   
is feasible by innovative inflow and outflow restorative technique. Selected   
patient can be managed off anticoagulation. Non-compliant patient can be   
managed with invasive radiological interventions.  
  
RECORD 283  
Splancnic vein thrombosis associated with myeloproliferative neoplasms. A   
study of the IWG-MRT in 494 subjects  
Pieri L. Guglielmelli P. Primignani M. Randi M.L. Santarossa C. Cazzola M.   
Rumi E. Cervantes F. Ellis M. Chen F. Tripathi D. Rajoriya N. Barbui T.   
Delaini F. De Stefano V. Rossi E. Betti S. Harrison C. Curto Garcia N.   
Specchia G. Ricco A. Gisslinger H. Gisslinger B. Vianelli N. Nicola P.   
Ruggeri M. Girodon F. Tefferi A. Vannucchi A.M.  
Haematologica (2014) 99 SUPPL. 1 (128). Date of Publication: 1 Jun 2014  
  
Background: Philadelphia-negative Myeloproliferative Neoplasms (MPN) include   
Polycythemia Vera (PV), Essential Thrombocythemia (ET), Myelofibrosis both   
Primary (PMF) and secondary to PV and ET (PPV-, PET-MF) as well as   
unclassified MPN (U-MPN). An increased risk of venous thrombosis in unusual   
sites, ie splanchnic vessels (SVT), is particularly associated with MPN. SVT   
can lead to complications such as portal hypertension, esophageal and   
gastric varices, ascites and hepatic failure. A recent meta-analysis   
reported that a MPN is the underlying cause of portal vein thrombosis in   
31.5% and of Budd Chiari syndrome in 40.9% of cases (Smalberg, 2012);   
however analysis of disease characteristics and outcome has been hampered by   
heterogeneity of available patients' cohorts comprising relatively small   
number of cases. Aims: We conducted a retrospective multicenter study   
collecting clinical and biological data of patients (pts) with SVT   
associated with WHO2008-diagnosed MPN, with the aim to describe patients'   
characteristics, trends and prognostic factors that may have implications   
for clinical practice. Methods: Data were collected from 16 international   
hematologic centers in the framework of IWG-MRT. Results: A total of 494   
cases of portal, splenic or mesenteric vein thrombosis (75.2%) or Budd   
Chiari syndrome (24.8%) associated with MPN were collected. Current analysis   
refers to 475/494 cases, and final data will be presented at EHA meeting.   
Frequency of MPN associated subtype was 38.1% ET (n=181), 34.9% PV (n=166),   
16.2% MF (n=77), 10.8% U-MPN (n=51). Median follow-up 87.9 mo (range   
0.5-430); female 61.3% (n=292; P<0.0001 vs male); median age at MPN   
diagnosis (dg) 44.4 y (range 12-90), significantly younger than non-SVT   
associated MPN. In 229 cases (48%) MPN and SVT dg were coincident, while in   
104 (22%) SVT occurred before MPN dg (median 40 mo, range 5-335) and in 129   
(27%) during MPN follow up (median 79 mo, range 5-394). Biological featured   
included JAK2V617F mutation present in 99% PV, 84.7% ET, 88.1% PMF and 92.9%   
U-MPN pts, while erythropoietin-independent colonies (EEC) were present at   
diagnosis in 80/110 evaluated cases (72.7%), 38/47 PV (84.4%), 32/45 ET   
(71.1%), 8/11 PMF (72.7%) and 2/7 U-MPN (28.6%). A concurrent thrombophilic   
status was found in 38.9% of cases. Therapy after SVT included   
anticoagulation in 77% of pts, antiaggregant therapy in 23.5% and both in   
1.5%; 68.8% of pts received cytotoxic drugs, 11.4% were treated with trans   
jugular portosystemic shunt. No differences in survival were noted with   
these approaches. Beta blockers was used in 48.5% of pts and correlated with   
improved survival (p=0.041). At last follow up 70/473 pts (14.8%) died;   
causes of death are evolution to AL (16.4%), other cancers (14.5%), disease   
progression without AL (12.7%), SVT (10.9%), hepatic failure and venous   
thrombosis other than SVT (9.1% each), heart failure and arterial thrombosis   
(7.3% each), hemorrhage (5.5%), renal failure and infection (3.6% each).   
After 10 y follow up 8/166 PV (5%), 14/181 ET (8%), 14/77 PMF (18%) and 1/51   
U-MPN (1.96%) pts died (p<0.01). Survival was significantly affected by   
occurrence of thrombosis other than SVT (p<0.0001), that occurred in 35.8%   
of pts but not by recurrence in splanchnic vessels (p=0.068). Summary and   
Conclusions: This large study describes characteristics, therapeutic options   
and outcome of SVT associated with MPN, pointing to an overall good   
prognosis compared with non-SVT associated MPN and identified thrombosis in   
districts other than splancnic district as the leading cause of death,   
suggesting the need to potentiate antithrombotic therapy.  
  
RECORD 284  
portal vein thrombosis after hepatectomy  
Yoshiya S. Shirabe K. Nakagawara H. Soejima Y. Yoshizumi T. Ikegami T.   
Yamashita Y.-I. Harimoto N. Nishie A. Yamanaka T. Maehara Y.  
World journal of surgery (2014) 38:6 (1491-1497). Date of Publication: 1 Jun   
2014  
  
BACKGROUND: Although various complications after hepatectomy have been   
reported, there have been no large studies on postoperative portal vein   
thrombosis (PVT) as a complication. This study evaluated the incidence, risk   
factors, and clinical outcomes of PVT after hepatectomy.METHODS: The   
preoperative and postoperative clinical characteristics of patients who   
underwent hepatectomy were retrospectively analyzed.RESULTS: A total of 208   
patients were reviewed. The incidence of PVT after hepatectomy was 9.1 % (n   
= 19), including main portal vein (MPV) thrombosis (n = 7) and peripheral   
portal vein (PPV) thrombosis (n = 12). Patients with MPV thrombosis had a   
significantly higher incidence of right hepatectomy (p < 0.001), larger   
resection volume (p = 0.003), and longer operation time (p = 0.021) than   
patients without PVT (n = 189). Multivariate analysis identified right   
hepatectomy as a significant independent risk factor for MPV thrombosis   
(odds ratio 108.9; p < 0.001). Patients with PPV thrombosis had a   
significantly longer duration of Pringle maneuver than patients without PVT   
(p = 0.002). Among patients who underwent right hepatectomy, those with PVT   
(n = 6) had a significantly lower early liver regeneration rate than those   
without PVT (n = 13; p = 0.040), and those with PVT had deterioration of   
liver function on postoperative day 7. In all patients with MPV thrombosis   
who received anticoagulation therapy, PVT subsequently resolved.CONCLUSIONS:   
Postoperative PVT after hepatectomy is not rare. It is closely related to   
delayed recovery of liver function and delayed liver regeneration.  
  
RECORD 285  
Prophylactic anticoagulation in Guillain- Barré syndrome: Too much of a good   
thing?  
Lim E. Lilleker J.B. Richardson A.M.  
European Journal of Neurology (2014) 21 SUPPL. 1 (540). Date of Publication:   
May 2014  
  
Objectives: Venous thromboembolic complications are common during recovery   
from Guillain-BarréSyndrome (GBS). The use of prophylactic anticoagulation   
to reduce this risk is accepted as good practice although can be associated   
with a risk of haemorrhagic complications. We examined the current practice   
of prophylactic anticoagulation in patients with GBS admitted to a tertiary   
neurosciences centre. The frequency of venous thromboembolism and   
haemorrhagic complications were also recorded. Methods: A retrospective   
notes review of 50 consecutive patients admitted with GBS to the Greater   
Manchester Neurosciences Centre between 2008 and 2013 was performed. Disease   
severity, prophylactic anticoagulation type, dose and duration, and the   
frequency and timing of haemorrhagic and thromboembolic complications were   
recorded and analysed. Results: Details of prophylactic anticoagulation   
prescription were obtained for 42 of 50 patients. All nonambulant patients   
(95%, 40/42) received low molecular weight heparin (LMWH) at any dose at   
some point during a mean inpatient stay of 64 days. 14 haemorrhagic   
complications occurred in 10 patients. 7 of these coincided with the use of   
'treatment (high) dose' LMWH. A bleeding tracheostomy site contributed to   
the death of 1 patient. 1 thrombotic event was observed: a portal vein   
thrombosis. No deep vein thrombosis or pulmonary emboli occurred.   
Conclusions: Thromboembolic complications were infrequent in this   
population. However, a relatively high frequency of haemorrhagic   
complications were observed and these appeared to correlate with the use of   
'treatment (high) dose' LMWH. Systematic work is required to define the   
optimal prophylactic anticoagulation strategy in patients with GBS to ensure   
that the benefits outweigh risks.  
  
RECORD 286  
Prophylactic anticoagulation in Guillain-Barré syndrome: Too much of a good   
thing?  
Lim E. Lilleker J.B. Richardson A.M.  
Journal of Neurology (2014) 261 SUPPL. 1 (S354-S355). Date of Publication:   
May 2014  
  
Objectives: Venous thromboembolic complications are common during recovery   
from Guillain-Barré Syndrome (GBS). The use of prophylactic anticoagulation   
to reduce this risk is accepted as good practice although can be associated   
with a risk of haemorrhagic complications. We examined the current practice   
of prophylactic anticoagulation in patients with GBS admitted to a tertiary   
neurosciences centre. The frequency of venous thromboembolism and   
haemorrhagic complications were also recorded. Methods: A retrospective   
notes review of 50 consecutive patients admitted with GBS to the Greater   
Manchester Neurosciences Centre between 2008 and 2013 was performed. Disease   
severity, prophylactic anticoagulation type, dose and duration, and the   
frequency and timing of haemorrhagic and thromboembolic complications were   
recorded and analysed. Results: Details of prophylactic anticoagulation   
prescription were obtained for 42 of 50 patients. All non-ambulant patients   
(95 %, 40/42) received low molecular weight heparin (LMWH) at any dose at   
some point during a mean inpatient stay of 64 days. 14 haemorrhagic   
complications occurred in 10 patients. 7 of these coincided with the use of   
'treatment (high) dose' LMWH. A bleeding tracheostomy site contributed to   
the death of 1 patient. 1 thrombotic event was observed: a portal vein   
thrombosis. No deep vein thrombosis or pulmonary emboli occurred.   
Conclusions: Thromboembolic complications were infrequent in this   
population. However, a relatively high frequency of haemorrhagic   
complications were observed and these appeared to correlate with the use of   
'treatment (high) dose' LMWH. Systematic work is required to define the   
optimal prophylactic anticoagulation strategy in patients with GBS to ensure   
that the benefits outweigh risks.  
  
RECORD 287  
Additional value of C-arm CT in imaging patent ductus venosus (PDV) and its   
intra-procedural role in guiding endovascular occlusion  
Bedford C. Ponraj C. Masand P. Himes R. Hernandez A. Pimpalwar S.  
Pediatric Radiology (2014) 44 SUPPL. 1 (S159). Date of Publication: May 2014  
  
Purpose or Case Report: Children with symptomatic PDV are selected for   
endovascular occlusion based on the size of intrahepatic portal veins and   
portal pressures after balloon test occlusion. Recently, C-arm angiography   
systems have the capability to acquire CT-like 3D images of vascular anatomy   
which could be applied in this scen Our purpose is: 1. To illustrate the   
additional value of C-arm CT in imaging PDVand its intraprocedural role in   
guiding endovascular occlusion using vascular plug. 2. To illustrate the   
steps involved in endovascular PDVocclusion procedure and to report a   
complication of post-procedural portal vein thrombosis. A 4 years old girl   
with Trisomy 12 mosaicism presented with a 3 years history of recurrent   
episodes of altered mental status and hyperammonemia (49-136 micromol/L).   
MRV showed a 10 mm diameter PDV. Using a right jugular venous approach,   
C-arm CT superior mesenteric venography was performed pre and post balloon   
occlusion. Hemodynamic pressure measurements were obtained and the PDV was   
occluded using a 14 mm Amplatzer Vascular Plug II under C-arm CT guidance.   
C-arm CT (syngo DynaCT®, Siemens AG, Forchheim, Germany) imaging was   
performed using 8-s DR low dose body protocol. Fortymilliliter4 iodinated   
contrast (320 mgI/ml) was injected through a 4Fr catheter at 4 ml/s, 500 psi   
with 2-s X-ray delay. Initial C-arm CT venography was helpful in   
demonstrating the complex 3D relationship between the PDV, left portal vein   
and IVC. C-arm CT imaging further helped with confirmation of device size   
and position in relationship to the portal vein and IVC prior to actual   
deployment. Serum ammonia levels normalized within 2 days post occlusion.   
Twelve hours post procedure ultrasound demonstrated an occlusive left portal   
vein thrombus which was treated with intravenous heparin and subsequently   
transitioned to lovenox. Three months follow-up ultrasound showed complete   
occlusion of the PDV and resolution of the portal venous thrombus. C-arm CT   
venography is a useful intra-procedural imaging tool for guiding   
endovascular closure of PDV. The hypoplastic portal venous system is at risk   
of thrombosis which can be managed with appropriate anticoagulation. (Figure   
Presented).  
  
RECORD 288  
portal vein thrombosis and anticoagulation  
Leza D. Jesús Escobar M. Alkadi N. Arango L. Romero L. Cornudella R. Gaván   
O.  
Thrombosis Research (2014) 133 SUPPL. 3 (S80). Date of Publication: May 2014  
  
Background: Thrombosis in atypical locations is infrequent, although when it   
develops it is extremely severe and associated with significant morbidity   
and mortality. It has been associated with different pathologies such as   
hepatic cirrhosis, neoplasia and hereditary thrombophilia. It is crucial to   
carry out full and early anticoagulant treatment in attempting to reduce   
complications derived from this pathology. This review examines risk factors   
in patients under oral anticoagulant treatment for portal vein or   
spleno-portal axis thrombosis. Methods: Clinical histories from the years   
2000 to 2013 were revised and the data in the study of coagulation, the   
study of plasmatic thrombophilia (PC, PS, AT, RPCA, lupus anticoagulant),   
genetic thrombophilia with FV R506Q (Leiden) and FV H1299R (R2) mutations,   
and prothrombin G20210A and MTHFR mutations. In other determinations the   
V617F gen JAK2 mutation was studied. Results: A total of 25 patients were   
studied, of which 22 were male and 4 were female, with ages ranging from 37   
and 78 years old. The pathology associated with portal vein and   
spleno-portal axis thrombosis of highest incidence are haematological   
processes (SMPC) 32% (in 8 patients), followed closely by hepatic cirrhosis   
in 28% (7 patients), associated to surgical processes in 24% (3 hepatic   
post-transplant, 1 splenectomy, 1transjugular intrahepatic portosystemic   
derivation) and in relation to inflammatory processes 16% (in 4 patients). A   
study of plasmatic and genetic thrombophilia was carried out in 76% of the   
patients studied, with a resulting relevance of 17%, not evaluable for   
plasmatic thrombophilia due to oral anticoagulant treatment or hepatopathy   
in 59% and not pathological in 24%. In 2 patients (28%), the FV Leiden   
mutation was detected. The mutation V617F gen JAK2 was positive in 4   
patients with Essential Thrombocythemia (ET) (16% of patients studied).   
Conclusions: portal vein thrombosis is more prevalent in males. The most   
frequent haematological process associated with portal vein thrombosis is   
ET, with positive JAK2. In relation to surgical processes, a hepatic   
transplant supposes a high risk for the development of portal vein   
thrombosis. The FV Leiden mutation in portal vein thrombosis was detected   
associated to surgery (hepatic post-transplant) and another to hepatopathy.   
It is recommended to carry out, together with a study of thrombophilia, the   
mutation V617F gen JAK2 in all patients without associated hepatopathy to   
rule out haematological processes.  
  
RECORD 289  
Hypercoagulability in cirrhotic patients with hepatocellular carcinoma (HCC)   
and portal vein thrombosis (PVT)  
Zanetto A. Vitale A. Cillo U. Rodriguez K. Fadin M. Gavasso S. Radu C.M.   
Zerbinati P. Farinati F. Russo F.P. Germani G. Burra P. Simioni P. Senzolo   
M.  
Thrombosis Research (2014) 133 SUPPL. 2 (S201). Date of Publication: May   
2014  
  
Introduction: Studies which explore the hypercoagulable state associated   
with neoplastic disease and its correlation with the risk of PVT in patients   
with HCC are lacking. Aim: The aim of the present study was to evaluate the   
thrombophilic role of HCC in cirrhotics with and without HCC and to   
correlate the presence of HCC and the coagulation profile with PVT   
incidence. Materials and Methods: Cirrhotic patients with and without HCC   
were prospectively enrolled in the study. Age- and sex-matched healthy   
individuals constituted the control group for ROTEM parameters All patients   
underwent: thromboelastometry (ROTEM), platelet count, determination of   
prothrombin time and of levels of pro and anticoagulation factors. During   
follow-up, PVT onset in both patients with and without HCC was recorded.   
Results: 76 cirrhotics, 41 with HCC and 35 without HCC, were included.   
Forty-eight healthy volunteers were included as the control group. Volume of   
active HCC was >5 cm(3) in 18 patients. Levels of pro- and anti-coagulation   
factors were similar between patients with and without HCC, but fibrinogen   
was increased in HCC patients with active volume >5cm(3) HCC compared to   
those with <5cm(3) HCC bulk (348.72 ±124.06mg/dL vs 237.64±99.18mg/dL) and   
to cirrhotics without HCC (260.57±126.07 mg/dL; p=0.006). Platelet count was   
significantly increased in HCC patients compared to non-HCC patients   
(125.41±67.88/ μL vs 86.89±54.07/μL; p=0.046), and this was especially true   
in Child Class A subjects (152.6 ± 66.14/μL vs 92 ±46.73/μL; p=0.038).   
Patients with HCC showed significantly lower clotting time and maximum clot   
formation at ROTEM compared to healthy controls. The hypercoagulable state   
was present even when HCC patients were compared to cirrhotics without HCC,   
and was more evident when performing a subgroup analysis of Child Class A   
patients, with statistically significant differences in MCF EXTEM, MCF NATEM   
e CFT NATEM. The incidence of PVT was 24.4% (10/41) and 11.4% (4/35) in HCC   
and non-HCC patients, respectively (OR: 2.5; 95%, CI 0.70-8.83). In the HCC   
group, 5/10 portal vein thromboses occurred in patients in Child Class A. In   
contrast with HCC patients who did not develop PVT, at fibrinogen test of   
ROTEM, MCF and AUC were statistically higher in HCC patients who later   
developed PVT (23.71 ±12.82 mm vs 16.30 ±7.08mm p=0.047 and 2,359±1,272.62   
vs 1,535±640.20 p=0.022; respectively). Conclusions: Cirrhotics with HCC   
demonstrate a prothrombotic hemostatic balance resulting in an increased   
risk of PVT development. The unstable hemostatic balance in cirrhotic   
patients can easily tip towards hypercoagulability due to the contribution   
of increased fibrinogen synthesis and an increased platelet count. ROTEM   
seems to be a sensitive method to identify hypercoagulability, that would   
otherwise be undetected by routine laboratory testing. Further   
investigations are needed to determine whether patients with HCC should   
receive prophylactic anticoagulation for PVT prevention.  
  
RECORD 290  
Circulating microparticles in cirrhotic patients with hepatocellular   
carcinoma (HCC) and portal vein thrombosis  
Campello E. Zanetto A. Radu C.M. Gavasso S. Spiezia L. Rodriguez K. Senzolo   
M. Simioni P.  
Thrombosis Research (2014) 133 SUPPL. 2 (S195). Date of Publication: May   
2014  
  
Introduction: Hepatocellular carcinoma (HCC) is a hypervascular tumor with   
high levels of apoptosis and tumor necrosis. Microparticles (MP) are small   
membrane vesicles released from the cell plasma membrane, particularly in   
cell stress, apoptosis and altered cellular viability. Aim: We investigated   
(1) the levels of circulating MP of different cellular origins in patients   
with cirrhosis with and without HCC and compared them with healthy people,   
and (2) the correlation between MP levels and portal vein thrombosis (PVT)   
incidence in HCC patients. Materials and Methods: Seventy-six cirrhotic   
patients [Child: A 29, B 29, C 18]: 41 with HCC (M/F 22/19; mean age 64±12   
years) and 35 without HCC (M/F 18/17; mean age 56±14) were prospectively   
enrolled. Age- and sex-matched healthy individuals constituted the control   
group. MP were identified by size and Annexin V-FITC labelling, using   
flow-cytometry. Endothelial-derived activated MP (E-Selectin+) were   
identified using anti-CD62E-PE; Tissue factor-bearing (TF+) with   
anti-CD142-PE; and Thrombomodulin-bearing MP (TM+) with anti-CD141-FITC   
antibodies. MP procoagulant activity was measured using the STA Procoag PPL   
assay. PVT onset in both patients with and without HCC was recorded.   
Results: Patients with HCC showed significantly higher levels of E-Selectin+   
(median 23380 [Interquartile range 1,9487-4,1172] MP/μL), TF+ (102 [94-168]   
MP/μL) and Annexin V-MP (5146 [1,619-6,264] MP/μL) compared to healthy   
controls (4,395 [3,329-7,253 MP/μL; 68 [17-93] MP/μL; 1,900 [1,154-2,114]   
MP/μL; all p<0.001). Moreover we showed a significant increase in   
E-Selectin+ and in TF+ in HCC patients compared to cirrhotics without HCC   
(p<0.001 and 0.05, respectively). The PPL clotting time was significantly   
shorter (p<0.05) in HCC patients (61 [48- 71] sec) compared to cirrhotics   
(72 [67-80] sec) and controls (81 [68- 101] sec). As for TM+, cirrhotics had   
lower levels (42 [27-85] MP/μL) than HCC patients (60 [52-76] MP/μL) and   
controls (86 [70-102] MP/μL), the differences were not significant. MP   
circulating levels did not significantly differ between Child A versus B and   
C in HCC patients. The incidence of PVT was 24.4% (10/41) and 11.4% (4/35)   
in HCC and non-HCC patients, respectively (OR: 2,5; 95%, CI 0.70-8.83). The   
levels of E-Selectin+ (24,561 [20,006-31,765] MP/μL) and TF+ (124 [101-188]   
MP/μL) were statistically higher in HCC patients who later developed PVT   
(p=0.002 and p=0.04; respectively) compared to HCC patients who did not   
develop PVT. Conclusions: cirrhosis is associated with an increase in   
endothelial and TF+ MP and a decrease of TM+MP compared to healthy controls.   
Moreover, cirrhotics with HCC showed higher MP circulating plasma levels   
than cirrhotic patients without HCC. MP may have a role in the   
hypercoagulability that characterizes the unstable haemostatic balance in   
cirrhotic patients and could be considered a sensitive method to identify   
hypercoagulability in HCC patients that should receive prophylactic   
anticoagulation for PVT prevention.  
  
RECORD 291  
Gastrointestinal ischemia in patients with acute and chronic portal vein   
thrombosis: A prospective study  
Harki J. Plompen E.P. Van Noord D. Hoekstra J. Kuipers E.J. Janssen H. Tjwa   
E.T.  
Gastroenterology (2014) 146:5 SUPPL. 1 (S-478). Date of Publication: May   
2014  
  
Introduction: portal vein thrombosis (PVT) patients often experience   
abdominal pain, in part of the cases caused by gastrointestinal ischemia   
(GI). Little is known about the frequency of GI as result of venous   
congestion. GI is characterized by specific complaints and mucosal   
desaturation. The aim of this study therefore was to evaluate the prevalence   
of GI in acute and chronic PVT. Methodology: A prospective cohort study   
between 2009 and 2013. Patients with non-cirrhotic, non-malignant PVT   
received the standard work-up for PVT and were assessed for clinical   
symptoms of GI along with radiological evaluation and state-ofthe- art   
mucosal intraluminal saturation measurements by means of visible light   
spectroscopy (VLS). Differences between groups were assessed using   
descriptive and non-parametric statistics. Results: We included 17 PVT   
patients (median 48 (IQR31-54) years; 59% female). Acute PVT was present in   
5 patients and chronic PVT in 12 patients. VLS measurements below the   
established cut-off of 58% saturation (i.e. indicating ischemia) for   
duodenal mucosa were found in 12/16 patients (75%). They were more frequent   
in patients with chronic PVT compared to acute PVT (82% vs. 60%, p=0.37 and   
median saturation 53% (IQR49-58) vs. 58% (IQR55-63), respectively, p=0.07,   
see Figure 1). Exercise-induced pain and weight loss, both typical for GI,   
were present in 47% and 41% of the patients and were more present in   
patients with chronic compared to acute PVT (58% vs. 20%, p=0.16 and 50% vs.   
20% , p=0.27 respectively). There was a marked difference in   
exercise-induced pain, but not weight loss, between patients with normal and   
decreased VLS measurements (0% vs. 67%, p= 0.025). Postprandial pain was   
present in 9 patients (53%) and patients with decreased VLS measurements   
tend to experience postprandial pain more often than patients with normal   
VLS measurements (67% vs. 25%, p=0.16). No clear relationship was observed   
between mucosal ischemia and degree of PVT occlusion, use of anticoagulation   
or extrahepatic venous involvement. None of the patients had   
gastrointestinal arterial involvement. Conclusions: Typical symptoms of   
gastrointestinal ischemia are frequently observed in patients with PVT and   
more prevalent in patients with chronic PVT. Mucosal ischemia is present in   
the majority of patients with PVT and exercise-induced pain seems to be the   
most specific indicator of mucosal ischemia. In patients with PVT, VLS   
measurements should be considered to assess ischemia and tailor patient   
management. (Figure Presented).  
  
RECORD 292  
Resolution of left ventricular thrombus in a patient with dilated   
cardiomyopathy by rivaroxaban  
Padilla-Pérez M. Almagro-Torres F. Sanchez-De Castro M. Lozano-Cabezas C.   
Salas-Bravo D. Torres Llergo J. Carlos Fernandez-Lozano J. Vazquez-Ruiz De   
Castroviejo E.  
Thrombosis Research (2014) 133 SUPPL. 3 (S55). Date of Publication: May 2014  
  
Background: Left ventricular (LV) thrombosis is a potentially   
lifethreatening condition, with a high risk of embolic complications. Many   
conditions increase the risk of LV thrombi (dilated cardiomyopathy (severe   
LV dysfunction, myocarditis, myocardial infarction or ventricular aneurysm).   
Transthoracic echocardiogram (TTE) usually gives the diagnosis, being the   
use of ultrasound contrast media useful to enhance its diagnostic accuracy.   
The information available about the treatment of intraventricular thrombosis   
is scarce. Despite the lack of evidence, the empiric use of vitamin K   
antagonists for up to 6 months is recommended in patients with myocardial   
infarction and mural thrombi. In the heart failure scenario, oral   
anticoagulation is only indicated in patients with atrial fibrillation (AF).   
The efficacy and safety of new oral anticoagulants in this context is   
unknown. It has been reported that rivaroxaban, oral direct factor Xa   
inhibitor currently indicated for the prevention of stroke and systemic   
embolism in patients with non-valvular AF, can be successfully used to treat   
thrombosis in other locations, such as left atrial appendage thrombus and   
acute portal vein thrombosis. Methods: We present the case of a 78 year-old   
patient admitted in our hospital for congestive heart failure. The ECG   
showed AF (first known episode) and complete left bundle branch block. His   
creatinine clearance was 40 ml/min by MDRD. Oral anticoagulation was   
initiated with Rivaroxaban 15 mg/day. The TTE showed dilated LV with severe   
sistolic dysfunction, and 2 images of thrombi inside the LV cavity, one in   
the basal inferior segment and other in the apex (figure 1 A y B).   
Carvedilol 6.25mg b.i.d., enalapril 10mg b.i.d. and intravenous furosemide   
were added to the treatment. He refused to undergo an invasive   
coronariography. Patient was discharged following clinical stabilization.   
After 4 weeks of treatment, the new TTE showed complete resolution of both   
thrombi (figure 1 C y D) and patient was asymptomatic. Results: Results of   
the Transtoracic Echocardiogram will be shown in the figure. Conclusions: To   
our knowledge, this is the first case reported about the resolution of LV   
thrombosis with Rivaroxaban. No randomized clinical trials have been   
specifically performed, but are needed, on the use of new oral   
anticoagulants in the clinical scenarios of dilated cardiomyopathy and   
thrombosis in the LV.  
  
RECORD 293  
Percutaneous microwave ablation of hepatocellular carcinoma: Clinical   
results with 118 tumors treated over 3 years  
Ziemlewicz T. Hinshaw J.L. Lubner M.G. Kitchin D.R. Brace C.L. Alexander M.   
Agarwal P. Lee F.T.  
Journal of Vascular and Interventional Radiology (2014) 25:5 (817.e4). Date   
of Publication: May 2014  
  
Objectives: Microwave (MW) ablation is a promising technology that offers   
several advantages over radiofrequency (RF) ablation. However, clinical   
evaluation of microwave ablation is limited currently. The purpose of this   
study was to retrospectively review the results in the first 79 patients   
with hepatocellular carcinoma (HCC) treated with a high-power, gas-cooled MW   
device at a single center. Methods: Between December 2010 and November 2013   
we treated 118 hepatocellular carcinomas in 85 sessions in 79 BCLC stage A   
patients via a percutaneous approach utilizing US and/or CT guidance. There   
were 64 male and 15 female patients with mean age of 60.5 years (range   
44-82). All procedures were performed with a highpowered, gas-cooled   
microwave system (Certus 140, Neuwave Medical, Madison, WI). Complications   
were recorded according to the Clavien-Dindo classification. Follow- up with   
contrast-enhanced CT or MR was planned at 1, 3, 6, 12, 18, 24, 30, and 36   
months post-procedure. Results: Tumors ranged in size from 0.5 to 4.2 cm   
(mean 2.1 cm) and median followup was 12 months (range 1-35). All treatments   
were completed in a single session and considered technically successful   
with no evidence of residual tumor at immediate post-procedure CECT. Mean   
power was 73 Watts (range 30-140 Watts) and mean ablation time was 5.1   
minutes (range 1-11.5 minutes). Primary treatment effectiveness by imaging   
was 93.2% (110/118), 93.1% (95/102) for tumors < 3 cm, 100% (14/14) for   
tumors 3-4 cm, and 50% (1/2) for tumors > 4 cm. Of the 8 tumor progressions,   
2 were treated with repeat ablation, 2 were noted at explant pathology, and   
4 were treated with intra-arterial therapy as they were abutting an adjacent   
critical structure, precluding more aggressive ablation, or multifocal HCC   
had developed in the interval. Distant intrahepatic progression occurred in   
20.2% of patients during the follow-up period with 5 patients undergoing   
repeat ablation and the other 11 developing multifocal disease treated with   
intra-arterial or systemic therapy. A single Grade II complication occurred   
(1.2%), a main portal vein thrombus following ablation of a caudate lobe   
lesion, which was noted at 1-month follow-up and resolved with low dose   
anti-coagulation (target INR 1.5-2.0). There were no Grade III or higher   
complications. There was no procedure related mortality. Overall survival is   
78.8% with most deaths related to end stage liver disease (n=8) or   
multifocal HCC (n=5). Conclusions: Treating hepatocellular carcinoma using   
percutaneous microwave ablation is safe, and in our experience is as   
effective with small (<3 cm) tumors, but more effective with larger tumors   
(excellent efficacy maintained up to 4 cm) as compared to other more   
established ablation modalities.  
  
RECORD 294  
Gastrointestinal ischemia in patients with acute and chronic portal vein   
thrombosis  
Harki J. Plompen E.P. Van Noord D. Hoekstra J. Kuipers E.J. Janssen H.L.   
Tjwa E.T.  
Journal of Hepatology (2014) 60:1 SUPPL. 1 (S239-S240). Date of Publication:   
April 2014  
  
Background and Aims: portal vein thrombosis (PVT) patients often experience   
abdominal pain. Little is known about the frequency of gastrointestinal   
ischemia (GI) as result of venous congestion. GI is characterized by   
specific complaints, presence of thrombosis and mucosal desaturation. The   
aim of this study was to evaluate GI prevalence in acute and chronic PVT.   
Methods: A prospective cohort study in patients with non-cirrhotic,   
non-malignant PVT who were assessed for clinical symptoms of GI along with   
radiological evaluation and state-of-the-art mucosal intraluminal saturation   
measurements (VLS). Results: We included 17 PVT patients (median 48   
(IQR31-54) years). Acute PVT was present in 5 patients and chronic PVT in 12   
patients. VLS measurements below the established cut-off of 58% (i.e.   
indicating ischemia) for duodenal mucosa were found in 75% of the patients   
and were lower in patients with chronic PVT compared to patients with acute   
PVT (see Figure1). VLS measurements were also decreased in 57% of patients   
without symptoms. Exerciseinduced pain and weight loss, both typical for GI,   
were more present in patients with chronic compared to acute PVT (58% vs.   
20% and 1.0±2.2 kg/month vs. 4.5±2.5 kg/month). Postprandial pain was   
present in 9 patients and similar between groups. No clear relationship was   
observed between GI and degree of PVT occlusion, use of anticoagulation or   
extrahepatic venous involvement. None of the patients had mesenteric   
arterial involvement. Conclusions: Characteristics of gastrointestinal   
ischemia are frequently observed in patients with PVT and more prevalent in   
patients with chronic PVT. In patients with PVT, VLS measurements should be   
considered to assess ischemia and tailor patient management. (Figure   
Presented).  
  
RECORD 295  
Prospective evaluation of the correlation between hemostatic alterations and   
incidence of portal vein thrombosis in patients with liver cirrhosis and   
hepatocellular carcinoma  
Zanetto A. Ferrarese A. Vitale A. Cillo U. Rodriguez K.-I. Fadin M. Gavasso   
S. Radu C. Zerbinati P. Simioni P. Farinati F. Germani G. Russo F.P. Burra   
P. Senzolo M.  
Journal of Hepatology (2014) 60:1 SUPPL. 1 (S14). Date of Publication: April   
2014  
  
Background and Aims: Studies which explores the hypercoagulable induced by   
HCC in cirrhosis are lacking. The aim of the present study was to evaluate   
the thrombophilic role of HCC as risk factor for development of PVT.   
Methods: Cirrhotic patients with and without HCC were prospectively enrolled   
in the study and underwent: thromboelastometry (ROTEM), platelet count,   
determination of prothrombin time and of levels of pro and anticoagulation   
factors. During follow-up, PVT onset in both patients with and without HCC   
was recorded. Results: 76 cirrhotics, 41 with HCC, were included. Volume of   
active HCC was >5cm(3) in 18 patients. Levels of pro and anticoagulation   
factors were similar between patients with and without HCC, but fibrinogen   
was increased in HCC patients with active volume >5cm(3) HCC compared to   
those with ≤5cm(3) HCC bulk (348.72±124.06 mg/dL vs 237.64±99.18 mg/dL) and   
to cirrhotics without HCC (260.57±126.07 mg/dL) (p = 0.006). Platelet count   
was significantly increased in HCC compared to non-HCC patients, and this   
was especially true in Child A group. ROTEM demonstrated a significantly   
lower clotting time and maximum clot formation in HCC patients compared to   
controls and non-HCC cirrhotics, especially in Child A group. One-year   
incidence of PVT was 19.5% (8/41) and 5.7% (2/35) in HCC and non-HCC   
patients, respectively (p = 0.04). Fibrinogen test of ROTEM, MCF and AUC   
were statistically greater in HCC patients who later developed PVT.   
Conclusions: Cirrhotics with HCC demonstrate a prothrombotic hemostatic   
balance resulting in an increased risk of PVT development. This   
prothrombotic state seems to be detectable by ROTEM and thus possibly   
suggest those who could benefit from thromboprophylaxis.  
  
RECORD 296  
portal vein thrombosis in cirrhotic patients undergoing orthotopic liver   
transplantation: A single centre experience  
Stradella D. Risso A. Martini S. Rizzetto M. Salizzoni M.  
Journal of Hepatology (2014) 60:1 SUPPL. 1 (S380-S381). Date of Publication:   
April 2014  
  
Background and Aims: portal vein thrombosis (PVT) is a complication of   
cirrhosis that may increase surgical complexities during Orthotopic Liver   
Transplantation (OLT). We retrospectively evaluated the management of PVT   
before, during and after OLT in our centre. Methods: Among all the cirrhotic   
patients who underwent OLT between 2005 and 2011 in Liver Transplant Turin   
Centre, we retrospectively included all the patients with US and CT   
diagnosis of pre-OLT non-neoplastic PVT. Extension of thrombosis (according   
to Yerdel classification), pre-OLT clinical and US course, use of   
anticoagulation therapy (AT), surgical technique for portal vein   
anastomosis, complications and US follow-up after OLT were collected for   
each patient. Results: 70/997 (7%) patients were included. PVT was:   
intrahepatic in 22.9%, grade 1 in 32.9%, grade 2 in 24.3%, grade 3 in 7.1%   
and grade 4 in 12.8%. Due to thrombosis, 72% of patients started AT   
(complications rate: 17%, all minor bleedings) and 40% underwent TIPS,   
without complications. Pre-OLT complete resolution or regression of   
thrombosis occurred in 74% of patients under AT vs 40% of patients not   
treated (p = 0.04). During OLT 97% of patients underwent porto-portal   
anastomosis, 29% of them needing thrombectomy. PVT extension (both at   
diagnosis and at OLT) and AT didn't statistically impact in terms of   
survival and complications during and after OLT. Conclusions: PVT is a   
frequent issue in cirrhotic patients waiting for OLT. In our experience AT   
can be safely managed allowing a pre-OLT understaging of PVT and the need   
for special surgical techniques at OLT is very uncommon.  
  
RECORD 297  
Impact of anticoagulant therapy on upper gastrointestinal bleeding (UGI) in   
patients with liver cirrhosis. results from a retrospective multicentric   
case-control study  
Cerini F. Martińez Gonzalez J. Puente Á. Casas M. Vinaixa C. Berenguer M.   
Villanueva C. Augustin S. Llop E. Bañares R. Albillos A. Bosch J.   
Hernández-Gea V. Garcia-Pagan J.C.  
Journal of Hepatology (2014) 60:1 SUPPL. 1 (S8). Date of Publication: April   
2014  
  
Background and Aims: Recent studies have shown that LC is actually an   
acquired hypercoagulable state with increased thrombotic risk and   
anticoagulation therapy (AT) is most frequently used in LC pts. Variceal   
bleeding is a severe complication of LC. It is unknown if AT may impact the   
outcome of UGIB in these pts. Methods: 52 pts on AT with UGIB were enrolled   
in the study. portal vein thrombosis (PVT) and other reasons were the   
indication for AT in 14 and 38 pts respectively. 104 pts with LC and UGIB   
not under AT were matched, for severity of LC, age, sex, source of UGIB and   
SOFA score, as controls. Results: There were no differences between groups   
except for INR/MELD. UGIB was attributed to portal hypertension (PH) in 63%   
of pts (gastroesophageal varices in 56%) and peptic/vascular lesions in 37%.   
17% of Pts experienced 5-days-failure being independent predictors age and   
presence of PVT, but not AT. There were no differences between pts   
with/without AT in needs for rescue therapies, ICU admission, transfusions,   
hospital stay and 6wmortality. 11% of pts had 6w-mortality. These patients   
had worse Child, MELD, MELD-XI, SOFA and use of AT for other reason than PVT   
(21% vs 7% in PVT and 8% in non-AT; p = 0.03) than survivors. Independent   
predictors of 6-weeks-mortality were SOFA score and use of PVT for other   
reason than PVT. Conclusions: Our study suggests that factors that impact   
the outcome of UGIB in pts under AT, are the degree of liver failure and   
comorbidity, but not AT itself.  
  
RECORD 298  
Successful treatment of diffuse portal vein thrombosis after splenectomy   
following living donor liver transplantation patient  
Kang S.H. Hwang S. Kim K.-H. Ahn C.-S. Moon D.-B. Ha T.-Y. Lee S.-G.  
HPB (2014) 16 SUPPL. 2 (552). Date of Publication: March 2014  
  
Introduction: Splenectomy is performed after living donor liver   
transplantation (LDLT) for various resons, including pancytopenia, ascites,   
left sided portal hypertension. Complications of splenectomy include   
bleeding, pancreatic injury, infection, portal vein thrombosis. Portal vein   
thrombosis (PVT) is rare but dreaded complication after splenectomy in LDLT   
recipients that can compromise patient and graft survival. Several treatment   
modality of PVT after splenectomy are reported, including anticoagulation,   
thrombolysis and surgical thrombectomy. Method: We recently experienced a   
case of acute and diffuse PVT after splenectomy in LDLT recipient who was   
successfully treated with thrombectomy and systemic anticoagulation therapy.   
The patient was a 56- year-old female with hepatitis B virus-associated   
liver cirrhosis. She underwent LDLT using modified right lobe graft on June   
2, 2006. Recently she developed thrombocytopenia on routine laboratory exam.   
A CT scan showed splenomegaly. We performed splenectomy to resolve   
thrombocytopenia. On postoperative fifth day, she complained pain on her   
shoulder. A CT scan was performed and showed diffuse splenic, main portal   
and intra-hepatic portal vein thrombosis with ischemic change in anterior   
section. Results: The patient was taken immediately to the operating room.   
We opened splenic stump and placed a 12-Fr Fogarty catheter. With the   
assistance of vascular surgeon, thrombectomy of the main portal vein was   
attempted via catheter under intra-operative ultrasound guiding. After   
thrombectomy, intra-operative portogram revealed recanalization of the   
splenic vein and main portal vein but still remained intra-hepatic portal   
vein thrombosis. An interventional radiologist put the catheter into   
intra-hepatic portal vein via inferior mesenteric vein. After several times   
of aspiration through the catheter, portogram showed complete recanalization   
of intra-hepatic portal vein. We put the stent into spleno-mesenteric   
junction to prevent recurrent PVT. Systemic heparinization war started   
immediately after operation and was converted warfarin. A postoperative   
Doppler ultrasound and CT scan showed patent main and intra-hepatic portal   
vein. Conclusions: This case showed that PVT after splenectomy can be   
treated with surgical thrombectomy, intra-operative interventional procedure   
and systemic anticoagulation therapy.  
  
RECORD 299  
Prospective evaluation of the correlation between hemostatic status and   
incidence of portal vein thrombosis in patients with liver cirrhosis and   
hepatocellular carcinoma  
Zanetto A. Ferrarese A. Vitale A. Cillo U. Rodriguez K. Fadin M. Gavasso S.   
Radu C. Zarbinati P. Simioni P. Farinati F. Germani G. Russo F.P. Burra P.   
Senzolo M.  
Digestive and Liver Disease (2014) 46 SUPPL. 2 (S11). Date of Publication:   
March 2014  
  
Background and aim: Studies which explores the hypercoagulable induced by   
HCC in cirrhosis are lacking. The aim of the present study was to evaluate   
the thrombophilic role of HCC as risk factor for development of PVT.   
Material and methods: Cirrhotic patients with and without HCC were   
prospectively enrolled in the study and underwent: thromboelastometry   
(ROTEM), platelet count, determination of prothrombin time and of levels of   
pro and anticoagulation factors. During follow-up, PVT onset in both   
patients with and without HCC was recorded. Results: 76 cirrhotics, 41 with   
HCC and 35 without HCC, were included. Forty-eight healthy volunteers were   
included as the control group. Volume of active HCC was >5 cm(3) in 18   
patients. Levels of pro and anticoagulation factors were similar between   
patients with and without HCC, but fibrinogen was increased in HCC patients   
with active volume >5 cm(3) HCC compared to those with ≤5 cm(3) HCC bulk   
(348.72 mg/dL±124.06 mg/dL vs 237.64 mg/dL ±99.18 mg/dL) and to cirrhotics   
without HCC (260.57 mg/dL±126.07 mg/dL) (p=0.006). Platelet count was   
significantly increased in HCC patients compared to non-HCC patients,   
especially in Child Class A subjects. Patients with HCC showed significantly   
lower clotting time and maximum clot formation at ROTEM compared to healthy   
controls. The hypercoagulable state was present even when HCC patients were   
compared to cirrhotics without HCC, and was more evident when performing a   
subgroup analysis of Child Class A patients, with statistically significant   
differences in MCF EXTEM/NATEM e CFT NATEM. One-year-incidence of PVT was   
19.5% (8/41) and 5.7% (2/35) in HCC and non-HCC patients, respectively   
(p=0.04). In the HCC group, 4/8 PVT occurred in patients in Child Class A.   
Fibrinogen test of ROTEM, MCF and AUC were statistically elavated in HCC   
patients who later developed PVT. Conclusions: Cirrhotics with HCC   
demonstrate a prothrombotic hemostatic balance resulting in an increased   
risk of PVT development. ROTEM seems to be a sensitive method to identify   
hypercoagulability. Further investigations are needed to determine whether   
patients with HCC should receive prophylactic anticoagulation for PVT   
prevention.  
  
RECORD 300  
Combined surgical and interventional therapy of acute portal vein thrombosis   
without cirrhosis: A new effective hybrid approach for recanalization of the   
portal venous system  
Loss M. Lang S.A. Uller W. Wohlgemuth W.A. Schlitt H.J.  
Journal of the American College of Surgeons (2014) 218:3 (e79-e86). Date of   
Publication: March 2014  
  
RECORD 301  
portal vein thrombosis secondary to embolization of superior mesenteric   
arteriovenous fistula  
Zhao Y. Li Z. Zhang L. Wei B. Zeng X. Fu P.  
Annals of Vascular Surgery (2014) 28:2 (490.e9-490.e12). Date of   
Publication: February 2014  
  
Superior mesenteric arteriovenous fistula is a rare vascular disorder.   
Endovascular embolization has been widely used to treat this disease.   
Patients receiving successful fistula embolization generally have good   
prognoses. We present a man with iatrogenic superior mesenteric   
arteriovenous fistula who received endovascular embolization. Portal   
thrombus was detected on postoperative day 2, and the patient eventually   
died of multiple organ failure on postoperative day 13 despite having   
received antithrombotic and antiplatelet therapy. We identified portal   
thrombosis as a serious complication of transcatheter superior mesenteric   
arteriovenous fistula embolization. © 2014 Elsevier Inc. All rights   
reserved.  
  
RECORD 302  
Hemostatic status and portal vein thrombosis (PVT) in cirrhotic patients   
with hepatocellular carcinoma (HCC)  
Ferrarese A. Vitale A. Cillo U. Rodriguez K.I. Fadin M. Gavasso S. Radu C.   
Zerbinati P. Simioni P. Farinati F. Germani G. Russo F.P. Burra P. Senzolo   
M.  
Digestive and Liver Disease (2014) 46 SUPPL. 1 (e36). Date of Publication:   
February 2014  
  
Background and aim: Studies exploring the hypercoagulable state induced by   
HCC and its correlation with the risk of PVT are lacking. The aim of the   
present study was to evaluate the thrombophilic role of HCC as risk factor   
for PVT development. Methods: Cirrhotic patients with and without HCC were   
prospectively enrolled. Age- and sex-matched healthy individuals constituted   
the control group for thromboelastometry (ROTEM). All patients underwent:   
ROTEM, platelet count, determination of prothrombin time and of levels of   
pro and anticoagulation factors. During follow-up, PVT onset was recorded.   
Results: 76 cirrhotics, 41 with HCC, and 48 healthy controls were included.   
Volume of active HCC was >5cm(3) in 18 patients. Levels of pro and   
anticoagulation factors were similar between patients with and without HCC,   
but fibrinogenwasincreased inHCCpatients with active volume >5cm(3) HCC   
compared to those with ≤5cm(3) HCC (348.72±124.06 mg/dL vs 237.64±99.18   
mg/dL) and to cirrhotics without HCC (260.57±126.07 mg/dL) (p = 0.006).   
Platelet countwassignificantly increased inHCCcompared to non-HCC, and this   
was especially true in Child Class A subjects. Patients with HCC showed   
significantly lower clotting time and maximum clot formation at ROTEM   
compared to controls. The hypercoagulable state was present even when HCC   
patients were compared to cirrhotics without HCC, especially in Child A   
patients, with statistically significant differences in MCF EXTEM/NATEM. One   
year-incidence of PVT was 19.5% (8/41) and 5.7% (2/35) in HCC and non-HCC   
patients, respectively (p = 0.04). In theHCCgroup, 4/8 portal vein   
thromboses occurred in patients in Child A group. Fibrinogen test of ROTEM,   
MCF and AUC were statistically greater in HCC patients who later developed   
PVT. Conclusions: Cirrhotics with HCC demonstrate a prothrombotic hemostatic   
balance resulting in an increased risk of PVT. This prothrombotic state   
seems to be detectable by ROTEM and thus possibly suggest those who could   
benefit from thromboprophylaxis.  
  
RECORD 303  
Antithrombotic treatment of splanchnic vein thrombosis: Results of an   
international registry  
Ageno W. Riva N. Schulman S. Bang S.M. Sartori M.T. Grandone E.   
Beyer-Westendorf J. Barillari G. Di Minno M.N.D. Dentali F.  
Seminars in Thrombosis and Hemostasis (2014) 40:1 (99-105). Date of   
Publication: February 2014  
  
Treatment of splanchnic vein thrombosis (SVT) is a clinical challenge due to   
heterogeneity of clinical presentations, increased bleeding risk, and lack   
of evidences from clinical trials. We performed an international registry to   
describe current treatment strategies and factors associated with   
therapeutic decisions in a large prospective cohort of unselected SVT   
patients. A total of 613 patients were enrolled (mean age 53.1 years,   
standard deviation ± 14.8); 62.6% males; the majority (468 patients) had   
portal vein thrombosis. Most common risk factors included cirrhosis (27.8%),   
solid cancer (22.3%), and intra-abdominal inflammation/infection (11.7%); in   
27.4% of patients, SVT was idiopathic. During the acute phase, 470 (76.7%)   
patients received anticoagulant drugs, 136 patients (22.2%) remained   
untreated. Incidental diagnosis, single vein thrombosis, gastrointestinal   
bleeding, thrombocytopenia, cancer, and cirrhosis were significantly   
associated with no anticoagulant treatment. Decision to start patients on   
vitamin K antagonists after an initial course of parenteral anticoagulation   
was significantly associated with younger age, symptomatic onset, multiple   
veins involvement, and unprovoked thrombosis. Although a nonnegligible   
proportion of SVT patients did not receive anticoagulant treatment, the   
majority received the same therapies recommended for patients with usual   
sites thrombosis, with some differences driven by the site of thrombosis and   
the pathogenesis of the disease. © 2014 by Thieme Medical Publishers, Inc.  
  
RECORD 304  
Liver transplantation in cirrhotic patients with portal vein thrombosis: A   
single centre experience  
Risso A. Stradella D. Martini S. Rizzetto M. Salizzoni M.  
Digestive and Liver Disease (2014) 46 SUPPL. 1 (e40). Date of Publication:   
February 2014  
  
Introduction: portal vein thrombosis (PVT) is a complication of cirrhosis   
that may increase surgical complexities during orthotopic liver   
transplantation (OLT) and cause complications after surgery. Aim: To   
evaluate the management of PVT before, during and after OLT in our centre.   
Materials and methods: Among all the cirrhotic patients who underwent OLT   
between 2005 and 2011 in Turin Liver Transplantation Centre, we   
retrospectively included all the patients with US and CT diagnosis of   
pre-OLT non-neoplastic PVT. Extension of thrombosis (according to Yerdel   
classification), presence of genetic prothrombotic risk factors, pre-OLT   
clinical and US course, use of anticoagulation therapy (AT), surgical   
technique for portal vein anastomosis, complications and US follow-up after   
OLT were collected for each patient. Results: 70/997 (7%) patients were   
included. PVT was: intrahepatic in 22.9% of them, grade 1 in 32.9%, grade 2   
in 24.3%, grade 3 in 7.1% and grade 4 in 12.8%. We found very small   
prevalence of genetic prothrombotic risk factors, and their presence did not   
correlate with extension of thrombosis and US course. Due to thrombosis, 72%   
of patients started AT (complications rate: 17%, all minor bleedings) and   
40% underwent TIPS, without complications. Pre-OLT complete resolution or   
regression of thrombosis occurred in 74% of patients under AT vs 40% of   
patients not treated (p = 0.04). During OLT 97% of patients underwent   
portoportal anastomosis, 29% of them needing thrombectomy. PVT extension   
(both at diagnosis and at OLT) and use of AT did not statistically impact in   
terms of survival and complications during and after OLT. Conclusions: PVT   
is a frequent issue in cirrhotic patients waiting for OLT and its   
development seems to be unrelated to the presence of prothrombotic risk   
factors. In our experience AT can be safely managed allowing a pre-OLT   
understaging of PVT and the need for special surgical techniques at OLT is   
very uncommon.  
  
RECORD 305  
Liver transplant in budd-chiari syndrome: A single-center experience in   
Saudi Arabia  
Saleh Y. Eldeen F.Z. Kamel Y. Kabbani M. Alsebayel M. Broering D.  
Experimental and Clinical Transplantation (2014) 12:1 (52-54). Date of   
Publication: February 2014  
  
Objectives: If they do not respond to other treatments, patients with   
Budd-Chiari syndrome are potential candidates for a liver transplant. Timing   
for transplant is controversial; however, before other systems deteriorate,   
early intervention in relatively stable patient may improve the outcome and   
survival of these patients. Materials and Methods: Six patients (2 women and   
4 men) had Budd-Chiari syndrome (1.2%) among 475 patients who had undergone   
a liver transplant at our center between 2001 and 2012. Imaging modalities   
including duplex ultrasound, abdominal computed tomography angiography, and   
hematologic evaluation were part of our routine diagnostic work-up. Although   
we perform mostly living-donor liver transplants, these patients received a   
liver transplant from a deceased donor, because there was not enough   
evidence to justify a living-donor liver transplant. We thought that not   
replacing the caval vein might negatively influence the outcome.   
Postoperatively, these recipients were started on a heparin infusion and   
triple therapy immunosuppression; only then was warfarin introduced as   
long-term anticoagulant. Results: Two patients died, 1 from uncontrollable   
bleeding and disseminated intravascular coagulopathy, and the other died in   
the intensive care unit after 5 months because of multiorgan failure and   
sepsis. One patient had portal vein thrombosis 9 months after the liver   
transplant; the other patient needed a liver retransplant after 5 years   
owing to liver failure, secondary to chronic rejection. Graft survival rate   
was 75%, and patient survival rate was 66.6%. Conclusions: This is the first   
article from Saudi Arabia to describe the outcome of a liver transplant in   
this subgroup of patients with Budd-Chiari syndrome. Treatment of   
Budd-Chiari syndrome follows a therapeutic algorithm that should start with   
anticoagulation and may end up with liver transplant; however, it should be   
considered early if other treatments fail. © Başkent University 2014 Printed   
in Turkey. All Rights Reserved.  
  
RECORD 306  
anticoagulation policy after venous resection with a pancreatectomy: A   
systematic review  
Chandrasegaram M.D. Eslick G.D. Lee W. Brooke-Smith M.E. Padbury R. Worthley   
C.S. Chen J.W. Windsor J.A.  
HPB (2014) 16:8 (691-698). Date of Publication: August 2014  
  
Background Portal vein (PV) resection is used increasingly in pancreatic   
resections. There is no agreed policy regarding anticoagulation. Methods A   
systematic review was performed to compare studies with an anticoagulation   
policy (AC+) to no anticoagulation policy (AC-) after venous resection.   
Results There were eight AC+ studies (n = 266) and five AC- studies (n =   
95). The AC+ studies included aspirin, clopidogrel, heparin or warfarin.   
Only 50% of patients in the AC+ group received anticoagulation. There were   
more prosthetic grafts in the AC+ group (30 versus 2, Fisher's exact P <   
0.001). The overall morbidity and mortality was similar in both groups.   
Early PV thrombosis (EPVT) was similar in the AC+ group and the AC- group   
(7%, versus 3%, Fisher's exact P = 0.270) and was associated with a high   
mortality (8/20, 40%). When prosthetic grafts were excluded there was no   
difference in the incidence of EPVT between both groups (1% vs 2%, Fisher's   
exact test P = 0.621). Conclusion There is significant heterogeneity in the   
use of anticoagulation after PV resection. Overall morbidity, mortality and   
EPVT in both groups were similar. EPVT has a high associated mortality.   
While we have been unable to demonstrate a benefit for anticoagulation, the   
incidence of EPVT is low in the absence of prosthetic grafts. © 2013   
International Hepato-Pancreato-Biliary Association.  
  
RECORD 307  
Prothrombotic disorders in a cohort of 25 patients undergoing   
transplantation: Investigation and management implications  
Pither C. Middleton S. Gao R. Sharkey L. Jamieson N. Butler A.  
Transplantation Proceedings (2014) 46:6 (2133-2135). Date of Publication:   
2014  
  
Background. Many patients referred for intestinal transplantation have a   
history of thrombosis. We undertook an analysis of transplanted patients to   
describe the history and frequency of thrombosis, clinical course, and   
management strategies used. Results. Twenty-five patients underwent   
transplantation of intestine containing blocks between 2007 and 2012; 20 of   
25 are still alive. Five of 25 patients were transplanted with history of   
portomesenteric thrombosis, 6 of 25 had experienced loss of venous access   
due to thrombosis, and 6 of 25 had history of mesenteric ischemia.   
Pretransplantation, 16 of 25 patients were anticoagulated. Thrombophilia   
screens identified 3 of 16 patients who were JAK2 positive, 1 of 25 who had   
antithrombin deficiency, and 1 of 25 who had a factor V Leiden heterozygote.   
Post-transplantation, of all 16 patients who were anticoagulated   
pretransplantation and continued postoperatively, 1 of 16 infarcted their   
small bowel graft and 4 of 16 developed a further venous thrombosis despite   
anticoagulation. Of the 9 without a previous history of thrombosis, 1 had a   
pulmonary embolus more than a decade after transplantation and another had   
an upper limb deep vein thrombosis associated with a line. Both were then   
anticoagulated. Seven of 25 are not anticoagulated, although they are   
administered antiplatelet prophylaxis. Postoperative bleeding complications   
of anticoagulation occurred in 3 patients. After a subarachnoid hemorrhage   
in 1 of those 3 patients, anticoagulation was stopped. The other 2 patients   
bled during ileal biopsy, and both remain on low molecular weight heparin   
treatment. Conclusion. Those with identifiable thrombophilic tendency and a   
history of venous or arterial thrombosis are considered to be at high risk   
for recurrent thrombosis. Those without such a history could be considered   
low risk. Our practice is to anticoagulate all high-risk individuals before   
and after transplantation and offer antiplatelet prophylaxis to low-risk   
patients as the risk of anticoagulation probably outweighs the risk of   
thrombosis for them. Early input from hematologists is vital in the   
management of high-risk patients, particularly those who thrombose when   
anticoagulated.  
  
RECORD 308  
Clinical management of acute portal/mesenteric vein thrombosis  
Lang S.A. Loss M. Wohlgemuth W.A. Schlitt H.J.  
Viszeralmedizin: Gastrointestinal Medicine and Surgery (2014) 30:6   
(394-400). Date of Publication: 21 Jan 2014  
  
Background: Acute thrombosis of the portal vein (PV) and/or the mesenteric   
vein (MV) is a rare but potentially life-threatening disease. A multitude of   
risk factors for acute portal vein thrombosis (PVT)/mesenteric vein   
thrombosis (MVT) have been identified, including liver cirrhosis,   
malignancy, coagulation disorders, intra-abdominal infection/inflammation,   
and postoperative condition. Methods: This article analyses the treatment   
options for acute PVT/MVT. Results: Initially, the clinical management   
should identify patients with an intra-abdominal focus requiring immediate   
surgical intervention (e.g. bowel ischaemia). Subsequently, emphasis is   
placed on the recanalization of the PV/MV or at least the prevention of   
thrombus extension to avoid long-term complications of portal hypertension.   
Several therapeutic options are currently available, including   
anticoagulation therapy, local/systemic thrombolysis, interventional or   
surgical thrombectomy, and a combination of these procedures. Due to the   
lack of prospective randomized studies, a comparison between these   
therapeutic approaches regarding the efficacy of PV/MV recanalization is   
difficult, if not impossible. Conclusion: In patients with acute PVT/MVT, an   
individualized treatment based on the clinical presentation, the underlying   
disease, the extent of the thrombosis, and the patients' comorbidities is   
mandatory. Therefore, these patients should be considered for an   
interdisciplinary therapy in specialized centres with the option to utilise   
all therapeutic approaches currently available.  
  
RECORD 309  
Management of portal/mesenteric vein occlusion  
Sauerbuch T. Hopt U.T. Neeff H. Pötzsch B. Rössle M. Valla D.  
Viszeralmedizin: Gastrointestinal Medicine and Surgery (2014) 30:6   
(417-420). Date of Publication: 21 Jan 2014  
  
RECORD 310  
Nephrotic syndrome complicated with portal, splenic, and superior mesenteric   
vein thrombosis  
Park B.S. Park S. Jin K. Choi G. Park K.M. Jo K.M. Kim Y.W.  
Kidney Research and Clinical Practice (2014) 33:3 (161-164). Date of   
Publication: 1 Sep 2014  
  
Thromboembolism is a major complication of nephrotic syndrome. Renal vein   
thrombosis and deep vein thrombosis are relatively common, especially in   
membranous nephropathy. However, the incidence of portal vein and superior   
mesenteric vein (SMV) thrombosis in patients with nephrotic syndrome is very   
rare. To date, several cases of portal vein thrombosis treated by   
anticoagulation therapy, not by thrombolytic therapy, have been reported as   
a complication of nephrotic syndrome. Here, we report a case of portal,   
splenic, and SMV thrombosis in a patient with a relapsed steroid dependent   
minimal change disease who was treated successfully with anticoagulation and   
thrombolytic therapy using urokinase. Radiologic findings and his clinical   
conditions gradually improved. Six months later, a complete remission of the   
nephrotic syndrome was observed and the follow-up computed tomography scan   
showed the disappearance of all portal vein, splenic vein, and SMV thrombi.  
  
RECORD 311  
Management of coagulation abnormalities in liver disease  
Potze W. Porte R.J. Lisman T.  
Expert Review of Gastroenterology and Hepatology (2014) 9:1 (103-114). Date   
of Publication: 1 Jan 2014  
  
Liver disease is characterized by changes in all phases of hemostasis. These   
hemostatic alterations were long considered to predispose patients with   
liver disease towards a bleeding tendency, as they are associated with   
prolonged conventional coagulation tests. However, these patients may also   
suffer from thrombotic complications, and we now know that the hemostatic   
system in patient with liver disease is, in fact, in a rebalanced state. In   
this review we discuss the concept of rebalanced hemostasis and its   
implications for clinical management of patients with liver disease. For   
instance, there is no evidence that the use of prophylactic blood product   
transfusion prior to invasive procedures reduces bleeding risk. Clinicians   
should also be aware of the possibility of thrombosis occurring in patients   
with a liver disease, and regular thrombosis prophylaxis should not be   
withheld in these patients.  
  
RECORD 312  
Do postliver transplant patients need thromboprophylactic anticoagulation?  
Mukerji A.N. Karachristos A. Maloo M. Johnson D. Jain A.  
Clinical and Applied Thrombosis/Hemostasis (2014) 20:7 (673-677). Date of   
Publication: 2014  
  
Postoperative thromboprophylactic anticoagulation against Deep Vein   
Thrombosis (DVT) and Pulmonary Embolism (PE) is standard of care with   
current evidence-based guidelines. However, majority of liver transplant   
(LT) patients have thrombocytopenia and/or prolonged INR before surgery.   
Studies or guidelines regarding role of prophylactic anticoagulation after   
LT are lacking. There is a need to balance the risk of thrombosis with   
significant hemorrhage, implying those needing transfusion or return to OR   
due to bleeding. We conclude that after LT, anticoagulation is not required   
routinely for DVT/PE prophylaxis. Rather, it is indicated in specific   
circumstances, chiefly for prophylaxis of hepatic artery thrombosis or   
portal vein thrombosis in cases with use of grafts, pediatric cases, small   
size vessels, Budd Chiari syndrome, amongst others.  
  
RECORD 313  
Venous thromboembolism at uncommon sites in neonates and children  
Pergantou H. Avgeri M. Komitopoulou A. Xafaki P. Kapsimali Z. Mazarakis M.   
Adamtziki E. Platokouki H.  
Journal of Pediatric Hematology/Oncology (2014) 36:8 (624-629). Date of   
Publication: 8 Nov 2014  
  
We retrospectively analyzed the data of 24 children (whereof 11 neonates),   
with non-central venous line-related and nonmalignancy-related venous   
thromboembolism (VTE) at uncommon sites, referred to our Unit from January   
1999 to January 2012. Thirty patients who also suffered deep vein   
thrombosis, but in upper/low extremities, were not included in the analysis.   
The location of rare site VTE was: portal (n=7), mesenteric (n=2) and left   
facial vein (n=1), spleen (n=3), lung (n=3), whereas 10 neonates developed   
renal venous thrombosis. The majority of patients (91.7%) had at least 1   
risk factor for thrombosis. Identified thrombophilic factors were:   
antiphospholipid antibodies (n=2), FV Leiden heterozygosity (n=6), MTHFR   
C677T homozygosity (n=4), protein S deficiency (n=2), whereas all neonates   
had agerelated low levels of protein C and protein S. All but 6 patients   
received low-molecular-weight heparin, followed by warfarin in 55% of cases,   
for 3 to 6 months. Prolonged anticoagulation was applied in selected cases.   
During a median follow-up period of 6 years, the clinical outcome was: full   
recovery in 15 patients, evolution to both chronic portal hypertension and   
esophageal varices in 2 children, and progression to renal failure in 7 of   
10 neonates. Neonates are greatly vulnerable to complications after VTE at   
uncommon sites, particularly renal. Future multicentre long-term studies on   
neonatal and pediatric VTE at unusual sites are considered worthwhile.  
  
RECORD 314  
Management of portal vein thrombosis in liver cirrhosis  
Qi X. Han G. Fan D.  
Nature Reviews Gastroenterology and Hepatology (2014) 11:7 (435-446). Date   
of Publication: July 2014  
  
portal vein thrombosis (PVT) is a fairly common complication of liver   
cirrhosis. Importantly, occlusive PVT might influence the prognosis of   
patients with cirrhosis. Evidence from a randomized controlled trial has   
shown that anticoagulation can prevent the occurrence of PVT in patients   
with cirrhosis without prior PVT. Evidence from several case series has also   
demonstrated that anticoagulation can achieve portal vein recanalization in   
patients with cirrhosis and PVT. Early initiation of anticoagulation therapy   
and absence of previous portal hypertensive bleeding might be positively   
associated with a high rate of portal vein recanalization after   
anticoagulation. However, the possibility of spontaneous resolution of   
partial PVT questions the necessity of anticoagulation for the treatment of   
partial PVT. In addition, a relatively low recanalization rate of complete   
PVT after anticoagulation therapy suggests its limited usefulness in   
patients with complete PVT. Successful insertion of a transjugular   
intrahepatic portosystemic shunt (TIPS) not only recanalizes the thrombosed   
portal vein, but also relieves the symptomatic portal hypertension. However,   
the technical difficulty of TIPS potentially limits its widespread   
application, and the risk and benefits should be fully balanced. Notably,   
current recommendations regarding the management of PVT in liver cirrhosis   
are insufficient owing to low-quality evidence. © 2014 Macmillan Publishers   
Limited. All rights reserved.  
  
RECORD 315  
Associations of Coagulation Factor V Leiden and Prothrombin G20210A   
Mutations With Budd-Chiari Syndrome and portal vein thrombosis: A Systematic   
Review and Meta-analysis  
Qi X. Ren W. De Stefano V. Fan D.  
Clinical Gastroenterology and Hepatology (2014) 12:11 (1801-1812). Date of   
Publication: 1 Nov 2014  
  
Background & Aims: We conducted a systematic review and meta-analysis to   
evaluate the associations of the coagulation factor V (encoded by F5) Leiden   
(FVL) or prothrombin (encoded by F2) G20210A mutation with Budd-Chiari   
syndrome or portal vein thrombosis (PVT). Methods: Relevant articles were   
identified in searches of the PubMed, EMBASE, Cochrane Library, and   
ScienceDirect databases. The prevalence of the FVL and prothrombin G20210A   
mutations were compared between patients with Budd-Chiari syndrome or PVT   
without cirrhosis and healthy individuals (controls) and between patients   
with cirrhosis, with and without PVT. Odds ratios (ORs) with 95% confidence   
intervals (CIs) were calculated. Results: We initially identified 869   
articles, and included 27 in our final analysis. Compared with controls,   
patients with Budd-Chiari syndrome had a significantly higher prevalence of   
the FVL mutation (OR, 6.21; 95% CI, 3.93-9.79) and a similar prevalence of   
the prothrombin G20210A mutation (OR, 1.90; 95% CI, 0.69-5.23); patients   
with PVT without cirrhosis had a significantly higher prevalence of the FVL   
mutation (OR, 1.85; 95% CI, 1.09-3.13) or the prothrombin G20210A mutation   
(OR, 5.01; 95% CI, 3.03-8.30). Compared with patients with cirrhosis without   
PVT, patients with cirrhosis and PVT had a significantly higher prevalence   
of the FVL mutation (OR, 2.55; 95% CI, 1.29-5.07). We observed a trend   
toward a higher prevalence of the prothrombin G20210A mutation in patients   
with cirrhosis and PVT, but the difference was not statistically significant   
(OR, 2.93; 95% CI, 0.94-9.07). Conclusions: Based on a meta-analysis, the   
FVL mutation is associated with an increased risk of Budd-Chiari syndrome,   
PVT without cirrhosis, and PVT in cirrhosis. The prothrombin G20210A   
mutation is associated with PVT, but not Budd-Chiari syndrome. Studies are   
needed to confirm these findings in different racial and ethnic groups.  
  
RECORD 316  
Gastric and ectopic varices  
Henry Z. Uppal D. Saad W. Caldwell S.  
Clinics in Liver Disease (2014) 18:2 (371-388). Date of Publication: May   
2014  
  
Although often considered together, gastric and ectopic varices represent   
complications of a heterogeneous group of underlying diseases. Commonly,   
these are known to arise in patients with cirrhosis secondary to portal   
hypertension; however, they also arise in patients with noncirrhotic portal   
hypertension, most often secondary to venous thrombosis of the portal venous   
system. One of the key initial assessments is to define the underlying   
condition leading to the formation of these portal-collateral pathways to   
guide management. In the authors' experience, these patients can be grouped   
into distinct although sometimes overlapping conditions, which can provide a   
helpful conceptual basis of management. © 2014 Elsevier Inc.  
  
RECORD 317  
Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy  
Salinas J. Barros D. Salgado N. Viscido G. Funke R. Pérez G. Pimentel F.   
Boza C.  
Surgical Endoscopy and Other Interventional Techniques (2014) 28:4   
(1083-1089). Date of Publication: April 2014  
  
Introduction: Portal and mesenteric vein thrombosis are relatively uncommon   
surgical complications, with difficult diagnosis and potentially severe   
consequences due to higher risk of bowel infarction. The purpose of this   
study was to present a series of patients who developed postoperative portal   
vein thrombosis after laparoscopic sleeve gastrectomy. Methods: This is a   
retrospective analysis of patients who underwent sleeve gastrectomy between   
June 2005 and June 2011 who developed portal vein thrombosis. Demographic   
data, personal risk factors, family history of thrombosis, and postoperative   
results of thrombophilia study were analyzed in this study. Results: A total   
of 1,713 laparoscopic sleeve gastrectomies were performed. Seventeen   
patients (1%) developed portal vein thrombosis after surgery. Of the 17   
patients, 16 were women, 8 had a history of smoking, 7 used oral   
contraceptives, and 2 had a family history of deep vein thrombosis of the   
lower limbs. All patients were discharged on the third day of surgery with   
no immediate complications. Symptoms presented at a median of 15 (range,   
8-43) days after surgery with abdominal pain in most cases. One case   
required emergency laparotomy and splenectomy because of an active bleeding   
hematoma with massive portomesenteric vein thrombosis. In 11 cases, a   
thrombosis of the main portal vein was identified, in 15 the right portal   
branch was compromised, and in 10 the left portal branch. Eleven patients   
presented thrombosis of the superior mesenteric vein, and ten patients   
presented a concomitant thrombosis of the splenic vein. A massive PMVT was   
presented in six cases. Seven patients had a positive thrombophilia study.   
Conclusions: portal vein thrombosis and/or mesenteric thrombosis are   
relatively uncommon complications in patients undergoing bariatric surgery.   
In this series, the portomesenteric vein thrombosis was the most common   
complication after LSG in a high-volume center. © 2014 Springer   
Science+Business Media.  
  
RECORD 318  
Long-term outcome of percutaneous transhepatic balloon angioplasty for   
portal vein stenosis after pediatric living donor liver transplantation: A   
single institute's experience  
Yabuta M. Shibata T. Shibata T. Shinozuka K. Isoda H. Okamoto S. Uemoto S.   
Togashi K.  
Journal of Vascular and Interventional Radiology (2014) 25:9 (1406-1412).   
Date of Publication: September 2014  
  
Purpose To evaluate retrospectively the long-term outcomes of percutaneous   
transhepatic balloon angioplasty performed for portal vein stenosis (PVS)   
after pediatric living donor liver transplantation (LDLT). Materials and   
Methods Between October 1997 and December 2013, of 527 pediatric patients   
(age < 18 y) who underwent LDLT in a single institution, 43 patients (19   
boys, 24 girls; mean age, 4.1 y ± 4.1) were confirmed to have PVS at direct   
portography with or without manometry and underwent percutaneous   
interventions, including balloon angioplasty with or without stent   
placement. Technical success, clinical success, laboratory findings,   
manometry findings, patency rates, and major complications were evaluated.   
Follow-up periods after initial balloon angioplasty ranged from 5-169 months   
(mean, 119 mo). Results Technical success was achieved in 65 of 66 sessions   
(98.5%) and in 42 of 43 patients (97.7%), and clinical success was achieved   
in 37 of 43 patients (86.0%). Platelet counts improved significantly. Of 32   
patients undergoing manometry, 19 showed significant improvement of pressure   
gradient across the stenosis after percutaneous transhepatic balloon   
angioplasty. At 1, 3, 5, and 10 years after balloon angioplasty, the rates   
of primary patency were 83%, 78%, 76%, and 70%, and the rates of   
primary-assisted patency were 100%, 100%, 100%, and 96%. Two major   
complications subsequent to balloon angioplasty were noted: severe asthma   
attack and portal vein thrombosis. Conclusions Percutaneous transhepatic   
balloon angioplasty is a safe and effective treatment with long-term patency   
for PVS after pediatric LDLT. © 2014 SIR.  
  
RECORD 319  
Analysis of risk factors for portal venous system thrombosis formation and   
treatment for patients with posthepatitic cirrhosis complicating portal   
hypertension after splenectomy and pericardial devascularization  
Wu S.-L. Wu Z. Wang R.-T. Bai J.-G.  
Journal of Xi'an Jiaotong University (Medical Sciences) (2014) 35:5   
(714-717). Date of Publication: 1 Sep 2014  
  
Objective: To analyze the risk factors for portal venous system thrombosis   
(PVST) formation and the effect of thrombolytic therapy in patients with   
posthepatitic cirrhosis complicating portal hypertension after splenectomy   
and pericardial devascularization. Methods: We retrospectively reviewed our   
records of 71 patients with posthepatitic cirrhosis complicating portal   
hypertension who underwent splenectomy and pericardial devascularization at   
our hospital between January 2005 and December 2011. The 71 patients were   
divided into three groups: Group A (23 who received anticoagulation therapy   
in the early period of postoperation), Group B (29 who received   
anticoagulation therapy when their postoperative platelet count was   
>300×10(9)/L), and Group C (19 who did not receive postoperative   
anticoagulation therapy). The incidence of PVST, the anatomic distribution   
of thrombosis, and the effect of thrombolytic therapy were compared among   
the three groups and the relationship between PVST and various factors   
before and during operation was determined. Results: Multivariate analysis   
showed that PVST after splenectomy and pericardial devascularization was   
related to the diameter of main portal vein and low preoperative platelet   
counts. The total incidence of PVST was 40.8 % (29/71). The incidence of   
portal venous system thrombosis in Groups A, B and C was 26.1% (6/23), 44.8%   
(13/29), and 52.6% (10/19), respectively, without obvious differences among   
these groups. Thrombosis of the portal vein and its branches was found   
postoperatively. Splenic vein thrombosis accounted for 72.4% of all PVST.   
The rate of complete resolution of portal and superior mesenteric venous   
thrombosis was 76.2% (16/21), while that was only 23.8% (5/21) for splenic   
vein thrombosis (χ(2)=11.524, P=0.001), which was significantly different   
from the former (χ(2)=11.524,P=0.001). Conclusion: The diameter of main   
portal vein and low preoperative platelet counts were independent risk   
factors for PVST in patient with posthepatitic cirrhosis complicating portal   
hypertension after splenectomy and pericardial devascularization. Preventive   
anticoagulation therapy does not affect PVST formation. After thrombolytic   
therapy, the rate of complete resolution of portal and superior mesenteric   
venous thrombosis is higher than that of splenic vein thrombosis.  
  
RECORD 320  
portal vein thrombosis in cirrhosis  
Raja K. Jacob M. Asthana S.  
Journal of Clinical and Experimental Hepatology (2014) 4:4 (320-331). Date   
of Publication: 1 Dec 2014  
  
portal vein thrombosis (PVT) is being increasingly recognized in patients   
with advanced cirrhosis and in those undergoing liver transplantation.   
Reduced flow in the portal vein is probably responsible for clotting in the   
spleno-porto-mesenteric venous system. There is also increasing evidence   
that hypercoagulability occurs in advanced liver disease and contributes to   
the risk of PVT. Ultrasound based studies have reported a prevalence of PVT   
in 10-25% of cirrhotic patients without hepatocellular carcinoma. Partial   
thrombosis of the portal vein is more common and may not have   
pathophysiological consequences. However, there is high risk of progression   
of partial PVT to complete PVT that may cause exacerbation of portal   
hypertension and progression of liver insufficiency. It is thus, essential   
to accurately diagnose and stage PVT in patients waiting for transplantation   
and consider anticoagulation therapy. Therapy with low molecular weight   
heparin and vitamin K antagonists has been shown to achieve complete and   
partial recanalization in 33-45% and 15-35% of cases respectively. There are   
however, no guidelines to help determine the dose and therapeutic efficacy   
of anticoagulation in patients with cirrhosis. anticoagulation therapy   
related bleeding is the most feared complication but it appears that the   
risk of variceal bleeding is more likely to be dependent on portal pressure   
rather than solely related to coagulation status. TIPS has also been   
reported to restore patency of the portal vein. Patients with complete PVT   
currently do not form an absolute contraindication for liver   
transplantation. Thrombectomy or thromboendovenectomy is possible in more   
than 75% of patients followed by anatomical end-to-end portal anastomosis.   
When patency of the portal vein and/or superior mesenteric vein is not   
achieved, only non-anatomical techniques (reno-portal anastomosis or   
cavo-portal hemitransposition) can be performed. These techniques, which do   
not fully reverse portal hypertension, are associated with higher morbidity   
and mortality risks in the short term.  
  
RECORD 321  
Efficacy and safety of the anticoagulant drug, danaparoid sodium, in the   
treatment of portal vein thrombosis in patients with liver cirrhosis  
Naeshiro N. Aikata H. Hyogo H. Kan H. Fujino H. Kobayashi T. Fukuhara T.   
Honda Y. Nakahara T. Ohno A. Miyaki D. Murakami E. Kawaoka T. Tsuge M.   
Hiraga N. Hiramatsu A. Imamura M. Kawakami Y. Ochi H. Chayama K.  
Hepatology Research (2014). Date of Publication: 2014  
  
Aim: To assess the efficacy and safety of the anticoagulant drug, danaparoid   
sodium, in the treatment of portal vein thrombosis (PVT) in patients with   
liver cirrhosis. Methods: A consecutive 26 cirrhotic patients with PVT were   
enrolled in this retrospective cohort study. The etiologies of cirrhosis   
were hepatitis B virus-related, hepatitis C virus-related, alcoholic and   
cryptogenic in five, 14, three and four patients, respectively. Child-Pugh   
grade A, B and C was noted in 13, eight and five patients, respectively.   
Patients were treated with 2 weeks' administration of danaparoid sodium   
followed by the evaluation of PVT reduction and adverse events. Results: All   
patients experienced reduction of PVT through the treatment. The median   
volume of PVT before and after treatment was 2.40cm(3) (range, 0.18-16.63)   
and 0.37cm(3) (range, 0-5.74), respectively. The median reduction rate of   
PVT volume was 77.3% (range, 18-100%). According to the reduction rate,   
complete reduction (CR), partial reduction (PR, ≥50%) and stable disease   
(SD, <50%) were observed in four (15%), 16 (62%) and six patients (23%),   
respectively. The median volume of PVT before treatment was significantly   
different between CR+PR and SD (2.09 vs 4.35cm(3), P=0.045). No severe   
adverse events such as bleeding symptoms (e.g. gastrointestinal bleeding and   
cerebral hemorrhage) and thrombocytopenia were encountered. Conclusion:   
Danaparoid sodium for the treatment of PVT in patients with liver cirrhosis   
was safe and effective. Therefore, anticoagulation therapy with danaparoid   
sodium could have potential as one of the treatment options in PVT   
accompanied by cirrhosis.  
  
RECORD 322  
An overview of current treatment methods for Budd-Chiari syndrome  
Seijo S. Garcia-Pagan J.C.  
Expert Opinion on Orphan Drugs (2014) 2:2 (147-157). Date of Publication:   
February 2014  
  
Introduction: The Budd-Chiari Syndrome (BCS) is a rare and life-threatening   
disorder caused by the obstruction of hepatic venous outflow. The clinical   
presentation of BCS can range from the absence of symptoms to the   
development of end-stage liver disease or fulminant liver failure. Areas   
covered: This review provides an overview of the available treatments for   
BCS. Long-term anticoagulation is mandatory in all patients with BCS. The   
need for an additional intervention, such as hepatic vein angioplasty,   
thrombolysis, transjugular intrahepatic portosystemic shunt, surgical shunts   
or liver transplantation depends on the severity of symptoms and response to   
treatment. Due to the low prevalence of the disease, knowledge of management   
of BCS is mostly based on retrospective series and expert opinion and   
hampered the development of randomized controlled trials. Expert opinion:   
Outcome of BCS has improved in the last decades, mainly due to the increased   
suspicion and early treatment instauration; the frequent recognition of an   
underlying systemic prothrombotic disorder and its ensuing adequate   
treatment; the widespread use of long-term anticoagulation and the   
implementation of a stepwise management strategy based on the clinical   
response to the previous step of treatment. Further studies are needed to   
better define treatment failure and the optimal timing for scaling in   
treatment. © Informa UK, Ltd.  
  
RECORD 323  
anticoagulation for portal vein thrombosis in cirrhosis  
Seijo S. García-Pagan J.C.  
Revista Espanola de Enfermedades Digestivas (2014) 106:7 (491). Date of   
Publication: 2014  
  
RECORD 324  
Therapy algorithm for portal vein thrombosis in liver cirrhosis: The   
internist's point of view  
Rössle M. Bausch B. Klinger C.  
Viszeralmedizin: Gastrointestinal Medicine and Surgery (2014) 30:6   
(401-408). Date of Publication: 21 Jan 2014  
  
Background: Treatment of non-malignant portal vein thrombosis (PVT) in   
patients with cirrhosis has been neglected in the past because of the fear   
of bleeding complications when using anticoagulation and due to the   
technical difficulties associated with the implantation of the transjugular   
intrahepatic portosystemic shunt (TIPS). However, PVT has a negative impact   
on outcome and compromises liver transplantation, warranting treatment by   
using anticoagulation and TIPS. Methods: This review considers studies on   
the treatment of PVT in cirrhosis published in the last 10 years.   
Unfortunately, many of these studies are limited by their retrospective   
design and a small sample size. Results: anticoagulation using   
low-molecular-weight heparin (LMWH) or vitamin K antagonists is effective in   
the treatment of patients with limited and recent PVT, resulting in a   
recanalization in up to 50% of the patients. TIPS (plus local measures)   
results in a recanalization of up to 100% and reduces the rebleeding rate   
considerably in patients with recent or chronic PVT. Conclusion: Based on   
the presently limited knowledge, a therapy algorithm is suggested favouring   
the TIPS as a first-line treatment for PVT in patients with symptomatic   
portal hypertension. Patients with thus far asymptomatic portal hypertension   
may first receive anticoagulation, preferably using LMWH. If these patients   
have a condition where anticoagulation is not promising (complete, extended,   
chronic PVT) or ineffective, or if they are candidates for liver   
transplantation, the TIPS may be implanted without delay.  
  
RECORD 325  
Nonsurgical therapeutic options in portal vein thrombosis  
Schultheiß M. Bettinger D. Thimme R.  
Viszeralmedizin: Gastrointestinal Medicine and Surgery (2014) 30:6   
(388-392). Date of Publication: 21 Jan 2014  
  
Background: portal vein thrombosis (PVT) is a rare but severe vascular   
disorder with an acute and a chronic course. Most patients have underlying   
liver cirrhosis; furthermore, thrombophilia is an important risk factor.   
However, idiopathic forms are also known. Methods: This review discusses   
nonsurgical treatment options in PVT. Results and Conclusion: Therapy of   
acute PVT is based on anticoagulation with heparin that is switched to oral   
anticoagulants, if applicable. Catheter-guided invasive therapy should be   
considered; however, patients with liver cirrhosis should be screened for   
portal hypertension before anticoagulation is mandatory. Therapy of chronic   
PVT is discussed controversially; therefore, a strict patient selection and   
an individual therapeutic decision are warranted depending on the etiology   
of PVT. Special forms of PVT including septic and malignant thrombosis as   
well as PVT in patients waiting for liver transplantation require particular   
therapy algorithms.  
  
RECORD 326  
Analysis of factors associated with portal vein thrombosis in pediatric   
living donor liver transplant recipients  
Neto J.S. Fonseca E.A. Feier F.H. Pugliese R. Candido H.L. Benavides M.R.   
Porta G. Miura I.K. Danesi V.B. Guimaraes T. Porta A. Borges C. Godoy A.   
Kondo M. Chapchap P.  
Liver Transplantation (2014) 20:10 (1157-1167). Date of Publication: 1 Oct   
2014  
  
The technique of vascular reconstruction plays a major role in the outcome   
of living donor liver transplantation (LDLT). An increased use of vascular   
grafts (VGs) as replacements for sclerotic portal veins has become a   
standard technique for our group. The aim of this study was to analyze the   
factors associated with portal vein thrombosis (PVT) in pediatric LDLT. We   
performed a retrospective analysis of 486 primary pediatric LDLT procedures   
performed between October 1995 and May 2013. VGs used for portal   
reconstruction included living donor inferior mesenteric veins, living donor   
ovarian veins, recipient internal jugular veins, deceased donor iliac   
arteries, and deceased donor iliac veins. Thirty-four patients (7.0%)   
developed PVT. The incidence of PVT dropped from 10.1% to 2%; the overall   
utilization of VGs increased from 3.5% to 37.1%. In a multivariate analysis,   
only the use of VGs remained an independent risk factor for the occurrence   
of PVT (hazard ratio=7.2, 95% confidence interval=2.8-18.7, P<0.001). There   
was no difference in survival rates between patients with PVT and patients   
without PVT. No patient with PVT underwent retransplantation. In conclusion,   
the use of VGs was independently associated with the development of PVT.   
Over time, there was a reduction in the incidence of early PVT in this   
cohort, and there was a trend toward a reduction in total PVT. The   
occurrence of isolated PVT in this study was not associated with decreased   
patient or graft survival.  
  
RECORD 327  
Treatment of acute portal vein thrombosis by nontraditional anticoagulation  
Martinez M. Tandra A. Vuppalanchi R.  
Hepatology (2014) 60:1 (425-426). Date of Publication: July 2014  
  
RECORD 328  
Treatment of thromboembolic events coincident with the diagnosis of   
myeloproliferative neoplasms: A physician survey  
Ellis M.H. Lavi N. Vannucchi A. Harrison C.  
Thrombosis Research (2014) 134:2 (251-254). Date of Publication: 2014  
  
The BCR-ABL1 negative myeloproliferative neoplasms (MPNs) are associated   
with an increased risk of both venous and arterial thromboembolic events.   
Thromboses may be the presenting clinical feature of an MPN or may occur   
during the course of the disease. Treatment comprises anticoagulant and   
antiaggregant agents as in non- MPN thromboses, and treatment of the   
particular MPN. The duration of anticoagulant treatment that is required for   
MPN thrombosis is unknown. This study was performed to survey the opinion of   
hematologists who treat patients with MPN regarding the duration of   
anticoagulation or antiaggregant therapy in patients in whom thrombosis is   
the presenting feature of MPN. Five clinical scenarios in which   
thromboembolism (cerebral vein thrombosis, pulmonary embolism,   
cerebrovascular accident, splanchnic vein thrombosis, portal vein   
thrombosis) was a presenting feature of MPN were created using a web-based   
tool and were sent by email to hematologists in Israel, Italy and England   
and to hematologists identified as key opinion leaders in the field of MPN.   
Physicians were asked to recommend duration of anticoagulation and/or   
aspirin use choosing from 4 alternatives provided. Seventy-three physicians   
responded to the survey. 42 physicians considered MPNs to be their main area   
of clinical interest, and 31 did not. 21 physicians saw more than 20 MPN   
patients per week, and 50 physicians had been in hematology practice for   
more than 10 years. Responses regarding the duration of anticoagulation   
and/or the use of aspirin varied for all of the clinical vignettes. Neither   
physician area-of-interest, volume of MPN patients treated nor years in   
practice were related to the responses obtained. This study demonstrates   
that hematologists, including those specializing in MPNs, lack consensus in   
their approach to the long-term treatment of thromboses as the presenting   
feature of an MPN. Controlled clinical studies are needed to inform   
appropriate decision making in this area. © 2014 Elsevier Ltd.  
  
RECORD 329  
Portal and splenic vein thrombosis successfully treated with anticoagulants   
in acute pancreatitis  
Seong J.S. Song J.H. Cho K.P. Lee J.S. Woo Y.M. Jeong B.J. Cho Y.J. Han Y.J.  
EWHA Medical Journal (2014) 37:2 (116-120). Date of Publication: 2014  
  
Splanchnic vein thrombosis arising from complications of acute pancreatitis   
is very rare. It usually occurs as a form of portal, splenic and superior   
mesenteric vein thrombosis, either in combination or separately. It could   
develop portal hypertension, bowel ischemia and gastrointestinal variceal   
bleeding. Treatment of splanchnic vein thrombosis includes anticoagulants,   
thrombolysis, insertion of shunts, bypass surgery and liver transplantation.   
In some cases, anticoagulation therapy may be considered to prevent   
complications. However, the standard protocol for anticoagulation in   
splanchnic vein thrombosis has not been determined yet. We report a case of   
43-year-old man who had portal and splenic vein thrombosis in acute   
pancreatitis. The patient was successfully treated with oral anticoagulants   
following low molecular weight heparin therapy.  
  
RECORD 330  
Managing periprocedural thrombocytopenia in cirrhosis: Aiming for a safety   
window  
Bissonnette J. Valla D. Rautou P.-E.  
Journal of Hepatology (2014) 61:6 (1199-1201). Date of Publication: 1 Dec   
2014  
  
RECORD 331  
Safety, efficacy, and response predictors of anticoagulation for the   
treatment of nonmalignant portal-vein thrombosis in patients with cirrhosis:   
A propensity score matching analysis  
Chung J.W. Kim G.H. Lee J.H. Ok K.S. Jang E.S. Jeong S.-H. Kim J.-W.  
Clinical and Molecular Hepatology (2014) 20:4 (384-391). Date of   
Publication: 2014  
  
Background/Aims: Portal-vein thrombosis (PVT) develops in 10–25% of   
cirrhotic patients and may aggravate portal hypertension. There are few data   
regarding the effects of anticoagulation on nonmalignant PVT in liver   
cirrhosis. The aim of this study was to elucidate the safety, efficacy, and   
predictors of response to anticoagulation therapy in cirrhotic patients.   
Methods: Patients with liver cirrhosis and nonmalignant PVT were identified   
by a hospital electronic medical record system (called BESTCARE). Patients   
with malignant PVT, Budd-Chiari syndrome, underlying primary hematologic   
disorders, or preexisting extrahepatic thrombosis were excluded from the   
analysis. Patients were divided into two groups (treatment and   
nontreatment), and propensity score matching analysis was performed to   
identify control patients. The sizes of the thrombus and spleen were   
evaluated using multidetector computed tomography. Results: Twenty-eight   
patients were enrolled in this study between 2003 and 2014: 14 patients who   
received warfarin for nonmalignant PVT and 14 patients who received no   
anticoagulation. After 112 days of treatment, 11 patients exhibited   
significantly higher response rates (complete in 6 and partial in 5)   
compared to the control patients, with decreases in thrombus size of >30%.   
Compared to nonresponders, the 11 responders were older, and had a thinner   
spleen and fewer episodes of previous endoscopic variceal ligations, whereas   
pretreatment liver function and changes in prothrombin time after   
anticoagulation did not differ significantly between the two groups. Two   
patients died after warfarin therapy, but the causes of death were not   
related to anticoagulation. Conclusions: Warfarin can be safely administered   
to cirrhotic patients with nonmalignant PVT. The presence of preexisting   
portal hypertension is a predictor of nonresponse to anticoagulation.  
  
RECORD 332  
Treatment of nontumoral portal vein thrombosis in cirrhosis  
Bañares R. Catalina M.-V.  
Gastroenterologia y Hepatologia (2014) 37:S2 (62-67). Date of Publication:   
2014  
  
portal vein thrombosis in cirrhosis is a relatively common complication   
associated with the presence of an accompanying prothrombotic phenotype of   
advanced cirrhosis. The consequences of portal vein thrombosis are relevant   
because it can be associated with impaired hepatic function, might   
contraindicate hepatic transplantation and could increase morbidity in the   
surgical procedure. There is controversy concerning the most effective   
treatment of portal vein thrombosis, which is based on information that is   
seldom robust and whose primary objective is to achieve a return to vessel   
patency. Various studies have suggested that starting anticoagulation   
therapy early is associated with portal vein repatency more frequently than   
without treatment and has a low rate of complications. There are no proven   
data on the type of anticoagulant (low-molecular-weight heparins or   
dicoumarin agents) and the treatment duration. The implementation of TIPS is   
technically feasible in thrombosis without cavernous transformation and is   
associated with portal vein recanalization in a significant proportion of   
cases. Thrombolytic therapy does not appear to present an adequate balance   
between efficacy and safety; its use is therefore not supported for this   
indication. The proper definition of treatment for portal vein thrombosis   
requires properly designed studies to delimit the efficacy and safety of the   
various alternatives. © 2014 Elsevier España, S.L.  
  
RECORD 333  
Unexpected disappearance of portal cavernoma on long-term anticoagulation  
Silva-Junior G. Turon F. Hernandez-Gea V. Darnell A. García-Criado Á.   
García-Pagán J.C.  
Journal of Hepatology (2014) 61:2 (446-448). Date of Publication: August   
2014  
  
Idiopathic non-cirrhotic portal hypertension is a rare disease of unknown   
etiology. Patients with idiopathic non-cirrhotic portal hypertension have an   
increased risk of developing portal vein thrombosis and this is especially   
prevalent when HIV is also present. We describe a unique case of a patient   
with idiopathic non-cirrhotic portal hypertension associated to HIV, who   
developed acute portal vein thrombosis that despite anticoagulation   
transformed in portal cavernoma and disappeared completely after five years   
of follow-up on continuous anticoagulation.  
  
RECORD 334  
portal vein thrombosis in minimal change disease  
Kim G. Lee J.Y. Heo S.J. Kee Y.K. Han S.H.  
EWHA Medical Journal (2014) 37:2 (131-135). Date of Publication: 2014  
  
Among the possible venous thromboembolic events in nephrotic syndrome, renal   
vein thrombosis and pulmonary embolism are common, while portal vein   
thrombosis (PVT) is rare. This report describes a 26-year-old man with   
histologically proven minimal change disease (MCD) complicated by PVT. The   
patient presented with epigastric pain and edema. He had been diagnosed with   
MCD five months earlier and achieved complete remission with   
corticosteroids, which were discontinued one month before the visit.   
Full-blown relapsing nephrotic syndrome was evident on laboratory and   
clinical findings, and an abdominal computed tomography revealed PVT. He   
immediately received immunosuppressants and anticoagulation therapy. An   
eight-week treatment resulted in complete remission, and a follow-up   
abdominal ultrasonography showed disappearance of PVT. In conclusion, PVT is   
rare and may not be easily diagnosed in patients with nephrotic syndrome   
suffering from abdominal pain. Early recognition of this rare complication   
and prompt immunosuppression and anticoagulation therapy are encouraged to   
avoid a fatal outcome.  
  
RECORD 335  
Combined pharmacomechanical thrombolysis of complete portomesenteric   
thrombosis in a liver transplant recipient  
Lorenz J.M. Bennett S. Patel J. Van Ha T.G. Funaki B.  
CardioVascular and Interventional Radiology (2014) 37:1 (262-266). Date of   
Publication: February 2014  
  
Treatment options for portomesenteric venous thrombosis range from   
anticoagulation to surgery, depending on chronicity, severity of symptoms,   
extent of thrombosis, and the availability of local expertise. For acute and   
subacute cases, a variety of endovascular options have been described in   
limited published series and case reports, including thrombolysis and   
mechanical thrombectomy. We report what is to our knowledge the first case   
in which the Trellis pharmacomechanical thrombolysis device was used   
successfully to treat complete acute thrombosis of the entire superior   
mesenteric vein and the entire portal vein with extension into all segmental   
intrahepatic portal branches in a young adult after liver transplantation.   
This device, coupled with adjunctive techniques using balloon catheters,   
facilitated complete restoration of flow, resulting in graft salvage and   
long-term patency. © 2013 Springer Science+Business Media New York and the   
Cardiovascular and Interventional Radiological Society of Europe (CIRSE).  
  
RECORD 336  
Deep vein thrombosis and pulmonary embolism in cirrhotic patients:   
Systematic review  
Aggarwal A. Puri K. Liangpunsakul S.  
World Journal of Gastroenterology (2014) 20:19 (5737-5745). Date of   
Publication: 2014  
  
Patients with liver cirrhosis were traditionally believed to be protected   
against development of blood clots. Lately, studies have shown that these   
patients may probably be at an increased risk of venous thrombotic   
complications. Although the hemostatic changes in the chronic liver disease   
patients and the factors that may predict bleeding vs thrombotic   
complications remains an area of active research, it is believed that the   
coagulation cascade is delicately balanced in these patients because of   
parallel reduced hepatic synthesis of pro and anticoagulant factors.   
Thrombotic state in cirrhotic patients is responsible for not only portal or   
non-portal thrombosis [deep vein thrombosis (DVT) and pulmonary embolism   
(PE)]; it has also been associated with progression of liver fibrosis. The   
use of anticoagulants in cirrhosis patients is a challenging, and often a   
scary situation. This review summarizes the current literature on the   
prevalence of venous thrombosis (DVT and PE), risk factors and safety of   
prophylactic and therapeutic anticoagulation in patients with chronic liver   
disease. © 2014 Baishideng Publishing Group Inc. All rights reserved.  
  
RECORD 337  
Efficacy of postoperative anticoagulation therapy with enoxaparin for portal   
vein thrombosis after hepatic resection in patients with liver cancer  
Yamashita Y.-I. Bekki Y. Imai D. Ikegami T. Yoshizumi T. Ikeda T. Kawanaka   
H. Nishie A. Shirabe K. Maehara Y.  
Thrombosis Research (2014) 134:4 (826-831). Date of Publication: 1 Oct 2014  
  
Backgrounds: Enoxaparin, low-molecular-weight heparin, has become a routine   
thromboprophylaxis in general surgery. Study design: A retrospective cohort   
study was performed in 281 patients who underwent hepatic resections for   
liver cancers from 2011 to 2013. These patients were divided into two   
groups; an enoxaparin (-) group (n = 228) and an enoxaparin (+) group (n =   
53). Short-term surgical results including venous thromboembolism (VTE) and   
portal vein thrombosis (PVT) were compared. Results: In the enoxaparin (+)   
group, the patients' age (65 vs. 69 years; p = 0.01) and BMI (22.9 vs. 24.4;   
p < 0.01) were significantly higher. According to the symptomatic VTE,   
symptomatic pulmonary embolism occurred in one patient (0.4%) in the   
enoxaparin (-) group, but the complication rate was not significantly   
different (p = 0.63). The complication rate of PVT was significantly lower   
in the enoxaparin (+) group (10 vs. 2%; p = 0.04). The independent risk   
factors for PVT were an operation time ≥ 300 minutes (Odds ratio 6.66) and   
non-treatment with enoxaparin (Odds ratio 2.49). Conclusions: Postoperative   
anticoagulant therapy with enoxaparin could prevent PVT in patients who   
underwent hepatic resection for liver cancers.  
  
RECORD 338  
Chronic idiopathic non-cirrhotic portal vein thrombosis treated with a   
mesocaval shunt procedure and anticoagulation  
Shaaban H. Shah N. Sidhom I.  
Indian Journal of Hematology and Blood Transfusion (2014) 30:3 (211-212).   
Date of Publication: September 2014  
  
portal vein thrombosis (PVT) was first reported in 1868 by Balfour and   
Stewart and is a medical condition in which the lumen of the portal vein is   
completely or partially obstructed due to the presence of a thrombus [1].   
Inherited (Factor V Leiden and Prothrombin gene mutation G201210A, Protein   
C, S and Anti thrombin III deficiency) and acquired thrombophilias (Lupus   
Anticoagulant, myeloproliferative diseases, malignancy, surgery and trauma)   
account for majority of the cases of PVT. © 2013 Indian Society of   
Haematology & Transfusion Medicine.  
  
RECORD 339  
portal vein thrombosis associated with an acute cytomegalovirus infection  
Galloula A. Rossi A. Gautier V. Minozzi C. Messas E. Mirault T.  
Journal des Maladies Vasculaires (2014) 39:3 (224-230). Date of Publication:   
May 2014  
  
portal vein thrombosis is an unusual condition and its association with an   
acute cytomegalovirus (CMV) infection is known but rarely reported. We   
present here the case of a 24-year-old woman suffering from a symptomatic   
portal vein thrombosis, confirmed by CT angiography, and acute CMV-related   
hepatitis. Besides a second generation oral contraceptive with estrogen and   
progesterone, not associated with smoking, the acute CMV infection was the   
only cause found to have provoked the venous thrombosis; a   
myeloproliferative disorder or biological thrombophilia were ruled out. The   
patient rapidly recovered with vitamin K antagonists (VKA) anticoagulant   
treatment. Eighteen cases of splanchnic vein thrombosis complicating acute   
CMV infection were found in the literature. All patients had acute   
hepatitis. The outcome was usually favorable with warfarin therapy for a   
period lasting 3to 7months. Antiviral treatment (anti-CMV) was used in three   
cases of severe infection. The antiviral therapy was given only in   
immunosuppressed patients. For immunocompetent patients, CMV infection is   
usually asymptomatic and clinical signs are often non-specific and mild, not   
requiring treatment. Conclusion: This case report and the review of the   
literature recall the need to search for acute CMV infection in patients   
with portal thrombosis so a possible transient trigger for venous   
thromboembolism can be identified, avoiding extended anticoagulation. © 2014   
Elsevier Masson SAS.  
  
RECORD 340  
Intra-abdominal venous thrombosis after colectomy in pediatric patients with   
chronic ulcerative colitis: Incidence, treatment, and outcomes  
Antiel R.M. Hashim Y. Moir C.R. Rodriguez V. Elraiyah T. Zarroug A.E.  
Journal of Pediatric Surgery (2014) 49:4 (614-617). Date of Publication:   
April 2014  
  
Purpose Children with chronic ulcerative colitis (CUC) are at increased risk   
for venous thromboembolism, especially after colectomy procedures. We aim to   
review our patients with CUC who underwent a colectomy and suffered   
intra-abdominal thrombosis; moreover we wanted to define thrombotic   
incidence and outcomes Methods In this is IRB approved retrospective study,   
we reviewed our patients who underwent colectomy for CUC from January 1999   
to December 2011 for development of intra-abdominal thrombosis. Results Of   
366 patients with CUC who underwent colectomy, 15 (4%) were diagnosed with a   
venous thromboembolism. All patients presented with acute abdominal pain.   
The locations of thrombus formation varied: 13 (87%) developed thrombi in   
the portal vein, 4 (27%) in the splenic vein, 2 (13%) in the superior   
mesenteric vein, 1 (7%) in the hepatic vein, and 1 (7%) in the hepatic   
artery. The mean number of post-operative days at diagnosis of thrombus was   
38.7 days (range 3-180 days). Fourteen patients (93%) underwent   
anticoagulation for treatment. The mean number of days of anticoagulant   
therapy until documented resolution of thrombus on imaging was 96.3 days   
(range 14-364 days). All thrombi resolved with therapy. There was no   
mortality during follow-up. Conclusions Four percent of our pediatric   
patients with chronic ulcerative colitis who underwent colectomy developed   
symptomatic intra-abdominal venous thromboembolism. 3 to 6 months of   
anticoagulant therapy is adequate treatment in almost all patients.   
Practitioners should have a high index of suspicion for intra-abdominal   
venous thrombus when these patients complain of abdominal pain   
postoperatively. Based on our experience, prophylactic anticoagulation   
should be strongly considered peri-operatively in this population. © 2014   
Elsevier Inc.  
  
RECORD 341  
Portal, mesenteric, and splenic vein thromboses after endovascular   
embolization for gastrointestinal bleeding caused by a splenic arteriovenous   
Fistula  
Ding P. Li Z. Han X.-W. Wang Z.-G. Zhang W.-G. Fu M.-T.  
Annals of Vascular Surgery (2014) 28:5 (1322.e1-1322.e5). Date of   
Publication: July 2014  
  
We present an unusual case of portal, mesenteric, and splenic vein   
thromboses after endovascular embolization for gastrointestinal bleeding   
caused by a splenic arteriovenous fistula. The thromboses were successfully   
treated with anticoagulation therapy. The patient was a 37-year-old woman   
who presented with portal hypertension manifested by gastrointestinal   
bleeding with no evidence of liver disease. Splenic arteriography confirmed   
the presence of a high-flow arteriovenous fistulous communication from the   
splenic artery directly into the splenic vein. The arteriovenous fistula was   
successfully treated with percutaneous transarterial embolization by   
embolization coils and the patient achieved effective hemostasis.   
Low-molecular-weight heparin and warfarin were administrated to prevent   
thrombosis in the portal venous system after the procedure. Although   
anticoagulants were immediately administered, thromboses of the portal,   
mesenteric, and splenic veins were diagnosed by contrast-enhanced computed   
tomography after 10 days. Complete recanalization of the portal venous   
system confirmed by contrast-enhanced computed tomography was achieved by   
administering warfarin orally for 3 months. © 2014 Elsevier Inc. All rights   
reserved.  
  
RECORD 342  
portal vein thrombosis: A clinician-oriented and practical review  
Handa P. Crowther M. Douketis J.D.  
Clinical and Applied Thrombosis/Hemostasis (2014) 20:5 (498-506). Date of   
Publication: July 2014  
  
With advances in modern imaging techniques, portal vein thrombosis (PVT) is   
being increasingly diagnosed. It has a wide ranging clinical spectrum from   
being an asymptomatic state to a potentially life-threatening situation. It   
is not unusual to find it as an incidental finding in the abdominal imagings   
done for other reasons. It is commonly associated with cirrhosis and   
abdominal malignancies and also has a strong association with prothrombotic   
disorders. It is often difficult for the clinicians to decide whether PVT is   
acute or chronic. This poses great challenges to its management strategies   
that include anticoagulants, thrombolysis, and surgical options. Timely   
diagnosis and appropriate management have great bearings on its outcomes of   
morbidity and mortality. In this clinician-oriented review, we have provided   
a concise review of clinical aspects of PVT and discussed various management   
strategies while addressing the common questions that come to a physician's   
mind dealing with such a patient. © 2013 The Author(s).  
  
RECORD 343  
Imaging in clinical decision-making for portal vein thrombosis  
Berzigotti A. García-Criado Á. Darnell A. García-Pagán J.-C.  
Nature Reviews Gastroenterology and Hepatology (2014) 11:5 (308-316). Date   
of Publication: May 2014  
  
Thrombosis of the portal venous system is a frequent and potentially   
life-threatening condition that can take place in a number of different   
clinical settings including liver cirrhosis, hepatocellular carcinoma, other   
solid tumours, abdominal septic foci, acute pancreatitis, haematological   
malignancies and congenital or acquired prothrombotic disorders. Clinical   
decision-making in patients with thrombosis of the portal venous system is a   
particularly complex process owing to the heterogeneity of the population   
affected by this condition and the lack of high-quality evidence from   
randomized controlled trials for the use of anticoagulation therapy in these   
patients. This Review discusses the available data regarding how imaging can   
provide assistance to physicians involved in this decision-making process in   
different clinical settings. A flowchart illustrating how to use imaging in   
this setting, based on current evidence and on the experience of the   
Vascular Liver Diseases Group of the Hospital Clinic in Barcelona, is also   
presented. © 2014 Macmillan Publishers Limited. All rights reserved.  
  
RECORD 344  
Inherited Thrombophilia and the Risk of portal vein thrombosis: Progress   
Toward Individualized anticoagulation in cirrhosis?  
Fallon M.B. Batra S.  
Clinical Gastroenterology and Hepatology (2014) 12:11 (1813-1814). Date of   
Publication: 1 Nov 2014  
  
RECORD 345  
Therapeutic effects of laparoscopic splenectomy and esophagogastric   
devascularization on liver cirrhosis and portal hypertension in 204 cases  
Cheng Z. Li J.-W. Chen J. Fan Y.-D. Guo P. Zheng S.-G.  
Journal of Laparoendoscopic and Advanced Surgical Techniques (2014) 24:9   
(612-616). Date of Publication: 1 Sep 2014  
  
Objective: To investigate the effects and technical points of laparoscopic   
splenectomy and esophagogastric devascularization (LS+ED) for portal   
hypertension (PH) due to liver cirrhosis. Subjects and Methods: In total,   
204 PH patients who underwent LS+ED from January 2008 to April 2013 in the   
Southwest Hospital of the Third Military Medical University were enrolled in   
this study. We retrospectively analyzed the clinical data and the key   
technical points and compared the results with other researchers. Results:   
LS+ED was successfully carried out on 188 patients. The mean duration of   
surgery was 232±59 minutes, the mean intraoperative blood loss was 189±137   
mL, the rate of blood transfusion was 19.6% (40/204), and no deaths occurred   
during surgery. The mean postoperative interval to passing of flatus was   
3.5±0.9 days, and the mean postoperative hospital stay was 8.7±2.2 days.   
Operative complications occurred in 100 patients, of whom 78 had portal vein   
system thrombosis (PVST). During a postoperative follow-up period of 2-65   
months, 15 cases were lost to follow-up, esophagogastric variceal bleeding   
re-occurred in 7 patients, encephalopathy occurred in 2 patients, and   
secondary liver cancer occurred in 3 patients. Five patients died during   
this period. Conclusions: The technical points of LS+ED include a combined   
surgical approach, a reasonable surgical procedure, and an appropriate   
laparoscopic operating plane. LS+ED is a safe and effective treatment for   
minimal trauma and rapid recovery. PVST is a common and potentially   
life-threatening complication after LS+ED, and anticoagulation therapy   
should be given early.  
  
RECORD 346  
Idiopathic portal hypertension: Natural history and long-term outcome  
Siramolpiwat S. Seijo S. Miquel R. Berzigotti A. Garcia-Criado A. Darnell A.   
Turon F. Hernandez-Gea V. Bosch J. Garcia-Pagán J.C.  
Hepatology (2014) 59:6 (2276-2285). Date of Publication: June 2014  
  
Idiopathic portal hypertension (IPH) is a rare cause of intrahepatic portal   
hypertension. Data on natural history and prognosis of IPH are limited. We   
sought to describe the complications and long-tem outcome of IPH by   
retrospectively studying 69 biopsy-proven cases of IPH. Mean duration of   
follow-up was 6.7±4.6 years. All patients had evidence of portal   
hypertension (PH) at diagnosis, and 42% were symptomatic. Variceal bleeding   
(VB) was the most common manifestation. In those without bleeding at   
diagnosis, 74% had varices at first endoscopy. In those with large varices,   
the 1-year probability of first bleeding despite primary prophylaxis was 9%.   
The 1-year probability of rebleeding was 22%. Ascites and hepatic   
encephalopathy was documented in 26% and 7% of patients, respectively, at   
least once during the clinical course. The 1-year probability of developing   
portal vein thrombosis (PVT) was 9%, and 53% of patients receiving   
anticoagulation achieved recanalization. Human immunodeficiency virus (HIV)   
infection and VB at diagnosis were the independent predictors of PVT. Seven   
patients died (6 as a result of an IPH-related cause) and 2 were   
transplanted. Probability of liver transplantation-free survival was 82% at   
10 years. Presence of a severe associated disorder and ascites as a   
presenting symptom were associated with poor survival. Conclusion: Variceal   
bleeding is a major complication of IPH. Using, in IPH patients, the same   
management approach for PH as in cirrhosis is safe and maintains a low   
incidence of first bleeding and rebleeding in IPH patients. PVT is a   
frequent complication, particularly in those with HIV infection. Despite   
several complications, overall survival of patients with IPH is considerably   
good. © 2014 by the American Association for the Study of Liver Diseases.  
  
RECORD 347  
Segmental grafts in adult and pediatric liver transplantation: Improving   
outcomes by minimizing vascular complications  
Rodriguez-Davalos M.I. Arvelakis A. Umman V. Tanjavur V. Yoo P.S. Kulkarni   
S. Luczycki S.M. Schilsky M. Emre S.  
JAMA Surgery (2014) 149:1 (63-70). Date of Publication: January 2014  
  
IMPORTANCE The use of technically variant segmental grafts are key in   
offering transplantation to increase organ availability. OBJECTIVE To   
describe the use of segmental allograft in the current era of donor   
scarcity, minimizing vascular complications using innovative surgical   
techniques. DESIGN, SETTING, AND PARTICIPANTS Retrospective study from   
August 2007 to August 2012 at a university hospital. A total of 218   
consecutive liver transplant patients were reviewed, and 69 patients (31.6%;   
38 males and 31 females; mean age, 22.5 years) received segmental grafts   
from living donors or split/reduced-size grafts from deceased donors. MAIN   
OUTCOMES AND MEASURES Graft type, vascular and biliary complications, and   
patient and graft survival. RESULTS Of 69 segmental transplants, 47 were   
living donor liver transplants: 13 grafts (27.7%) were right lobes, 22   
(46.8%) were left lobes, and 12 (25.5%) were left lateral segments.   
Twenty-two patients received deceased donor segmental grafts; of these, 11   
(50.0%) were extended right lobes, 9 (40.9%) were left lateral segments, 1   
(4.5%) was a right lobe, and 1 (4.5%) was a left lobe. Arterial anastomoses   
were done using 8-0 monofilament sutures in an interrupted fashion for   
living donor graft recipients and for pediatric patients. Most patients   
received a prophylactic dose of low-molecular-weight heparin for a week and   
aspirin indefinitely. There was no incidence of hepatic artery or portal   
vein thrombosis. Two patients developed hepatic artery stenosis and were   
treated with balloon angioplasty by radiology. Graft and patient survivals   
were 96% and 98%, respectively. CONCLUSIONS AND RELEVANCE Use of segmental   
allografts is essential to offer timely transplantation and decrease waiting   
list mortality. Living donor liver transplants and segmental grafts from   
deceased donors are complementary. It is possible to have excellent outcomes   
combining a multidisciplinary team approach, technical expertise, routine   
use of anticoagulation, and strict patient and donor selection. Copyright   
2014 American Medical Association. All rights reserved.  
  
RECORD 348  
Therapeutic strategies of iatrogenic portal vein injury after   
cholecystectomy  
Wang Z. Yu L. Wang W. Xia J. Li D. Lu Y. Wang B.  
Journal of Surgical Research (2013) 185:2 (934-939). Date of Publication:   
December 2013  
  
Background: The incidence of vascular injury after a cholecystectomy is   
often underestimated. Although injuries to the portal vein are rare, they   
are devastating. The aim of the present study was to analyze suitable   
therapeutic strategies regarding portal vein injury in the absence of   
biliary injury. Materials and methods: Eleven patients with portal vein   
injuries after laparoscopic or open cholecystectomy were referred to our   
hospital between 2004 and 2010. The clinical presentation, diagnosis, and   
management of patients with severe portal vein injuries were reviewed. All   
the patients were discharged without outstanding clinical conditions. During   
retrospective analysis, these patients were divided into early, middle, and   
late stages. Results: All the 11 patients had a portal vein and/or right   
hepatic artery injury, but no biliary injuries were observed. Among these   
patients, different management strategies were managed according to the   
stage of the injury. Eight patients received a direct suture at the time of   
injury by an experienced hepatobiliary surgeon. Two patients received   
thrombolytic and anticoagulation therapy after cholecystectomy, without   
additional surgery. One patient received a liver transplant 3 mo after the   
injury. After long-term follow-up, these patients had no clinical   
conditions. Conclusions: Direct repair or suture is important during the   
early stage of portal vein injury. Conservative thrombolytic and   
anticoagulation therapy may serve an important role in the treatment of   
acute massive thrombus in portal vein injury during the middle stage. Liver   
transplantation is a salvage therapy that should be used during the late   
stage. © 2013 Elsevier Inc. All rights reserved.  
  
RECORD 349  
portal vein thrombosis after partial splenic embolization in liver   
cirrhosis: Efficacy of anticoagulation and long-term follow-up  
Cai M. Zhu K. Huang W. Meng X. He K. Zhou B. Guo Y. Chen J. Shan H.  
Journal of Vascular and Interventional Radiology (2013) 24:12 (1808-1816).   
Date of Publication: December 2013  
  
Purpose To investigate the treatment and long-term outcome of portal vein   
thrombosis (PVT) after partial splenic embolization (PSE). Materials and   
Methods From January 2006 to December 2011, 145 patients with hypersplenism   
caused by cirrhotic portal hypertension underwent PSE. In 11 cases, PVT was   
detected 13-42 days after PSE. Among the 11 patients, 5 underwent   
anticoagulant therapy because of clinical symptoms, and 6 did not receive   
anticoagulation because they were symptom-free (4 patients) or experienced   
variceal bleeding (2 patients). The long-term follow-up data from these 11   
patients were analyzed retrospectively. Results The 11 patients with PVT had   
a mean splenic infarction ratio of 71.5%. The mean duration of follow-up was   
37.6 months. During the follow-up period, none of the 5 patients who   
underwent anticoagulation developed variceal hemorrhage despite presenting   
with large esophagogastric varices. Four of the five patients achieved   
complete resolution of thrombosis, and one did not develop thrombus   
progression. However, among the 6 patients who did not undergo   
anticoagulation, 2 developed esophagogastric variceal hemorrhage secondary   
to thrombus progression, 3 developed cavernous transformation of the portal   
vein and variceal progression, and 1 had partial calcification of the   
thrombus. Two patients who had variceal bleeding or rebleeding underwent a   
transjugular intrahepatic portosystemic shunt. Complete recanalization of   
the portal vein was achieved after the procedures. Conclusions PVT is a   
severe, potentially fatal complication of PSE. Early detection of PVT and   
prompt anticoagulation are effective to avoid serious consequences of PVT. ©   
2013 SIR.  
  
RECORD 350  
Usefulness of conventional mri sequences and diffusion-weighted imaging in   
differentiating malignant from benign portal vein thrombus in cirrhotic   
patients  
Sandrasegaran K. Tahir B. Nutakki K. Akisik F.M. Bodanapally U. Tann M.   
Chalasani N.  
American Journal of Roentgenology (2013) 201:6 (1211-1219). Date of   
Publication: December 2013  
  
OBJECTIVE. The objective of our study was to determine the value of   
diffusion-weighted imaging (DWI) and conventional MRI (non-DWI sequences) in   
differentiating benign portal vein thrombus (PVT) from malignant PVT in   
cirrhotic patients. MATERIALS AND METHODS. A retrospective search of the   
department of radiology's MRI database of examinations performed from   
October 2006 through December 2010 for "portal vein thrombosis" and   
"cirrhosis" and "hepatocellular cancer" was performed. Patients who   
underwent diagnostic DWI and had thrombus shown to be rapidly (< 3 months)   
increasing in size despite anticoagulation therapy were considered to have   
malignant PVT (n = 16 cases) and patients with MRI findings showing   
stability or reduction in the extent of thrombus over a 12-month follow-up   
were considered to have benign PVT (n = 20 cases). Two blinded and   
independent reviewers analyzed the DW images and conventional MR images.   
RESULTS. There was no difference in the distribution of patients by age (p =   
0.25) or sex (p = 0.68) between the benign and malignant PVT groups. On   
multivariate analysis, the only parameter to predict the type of PVT was the   
size of HCC (p = 0.05); other parameters were excluded from the model. There   
was substantial overlap in apparent diffusion coefficient (ADC) values and   
PVT/liver ADC ratios of benign PVT and malignant PVT. The presence of at   
least two of the three following MRI findings had a sensitivity of 100% and   
specificity of 90% for the diagnosis of malignant PVT: distance from tumor   
to PVT of less than 2 cm, HCC size of greater than 5 cm, and arterial   
enhancement of PVT. CONCLUSION. Signal-intensity characteristics on DWI and   
measured ADC values do not reliably differentiate benign PVT from malignant   
PVT. On the other hand, careful assessment of conventional MRI findings may   
allow this distinction, thus obviating biopsy.  
  
RECORD 351  
Case Series: Thrombus Resolution in 2 Patients with portal vein thrombosis   
Without anticoagulation—Do We Need to Anticoagulate Patients with Portal   
Vein Thrombosis?  
Sule A. A. Borja A. M. Xing W. Lymen E. Azucena B. Chin T. J. Lymen E.  
Annals of the Academy of Medicine, Singapore (2013) 42 No. 8 Supplement   
(S11-S11). Date of Publication: August 2011  
  
Introduction: portal vein thrombosis (PVT) is thrombosis that develops in   
the trunk of the portal vein which can extend to its branches. It results   
from a combination of local and systemic prothrombotic factors. Methods: We   
describe 2 cases for this study. Patient 1 is a 77-year-old male who was   
admitted for cholangitis and pancreatitis and was found to have an   
incidental PVT. Patient 1’s investigations and laboratory workup: total   
white count 23.0x10(9)/L (neutrophils 91.6%), haemoglobin 11.7g/dL,   
platelets 147x10(9)/L; total bilirubin 184umol/L, alanine transaminase   
111U/L, aspartate transaminase 113 U/L, gamma-glutamyltranspeptidase 515   
U/L; amylase 641 U/L; hepatitis screening was negative. Abdominal computed   
tomography (CT) scan showed cholangitis with common bile duct calculi and an   
incidental thrombosis of the segmental branches of the right portal vein.   
Thrombophilia screen was negative. Patient 2 is a 60-year-old female with   
child’s B cryptogenic liver cirrhosis and was admitted for gastroenteritis   
and left breast lump. She was found to have an incidental non-occlusive   
thrombus in the main portal vein. Her investigations and laboratory workup   
were as follows: total white cell 6.2x10(9)/L (neutrophils 73.1%),   
haemoglobin 9.1g/dL, platelets 116x10(9)/L; Na 133 mmol/L, K 4.6 mmol/L,   
creatinine 115 umol/L; albumin 29g/L, total bilirubin 25 umol/L, alanine   
transaminase 27U/L, aspartate transaminase 42 U/L, C-reactive protein   
15.5mg/L. CT scan showed left breast mass, cirrhosis with portal   
hypertension and non-occlusive portal vein thrombus. Results: Patient 1   
underwent endoscopic retrograde cholangiopancreatography (ERCP), removal of   
stones and was given antibiotics. Patient was not anticoagulated due to the   
ongoing infection. A repeat CT scan 6 months later showed no evidence of   
PVT. Patient 2 underwent peritoneal drainage and was given antibiotics. No   
anticoagulation was given due to low platelet count. Eleven months later, an   
ultrasound Doppler of the hepatobiliary system revealed no evidence of   
vascular thrombosis. The left breast mass was later noted to be an invasive   
adenocarcinoma. Conclusion: The decision to anticoagulate a patient with   
portal vein thrombosis depends on several factors. Spontaneous resolution is   
possible but is an uncommon occurrence.  
  
RECORD 352  
Splanchnic vein thrombosis in acute pancreatitis: A single-center experience  
Harris S. Nadkarni N.A. Naina H.V. Vege S.S.  
Pancreas (2013) 42:8 (1251-1254). Date of Publication: November 2013  
  
OBJECTIVES: This study aimed to estimate outcomes of splanchnic vein   
thrombosis (SVT) in hospitalized patients with acute pancreatitis (AP).   
METHODS: This was a retrospective study (January 1996 to December 2006) via   
chart review. RESULTS: Over 10 years, 1.8% (45/2454) of patients with AP   
with a mean (SD) age of 58 (15) years were diagnosed with SVT. Splenic vein   
thrombosis was the most common form of SVT (30/45 patients, 67%). Seventeen   
patients were anticoagulated with heparin, when the SVT was diagnosed in the   
acute stage followed by oral anticoagulation (AC). The thrombosis that was   
most commonly anticoagulated was portal vein thrombosis in 11 (65%) of 17   
patients. Of 17 patients in the AC group, 2 (12%) showed recanalization as   
compared with 3 (11%) of 28 patients in the non-AC group (P > 0.05). The   
mortality was 3 (7%) of 45 (2 from the AC group versus 1 in the non-AC   
group, P > 0.05). Two of these died of multiorgan failure, and the other,   
from septic shock. None of the deaths were due to bleeding complications.   
CONCLUSIONS: Splanchnic vein thrombosis occurred in 1.8% patients of AP. The   
use of AC was reasonably safe with no fatal bleeding complications. However,   
there was no significant difference in the recanalization rates in those   
with and without AC. Copyright © 2013 Lippincott Williams & Wilkins.  
  
RECORD 353  
Liver transplantation in budd-chiari syndrome: A single centre experience in   
Saudi Arabia  
Saleh Y. Eldeen F.Z. Kamel Y. Kabbani M. Al-Sebayel M. Broering D.  
Transplant International (2013) 26 SUPPL. 2 (307). Date of Publication:   
November 2013  
  
Background: Patients suffering from Budd Chiari Syndrome are considered as   
potential candidates for liver transplantation (LT) if not responding to   
other modalities. Early intervention in relatively stable can improve the   
outcome and survival. Patient and methods: This is the first article from   
Saudi Arabia to describe our experience in LT in patients with BCS. Data for   
patients, who underwent LT between Mar 2001 and Oct 2012, were analyzed. Six   
patients with BCS underwent LT (1.4%). Diagnostic work up such as imaging   
modalities and hematological evaluation was part of work up. Results: All   
patients received whole liver transplant from deceased donor. They were   
started on therapeutic heparin infusion and triple therapy   
immunosuppression, according to our protocol, then warfarin was introduced   
for long term control. Two patients (33%) died; one from bleeding caused by   
DIC; second succumbed after 5 months of pneumonia and multiorgan failure.   
One patient had recurrence after portal vein thrombosis nine months post LT.   
The predictors of mortality in our cases are renal failure, previous   
abdominal surgery and low BMI. Conclusion: Treatment of BCS follows a   
therapeutic algorithm that should start by anticoagulation and might end by   
liver transplantation, which seems to be feasible in our experience.  
  
RECORD 354  
Impact of untreated portal vein thrombosis on pre and post liver transplant   
outcomes in cirrhosis  
John B.V. Konjeti R. Aggarwal A. Lopez R. Atreja A. Miller C. Zein N.N.   
Carey W.D.  
Annals of Hepatology (2013) 12:6 (952-958). Date of Publication:   
November/December 2013  
  
Background and aims. Most portal vein thromboses (PVT) in cirrhotics are   
discovered incidentally. While case series demonstrate improved portal vein   
patency with anti-coagulation, there is little information on impact of PVT   
on morbidity and mortality. This study aimed to compare morbidity and   
mortality in cirrhotics with untreated PVT with those without PVT. Material   
and methods. Cirrhotics evaluated for orthotopic liver transplant in a   
single large transplant center were prospectively followed. Subjects had   
contrast CT or MRI at initial evaluation and serial imaging every 6 months   
until transplantation, removal from the list or death. Univariate and   
multivariate Cox regression analysis were used to assess associations   
between new PVT and factors of interest. Results. Of the 290 prospectively   
followed cirrhotics who met inclusion criteria, PVT was detected in 70   
(24.1%)-47 had PVT at the time of initial evaluation and 23 developed one   
during the pre-transplant study period. A third of the patients with PVT had   
re-canalization or spontaneous resolution of thrombus while awaiting   
transplantation. There was no difference in the pre or posttransplant   
mortality between cirrhotics with and without PVT. Conclusion. Cirrhotics   
with untreated PVT fared equally well as those without PVT before and after   
transplantation. Further studies with larger numbers of patients are needed   
to determine if anticoagulation therapy truly improves outcomes in   
cirrhotics with portal vein thrombosis.  
  
RECORD 355  
Occult pulmonary mucosa-associated lymphoid tissue lymphoma presenting as   
catastrophic antiphospholipid antibody syndrome  
Regunath H. Shortridge J. Raza S. Nistala P. Huffman B.M. Wang M.X. Xiang D.  
Oncology Letters (2013) 6:5 (1261-1264). Date of Publication: November 2013  
  
Catastrophic antiphospholipid antibody syndrome (CAPS) is characterized by   
fulminant thrombosis of the arterial and venous beds of multiple organ   
systems over a relatively short period of time and with a high mortality   
rate. Mucosa-associated lymphoid tissue (MALT) lymphoma of the lung has   
never been reported as a causative or precipitating factor for CAPS in the   
CAPS registry database. The present study describes a rare case of pulmonary   
MALT lymphoma of the lung that presented as CAPS. A 19-year-old Hispanic   
female presented with shortness of breath and abdominal pain. Computed   
tomography (CT) scans of the chest and abdomen revealed multiple portal vein   
thromboses and bilateral pulmonary nodules. Within one week of presentation,   
the patient developed a straight sinus thrombosis and upper extremity deep   
vein thrombosis, which led to shortness of breath. A biopsy of the lung   
nodule revealed MALT lymphoma. The present case illustrates a rarely   
reported pulmonary MALT lymphoma presenting as CAPS in a young female. The   
patient was successfully treated with 90 mg/m(2) bendamustine on days one   
and two and rituximab 375 mg/m(2) on day one of each 28-day cycle. Complete   
remission of the lung nodules was observed following three cycles of   
treatment, as visualized by positron emission tomography (PET)/CT scan.   
Fondaparinux was identified as a feasible anticoagulation drug of choice for   
this case. At seven months post-treatment, the patient continues to be   
stable with no further evidence of thrombosis and is currently undergoing   
rituximab maintenance therapy every six months for two years. A repeat lupus   
anticoagulant antibody assay turned and remained negative during the   
clinical follow-up period. A prompt diagnosis and early aggressive treatment   
is potentially curative and may dramatically decrease the mortality risk.   
Future studies should explore the role of rituximab in the management of   
CAPS-associated B-cell lymphoid malignancies.  
  
RECORD 356  
Risk factors, diagnosis, management, and outcomes for splanchnic vein   
thrombosis: A retrospective analysis  
Derman B.A. Kwaan H.C.  
Blood (2013) 122:21. Date of Publication: 21 Oct 2013  
  
Background There is a paucity of data on the incidence of risk factors for   
splanchnic vein thrombosis in current published literature. The present   
study is an attempt to determine the risk factors, diagnostic methods   
employed, treatment modalities, and outcomes in patients with splanchnic   
vein thrombosis in a single institution over a two-year period. Methods   
Retrospective chart review of patients, 18-90 years old, who were diagnosed   
with splanchnic vein thrombosis (SVT) at a single institution from January   
1, 2010 to November 10, 2012. They were grouped as those with Budd-Chiari   
syndrome (BCS) and those with portal vein thrombosis (PVT), including those   
combined with splenic vein thrombosis (SPVT) and those with mesenteric vein   
thrombosis (MVT). Results Among the 246 patients studied, 21 had BCS and 225   
had PVT. Associated risk factors in the order of frequency were liver   
disease being present in 48% of BCS, 69% of PVT, 45% of PVT+SPVT, and 52% of   
PVT+MVT. Next was regional cancer, being present in 24%in BCS and 47% of   
PVT. Third commonest was pancreatitis being present in 14% of BCS, 9% of   
PVT, 18% of PVT+SPVT, and 6% of PVT+MVT. Hereditary thrombophilias were   
found in 10% of the BCS group and 4% of PVT; however, it constituted 18% of   
the PVT+SPVT group, and 12% of the PVT+MVT group. 10% of patients in both   
the BCS and PVT groups had a liver transplant during their lifetime. The   
most common presenting symptom was abdominal pain occurring in 57% patients   
with BCS and 50% patients with PVT. The majority had laboratory findings of   
liver dysfunction at presentation with 86% in BCS group and 78% in PVT   
group. JAK2 V617F mutation, when tested, was present in 14% of those with   
BCS, 20% of the PVT group, 29% of those with PVT+SPVT and 22% of those with   
PVT+MVT. Diagnosis of SVT was most commonly made by computerized tomography   
(CT) with contrast (57% for BCS, 56% for PVT). Approximately 60% of BCS   
patients and 30% of PVT patients received either short-term or long-term   
anticoagulation; 20% of both groups received transjugular intrahepatic   
portal system (TIPS) catheterization. Recurrence of symptoms requiring a   
second hospitalization occurred in 24% of those with BCS and 15% of patients   
with PVT (36% of the PVT+SPVT and 27% of the PVT+MVT). In those patients   
with a greater comorbidity profile, including hypertension, diabetes, and   
malignancy, PVT is more likely than BCS to occur. Regional presence of   
inflammation or cancer, specifically underlying liver disease,   
hepatocellular carcinoma, pancreatic cancer, pancreatitis, as well as   
regional surgical procedures appear to play major role in splanchnic vein   
thrombosis, while hereditary thrombophilias and the JAK2 V617F mutation make   
up an important but small component of splanchnic vein thrombosis.   
Contrast-enhanced CT was the most commonly successful radiologic technique   
for diagnosis, though magnetic resonance imaging (MRI) provides a more   
accurate alternative. anticoagulation was largely limited to patients with   
the most severe cases of SVT, and symptomatic recurrence was also more   
likely in these populations. Conclusions The present findings of risk   
factors associated with SVT are at variance with those in the current   
published literature, with higher incidence of regional cancer and lower   
incidence of JAK2 V617F mutation. There are, however, limitations to this   
study, including the fact that this is a retrospective analysis with data   
from a single institution. Verification of these findings has to been made   
in a prospective multi-institutional study involving a larger number of   
patients and a longer period of observation.  
  
RECORD 357  
Splanchnic vein thrombosis associated with myeloproliferative neoplasms. A   
study of the IWG-MRT in 475 subjects  
Pieri L. Guglielmelli P. Primignani M. Brambilla C. Randi M.L. Santarossa C.   
Cazzola M. Rumi E. Cervantes F. Ellis M. Chen F. Tripathi D. Rajoriya N.   
Barbui T. Delaini F. De Stefano V. Rossi E. Betti S. Specchia G. Ricco A.   
Gisslinger H. Gisslinger B. Vianelli N. Polverelli N. Ruggeri M. Girodon F.   
Tefferi A. Vannucchi A.M.  
Blood (2013) 122:21. Date of Publication: 21 Oct 2013  
  
Philadelphia-negative Myeloproliferative Neoplasms (MPN) include   
Polycythemia Vera (PV), Essential Thrombocythemia (ET) and Myelofibrosis   
Primary (PMF) and secondary to PV and ET (PPV-, PET-MF); included are also   
some less characterized entities defined as unclassified MPN (U-MPN). Risk   
of arterial and venous thrombosis is increased in MPN patients, and   
thrombosis is one of most important causes of mortality and morbidity. The   
risk of venous thrombosis in unusual sites, such as splanchnic vessels   
(SVT), is particularly associated with MPN; SVT can lead to complications   
such as portal hypertension, esophageal and gastric varices, ascites and   
hepatic failure. A recent meta-analysis reported that a MPN is the   
underlying cause of portal vein thrombosis in 31.5% and of Budd Chiari   
syndrome in 40.9% of cases (Smalberg, 2012). A significant association of   
SVT with JAK2V617F mutated MPN was reported (Dentali, 2009) but study of   
other correlations has been hampered by heterogeneity of available patient   
cohorts comprising relatively small number of cases. We conducted a   
retrospective multicenter study collecting clinical and biological data of   
patients (pts) with SVT associated with MPN diagnosed according to WHO2008   
criteria, aiming to describe patients' characteristics, trends and   
prognostic factors, and their potential implications for clinical practice.   
Data were collected from 15 international hematology centers in the   
framework of IWG-MRT. We collected 475 cases of pts with portal, splenic or   
mesenteric vein thrombosis (75.2%) or Budd Chiari syndrome (24.8%)   
associated with MPN. In 32% of cases, simultaneous involvement of portal   
(69.1% of total thrombosis), splenic (30.5%) and mesenteric (25.3%) veins   
occurred, and in 1.7% they were associated with Budd Chiari syndrome.   
Frequency of MPN subtype: 38.1% ET (n=181), 34.9% PV (n=166), 16.2% MF   
(n=77), 10.8% U-MPN (n=51). Median follow-up 87.9 mo (range 0.5-430); female   
61.3% (n=292; P<0.0001 vs male); median age at MPN diagnosis (dg) 44.4 y   
(range 12-90), at SVT dg 44.9 y (range 17-85). In 229 cases (48%) MPN and   
SVT dg were coincident, while in 104 (22%) SVT occurred before MPN dg   
(median 40 mo, range 5-335) and in 129 (27%) during MPN follow up (median 79   
mo, range 5-394). JAK2V617F mutational status is available for 361 pts: 99%   
PV, 84.7% ET, 88.1% PMF and 92.9% U-MPN pts were JAK2V617F positive, with a   
mean allele burden of 56±27.4%, 33.1±25.5%, 39.3±19.4% and 23.8±11.9%,   
respectively. Erythropoietin-independent colonies (EEC) were present at   
diagnosis in 80/110 evaluated cases (72.7%), 38/47 PV (84.4%), 32/45 ET   
(71.1%), 8/11 PMF (72.7%) and 2/7 U-MPN (28.6%). A concurrent thrombophilic   
state was found in 38.9% of cases. A 12.3% of pts experienced a recurrence   
of SVT after a median of 29 mo (range 1-378.3) and 35.8% developed   
thrombosis in other sites (17.7% arterial, 19.3% venous). Esophageal varices   
were found in 70.6% from which 31.9% bled. MF transformation occurred in   
32/166 PV (19%) and in 23/181 ET (13%) pts, with median time to progression   
of 122.3 mo (range 5.4-377.3) and 125.1 mo (range 39.3-255.3), respectively.   
Evolution to acute leukemia (AL) occurred in 12 pts (2.7%), of which 2 PMF,   
6 PV and 4 ET. In 3 PV and 1 ET pts a PPV and PET-MF transformation occurred   
before AL. After SVT, 77% of pts received anticoagulation, 23.5%   
antiaggregant therapy and 1.5% both; 68.8% received cytotoxic drugs, 11.4%   
of pts were treated with trans jugular porto-systemic shunt. No differences   
in survival were noted with these approaches. Beta blocker therapy was used   
in 48.5% of pts and correlated with improved survival (p=0.041) At last   
follow up 70/473 pts (14.8%) died; causes of death are evolution to AL   
(16.4%), other cancers (14.5%), disease progression without AL (12.7%), SVT   
(10.9%), hepatic failure and venous thrombosis other than SVT (9.1% each),   
heart failure and arterial thrombosis (7.3% each), hemorrhage (5.5%), renal   
failure and infection (3.6% each). After 10 y follow up 8/166 PV (5%),   
14/181 ET (8%), 14/77 PMF (18%) and 1/51 U-MPN (1.96%) pts died (p<0.01).   
Survival was significantly affected by occurrence of thrombosis other than   
SVT (p<0.0001) but not recurrence in splanchnic vessels (p=0.068). This   
large study confirms the strong association between JAK2V617F-mutated MPN   
and SVT and identifies the category of U-MPN as the prognostically more   
favorable; thrombosis at sites outside the splanchnic vasculature remains   
the leading cause of death.  
  
RECORD 358  
Paroxysmal nocturnal hemoglobinuria with budd-chiari syndrome treated with   
complement inhibitor eculizumab; a case report  
Valeri F. Borchiellini A. Schinco P. Boccadoro M.  
Blood (2013) 122:21. Date of Publication: 21 Oct 2013  
  
Introduction Paroxysmal nocturnal hemoglobinuria (PNH) is a rare, acquired   
haemolytic anaemia caused by somatic mutation in the phosphatidylinositol   
glycan-complementation class A gene, resulting in absence of a key   
complement regulatory protein, CD59. Thrombosis occurs in up to 40% of PNH   
patients; it usually involves abdominal and cerebral veins and it is the   
leading cause of death disease related. Methods We describe the response to   
Eculizumab (Soliris, Alexion) in 28 years old male with PNH diagnosed as a   
consequence of Budd Chiari Syndrome, acute liver dysfunction, mild   
haemolytic anaemia and thrombocytopenia. Results The patient was admitted to   
the gastroenterology department with acute abdominal pain, fatigue,   
hemolytic anaemia, thrombocytopenia and transaminitis. Abdominal doppler   
ultrasonography (US) was immediately performed with detection of Budd Chiari   
Syndrome, portal vein thrombosis, initial portal hypertension and ascites.   
He was started on low dose low molecular weight heparin (platelets <   
40x10-9/L), but despite anticoagulation progressive liver damage occurred,   
with poor pain control and worsening ascites. At the same time, we observed   
rapid exacerbation of thrombocytopenia and increasing in hemolysis tests   
with lactate dehydrogenase (LDH) reaching 1766 U/L, unresponsive to steroids   
administration. Bone marrow biopsy was negative but peripheral blood flow   
cytometry characterized a large PNH clone (85% total red blood cells).   
Furthermore, liver biopsy identified advanced stage of idiopathic cirrosis.   
Eculizumab therapy was then initiated at a dose of 600 mg weekly for 4 weeks   
and then 900 mg every 14 days. During the first month, transaminases   
progressively normalized and platelets settled permanently above 40x10-9/L,   
allowing therapeutic dose of anticoagulation. LDH dropped from basal value   
of >1000U/L to 600U/L and progressive reduction in abdominal pain was   
observed. Recanalization of portal vein thrombosis was found out at the US   
doppler after 6 weeks of anticoagulation, but recanalization of sovraepatic   
veins was not yet detectable. Conclusions Currently, after 17 Eculizumab   
administrations, platelets are 44 x 10-9/L, Hb 11.9 g/dl, AST 26 mg/dl, ALT   
55 mg/dl, GGT 123 mg/dl, LDH 518 U/L. No further thrombotic episodes   
occurred, no ascites was detected as well as portal hypertension signs,   
performing ultrasonography monitoring. This case shows that Eculizumab can   
block intravascular haemolysis and platelet consumption and can improve   
hepatic failure, allowing full dose of anticoagulant as therapy for current   
thrombosis or as prophylaxis for future events.  
  
RECORD 359  
A phase 2 study of ruxolitinib in patients with splanchnic vein thrombosis   
associated with myeloproliferative neoplasm. Preliminary results  
Pieri L. Paoli C. Guglielmelli P. Fjerza R. Arena U. Marra F. Colagrande S.   
Mori F. Marchioli R. Pioggiarella R. Ruggeri M. Nichele I. Finazzi G.   
Ferrari M.L. Rosti V. De Stefano V. Rumi E. Mannarelli C. Fanelli T. Bosi A.   
Rambaldi A. Barosi G. Cazzola M. Barbui T. Vannucchi A.M.  
Blood (2013) 122:21. Date of Publication: 21 Oct 2013  
  
Philadelphia-negative Myeloproliferative Neoplasms (MPN) include   
Polycythemia Vera (PV), Essential Thrombocythemia (ET) and Myelofibrosis,   
both Primary (PMF) and secondary to PV or ET (PPV-MF and PET-MF). A MPN is   
frequently the underlying cause of splanchnic vein thrombosis (SVT),   
accounting for 31.5% of portal vein thrombosis (PVT) and 40.9% of Budd   
Chiari syndrome (BCS). In patients (pts) with MPN and SVT, splenomegaly can   
arise as the consequence of the hematological disease and/or blood flow   
abnormalities consequent to the thrombosis itself. Splenomegaly and the   
compensatory enlarged splanchnic vessels are responsible for several   
complications including esophageal and gastric varices. Splenomegaly may   
cause abdominal discomfort; furthermore pts may present symptomatic burden   
due to the MPN. Current treatment strategies for MPN pts with SVT include   
anticoagulants and cytoreductive therapy (ie hydroxyurea, interferon) that   
have little influence in the control of splenomegaly and symptoms and do not   
improve flow abnormalities. Ruxolitinib, a JAK1/2 inhibitor, was highly   
effective in reducing spleen volume and improving symptoms in patients with   
MF and PV in phase II and III studies. We hypothesized that the decrease of   
the enlarged spleen determined by Ruxolitinib could result in a reduction of   
the local pressure in splanchnic vessels, producing both symptomatic   
improvement of splenomegaly-related symptoms and of splanchnic circulation.   
We designed an investigator-initiated multicentre phase 2 study of   
Ruxolitinib in pts with splenomegaly due to an underlying MPN associated   
with SVT. The drug was provided free of charge by Novartis, that had no role   
in trial design nor in data analysis. The primary study objective was to   
evaluate the proportion of subjects achieving ≥ 50% reduction in spleen   
length from left costal margin (LCM) measured by palpation at any time from   
baseline to week 24 (w24) and at w24, or a ≥ 35% reduction in spleen volume   
by MRI or CT at week 24. The secondary objectives included: evaluation of   
safety of Ruxolitinib in MPN-associated SVT; assessment of splanchnic   
circulation through Doppler analysis, measurement of hyperdynamic arterial   
circulation by echocardiography and stiffness of hepatic/splenic parenchyma   
by fibroscan; status of esophageal varices at w24 compared to baseline.   
Quality of Life assessment was performed using MPN-SAF questionnaire.   
Exploratory objectives include evaluations of changes in JAK2V617F or   
MPLW515 allelic burden, association of baseline mutations with response to   
treatment, changes in cytokine and microRNAs profiles, quantification of   
circulating endothelial cells. At the time of abstract submission 7 out of   
21 pts have been enrolled, of which 5 completed the 24 weeks of treatment;   
two additional pts are in screening phase. Three pts had PMF, two ET, one PV   
and one PPV-MF, associated to spleno-porto-mesenteric thrombosis (5 pts) and   
Budd Chiari syndrome (2 pts). All pts were under oral anticoagulation   
therapy. Initial dose of Ruxolitinib was 10 mg BID for PV, 25 mg BID for ET,   
15 mg BID for MF pts with baseline platelet count of 100 to 200x109/L and 20   
mg BID for those with baseline platelet count >200x109/L. A palpable   
splenomegaly greater than 5 cm below LCM was a criterion for enrollment; the   
5 patients who completed the 24 weeks of treatment had a median splenomegaly   
of 8 cm below LCM at baseline, and obtained a median reduction of 69%   
measured by palpation at week 24, associated with a significant reduction in   
abdominal discomfort as measured by MPN-SAF questionnaire (median score at   
screening 5 vs 1.5 at week 24). The total symptom score calculated by using   
BFI and MPN-SAF was reduced from 50 at screening to 35 at week 24.   
Instrumental evaluations of splanchnic and systemic circulation showed that   
3 pts obtained a reduction of the spleen stiffness from a median value of 66   
to 49.6 kilopascals (KPa), 2 pts had a reduction of the liver stiffness from   
a median value of 23.85 to 18.2 KPa and 1 pt a reduction of the cardiac   
output from 5.871 to 4.6 L/min. Evaluation of esophageal varices at week 24   
showed stabilization with neither worsening nor need of banding. Ruxolitinib   
was well tolerated, with no SAE reported; one pt developed anemia G2 and one   
G3 leading to dose reduction. Other adverse events include G1 asthenia and   
G≤2 AST/ALT increase in 3 pts, one case of Herpes Zoster and one case of   
abdominal pain both G1. Updated results will be presented at the meeting.  
  
RECORD 360  
Extramedullary hematopoiesis and splenic vein thrombosis, a unique   
presentation of pre-clinical essential thrombocythemia  
Yacoub A. Brockman A.  
Blood (2013) 122:21. Date of Publication: 21 Oct 2013  
  
Essential thrombocythemia (ET) is of the BCR-ABL-Negative myeloproliferative   
neoplasms (MPN). The incidence of ET is approximately 2.5 in every 100,000   
person per year. However, given the good prognosis, associated long life   
expectancy increasing detection in younger populations,ET is associated with   
a higher prevalence rate estimated to be 24 in every 100,000 person per   
year. ET is characterized by thrombocytosis, vasomotor symptoms a variable   
but increased risk of thrombosis and bleeding. Half of all ET patients will   
have a positive JAK2 and/or MPL mutation(s). Extramedullary hematopoiesis   
(EMH) is not a common finding in ET. Nonetheless, ET and other MPNs are   
associated with the mobilization of CD34+ cells into the peripheral blood.   
This process can ultimately lead to the seeding of extramedullary sites with   
primitive hematopoietic capacity, resulting in EMH within the spleen and   
liver, as well as a variety of other organs. Herein we describe a case that   
presented with life-threatening thrombosis and was found to have hepatic EMH   
several months prior to a clinical and pathologic diagnosis of ET. Case   
description A 22 year-old woman presented 10 days post Cesarean section with   
abdominal pain and hematemesis. Abdominal imaging showed hepatomegaly,   
splenomegaly, along with splenic and portal vein thrombosis. The patient   
underwent an emergency surgical splenectomy due to severe portal   
hypertension and endoscopic evidence of gastric variceal bleeding. A random   
liver biopsy was also performed intra-operatively. The splenectomy resulted   
in resolution of the GI bleeding and the varices normalized on follow up.   
Her platelet count was normal at the time of operation, but post-splenectomy   
her platelet count peaked at 1,217 K/ μL. Extensive testing did not unravel   
any identifiable inherited and/or acquired hypercoaguable factors.   
Subsequently anticoagulation therapy was recommended for 6 months. On   
pathology review, the spleen histology showed congestion, but otherwise no   
diagnostic abnormalities were noted. The liver biopsy showed evidence of EMH   
but did not identify any liver parenchymal disease. On subsequent follow up,   
the patient had persistent and marked thrombocytosis for over a year. A bone   
marrow biopsy was performed which showed a hypercellular bone marrow and   
megakaryocytic hyperplasia with a few large forms. There was no dysplasia or   
significant reticulin fibrosis. JAK2 mutation and BCR-ABL translocation were   
negative. Hydroxyurea and aspirin were started due to high risk of   
thrombosis. Discussion We report this unique case in which there was   
evidence of extramedullary hematopoiesis, along with pathologic and life   
threatening visceral thrombosis several months before the patient met   
criteria for diagnosis of ET. This supports the notion that neoplastic cells   
can mobilize and seed other organs early in the course of MPNs, including   
ET. Thrombotic risk in MPNs can also occur in the preclinical phase of MPNs   
as has been suggested in other reports. We also conclude that the   
demonstration of EMH in individuals with no preexisting hematologic neoplasm   
should warrant close follow up and assessment.  
  
RECORD 361  
Alcoholic pancreatitis-induced extrahepatic portal venous system th rombosis   
(EPVST): A pair of illustrative cases  
Patel V. Patel J. Afshari M. Cervellione K. Mehta A.  
American Journal of Gastroenterology (2013) 108 SUPPL. 1 (S257). Date of   
Publication: October 2013  
  
Introduction: Vascular complications of recurrent acute or chronic alcoholic   
pancreatitis are well-known, including extrahepatic portal venous system   
thrombosis (EPVST), which occurs at a rate of approximately 13% in this   
population. The splenic vein is the most commonly affected site in these   
vascular complications, with the portal and superior mesenteric veins being   
less common. Prognosis of patients with EPVST depends on early diagnosis and   
prompt treatment with anticoagulation. Here, we present two cases of   
patients presenting with EPVST, one in the portal vein and one in the   
splenic vein, illustrating the manifestations and the course of these   
important clinical entities. Case 1: A 48-year-old, alcoholic, HIV-positive   
male presented with epigastric pain, severe epigastric tenderness, and   
nausea. He had a history of admissions for acute alcoholic pancreatitis.   
Initial blood work showed evidence of hemoconcentration with elevated   
amylase and lipase levels suggestive of acute pancreatitis. Abdominal CT   
scan showed pancreatic inflammation with portal vein thrombosis (Figure 1).   
The patient was started on therapeutic anticoagulation and treated with   
aggressive fluid hydration with pain management. The patient improved   
symptomatically and repeat CT scan 8 weeks later showed resolution of portal   
vein thrombosis. Case 2: A 37-year-old alcoholic male with multiple past   
admissions for recurrent pancreatitis presented with epigastric pain, mild   
epigastric tenderness, nausea, and vomiting for 2 days. Initial labs showed   
minimal elevations in pancreatic enzyme levels. Abdominal CT scan revealed   
pancreatic inflammation and calcification with splenic and right common   
iliac vein thrombosis (Figure 2). Therapeutic anticoagulation was started   
with significant improvement in the patient's clinical condition.   
Conclusion: Despite being a well-known complication of recurrent acute or   
chronic alcoholic pancreatitis, the exact mechanism(s) causing venous   
thrombosis in this patient population is unclear. Venous stasis, spasm, and   
mass effects from inflamed pancreas are three of the possible causes. Prompt   
treatment with therapeutic anticoagulation is essential to prevent   
complications such as chronic portal vein thrombosis, portal venous   
hypertension, mesenteric ischemia, and infarction. The optimal duration of   
anticoagulation remains unknown and warrants further study. (Figure   
Presented).  
  
RECORD 362  
An uncommon cause of portal vein thrombosis  
De Jong I.M. Muller M.C.A. Peterson G.M. Polle S.W.  
Netherlands Journal of Medicine (2013) 71:8 (431). Date of Publication:   
October 2013  
  
RECORD 363  
Splanchnic vein thrombosis: Focus on antithrombotic treatment  
Riva N. Ageno W.  
Haematologica (2013) 98 SUPPL. 3 (262-264). Date of Publication: 1 Oct 2013  
  
Splanchnic vein thrombosis (SVT) is a manifestation of unusual site venous   
thromboembolism (VTE). Veins draining from different abdominal organs may be   
involved, leading to portal vein thrombosis (PVT), mesenteric veins   
thrombosis (MVT), splenic vein thrombosis (SPVT) and Budd-Chiari syndrome   
(BCS). Pathophysiology, clinical presentation and prognosis vary according   
to the site of thrombosis, although showing some common features.   
Epidemiology The epidemiology of SVT is poorly defined and varies greatly   
depending on data sources. PVT is the most common manifestation in the   
spectrum of SVT, with a reported annual incidence of less than 4 per million   
people in hospital registry data in the 1980s and a population prevalence of   
approximately 1% in a recent large autopsy study1. Viceversa, BCS is the   
least frequent disease, with an incidence ranging from 0.1 to 0.4-0.8 per   
million people per year (in Japan and Western countries, respectively) and,   
inversely, a prevalence ranging from 1.4 to 2.4 per million people (in   
Western countries and Japan, respectively)1. SVT has also a non-negligible   
rate of asymptomatic incidental findings, in imaging studies performed for   
other indications, such as follow-up of patients with cancer or liver   
cirrhosis. Risk factors SVT may be associated with different underlying   
disorders, either local or systemic. Abdominal cancer (mainly in the   
pancreatic, hepatobiliary or gastrointestinal system) and liver cirrhosis   
are the most common risk factors for PVT, being present in 31% and 34% of   
patients in a recently published study.2 The most common local risk factors   
for isolated MVT are cancer and abdominal inflammations or infections, each   
being present in about 20% of cases.2 Isolated sPVT was associated with   
underlying acute pancreatitis in nearly half of the patients, followed by   
cancer, cirrhosis and splenectomy.2 Myeloproliferative neoplasms (MPNs) are   
the leading systemic cause of SVT, diagnosed in 40% of BCS patients and   
approximately 30% of patients with non-cirrhotic non-malignant PVT.3   
Moreover, MPN subtypes showed different frequency according to the site of   
thrombosis: polycythemia vera and myelofibrosis were more prevalent in BCS   
than in PVT patients; while no difference has been reported in the   
prevalence of essential thrombocythemia and unclassifiable MPNs.3 Moreover,   
the JAK2 V617F mutation, the main molecular marker of the   
Philadelphia-negative MPN, emerged as an independent factor for SVT.4 Among   
inherited thrombophilias, higher prevalence of prothrombin G20120A mutation   
has been reported in patients with extra-hepatic PVT, while Factor V Leiden   
mutation was more frequent in BCS patients.1 Common systemic risk factors   
for BCS are also hormonal stimuli, such as the use of oral contraceptives   
and pregnancy or puerperium.1 Recently, an association between SVT and   
paroxysmal nocturnal hemoglobinuria has also been reported.1 Overall,   
permanent or transient risk factors are identified in at least 80% of   
patients, thus leaving a minority of events classified as unprovoked SVT.2   
Clinical presentation The clinical presentation of SVT is heterogeneous and   
varies accord- ing to the characteristics of the onset and the involved   
veins. Abdominal pain is the most frequent symptom, with a prevalence   
ranging from 40% in patients with PVT to more than 60% in patients with   
MVT.2 Acute MVT is indeed associated with intestinal infarction in almost   
onethird of patients.1Gastroesophageal varices and gastrointestinal   
bleeding, triggered by portal hypertension, are reported in one-quarter of   
patients, mainly with PVT or SPVT, and represent a challenge for treatment   
decisions. 2 Chronic PVT is also associated with the finding of portal   
cavernoma, portal cholangiopathy and hepatic encephalopathy1. In the   
majority of BCS patients, ascites, hepatomegaly, splenomegaly and right   
upper abdominal pain are reported.1 In a large cohort of 832 patients   
diagnosed with SVT over a 20-year period, 18% were asymptomatic.2   
Antithrombotic treatment The choice of the optimal treatment in patients   
with SVT is challenging. These patients have an increased risk of bleeding,   
due to the presence of oesophageal varices or thrombocytopenia, but in the   
meantime they also have a prothrombotic predisposition, resulting from the   
underlying cirrhosis or malignancy. In literature, there is a lack of   
randomized clinical trials to guide treatment decision, and contrasting   
evidence emerged from observational studies in the last decades. The   
majority of studies evaluated the antithrombotic treatment only in patients   
with non-malignant non-cirrhotic PVT. The retrospective study performed by   
Condat et al.5 included 136 patients, with a median follow-up duration of 46   
months. Anticoagulant treatment with heparin or vitamin K antagonists (VKAs)   
has been administered only in 84 patients, of whom 54 continued throughout   
the follow-up period and 30 discontinued the treatment before, but its   
duration was not reported. The anticoagulant treatment reduced the risk of   
recurrent thrombotic events in the portal venous system by two thirds   
(0.64/100 patient-years vs 1.87/100 patientyears, with and without   
anticoagulant therapy, respectively), without increasing the risk or   
severity of gastrointestinal bleeding. Indeed, the incidence of   
gastrointestinal bleeding in the overall cohort was high (12.5/100   
patient-years) and the only independent predictor of bleeding was the   
presence of moderate or large esophagogastric varices without adequate   
prophylactic measures. Plessier et al.6 evaluated the early initiation of   
heparin therapy, followed by oral anticoagulation, in 95 patients with acute   
PVT enrolled in a prospective European study. The anticoagulant treatment   
has been prescribed for at least 6 months, prolonged to long-term if   
mesenteric vein thrombosis or permanent prothrombotic disorder, for a median   
treatment duration of 234 days. At 1- year follow-up, recanalization was   
detected in one-third of PVT patients, and more than half of MVT and SPVT   
patients. Although major bleeding occurred in 5% of patients, no death   
resulted from haemorrhage. Opposite results emerged from the largest   
unselected cohort of SVT patients, diagnosed and followed up at a single   
institution, the Mayo Clinic, over a 20-year period.2 This retrospective   
study enrolled 832 patients with thrombosis of different splanchnic veins   
(including hepatic, splenic, portal or mesenteric) and different aetiologies   
(particularly malignancy and cirrhosis). Warfarin has been provided to 235   
patients (28%), of whom 175 lifelong, but no information is available on the   
use of alternative anticoagulant drugs such as heparins. After a mean follow   
up of 27 months, the incidence of recurrent venous thrombosis was 3.5/100   
patient-years, but the recurrence-free survival was not improved by the   
anticoagulant treatment (0.89 vs 0.77, p=0.38). The overall incidence of   
major bleeding was 6.9/100 patient-years and these complications were   
significantly higher in patients receiving warfarin compared with   
not-anticoagulated patients (26.2% vs 18.9%, p<0.05). More recently,   
Spaander et al.7 retrospectively collected information on 120 patients with   
non-malignant non-cirrhotic PVT. Only 66 patients (55%) were anticoagulated,   
with heparin or VKAs, for a median treatment duration of 1.9 years. The   
anticoagulant therapy showed a tendency to prevent recurrent venous   
thrombotic events (HR 0.2, p=0.1) but significantly increased the risk of   
gastrointestinal bleeding (HR 2.0, p<0.01). Indeed, 58 bleeding episodes   
happened in 66 patients on anticoagulant therapy vs 25 bleeding episodes in   
54 patients without anticoagulant therapy. At multivariate analysis,   
independent predictors of bleeding included also gastrointestinal bleeding   
at baseline (HR 2.1, p<0.01) and ascites at baseline (HR 2.0, p=0.01).   
Again, these findings are not generalizable to the whole population of SVT   
patients, given the highly selected population included in this study.   
Currently available guidelines recommend, in the absence of major   
contraindications, to start the anticoagulant therapy in all patients   
presenting with acute symptomatic SVT,8-9 with the aim to prevent the   
intestinal infarction and the long-term complications of chronic portal   
hypertension. After an initial period with either lowmolecular weight   
heparin (LMWH) or unfractionated heparin, most of the patients are   
candidates to VKAs. However, LMWH should be considered for extended   
treatment, if there is active malignancy, liver cirrhosis or   
thrombocytopenia.9 There is no consensus about the use of anticoagulant   
drugs in chronic SVT, which presents with variceal bleeding and   
hypersplenism but without signs of recent occlusion.8 Moreover, current   
guidelines suggest not to treat patients with asymptomatic incidentally   
detected SVT, even though the level of evidence is low.9 A recent   
prospective international registry evaluated the use of antithrombotic   
treatment for SVT patients in real life clinical practice.10 This unselected   
cohort included 613 patients, with a non-negligible proportion of liver   
cirrhosis or solid cancer (27.8% and 22.3%, respectively). The most commonly   
site of thrombosis was PVT (76.3%), with or without the involvement of other   
venous segment, but there was also a minority of patients with isolated MVT   
(10.9%), BCS (8.3%) or SPVT (3.1%). During the acute phase, 136 patients   
(22.2%) remained untreated. Factors associated with the decision not to   
administer anticoagulant therapy were: incidental diagnosis, single vein   
thrombosis, gastrointestinal bleeding at onset, solid cancer, liver   
cirrhosis and thrombocytopenia. Excluding a minority of patients that   
underwent interventional procedures, parenteral or oral anticoagulation has   
been administered to 470 patients (76.7%), of whom 295 continued with VKAs.  
  
RECORD 364  
Clinical features and natural history of portal vein thrombosis after   
radiofrequency ablation for hepatocellular carcinoma in Japan  
Shimada T. Maruyama H. Kondo T. Sekimoto T. Takahashi M. Motoyama T.   
Ogasawara S. Suzuki E. Ooka Y. Tawada A. Chiba T. Kanai F. Okabe S.   
Yoshikawa M. Yokosuka O.  
Hepatology International (2013) 7:4 (1030-1039). Date of Publication:   
October 2013  
  
Purpose: Little is known about portal vein thrombosis (PVT) after   
radiofrequency ablation (RFA) for hepatocellular carcinoma (HCC). We aimed   
to determine the incidence, background, and natural history of RFA-related   
PVT. Methods: This is a retrospective study of 317 patients (219 males and   
98 females) with HCC treated by RFA. Clinical data were compared between   
patients with and without PVT detected by ultrasound/CT. The median   
follow-up period after RFA was 15.8 months. Results: PVT was detected in 6   
(1.9 %) of 317 patients, 6 (0.8 %) of 802 treatments for HCC, and 6 (0.6 %)   
of 964 sessions of RFA. Body mass index was significantly higher in patients   
with PVT (26.9 ± 3.1 kg/m(2)) than in those without (22.9 ± 3.5 kg/m(2), p =   
0.0075). PVT was significantly more frequent in RFA for the left lobe of the   
liver (2.7 %) than for the other sites (0 %, p < 0.0001). Five of the six   
patients received no treatment for PVT, with natural outcomes of   
disappearance in one patient, improvement in one patient, and unchanged   
appearance in three patients. anticoagulation was applied in the one   
remaining patient and resulted in a successful recanalization. In the six   
patients, there was no significant difference in hepatic functional reserve   
between baseline and time of detection of PVT. Conclusions: These results   
indicated that a high body mass index and RFA for HCC in the left lobe might   
be significant risk factors for PVT and that RFA-related PVT was rarely   
progressive with little influence on liver function. © 2013 Asian Pacific   
Association for the Study of the Liver.  
  
RECORD 365  
Portal and mesenteric vein thrombosis in inflammatory bowel disease outside   
the surgical setting  
Arora Z. Navaneethan U. Shen B.  
American Journal of Gastroenterology (2013) 108 SUPPL. 1 (S526). Date of   
Publication: October 2013  
  
Purpose: Patients with Inflammatory Bowel Disease (IBD) are at an increased   
risk for portal vein thrombosis (PVT) & mesenteric vein thrombosis (MVT).   
Although PVT and MVT commonly occur during or after restorative colectomy,   
they can also occur during the course of medical management of IBD. The aim   
of the study was to evaluate the clinical characteristics & clinical   
outcomes of IBD patients with PVT/MVT without a history of recent abdominal   
surgery. Methods: A retrospective chart review was performed for all IBD   
patients seen at the Cleveland Clinic who were also diagnosed with PVT or   
MVT. Patients with abdominal surgery within 6 months prior to diagnosis,   
cirrhosis and malignancy were excluded. Comparison between groups was   
performed with t-test for continuous variables or with Fisher's Exact tests   
or Pearson's chi-square tests for categorical data. Results: Out of total 19   
patients, 10 were male and 9 female with a mean age of 45.3 ± 16 yrs and   
mean duration of IBD of 14.6 ± 15.3 yrs. 15 patients had Crohn's Disease   
while 4 had Ulcerative Colitis. Risk factors for thrombosis were present in   
only 10 patients including 2 patients on oral contraceptive pills & 1   
patient with abnormal hypercoagulability testing. None of the patients had a   
prior history of DVT/PE. Presenting symptoms were non-specific and included   
abdominal pain, fever, diarrhea or nausea/vomiting and required   
hospitalization in 13 patients. 12 patients (63.2%) had received steroids   
and 10 patients (52.6%) had received biological agent in the last six months   
prior to being diagnosed with PVT. Patients with involvement of the main   
portal vein were more likely to be treated with anticoagulation, however   
this trend did not reach statistical significance (p=0.06). Mean duration of   
follow up was 26.3 ± 23.9 months. There was no significant difference in the   
rate of resolution of thrombosis between the treated and non-treated   
patients (p=0.19). In the 3 months following diagnosis of PVT, IBD   
medications were escalated in 9 patients (47.4%) and 4 patients required   
re-hospitalization including 3 for IBD related surgery. There was no   
evidence of Esophageal/Gastric Varices in any of the 9 patients who   
underwent EGD after being diagnosed with PVT. Conclusion: PVT can be seen in   
IBD patients outside the post-operative setting and can occur without the   
presence of any other risk factors. Symptoms of PVT are non-specific but   
frequently severe enough to warrant hospitalization. Occurrence of PVT is   
frequently associated with escalation of IBD therapy which may indicate   
worsening disease course. Our findings also suggest that not all PVT in IBD   
patients need anticoagulation, especially if the clot is small, peripheral   
or discovered incidentally on imaging and if the patient is asymptomatic.   
(Table Presented).  
  
RECORD 366  
Pylephlebitis: An uncommon and dangerous cause of right upper quadrant pain  
Ori T. Sherner J.  
Chest (2013) 144:4 MEETING ABSTRACT. Date of Publication: October 2013  
  
INTRODUCTION: Pylephlebitis, septic thrombophlebitis of the portal vein, is   
an uncommon yet severe complication of bacteremia secondary to   
intra-abdominal and pelvic infections. The non-specific presentation and low   
incidence makes recognition challenging, yet relatively high morbidity and   
mortality rates make diagnosis and treatment critical. CASE PRESENTATION:   
Our patient is a 61 year old male with a history of diverticular bleed   
presenting with a 10 day history of fever, nausea and vomiting. 4 days   
earlier, he presented with similar symptoms and was diagnosed with suspected   
viral gastroenteritis. The patient was unresponsive to symptomatic therapy   
however, and he returned upon developing rigors and anorexia. At   
presentation, the patient was febrile while all other vital signs were   
normal. Laboratory evaluation revealed leukocytosis, elevated AST, ALT, and   
alkaline phosphatase, and hypokalemia and hyponatremia. Blood cultures drawn   
during the previous presentation confirmed Bacteroides fragilis bacteremia.   
Abdominal computed tomography demonstrated heterogeneous hepatic parenchyma   
and a left portal vein thrombus, confirmed on PET imaging. Antibiotic   
therapy with pipercillin-tazobactam, and anticoagulation therapy with   
intravenous heparin, was initiated. The patient remained intermittently   
febrile with rigors for 72 hours before clinical improvement was evident.   
After a two week hospitalization, during which the fever and abdominal   
discomfort resolved, the patient was transitioned to oral therapies. He   
completed 6 weeks of antibiotic therapy and 3 months of anticoagulation   
without evidence of recurrence. DISCUSSION: While pylephlebitis remains a   
relatively uncommon diagnosis, early recognition in the critical care   
setting is imperative to successful outcomes as clinical response to therapy   
may take several days. Diverticulitis and appendicitis remain the most   
commonly associated infections, though no etiology was identified here.   
Fever and abdominal discomfort are hallmarks of the non-specific   
presentation, and leukocytosis, transaminitis and elevated alkaline   
phosphatase are routinely seen as in this case. Treatment consists of   
prolonged parenteral antibiotics covering both gram negative enteric   
organisms and anaerobes, and while there is no clear consensus regarding the   
role of anticoagulation, this patient was treated successfully. CONCLUSIONS:   
We present a case of B. fragilis pylephlebitis, initially suspected to be   
common gastroenteritis, treated successfully with targeted antibiotic   
therapy and anticoagulation.  
  
RECORD 367  
Management of portal hypertension in children with portal vein thrombosis  
Giouleme O. Theocharidou E.  
Journal of Pediatric Gastroenterology and Nutrition (2013) 57:4 (419-425).   
Date of Publication: October 2013  
  
portal vein thrombosis (PVT) is a common cause of portal hypertension in   
children. Predisposing conditions for PVT are obscure in more than half of   
the cases. Variceal bleeding and splenomegaly are the most frequent initial   
manifestations. Radiologic imaging studies are the mainstay for diagnosis.   
Treatment includes pharmacologic, endoscopic, and surgical modalities.   
β-Adrenergic blockers are not routinely used in children because of unproven   
efficacy and significant adverse effects. Endoscopic methods, such as   
sclerotherapy and endoscopic variceal ligation (EVL), are highly effective   
in the treatment of acute variceal bleeding and eradication of varices. EVL   
is the treatment of choice because of minimal complications and the need for   
few endoscopic sessions. EVL facilitates portal decompression either by the   
formation of collateral vessels or by surgical portosystemic shunting, when   
vessels grow to the proper diameter for anastomosis. Surgical portosystemic   
shunts are reserved for refractory cases because of significant   
complications and technical difficulties. Transjugular portosystemic shunts   
have an emerging role in the management of portal hypertension caused by   
PVT. PVT may occur in the posttransplant setting, but optimal management is   
not defined yet. Copyright © 2013 by European Society for Pediatric   
Gastroenterology, Hepatology, and Nutrition and North American Society for   
Pediatric Gastroenterology, Hepatology, and Nutrition.  
  
RECORD 368  
portal vein thrombosis in a 60 year old white female found to have MTHFR   
mutation heterozygosity  
Girithari G. Batista R. Simoes J. Gil E. Goncalves C.  
European Journal of Internal Medicine (2013) 24 SUPPL. 1 (e82). Date of   
Publication: October 2013  
  
Background: portal vein thrombosis (PVT) is being recognized with increasing   
frequencywith the use of ultrasonography. The major causes are hepatic   
parenchymal disease, hypercoagulable syndromes and abdominal sepsis. It can   
be asymptomatic and discovered by accident during imaging tests, or in rare   
cases manifested as abdominal pain. Methods and results: The authors present   
a case of a 60 year old Portuguese female referred to the internal medicine   
external consultation because of persistent thrombocytopenia. Three months   
prior, she was admitted at our hospital due to extensive necrosis of small   
intestine complicated with septic shock with need of ventilator and   
hemodynamic support. She has denied history of spontaneous abortions and   
other health or family history was noncontributory. An exhaustive   
differential diagnosis was performed. A CT scan of the abdomen was done,   
revealing a cavernoma on the right branch of the portal vein resulting from   
partial thrombosis. Coagulation study was performed, revealing that the   
patient was heterozygous for MTHFR gene (C677-T). anticoagulation with   
warfarin was initiated with total reversion of laboratory abnormalities.   
Conclusion: Methylenetetrahydrofolate reductase (MTHR) is an important   
enzyme in human physiology. Genetic variation in this gene may result in   
deficiencies in production or function of this enzyme has been associated   
with increased risk of myocardial infarction, stroke, venous thrombosis,   
several types of cancer, congenital defects, inflammatory bowel disease and   
several neuropsychiatric conditions. Corrective treatment must be   
individualized based on genotype expression. After presenting the case, the   
author makes a brief theoretical review.  
  
RECORD 369  
APVT after EUS-FNA: A rare presentation in advanced pancreatic cancer at   
increased risk  
Ngiu C.S. Chow P.K.H. Too C.W. Tan D.  
Journal of Gastroenterology and Hepatology (2013) 28 SUPPL. 3 (428). Date of   
Publication: October 2013  
  
Objective: Endoscopic ultrasound guided fine needle aspiration (EUSFNA) has   
become an important procedure to acquire tissue diagnosis for pancreatic   
tumour with low procedural complication. Uncommon reported complications of   
EUS-FNA for pancreatic tumour were infection, bleeding, perforation, and   
acute pancreatitis. Acute portal vein thrombosis (APVT) as rare complication   
of EUS-FNA was reported once only in a case of advance metastatic pancreatic   
cancer. Local tumour infiltration of portal vein with post EUS-FNA   
bacteremia was presumably the causative factors and intravenous antibiotic   
prior to EUS-FNA was suggested as preventive measures. Methods: We present a   
middle age lady with advance metastatic pancreatic cancer referred for   
EUS-FNA. Preprocedural imaging studies showed a pancreatic head mass,   
measuring 3.8 x 3.3 cm with thick enhancing wall and central hypodensity.   
The portovenous and splenomesenteric vessels were patent. Several hepatic   
masses were noted, in keeping with metastases. Antibiotic was given to the   
patient in view of cystic nature of pancreatic tumour prior to EUS-FNA. The   
EUS-FNA was performed with linear endoscopic ultrasound (Olympus,   
GF-UCT140-AL5, Japan). EUS-FNA was performed on the lymph node initially,   
and followed by pancreatic tumour with 22 G FNA needle (Cook Medical Inc,   
Limerick Ireland). The pancareatic tumour was difficult to assess despite   
changing to pancreatic tumour with 25 G FNA needle (Cook Medical Inc,   
Limerick Ireland). The technical difficulty in assessing the lesion led to   
prolonged procedural time. Results: She presented three days later with   
abdominal pain, which later diagnosed as acute portovenous thrombosis based   
on repeated computer tomogram. anticoagulation was initiated and   
subsequently patient was arranged for palliative chemotherapy. Conclusion:   
In conclusion, prothrombotic state in advance pancreatic cancer, venous   
stasis from endoscope manipulation and micro-endothelial injury from   
mechanical manipulation during EUS-FNA can lead to acute portal vein   
thrombosis. Our experience showed acute portal vein thrombosis can occur in   
naïve portovenous vessels in advanced pancreatic cancer.  
  
RECORD 370  
anticoagulation for acute portal vein thrombosis in liver cirrhosis is safe   
and does not increase the mortality  
Sliwa K. Malek N. Plentz R.R.  
United European Gastroenterology Journal (2013) 1:1 SUPPL. 1 (A154). Date of   
Publication: October 2013  
  
INTRODUCTION: portal vein thrombosis (PVT) is caused by liver cirrhosis,   
inflammatory diseases, cancer, myeloproliferative and coagulation disorders.   
Acute PVT can be distinguished from chronic PVT. Clinical presentation   
depends on the onset and the extent of the thrombosis and the development of   
collateral circulation / portal hypertension. For acute PVT early initiation   
of anticoagulation (AC) or thrombolytic therapy is recommended. The   
therapeutic approach in chronic PVT, especially in patients with liver   
cirrhosis, is controversial. AIMS&METHODS: Our analysis was designed to   
validate retrospectively the managment of PVT in patients with different   
underlying illness. Therefore we reviewed all patients with diagnosed PVT   
(n=149) in a period of 2005 to 2012 at our Department of Medicine. Patient   
characteristics, including demographics, acute or chronic PVT, underlying   
disease, therapeutic managment and complications were analyzed. RESULTS: PVT   
occurred in 102 men and 47 women. PVT was common in patients with   
gastrointestinal cancer, liver cirrhosis, inflammatory diseases, abdominal   
surgery, myeloproliferative and coagulation disorders. 76 patients had acute   
and 73 chronic PVT. 36 patients with acute PVT were treated by AC. AC   
(heparin, marcumar, thrombolytic therapy) could achieve in 20 patients   
recanalisation. In 7 patients AC caused impairment and 9 patients died. 20   
patients with chronic PVT were treated by AC. AC could achieve in 3 patients   
recanalisation In 1 patient AC caused impairment, 16 patients showed no   
change and 5 patients died. CONCLUSION: Patients with acute PVT benefit   
significantly from AC. AC is safe, especially in patients with liver   
cirrhosis (CHILD A & B) and had no significant impact on side effects and   
mortality. Patients with cancer (HCC, pancreatic cancer) and acute PVT have   
no advantage of AC.  
  
RECORD 371  
Paroxysmal nocturnal hemoglobinuria with Budd-Chiari syndrome treated with   
complement inhibitor eculizumab; a case report  
Valeri F. Borchiellini A. Beggiato E. Schinco P.  
Haematologica (2013) 98 SUPPL. 3 (106). Date of Publication: 1 Oct 2013  
  
Paroxysmal Nocturnal Haemoglobinuria (PNH) is a rare, acquired haemolytic   
anaemia caused by somatic mutation in phosphatidylinositol   
glycan-complementation class A gene, resulting in absence of two key   
complement regulatory proteins CD59 and CD55. Thrombosis occurs in up to 40%   
of PNH patients; it commonly involves abdominal and cerebral veins and is   
the leading cause of disease related death. We describe response to   
Eculizumab (Soliris, Alexion) in a 28 year old male with PNH, Budd-Chiari   
Syndrome, acute liver dysfunction, haemolytic anaemia and thrombocytopenia.   
The patient was admitted to the gastroenterology department with acute   
abdominal pain, haemolitic anaemia, thrombocytopenia and transaminitis.   
Abdominal doppler ultrasound (US) was immediately performed, detecting of   
venous sovrahepatic thrombosis (Budd-Chiari Syndrome), portal vein   
thrombosis, portal hypertension and ascites. He was started on low dose low   
molecular weight heparin (platelets <40x109/L), but despite anticoagulation   
progressive liver failure occurred, with poor pain control and worsening   
ascites. We observed worsening thrombocytopenia and haemolysis, with lactate   
dehydrogenase (LDH) reaching 1766 IU/L, unresponsive to steroids   
administration. Bone marrow biopsy showed increased red cell turnover, and   
peripheral blood flow cytometry characterized a large PNH clone (85% total   
red blood cells). Liver biopsy revealed advanced stage idiopathic cirrhosis.   
Eculizumab therapy was then started at the dose of 600 mg weekly for 4 weeks   
and then 900 mg every 15 days. During the first month clinical conditions   
improved and progressive reduction in abdominal pain was observed;   
transaminases progressively normalized, LDH dropped to 518 IU/L and   
platelets reached 40x109/L, allowing therapeutic anticoagulation with   
warfarin. Recanalization of the portal vein thrombosis was found at the   
Doppler US after 6 weeks' anticoagulation, but recanalization of   
sovrahepatic veins was not achieved. Currently, after 12 Eculizumab   
administrations, the patient is well and pain free, platelets are stable   
>40x109/L, Hb 11.9 mg/dL, AST 36 IU/dL, ALT 60 IU/dL, GGT 169 IU/dL, LDH 649   
IU/L. No further thrombotic episode has occurred. This case shows that   
Eculizumab can block intravascular haemolysis and platelet consumption and   
can improve hepatic failure, allowing full dose of anticoagulants as therapy   
for current thrombosis or as prophylaxis for future events.  
  
RECORD 372  
Successful liver transplantation in a patient with splanchnic vein   
thrombosis and pulmonary embolism due to polycythemia vera with Jak2v617f   
mutation and heparin-induced thrombocytopenia  
Biagioni E. Pedrazzi P. Marietta M. Benedetto F.D. Villa E. Luppi M.   
Girardis M.  
Journal of Thrombosis and Thrombolysis (2013) 36:3 (352-354). Date of   
Publication: October 2013  
  
Heparin-induced thrombocytopenia (HIT) is a rare complication of heparin   
treatment resulting in a severe acquired thrombophilic condition with an   
associated mortality of about 10 %. We report the first case of successful   
urgent liver transplantation (LT) in a patient with end-stage liver disease   
due to a Budd-Chiari syndrome, portal vein thrombosis and pulmonary embolism   
due to acquired thrombophilia associated to polycythemia vera carrying   
JAK2V617F gene mutation and HIT in the acute phase. Lepirudin was used to   
provide anticoagulation in the LT perioperative period that was performed   
without haemorrhagic and thrombotic complications despite the donor received   
heparin during liver explantation. © 2012 Springer Science+Business Media   
New York.  
  
RECORD 373  
The vanishing liver mets  
O'Connell B. Wilford R.  
American Journal of Gastroenterology (2013) 108 SUPPL. 1 (S335). Date of   
Publication: October 2013  
  
Introduction: Clinicians have become increasingly reliant on imaging studies   
to make diagnoses. Despite improved sensitivity of these imaging modalities,   
some diagnoses cannot be made without biopsy. We present a case of   
pylephlebitis, in which the radiographic evidence pointed overwhelmingly to   
metastatic disease. To our knowledge, this is the first reported case of   
pylephlebitis mimicking colon cancer with liver metastases. Case: A   
65-year-old female with a history of hypertension and hypothyroidism   
presented with six weeks of fatigue, weight loss, fever, and jaundice. She   
had a laparoscopic cholecystectomy three years ago for cholelithiasis.   
Initial workup revealed leukocytosis with bandemia (WBC 20,200, 17% bands),   
elevated bilirubin (7.4 mg/dl), and liver enzymes with an obstructive   
pattern (alkaline phosphatase 503 U/L, AST 82 U/L, ALT 96 U/L). Blood and   
urine cultures were obtained, and ertapenem was begun empirically. Abdominal   
CT with oral and intravenous contrast showed portal vein thrombosis,   
multiple liver lesions consistent with metastases, and an ascending colon   
stricture, leading to extensive oncologic and gastroenterological   
evaluation. No intrahepatic or extrahepatic biliary dilation or stricture   
was seen on MRCP, but it showed numerous hepatic masses, concerning for   
metastases. Liver biopsy showed biliary obstruction, but no neoplasm. She   
remained febrile, despite negative blood and urine cultures. After expanding   
antibiotic coverage to piperacillin/tazobactam and azithromycin, she   
improved clinically, and her bilirubin and transaminases decreased.   
Colonoscopy showed diverticulosis without masses or strictures. She was   
diagnosed with pylephlebitis and discharged home on amoxicillin/clavulanate   
and anticoagulation. One month later, she was asymptomatic, her jaundice had   
resolved, and repeat abdominal CT showed a patent portal vein and complete   
resolution of the hepatic lesions. Conclusion: Pylephlebitis is a rare   
disorder that carries a high morbidity and mortality, despite early   
detection with CT imaging and broad spectrum antibiotics. Greater than 80%   
of documented cases contain an identifiable infectious or inflammatory   
etiology. Our patient lacked demonstrable intra-abdominal, urinary, and   
blood-borne infection, but rather had radiographic findings that strongly   
suggested colon cancer with liver metastases. Multiple lesions in the liver   
are often malignant, but clinicians should keep their differentials open to   
other etiologies. Pylephlebitis should be considered in a patient with   
fever, abnormal liver function tests, and portal vein thrombosis.  
  
RECORD 374  
Incidence of thrombotic events in chronic liver disease  
Mocanu I. Amaral M.S. Alves J.D.  
European Journal of Internal Medicine (2013) 24 SUPPL. 1 (e89). Date of   
Publication: October 2013  
  
Introduction: Chronic liver disease is labeled as a classic acquired   
bleeding disorder, however it has been demonstrated that these patients   
present thrombin production within normal range, which raises questions   
about the International Normalized Ration included in several prognosis   
stratification scores. Although this population does not seem to be   
protected from thrombotic events, the prophylaxis of deep vein thrombosis is   
mostly withheld from patients with chronic liver disease. Objectives: Our   
aim is to analyze the incidence of thrombotic events and the percentage of   
prophylaxis use in in-patients with diagnosis chronic liver disease.   
Methods: Retrospective study from clinical data-base of patients with   
diagnosis of chronic liver disease and thromboembolism at discharge during   
one year. We also evaluated the use of thromboprophylaxis, mortality,   
admission time, and Child-Pugh score, among other variables. Results: 34   
hospitalizations, 67% male with a mean age of 59 years and mean value of   
stay of 16 days. 23% of these were given prophylactic anticoagulants. We   
identified two cases of portal vein thrombosis one of which received oral   
anticoagulant at discharge. There was no evidence of hemorrhagic   
complications in patients medicated with anticoagulants. Conclusion: The   
need for considering anticoagulation in patients with chronic liver disease   
is an emerging issue due to the increase in life expectancy of these   
patients with underlying co-morbidities that boost the risk of thrombotic   
events. Although our study has limitations, as the short period of time is   
included, we considered that there is a need of a larger study to identify   
the true incidence of thrombotic events in this population.  
  
RECORD 375  
A case report of a patient with recurrent pouchitis and large pulmonary   
emboli  
Yoo L. Elwir S. Tinsley A. Williams E.  
American Journal of Gastroenterology (2013) 108 SUPPL. 1 (S421). Date of   
Publication: October 2013  
  
Purpose: We report a case of a 33-year-old woman with a history of   
ulcerative colitis diagnosed 10 years prior to presentation status post   
total abdominal colectomy with ileal pouch anal anastomosis (IPAA) 6 years   
prior to presentation. The patient suffered recurrent severe antibiotic   
resistant pouchitis shortly after her surgery. Two years ago, a pouchoscopy   
demonstrated continued pouchitis with more extensive inflammation above the   
pouch. The patient was started on Azathioprine and responded to a course of   
steroids and antibiotics. After her endoscopic evaluation, the developed an   
idiopathic left upper extremity deep vein thrombosis at her IV site and   
completed 6 months of warfarin therapy. One month prior to presentation, the   
patient had another episode of severe pouchitis and was treated with   
antibiotics and a steroid taper. During her taper, the patient presented   
with dyspnea on exertion. Chest CT scan revealed large bilateral pulmonary   
emboli (PE) with severe right heart strain, enlargement of the right atrium   
and main pulmonary artery consistent with pulmonary hypertension. She was   
treated with enoxaparin and warfarin and discharged home. Our patient   
appeared to have developed thromboemboli in the setting of two episodes of   
pouchitis. Hospitalized inflammatory bowel disease (IBD) patients are known   
to be at increased risk for thromboembolism. The most frequent complication   
in ulcerative colitis patients after ileal pouch anal anastomosis (IPAA) is   
pouchitis, a nonspecific inflammation of the ileal pouch reservoir, having a   
cumulative prevalence of 50%. It is unknown if this inflammatory state   
increases hypercoagulability. While portal vein thrombi (PVT) have recently   
been linked to IPAA and patients found with PVT had a higher incidence of   
postoperative pouchitis, an association with PE has not been described.   
Hospitalized patients with IBD and pouchitis, such as our patient, must be   
considered at high risk for thromboembolism and receive appropriate   
prophylaxis or be considered for long-term anticoagulation regardless of   
history of thromboembolism.  
  
RECORD 376  
Budd Chiari Syndrome (BCS): The Austin experience  
French J. Mo A. Testro A. Gow P. Grigg A.  
Journal of Gastroenterology and Hepatology (2013) 28 SUPPL. 2 (62). Date of   
Publication: October 2013  
  
Aim: Budd Chiari Syndrome 'BCS' is a rare disorder, with an annual incidence   
of 0.2-0.8 per million.1 The few available studies report liver   
transplantation rates of 12.7%2 to 42%3 and poor 5-year transplantationfree   
survival of 28%3 for primary BCS. We aimed to investigate the epidemiology,   
natural history and outcomes of BCS patients at Austin Health. Method: This   
study was retrospective and was performed at the Austin Hospital. We   
searched the hospitals computerised diagnosis database and the hospital's   
liver transplant database for cases of Budd Chiari syndrome from January   
2000 until August 2012. Patients with hepatic venous outflow obstruction at   
any point from the small hepatic veins to the inferior vena cava were   
included. Patients with secondary Budd Chiari syndrome were excluded.   
Results: Median age at diagnosis was 42 years (range 21-76). 59% were   
female. Eight patients (30%) had concomitant portal vein thrombosis (PVT).   
Twenty four patients (89%) had at least one identifiable risk factor. The   
most common risk factor was myeloproliferative neoplasm (MPN, n = 16) with   
polycythaemia rubra vera (PRV) being the most common subtype. JAK-2 was   
positive in 12 of 18 patients tested. The primary intervention was   
transjugular intrahepatic portosystemic shunting (TIPS) in thirteen patients   
(48%) and angioplasty/stenting in eleven (41%). One patient had a   
splenorenal shunt. No patients required transplantation during the 10 year   
follow up period. At median follow-up of 5 years 16 patients had compensated   
liver disease, 3 had decompensated liver disease, 2 patients died a liver   
related death (one from hepatorenal syndrome and bilateral pulmonary emboli,   
one death secondary to hepatic encephalopathy) , 4 died from a non liver   
related death and 2 patients were lost to follow-up. The overall transplant   
free one year survival was 96% and 81% at five years. Discussion: In this   
retrospective study, we aimed to characterise the aetiology and treatment   
outcomes of patients with Budd Chiari syndrome treated in our institution.   
This is the only published cohort of Budd Chiari patients where no liver   
transplantations were required. We postulate that this is due to intensive   
TIPS surveillance at our hospital to prevent TIPS failure. MPN is the most   
common aetiological factor in BCS. This can be missed at diagnosis, and all   
patients should have JAK2 testing or bone marrow biopsy. TIPS or   
angioplasty/stenting, together with anticoagulation and treatment of any   
MPN, results in favourable long term transplantation-free outcomes and   
represents optimal standard of care.  
  
RECORD 377  
Complete portal vein thrombosis in a patient with active crohn's disease  
Cornish C. Amundson W. Mason D. Dogra V. Kaul V. Shah A.  
American Journal of Gastroenterology (2013) 108 SUPPL. 1 (S422). Date of   
Publication: October 2013  
  
Introduction: portal vein thrombosis is an uncommon occurrence in patients   
with inflammatory bowel disease, but when it does occur it is usually soon   
after intra-abdominal surgery. We describe a case of complete portal vein   
thrombosis in a patient with active Crohn's disease. Case Summary: A   
41-year-old man with a 10-year history of Crohn's colitis, treated with   
sulfasalazine 1,500 mg BID and mercaptopurine 75 mg daily, presented to the   
office with a 3-week history of generalized, constant abdominal pain, with   
associated anorexia and 10-12 loose, bloody bowel movements per day, which   
began localizing to the right upper quadrant (RUQ) 4 days prior to being   
seen. His physical exam was notable for RUQ tenderness to even light   
palpation with guarding and mild hepatomegaly. His labs were significant for   
AST 209 U/L, ALT 693 U/L, and CRP 62 mg/L. His initial abdominal ultrasound,   
obtained 1 week after his clinic visit, demonstrated a non-occlusive left   
portal vein thrombus extending into the bifurcation of the main portal vein.   
The right and main portal vein were both patent. His abdominal pain   
gradually resolved and his liver profile returned to normal. Five weeks   
later, he presented to the emergency department with intense RUQ abdominal   
pain. His AST, ALT, and total bilirubin were found to be 1,406 U/L, 2,527   
U/L, and 1.7 mg/dL (direct 0.9 mg/dL), respectively. An abdominal ultrasound   
was obtained, which demonstrated partial thrombosis of the right portal vein   
and complete thrombosis of the main portal vein. He was subsequently started   
on a heparin drip after labs to check for a hypercoagulable state, which   
were unremarkable, had been drawn. Two days later, the patient was started   
on warfarin, as well as an enoxaparin bridge, and discharged to home. The   
patient's liver profile quickly improved. A repeat abdominal ultrasound   
after 6 months of anticoagulation therapy has not yet been performed.   
Discussion: portal vein thrombosis is a potentially life-threatening   
hepatobiliary manifestation associated with inflammatory bowel disease (IBD)   
and occurs more often in patients with Crohn's disease than ulcerative   
colitis. The formation of a portal vein thrombus in patients with IBD is   
rare, especially in those who have not recently undergone intra-abdominal   
surgery. Given that the patient presented with portal vein thrombosis in the   
setting of a Crohn's disease flare, we hypothesize that ulceration of the   
bowel mucosa allowed for translocation of bacteria into the portal venous   
system leading to portal pyelophlebitis followed by portal vein thrombosis.  
  
RECORD 378  
Risk factors of arterial or venous thromboembolism in cirrhotic patients  
Laabidi A. Baccouche H. Fekih M. Ben Mustapha N. Serghini M. Boubaker J. Ben   
Romdhane N. Filali A.  
United European Gastroenterology Journal (2013) 1:1 SUPPL. 1 (A298). Date of   
Publication: October 2013  
  
INTRODUCTION: cirrhosis results in a complex pattern of defects in   
haemostatic functions with reduced synthesis of pro and anticoagulant   
factors. As possible complication of coagulation disorders in cirrhosis,   
could be the development of arterial or venous thromboembolism (AVTE). The   
purpose of our study was to determine thrombotic risk factors in cirrhotic   
patients. AIMS&METHODS: Cirrhotic patients were enrolled. The presence of   
personal and familial history of AVTE was investigated. Patients were   
divided into 2 groups. Group1 included patients who developed arterial or   
venous thromboembolism after cirrhosis diagnosis and group 2 cirrhotic   
patients without thrombotic event. White blood cells, platelet count,   
prothrombine time, INR, albumin, urea, pro coagulant factors (VIII, XII,   
VII, II, V) were determined. Level of antithrombin, protein C and protein S   
were measured. Search for factor V Leiden and prothrombin gene mutation   
(G20210A) were performed with PCRRFLP. Anticardiolipin and   
antiB2glycoprotein antibodies were also investigated. Both groups of   
patients were compared with regard of clinical and biological findings.   
RESULTS: Fifty one cirrhotic patients were included. Their mean age was 56.8   
years. They were men and women. Among the 51 cirrhotic patients, 7 (13.7%)   
had experienced AVTE after cirrhosis diagnosis: deep venous thrombosis   
(n=2), pulmonary embolism (n=1), Budd Chiari syndrome (n=1), portal   
thrombosis (n=3). They were compared to 46 cirrhotic patients without   
thrombosis. No patient with AVTE had neither personal nor familial history   
of thrombosis. In an univariate analysis, white blood cell count and   
platelet count were significantly higher in patients with AVTE than other   
cirrhotic patients (respectively 8795 vs 5032/mm(3), p < 0.018 and 91133 vs   
154375/mm(3), p=0.03) However, In a multivariate analysis only the platelet   
count was independently predictive of VTE in cirrhotic patients (P=0.05).   
Moreover, prothrombin time, INR, albumin, urea, level of pro and   
anticoagulant factors were not statistically different in both groups. There   
was no link between the presence of Factor VLeiden, prothrombin gene   
mutation (G20210A), anticardiolipin and antiB2glycoprotein antibodies to   
thrombosis. CONCLUSION: Approximately 13.7% of cirrhotic patients resulted   
in a thromboembolic event. Platelet count was predictive of increased risk   
of AVTE as it was supported by other studies. Understanding the factors   
predisposing to thrombosis in cirrhotic patients could play a role in   
identifying a subgroup of patients at high risk of thrombosis and making   
decisions regarding the utility of anticoagulation therapy.  
  
RECORD 379  
Abdominal and Pelvic Venous Thrombosis  
Veerreddy P.  
Hospital Medicine Clinics (2013) 2:4 (481-498). Date of Publication: October   
2013  
  
A lot is known and published about venous thrombosis in the lower   
extremities and upper extremities. But there is lack of awareness about   
abdominal and pelvic venous thrombosis. Hence, the focus of this article is   
to bring to light these underdiagnosed causes of venous thrombosis. This   
article discusses the definitions, epidemiology, systemic risk factors,   
local risk factors, history and examination, diagnosis, prognosis, and   
management of abdominal and pelvic venous thrombosis. © 2013 Elsevier Inc.  
  
RECORD 380  
Long term insulin independence after islet transplant alone (ITA) and   
pancreas transplant alone (PTA) in patients with type 1 diabetes (T1D) - A   
single institution experience  
Masharani U. Moassesfar S. Frassetto L. Szot G. Tavakol M. McElroy J. Ramos   
M. Johnson K. Stock P.G. Posselt A.  
Transplantation (2013) 96 SUPPL. 6S (S44). Date of Publication: 27 Sep 2013  
  
Background: We describe ITA in patients with T1D using two novel   
immunosuppressive regimens based on the anti-LFA 1 antibody, efalizumab, or   
the costimulation blocking antibody, belatacept, that permit long-term islet   
allograft survival without need for corticosteroids or calcineurin   
inhibitors (CNI). We also asked whether ITA using these protocols could   
achieve outcomes comparable to PTA performed at our institution. Methods:   
Ten T1D patients received ITA between 2007-2010. Insulin independence, renal   
function & adverse reactions were compared to 17 TID who received   
consecutive PTA between 2002-2011. All patients received thymoglobulin   
induction. Maintenance immunosuppression for ITA consisted of Efalizumab   
(n=5) or Belatacept (n=5), sirolimus, and mycophenolate mofetil (MMF). PTA   
patients received low-dose tacrolimus, MMF, sirolimus and prednisone. High   
insulin requirements and BMI > 30 were exclusion criteria for the ITA; and   
high cardiovascular risk is an exclusion criterion for PTA. Results: Six   
patients received one and four received two islet transplants. All 10 became   
insulin independent after the final transplant for amean of 46months   
(25-64). Seven (70%) remain insulin independent at most recent follow-up   
(3.1 - 5.8yrs), and 3 resumed insulin use at 24, 34, 34 months (see Table   
1). Mean duration of insulin independence in the 17 PTA recipients was 72.8   
months (12-136). Thus after final transplant, all ITA (100%) were insulin   
independent at 1 year and 7/10 (70%) at 3 years. For PTA, 16/17 (94%) were   
insulin independent at 1 year and 13/17 (76.5%) at 3 years. Significant   
complications in the ITA group included 1 partial portal vein thrombosis   
which resolved with anticoagulation, and 1 case of posttransplant   
lymphoproliferative disorder (PTLD) which resolved with therapy and did not   
result in graft loss. In the PTA group, there was 1 case of PTLD   
necessitating withdrawal of immunosuppression; 4 graft pancreatectomies for   
pancreatitis/rejection; 1 bowel obstruction; 3 incisional hernias; 1 soft   
tissue infection; and 2 conversions to enteric drainage. Renal function   
remained stable in 10/10 ITA and decreased in 5/17 PTA patients on CNI based   
regimens. Conclusions: Long term insulin independence following ITA   
performed in selected patients receiving co-stimulation/adhesion blockade is   
similar to that observed for PTA at our institution. Although selection   
criteria for ITA versus PTA are different, these data demonstrate increasing   
options to achieve long term insulin-free survival for people undergoing   
beta cell replacement for T1D. (Table presented).  
  
RECORD 381  
Coagulation in Liver Disease: A Guide for the Clinician  
Northup P.G. Caldwell S.H.  
Clinical Gastroenterology and Hepatology (2013) 11:9 (1064-1074). Date of   
Publication: September 2013  
  
The human hemostasis system is complex and poorly understood after decades   
of intense scientific study. Despite multiple defects in routine coagulation   
laboratory studies in patients with chronic liver disease, there is growing   
evidence that these patients are effectively "rebalanced" with regard to   
procoagulant and anticoagulant activity and that most of these patients   
remain in a tenuous but balanced state of hemostasis. A major difficulty in   
the assessment of these patients is that there are no established laboratory   
tests that accurately reflect the changes in both the procoagulant and   
anticoagulant systems; therefore, routine laboratory testing is misleading   
to the clinician and may prompt inappropriate or risky therapies with little   
real benefit to the patient. The international normalized ratio is an   
example of this type of misleading test. Although the international   
normalized ratio is inextricably linked to prognosis and severity of protein   
synthetic dysfunction in acute and chronic liver disease, it is a very poor   
marker for bleeding risk and should not be used in isolation for this   
purpose. Coagulation disorders are critical in the management of frequent   
clinical scenarios such as esophageal variceal bleeding, invasive and   
percutaneous procedures, portal vein thrombosis, venous thromboembolism, and   
acute liver failure. This article summarizes the pathophysiology of   
hemostasis in liver disease, describes the strengths and weaknesses of   
various laboratory tests in assessment of these patients, and outlines the   
optimal management of hemostasis for some common clinical scenarios. Further   
research is needed for proper understanding of hemostasis in liver disease   
to optimally and safely manage these complex patients. © 2013 AGA Institute.  
  
RECORD 382  
anticoagulation in cirrhosis: Ready ... set ... wait!  
Seijo S. Garcia-Pagan J.C.  
Hepatology (2013) 58:3 (1175-1176). Date of Publication: September 2013  
  
Background and Aims: We performed a randomized controlled trial to evaluate   
the safety and efficacy of enoxaparin, a lowmolecular-weight heparin, in   
preventing portal vein thrombosis (PVT) in patients with advanced cirrhosis.   
Methods: In a nonblinded, single-center study, 70 outpatients with cirrhosis   
(Child-Pugh classes B7-C10) with demonstrated patent portal veins and   
without hepatocellular carcinoma were assigned randomly to groups that were   
given enoxaparin (4000 IU/day, subcutaneously for 48 weeks; n 5 34) or no   
treatment (controls, n 5 36). Ultrasonography (every 3 months) and computed   
tomography (every 6 months) were performed to check the portal vein axis.   
The primary outcome was prevention of PVT. Radiologists and hepatologists   
that assessed outcomes were blinded to group assignments. Analysis was by   
intention to treat. Results: At 48 weeks, none of the patients in the   
enoxaparin group had developed PVT, compared with 6 of 36 (16.6%) controls   
(P 5 0.025). At 96 weeks, no patient developed PVT in the enoxaparin group,   
compared with 10 of 36 (27.7%) controls (P 5 0.001). At the end of the   
follow-up period, 8.8% of patients in the enoxaparin group and 27.7% of   
controls developed PVT (P 5 0.048). The actuarial probability of PVT was   
lower in the enoxaparin group (P 5 0.006). Liver decompensation was less   
frequent among patients given enoxaparin (11.7%) than controls (59.4%) (P 5   
0.0001); overall values were 38.2% vs 83.0%, respectively (P 5 0.0001). The   
actuarial probability of liver decompensation was lower in the enoxaparin   
group (P 5 0.0001). Eight patients in the enoxaparin group and 13 controls   
died. The actuarial probability of survival was higher in the enoxaparin   
group (P 5 0.020). No relevant side effects or hemorrhagic events were   
reported. Conclusions: In a small randomized controlled trial, a 12-month   
course of enoxaparin was safe and effective in preventing PVT in patients   
with cirrhosis and a Child-Pugh score of 7-10. Enoxaparin appeared to delay   
the occurrence of hepatic decompensation and to improve survival. © 2013 by   
the American Association for the Study of Liver Diseases.  
  
RECORD 383  
Case series: Thrombus resolution in 2 patients with portal vein thrombosis   
without anticoagulation-do we need to anticoagulate patients with portal   
vein thrombosis?  
Sule A.A. Borja A.M. Xing W. Lymen E. Azucena B. Chin T.J.  
Annals of the Academy of Medicine Singapore (2013) 42:8 SUPPL. 1 (S11). Date   
of Publication: August 2013  
  
Introduction: portal vein thrombosis (PVT) is thrombosis that develops in   
the trunk of the portal vein which can extend to its branches. It results   
from a combination of local and systemic prothrombotic factors. Methods: We   
describe 2 cases for this study. Patient 1 is a 77-year-old male who was   
admitted for cholangitis and pancreatitis and was found to have an   
incidental PVT. Patient 1's investigations and laboratory workup: total   
white count 23.0x109/L (neutrophils 91.6%), haemoglobin 11.7g/dL, platelets   
147x109/L; total bilirubin 184umol/L, alanine transaminase 111U/L, aspartate   
transaminase 113 U/L, gamma-glutamyltranspeptidase 515 U/L; amylase 641 U/L;   
hepatitis screening was negative. Abdominal computed tomography (CT) scan   
showed cholangitis with common bile duct calculi and an incidental   
thrombosis of the segmental branches of the right portal vein. Thrombophilia   
screen was negative. Patient 2 is a 60-year-old female with child's B   
cryptogenic liver cirrhosis and was admitted for gastroenteritis and left   
breast lump. She was found to have an incidental non-occlusive thrombus in   
the main portal vein. Her investigations and laboratory workup were as   
follows: total white cell 6.2x109/L (neutrophils 73.1%), haemoglobin   
9.1g/dL, platelets 116x109/L; Na 133 mmol/L, K 4.6 mmol/L, creatinine 115   
umol/L; albumin 29g/L, total bilirubin 25 umol/L, alanine transaminase   
27U/L, aspartate transaminase 42 U/L, C-reactive protein 15.5mg/L. CT scan   
showed left breast mass, cirrhosis with portal hypertension and   
non-occlusive portal vein thrombus. Results: Patient 1 underwent endoscopic   
retrograde cholangiopancreatography (ERCP), removal of stones and was given   
antibiotics. Patient was not anticoagulated due to the ongoing infection. A   
repeat CT scan 6 months later showed no evidence of PVT. Patient 2 underwent   
peritoneal drainage and was given antibiotics. No anticoagulation was given   
due to low platelet count. Eleven months later, an ultrasound Doppler of the   
hepatobiliary system revealed no evidence of vascular thrombosis. The left   
breast mass was later noted to be an invasive adenocarcinoma. Conclusion:   
The decision to anticoagulate a patient with portal vein thrombosis depends   
on several factors. Spontaneous resolution is possible but is an uncommon   
occurrence.  
  
RECORD 384  
portal vein thrombosis in neonates: Results of an anticoagulation protocol  
Williams S. Brandao L.R. Labarque V. Williams S.  
Journal of Thrombosis and Haemostasis (2013) 11 SUPPL. 2 (389). Date of   
Publication: July 2013  
  
Background: portal vein thrombosis (PVT) is a common thrombotic event in   
neonates. The majority of patients will have resolution of thrombus with a   
minority going on to develop complications such as portal hypertension. Due   
to the scarcity of published studies, the role of anticoagulation (ACT) in   
neonatal PVT is unclear. Aims: The aim was to describe the treatment and   
outcomes following portal vein thrombosis in a neonatal cohort treated   
according to an institutional anticoagulation protocol. Methods: The study   
was approved by the hospital research ethics board and written consent   
waived. A retrospective chart review of neonates presenting with PVT to the   
Hospital for Sick Children from January 2008 to September 2010, identified   
from the clinical thrombosis database, was conducted. All patients were < 30   
days at the time of diagnosis and treated according to the institutional   
protocol for anticoagulation for neonatal portal vein thrombosis. In the   
protocol, neonates with non-occlusive PVT are not treated with ACT unless   
thrombotic extension occurs or there is a concomitant other indication for   
ACT, and neonates with occlusive PVT without contraindication to ACT are   
treated. Clinical and radiologic data were collected. Poor outcome was   
defined as portal hypertension, hypersplenism, liver atrophy on follow-up.   
Descriptive statistics and Fisher exact testing were completed to compare   
the neonates by presenting features, treatment and outcome. Results: There   
were 94 patients identified. The mean age (± SD) was 10 days (± 5). The mean   
gestational age was 35 weeks (± 5). Fiftythree (56%) patients received ACT   
and 41 (44%) patients did not receive ACT. Of the patients treated with ACT   
6/53 (11%) received standard heparin, 34/53 (64%) received low molecular   
weight heparin and 13/53 (25%) received both standard and low molecular   
weight heparin. Follow-up occurred for a mean of 344 days (± 315). ACT was   
continued for a mean 41 days (± 55). Complete resolution of thrombus   
occurred in 48/94 (51%), partial resolution in 15/94 (16%) and progression   
in 25/94 (27%). Progression of PVT was associated with initial non-occlusive   
thrombus occurring in 15/36 (42%) of patients with non-occlusive thrombus   
vs. 9/57 (16%) of patients with initial occlusive thrombosis (P = 0.008).   
Thrombosis involving the inferior vena cava (IVC) was associated with   
thrombotic progression occurring in 8/12 (67%) of patients with IVC   
involvement compared to 16/83 (67%) of patients without IVC involvement (P =   
0.001). There was no association of anticoagulation with thrombus resolution   
or decreased rates of poor outcome. Liver atrophy occurred in 25/94 (27%).   
Portal hypertension or hypersplenism occurred in 6/94 (6%) patients. Major   
bleeds occurred in 7/53 (13%) of patients who received ACT. Conclusions: The   
majority of neonates had a good outcome, and anticoagulation was not   
associated with decreased rates of poor outcome in this cohort of patients   
treated according to an institutional protocol for neonatal PVT. Prospective   
studies may identify a subset of neonates who benefit from anticoagulation.  
  
RECORD 385  
Thrombotic complications of myeloproliferative neoplasms: Risk assessment   
and risk-guided management  
Casini A. Fontana P. Lecompte T.P.  
Journal of Thrombosis and Haemostasis (2013) 11:7 (1215-1227). Date of   
Publication: July 2013  
  
Philadelphia-negative myeloproliferative neoplasms are considered to be   
acquired thrombophilic states. Thromboses, both arterial and venous (not   
rarely in unusual sites), are often the initial events leading to the   
diagnosis. After diagnosis, the yearly incidence of thrombotic events is   
highly variable, and ranges from approximately 1% to 10%. The identification   
of patients at risk who may benefit from antithrombotic therapy remains a   
challenge, and it is currently based on age and history of thrombotic   
events. However, the predictive value of these clinical characteristics is   
rather limited. Few prospective studies and even fewer interventional   
randomized studies are available, and there are no studies designed to   
formally validate the use of risk stratification. The implementation of   
laboratory parameters such as leukocytosis and/or the JAK2 V617F mutation   
into a scoring system may be of interest. The mechanisms at work leading to   
thrombosis remain largely speculative, but are likely to be complex and   
multifactorial, with a prominent role of cell-cell interactions, mostly   
owing to qualitative changes. The long-term treatment options to prevent   
thrombosis are, schematically, aspirin alone as primary prevention for the   
low-risk patients, and cytoreduction combined with aspirin for the other   
patients. In very low-risk young essential thrombocythemia patients,   
abstention can even be considered. The optimal duration of anticoagulation   
after a thrombotic event is not established. All antithrombotic therapies   
should be balanced with the hemorrhagic risk, which can also be increased in   
these patients. © 2013 International Society on Thrombosis and Haemostasis.  
  
RECORD 386  
portal vein thrombosis in children and adolescents  
Williams S. Fayea N. Al Hinai K. Brandao L.R. Labarque V.  
Journal of Thrombosis and Haemostasis (2013) 11 SUPPL. 2 (1012). Date of   
Publication: July 2013  
  
Background: portal vein thrombosis (PVT) is an important cause of portal   
hypertension (PH) in children. Complications include upper gastrointestinal   
bleeding from the rupture of esophageal varices, hypersplenism secondary to   
splenomegaly, growth retardation, and portal biliopathy. Management may   
include portosystemic shunting. Unlike neonatal portal vein thrombosis which   
has low reported rates of portal hypertension of <5%, while gastrointestinal   
bleeding occurs in up to 80% of children with PVT. Aims: The aim was to   
describe the presentation, treatment, and outcomes of an unselected cohort   
of children presenting with portal vein thrombosis, to determine if the   
outcome differed with age of presentation. Methods: The study was approved   
by the hospital research ethics board and written consent waived. A   
retrospective chart review of infants and children with PVT presenting to   
the Hospital for Sick Children from January 2008 to January 2012, identified   
from the clinical thrombosis database was conducted. Neonates were excluded,   
and age was limited to 31 days to 18 years at time of presentation. Clinical   
and radiologic data were collected. Descriptive statistics and Fisher exact   
testing were completed to compare the two groups; infants (age ≤1 year) and   
children (age. >1 year). Results: 36 children with PVT were identified. At   
the time of presentation, 14/36 patients (38%) had underlying liver disease   
(biliary atresia (n = 6), sclerosing cholangitis (n = 3), metabolic disease   
(n = 1), chemotherapy induced liver disease (n = 2), hepatic infiltration (n   
= 2). The majority of PVTs, 19/36 (52%), were identified incidentally, on   
abdominal ultrasound completed during work up for other medical illness;   
(fever and sepsis (n = 7), pre- or post- liver transplant (n = 5), elevated   
liver enzymes (n = 2), pre Kasai (n = 1), abdominal pain (n = 10). Risk   
factors (intra-abdominal infection, sepsis or abdominal surgery) were   
present in 25/36 (70%). Infants (n = 20) had a mean age (±SD) of 5 months   
(±3), children (n = 16) had a mean age of 10 years (±5). Mean follow-up in   
infants was 24 months (±24) and in children was 36 months (±37). There was a   
history of a prior umbilical venous catheter in 10/20 (50%) infants and 1/16   
(6%) children (P = 0.0091). Cavernous transformation was found on initial   
imaging in 2/20 (10%) infants and 7/16 (44%) children (P = 0.049).   
Gastrointestinal bleeding at time of initial presentation was present in   
0/20 infants and in 5/16 (31%) children (P = 0.012). There was no difference   
between infants and children in hypersplenism (19%) gastrointestinal   
bleeding in follow- up (11%), PH at time of presentation (20%), PH in   
follow-up (31%), thrombus resolution (58%), need for portosystemic shunting   
(8%), growth failure (20%) or thrombophilia [low protein C, protein S,   
antithrombin] (44%). There was no difference in radiologic resolution with   
anticoagulation (67%) or without anticoagulation (57%). Conclusions: In this   
single institution cohort, limited by the small number of patients, rates of   
portal hypertension and gastrointestinal bleeding were similar in infants   
and children, but higher than reported rates in neonates. PVT in infants had   
a greater association with previous umbilical catheter, and presented less   
often with cavernous transformation or gastrointestinal bleeding than in   
children.  
  
RECORD 387  
Thrombotic risk factors in cirrhotic patients  
Baccouche H. Labidi A. Kaabi H. Mahjoub S. Hsouna K. Fekih M. Slama H.   
Filali A. Ben Romdhane N.  
Journal of Thrombosis and Haemostasis (2013) 11 SUPPL. 2 (1170-1171). Date   
of Publication: July 2013  
  
Background: cirrhosis results in a complex pattern of defects in haemostatic   
functions with reduced synthesis of pro and anticoagulant factors. As   
possible complication of coagulation disorders in cirrhosis, could be the   
development of arterial and venous thromboembolism (AVTE). The purpose of   
our study was to determine thrombotic risk factors in cirrhotic patients.   
Methods: Fifty one cirrhotic patients were enrolled into a case control   
study. The presence of personal and familial history of AVTE were   
investigated. White blood cells, platelet count, prothrombine time, INR,   
albumin, urea, procoagulant factors (VIII, XII, VII, II, V) were determined.   
Level of antithrombin, protein C and protein S were measured (respectively   
STACHROM AT, STACLOT PC, STACLOT PS; DIAGNOSTICA STAGO). Search for factor V   
Leiden and prothrombin gene mutation (G20210A) were performed with PCR-RFLP.   
Anticardioloipin and antiB2glycoprotein antibodies were also investigated.   
Results: Mean age was 56.8 years old (range16-86 years old). Sex ratio was   
0.9. Among the 51 cirrhotic patients, 7 patients (13.7%) had experienced   
AVTE after cirrhosis diagnosis: deep venous thrombosis (n = 2), pulmonary   
embolism (n = 1), Budd Chiari syndrome (n = 1), portal thrombosis (n = 3).   
They were compared to 46 cirrhotic patients without thrombosis. No patient   
with AVTE had neither personal nor familial history of thrombosis. In an   
univariate analysis, white blood cell count and platelet count were   
significantly higher in patients with AVTE than other cirrhotic patients   
(respectively, 8795 vs. 5032/mm(3), P < 0.018 and 91133 vs. 154375/mm(3), P   
= 0.03) However, In a multivariate analysis only the platelet count was   
independently predictive of AVTE in cirrhotic patients (P = 0.05). White   
blood count was not an independent predictive factor of thrombosis in   
cirrhotic patients (P = 0.07). Moreover, prothrombin time, INR, albumin,   
urea, level of pro and anticoagulant factors were not statistically   
different in both groups. There was no link between the presence of Factor V   
Leiden, prothrombin gene mutation (G20210A), anticardiolipin and   
antiB2glycoprotein antibodies to thrombosis. Conclusions: Approximately   
13.7% of cirrhotic patients resulted in a thromboembolic event. Platelet   
count was predictive of increased risk of AVTE as it was supported by other   
studies. Understanding the factors predisposing to thrombosis in cirrhotic   
patients could play a role in identifying a subgroup of patients at high   
risk of thrombosis and making decisions regarding the utility of   
anticoagulation therapy.  
  
RECORD 388  
Use of the AngioJet percutaneous thrombectomy system for the treatment of   
acute Budd-Chiari syndrome  
Doyle A. Nicoll A. Dowling R.  
BMJ Case Reports (2013). Date of Publication: 3 Jun 2013  
  
A 31-year-old woman presented to our emergency department with an acute   
liver injury secondary to acute Budd-Chiari (BC) syndrome from hepatic vein   
thrombosis. After a thorough discussion of the risks involved, we proceeded   
to treatment with a novel approach, performing a mechanical hepatic vein   
thrombectomy with the AngioJet percutaneous thrombectomy system. Restoration   
of hepatic vein flow was confirmed with on-table Doppler ultrasound. There   
were no complications following the procedure. The patient was initiated on   
anticoagulation, and showed progressive clinical and laboratory improvement.   
She was discharged home on day 20 with normal liver function and   
biochemistry. This is the first reported case of successful mechanical   
thrombectomy in acute BC syndrome without the addition of angioplasty or   
chemical thrombolysis. Copyright 2013 BMJ Publishing Group. All rights   
reserved.  
  
RECORD 389  
Intrahepatic portal vein thrombosis: Is gastric surgery a risk factor?  
Mainali N.R. Aryal M.R. Badal M. Alweis R.  
Journal of General Internal Medicine (2013) 28 SUPPL. 1 (S345-S346). Date of   
Publication: June 2013  
  
LEARNING OBJECTIVE 1: Recognize the clinical features and diagnosis of   
Intrahepatic portal vein thrombosis. LEARNING OBJECTIVE 2: Describe the   
treatment and prognosis of Intrahepatic portal vein thrombosis. CASE: Portal   
Vein thrombosis is frequently being diagnosed these days, with life time   
risk of 1 % in the general population. Intrahepatic portal vein thrombosis,   
however, is a less common entity than extrahepatic portal vein thrombosis.   
Usually, intrahepatic portal vein thrombosis is associated with   
hepatocellular carcinoma. In adults, approximately 25 % of patients with   
extrahepatic portal vein thrombosis have underlying cirrhosis that might   
extend into intrahepatic portal veins. Other common causes of intrahepatic   
portal vein thrombosis include prothrombotic disorder and abdominal   
inflammation. It might be complicated with splenomegaly, esophageal or   
gastric varices, portal hypertensive gastropathy or ascites. A 38-year-old   
morbidly obese female, with recent laparoscopic sleeve gastrectomy for   
weight control presented in the emergency department with severe abdominal   
pain for 2 days. Initially, the pain was crampy in nature and associated   
with burning sensations in the epigastric area. It was also associated with   
nausea and vomiting. She was passing flatus with normal bowel movements.   
There was no history of melena, hematochezia, hematemesis or dysuria. She   
denied fever, chills, chest pain and shortness of breath. In the meantime,   
she was being treated for left lower lobe pneumonia. Physical exam revealed   
mild tenderness in the mid-epigastric area but no obvious   
hepatosplenomegaly. Laboratory investigations revealed hemoglobin 12.1   
gm/dL, WBC 7800/μL, platelets 232000/μL and normal abdominal X-ray and serum   
electrolytes. D-Dimer was elevated at 1000 mg/dL. Abdominal CTscan showed   
intrahepatic portal vein thrombosis in the left lobe of the liver. Protein   
C, protein S, antithrombin III were all normal and factor V Leiden,   
anti-cardiolipin antibodies and lupus anticoagulant antibodies were found to   
be negative. anticoagulation with heparin and warfarin was initiated and she   
was discharged on warfarin for 6 months. On follow up exam after 6 months,   
she was asymptomatic without any evidence of thrombosis on the abdominal CT   
scan. DISCUSSION: The patient presented with several risk factors, including   
recent surgery. Some studies suggest the possibility that the CO2 used for   
pneumoperitoneum may increase risk, but tissue damage from the surgery   
itself is a well-recognized risk factor for thrombophilia. Tissue damage   
from gastric surgery may lead to release of procoagulant products in the   
portal venous system eliciting portal vein thrombosis. Additionally,   
infection like pneumonia and obesity itself are risk factors for thrombosis.   
Since her pneumonia was almost resolved with treatment, negative   
prothrombotic work-up, recent surgery was the most likely explanation of her   
condition. Treatment consists of anticoagulation for at least 6 months. It   
is prudent to repeat the abdominal CT scan after 6 months to confirm the   
resolution of thrombus. It is unclear whether a follow-up D-dimer is useful   
in the decision to discontinue anticoagulation. Despite this increasingly   
common condition, there remains a paucity of studies to guide clinicians.  
  
RECORD 390  
Management dilemma: Progressive thrombosis in a patient with sarcoidosis,   
cirrhosis and a history of bleeding esophageal varices  
Pan L. Iheanyichukwu O. Desilets D. Canty L.J.  
Journal of General Internal Medicine (2013) 28 SUPPL. 1 (S358). Date of   
Publication: June 2013  
  
LEARNING OBJECTIVE 1: Recognize the common causes of portal vein thrombosis   
in the setting of liver cirrhosis. LEARNING OBJECTIVE 2: Understand the   
risks and benefits of anticoagulation vs. conservative management in   
patients with chronic portal vein thrombosis secondary to liver cirrhosis.   
CASE: A 60 year old female with past medical history significant for liver   
cirrhosis secondary to sarcoidosis, upper GI bleeding due to esophageal   
varices which were subsequently banded, iron-deficiency anemia, portal vein   
thrombosis (PVT) and superior mesenteric vein thrombosis that was diagnosed   
seven months prior presented with subacute worsening of abdominal pain. The   
abdominal pain was severe and localized to the midepigastric region   
associated with nausea without vomiting, BRBPR or melena. On physical   
examination, patient had stable vital signs. Her abdomen was soft, nontender   
and nondistended with active bowel sounds. CT scan revealed a greater degree   
of thrombosis in the superior mesenteric vein and portal vein (PV) with   
tortuous mesenteric vessels presumably collaterals. Hypercoagulable workup   
revealed a mildly elevated antidcardiolipin (aCL) IgM of 19 MPL/ml, while   
the remainder of the antiphospholipid panel was negative. Factor V Leiden,   
Factor II Prothrombin, Protein S deficiency and JAK-2 mutation (screening   
for myeloproliferative disorder) were negative. Protein C was low at 49 %   
(normal range 74-151). The patient was managed conservatively with IV fluids   
and pain control. Her abdominal pain improved during the hospital stay.   
DISCUSSION: anticoagulation therapy was contemplated for several reasons.   
anticoagulation in patients with PVT could lead to recanalization of the PV   
and prevent progression of PVT. The worsening thrombosis, reduced level of   
protein C and elevated aCL IgM were concerning for a possible underlying   
prothrombotic disorder contributing to the PVT. However, the lowered protein   
C could have been an acquired condition due to the patient's liver   
cirrhosis. In addition, aCL IgMhas been found in 18.8 % of patients with   
chronic liver disease unrelated to thrombosis and was considered an   
epiphenomenon of chronic liver damage. Even among patients with systemic   
lupus erythematosus, aCL antibodies were not an independent risk factor for   
thrombosis-related event. Furthermore, portal vein recanalization is more   
likely to be achieved if anticoagulation was initiated within 6 months of   
diagnosis. Our patient was diagnosed with PVT seven months prior. Finally,   
the clinical benefits of PVrecanalization in this particular patient are   
uncertain. Mortality benefits have only been demonstrated in individuals   
with PVTon the waiting list for liver transplant. Even though patients with   
PVT treated with anticoagulation did not have significant esophageal   
variceal bleeding, the risk of bleeding was taken into consideration given   
this patient's known prior bleeding. Finally, the PVT is likely due to   
reduced flow in the portal system. After careful deliberation, the decision   
was made not to anticoagulate. As of nine months of follow-up, no further   
complications have developed.  
  
RECORD 391  
Do all post-liver transplant patients need thrombo-prophylactic   
anticoagulation?  
Mukerji A. Karachristos A. Maloo M. Johnson D. Jain A.  
Liver Transplantation (2013) 19:6 SUPPL. 1 (S245). Date of Publication: June   
2013  
  
Background: Thromboprophylactic anticoagulation against Deep Vein Thrombosis   
(DVT) and Pulmonary Embolism (PE) is standard of care in general surgery   
with. Majority liver transplant (LT) patients have thrombocytopenia and/or   
prolonged INR before surgery, which is a special challenger There is a lack   
of studies or guidelines regarding role of prophylactic anticoagulation   
after LT. Aim: Discuss routine prophylactic anticoagulation after LT,   
Discussion: Risk of DVT after major general surgery without prophylaxis is   
25%, of PE 1,6% and fatal PE 0,9% and are considered preventable. With   
prophylaxis the incidence of DVT is 7-8% (70% decrease) and fatal PEs   
decreased two-thirds. On the other hand, the incidence of major bleeding   
following surgery with prophylactic anticoagulation was 3,6-4.S% with no   
significant increase due to anticoagulation. Occurrence of wound hematomas   
Increased, but did not affect mortality. However, after LT without   
prophylactic anticoagulation, incidence of major bleeding needing   
transfusions/reoperation was 8.4-27%, which was significantly higher than   
after general surgery. The incidence of DVT was 2,7% and PF. 1%, presenting   
after y mean of 70 and 128 days following the surgery. This was   
significantly lower than in general surgery and also presented later. Most   
proven fatal PEs In Lis were intra-operative. Of special importance after   
LT. the incidence of hiepatic artery thrombosis (HAT) was 4.2% with 52,8%   
long term mortality and 53.1% re-transplant rate. The incidence of portal   
vein thrombosis (PVT) following LT was 22-2.6%, with in hospital mortality   
of 30% and 5-year survival of 65.5 After LT, anticoagulation is not required   
routinely for DVT/PE prophyiaxis. Rather, it is required in specific   
circumstances outlined in Table 1. Even in them anticoagulation must be   
started cautiously after INK decreases below -1,5, platelet count is above   
-50,000 and clinical bleeding ceases. (Table presented) Conclusion: After LT   
routine anticoagulation for DVT/PL is not needed. It is io be considered   
judiciously for prophylaxis of HAT and PVT in high risk cases (Table 1).  
  
RECORD 392  
Optimal treatment duration of venous thrombosis  
Ageno W. Dentali F. Donadini M.P. Squizzato A.  
Journal of Thrombosis and Haemostasis (2013) 11:SUPPL.1 (151-160). Date of   
Publication: June 2013  
  
Randomized controlled trials have shown that patients with venous   
thromboembolism benefit from a minimum of three months of anticoagulant   
therapy. After this period, it was suggested that patients with an expected   
annual recurrence rate of < 5% could safely discontinue treatment. Using a   
population-based approach for stratification, these patients are those with   
major transient risk factors, and represent the minority. For all other   
patients, including those with previous episodes of venous thromboembolism,   
cancer, or unprovoked events, this treatment duration may not be   
sufficiently protective. Because extending anticoagulation for additional   
three to nine months does not result in further, long-term reduction of   
recurrences, indefinite treatment duration should be considered. However,   
case-fatality rate for major bleeding in patients taking warfarin for more   
than three months is higher than case-fatality rate of recurrent venous   
thromboembolism. Thus, an individual patient approach to improve and   
increase the identification of those who can safely discontinue treatment at   
three months becomes necessary. Clinical prediction rules or management   
strategies based on D-dimer levels or residual vein thrombosis have been   
proposed and need further refinement and validation. Specific bleeding   
scores are lacking. Meanwhile, the oral direct inhibitors have been proposed   
as potential alternatives to the vitamin K antagonists, and aspirin may   
provide some benefit in selected patients who discontinue anticoagulation.   
Deep vein thrombosis in unusual sites is associated with less, but   
potentially more severe recurrences, in particular in patients with   
splanchnic vein thrombosis who also face an increased risk of bleeding   
complications while on treatment. © 2013 International Society on Thrombosis   
and Haemostasis.  
  
RECORD 393  
Pus, thrombosis and fusobacterium necrophorum infection: A recurrent theme  
Sandouk Z. Montezuma D. Uduman A.K. Weinmann A.  
Journal of General Internal Medicine (2013) 28 SUPPL. 1 (S377). Date of   
Publication: June 2013  
  
LEARNING OBJECTIVE 1: Recognize Fusobacterium necrophorum infection as a   
possible etiology for portal vein thrombosis. LEARNING OBJECTIVE 2: Manage   
Fusobacterium necrophorum infection and its complications: thromboembolic   
events, abscess and sepsis. CASE: A 56 years old man with history of heavy   
alcohol use, presented to the hospital with complaints of weight loss, right   
upper quadrant pain and night sweats for 1 month. His laboratory findings   
were normal except for normocytic anemia. Hepatitis panel, HIV, blood and   
urine cultures were negative. Tuberculosis was ruled out. Ultrasound of the   
abdomen revealed right portal vein thrombosis without liver or gallbladder   
abnormalities. anticoagulation was started and malignancy work up was   
performed, including CT chest, abdomen and pelvis, endoscopy and   
colonoscopy. No suspicious masses or lymph nodes were detected. Thrombotic   
work up was negative. The patient did not have any recorded fever while in   
the hospital, was stable and discharged with warfarin. He returns a month   
later with the same complaints. Work up included blood, urine and fungal   
cultures (fungitell, galactoman, and urine for histoplasma) all of which   
were negative. A repeat CT abdomen reveals interval development of an   
abscess within the right hepatic lobe measuring 8.8×6.8 cm. He was started   
on empiric ceftriaxone and metronidazole. One hundred milliliters of white   
purulent fluid was aspirated. Gram stain of the fluid was negative as were   
aerobic cultures, but the anaerobic culture grew Fusobacterium necrophorum.   
Hospital course was complicated by septic shock and development of right   
sided empyema attributed to contiguous spread of infection from the liver,   
requiring video assisted thoracoscopy with decortication. Due to the size of   
the hepatic abscess complete drainage was not feasible, it was decided to   
treat with oral metronidazole for several weeks, with planned reevaluation   
by CT as outpatient. DISCUSSION: Fusobacterium necrophorum is an anaerobic   
gramnegative bacillus that belongs to the normal oropharyngeal flora. It is   
associated with septic venous thrombosis, Lemierre's disease, in which   
thrombosis of the internal jugular vein is precipitated by an upper   
respiratory infection. Primary foci of F. necrophorum infection in sites   
other than the head are uncommon, but can occur in the urogenital or   
gastrointestinal tracts. Clinical features include fever, dyspnea, malaise,   
and night sweats. The infection is most often recognized with isolation of   
the bacteria from a sterile body site (blood or abscesses). Compared with   
Lemierre's syndrome, illness due to primary foci caudal to the head carries   
a higher mortality rate. Complications include abscesses and septicemia.   
Metronidazole has been found to be the drug of choice, with duration of   
treatment from 3 to 6 weeks. Response to antibiotics is slow because of the   
endovascular nature of the infection. Our patient is responding slower than   
expected with planned longer duration of antimicrobials in the setting of an   
undrainable abscess. Therapeutic anticoagulation to prevent thromboembolic   
complications is controversial. It is used most frequently for patients with   
an underlying thrombophilia, a cerebral infarct, cavernous sinus thrombosis,   
and refractory disease. The most beneficial role of surgery is associated   
with drainage of the abscess within the neck, lung or liver. Finally, it is   
important to exclude underlying malignancy with nonhead primary foci as up   
to 69 % of patients have underlying malignancies of the affected system.  
  
RECORD 394  
Living donor liver transplantation in a case of Budd Chiari syndrome with   
IVC stent and portal vein thrombosis: A case report  
Sood G. Chorasiya V. Makki K. Lalwani S. Dargan P. Vij V.  
Liver Transplantation (2013) 19:6 SUPPL. 1 (S307-S308). Date of Publication:   
June 2013  
  
Background: portal vein thrombosis is seen in 15-20% eases of Budd Chiari   
Syndrome (BCS) and it generally signifies poor prognosis. Traditionally   
cadaveric liver transplantation has been advocated for these patients as   
most of them require excision of Inferior Vena Cava (IVC), However scarcity   
of cadaveric organs and development of innovative techniques have allowed   
the performance of living donor liver transplantation (LDLT) in this group.   
The presence of metal stent in IVC further complicates the issue. We herein   
report our experience of performing LDLT in one such ease. Methods: We have   
performed LDLT in 2 cases of BCS oat of 37 from December 2011 till December   
2012. We report a case of 33 year old male with protein C deficiency that   
was diagnosed with BCS and portal vein thrombosis (PVT) three years ago. He   
had a (ailed attempt of TIPS and an IVC stent was inserted in a different   
center. As his bilirubin was progressively increasing and he had failed   
medical and interventional therapy he was listed for LDLT. His sister was   
evaluated as a donor and screened for Protein C deficiency which was   
negative. Surgery: Eversion thrombectomy was done to completely remove the   
thrombus from the portal vein. IVC was mobilized up to the atrium after   
phreno-caval dissociation. The anastomoses of the Right hepatic vein, Middle   
hepatic vein and inferior hepatic vein was done above the stent after   
extending these veins with cadaveric iliac vessels. Results: The   
post-operative recovery was uneventful. His protein C level was normal in   
the 2nd week, after transplantation and he was discharged in the 3rd week   
without any anticoagulation. Follow up triple phase CT revealed patent flow   
in the portal as well as hepatic veins. He continues to do well f months   
after the surgery and his serum bilirubin is normal Conclusions This paper   
is an attempt to add to already emerging data that LDLT tit BCS is safe and   
effective even in the presence of portal vein thrombosis. Even in difficult   
cases with IVC stent in situ, it might be possible to preserve the IVC.  
  
RECORD 395  
Catastrophe!  
Atreya A.R. Kitt E. Besharatian B. Verma A. DeMatteo M.  
Journal of General Internal Medicine (2013) 28 SUPPL. 1 (S304-S305). Date of   
Publication: June 2013  
  
LEARNING OBJECTIVE 1: Recognize early the challenging diagnosis of   
Catastrophic Antiphospholipid Antibody Syndrome (CAPS), in order to direct   
management appropriately as the condition is life-threatening. CASE: KP, a   
26 years old African-American male with SLE, noncompliant with his   
outpatient steroid therapy, presented with 3 days of constitutional symptoms   
and was found to have acute kidney injury, pancreatitis, profound anemia   
needing transfusion (Hb 5.7), thrombocytopenia, acute decompensated heart   
failure with troponin elevation. Echocardiogram showed global hypokinesis   
with regional areas of akinesis, suggestive of infarction. He was started on   
high dose steroids for presumed SLE flare. Subsequently, he had massive   
upper GI bleed; EGD revealed gastritis as well as esophageal varices. US of   
liver revealed portal vein thrombosis with cavernous transformation and   
splenic vein thrombosis. His renal function worsened necessitating dialysis.   
He became more somnolent with development of neurological deficits; CSF   
analysis showed no signs of infection and an MRI demonstrated disseminated   
sub-acute ischemic infarcts. At this point, concern for a more serious   
condition such as concomitant antiphospholipid syndrome was raised. Renal   
biopsy showed multifocal arterial and arteriolar thrombi, consistent with   
CAPS. There was no evidence of lupus nephritis. Positive serology   
(anti-cardiolipin IgM 25 ug/mL) helped confirm diagnosis of CAPS and he was   
started on anticoagulation, cyclophosphamide and plasmapheresis. Eventually,   
KP recovered from his cataclysmic disease and was discharged to a   
rehabilitation center with long-term warfarin and prednisone therapy.   
DISCUSSION: Catastrophic antiphospholipid syndrome (CAPS) was first   
described by Ronald Asherson in 1992 and is diagnosed using the   
classification criteria proposed by the International Congress on   
Antiphospholipid in 2011. This includes the presence of all of the   
following: evidence of involvement of≤3 organs; manifestations occurring   
simultaneously or in less than 1 week; histopathological confirmation and   
serological confirmation (anticardiolipin/lupus anticoagulant/anti-beta2   
glycoprotein antibodies). Although this condition is fairly uncommon in   
patients with antiphospholipid syndrome (<1 %), it is a life-threatening   
condition with greater than 50 % mortality. The pathogenesis of this disease   
is still unclear, but it is postulated that certain triggers (eg. infection)   
facilitate a 'cytokine storm' that promotes inflammation and thrombosis.   
Treatment options include anticoagulation, steroids, cyclophosphamide,   
plasmapheresis. Additional therapies such as rituximab, eculizumab and IVIG   
have been used with some success for refractory CAPS. In our patient, the   
presence of cerebral infarcts, myocardial infarcts, renal thrombosis,   
ischemic gastritis, portal vein thrombosis, pancreatic injury within a short   
duration raised concerns for a serious disorder. Histological and   
serological tests confirmed the diagnosis. Despite lack of experience with   
this disorder, prompt review of medical literature and early sub-specialist   
consultation were vital to ensure a favorable outcome.  
  
RECORD 396  
Vascular outcomes in segmental liver transplantation  
Rodriguez-Davaios M.I. Arvelakis A. Umman V. Tanjavur V. Yoo P. Kulkarni S.   
Luczycki S. Schilsky M. Emre S.  
Liver Transplantation (2013) 19:6 SUPPL. 1 (S245-S246). Date of Publication:   
June 2013  
  
Aim: Describe the use of segmental allograft in the current era of liver   
transplantation, and the importance of minimizing vascular complications to   
obtain excellent outcomes. Methods: Liver transplants performed from 8/07 to   
12/12 at the Yale New Haven Transplantation Center were reviewed. Recipient   
demographics, donor and graft type, operative techniques, vascular   
complications, graft and patient outcomes were analyzed. Donor selection   
criteria for splitting and living donor selection were strict: in-situ   
splitting is our preference, we advocate left lobe hepntectomy for living   
donation if possible in view of donor safety Surgical techniques typically   
included the use of microsurgical instruments and loupes (3.5X), For hepatic   
artery anastomoses interrupted 8-0 polypropylene sutures are used, for   
hepatic vein out flow, we dont perform complex hack table venoplasty;   
instead our technique was to drain segmental veins directly into vena cava   
or use interposition grafts. anticoagulation protocol included the use of   
low molecular weight heparin for a week, and baby aspirin indefinitely.   
Demographics, mean follow-up and outcomes. (Table presented) There was no   
hepatic artery or portal vein thrombosis. Two patients developed hepatic   
artery stenosis, treated with angioplasty. Graft/Patient survival was   
97.2%/98.6% respectively Conclusion: Minimizing vascular complications in   
segmental grafts is fundamental to obtain excellent outcomes, we promote   
strict donor and patient selection and routine anticoagulation.  
  
RECORD 397  
Role of anticoagulant therapy in liver disease  
Plompen E.P.C. Schouten J.N.L. Janssen H.L.A.  
Hepatology International (2013) 7:2 (369-376). Date of Publication: June   
2013  
  
Anticoagulant therapy is a cornerstone in the treatment of different liver   
diseases. In Budd-Chiari syndrome (BCS), survival rates have increased   
considerably since the introduction of a treatment strategy in which   
anticoagulation is the treatment of first choice. In all patients diagnosed   
with acute portal vein thrombosis (PVT), anticoagulant therapy for at least   
3 months is indicated. anticoagulation should also be considered in patients   
with chronic PVT and a concurrent prothrombotic risk factor. Current   
evidence suggests that patients with PVT in cirrhosis will benefit from   
treatment with anticoagulation as well. In severe chronic liver disease the   
levels of both pro- and anticoagulant factors are decreased, resetting the   
coagulant balance in an individual patient and making it more prone to   
deviate to a hypo- or hypercoagulable state. An increased activity of the   
coagulation cascade is not solely a feature of chronic liver disease; it   
influences the development of liver fibrosis as well. Several studies in   
animals and humans have shown that anticoagulation could prevent or improve   
fibrogenesis and even disease progression in cirrhosis. anticoagulation is   
therefore a promising antifibrotic treatment modality. © 2013 Asian Pacific   
Association for the Study of the Liver.  
  
RECORD 398  
portal vein thrombosis in patients with end stage liver disease awaiting   
liver transplantation: Outcome of anticoagulation  
Werner K.T. Sando S. Carey E.J. Vargas H.E. Byrne T.J. Douglas D.D. Harrison   
M.E. Rakela J. Aqel B.A.  
Digestive Diseases and Sciences (2013) 58:6 (1776-1780). Date of   
Publication: June 2013  
  
Background: The prevalence of portal vein thrombosis (PVT) increases with   
the severity of liver disease. Development of PVT is often accompanied by   
increased rate of morbidity and mortality and may affect patient candidacy   
for liver transplant. There is limited data regarding the role of   
anticoagulation therapy in patients with PVT and liver cirrhosis.   
Objectives: The aims of this study were to describe the prevalence of   
hypercoagulable disorders in patients with liver cirrhosis and PVT, and to   
describe the outcome of anticoagulation in patients with liver cirrhosis and   
PVT. Methods: A retrospective chart review was conducted of patients with   
liver cirrhosis awaiting liver transplant who were diagnosed with PVT   
between January 2005 and November 2011. Results: During the study period,   
537 patients were evaluated for liver transplant. Sixty-nine (13 %) patients   
were diagnosed with portal vein thrombosis. Chronic hepatitis C was the   
cause of liver disease in 24/69 (35 %) patients, and hepatocellular   
carcinoma was present in 39 % of patients. In 22 patients screened for   
hypercoagulable disorders, hypercoagulable disorder was diagnosed in one   
patient (5 %). Twenty-eight (28/69) patients were treated during the study   
period with warfarin: PVT resolved in 11/28 (39 %), no change in 5/28 (18   
%), and 12/28 (43 %) patients showed partial resolution of thrombus. Eight   
patients received liver transplant while on anticoagulation, and operative   
notes confirmed patency of PV in all eight patients. Conclusions: PVT is   
frequently seen in patients with end stage liver disease with prevalence of   
13 %. Hypercoagulable disorder was detected in 5 % of the patients screened.   
Careful use of anticoagulation is safe and effective in patients with PVT. ©   
2013 Springer Science+Business Media New York.  
  
RECORD 399  
Antiphospholipid antibodies: An under-recognized cause of morbidity in   
patients transplanted for end-stage liver disease  
Villamil A. Galdame O.A. Bandi J.C. De Santibanes E. Gadano A.C.  
Liver Transplantation (2013) 19:6 SUPPL. 1 (S96). Date of Publication: June   
2013  
  
Circulating antiphospholipid antibodies (aPL-ab) are often detected In   
patients with liver discase. Aim: To establish prevalence of aPL-ab in   
patients transplanted for chronic liver disease and to assess their impact   
at 1 year post-OLT. Methods: Between Jan 2006 and Dec 2010, 150 patients   
transplanted for chronic liver disease were screened for aPL-ab (IgG and IgM   
isotypes) and lupus anticoagulant activity. Clinical and Doppler-ultrasound   
evaluations were performed before OLT and at different time-points   
post-transplant. Results were compared with aPL-ab negative patients. All   
patients received aspirin and/or low weight heparin post-OLT. Median   
follow-up: 26 months (12-56), Results: 39/150 patients (24%) evidenced   
increased levels of aPL-ab pre-OLT. Child C patients had a lower prevalence   
of aPL-ab than Child B patients (21 vs 32 %. p NS). No difference was   
observed in renal or liver function tests, except for bilirrubin levels   
which were higher in aPL-ab + patients (5.9 vs 3.6 mg/dl, p=0.04). Seven   
thrombotic complications were observed in 6/36 aPL + patients post-OLT   
(humeral thrombosis, n=1, cerebrovascular ischemia n=3, hepatic artery   
thrombosis n=1, retinal thrombosis=1, intestinal ischemia n=1) resulting in   
one graft loss and one death, compared to nine thrombotic complications in   
8/114 patients aPL negative resulting in one graft loss and   
re-transplantation (p<0.05). Four patients in the ApL+ group developed   
catastrophic antiphospholipid syndrome and 3/4 died in spite of early   
plasmapheresis and anticoagulation. No differences were observed between   
both groups in infection rates, thrombocytopenia, acute cellular rejection   
or bleeding complications. In 3/4 patients acute cellular rejection was a   
potentially triggering factor of CAPS. No patient with aPL antibodies   
pre-OLT developed portal vein thrombosis on follow-up. Only one patient that   
was aPL negative pre-OLT presented “de novo” anticardiolipin antibodies   
post-OLT and developed an aPL associated vascular complication   
(cerebrovascular ischemia). Conclusion: Patients with end-stage liver   
disease have a high prevalence of aPL antibodies. The presence of aPL   
antibodies is associated with a higher risk of morbidity post-OLT Pre-OLT   
screening for anticardiolipin and lupus anticoagulant, and a high index of   
suspicion of ApL vascular complications post OLT is recommended to improve   
outcome.  
  
RECORD 400  
Single dose of steriod combined with two dose of basiliximab for immune   
induction in liver transplantation with donation after cardiac death: Single   
centre experience in China  
Deng F. Zhen Z. Chen H. Zhu X. Ji Y. Chen Y. Li J. Wang F. Li Q. Li M. He Y.  
Liver Transplantation (2013) 19:6 SUPPL. 1 (S267). Date of Publication: June   
2013  
  
Objective: To explore the immune induction role of single dose of sic nod   
combined with two dose of basiliximab in liver transplantation with donation   
a Her cardiac death (DCD). Material and Methods: Fifteen liver   
transplantation with DCD were performed in our centre between November 5   
2011 and December 31 2012, Liver transplant recipients including twelve   
cases with hepatitis B virus infection (four with hepatocellular carcinoma   
and two with fulminant hepatic failure), one with hepatitis C virus   
infection, one with polycystic liver and polycystic kidney, and one with   
autoimmune disease associated liver cirrohsis and hepatocellular carcinoma.   
Four recipients underwent classic orthotopic liver transplantation and   
eleven recipients underwent modified piggyback liver transplantation. Single   
dose of methylprednisolone 500mg was Injected at anhepatic phase combined   
with two dose of basiliximab 20mg (day 0 and day 4 after transplantation).   
No more steriod was used. Tacrolimus dose of 2mg combined with mycophenolate   
mofetil tablets dose of 0.5g twice a day was started at day 2 after   
transplantation. The blood drug level of tacrolimus was monitored   
regularity, which was maintained at 8-13ng/ml within 3 months, 7-10ng/ml for   
3-6 months, 6-8ng/ml for 6-12 months, and 5ng/m1 for more than 1 year.   
Results: All the recipients recovered well after transplantation except one   
died of multiple organ failure at day 8 postoperation. One recipient occured   
acute rejection at day 30 after transplantation as for low tacrolimus level   
leading by the severe diarrhea, and was inversed with tacrolimus dose   
adjustment. No clinical manifestation or biospy proven rejection occured in   
the other recpients, the liver function recovered to normal level at day   
6-10 postoperation. All the survival recipients with good liver Junction   
until now. Hypertension, renal injury or diabetes mellitus occurrence was   
not found in the recipients. Conclusion: The single dose of steriod combined   
with two dose of basiliximab for immune induction was safe in liver   
transplantation with DCD.  
  
RECORD 401  
Portal venous thrombosis after distal pancreatectomy: Risk factors and   
outcomes  
Kamath A.S. Kendrick M.L. Sarr M.G. Nagorney D.M. McBane R. Farnell M.B.   
Lombardo K.M.R. Que F.G.  
Gastroenterology (2013) 144:5 SUPPL. 1 (S1080). Date of Publication: May   
2013  
  
Aim: Outcomes of patients developing portal vein (PV) thrombosis (PVT) after   
distal pancreatectomy (DP) are unknown. The goal of this study was to   
identify risk factors for PVT and describe the long term outcomes in these   
patients. Methods: Patients undergoing DP without repair or reconstruction   
of the PV between 2001 and 2011 were included. Patients that showed evidence   
of PVT on pre-operative imaging were excluded from the study. Location and   
extent of thrombosis was determined by postoperative CT or ultrasound   
imaging in all patients. Evidence of systemic thrombosis (if present) in   
addition to PVT was also documented. Results: In the study period, 991   
patients underwent DP and 21 (2.1 %) patients were diagnosed with PVT.   
Pancreatic neoplasm was the most frequent indication for operation (n = 11).   
Thrombus occurred in the main PV in 15 and the right branch of the PV in 8   
patients. Complete PV occlusion occurred in 9 patients with a median time to   
diagnosis of 16 days (range 5 - 85 days). Seventeen patients were   
anticoagulated for a median duration of 6 months (range 3.3 - 36 months)   
after the diagnosis of PVT. Over a median follow up of 22 months, resolution   
of PVT occurred in 7 patients. Predictors of non-resolution of PVT included   
anesthesia time .180 minutes (p = 0.025), DM type II (p = 0.03), BMI .30   
Kg/m2 (p = 0.03), occlusive PVT (p <0.001), or thrombus in a sectoral branch   
(p = 0.02). anticoagulation therapy did not influence the frequency of   
thrombus resolution and was complicated by gastrointestinal hemorrhage in 4   
patients. There was no mortality as a direct result of PVT or   
anticoagulation. Conclusion: PVT after distal pancreatectomy is a rare   
complication. Serious complications as a direct result of PVT in this   
setting are uncommon and are not dependent on thrombus resolution. Although   
anticoagulation does not appear to influence the rate of PVT resolution in   
this small retrospective series, we support the use of anticoagulation until   
larger, controlled-studies define clear advantages or disadvantages.  
  
RECORD 402  
Splanchnic vein thrombosis: A difficult management  
Casali A. Arioli D. Leone M.C. Pizzini A.M. Romagnoli E. Iori I.  
Italian Journal of Medicine (2013) 7 SUPPL. 2 (21-22). Date of Publication:   
May 2013  
  
Clinical report: We describe the case of a 42 years old woman affected by a   
myeloproliferative neoplasm diagnosed 20 years ago, treated with   
oncocarbide. In September 2012 the patient experienced abdominal pain and a   
complete extrahepatic portal vein thrombosis with patent hepatic veins and   
important splenomegaly was diagnosed by CT-scan. She had a severe portal   
hypertension with esophageal varices F2-F3 with cherry red spots and gastric   
varices F2. We excluded inherited thrombophilia. The JAK2 mutation was   
found. Management: The patient underwent endoscopic variceal ligation. She   
was treated with prophylactic dose of LMWH for the severe portal   
hypertension, then with LMWH at the dose of 4.000 U bid in a 60 kg patient.   
When we get complete eradication of esophageal varices,a long-term   
anticoagulant therapy will be indicated. Conclusions: The close relationship   
between myeloproliferative neoplasms and splanchnic vein thrombosis has been   
confirmed by the current one third prevalence of the JAK2 mutation among   
patients with Budd Chiari syndrome and extrahepatic portal vein thrombosis.   
The JAK2 mutation is associated with hypercoagulability and carriers are   
more prone to thrombosis. The management of anticoagulant therapy in   
patients with extrahepatic portal vein thrombosis and esofaeal varices is   
difficult because of the balance between bleeding and thrombotic risk.   
Long-term oral anticoagulation with vitamin K antagonists is recommended in   
patients with extrahepatic portal vein thrombosis and permanent   
prothrombotic state.  
  
RECORD 403  
Shunt surgery for extra hepatic portal venous obstruction: Keeping it simple  
Bhalla V.P. Vij J.C. Vats R. Goel D.  
Gastroenterology (2013) 144:5 SUPPL. 1 (S914). Date of Publication: May 2013  
  
Aim To present a simple cost effective and innovative approach for surgical   
shunting to lower portal pressures and compare the same with the more   
complex REX shunt. Background Beginning with the Eck fistula, surgical   
shunts for lowering portal pressures have always been an interesting   
proposition. During the 80's and 90's many centres had impressive series of   
portosystemic shunts. About this time endoscopic management strategies   
developed rapidly and proved invaluable in management of bleeding varices   
associated with portal hypertension (PHT) and interest in surgical shunting   
waned. Introduction of the REX shunt for bypassing an extrahepatic portal   
block by performing a mesenterico left portal shunt has again brought   
surgery for patients with extrahepatic portal obstruction with good liver   
function back into reckoning. The REX shunt was initially described for   
paediatric patients with extrahepatic obstruction. While it is a good shunt   
to lower portal pressure even while it maintains hepatofugal blood flow it   
is challenging to plan and technically demanding to learn and perform.   
Indian experience suggests that the proximal leinorenal shunt is a simpler   
shunt which is relatively easy to learn and do. There is no need for   
elaborate imaging studies to plan the shunt and often a simple abdominal   
ultrasonography has been used for planning surgery. Also pressure monitoring   
including wedge hepatic venous pressure gradient is possibly not essential   
in making management decisions for diagnosed PHT with good liver function.   
Compared to this the Rex shunt needs elaborate angiography and involves   
operating in the region of the porta hepatis often in the presence of   
collaterals. The left portal branch may also be involved by the extending   
main portal vein thrombus and there is a need for an interposition   
autologous or synthetic graft. Patients and methods The comparison with the   
REX shunt is based on a twenty year experience from 1993-2012 of 92 patients   
who underwent surgical shunting for portal hypertension with good liver   
function. The commonest causes of PHT were portal vein thrombosis and non   
cirrhotic portal fibrosis in 72/ 92 cases. GI bleeding in 66/92 patients was   
the commonest indication for shunting. Diagnosis of PHT in a patient with a   
GI bleed was based on the presence of splenomegaly on clinical examination   
and demonstrated esophageal varices on endoscopy. The abdominal ultrasound   
provided adequate information to plan surgery. The procedure performed was   
splenectomy with a Proximal Leino Renal shunt. No routine post shunt   
anticoagulation was used. No post shunt hepatic encephalopathy was   
encountered in this group. Shunt patency rate was 86% at one year follow up.   
Conclusion The simple management plan suggested may well be a better   
treatment option than the REX shunt.  
  
RECORD 404  
Percutaneous microwave ablation of hepatocellular carcinoma with   
high-powered, gas-cooled antennas: 24-month experience in 63 patients  
Ziemlewicz T. Hinshaw L. Lubner M. Brace C.L. Alexander M. Sampson L. Lee   
Jr. F.T.  
Journal of Vascular and Interventional Radiology (2013) 24:5 (759.e9). Date   
of Publication: May 2013  
  
Objectives: Microwave (MW) ablation is a promising technology that offers   
several advantages over radiofrequency (RF) ablation including: faster   
heating, higher (more lethal) tissue temperatures, improved consistency in   
different tissue types, and potentially greater ablation zone sizes. The   
purpose of this study was to retrospectively review the results in the first   
63 patients with hepatocellular carcinoma (HCC) treated with a high-power,   
gas-cooled MW device at a single center. Methods: Between December 2010 and   
December 2012 we treated 91 hepatocellular carcinomas in 63 patients via a   
percutaneous approach utilizing US and/or CT guidance. There were 54 male   
and 9 female patients with mean age of 61 years (range 44-83). All   
procedures were performed with a high-powered, gas-cooled microwave system   
(Certus 140, Neuwave Medical, Madison, WI) utilizing 1-3 (mean 1.7) 17-   
gauge antennas. Antenna power and ablation time was determined by the   
performing physician based on lesion size, location, and imaging findings.   
Mean power was 78 W (range 35-140 W) and mean ablation time was 5.7 minutes   
(range 1-15 minutes). 12 tumors in 10 patients were treated with   
chemoembolization at the time of or within 3 weeks preceding microwave   
ablation (7 patients with tumors >4 cm and 3 patients where the tumor(s)   
could not be identified by ultrasound). Follow-up imaging was performed   
immediately post-ablation and at 1, 3, 6, 9, and 12 months with   
contrast-enhanced CT or MRI. Results: Tumors ranged in size from 0.5 to 6.0   
cm (mean 2.5 cm) and median followup was 10 months. All treatments were   
considered technically successful with no evidence of residual tumor at   
immediate post-procedure CECT. Primary treatment effectiveness was 89.0%   
(80/91) for all tumors, 94.7% (74/79) for tumors < 4 cm, and 58.3% (7/12)   
for tumors > 4 cm. Primary treatment effectiveness for tumors > 3 cm and < 4   
cm was 100% (9/9). Secondary effectiveness via local regional therapy (LRT)   
was 98.8% (85/86), with one patient awaiting repeat microwave ablation. 5   
tumor progressions were excluded from secondary effectiveness analysis as   
they were noted only at explant pathology and therefore there was not   
opportunity for retreatment. Of the tumor progression in lesions <4 cm   
(n=5); 3 were treated with little or no margin due to compromised hepatic   
function or proximity to a critical structure and 2 were identified as only   
microscopic foci at explant pathology (by H&E staining, no viability   
staining was performed). A single minor complication occurred (1.6%), a main   
portal vein thrombus following ablation of a caudate lobe lesion which was   
noted at 1 month follow-up and resolved with anti-coagulation. There were no   
major complications. A patient died 8 days following the procedure secondary   
to a pneumonia for which he refused treatment. There was no procedure   
related mortality. Overall survival is 85.7% at median 10 month follow-up   
with deaths related to end stage liver disease (n=4), multifocal HCC/ESLD   
(n=3), transplant complications (n=1), or pneumonia (n=1). Conclusions: 24   
month experience treating hepatocellular carcinoma using a highpowered,   
gas-cooled microwave ablation system is safe with excellent local control.   
Prior RF studies have noted a substantial drop in efficacy when tumors   
exceed 3.0 cm in diameter. In this study local control of HCC by MW ablation   
was excellent in tumors up to 4.0 cm in size. Continued study is warranted   
to determine durability of treatment and survival with longer follow-up.  
  
RECORD 405  
Isolated superior mesenteric venous thrombophlebitis with acute appendicitis  
Karam M.M. Abdalla M.F. Bedair S.  
International Journal of Surgery Case Reports (2013) 4:4 (432-434). Date of   
Publication: 2013  
  
INTRODUCTION: Isolated superior mesentericveinous thrmbophlebitis is a   
rarely recognised condition associated with a high morbidity. It usually   
develops secondary to infection in the drainage area of the portal venous   
system, like appendix. PRESENTATION OF CASE: We report a case of neglected   
perforated acute appendicitis complicated by superior mesenteric venous   
pyelephlebitis patiant represented with a vague pain to right of umlicus,   
which is atypical this why cat scan was done and showed obstructed   
superiormesentric vein, portal vein was free with acute appendicitis.   
Appendicectomy and treatment with broad-spectrum antibiotics,   
anticoagulation, and platelets led to a full recovery. Follow-up imaging   
after one month revealed complete canalization of superior mesentric vein.   
DISCUSSION: Abdominal pain if atypical like our case report need imaging   
diagnosis. Modern diagnostic imaging techniques help the early diagnosis of   
acute phase pylephlebitis. CT can detect primary source of infection, extent   
of pylephlebitis, CT scan is the most reliable initially. Ultrasound scan   
with color flow Doppler is also a sensitive test for confirming partial   
patency of the portal vein and portal vein thrombosis accidentally   
discovered complete obliteration of superior mesenteric vein with thrombosis   
which remained not propagated by serial Doppler ultrasound of liver.   
Appropriate treatment should be initiated as soon as possible. To avoid   
extension to portal vein. The principal treatment for pylephlebitis is to   
remove the source of infection as appendicectomy. Anticoagulants must be   
used. Regarding the treatment of portal thrombosis, post operative use of   
heparin has been advocated. CONCLUSION: Cat scan play an important role in   
case of atypical abdominal pain. © 2013 Published by Elsevier Ltd on behalf   
of Surgical Associates Ltd.  
  
RECORD 406  
Predictors of response to anticoagulant therapy for the treatment of portal   
vein thrombosis (PVT) in cirrhosis patients  
Rodríguez-Castro K.I. Sartori M.T. Radu C.M. Gavasso S. Zerbinati P.   
Bortoluzzi I. Nadal E. Simioni P. Burra P. Senzolo M.  
Journal of Hepatology (2013) 58 SUPPL. 1 (S101). Date of Publication: April   
2013  
  
Background and Aims: anticoagulation has been demonstrated to be effective   
in the treatment of PVT; however, it is not known which factors predict the   
therapeutic response. The purpose of this study was to assess hemostatic   
status (pro- and anti-coagulant factors), and thrombus and patient   
characteristics as predictors of therapeutic efficacy of anticoagulation.   
Patients and Methods: 46 cirrhotics with PVT who received anticoagulation   
therapy with LMWH were retrospectively evaluated. Nadroparin 95 IU/Kg was   
administered to all patients (40% dose reduction if <50.000×109/L   
platelets). Interval between PVT onset and start of anticoagulation was   
estimated. All patients underwent thrombophilia screening and dosing of   
plasmatic proand anti-coagulation factors. Coagulation imbalance was further   
evaluated using the FactorVIII/Protein C ratio. Vessel recanalization was   
evaluated monthly using abdominal ultrasound and every 3 months by CT scan.   
Results: 34 patients were male and mean age was 58±11 years. Etiology of   
cirrhosis was viral in 47.8% and alcohol-related in 32.6% of cases. Partial   
PVT was found in 36/46 patients. Estimated interval from appearance of PVT   
and start of anticoagulation was ≤6 months in 35/46, and >6 months in the   
remaining 11 cases. Thrombophilic mutations were found in 4 patients.   
Recanalization of the portal vein was obtained in 30 patients (24 complete   
recanalization) after a mean time of 4.5±3.1 months of therapy. No   
correlation was found between standard coagulation parameters, plasmatic   
activity of factors VII, IX, XI, AT, PS, PC, fibrinogen, or factor VIII/PC   
ratio, and thrombus disappearance. Likewise, repermeation did not correlate   
with the extension of PVT, presence of thrombophilic mutations, severity of   
liver disease, or etiology of cirrhosis. An interval between development of   
PVT and start of anticoagulation therapy <6 months was the only significant   
predictor of anticoagulation efficacy (93% versus 15.2%, p < 0.001) with   
only 2 patients with older thrombus achieving recanalization after 6 months   
of therapy. Conclusions: The interval between PVT occurrence and start of   
anticoagulation therapy is the only predictor of recanalization; on the   
contrary, hemostatic imbalance does not correlate with anticoagulant   
response. For patients with recent thrombus, continuation of anticoagulant   
therapy beyond 6 months could increase the possibility of repermeation.  
  
RECORD 407  
Prophylactic enoxaparin in decompensated cirrhosis: A prevention of portal   
hypertension-related complications?  
Rudler M. Thabut D.  
Clinics and Research in Hepatology and Gastroenterology (2013) 37:2   
(115-116). Date of Publication: April 2013  
  
RECORD 408  
anticoagulation in patients with liver cirrhosis: Complication or   
therapeutic opportunity?  
Jairath V. Burroughs A.K.  
Gut (2013) 62:4 (479-482). Date of Publication: April 2013  
  
RECORD 409  
Vascular complications after orthotopic liver transplantation in Estonia  
Väli T. Tein A. Tiganik V. Ulst K.  
Transplantation Proceedings (2013) 45:3 (1201-1203). Date of Publication:   
April 2013  
  
The aim of this study was to analyse vascular complications (VC)   
accompanying the introduction in Estonia of orthotopic liver transplantation   
(OLT) for treatment of end-stage liver disease. We present the incidence and   
treatment of VC occurring among our first 23 OLT in 22 patients. The 11   
female and 11 male patients were aged 12 to 67 years. Their diagnoses were   
cholestatic disease (n = 8); hepatitis C virus (HCV) cirrhosis (n = 6);   
tumor (n = 3); Budd-Chiari syndrome (n = 2); autoimmune hepatitis (n = 1);   
cystic fibrosis (n = 1); or fulminant hepatic failure (n = 1). Only   
end-to-end vascular reconstructions were used in OLT. The patients' 1-year   
post-OLT survival rate was 86%. VC were confirmed using computed tomography   
(CT) or magnetic resonance imaging (MRI). In cases of VC, we started a   
1-week course of subcutaneous anticoagulant therapy with low-molecular   
weight heparin (LMWH) immediately followed by permanent oral treatment. The   
incidence of VC was 14% (n = 3). There was no hepatic artery thrombosis. One   
patient developed hepatic venous thrombosis at 3 weeks after   
retransplantation. She was treated successfully with immediate LMWH followed   
by a permanent oral anticoagulation. Two patients experienced portal vein   
complications: 1 with pre-OLT portal vein thrombosis developed right   
intrahepatic portal vein thrombosis at 5 weeks after OLT requiring portal   
thrombectomy. He was treated successfully with immediate LMWH followed by   
permanent oral anticoagulation. The other subject displayed left   
intrahepatic portal vein thrombosis at 1 week after OLT. Despite immediate   
LMWH treatment followed by a permanent oral anticoagulation, he required   
left lobe necrectomy and Roux-Y choledochojejunostomy for recovery. The   
survival and recovery of all studied patients with VC allow us to recommend   
immediate subcutaneous anticoagulant therapy for post-OLT portal or hepatic   
venous thrombosis. © 2013 Elsevier Inc.  
  
RECORD 410  
Portomesenteric thrombosis following laparoscopic bariatric surgery:   
Incidence, patterns of clinical presentation, and etiology in a bariatric   
patient population  
Goitein D. Matter I. Raziel A. Keidar A. Hazzan D. Rimon U. Sakran N.  
JAMA Surgery (2013) 148:4 (340-346). Date of Publication: April 2013  
  
Objective: To describe the incidence of, the patterns of clinical   
presentation of, and the reasons for portomesenteric vein thrombosis among   
patients who underwent laparoscopic bariatric surgery. Design:   
Retrospective, multicenter study. Setting: Six academic bariatric centers.   
Patients: Morbidly obese patients diagnosed with portomesenteric vein   
thrombosis following laparoscopic bariatric surgery between January 2007 and   
June 2012. Main Outcome Measures: Clinical presentation, diagnostic measures   
used, treatments employed, outcome, and hematologic workup of patients.   
Results: Of 5706 patients who underwent laparoscopic bariatric surgery, 17   
(0.3%) had portomesenteric vein thrombosis, 16 after sleeve gastrectomy and   
1 following adjustable gastric banding. Seven patients were women, the mean   
age was 38 years, and the mean body mass index was 44.3. The median time to   
presentation was 10.1 days, and the median time to diagnosis was 11.7 days.   
New-onset epigastric pain was present in all patients, whereas other signs   
and symptoms were sporadically found. Computed tomography was performed and   
was diagnostic in 16 cases. Ultrasonography was used for 9 patients, and   
positive results were found for 8 of these patients. Patients were treated   
by anticoagulation with subcutaneous low-molecular-weight heparin (n=15) or   
intravenous heparin (n=2), followed by warfarin sodium. One patient   
underwent transhepatic portal infusion of streptokinase. Three patients   
required surgery: laparoscopic splenectomy due to infarct and abscess for 1   
patient and laparotomy for 2 patients (with necrotic small-bowl resection   
for 1 of these patients). There were no deaths. Conclusions: Portomesenteric   
vein thrombosis is rare after laparoscopic bariatric surgery. Familiarity   
with this dangerous entity is important. Prompt diagnosis and care,   
initiated by a high index of suspicion, is crucial. © 2013 American Medical   
Association.  
  
RECORD 411  
Thrombolysis of portal vein thrombosis after splenectomy following liver   
transplantation  
Brown L. Abbass A.A. Nagai S. Patil V. Abouljoud M. Getzen T. Yoshida A.   
Kazimi M. Kim D.Y.  
Liver Transplantation (2013) 19:3 (346-348). Date of Publication: March 2013  
  
RECORD 412  
Anticoagulant therapy in patients with non-cirrhotic portal vein thrombosis:   
Effect on new thrombotic events and gastrointestinal bleeding  
Spaander M.C.W. Hoekstra J. Hansen B.E. Van Buuren H.R. Leebeek F.W.G.   
Janssen H.L.A.  
Journal of Thrombosis and Haemostasis (2013) 11:3 (452-459). Date of   
Publication: March 2013  
  
Background and aims: It remains unclear when anticoagulant therapy should be   
given in patients with non-cirrhotic portal vein thrombosis (PVT). The aim   
of this study was to assess the effect of anticoagulation on recurrent   
thrombotic events and gastrointestinal bleeding in non-cirrhotic PVT   
patients. Methods: Retrospective study of all patients with non-cirrhotic   
PVT (n = 120), seen at our hospital from 1985 to 2009. Data were collected   
by systematic chart review. Results: Sixty-six of the 120 patients were   
treated with anticoagulants. Twenty-two recurrent thrombotic events occurred   
in 19 patients. The overall thrombotic risk at 1, 5 and 10 years was 4%, 8%   
and 27%, respectively. Seventy-four percent of all recurrent thrombotic   
events occurred in patients with a prothrombotic disorder. Anticoagulant   
therapy tended to lower the risk of recurrent thrombosis (hazard ratio [HR]   
0.2, P = 0.1), yet the only significant predictor of recurrent thrombotic   
events was the presence of a prothrombotic disorder (HR 3.1, P = 0.03). In   
37 patients, 83 gastrointestinal bleeding events occurred. The re-bleeding   
risk at 1, 5 and 10 years was 19%, 46% and 49%, respectively.   
anticoagulation therapy (HR 2.0, P ≤ 0.01) was a significant predictor of   
(re)bleeding. anticoagulation therapy had no effect on the severity of   
gastrointestinal bleeding. Poor survival was associated with recurrent   
thrombotic events (HR 3.1 P = 0.02), whereas bleeding (HR 1.6 P = 0.2) and   
anticoagulant treatment (HR 0.5 P = 0.2) had no significant effect on   
survival. Conclusions: In non-cirrhotic PVT patients recurrent thrombotic   
events are mainly observed in patients with underlying prothrombotic   
disorders. anticoagulation therapy tends to prevent recurrent thrombosis but   
also significantly increases the risk of gastrointestinal bleeding. © 2013   
International Society on Thrombosis and Haemostasis.  
  
RECORD 413  
Management of Venous Thromboembolism  
Burnett B.  
Primary Care - Clinics in Office Practice (2013) 40:1 (73-90). Date of   
Publication: March 2013  
  
This article describes the risk factors, diagnostic tools, and therapeutic   
approaches for venous thromboembolism (VTE), which includes primarily deep   
vein thrombosis and pulmonary embolism, as well as VTE occurring at other   
sites. Outpatient management strategies are emphasized. © 2013 Elsevier Inc.  
  
RECORD 414  
Survival of patients with portal vein thrombosis: Analysis based on disease   
onset  
Khayyat Y.M.  
Hepato-Gastroenterology (2013) 60:122 (65-69). Date of Publication:   
March-April 2013  
  
Background/Aims: To identify prevalent causes and determine survival rates   
of patients with portal vein thrombosis (PVT) in the Western Saudi Arabia.   
Methodology: Retrospective chart review of patients diagnosed with chronic   
liver disease and portal vein thrombosis in two major cities in Western   
Saudi Arabia during the period 2000-2009. Results: Among 1349 patients   
screened, 109 patients met the inclusion criteria, 67 patients had acute PVT   
and 42 patients had chronic PVT. The relative risk of developing PVT is   
higher in males in whom acute PVT is 1.32 vs. 0.68 in females (95% CI:   
090-1.94). Mean survival age of acute and chronic PVT (Kaplan-Meier) is 5.61   
years (95% CI: 4.52-6.70). Univariate and multivariate regression analysis   
identified several variables in which international normalized ratio (INR)   
level on presentation was found to be a significant variable in survival.   
Conclusions: Mean survival for acute or chronic PVT is about five years. The   
first determination of INR level is a useful predictor of survival. © H.G.E.   
Update Medical Publishing S.A.  
  
RECORD 415  
Clinical outcome of partial portal vein thrombosis in cirrhotic patients: To   
observe or to treat?  
Caracciolo G. Garcovich M. Zocco M.A. Ainora M.E. Roccarina D. Annicchiarico   
B.E. Ponziani F.R. Siciliano M. Gasbarrini A.  
Digestive and Liver Disease (2013) 45 SUPPL. 2 (S171). Date of Publication:   
March 2013  
  
Background and aim: Recently, safety and efficacy of anticoagulation therapy   
(AT) in cirrhotic patients with complete PVT have been shown, but little is   
known about long-term outcome and resolution of partial PVT (pPVT) with or   
without therapeutic intervention. The aim of this study was to compare   
cirrhotic patients with pPVT undergoing either therapy with low molecular   
weight heparin (LMWH) or only clinical observation. Material and methods:We   
retrospectively reviewed data on cirrhotic patients with pPVT followed in   
our Unit and selected two cohorts of patients well matched for clinical and   
demographic characteristics: patients treated with LMWH (group A) and   
patients who didn't receive AT (group B). Exclusion criteria were advanced   
liver cirrhosis (Child-Pugh C), liver transplantation during follow-up,   
cavernomatous transformation of PVT, presence of neoplasms and active   
variceal bleeding or high-risk esophageal varices. Imaging of PVT with   
Doppler ultrasound or spiral CT/MRI was evaluated at baseline and 6-12   
months after inclusion. Thrombosis was considered partial when involving   
<50% of the vessel with or without extension in the superior mesenteric   
vein; response was defined as complete or almost complete recanalization.   
Results: A total of 52 cirrhotic patients with pPVT were evaluated in order   
to select 12 patients with pPVT receiving LMWH for 3-6 months and 15   
patients with pPVT who didn't receive AT. LMWH therapy was administered for   
3-6 months or until resolution of thrombosis, with no major side effects   
such as uncontrolled bleeding reported. Complete portal recanalization   
occurred in 8 out 12 patients in group A and in 8 out of 14 patients in   
group B (66% vs 57% complete resolution; p=0.61), suggesting no clear   
advantage for AT. Conclusions: As cirrhosis is characterized by a complex   
haemostasis defect including primary haemostasis, coagulation and   
fibrinolysis, clinical outcome of PVT may not always be easily predictable,   
especially in patients with partial or minimal PVT.  
  
RECORD 416  
portal vein thrombosis in cirrhosis: Ignore, prevent, or treat?  
Senzolo M. Caldwell S.  
Gastroenterology (2013) 144:2 (e19-e20). Date of Publication: Feb 2013  
  
RECORD 417  
portal vein thrombosis secondary to hepatopancreaticobiliary malignancy: An   
assessment of incidence, risk factors, and clinical management  
Dunki-Jacobs E.M. Priddy E.E. Philips P. Egger M.E. Scoggins C.R. Callender   
G.G. McMasters K.M. Martin R.C.  
HPB (2013) 15 SUPPL. 1 (20). Date of Publication: February 2013  
  
Introduction: portal vein thrombosis (PVT) has not been well described in   
the setting of hepato-pancreatico-biliary (HPB) malignancy. The aim of this   
study is to assess the incidence and risk factors of PVT secondary to HPB   
malignancy and to evaluate the effectiveness of systemic anticoagulation.   
Methods: All patients with a diagnosis of an HBP neoplasm from January 2009   
and December 2011 were evaluated using a prospective database. Patients with   
a coexisting diagnosis of PVT were matched in a 1 : 1 ratio with controls   
from the same database. Data collected included tumor location (pancreas vs   
hepatic), stage, methods of clinical management, timing of PVT and clinical   
management. Results: 1072 patients with HPB malignancies (630 pancreas and   
442 intrahepatic) were evaluated. Tumor location and cancer stage were the   
only predictors of PVT (p = 0.049 and p = 0.042, respectively). Surgical   
resection, RFA ablation, chemoradiation therapy, and hepatic arterial   
therapy (HAT) did not potentiate PVT (Table 1). Eleven of 41 patients (27%)   
were treated with anticoagulation for the management of their PVT. Portal   
vein recanalization was not achieved in any patient and thrombus progression   
was seen in 64% of patients despite therapeutic anticoagulation. Conclusion:   
Hepatic malignancies are more likely to present with PVT whereas pancreatic   
malignancies are more likely to develop PVT as a sign of progression to   
stage IV disease. The mechanism of PVT secondary to HPB malignancy appears   
to be different than PVT of benign conditions and is not amendable to   
systemic anticoagulation. (Table Presented).  
  
RECORD 418  
Predictors of response to anticoagulant therapy for the treatment of portal   
vein thrombosis (PVT) in cirrhosis patients  
Rodriguez K.I. Sartori M.T. Radu C. Gavasso S. Zerbinati P. Bortoluzzi I.   
Nadal E. Simioni P. Burra P. Senzolo M.  
Digestive and Liver Disease (2013) 45 SUPPL. 1 (S41). Date of Publication:   
February 2013  
  
Introduction: It is not known which factors predict efficacy of   
anticoagulation in the treatment of PVT. Aim: To assess hemostatic status   
(pro- and anti-coagulant factors), and thrombus and patient characteristics   
as predictors of therapeutic efficacy of anticoagulation. Materials and   
methods: 46 cirrhotics with PVT who were anticoagulated with low molecular   
weight heparin (LMWH) were retrospectively evaluated. Nadroparin 95 IU/kg   
was administered to all patients (40% dose reduction if <50,000×109/L   
platelets). Interval between PVT onset and start of anticoagulation was   
estimated. All patients underwent thrombophilia screening and dosing of   
plasmatic pro-and anti-coagulation factors. Coagulation imbalance was   
further evaluated using the FactorVIII/Protein C ratio. Vessel   
recanalization was evaluated monthly using abdominal ultrasound and every 3   
months by CT scan. Results: 34 patients were male and mean age was 58±11   
years. Etiology of cirrhosis was viral in 47.8% and alcohol-related in 32.6%   
of cases. Partial PVT was found in 36/46 patients. Estimated interval from   
appearance of PVT and start of anticoagulation was £6 months in 35/46.   
Thrombophilic mutations were found in 4 patients. Recanalization was   
obtained in 30 patients (24 complete recanalization) after a mean time of   
4.5±3.1 months of therapy. No correlation was found between standard   
coagulation parameters, plasmatic activity of factors VII, IX, XI, AT, PS,   
PC, fibrinogen, or FVIII/PC ratio, and thrombus disappearance. Likewise,   
recanalization did not correlate with the extension of PVT, presence of   
thrombophilic mutations, severity or etiology of liver disease. An interval   
between development of PVT and start of anticoagulation therapy <6 months   
was the only significant predictor of anticoagulation efficacy (93% versus   
15.2%, p<0.001) Conclusions: the interval between PVT onset and start of   
anticoagulation is the only predictor of recanalization. Hemostatic   
imbalance does not correlate with anticoagulant response. For patients with   
recent thrombus, continuation of anticoagulant therapy beyond 6 months could   
increase the possibility of recanalization.  
  
RECORD 419  
portal vein thrombosis: Should anticoagulation be used?  
Congly S.E. Lee S.S.  
Current Gastroenterology Reports (2013) 15:2 Article Number: 306. Date of   
Publication: February 2013  
  
portal vein thrombosis (PVT) can contribute to significant morbidity and   
mortality; in patients with cirrhosis, this can make transplant more   
technically challenging. Additionally, the clot may extend further into the   
mesenteric and splenic veins, and disturbance of the hepatic blood flow may   
lead to faster progression of the cirrhosis. Development of PVT is   
associated with local risk factors, and many patients have associated   
systemic prothrombotic factors. anticoagulation in noncirrhotic patients   
should be initiated at diagnosis, using low-molecular-weight heparin   
overlapping with vitamin K antagonists. Cirrhotic patients with PVT should   
be screened for varices and then anticoagulated with low-molecularweight   
heparin for at least a 6-month period. All patients should be assessed for   
triggering factors and tumors, as well as systemic prothrombotic factors.   
Newer evidence suggests that prophylactic anticoagulation in patients with   
cirrhosis may have a role in clinical management with decreased incidence of   
PVT and improved survival; further study is needed. © 2013 Springer   
Science+Business Media New York.  
  
RECORD 420  
Feasibility of anticoagulation in patients of Budd-Chiari syndrome with   
gastroesophageal varices and portal hypertension  
Dabbous H. Sakr M. Abdelhakam S. Youssef S. Gharib M. Shaker M. Eldorry A.  
Journal of Gastroenterology and Hepatology Research (2013) 2:5 (581-584).   
Date of Publication: 2013  
  
AIM: Budd-Chiari syndrome (BCS) is characterized by hepatic venous outflow   
obstruction. Patients with BCS are found to have oesophageal varices (OV),   
gastric varices (GV) as well as portal hypertensive gastropathy (PHG).   
anticoagulation is recommended in BCS though not evaluated in randomized   
trials. The aim of work is to determine feasibility of anticoagulation in   
patients with BCS with gastro-esophageal varices. METHODS: 150 patients with   
BCS were included. All had upper endoscopy. Band ligation was planned for   
(OV) with red signs or recent bleeding and cyanoacrylate injection for   
bleeding (GV) or signs of impending hemorrhage before anticoagulation.   
RESULTS: 30 patients (20%) were presented by GI bleeding, 12/30(40%) had   
large sized OV with (GV) in 3 of them, all had band ligation with   
cyanoacrylate injection of the (GV) before anticoagulation. The remaining 18   
had PHG with medium sized non risky varices in 12/30 (40%) and small OV in   
6/30 (20%). Only 2/30 (6.6%) had GI bleeding after anticoagulation with   
overall survival of 10.4 months. Among 120 patients who were not presented   
by GI bleeding, 18/120 (15%) had large OV, 57/120 (47.5%) had PHG with small   
or medium sized OV, one patient had isolated large (GV), and 44 patients had   
neither OV nor PHG. All were anticoagulated after band ligation of risky   
varices. 23 (19%) out of 120 died, only 3 (2.5%) due to GI bleeding with   
overall survival of 12.3 months. CONCLUSIONS: anticoagulation in BCS is   
feasible after band ligation of large or medium sized OV with red signs.   
History of GI bleeding should not be a contraindication for anticoagulation   
in BCS. © 2013 ACT.  
  
RECORD 421  
Diagnosis and endovascular treatment of common vascular complications in the   
post liver transplant patient  
Lancaster M. Rosenkrantz J. Salsamendi J. Pereira K.  
Journal of Vascular and Interventional Radiology (2013) 24:1   
(145.e4-145.e5). Date of Publication: January 2013  
  
Purpose: To describe and review the diagnosis and endovascular treatment of   
common vascular complications of liver transplant. Materials and Methods: 1)   
Review of the literature regarding vascular complications of liver   
transplant, with particular attention to those complications commonly   
diagnosed and treated by the Interventional Radiologist. 2) Discussion of a   
case example of one such complication, portal vein stenosis, recently   
treated by balloon dilatation and stenting at our institution. 3) Review and   
discussion of other case examples from the literature or our institutional   
experience, pertaining to other vascular complications. Results: Vascular   
complications after liver transplant are not infrequently encountered. These   
include hepatic artery thrombosis and stenosis, as well as stenosis of the   
portal vein, hepatic veins, and IVC. portal vein thrombosis can also be   
seen. With the exception of hepatic artery thrombosis, these are often   
treated with balloon dilatation and/or stenting with satisfactory results.   
In the case of hepatic artery thrombosis, selective thrombolytic injection   
can be employed in the affected branch, although stenting and/or balloon   
dilatation may still be required for a concomitant hepatic artery stenosis.   
In addition, combinations of anti-platelet therapy and anticoagulation are   
often utilized in conjunction with stent placement. As an example of one   
such complication, we discuss a relatively uncommon case of portal vein   
stenosis. Incidentally, the stenosis was of such severity that it caused   
post stenotic aneurysmal dilatation of more proximal portion, which has not   
been previously reported to our knowledge. Portal venous stenoses are   
relatively uncommon, being reported in approximately 3% of cases. There are   
various post treatment medical management related issues to address as well,   
such as the type and duration of anticoagulation to use and when the patient   
should return for routine follow-up. Conclusion: In conclusion, we review   
the common vascular complications that can be seen after liver transplant,   
using a case of portal vein stenosis and associated post stenotic dilatation   
of the portal vein as an example. Generally speaking, they are relatively   
infrequent, occurring in as many as 5% of patients. Yet, a working   
familiarity with these complications and expected subsequent management is   
useful, particularly at smaller centers or community hospitals where there   
is no transplant program. These important considerations are discussed and   
addressed in this concise review of the topic.  
  
RECORD 422  
Impact of anticoagulation on outcomes in acute non-cirrhotic and   
non-malignant portal vein thrombosis: A retrospective observational study  
Hall T.C. Garcea G. Metcalfe M. Bilk D. Rajesh A. Dennison A.  
Hepato-Gastroenterology (2013) 60:121 (311-317). Date of Publication:   
January-February 2013  
  
Background/Aims: No definitive evidence exists regarding the treatment of   
acute portal vein thrombosis (PVT). Treatment modalities that have been   
employed and investigated include conservative management, anticoagulation,   
thrombolysis and thrombectomy. This observational study examines the impact   
of anticoagulation on PVT. Methodology: The electronic radiology database   
was searched with keywords 'portal vein' and 'thrombosis'. Relevant patient   
notes and imaging were reviewed to collect data from those with acute PVT.   
The primary end point was portal vein recanalisation. Secondary outcome   
measures were morbidity and the development of portal hypertension and its   
sequelae (including variceal bleeding). Data from patients with PVT in the   
context of cirrhosis, malignancy or liver transplant were excluded. Results:   
Partial or complete recanalization of the portal vein occurred in 81.8% of   
anticoagulated patients and 37.5% of the non-treatment group. Five patients   
died, 1 following an intracranial haemorrhage whilst anticoagulated and   
another who was not treated and developed secondary small bowel ischaemia   
and peritonitis. The remaining 3 died from their underlying pathology. Late   
complications, such as varices and ascites occurred more frequently in the   
patients in whom the portal vein failed to recanalize (83.3% vs. 27.3%).   
Conclusions: Spontaneous resolution of acute portal vein thrombosis is   
uncommon. Early anticoagulation results in a higher rate of recanalisation   
with minimal associated morbidity when compared with no treatment. © H.G.E.   
Update Medical Publishing S.A.  
  
RECORD 423  
The management of mesenteric vein thrombosis: A single institution's   
experience  
Yanar F. Aǧcaoǧlu O. Gök A.F.K. Sarici I.S. Özçinar B. Aksakal N. Aksoy M.   
Özkurt E. Kurtoǧlu M.  
Ulusal Travma ve Acil Cerrahi Dergisi (2013) 19:3 (223-228). Date of   
Publication: 2013  
  
BACKGROUND Mesenteric vein thrombosis occurs rarely and is responsible for   
approximately 5-15% of all cases of acute mesenteric ischemia. The aim of   
this report was to discuss the management of mesenteric vein thrombosis   
based on our experience with 34 patients. METHODS In the present study, 34   
patients who were admitted to our emergency surgery department between   
January 2007 and January 2010 with a diagnosis of acute mesenteric vein   
thrombosis were assessed retrospectively. Patients with peritoneal signs   
first underwent diagnostic laparoscopy to rule out perforation or bowel   
gangrene. We performed a second-look laparoscopy within 72 hours of the   
first operation. All patients were administered 100 mg/kg of the   
anticoagulant enoxaparin twice daily. In the 6th and 12th months of follow   
up, CT angiography was performed to evaluate recanalization of the veins.   
RESULTS CT angiography revealed superior mesenteric vein thrombosis in 25   
(73%) patients, portal vein thrombosis in 24 (70%) patients, and splenic   
vein thrombosis in 12 (35%) patients. Eleven patients with peritoneal signs   
underwent diagnostic laparoscopy; eight of the patients underwent small   
bowel resection, anastomosis, and trocar insertion. During second-look   
laparoscopy, small bowel ischemia was found in two patients and re-resection   
was performed. CONCLUSION Early diagnosis with CT angiography, surgical and   
nonsurgical blood flow restoration, proper anticoagulation, and supportive   
intensive care are the cornerstones of successful treatment of mesenteric   
vein thrombosis.  
  
RECORD 424  
anticoagulation prevents portal vein thrombosis and decompensation in   
patients with cirrhosis  
Pariente A.  
Hepato-Gastro and Oncologie Digestive (2013) 20:1 (62-65). Date of   
Publication: 1 Jan 2013  
  
RECORD 425  
anticoagulation prevents portal vein thrombosis and decompensation in   
patients with cirrhosis  
Pariente A.  
Hepato-Gastro (2013) 20:1 (62-65). Date of Publication: January 2013  
  
RECORD 426  
Rex shunt for portal vein thrombosis after adult living donor liver   
transplantation.  
Soejima Y. Shirabe K. Yoshizumi T. Uchiyama H. Ikegami T. Yamashita Y. Ikeda   
T. Kawanaka H. Sugimachi K. Mimori K. Watanabe M. Morita M. Oki E. Saeki H.   
Maehara Y.  
Fukuoka igaku zasshi = Hukuoka acta medica (2013) 104:11 (464-468). Date of   
Publication: Nov 2013  
  
portal vein thrombosis (PVT) after liver transplantation is a relatively   
common but serious complication which could lead to portal hypertension or a   
direct graft loss. A "Rex" shunt created between the superior mesenteric   
vein (SMV) and the umbilical portion of the left portal vein can be a useful   
option to treat PVT after pediatric liver transplantation, however, its   
application to adult patients has not been reported so far because   
appropriate vein grafts are hardly available. Herein we present a case of   
PVT after left lobe living donor liver transplantation (LDLT) who underwent   
the procedure using the own inferior jugular vein and the gonadal vein as a   
shunt graft. The shunt was patent immediately after the procedure but was   
thrombosed 2 days after probably due to the insufficient inflow from the SMV   
and the absence of anticoagulation therapy, for which emergent thrombectomy   
and ligation of the significant hepatofugal collateral veins followed by   
full anti-coagulation therapy were performed. The shunt remains open at 8   
month after the procedure with a normal anmonia level and liver function. In   
conclusion, the Rex shunt using recipient's autologous vein grafts is a   
feasible and valuable option for adult patients to treat PVT after LDLT.  
  
RECORD 427  
Q: Is anticoagulation appropriate for all patients with portal vein   
thrombosis?  
Confer B.D. Hanouneh I. Gomes M. Chadi Alraies M.  
Cleveland Clinic Journal of Medicine (2013) 80:10 (611-613). Date of   
Publication: 2013  
  
RECORD 428  
Venous thromboembolism in cirrhosis: A review of the literature  
Buresi M. Hull R. Coffin C.S.  
Canadian Journal of Gastroenterology (2012) 26:12 (905-908). Date of   
Publication: December 2012  
  
Although hemorrhage has traditionally been regarded as the most significant   
hemostatic complication of liver disease, there is increasing recognition   
that hypercoagulability is a prominent aspect of cirrhosis. Identifying   
markers of coagulability and monitoring anticoagulation therapy in the   
setting of cirrhosis is problematic. The bleeding risk of venous   
thromboembolism (VTE) prophylaxis and treatment in patients with chronic   
liver disease is unclear and there are currently no recommendations to guide   
practice in this regard. In the present report, the mechanism of coagulation   
disturbance in chronic liver disease is reviewed with an examination of the   
evidence for an increased VTE risk in cirrhosis. Finally, the available   
evidence is assessed for prophylaxis and therapy of VTE in chronic liver   
disease, and the role it may play in decreasing clinical decompensation and   
improving survival. ©2012 Pulsus Group Inc. All rights reserved.  
  
RECORD 429  
Non-cirrhotic, non-malignant acute idiopathic portal vein thrombosis leading   
to consumptive thrombocytopenia and massive upper gastrointestinal bleed  
Tirmizi A.  
Critical Care Medicine (2012) 40:12 SUPPL. 1 (319). Date of Publication:   
December 2012  
  
Case Reports: A 71 years old Caucasian female was transferred to intensive   
care unit with acute abdominal pain and hematemesis. Patient was admitted to   
medical floor few days ago with the diagnosis of acute idiopathic portal   
vein thrombosis and thrombocytopenia with platelet count of 35,000.   
Extensive evaluation did not reveal any specific etiology of   
thrombocytopenia and hence was diagnosed as immune (idiopathic)   
thrombocytopenic purpura (ITP). Prior to her hematemesis, patient was on   
intravenous argatroban and oral warfarin for idiopathic portal vein   
thrombosis and on intravenous solumedrol for possible ITP. Emergent CT of   
the abdomen and pelvis revealed further extension of portal vein thrombus   
into the distal left splenic and superior mesenteric veins with no evidence   
of splenomegaly or ascites. INR was 2.5 and platelet count 75,000.   
Hematocrit dropped from 35.2 to 20.6. All the medications were discontinued   
and intravenous pantoprazole and octreotide were started. EGD revealed   
multiple fundic varices and bleeding. Later that day patient developed   
respiratory failure from fluid overload due to administration of multiple   
blood transfusions. Patient required intubation and mechanical ventilation   
but was successfully weaned off after three days. There were no further   
episodes of variceal bleeding and hematocrit stabilized at 30 and platelet   
count at 100,000. Patient was subsequently discharged home. Recanalization   
of portal and superior mesenteric veins was demonstrated on repeat abdominal   
CT in seven weeks. Platelet count was normal at 219,000 suggesting   
consumptive thrombocytopenia during patient's hospitalization due to acute   
thrombosis. This is the first reported case of non-cirrhotic, non-malignant   
acute idiopathic portal vein thrombosis occurring simultaneously with   
consumptive thrombocytopenia. Aggressive management of the consequent life   
threatening variceal bleed in the ICU led to a favorable outcome. In   
conclusion, acute idiopathic portal vein thrombosis is easily treatable with   
anticoagulation in patients without cirrhosis or malignancy but management   
becomes extremely challenging when there is associated variceal bleeding and   
consumptive thrombocytopenia.  
  
RECORD 430  
TFPI resistance related to inherited or acquired protein S deficiency  
Tardy-Poncet B. Piot M. Brunet D. Chapelle C. Bonardel M. Mismetti P.   
Morange P. Tardy B.  
Thrombosis Research (2012) 130:6 (925-928). Date of Publication: December   
2012  
  
Background: Protein S (PS) is an essential component of the protein C   
pathway and PS deficiency can explain a poor response to activated protein   
C. It has recently been shown that PS also acts as a cofactor of Tissue   
Factor Pathway Inhibitor (TFPI). Objectives: In the present study, we   
investigated whether PS deficiency could be responsible for a poor response   
to TFPI. Patients/Methods: Thirty-one patients with inherited PS deficiency,   
seven pregnant women and 36 controls were enrolled in the study. We measured   
the plasma response to added TFPI using a two-step diluted prothrombin time   
(dPT) assay. The response of the different plasmas to the anticoagulant   
activity of TFPI was expressed as TFPI Normalised Ratio (TFPI NR). Results:   
The median TFPI NR was statistically significantly lower in patients with   
inherited PS deficiency (0.5) than in controls (1.0) (p < 0.0001). It was   
statistically significantly lower in patients with type I inherited PS   
deficiency (0.47) compared to patients with type III inherited PS deficiency   
(0.58) (p = 0.018). In contrast, it did not differ between patients with and   
without thrombosis. Median TFPI NR values were statistically significantly   
lower during pregnancy (0.54) than 3 months after delivery (0.71) (p =   
0.016). TFPI NR values correlated well with PS activity values (R(2) =   
0.681) whatever the nature of the PS deficiency. Conclusions: Our findings   
confirm that PS deficiency results in a poor anticoagulant response to TFPI,   
demonstrating again the cofactor role of PS in TFPI activity. © 2012   
Elsevier Ltd. All rights reserved.  
  
RECORD 431  
Immediate use of an arteriovenous prosthetic graft for life-saving dialysis   
in a child  
Grimaldi C. Crocoli A. De Galasso L. Picca S. Natali G.L. De Ville De Goyet   
J.  
Pediatric Nephrology (2012) 27:12 (2311-2313). Date of Publication: December   
2012  
  
Background: Autologous arteriovenous fistulas (AVFs) are the current gold   
standard for vascular access in hemodialysis (HD). However, in pediatric   
patients, specific clinical settings may contraindicate the procedure, thus   
mandating the use of a prosthetic graft (PG). Case-Diagnosis/Treatment: We   
report a case of successful polycarbonate urethane graft implantation and   
subsequent resumption of HD 12 h after the procedure in a young girl with   
end-stage renal disease (ESRD), challenging vascular anatomy and the absence   
of vascular access. Conclusions: The use of polycarbonate urethane PGs in   
children with ESRD and difficult vascular accesses may represent a valid   
alternative for early resumption of HD. © 2012 IPNA.  
  
RECORD 432  
Acute portal vein thrombosis complicating in vitro fertilization  
Mmbaga N. Torrealday S. McCarthy S. Rackow B.W.  
Fertility and Sterility (2012) 98:6 (1470-1473). Date of Publication:   
December 2012  
  
Objective: To describe a case of acute portal vein thrombosis after IVF   
treatment. Design: Case report. Setting: University teaching hospital.   
Patient(s): A 39-year-old woman experienced worsening, right upper quadrant   
pain several days after oocyte retrieval; ET was withheld. Imaging studies   
revealed acute portal vein thrombosis with extension into the splenic and   
superior mesenteric veins. Intervention(s): Therapeutic anticoagulation; no   
ET was performed. Main Outcome Measure(s): Improvement in symptoms, accurate   
diagnosis of condition. Result(s): Decreased size of portal vein thrombosis   
and partial vessel recanalization. Conclusion(s): Thromboembolic events are   
a rare complication of assisted reproductive technology (ART). In women who   
present with upper abdominal pain during ART, portal vein thrombosis should   
be considered in the differential diagnosis. © 2012 by American Society for   
Reproductive Medicine.  
  
RECORD 433  
portal vein thrombosis as a late-diagnosed, rare cause of bowel infarction:   
A case report  
Budzynski J. Wisniewska J. Pulkowski G.  
Acta Angiologica (2012) 18:4 (183-188). Date of Publication: 2012  
  
This case report presents a female patient in the puerperal period with   
abdominal pain recurring for 10 days in whom bowel infarction occurred. In a   
computerized tomography angiography (CTA) made after bowel resection,   
superior mesenteric artery (SMA) occlusion was diagnosed. The patient was   
referred for SMA stenting. However, analysis of the clinical course of the   
disease and ultrasonographic examination suggested the possibility of portal   
and superior mesenteric vein (SMV) thrombosis as a cause of bowel   
infarction. This was confirmed in the second CTA. Additionally, signs of   
portal hypertension in CTA and panendoscopy were diagnosed. anticoagulation   
with warfarin for thrombosis and carvedilolum for portal hypertension   
reduction were recommended. Sclerotherapy was performed three times with   
histoacryl and polidocanol due to the progression of the gastric fundal   
varices. A diagnostic examination made in order to determine the cause of   
the thrombotic process showed only a mutation in the heterozygous   
tetrahydrofolate reductase (MTHFR) gene. Diagnostic difficulties were   
described, and practical suggestions for the diagnosis of the basic disorder   
and its complications were made. Rationales for therapy were discussed.   
Copyright © 2012 Via Medica.  
  
RECORD 434  
Pregnancy in women with portal vein thrombosis: Results of a multicentric   
European study on maternal and fetal management and outcome  
Hoekstra J. Seijo S. Rautou P.E. Ducarme G. Boudaoud L. Luton D.   
Alijotas-Reig J. Casellas-Caro M. Condat B. Bresser E. Thabut D. Larroque B.   
Gárcia-Pagán J.C. Janssen H.L.A. Valla D.C. Plessier A.  
Journal of Hepatology (2012) 57:6 (1214-1219). Date of Publication: December   
2012  
  
Background & Aims: Women of childbearing age account for approximately 25%   
of patients with non-cirrhotic portal vein thrombosis (PVT). We aimed at   
assessing maternal and fetal outcome in pregnant women with known PVT.   
Methods: We performed a retrospective analysis of the files of women with   
chronic PVT in three European referral centers between 1986 and 2010.   
Results: Forty-five pregnancies, 28 (62%) treated with low molecular weight   
heparin, occurred in 24 women. Nine (20%) were lost before gestation week   
20. Preterm birth occurred in 38% of deliveries: there were 3 births at week   
24-25, 7 at week 32-36, and 26 after week 37. A term birth with a healthy   
infant occurred in 58% of pregnancies. Cesarean section was used in 53% of   
deliveries. Two women developed HELLP syndrome. A favorable outcome happened   
in 64% of pregnancies. Pregnancies with an unfavorable outcome were   
associated with a higher platelet count at diagnosis. Bleeding from   
esophageal varices occurred in 3 patients during pregnancy, all without   
adequate primary prophylaxis. Genital or parietal bleeding occurred   
postpartum in 4 patients, only one being on anticoagulation therapy.   
Thrombotic events occurred in 2 patients, none related to lower limbs or   
mesenteric veins. There were no maternal deaths. Conclusions: In pregnant   
PVT patients treated with anticoagulation on an individual basis, the rate   
of miscarriage and preterm birth appears to be increased. However, fetal and   
maternal outcomes are favorable for most pregnancies reaching gestation week   
20. High platelet counts appear to increase the risk for unfavorable   
outcome. Pregnancy should not be contraindicated in stable PVT patients. ©   
2012 European Association for the Study of the Liver. Published by Elsevier   
B.V. All rights reserved.  
  
RECORD 435  
Cerebral venous thrombosis and portal vein thrombosis associated with   
ulcerative colitis in a child: A case report  
Song S.M. Yum M.-S. Ko T.-S. Kim K.M.  
Journal of Gastroenterology and Hepatology (2012) 27 SUPPL. 5 (168). Date of   
Publication: December 2012  
  
Introduction: Patients with ulcerative colitis (UC) are known to have   
hypercoagulability and an increased risk of venous thromboembolism. The deep   
veins of the lower extremities and the pulmonary veins are the most common   
sites of thrombosis in UC. However, cerebral venous sinus thrombosis and   
portal vein thrombosis are very rare extra-intestinal complications of UC in   
children. We report the case of a child with newly diagnosed UC who   
developed both cerebral sinus thrombosis and portal vein thrombosis during   
an acute exacerbation of disease. Case report: A 12-year-old girl was   
referred to our hospital with a 1-month history of bloody diarrhea and   
abdominal pain and a 10-day history of headache, accompanied with nausea and   
vomiting. The patient had been diagnosed with UC in one week prior to her   
transfer. At that time she was placed on intravenous steroids and   
sulfasalazine. She continued to have a worsening headache, and 4 days after   
admission, she began to complain of right-sided progressive hemiparesis,   
numbness, and hemiparesthesia. An urgent magnetic resonance imaging   
angiograph showed superior sagittal sinus thrombosis and cortical vein   
thrombosis with associated cerebral edema in the left frontal area. Doppler   
ultrasonography and an abdominal CT scan revealed the portal vein   
thrombosis. These lesions were successfully treated with conventional   
management for UC and anticoagulation therapy. The patient was discharged   
without neurologic sequelae 21 days after admission. Conclusion: This is a   
rare case of cerebral venous thrombosis and portal vein thrombosis   
complicated by active UC, in which anticoagulation therapy was successful.   
Disease activity may play a major role in the occurrence of thrombosis.  
  
RECORD 436  
Tamoxifen-associated Budd-Chiari syndrome complicated by heparin-induced   
thrombocytopenia and thrombosis: A case report and literature review  
Chayanupatkul M. Rhee J.H. Kumar A.R. Varadi G.  
BMJ Case Reports (2012). Date of Publication: 2012  
  
We reported a rare case of Budd-Chiari syndrome (BCS) associated with   
tamoxifen use, which was later complicated by heparin-induced   
thrombocytopenia and thrombosis (HITT). The patient was a 44 year-old woman   
with a medical history of lobular carcinoma in situ, who had been on   
tamoxifen for 2 years, presented with abdominal pain and distention. Imaging   
studies followed by a liver biopsy confirmed the diagnosis of BCS. On   
extensive work-up, the patient was found to have an unclassified   
myeloproliferative disorder with positive JAK-2 V617 mutation. After   
discontinuing tamoxifen, the patient was started on intravenous heparin.   
However, later in the course, she developed HITT. Myeloproliferative   
disorder, in conjunction with tamoxifen, predisposed the patient to be   
highly thrombophilic resulting in BCS. HITT was found to be relatively   
common in BCS. anticoagulation and blood count need to be carefully   
monitored, and the possibility of HITT emergence in these patients should   
always be kept in mind. Copyright 2012 BMJ Publishing Group. All rights   
reserved.  
  
RECORD 437  
Gastrointestinal bleeding caused by extrahepatic arterioportal fistula   
associated with portal vein thrombosis  
Nie L. Luo X.-F. Li X.  
World Journal of Gastroenterology (2012) 18:44 (6501-6503). Date of   
Publication: 2012  
  
An extrahepatic arterioportal fistula (APF) involving the gastroduodenal   
artery and superior mesenteric vein is rare and mostly results from   
iatrogenic injuries. The clinical symptoms associated with APFs may include   
abdominal pain, gastrointestinal bleeding, ascites, nausea, vomiting,   
diarrhea, or even congestive heart failure. We present the case of a   
70-year-old man who presented with chronic abdominal pain and   
gastrointestinal bleeding secondary to APF and portal vein thrombosis. The   
endovascular embolization of APF was accomplished successfully, and symptoms   
of portal hypertension resolved immediately after intervention.   
Unfortunately, the patient did not respond well to anticoagulation therapy   
with warfarin. Therefore, the patient underwent implantation of a   
transjugular intrahepatic portosystemic shunt, and the complications of   
portal hypertension resolved. In conclusion, the embolization of APF is   
technically feasible and effective and can be considered the first-choice   
therapy in selected patients. © 2012 Baishideng. All rights reserved.  
  
RECORD 438  
The significance of antiphospholipid antibodies as a marker of thrombosis in   
patients after liver transplantation-a single center experience  
Furmańczyk A. Tronina O. Sadowska A. Ba¸czkowska T. Pacholczyk M. Chmura A.   
Durlik M.  
Transplantation (2012) 94 SUPPL. 10S (660). Date of Publication: 27 Sep 2012  
  
Introduction: Antiphospholipid antibodies (APLA) are the most common cause   
of acquired thrombophilia. After liver transplantation hepatic artery   
thrombosis (HAT) and portal vein thrombosis (PVT) frequently lead to graft   
failure and retransplantation. Objective: The aim of the study was to   
determine the relation between APLA and liver graft thrombosis. Patients and   
methods: The study included 33 Caucasian patients after liver   
transplantation (21 women/12 men, mean time after transplantation 33,9   
months) aged 22-74 years. Most patients (57,57%) were given   
steroids+CNI+MMF, 30% remained on steroids+CNI, 12% - CNI. The patients were   
divided into 2 groups: 25 patients with no clinical history of thrombosis   
T(-) and 8 patients T(+) with previous strong thrombotic events (thrombosis   
1): HAT or PVT in previous graft with retransplantation or idiopathic   
thrombocytopenia (Budd-Chiari syndrome) or deep vein thrombosis (3 episodes)   
in patients with autoimmune diseases. APLA consist of LA (lupus   
anticoagulant), ACL (anticardiolipin antibodies), anti-β2Glicoprotein I   
(anty-β2GPI), anti-prothrombin antibodies (anti-PT). APLA IgM and IgG were   
detected in serum twice in 6 months interval. Mean observational time was 14   
months. Results: APLA incidence in patients after liver transplantation is   
higher than in general population and ranges from 0-18,75%. In three T(+)   
patients an episode of thrombosis occurred during observation time   
(thrombosis 2). Two patients developed HAT, but only in one with AIH in   
native liver (retransplanted due to HAT complicated by biliary ischemia and   
hepatic abscess in the first graft, with recurrence of early HAT after   
retransplantation) ACL were detected in highly positive titer. In the second   
patient on aspirin with no APLA, late, partial HAT appeared during treatment   
with INF/RBV due to hepatitis C recurrence. The 3rd patient (AIH in native   
liver) developed splenic artery thrombosis despite treatment with VKA (INR   
2-3). No thrombotic events were observed in T(- ). There were statistically   
significant difference in ACL IgM between T(-) and T(+) in examination 1   
(p=0,0194) and in examination 2 (p=0,0090). In patients with thrombosis 2   
liver graft function remained above upper normal limits (ASP 40,7+/-8,1, ALT   
49,7+/-20). In the whole group liver graft function remained stable with no   
significant difference in ASP/ALT activity between T(-) and T(+).   
Discussion: ACL IgM and IgG, anty-β2GPI IgM and anty-PT IgG were detected in   
highly positive titers in both examinations, what confirms a constant   
production of APLA. Anti-β2GPI are representative for autoimmune diseases   
and their IgM concentration detected in liver recipients was also higher   
than in general population, but with no statistically significant difference   
between T(-) and T(+). As a potential causes of not detecting APLA in two   
T(+) patients with thrombosis 2, we consider immunosuppressive therapy,   
spontaneous APLA elimination from the circulation or thrombosis was due to   
surgical complication with no correlation with APLA. ACL was persistently   
present only in one T(+) patient. All thrombotic events occurred during   
anticoagulation, what indicate that the strength of this treatment was   
inadequate. Conclusion: Liver graft thrombosis has multifactorial etiology,   
but APLA detection may be useful tool to determine the additional thrombotic   
risk factor. A study designed on larger group is required.  
  
RECORD 439  
Antiphospholipid antibodies: An under-recognized cause of morbidity and   
mortality in patients transplanted for end stage liver disease  
Villamil A. Bandi J.C. Galdame O. Carballo G. De Santibañes E. Gadano A.  
Transplantation (2012) 94 SUPPL. 10S (223). Date of Publication: 27 Sep 2012  
  
Circulating antiphospholipid antibodies(aPL-ab) are often detected in   
patients with liver disease. Aim: To establish prevalence of aPL-ab in   
patients transplanted for chronic liver disease (OLT) and to assess their   
impact in the outcome of patients at 1 year post-OLT. Methods: Between Jan   
2006 and Dec 2010, 150 patients transplanted for chronic liver disease (88   
female, 62 male) were screened for aPLab. Anticardiolipin antibodies (IgG   
and IgM isotypes) were assayed by ELISA. Levels < 20 IU were considered   
normal. Plasma samples were evaluated for lupus anticoagulant activity (LA).   
Clinical and Dopplerultrasound evaluations were performed before OLT and at   
different timepoints post-OLT (weekly the first month and monthly   
thereafter). Results were compared with aPL-ab negative patients.   
Immunosuppressive regimen: cyclosporin or tacrolimus + mycophenolate-mofetil   
+ steroids. All patients received aspirin and/or low weight heparin   
post-OLT. Median follow-up: 26 months (12-56). Results: 39 /150 patients   
(24%) evidenced increased levels of aPL-ab pre-OLT. Etiology of liver   
disease was: HCV (n=12), PBC (n=7), alcohol (n=7), PSC (n=2), autoimmune   
(n=4), cryptogenic (n=3) and other (n=4). Child C patients had a lower   
prevalence of aPL-ab than Child B patients (21 vs 32 %, p NS). No difference   
was observed in renal or liver function tests, except for bilirrubin levels   
which were higher in aPL-ab + patients (5.9 vs 3,6 mg/dl, p=0.04). Seven   
thrombotic complications were observed in 6/36 aPL + patients post-OLT   
(humeral thrombosis, n=1, cerebrovascular ischemia n=3, hepatic artery   
thrombosis n=1, retinal thrombosis=1, intestinal ischemia n=1) resulting in   
one graft loss and one death, compared to nine thrombotic complications in   
8/114 patients aPL negative (cerebrovascular ischemia n=3, deep vein   
thrombosis n=2, hepatic artery thrombosis n=1, intestinal ischemia n=1,   
humeral thrombosis n=1, femoral thrombosis n=1) resulting in one graft loss   
and re-transplantation (p< 0.05). Five patients in the ApL+ group developed   
catastrophic antiphospholipid syndrome (CAPS) and 4/5 died in spite of early   
plasmapheresis, higher immunosuppression and anticoagulation. No differences   
were observed between both groups in infection rates, thrombocytopenia,   
acute cellular rejection or bleeding complications. 3/5 patients that   
developed CAPS presented a thrombotic event pre-OLT. 4:5 patients acute   
cellular rejection was a potentially triggering factor of aPL. No patient   
with aPL antibodies pre-OLT developed portal vein thrombosis on follow-up.   
Only one patient that was aPL negative pre-OLT presented “de novo”   
anticardiolipin antibodies post-OLT and developed an aPL associated vascular   
complication (cerebrovascular ischemia). Conclusion: Patients with end-stage   
liver disease have a high prevalence of aPL antibodies. The presence of aPL   
antibodies is associated with a higher risk of morbidity and mortality   
post-OLT Pre-OLT screening for anticardiolipin and lupus anticoagulant, and   
a high index of suspicion of ApL vascular complications post OLT is   
recommended to improve outcome.  
  
RECORD 440  
Antiphospholipid antibodies: An under-recognized cause of morbidity and   
mortality in patient's transplanted for end stage liver disease  
Villamil A. Bandi J.C. Galdame O. Carballo G. De Santibañes E. Gadano A.  
Transplantation (2012) 94 SUPPL. 10S (427). Date of Publication: 27 Sep 2012  
  
Circulating antiphospholipid antibodies(aPL-ab) are often detected in   
patients with liver disease. Aim: To establish prevalence of aPL-ab in   
patients transplanted for chronic liver disease (OLT) and to assess their   
impact in the outcome of patients at 1 year post-OLT. Methods: Between Jan   
2006 and Dec 2010, 150 patients transplanted for chronic liver disease (88   
female, 62 male) were screened for aPLab. Anticardiolipin antibodies (IgG   
and IgM isotypes) were assayed by ELISA. Levels < 20 IU were considered   
normal. Plasma samples were evaluated for lupus anticoagulant activity (LA).   
Clinical and Dopplerultrasound evaluations were performed before OLT and at   
different timepoints post-OLT (weekly the first month and monthly   
thereafter). Results were compared with aPL-ab negative patients.   
Immunosuppressive regimen: cyclosporin or tacrolimus + mycophenolate-mofetil   
+ steroids. All patients received aspirin and/or low weight heparin   
post-OLT. Median follow-up: 26 months (12-56). Results: 39 /150 patients   
(24%) evidenced increased levels of aPL-ab pre-OLT. Etiology of liver   
disease was: HCV (n=12), PBC (n=7), alcohol (n=7), PSC (n=2), autoimmune   
(n=4), cryptogenic (n=3) and other (n=4). Child C patients had a lower   
prevalence of aPL-ab than Child B patients (21 vs 32 %, p NS). No difference   
was observed in renal or liver function tests, except for bilirrubin levels   
which were higher in aPL-ab + patients (5.9 vs 3,6 mg/dl, p=0.04). Seven   
thrombotic complications were observed in 6/36 aPL + patients post-OLT   
(humeral thrombosis, n=1, cerebrovascular ischemia n=3, hepatic artery   
thrombosis n=1, retinal thrombosis=1, intestinal ischemia n=1) resulting in   
one graft loss and one death, compared to nine thrombotic complications in   
8/114 patients aPL negative (cerebrovascular ischemia n=3, deep vein   
thrombosis n=2, hepatic artery thrombosis n=1, intestinal ischemia n=1,   
humeral thrombosis n=1, femoral thrombosis n=1) resulting in one graft loss   
and re-transplantation (p< 0.05). Five patients in the ApL+ group developed   
catastrophic antiphospholipid syndrome (CAPS) and 4/5 died in spite of early   
plasmapheresis, higher immunosuppression and anticoagulation. No differences   
were observed between both groups in infection rates, thrombocytopenia,   
acute cellular rejection or bleeding complications. 3/5 patients that   
developed CAPS presented a thrombotic event pre-OLT. 4:5 patients acute   
cellular rejection was a potentially triggering factor of aPL. No patient   
with aPL antibodies pre-OLT developed portal vein thrombosis on follow-up.   
Only one patient that was aPL negative pre-OLT presented “de novo”   
anticardiolipin antibodies post-OLT and developed an aPL associated vascular   
complication (cerebrovascular ischemia). Conclusion: Patients with end-stage   
liver disease have a high prevalence of aPL antibodies. The presence of aPL   
antibodies is associated with a higher risk of morbidity and mortality   
post-OLT Pre-OLT screening for anticardiolipin and lupus anticoagulant, and   
a high index of suspicion of ApL vascular complications post OLT is   
recommended to improve outcome.  
  
RECORD 441  
Thrombosis in newborns: Experience from 31 cases  
Nosan G. Groselj-Grenc M. Paro-Panjan D.  
Signa Vitae (2012) 7:2 (29-32). Date of Publication: 2012  
  
Thrombosis is the result of congenital or acquired prothrombotic risk   
factors. The incidence of thrombosis in the paediatric population is highest   
in newborns, as about 10% of thrombotic events occur in the first four weeks   
of life. Haemostasis in a newborn, though still developing, is a well   
balanced mechanism. About 90% of all thrombotic events are due to acquired   
and the rest to congenital risk factors. The aim of our study was to   
estimate the incidence of thrombosis in a population of Slovenian newborns   
and to study risk factors, location and treatment of thrombotic events.   
Inpatient charts of newborns with thrombosis, admitted to a tertiary   
neonatology centre and paediatric intensive care unit between 2004 and 2011,   
were studied retrospectively. Family history, location, aetiology and   
treatment of thrombosis were analysed. Thirty one newborns, 17 boys (54.8%)   
and 14 girls (45.2%), with 31 thrombotic events were found. There were 17   
cases (54.8%) of arterial and 14 cases (45.2%) of venous thrombosis. A   
family history of thrombophilia was found in two cases (6.5%). Twenty six   
cases (83.9%) were contributed to acquired risk factors and five (16.1%) to   
congenital aetiology. Four cases (12.8%) were treated, two with   
anticoagulation, one with thrombolysis and one with both. The estimated   
incidence of thrombosis was 0.17 per 1000 live births. Our data showed a   
higher incidence of thrombosis in Slovenian newborns and a higher incidence   
of congenital prothrombotic risk factors than in the data published so far.  
  
RECORD 442  
Treatment irrespective of symptoms is a major predictive factor for thrombus   
resolution in patients with portal vein thrombosis  
Yu Y.-B. Yang C.-W. Liu C.-Y. Gau J.-P. Hong Y.-C. Hsiao L.-T. Liu J.-H.   
Chiou T.-J. Hsu H.-C. Tzeng C.-H.  
Blood (2012) 120:21. Date of Publication: 16 Nov 2012  
  
Background: portal vein thrombosis (PVT) typically presents with abdominal   
pain, ascites, and splenomegaly, but it is frequently asymptomatic. Current   
guidelines recommend that anticoagulation be used for symptomatic patients   
with PVT. However, it remains controversial regarding the benefits of   
treatments for asymptomatic patients with PVT. Methods: We retrospectively   
enrolled 933 patients with suspicious PVT in the Taipei Veterans General   
Hospital from January 2002 to December 2011. At total of 93 patients were   
confirmed using either doppler sonography, computed tomography, or magnetic   
resonance imaging. Response to treatment was defined as the recanalization   
or cavernous formation of portal veins. Logistic regression was used to   
investigate the clinic-laboratory parameters that were predictive for the   
resolution of PVT. Results: Among the 93 patients, the median age was 63   
years old (range 1-91), and 61 (66%) of the patients were male. Abdominal   
pain was the most common symptom, occurring in the 53 patients (57%).   
Twenty-nine (31%) patients were asymptomatic at the time of diagnosis. For   
35 of the treated patients, anticoagulation (71%), anti-platelet agents   
(20%), and catheter-directed urokinase infusion (26%) were the common   
modalities. Bleeding was noted in 6 (17%) of the patients, and all of the   
bleeding incidents were in the gastrointestinal tract. In the multivariate   
analysis, treatment was the only independent factor for thrombus improvement   
[odds ratio 8.54, 95% confidence interval 2.61-28.0, P < 0.001]. The results   
were the same when we analyzed the symptomatic and asymptomatic subgroups.   
The cumulative probability of improvement was higher among the treated   
patients compared to untreated patients at 2 years (62.8% vs. 26.2%, P<   
0.001), and the benefits of the treatment were evident among the symptomatic   
and asymptomatic patients. Conclusions: Asymptomatic PVT patients may   
benefit from treatment. Further large-scale or prospective studies are   
necessary.  
  
RECORD 443  
Treatment of ascites, portal vein thrombosis and hepatic encephalopathy in   
patients with cirrhosis of the liver  
Gerbes A.L. Glberg V.  
Viszeralmedizin: Gastrointestinal Medicine and Surgery (2012) 28:5   
(297-303). Date of Publication: Oktober 2012  
  
Background: Ascites, portal vein thrombosis and hepatic encephalopathy are   
important complications of cirrhosis of the liver. Guidelines for the   
treatment of ascites have recently been published. Method: This manuscript   
summarizes up-to-date recommendations on the basis of the DGVS S3 guideline   
and of other guidelines as well as of the authors' experience. Results and   
Conclusions: TIPS (transjugular intrahepatic portosystemic shunt) is the   
preferred treatment for refractory or recidivant ascites unless there are   
contraindications. The therapy of hepatorenal syndrome type 1 with albumin   
and the vasoconstrictor Terlipressin has been proven effective. Treatment of   
portal vein thrombosis comprises a strategy of anticoagulation, TIPS and   
liver transplantation. The most important therapeutic strategy for hepatic   
encephalopathy is the search for as well as the treatment of trigger events.   
Rifaximin is being increasingly used for the treatment and prophylaxis of   
hepatic encephalopathy. © 2012 S. Karger GmbH, Freiburg.  
  
RECORD 444  
Prediction of venous thromboembolism (VTE) in patients with pancreatic   
cancer using clinical data, biomarkers, and VTE risk models  
Ruch J.M. Bellile E. Hawley A.E. Anderson M.A. Wakefield T.W. Sood S.L.  
Blood (2012) 120:21. Date of Publication: 16 Nov 2012  
  
INTRODUCTION: VTE is common in patients with cancer and causes significant   
morbidity and mortality. Clinical risk models and biomarkers including   
C-reactive protein (CRP), soluble P-selectin (sPsel), and D-dimer have been   
used to predict VTE in diverse groups of cancer patients at varying risk for   
VTE. The applicability of these findings to specific high risk subtypes of   
cancer has not been established. Therefore, we sought to identify the value   
of clinical factors, plasma biomarkers, and risk models in predicting VTE in   
patients with pancreatic cancer, a malignancy with a high predilection for   
VTE. METHODS: Patients seen at the University of Michigan Comprehensive   
Cancer Center (UMCCC) and previously consented and enrolled in a prospective   
cohort study were eligible. Inclusion criteria are diagnosis of pancreatic   
adenocarcinoma, evaluation at UMCCC, no VTE within a month prior to cancer   
diagnosis, and documentation in the Electronic Medical Record (EMR) at least   
every 6 months until death. Primary objective was to identify factors   
predictive of VTE. Secondary objectives were to develop a VTE predictive   
model, assess the utility of published VTE risk models, and evaluate factors   
associated with overall survival (OS). Demographics, clinical data, and VTE   
(deep vein thrombosis [DVT], portal vein thrombosis [PVT], or pulmonary   
embolism [PE]) rate were obtained from the EMR. ELISAs were performed for   
CRP, D-dimer, Mac-2 binding protein, soluble E-selectin (sEsel), and sPsel   
using banked plasma specimens drawn at diagnosis. A retrospective cohort   
study was performed including univariate and multivariate regression   
analysis. The utility of predictive models by Khorana, et al (Blood, 2008.   
111:4902-4907), which includes cancer site, body mass index (BMI),   
hemoglobin (Hb), platelet (plt) count, and white blood cell count, and the   
expanded model by the Vienna Cancer and Thrombosis Study (CATS) (Blood,   
2010. 116:5377-5382), which additionally includes sPsel and D-dimer, were   
assessed. RESULTS: Between 2005 and 2011, 89 patients were eligible for   
analysis. Median follow-up was 268 (18-2433) days. Twenty (22%) cases had a   
VTE; 10 (50%) DVT, 2 (10%) PE, 4 (20%) PVT, and 4 (20%) multiple VTEs. Mean   
(SD) age was 63.4 (8.9) in cases and 65.3 (11.2) in controls. Women   
accounted for 55% of cases and 48% of controls. Higher BMI (median 28.8   
[21.2-44.7] in cases vs. 25.4 [16.4-43.3] in controls, p=0.03) and lower plt   
count (median 241 [145-323] in cases vs. 289 [97-648] in controls, p=0.001)   
were associated with VTE on univariate analysis. On multivariate regression   
analysis, lower plt count (β -0.01, SE 0.004) and lower Hb (β -0.43, SE   
0.20) were predictive of VTE after adjusting for BMI, tumor location, and   
treatment with surgery, chemotherapy or radiation (AUC 0.78). None of the   
biomarkers were significantly associated with VTE on univariate analysis,   
although there was a trend with D-dimer (p=0.09). The Khorana score was   
determined in 85 patients; 48 were intermediate (2 points) and 37 high risk   
(3 points) with VTE rates of 20.8% and 24.3%, respectively (p=0.70). The AUC   
of this model was 0.63. The risk score from CATS was calculated for 84   
patients; 54 were intermediate (2 or 3 points), 17 high (4 points), and 13   
highest risk (5 points). VTE incidence was not different among these groups   
and the AUC was 0.65. Factors associated with poor OS on univariate analysis   
were: age (per 10-year increment) (HR [95% confidence interval], p-value)   
(1.35 [1.07-1.71], 0.013), chronic kidney disease (5.67 [2.62-12.25],   
<0.0001), use of anticoagulation (3.14 [1.33-7.41], 0.009), stage III/IV vs.   
I/II pancreas cancer (2.05 [1.27-3.32], 0.003), and INR (1.65 [1.04-2.63],   
0.035); elevated Hb (0.87 [0.76-0.99], 0.041) and sEsel (0.46 [0.29-0.72],   
0.0007) were protective. CONCLUSIONS: Pancreatic cancer patients with higher   
BMI, lower plt count, and lower Hb were more likely to develop VTE. Other   
clinical variables and biomarkers did not add additional predictive   
information. Elevated sEsel, important for neutrophil trafficking to sites   
of inflammation, was found to be protective on survival analysis. The risk   
models developed by Khorana, et al and CATS in a diverse group of patients   
with cancer were not able to further differentiate VTE risk among this   
already high risk group. Additional work is needed to determine which   
patients with pancreatic cancer are at highest risk for VTE and who may   
benefit most from thromboprophylaxis.  
  
RECORD 445  
Should anticoagulants be administered for portal vein thrombosis associated   
with acute pancreatitis?  
Park W.-S. Kim H.-I. Jeon B.-J. Kim S.-H. Lee S.-O.  
World Journal of Gastroenterology (2012) 18:42 (6168-6171). Date of   
Publication: 14 Nov 2012  
  
Venous complications in patients with acute pancreatitis typically occur as   
a form of splenic, portal, or superior mesenteric vein thrombosis and have   
been detected more frequently in recent reports. Although a well-organized   
protocol for the treatment of venous thrombosis has not been established,   
anticoagulation therapy is commonly recommended. A 73-year-old man was   
diagnosed with acute progressive portal vein thrombosis associated with   
acute pancreatitis. After one month of anticoagulation therapy, the patient   
developed severe hematemesis. With endoscopy and an abdominal computed   
tomography scan, hemorrhages in the pancreatic pseudocyst, which was   
ruptured into the duodenal bulb, were confirmed. After conservative   
treatment, the patient was stabilized. While the rupture of a pseudocyst   
into the surrounding viscera is a well-known phenomenon, spontaneous rupture   
into the duodenum is rare. Moreover, no reports of upper gastrointestinal   
bleeding caused by pseudocyst rupture in patients under anticoagulation   
therapy for venous thrombosis associated with acute pancreatitis have been   
published. Herein, we report a unique case of massive upper gastrointestinal   
bleeding due to pancreatic pseudocyst rupture into the duodenum, which   
developed during anticoagulation therapy for portal vein thrombosis   
associated with acute pancreatitis. © 2012 Baishideng. All rights reserved.  
  
RECORD 446  
anticoagulation for the treatment of thrombotic complications in patients   
with cirrhosis  
Rodriguez-Castro K.I. Simioni P. Burra P. Senzolo M.  
Liver International (2012) 32:10 (1465-1476). Date of Publication: November   
2012  
  
Cirrhotic patients can develop thrombotic complications, which in this group   
of patients occur with a greater frequency than in the general population.   
portal vein thrombosis (PVT) is the most common thrombotic phenomenon,   
although deep venous thrombosis and pulmonary embolism can also occur. Risk   
factors for thrombosis include inherited and acquired deficiency of factors   
involved in anticoagulation mechanisms, venous stasis of the portal vein   
owing to architectural derangement of the liver and possibly local factors   
related to the endothelium. Clinical manifestations of PVT range from   
asymptomatic disease to a life-threatening complication, and although it is   
no longer considered an absolute contraindication for liver transplant, its   
presence may require challenging surgical techniques, which entail greater   
morbidity. anticoagulation therapy is henceforth an important strategy to   
treat cirrhotic patients with PVT, although experience in this group of   
patients is limited. Vitamin K antagonists and low-molecular-weight heparin   
have been used successfully, achieving recanalization of the thrombosed   
vessel in patients with cirrhosis; however, the precise drug regimen   
management and monitoring has not be fully explored in this group of   
patients. © 2012 John Wiley & Sons A/S.  
  
RECORD 447  
Prophylactic anticoagulation in cirrhotics: A paradox for prime time?  
Fontana R.J.  
Gastroenterology (2012) 143:5 (1138-1141). Date of Publication: November   
2012  
  
RECORD 448  
Diagnosis and treatment of portal thrombosis in liver cirrhosis  
Seijo S. García-Criado T. Darnell A. García-Pagán J.C.  
Gastroenterologia y Hepatologia (2012) 35:9 (660-666). Date of Publication:   
November 2012  
  
Improved imaging techniques and the routine use of color Doppler ultrasound   
in the follow-up of patients with liver cirrhosis has increased diagnosis of   
portal vein thrombosis (PVT) in these patients. The extension of PVT should   
be evaluated with computed tomography angiography or magnetic resonance   
angiography. The natural history of PVT in cirrhosis and its impact on liver   
disease is unknown but it seems clear that PVT could increase the morbidity   
and mortality associated with liver transplantation and can even be a   
contraindication to this procedure when the thrombus extends to the superior   
mesenteric vein. anticoagulation is a relatively safe and effective   
treatment in achieving recanalization of the splenoportal axis or in   
preventing progression of thrombosis and is therefore frequently used. The   
use of transjugular intrahepatic portosystemic shunts (TIPS) is reserved for   
patients unresponsive to anticoagulation or in those with severe   
complications of portal hypertension. © 2012 Elsevier España, S.L. and AEEH   
y AEG.  
  
RECORD 449  
Inferior vena cava clip migration: Unusual cause of duodenal foreign body  
Antonoff M.B. Beilman G.J.  
Annals of Vascular Surgery (2012) 26:8 (1129.e5-1129.e8). Date of   
Publication: November 2012  
  
Before the development of the inferior vena cava (IVC) filter, various   
techniques of IVC interruption were described for the management of patients   
at high risk for thromboembolic events, and for whom anticoagulation was   
either inadequate or contraindicated. In this report, we describe the   
enteric migration of a Miles IVC clip, occurring 27 years after IVC   
interruption. This previously undescribed complication and the patient's   
prolonged follow-up period render this case of significant interest. ©   
Annals of Vascular Surgery Inc.  
  
RECORD 450  
portal vein thrombosis - experience in a single centre  
Swallow G. Pavord S.  
British Journal of Haematology (2012) 159:4 (482-484). Date of Publication:   
November 2012  
  
RECORD 451  
Splenectomy combined with anticoagulation therapy for antithrombin defi   
ciency with portal vein thrombosis and refractory thrombocytopenia in   
children  
Sung S.-Y. Hsu K.-F. Yu J.-C. Chan D.-C. Chen Y.-C. Chen T.-W.  
Journal of Medical Sciences (Taiwan) (2012) 32:5 (243-246). Date of   
Publication: 20 Oct 2012  
  
Antithrombin defi ciency with portal vein thrombosis is an unusual disease   
in clinic. A 16-year-old adolescent with a history of frequent spontaneous   
epistaxis and hypersplenism in his childhood presented with abdominal pain   
and distension. Chronic portal vein thrombosis, portal hypertension, and   
hypersplenism were caused by antithrombin defi ciency based on laboratory   
data and image fi ndings. He underwent splenectomy and subsequent   
anticoagulation therapy with warfarin. Postoperative course was   
uneventfully. During 1 year follow-up, he had no epistaxis and epigastric   
pain and platelet count showed normal value (366,000/μL). © 2012 JMS.  
  
RECORD 452  
Comparison of two strategies of management of portal thrombosis in the   
absence of cirrhosis  
Zaoui S. Trillot N. Louvet A. Cambier N. Colin M. Wemeau M. Canva V.   
Tintillier V. Mathurin P. Biernat J. Jude B. Susen S. Dharancy S.  
Hepatology (2012) 56 SUPPL. 1 (755A). Date of Publication: October 2012  
  
Portal thrombosis (PT) in the absence of cirrhosis is often related to a   
combination of local causes and general prothrombotic conditions. The   
duration of anticoagulation therapy has been recommended for 6 months taking   
into account the thrombotic underlying conditions. In the absence of a   
strong prothrombic risk factor, the indications for permanent   
anticoagulation are still unclear. The aim of this study was to compare 2   
strategies of treatment: anticoagulation withdrawal (AW) versus permanent   
anticoagulation (PA). Patients and Methods: Retrospective controlled study   
performed from 2007 to 2011 in consecutive pts with PT without cirrhosis.   
Investigations for thrombotic risk factors were conducted and   
myeloproliferative neoplasms and JAK2 mutation were ruled out. Abdominal CT   
scan was planned at M1, M6 then annually or as required. In the two groups,   
low molecular weight heparin was early initiated at diagnosis and shifted to   
vitamin K antagonists targeting an INR 2 to 3. Treatment was withdrawn (AW)   
or continued (PA) according to a multidisciplinary committee decision. End   
points were the rates of recurrent thrombosis and hemorrhagic events.   
Results: Fifty two pts (mean age 47.2±14 yrs) followed for a mean of 31±17   
months were enrolled. PT was extra-hepatic in 32%, segmental in 14% and   
mixed in 54% of cases. Thirteen % of pts had no risk factor of deep vein   
thrombosis, 57% had one and 29% had at least 2 risk factors. The most   
prevalent risk factors were past medical history of vein thrombosis (50%),   
inflammatory condition (44%) and oral contraceptive (31%). anticoagulation   
therapy was continued in 32 pts (61% of cases) whereas the duration of   
therapy was 8.9±3 months in AW group. AW and PA groups differed in terms of   
circumstances of diagnosis (fortuitous in 60% vs 12.5% p=0.001), presence   
factor II /V heterozygous mutation (5% vs 34%, p=0.01) and location of PT   
(extrahepatic PT: 45% vs 81%, p=0.006). CT scan at M6 was available for 37   
pts (71%), recanalisation was obtained at similar rates in the 2 groups   
(52.6% vs 55.6%, p=ns). Hemoglobin/platelet counts did not differ at any   
time of analysis. An extension of the thrombus was more frequently detected   
in the AW group at month 36 (p=0.01) whereas the cumulative rate of   
hemorrhagic events and the rate of severe events were not different between   
the two groups (17.6% vs 32% and 6 vs 10%, p=ns). Conclusion:   
anticoagulation therapy is efficient in the majority of pts with PT in the   
absence of cirrhosis and myeloproliferative neoplasms. In this study, the   
maintenance of anticoagulation was not associated with an excess risk of   
bleeding and prevented the risk of thrombosis extension at middle term.  
  
RECORD 453  
Pylephlebitis: A rare cause of intrabdominal sepsis  
Bhatia A. Kathpalia P. Pillai A. Ahn J. Cohen S.  
American Journal of Gastroenterology (2012) 107 SUPPL. 1 (S440). Date of   
Publication: October 2012  
  
Purpose: Intra-abdominal infections can be associated with a variety of   
complications. We present an unusual case of an infection of the portal vein   
(pylephlebitis). A 72 year-old Caucasian male with hypertension presented   
with epigastric pain and fevers. Initial workup revealed leukocytosis (19   
K/μL) as well as mildly elevated bilirubin (1.9 mg/dL). CT scan revealed an   
acute leftportal vein thrombosis as well as concern for a fluid collection   
near the hilum of the liver, possibly consistent with a hepatic abscess.   
Treatment was initiated with intravenous heparin as well as broad-spectrum   
antibiotics. The patient subsequently developed hypotension requiring   
pressor support, progressive renal failure, and atrial fibrillation   
necessitating cardioversion. Blood cultures revealed gram negative   
bacteremia with Veillonella species. The patient then improved with the   
antibiotics and anticoagulation over the next 48 hours. It was unclear   
whether the hilar fluid collection represented a cystic hepatic lesion or a   
hepatic abscess through direct extension of the pylephlebitis. Given the   
patient's clinical improvement, drainage of this hilar lesion was not   
attempted. The patient received 6 weeks of intravenous antibiotic therapy as   
well as Coumadin therapy. At long-term follow-up, he had no further   
symptoms. Repeat CT scan showed a small complex hepatic cyst, and   
persistence of the leftportal vein thrombosis. The plan was to continue at   
least 6 months of anticoagulation and repeat imaging studies 6 months later.   
Pylephlebitis, or septic portal vein thrombosis, can complicate any   
intra-abdominal infection including diverticulitis, appendicitis, biliary   
process or even inflammatory bowel disease. It is rarely associated with an   
underlying hypercoagulable condition. Given its significant mortality (up to   
30% even with antibiotic therapy), pylephlebitis should be considered in any   
patient that presents with fevers, abdominal pain, and jaundice. Early   
intravenous antibiotic therapy remains the mainstay of treatment. The role   
of anticoagulation is controversial, but appears to have a beneficial role   
through improved vascular recanalization, which may result in improved   
mortality.  
  
RECORD 454  
Hemostatic imbalance does not predict response to anticoagulant therapy in   
cirrhosis patients with portal vein thrombosis (PVT)  
Rodriguez-Castro K.I. Sartori M.T. Pizzuti D. Fadin M. Spiezia L. Simioni P.   
Russo F.P. Burra P. Senzolo M.  
Hepatology (2012) 56 SUPPL. 1 (924A). Date of Publication: October 2012  
  
Aim: The purpose of this study was to assess hemostatic status in terms of   
pro- and anti-coagulant factors, as well as thrombus, and patient   
characteristics, as predictors of therapeutic efficacy of anticoagulation to   
treat PVT in cirrhotics. Patients and methods: A cohort of 43 cirrhosis   
patients who were diagnosed with PVT and received anticoagulation therapy   
with low molecular weight heparin were retrospectively evaluated. Nadroparin   
95 IU/Kg was administered to all patients, with a 40% dose reduction in   
patients with platelet count below 50.000x109/L. Estimation of interval   
between appearance of PVT and start of anticoagulation therapy was made,   
along with determination of PVT characteristics and extension. All patients   
underwent screening for thrombophilia and dosing of plasmatic pro-and   
anti-coagulation factors. Imbalance between pro- and anti-coagulant factors   
was further evaluated using the ratio FactorVIII/ Protein C. Vessel   
recanalization was evaluated monthly using abdominal ultrasound and every 3   
months by CT scan. Results: 31 patients were males and mean age was 58±11   
years. Etiology of liver disease was viral in 48.8% and alcoholrelated in   
30.2% of cases. Partial PVT was found in 34/43 patients, with extension into   
superior mesenteric or splenic veins in 14/43. Upon starting of   
anticoagulation therapy, estimated interval from appearance of PVT was >6   
months in 32/43, and longer than 6 months in the remaining 11 cases. Genetic   
thrombophilic mutations were found in 7 patients. Twenty-five patients   
responded to anticoagulant therapy, obtaining repermeation of the portal   
vein (16 achieved complete recanalization) after a mean time of 6±9.6 months   
of therapy. No correlation was found between standard coagulation   
parameters, plasmatic activity of factors VII, IX, XI, AT, PS, PC,   
fibrinogen, or factor VIII/PC ratio, and thrombus disappearance. Likewise,   
repermeation did not correlate with the extension of PVT, presence of   
thrombophilic mutations, severity of liver disease, or etiology of   
cirrhosis. An interval between development of PVT and start of   
anticoagulation therapy >6 months was the only significant predictor of   
anticoagulation efficacy (75% versus 18%, p>.001) with no patients with   
older thrombus achieving repermeation after 6 months of therapy.   
Conclusions: the interval between PVT occurrence and start of   
anticoagulation therapy is the only predictor of recanalization; on the   
contrary, hemostatic imbalance does not correlate with anticoagulant   
response. For patients with recent thrombus, continuation of anticoagulant   
therapy beyond 6 months could increase the possibility of repermeation.  
  
RECORD 455  
Portal and mesenteric vein thrombosis in hereditary spherocytosis  
Maheshwari N. Kulkarni S. Ikwueke I. Danve A. Anand S. Awasthi S. Kumari D.   
Harley J.  
American Journal of Gastroenterology (2012) 107 SUPPL. 1 (S443). Date of   
Publication: October 2012  
  
Introduction: Hereditary spherocytosis (HS) is an inherited hemolytic   
disorder and complications associated with HS include extra medullary   
hematopoiesis, gallstones, aplastic crisis, leg ulcers but venous or   
arterial thrombosis are uncommon. We report clinical and laboratory findings   
of HS patient with portal and superior mesenteric vein thrombosis. Case   
Report: 36-year-old male with history of HS, splenectomy at 5 years of age,   
hypertension, hyperlipidemia admitted with complaints of peri umbilical   
abdominal pain, nausea and vomiting for one day after eating cheeseburger.   
Denies alcohol, smoking or drug abuse and was not on medications at home.   
Review of systems was unremarkable except for above. On physical   
examination, he was febrile of 101F, abdomen was soft , non-distended,   
moderate to severe diffuse tenderness with prominence in epigastric and   
periumbilical areas, no rebound tenderness and normal bowel sounds, rest of   
examination was unremarkable. Laboratory findings were pertinent for   
leukocytosis with left shift , mildly elevated creatine, normal lactate,   
amylase and lipase. CAT scan of abdomen showed acute portal vein and   
superior mesenteric vein thrombosis, wall edema of stomach (antrum, pylorus)   
and duodenum. Hypercoaguable work up including antithrombin III, factor V,   
factor V Leiden mutation, anti-phospholipids antibody panel, protein C and   
S, serum homocysteine, coombs test, prothrombin gene mutation,   
anticardiolipin antibodies, LDH, and haptoglobin were negative. Patient was   
started on anti-coagulation and symptoms improved in 2-3 days with good oral   
tolerance and discharged on oral anticoagulation to followup in hematology   
clinic. Discussion: There are few reported cases of arterial and venous   
thromboembolic events in splencetomized HS patients. These vascular events   
involve brain, lungs, and to our knowledge, only a few cases of portal and   
superior mesenteric vein thrombosis have been reported. First case of HS and   
portal vein thrombosis was of a 39-year-old male with HS and splenectomy who   
developed hematemesis and portal and superior mesenteric vein thrombosis.   
The precise mechanism underlying hypercoaguable state in patients with HS is   
not fully understood yet though a few suggested mechanisms have been   
described like accelerated lipid loss with increased substrate for   
procoagulant activation and platelet activation causing transfer of anionic   
phospholipids from inner to the outer membrane accelerating coagulation   
process. Physicians should keep this rare complication in differential   
diagnosis for HS patients presenting with abdominal pain and prompt   
diagnosis may prevent further clinical deterioration.  
  
RECORD 456  
Predictive factors of portal vein thrombosis (PVT) in cirrhotic patients:   
Importance of non-invasive measurement of splanchnic blood flow with MRI  
Sogni P. Vauthier A. Gouya H. Corouge M. Mallet V. Vallet-Pichard A.   
Fontaine H. Trabut J.-B. Tripon S. Vignaux O. Legmann P. Pol S.  
Hepatology (2012) 56 SUPPL. 1 (950A). Date of Publication: October 2012  
  
Background: A non-tumoral PVT in cirrhotic patients worsens the prognosis.   
It's important to determine predictive factors, in the setting of future   
preventive anticoagulation studies. The aim of this study was the   
identification of predictive factors of PVT with a focus on splanchnic and   
systemic blood flow measurement with MRI which is a non-invasive and a   
non-operator dependent method. Patients and Methods: All cirrhotic patients   
with non-invasive MRI validated measurement of blood flows (1) were   
included. Initial clinical, biological and endoscopic data were collected   
and the follow-up was performed with ultrasound every 6 months or earlier if   
a complication occurred, until the diagnosis of PVT (complete or partial) or   
the end of follow- up, death or transplantation. Patients with tumoral PVT   
were excluded. Results: Forty-seven cirrhotic patients (58±2 years; 72%   
male; 18 alcohol, 16 HCV and 13 associated or others) with an initial MRI   
measurement of portal vein, azygos and abdominal aortic blood flows were   
prospectively included. At the inclusion, 13, 21 and 12 patients were   
Child-Pugh A, B and C respectively, 60% with an ascites, 9% with an hepatic   
encephalopathy and 28 % received beta-blockers. Esophageal varices were of   
grade 0-1 in 21 patients and grade 2-3 in 26. During the follow-up (17.7±1.7   
months), a PVT occurred in 10 patients with a delay of 21.3±4.3 months. In   
univariate analysis, the only initial factor associated with PVT was the   
Child- Pugh score (p=0.047). Factors associated with PVT in univariate   
analysis with p<0.20 were included in multivariate analysis (Child-Pugh,   
ascites, portal flux direction, portal blood flow and azygos blood flow).   
The 2 independent and initial factors associated with PVT were azygos blood   
flow p=0.015) and ascites (p=0.043). Conclusion: The presence of ascites and   
a low speed azygos blood flow were the 2 independent and initial factors   
associated with the occurrence of PVT in cirrhotic patients. In the future   
anticoagulation studies for the prevention of PVT in cirrhotic patients,   
non-invasive measurement of blood flows, especially for the azygos vein,   
could be of interest for selecting at risk population.  
  
RECORD 457  
Colonic endometriosis: A case of chronic abdominal pain  
Horsley-Silva J. Vazquez Roque M.  
American Journal of Gastroenterology (2012) 107 SUPPL. 1 (S477-S478). Date   
of Publication: October 2012  
  
Purpose: A 43-year-old white female presented to clinic for further   
evaluation of two years of abdominal pain mostly on the left side, dull,   
sometimes sharp, worsening after eating, improving after bowel movements and   
no associated bloody stools. Patient had a history of deep venous thrombosis   
and pulmonary embolism three years prior treated with coumadin for one year.   
A few months prior to presentation patient had acute right upper quadrant   
pain leading to computer tomography (CT) of the abdomen demonstrating portal   
vein thrombosis and superior mesenteric vein (SMV) thrombosis causing   
treatment initiation with coumadin again. Patient underwent evaluation for   
thrombophilia disorders and malignancy resulting in a colonoscopy, which   
revealed a nonbleeding mucosal ulceration in the proximal ascending colon   
and initial biopsy consistent with chronic inflammation. Repeat colonoscopy   
one month later demonstrated a persistent ulcer in the mid-ascending colon   
with biopsies revealing endometriosis. Patient's gynecologic history   
involved two previous gestations, a caesarean section, uterine fibroids and   
hysterectomy many years prior. No history of endometriosis. Further   
evaluation at our hospital involved CT enterography demonstrating a   
nonocclusive thrombus in the SMV and occlusive thrombus in the right   
ileocolic branch. Colonoscopy showed granularity and nodularity in the   
ascending colon without ulceration, and biopsy revealed colonic mucosa with   
associated inflammation with stroma and focal glands suggestive of   
endometriosis. Patient underwent evaluation by vascular surgery concluding   
no surgery was necessary since adequate vessels were present to supply gut   
through collaterals. Hematology evaluation revealed a negative complete   
coagulation survey. Gynecology desired to pursue a trial with lupron   
(leuprolide) injections. Colorectal surgery recommended to continue   
anticoagulation longer and to monitor on hormonal suppression and if   
improvement plan for laparoscopic right colon resection. Colonic   
endometriosis is a rare entity that can present with a variety of   
manifestations making it difficult to diagnose. It most commonly affects   
pre-menopausal women in early forties around half of which demonstrate   
previous pelvic endometriosis. Most commonly it presents with abdominal pain   
and can be associated with stenosis, polyps, mural masses, and ulcers.   
However, intestinal endometriosis is rarely found within the superficial   
mucosa of the intestine, with estimates around 30% confirmed on endoscopy.   
Surgical resection is the gold standard for those with refractory symptoms,   
and is often when a tissue diagnosis becomes available. Endoscopic   
ultrasound with tissue sampling may provide diagnosis if routine endoscopy   
fails.  
  
RECORD 458  
Thrombosis in hematologic malignancies: Risks and consequences  
Kwaan H.C.  
International Journal of Hematologic Oncology (2012) 1:1 (87-95). Date of   
Publication: October 2012  
  
Thrombotic complications in hematologic malignancies have been found to be   
high among the various forms of cancer. Thrombosis not only increases the   
morbidity, but also has an adverse impact on survival. The pathogenesis   
among the different forms of hematologic malignancies is reviewed in this   
article. The thrombogenicity of the individual malignant cells, the tumor   
burden, treatment modalities and presence of comorbidities are among the   
major risk factors. These factors vary with the acute leukemias, lymphomas,   
multiple myeloma and myeloproliferative neoplasms. The thrombogenetic   
factors in the more common hematologic malignancies are discussed. Results   
of recent randomized controlled clinical trials are beginning to provide   
data for meaningful therapeutic guidelines on thromboprophylaxis. They also   
enable the clinician to assess the risk factors in each individual patient.   
More clinical trials are needed to provide better risk stratification and to   
devise risk-adapted treatment regimens. © 2012 Future Medicine Ltd.  
  
RECORD 459  
Guidelines on the investigation and management of venous thrombosis at   
unusual sites  
Tait C. Baglin T. Watson H. Laffan M. Makris M. Perry D. Keeling D.  
British Journal of Haematology (2012) 159:1 (28-38). Date of Publication:   
October 2012  
  
RECORD 460  
Prophylactic anticoagulation following splenectomy in cirrhotic patients  
Chen P. Wang W. Yan L.  
Hepato-Gastroenterology (2012) 59:119 (2042-2044). Date of Publication:   
October 2012  
  
Background/Aims: The aim of the study is to address the impact of   
prophylactic anticoagulation on the incidence of PVT in cirrhotic patients   
compared with no prophylactic anticoagulation after splenectomy.   
Methodology: Randomized controlled trials (RCTs) comparing prophylactic   
anticoagulation and no prophylactic anticoagulation after splenectomy were   
included by a systematic literature search. Two authors independently   
assessed the trials for inclusion and extracted the data. Results: A total   
of 1406 studies were searched and none met our inclusion criteria.   
Conclusions: Most current studies were not prospective control trials based   
on small sample sizes and single center experiences. Therefore, it is hard   
to draw the conclusion that prophylactic anticoagulation following   
splenectomy should be recommended in cirrhotic patients. More attention to   
the problem is required and the administration of routine postoperative   
anticoagulation needs to be standardized. © H.G.E. Update Medical Publishing   
S.A.  
  
RECORD 461  
Management of acute portomesenteric venous thrombosis induced by protein S   
deficiency: Report of a case  
Lin H.-Y. Ho C.-M. Lai H.-S. Lee P.-H.  
Surgery Today (2012) 42:10 (1014-1018). Date of Publication: October 2012  
  
Hereditary protein S deficiency is a risk factor which may predispose   
patients to venous thrombosis. Deep venous thrombosis of the lower   
extremities can result in painful congestion, while the presence of   
mesenteric venous thrombosis (MVT) can cause abdominal emergencies. We   
herein report a protein S-deficient patient presenting with acute   
portomesenteric venous thrombosis. Early management using anticoagulant   
therapy was initially successful. However, the subsequent bowel stricture   
resulting from the ischemic insult was further managed with a surgical   
bypass. The patient was kept on long-term thrombophylaxis. The treatment   
strategy for MVT with bowel ischemia has evolved from aggressive   
portomesenteric thrombectomy with resection of the involved bowel, to   
conservative anticoagulation to recanalize thrombotic mesenteric veins with   
bowel preservation. Surgical intervention is reserved for transmural   
necrosis or bowel perforation. The perioperative thrombophylaxis of   
inherited thrombophilic patients is also important for preventing further   
thromboembolic events. © Springer 2012.  
  
RECORD 462  
portal vein thrombosis in splenectomized cirrhotic patient: A case report  
Panamonta N. Kijsirichareanchai K. Mankongpaisarnrung C. Rakvit A.  
American Journal of Gastroenterology (2012) 107 SUPPL. 1 (S401). Date of   
Publication: October 2012  
  
Purpose: portal vein thrombosis (PVT) and systemic venous thromboembolism   
are significantly increased in cirrhotic patients even without   
hepatocellular carcinoma. The risk of developing such complications is   
usually associated with advanced cirrhosis. When cirrhosis is mild and   
compensated, other possible causes of PVT should also be considered. We   
present a case of PVT in chronic hepatitis C infection with focal cirrhosis   
and portal hypertension with history of splenectomy. A 47-year-old Caucasian   
woman presented with chronic hepatitis C, genotype 1. She was treated with   
pegylated interferon and ribavirin for 48 weeks, but developed viral   
relapse. She had splenectomy when she was young for unclear reason. Her   
findings on pathologic examination of the liver biopsy were consistent with   
focal cirrhosis in mild to moderate chronic hepatitis; the Knodell's   
histologic activity index (HAI) score was 7-9/22. Her MELD score was 6. An   
annual esophagogastroduodenoscopy revealed non-bleeding grade II esophageal   
varices and cardial gastric varices. A computerized tomography scan revealed   
the sub-occlusive thrombus at the bifurcation of the main portal vein. The   
workup for hypercoagulable state showed low protein C, protein S, and   
antithrombin III levels. The inferior vena caval venogram showed thrombotic   
occlusion in left , right, and common portal vein with a portal systemic   
pressure gradient of 11 mmHg. Collateralization was present at portal   
hiatus. The ultrasound guided thrombus removal with interventional   
thrombolysis was attempted in order to avoid systemic anticoagulation   
therapy. However, the guidewire failed to pass through the obstructed portal   
vein. She eventually was evaluated for liver transplantation. This is a case   
that demonstrates the presence of PVT in a patient with mild compensated   
cirrhosis (based on both histological and clinical evaluation) without   
hepatocellular carcinoma. The incidence of PVT in well-compensated cirrhosis   
is reported between 0.6-16%. An increased frequency is reported in   
decompensated cirrhosis and in up to 35% of cirrhotic patients with   
hepatocellular carcinoma. The origin of PVT is multifactorial in most   
cirrhotic patients. Low serum protein C, protein S, and antithrombin III   
level secondary to decreased protein synthesis in cirrhosis can contribute   
to a prothrombotic state. Splenectomy is another possible cause of PVT in   
this case, since PVT after splenectomy occurs in 6-8%. However, the late   
onset of presentation makes it a less likely cause since most PVT occurs in   
days to weeks after splenectomy.  
  
RECORD 463  
Splanchnic vein thrombosis  
Riva N. Donadini M.P. Dentali F. Squizzato A. Ageno W.  
Phlebologie (2012) 41:3 (135-139). Date of Publication: 2012  
  
Splanchnic vein thrombosis (SVT) - including mesenteric, portal, splenic and   
supra-hepatic veins thrombosis - is an underdiagnosed disease, with   
heterogeneous clinical presentations and a non-negligible rate of incidental   
findings. The main risk factors include abdominal diseases or interventions   
(e.g. infections, cirrhosis, abdominal cancer or surgical procedures),   
haematological disorders (mainly myeloproliferative neoplasms), inherited   
thrombophilic states and hormonal imbalances. New biological markers of   
subclinical disorders have recently been identified: JAK2 mutation and flow   
cytometry for CD55 and CD59. Clinical manifestations are generally   
aspecific. During the acute phase, main symptoms can be abdominal pain,   
gastrointestinal bleeding and ascites; while long-term consequences include   
liver cirrhosis and portal hypertension. Advances in non-invasive vascular   
imaging (Doppler ultrasound, angio-computed tomography and magnetic   
resonance imaging), have improved the diagnosis of SVT. Alterations in blood   
tests may suggest an underlying haematological or hepatic disorder.The   
optimal treatment of SVT remains an open issue, since large clinical trials   
are lacking. Expert consensus recommend to treat acute symptomatic   
non-cirrhotic portal vein thrombosis with parenteral anticoagulation during   
the acute phase, followed by oral anticoagulants for at least 3 months,   
though lifelong treatment is recommended in case of persistent prothrombotic   
factors. In Budd-Chiari syndrome, anticoagulation is recommended for all   
patients in the absence of major contraindications. However, the risk to   
benefit-ratio of anticoagulant therapy, both in the acute phase and for the   
long-term secondary prevention, still needs to be better assessed. ©   
Schattauer 2012.  
  
RECORD 464  
How to manage portal vein stenosis  
Vidal V. Gaubert J.-Y.  
CardioVascular and Interventional Radiology (2012) 35 SUPPL. 1 (S133). Date   
of Publication: September 2012  
  
Learning Objectives 1. To present an update of recent trials and   
meta-analysis 2. To describe the indications, techniques and devices for   
recanalizing portal and mesenteric vein occlusion 3. To describe the   
techniques of thrombectomy and thrombolysis portal vein thrombosis (PVT)   
leads to complications of portal hypertension which causes bleeding through   
varices. Eight to 20% are idiopathic, in adults. PVT is associated with   
cirrhosis and its incidence increases as the disease progresses. The   
prevalence in patients with cirrhosis and hepatocarcinoma is as high as 44%.   
The other causes are neoplasm, coagulation disorders and inflammatory-   
infectious abdominal causes (1). The acute and chronic forms of PVT are   
differentiated by the length of time over which they develop. In acute   
stage, symptoms are generally nonspecific. If the thrombus extends as far as   
the distal mesenteric branches, it may cause ischemia or infarction of the   
mesenteric vein which leads to abdominal pain, nausea, vomiting and ascites.   
The existence of peritoneal irritation and ascites indicates necrosis of the   
wall and perforation. In these cases, surgery is required with 13 to 50%   
mortality reported. In the chronic form, symptoms are usually due to portal   
hypertension, principally bleeding through varices (2). Imaging techniques   
are essential for the diagnosis of PVT because the clinical manifestations   
are minor and unspecific. Four anatomic categories related to the extent of   
PVT have been defined and have clinical relevance for both prognosis and   
treatment: grade I, thrombus limited to the portal vein, grade II, thrombus   
extending to the superior mesenteric vein, grade III, thrombus spreading   
diffusely through the splanchnic venous system but with the presence of   
large collaterals, grade IV, as grade III but without collaterals. The first   
approach for treating acute PVT is to start anticoagulation with heparin at   
once. The aim is to maintain the activated partial thromboplastin time   
(aPTT) at twice its normal level. Once the thrombosis is resolved, heparin   
is replaced by oral anticoagulants for 6 months. anticoagulation alone   
results in resolution of PVT in up to 80% of cases. In chronic forms of PVT,   
anticoagulation is more controversial, because portosystemic variceal   
collaterals have developed, which confer a greater risk of bleeding.   
Selective administration of local fibrinolysis ensures that a high   
concentration of fibrinolytic is present in the portomesenteric venous   
system with lower systemic concentration. Local fibrinolysis can be   
performed through the superior mesenteric artery or directly through the   
portal vein via a transhepatic or transjugular route. Fibrinolysis through   
the SMA seems to be more effective to reach the distal branches of the   
mesenteric veins. In general, transhepatic approach is the most frequently   
used. It is easier than transjugular approach but has a greater risk of   
bleeding. The thrombus is recanalized with guide wires and a multiperforated   
catheter is inserted to perfuse urokinase or rtPA. Pharmacologic   
thrombolysis is an effective technique for treating acute PVT but severe   
hemorrhagic complications occurred in more than 30% of cases, so it is   
recommend to use this technique for severe cases. The efficacy of   
thrombolysis is related to the time over which the thrombus has developed.   
The best results are obtained when the thrombus is less than 14 days. Only   
very slight response can be expected after 40 days. The main complications   
of local fibrinolytic treatment are related to transhepatic puncture, which   
may cause intra-peritoneal bleeding (3). Mechanical percutaneous   
thrombectomy is performed usually in conjunction with other treatments as   
local fibrinolysis or balloon dilatation and stent placement. Balloon   
dilatation with stent placement is the technique of choice for dealing with   
residual thrombosis with stenosis or tumor invasion or local inflammatory   
processes. Mechanical recanalization devices can be divided into devices   
that perform thrombectomy by direct contact (angioplasty balloon, fixed or   
rotating wire baskets and pigtail catheters), hydrodynamic thrombectomy   
devices and rheolytic thrombectomy devices (based on flow). It is also   
useful to try to remove part of the thrombus by aspiration. Balloons used   
ranged usually from 8 to 10 mm diameter and stent is self-expandable. When   
mechanical thrombectomy devices are used, there is a potential risk of   
damaging the vessel, which may predispose to rethrombosis. Although the   
presence of PVT is regarded as a relative contraindication to TIPS, it can   
be very useful in cirrhotic patient with portal hypertension. TIPS in these   
cases need to be inserted by an experimented team.  
  
RECORD 465  
Radiological reporting of incidental portal vein thrombosis - Do we aid   
clinical management?  
Gopalan P. Vinayagam R.  
Clinical Radiology (2012) 67 SUPPL. 1 (S4). Date of Publication: September   
2012  
  
Purpose: portal vein thrombosis is detected incidentally on imaging. There   
is less clarity on the imaging follow up of these patients and their   
management with anticoagulant therapy. Methods and materials: 105 patients   
over 4yrs (2008-2011). CT, US and MR images, reports and selected case notes   
reviewed. Results: 60% detected on CT scan, 34% on ultrasound scan and 6% on   
MRI. Underlying causes included 34% malignancy, 30% cirrhosis and 36% sepsis   
of which 51% due to biliary and pancreatic cause. A third of reports   
mentioned acute/chronic nature. 72% thrombus located within main portal   
vein, 24% branch vessels, 11% SMV and 4.7% within splenic vein. 36%   
suspected acute thrombosis in relation to previous imaging, half were   
anticoagulated with variable benefits. Spontaneous complete resolution at 6   
months in 13%, none had malignancy. Interestingly all of these were within   
main portal vein and measured <1 cm in length. Conclusion: Radiology reports   
should elaborate on age of the thrombus, site, size and extent, involvement   
of other vessels and presence of collaterals, varices and ascites apart from   
any underlying cause. Higher chance of spontaneous complete resolution of   
thrombosis in benign conditions such as grade I/II cirrhosis and sepsis   
where follow-up imaging in 3-6 months could be considered over immediate   
anticoagulation, if clinically appropriate. We have formulated an   
investigative pathway which will help in management.  
  
RECORD 466  
Enoxaparin prevents portal vein thrombosis  
Villa E. Marietta M. Zecchini R. Bernabucci V. Lei B. Vukotic R. Ferrari A.   
De Maria N. Schepis F. Fornaciari G. Schianchi S.  
Blood Transfusion (2012) 10 SUPPL. 4 (s42). Date of Publication: September   
2012  
  
Background portal vein thrombosis (PVT) is a frequent complication of   
advanced cirrhosis, occurring in 8-25% of patients and leading to severe   
clinical deterioration, decompensation and death. anticoagulation has never   
been prospectively tested for its prevention. Patients and Methods We   
designed a prospective randomized trial of anticoagulant therapy in advanced   
cirrhotic patients with the following end-points: primary, evaluation of   
efficacy in preventing PVT; secondary, assessment of safety, prevention of   
decompensation and/or survival (ISRCTN32383354, Eudract 2007-007890-22).   
Cirrhotic patients, Child B7-C10, were randomized to receive enoxaparin 4000   
IU/die or placebo for 12 months followed by 12 months observation. Doppler   
US was performed every three months and CT every six months to check for   
portal vein axis. PVT was considered as relevant when complete or involving   
more than 50% of PV diameter and symptomatic. We report the events of the 70   
enrolled patients (34 randomized to treatment and 36 to placebo) at   
completion of the 24 months study. Results No major bleeding was reported in   
the treatment arm. Only one patient was withdrawn from active arm because of   
thrombocytopenia (<10.000/mmc). During the 1-year study period, PVT (3   
complete, 3 partial) occurred in 6 of 36 (16.7%) patients on placebo and in   
none on enoxaparin (p=0.023 χ2). During follow up, 6 additional thrombotic   
events occurred, 3 in the placebo group and 3 in the active arm, 2-6 months   
after enoxaparin discontinuation (p=0.746). Decompensation occurred during   
the study period significantly more in placebo than in enoxaparin-treated   
patients [placebo 19 of 36 (52.7%) vs. 4/34 (11.7% ), p=0.0007]. Conclusions   
Survival was significantly better in enoxaparintreated patients (log rank   
0.019). Cox's regression analysis showed that enoxaparin treatment (HR   
0.098, 95% CI: 0.014- 0.697, p=0.020) and lower protein C levels (HR 0.984,   
95% CI: 0.858-0.981, p=0.012) were independently associated with a decreased   
risk of developing PVT.  
  
RECORD 467  
A data profile of phenotypic features in 72 Klinefelter syndrome (KFS) males  
Ranganath V. Rajangam S.  
International Journal of Human Genetics (2012) 12:3 (139-143). Date of   
Publication: September 2012  
  
Klinefelter syndrome phenotype is associated with hypogonadism and   
infertility that results from 47,XXY or 46,XY/47,XXY karyotype. Men with   
mosaic status show milder phenotype than those of non-mosaics. The present   
study aimed to report, a data profile on the observed phenotypic features in   
72 cytogenetically confirmed Klinefelter syndrome male gathered from duly   
filled proforma. The reported phenotype from the literature were categorized   
into 14 groups (highly arched palate, winged scapula, thin long fingers,   
flat feet, prognathism, liver cirrohsis, seizures, mental illness, penis,   
gonads, axillary hair growth, and pubic hair growth, presence of   
gynaecomastia and semen analysis). The calculated total number of the 14   
features multiplied for the 72 samples was 1,008. Of the 1,008 features   
(14X72), KFS male manifested only 16.56% of abnormal features (167/1,008).   
Scanty axillary hair growth (25%, 18), scanty pubic hair growth (26.38%,   
19), small sized penis (25%, 18), small sized gonads (55.56%, 40), presence   
of gynaecomastia (45.83%, 33) were of highest percentage. It was noticed   
that, for the entire sample of 72, the manifestation of the 14 categorised   
features was only 16.56%, irrespective of the karyotype; out of which, with   
47,XXY, the manifestation of the phenotypic features was observed to be   
highest (18.52%, 153/ 826). The findings confirmed the reported observations   
that in Klinefelter syndrome, there seemed to be a wide variability in the   
phenotype. © Kamla-Raj 2012.  
  
RECORD 468  
The transjugular intrahepatic portosystemic shunt in the treatment of portal   
hypertension: Current status  
Pomier-Layrargues G. Bouchard L. Lafortune M. Bissonnette J. Guérette D.   
Perreault P.  
International Journal of Hepatology (2012) Article Number: 167868. Date of   
Publication: 2012  
  
The transjugular intrahepatic portosystemic shunt (TIPS) represents a major   
advance in the treatment of complications of portal hypertension. Technical   
improvements and increased experience over the past 24 years led to improved   
clinical results and a better definition of the indications for TIPS.   
Randomized clinical trials indicate that the TIPS procedure is not a   
first-line therapy for variceal bleeding, but can be used when medical   
treatment fails, both in the acute situation or to prevent variceal   
rebleeding. The role of TIPS to treat refractory ascites is probably more   
justified to improve the quality of life rather than to improve survival,   
except for patients with preserved liver function. It can be helpful for   
hepatic hydrothorax and can reverse hepatorenal syndrome in selected cases.   
It is a good treatment for Budd Chiari syndrome uncontrollable by medical   
treatment. Careful selection of patients is mandatory before TIPS, and   
clinical followup is essential to detect and treat complications that may   
result from TIPS stenosis (which can be prevented by using covered stents)   
and chronic encephalopathy (which may in severe cases justify reduction or   
occlusion of the shunt). A multidisciplinary approach, including the   
resources for liver transplantation, is always required to treat these   
patients. © 2012 Gilles Pomier-Layrargues et al.  
  
RECORD 469  
Transjugular intrahepatic portosystemic shunt may be superior to   
conservative therapy for variceal rebleeding in cirrhotic patients with   
non-tumoral portal vein thrombosis: A hypothesis  
Qi X. Han G. He C. Yin Z. Zhang H. Wang J. Xia J. Cai H. Yang Z. Bai M. Wu   
K. Fan D.  
Medical Science Monitor (2012) 18:8 (HY37-HY41). Date of Publication: August   
2012  
  
The presence of occlusive portal vein thrombosis (PVT) greatly changes the   
natural history of liver cirrhosis, because it not only significantly   
increases the incidence of variceal rebleeding but also negatively   
influences the survival. However, due to the absence of strong evidence, no   
standard treatment algorithm for the secondary prophylaxis of variceal   
bleeding in cirrhotic patients with non-tumoral PVT has been established.   
Previous randomized controlled trials have demonstrated that transjugular   
intrahepatic portosystemic shunt (TIPS) can significantly decrease the   
incidence of variceal rebleeding in cirrhotic patients without PVT, compared   
with conservative therapy (i.e., endoscopic plus pharmacological therapy).   
Further, several large cohort studies have confirmed that TIPS can   
effectively prevent variceal rebleeding in cirrhotic patients with   
non-tumoral PVT. On the other hand, TIPS can facilitate recanalizing the   
thrombosed portal vein by endovascular manipulations, even in the presence   
of cavernous transformation of the portal vein (CTPV). More importantly,   
successful TIPS insertions can maintain the persistent portal vein patency,   
and avoid thrombus extension into the portal venous system. By comparison,   
anticoagulation therapy can achieve portal vein recanalization only in   
patients with partial PVT, but not in those with occlusive PVT or CTPV, and   
the use of anticoagulants may aggravate the risk of variceal bleeding in   
cirrhotic patients with a history of variceal bleeding. Collectively, we   
hypothesize that TIPS may be superior to conservative therapy for the   
prevention of variceal rebleeding in cirrhotic patients with non-tumoral   
PVT. Randomized controlled trials should be conducted to evaluate the   
survival benefit of TIPS in these patients. © Med Sci Monit, 2012.  
  
RECORD 470  
portal vein thrombosis after total knee replacement: a case report.  
Martin G. Rashid A. Abdul-Jabar H.B. Jennings S.  
Journal of orthopaedic surgery (Hong Kong) (2012) 20:2 (276-278). Date of   
Publication: Aug 2012  
  
We present a 74-year-old woman who developed a portal vein thrombosis   
following an elective total knee replacement. She had atrial fibrillation   
for which she was taking warfarin for anticoagulation. Seven days prior to   
surgery, she was instructed to discontinue warfarin and replace it with   
prophylactic low-molecular-weight heparin. On postoperative day 1, routine   
blood tests revealed deranged hepatic synthetic function, despite standard   
anticoagulation management. Doppler ultrasonography confirmed a portal vein   
thrombosis. She was treated with therapeutic doses of low-molecular-weight   
heparin until her international normalised ratio reached therapeutic levels.   
Her liver function results had normalised 2 weeks later. Portal vein   
thrombosis is a potentially fatal complication that is reversible if   
identified and treated early.  
  
RECORD 471  
Management of anticoagulation for portal vein thrombosis in individuals with   
cirrhosis: A systematic review  
Huard G. Bilodeau M.  
International Journal of Hepatology (2012) Article Number: 672986. Date of   
Publication: 2012  
  
Non-neoplastic portal vein thrombosis (PVT) is an increasingly recognized   
complication of liver cirrhosis. It is often diagnosed fortuitously and can   
be either partial or complete. The clinical significance of PVT is not   
obvious except in some situations such as when patients are on the waiting   
list for liver transplantation. The only known therapy is anticoagulation   
which has been shown to permit the disappearance of thrombosis and to   
prevent further extension. anticoagulation is a challenging therapy in   
individuals with liver cirrhosis because of the well-recognized coagulation   
abnormalities observed in that setting and because of the increased risk of   
bleeding, especially from gastrointestinal tract caused by portal   
hypertension. We herein review the current knowledge on that topic in order   
to highlight the advantages and disadvantages of the currently proposed   
therapeutic attitudes in face of the diagnosis of PVT in individuals with   
cirrhosis. © Copyright 2012 Genevive Huard and Marc Bilodeau.  
  
RECORD 472  
Impact of anticoagulation on outcomes in non-malignant and non-cirrhotic   
portal vein thrombosis: A retrospective observational study  
Hall T. Bilku D. Metcalfe M. Rajesh A. Dennison A. Garcea G.  
HPB (2012) 14 SUPPL. 2 (548). Date of Publication: July 2012  
  
Introduction: No definitive evidence exists regarding the treatment of acute   
portal vein thrombosis (PVT). The natural history is also poorly understood.   
Treatment modalities described include conservative management,   
anticoagulation, thrombolysis and thrombectomy. This observational study   
examines the impact of anticoagulation on PVT. Methods: The electronic   
radiology database was searched with keywords 'portal vein' and   
'thrombosis'. Relevant patient notes and imaging were reviewed to collect   
data from those with acute PVT. The primary end point was portal vein   
recanalisation. Secondary outcome measures were morbidity and the   
development of portal hypertension and its sequelae (including variceal   
bleeding). Data from patients with PVT in the context of cirrhosis,   
malignancy or liver transplant were excluded. Results: Twenty two patients   
were included in the study. 45.5% were male and median age was 58.5 years   
(range 30-89). Acute pancreatitis was implicated as the precipitating cause   
in 50% of patients. 41.0% of patients were treated with anticoagulation. The   
remainder received no intervention. Partial or complete recanalisation of   
the portal vein occurred in 81.8% of anticoagulated patients and 37.5% of   
the non-treatment group. 5 patients died, 1 was secondary to an intracranial   
haemorrhage whilst anticoagulated and another who was not treated secondary   
developed small bowel ischaemia and peritonitis. The remaining 3 died from   
the underlying pathology. Conclusion: Spontaneous resolution of acute portal   
vein thrombosis is uncommon. Early anticoagulation results in a higher rate   
of recanalisation with minimal associated morbidity when compared with no   
treatment.  
  
RECORD 473  
portal vein thrombosis, cirrhosis, and liver transplantation  
Francoz C. Valla D. Durand F.  
Journal of Hepatology (2012) 57:1 (203-212). Date of Publication: July 2012  
  
portal vein thrombosis is not uncommon in candidates for transplantation.   
Partial thrombosis is more common than complete thrombosis. Despite careful   
screening at evaluation, a number of patients are still found with   
previously unrecognized thrombosis per-operatively. The objective is to   
recanalize the portal vein or, if recanalization is not achievable, to   
prevent the extension of the thrombus so that a splanchnic vein can be used   
as the inflow vessel to restore physiological blood flow to the allograft.   
anticoagulation during waiting time and transjugular intrahepatic   
portosystemic shunt (TIPS) are two options to achieve these goals. TIPS may   
achieve recanalization in patients with complete portal vein thrombosis.   
However, a marked impairment in liver function, which is a characteristic   
feature of most candidates for transplantation, may be a contraindication   
for TIPS. Importantly, the MELD score is artificially increased by the   
administration of vitamin K antagonists due to prolonged INR. When patency   
of the portal vein and/or superior mesenteric vein is not achieved, only   
non-anatomical techniques (renoportal anastomosis or cavoportal   
hemitransposition) can be performed. These techniques, which do not fully   
reverse portal hypertension, are associated with higher morbidity and   
mortality risks. Multivisceral transplantation including the liver and small   
bowel needs to be evaluated. In the absence of prothrombotic states that may   
persist after transplantation, there is no evidence that pre-transplant   
portal vein thrombosis justifies long term anticoagulation   
post-transplantation, provided portal flow has been restored through   
conventional end-to-end portal anastomosis. © 2012 European Association for   
the Study of the Liver. Published by Elsevier B.V. All rights reserved.  
  
RECORD 474  
Degree of portal vein thrombosis Might Be Associated With Recanalization   
During anticoagulation  
Qi X. Han G. Fan D.  
Clinical Gastroenterology and Hepatology (2012) 10:7 (820). Date of   
Publication: July 2012  
  
RECORD 475  
Efficacy and Safety of anticoagulation on Patients With cirrhosis and Portal   
Vein Thrombosis  
Delgado M.G. Seijo S. Yepes I. Achécar L. Catalina M.V. García-Criado T.   
Abraldes J.G. de la Peña J. Bañares R. Albillos A. Bosch J. García-Pagán   
J.C.  
Clinical Gastroenterology and Hepatology (2012) 10:7 (776-783). Date of   
Publication: July 2012  
  
Background & Aims: portal vein thrombosis (PVT) is a frequent event in   
patients with cirrhosis; it can be treated with anticoagulants, but there   
are limited data regarding safety and efficacy of this approach. We   
evaluated this therapy in a large series of patients with cirrhosis and   
non-neoplastic PVT. Methods: We analyzed data from 55 patients with   
cirrhosis and PVT, diagnosed from June 2003 to September 2010, who received   
anticoagulant therapy for acute or subacute thrombosis (n = 31) or   
progression of previously known PVT (n = 24). Patients with cavernomatous   
transformation were excluded. Thrombosis was diagnosed, and recanalization   
was evaluated by using Doppler ultrasound, angio-computed tomography, and/or   
angio-magnetic resonance imaging analyses. Results: Partial or complete   
recanalization was achieved in 33 patients (60%; complete in 25). Early   
initiation of anticoagulation was the only factor significantly associated   
with recanalization. Rethrombosis after complete recanalization occurred in   
38.5% of patients after anticoagulation therapy was stopped. Despite similar   
baseline characteristics, patients who achieved recanalization developed   
less frequent liver-related events (portal hypertension-related bleeding,   
ascites, or hepatic encephalopathy) during the follow-up period, but this   
difference was not statistically significant (P = .1). Five patients   
developed bleeding complications that were probably related to   
anticoagulation. A platelet count <50 × 109/L was the only factor   
significantly associated with higher risk for experiencing a bleeding   
complication. There were no deaths related to anticoagulation therapy.   
Conclusions: anticoagulation is a relatively safe treatment that leads to   
partial or complete recanalization of the portal venous axis in 60% of   
patients with cirrhosis and PVT; it should be maintained indefinitely to   
prevent rethrombosis. © 2012 AGA Institute.  
  
RECORD 476  
Increased platelet activation in cirrhosis via oxidative stress  
Celikbilek M. Dogan S. Gürsoy S. Güven K.  
Liver International (2012) 32:6 (1029). Date of Publication: July 2012  
  
RECORD 477  
Prospective evaluation of anticoagulation and transjugular intrahepatic   
portosistemic shunt for the management of portal vein thrombosis in   
cirrhosis  
Senzolo M. Sartori T.M. Rossetto V. Burra P. Cillo U. Boccagni P. Gasparini   
D. Miotto D. Simioni P. Tsochatzis E. Burroughs K.A.  
Liver International (2012) 32:6 (919-927). Date of Publication: July 2012  
  
Background: There is no established management algorithm for portal vein   
thrombosis (PVT) in cirrhotic patients. The aim of our study was to   
prospectively evaluate anticoagulation and transjugular intrahepatic   
portosystemic shunt (TIPS) to treat PVT. Methods: Cirrhotics with   
non-malignant PVT were included. Low weight molecular heparin   
anticoagulation was considered in all; TIPS was indicated if thrombosis   
progressed or anticoagulation was contraindicated. Patients who were not   
anticoagulated nor received TIPS served as controls. Results: Fifty-six   
patients (of whom 21 controls) were included. PVT was occlusive in 11/35,   
with extension to the superior mesenteric or splenic vein in 13/35. In the   
study group 33 patients were anticoagulated, with a recanalization rate of   
36% (12/33) compared with 1/21 among controls. A time interval between   
appearance of thrombosis and anticoagulation < 6 months predicted chance of   
repermeation. Thrombus progression occurred in 15/21 non anticoagulated   
patients and in 5/33 anticoagulated patients (P < 0.001). TIPS was placed in   
six patients. There were five variceal bleedings and two intestinal venous   
ischaemia episodes in the control group, compared with one variceal bleeding   
episode in the study group. Conclusions: In cirrhotics with PVT, a treatment   
algorithm using anticoagulation and TIPS achieves a good chance of complete   
repermeation, reduces portal hypertensive complications, and decreases the   
rate of thrombosis progression. © 2012 John Wiley & Sons A/S.  
  
RECORD 478  
anticoagulation therapy prevents portal-splenic vein thrombosis after   
splenectomy with gastroesophageal devascularization  
Lai W. Lu S.-C. Li G.-Y. Li C.-Y. Wu J.-S. Guo Q.-L. Wang M.-L. Li N.  
World Journal of Gastroenterology (2012) 18:26 (3443-3450). Date of   
Publication: July2012  
  
AIM: To compare the incidence of early portal or splenic vein thrombosis   
(PSVT) in patients treated with irregular and regular anticoagulantion after   
splenectomy with gastroesophageal devascularization.METHODS: We   
retrospectively analyzed 301 patients who underwent splenectomy with   
gastroesophageal devascularization for portal hypertension due to cirrhosis   
between April 2004 and July 2010. Patients were categorized into group A   
with irregular anticoagulation and group B with regular anticoagulation,   
respectively. Group A (153 patients) received anticoagulant mono-therapy for   
an undesignated time period or with aspirin or warfarin without   
low-molecular-weight heparin (LMWH) irregularly. Group B (148 patients)   
received subcutaneous injection of LMWH routinely within the first 5 d after   
surgery, followed by oral warfarin and aspirin for one month regularly. The   
target prothrombin time/international normalized ratio (PT/INR) was   
1.25-1.50. Platelet and PT/INR were monitored. Color Doppler imaging was   
performed to monitor PSVT as well as the effectiveness of thrombolytic   
therapy. RESULTS: The patients' data were collected and analyzed   
retrospectively. Among the patients, 94 developed early postoperative mural   
PSVT, including 63 patients in group A (63/153, 41.17%) and 31 patients in   
group B (31/148, 20.94%). There were 50 (32.67%) patients in group A and 27   
(18.24%) in group B with mural PSVT in the main trunk of portal vein. After   
the administration of thrombolytic, anticoagulant and anti-aggregation   
therapy, complete or partial thrombus dissolution achieved in 50 (79.37%) in   
group A and 26 (83.87%) in group B.CONCLUSION: Regular anticoagulation   
therapy can reduce the incidence of PSVT in patients who undergo splenectomy   
with gastroesophageal devascularization, and regular anticoagulant therapy   
is safer and more effective than irregular anticoagulant therapy. Early and   
timely thrombolytic therapy is imperative and feasible for the prevention of   
PSVT. © 2012 Baishideng. All rights reserved.  
  
RECORD 479  
anticoagulation for Cirrhotic portal vein thrombosis: Bold, Brave, and   
Possibly Beneficial  
Campbell S. Lachlan N.J.  
Clinical Gastroenterology and Hepatology (2012) 10:7 (784-785). Date of   
Publication: July 2012  
  
RECORD 480  
anticoagulation in cirrhosis  
Villa E. De Maria N.  
Liver International (2012) 32:6 (878-879). Date of Publication: July 2012  
  
RECORD 481  
Transradial approach for transcatheter selective superior mesenteric artery   
urokinase infusion therapy in patients with acute extensive portal and   
superior mesenteric vein thrombosis  
Wang Y. Wang M.-Q. Liu F.-Y. Wang Z.-J. Duan F. Song P.  
National Medical Journal of China (2012) 92:21 (1448-1452). Date of   
Publication: 5 Jun 2012  
  
Objective: To evaluate the feasibility and efficacy of urokinase infusion   
therapy via a transradial approach for transcatheter superior mesenteric   
artery (SMA) in patients with acute extensive portal and superior mesenteric   
venous thrombosis. Methods: During a period of 8 years, 47 patients with   
acute extensive thrombosis of portal vein (PV) and superior mesenteric veins   
(SMV) received urokinase infusion therapy by transcatheter selective SMA via   
radial artery. Their mean age was 44±13 years (range: 19-65). Through radial   
sheath, a 5F catheter was placed into SMA and subsequently the infusion of   
urokinase was given for 5-11 days (mean: 7.1±2.5). Adequate anticoagulation   
was initiated during treatment, throughout hospitalization and   
post-discharge. Follow-up contrast-enhanced computed tomography (CT) was   
performed in each patient every 3 days and before the removal of infusion   
catheter. Termination of urokinase infusion therapy was decided on the basis   
of clinical and radiographic findings. Results: Technical success was   
achieved in all patients. Two patients had worsening abdominal pain,   
developed the signs of peritonitis at 24 hours after interventional   
treatment and underwent eventual laparotomy with the resection of necrotic   
bowel. Substantial clinical improvement was observed in 45 (95.7%) of them   
after the procedure. Minor complications at the radial puncture site were   
observed in 7 patients (14.9%) and infusion therapy continued. Follow-up CT   
scans at pre-discharge demonstrated a nearly complete disappearance of   
PV-SMV thrombosis in 29 patients (64.4%) and partial recanalization of   
PV-SMV thrombosis in 16 patients(35.6%). They were discharged at 9-20 days   
(mean: 12±6) post-admission. The mean post-discharge duration of follow-up   
was 48±20 months. Recurrent episodes of PV and SMV thrombosis were observed   
in 2(4.4%) patients at 6 months and 5 years respectively post-discharge and   
they were treated successfully with urokinase infusion. Conclusion: The   
transcatheter SMA urokinase infusion therapy via a transradial approach for   
plus anticoagulation is both safe and effective for the management of   
patients with acute extensive PV-SMV thrombosis.  
  
RECORD 482  
Prediction of the therapeutic effects of anticoagulation for recent portal   
vein thrombosis: A novel approach with contrast-enhanced ultrasound  
Maruyama H. Ishibashi H. Takahashi M. Shimada T. Kamesaki H. Yokosuka O.  
Abdominal Imaging (2012) 37:3 (431-438). Date of Publication: June 2012  
  
Objective: To examine whether intra-thrombus enhancement on   
contrast-enhanced sonograms can predict the recanalization by   
anticoagulation for recent portal thrombosis. Methods: This prospective   
study included 10 patients with a recent portal thrombosis and 20 controls   
(10 cirrhosis patients and 10 healthy subjects, all without thrombosis). The   
diagnosis of thrombosis was based on clinical and ultrasound findings.   
Pre-anticoagulation intra-thrombus enhancement on the contrast-enhanced   
sonogram was examined with respect to the postanticoagulation results or   
portal enhancement in controls. Results: Complete recanalization was   
obtained in 4 patients with positive intra-thrombus enhancement. However, in   
4 other patients who had a thrombosis showing positive enhancement   
concurrent with one showing negative enhancement, anticoagulation   
recanalized the former and failed to recanalize the latter. Mean onset time   
of contrast enhancement measured from the beginning of hepatic arterial   
enhancement was significantly longer in the thrombus (6.6 ± 4.3 s, 3-16 s)   
than in the portal vein of controls (cirrhosis, 4.3 ± 1.4 s, 2-8 s, P =   
0.0035; healthy subjects, 2.4 ± 0.6 s, 1-3 s, P < 0.0001). anticoagulation   
failed to achieve recanalization in 2 patients with negative intra-thrombus   
enhancement. Sensitivity and specificity of contrast enhancement for the   
prediction of post-treatment recanalization was 100%. Conclusions:   
Intra-thrombus positive enhancement demonstrated on contrast-enhanced   
sonograms has promise as a successful predictor of recanalization for the   
recent portal thrombosis. © Springer Science+Business Media, LLC 2011.  
  
RECORD 483  
Emergency anticoagulation treatment for cirrhosis patients with portal vein   
thrombosis and acute variceal bleeding  
Maruyama H. Takahashi M. Shimada T. Yokosuka O.  
Scandinavian Journal of Gastroenterology (2012) 47:6 (686-691). Date of   
Publication: June 2012  
  
Objective: To determine the safety and efficacy of anticoagulation treatment   
for portal vein thrombosis in cirrhosis patients with acute variceal   
bleeding, with patient eligibility determined by contrast ultrasonography   
findings. Materials and methods: This prospective study included 23   
consecutive cirrhosis patients (63.8 ± 11.8 years old, 12 males and 11   
females) with emergency admission for acute variceal bleeding with or   
without portal vein thrombus. Eligibility for anticoagulation treatment was   
determined by positive intra-thrombus enhancement on contrast   
ultrasonography (perflubutane microbubble agent, 0.0075 mL/kg) performed   
before endoscopy. Low-molecular-weight heparin was administered after   
hemostasis was achieved by band ligation. Repeated band ligation or   
injection sclerotherapy combined with argon plasma coagulation was performed   
for variceal disappearance. Results: Hemostasis was achieved in all 10   
patients with active bleeding. Five of these patients had portal vein   
thrombus, and all showed positive intra-thrombus enhancement on contrast   
ultrasonography. anticoagulation treatment of these five patients resulted   
in complete recanalization of the portal vein within 2-11 days. There were   
no significant differences in the number of endoscopic treatment sessions or   
the length of hospital stay between the groups with and without thrombosis,   
and no complications including rebleeding were reported. Long term, none of   
the patients who continued oral anticoagulation treatment had recurrence of   
thrombosis (4/5). Variceal recurrence occurred only in the non-thrombosis   
group (2/18) during the follow-up period (median: 351 days). Conclusions:   
Early anticoagulation treatment in cirrhosis patients with portal vein   
thrombosis and acute variceal bleeding may be safe, tolerated, and effective   
in cases with positive intra-thrombus enhancement on contrast   
ultrasonography. © 2012 Informa Healthcare.  
  
RECORD 484  
Recurrent cerebral and abdominal thromboses and pulmonary embolism (PE) in a   
non-tranfusion dependent patient with paroxysmal nocturnal hemoglobinuria.   
Effectiveness of eculizumab treatment  
Carbone M. Luzzatto L. Rossi G.  
Haematologica (2012) 97 SUPPL. 1 (417-418). Date of Publication: 1 Jun 2012  
  
Background. Paroxysmal nocturnal hemoglobulinuria (PNH) is a rare, genetic,   
hematopoietic stem cell disorder characterized by chronic uncontrolled   
terminal complement activation, causing hemolysis, platelet activation, and   
inflammation, and ultimately leading to serious morbidities. Thromboembolism   
(TE) is among the most dangerous complications of PNH. Aims. To present a   
case highlighting severe recurrent TE occurring in a non-transfusion   
dependent PNH patient, despite prophylactic warfarin anticoagulation.   
Results. In July 2004, a 36-year-old woman in her second pregnancy presented   
with isolated low platelet count (37x109/L). Routine diagnostic work-up was   
unremarkable include ing tests recommended to detect causes of secondary   
thrombocytopenia. LDH level was between 1 and 1.5 ULN, a potential clue to   
an early diagnostic workup to exclude PNH Bone marrow aspiration was normal   
and idiopathic thrombocytopenic purpura was diagnosed. Despite treatment   
with high-dose steroids, intravenous immunoglobulin and cyclosporin A her   
platelet count did not improve. At term, a Caesarean section was performed   
along with a splenectomy. Following a massive post-partum hemorrhage   
requiring packed RBC transfusions the patient was discharged in September   
2004 with Hb 11.6 g/dL, but still low platelets (35x109/L ). Two years later   
the patient was hospitalized in the department of neurology of another   
hospital after experiencing drowsiness, headache and transient aphasia.   
Cerebral CT scan revealed thrombosis of the sagittal sinus, moreover hepatic   
enlargement, ascites, due to Budd Chiari syndrome ,along with asymptomatic   
PE were detected and warfarin treatment initiated. The patient was diagnosed   
with thrombophilia of unknown origin. Platelet count ranged between 50 and   
100x109/L. During follow-up, a series of diagnostic tests, including bone   
marrow biopsy (cellularity 15%), tumor markers and markers of congenital and   
acquired thrombophilia were performed. PNH was diagnosed by flow cytometry   
(99% GPI-linked CD14 and CD55 defect). The patient developed hepatic vein   
thrombosis in 2007 and was hospitalized again in January 2009 with   
persistent abdominal pain while on warfarin and with INR within the   
therapeutic range. D-dimer level was 5290 ng/ml and LDH 2N , Abdominal   
Doppler US revealed massive portal vein thrombosis with cavernoma of the   
liver. Eculizumab therapy was started in June 2009, as recommended in   
patients with recurrent abdominal and cerebral thromboses (Brodsky, Blood   
2011), and because oral warfarin had proven ineffective. The subsequent   
clinical course was unremarkable with no further reports of abdominal pain,   
or TE and normalization of all hematological values (LDH: 159-364 IU/L; Hb:   
12.6 g/dL; platelets: 231,000/mm3). D-dimer level range between N and   
2N.Conclusions: We suggest that in patients who have a history of   
potentially devastating recurrent venous thrombosis eculizumab treatment   
must be considered, even when haemolysis is not serious enough to require   
blood transfusion. In this particular patient, who had experienced at least   
3 major thrombotic events over a 5 year period, there has been no recurrence   
since eculizumab was started over 2 years ago.  
  
RECORD 485  
Pylephlebitis and acute mesenteric ischemia in a young man with inherited   
thrombophilia and suspected foodborne illness  
Pradka S.P. Trankiem C.T. Ricotta J.J.  
Journal of Vascular Surgery (2012) 55:6 (1769-1772). Date of Publication:   
June 2012  
  
We report on a young man who developed complicated pylephlebitis after   
foodborne illness. Despite antibiotics and resection of the focus of   
infectious colitis, he developed extensive small bowel infarction. He was   
treated with anticoagulation, local thrombolytic infusion, and resection of   
irreversibly ischemic small bowel. Thrombophilia workup demonstrated   
heterozygosity for factor V Leiden and the prothrombin G20210A mutation. The   
complications of pylephlebitis can be minimized by using systemic   
anticoagulation, thrombectomy, and/or local thrombolytic infusion along with   
antibiotics and surgical management of the infection. Evaluation for   
thrombophilic states should be considered, particularly if a patient does   
not respond to initial therapy. © 2012 Society for Vascular Surgery.  
  
RECORD 486  
Non cirrhotic portal vein thrombosis. Diagnosis and therapeutic algorithm  
Catalina-Rodríguez M.V. De García-Fernández C.P.  
Medicine (Spain) (2012) 11:12 (728-732). Date of Publication: June 2012  
  
Vascular disorders of the liver represent a heterogeneous group of diseases   
characterized by the frequent presence of a prothrombotic condition. Primary   
Budd-Chiari syndrome is characterized by the obstruction of the hepatic vein   
drainage at any topographical level. Clinical presentation is very   
heterogeneous ranging from the complete absence of symptoms to fulminant   
liver failure, depending on thrombosis extension, velocity of the appearance   
and on the development of compensatory mechanisms like formation of   
collateral vessels. portal vein thrombosis is usually the consequence of a   
combination of local and systemic risk factors, and its manifestations   
differ in acute or chronic thrombosis. In both cases diagnosis is based on   
the demonstration of the venous obstruction usually by imaging like   
abdominal ultrasound or CT scan. The main goal of therapy is to provide   
vessel recanalization and/or decompression of the affected vascular bed.  
  
RECORD 487  
Primary Budd-Chiari syndrome. Diagnosis and treatment algorithm  
Catalina-Rodríguez M.V. Díaz-Fontena F.  
Medicine (Spain) (2012) 11:12 (723-727). Date of Publication: June 2012  
  
Vascular disorders of the liver represent a heterogeneous group of diseases   
characterized by the frequent presence of a prothrombotic condition. Primary   
Budd-Chiari syndrome is characterized by the obstruction of the hepatic vein   
drainage at any topographical level. Clinical presentation is very   
heterogeneous ranging from the complete absence of symptoms to fulminant   
liver failure, depending on thrombosis extension, velocity of the appearance   
and on the development of compensatory mechanisms like formation of   
collateral vessels. portal vein thrombosis is usually the consequence of a   
combination of local and systemic risk factors, and its manifestations   
differ in acute or chronic thrombosis. In both cases diagnosis is based on   
the demonstration of the venous obstruction usually by imaging like   
abdominal ultrasound or CT scan. The main goal of therapy is to provide   
vessel recanalization and/or decompression of the affected vascular bed.  
  
RECORD 488  
Clinical and radiographic presentation of superior mesenteric vein   
thrombosis in Crohn's disease: A single center experience  
Kopylov U. Amitai M.M. Lubetsky A. Eliakim R. Chowers Y. Ben-Horin S.  
Journal of Crohn's and Colitis (2012) 6:5 (543-549). Date of Publication:   
June 2012  
  
Background: Mesenteric vein thrombosis (MVT) is a rare and frequently   
underdiagnosed complicationof Crohn's disease (CD). This study describes the   
clinical and radiological characteristics of CD /patients with superior   
mesenteric vein thrombosis (MVT) diagnosed by CT/MRI. Patients and methods:   
The database of Crohn's disease patients treated in Sheba Medical Center   
between 2005-2010 was searched for MVT diagnosis. Imaging studies of   
identified patients were retrieved and reviewed by an experienced abdominal   
radiologist. MVT was defined by superior mesenteric vein obliteration and/or   
thrombus in the vessel lumen on abdominal imaging. The clinical and   
radiologic data of these patients were collected from the medical records.   
Results: MVT was demonstrated in 6/460 CD patients. Five patients had   
stricturing disease, and one patient had a combined fistulizing and   
stricturing disease phenotype. All patients had small bowel disease, but 3/6   
also had colonic involvement. No patient had a prior thromboembolic history   
or demonstrable hypercoagulability. One patient had an acute SMV thrombus   
demonstrable on CT scanning, the remaining patients showed an obliteration   
of superior mesenteric vein. Two patients received anticoagulation upon   
diagnosis of thrombosis. No subsequent thromboembolic events were recorded.   
Conclusions: The incidence of mesenteric vein thrombosis is likely to be   
underestimated in patients with Crohn's disease. Both CT and MRI imaging   
demonstrate the extent of enteric disease and coincident SMV thrombosis. In   
our cohort, thrombosis was associated with stricturing disease of the small   
bowel. The clinical impact of SMV thrombosis and whether anticoagulation is   
mandatory for all of these patients remains to be determined. © 2011   
European Crohn's and Colitis Organisation.  
  
RECORD 489  
Is long term anticoagulation needed to prevent rethrombosis following LT in   
cirrhotic patients who had portal vein thrombosis prior to LT?  
Francoz C. Dondero F. Houssel P. Dokmak S. Belghiti J. Durand F.  
Liver Transplantation (2012) 18 SUPPL. 1 (S102). Date of Publication: May   
2012  
  
portal vein thrombosis (PVT) is not uncommon in cirrhosis as it is found in   
about 15% of candidates for LT. Several reports suggest that preLT   
anticoagulation may help to perform anatomic portal anastomoses. In those   
patients, there is a potential risk of rethrombosis. In the early post   
operative period, recurrence of PVT generally leads to graft failure. In   
contrast, delayed thrombosis does not lead to graft failure but results in   
portal hypertension. Theoretically, post LT anticoagulation may prevent the   
recurrence of PVT, however the risk of rethrombosis, without   
anticoagulation, has never been investigated. The aim of this study was to   
assess the risk of rethrombosis after LT in a cohort of patients who had   
preLT PVT. We retrospectively studied 91 patients who had pretransplant PVT   
and were transplanted between 1990 and 2011 in a single institution.Patients   
with myeloproliferative disorders or antiphospholipid syndrome were   
excluded. Until 2001, patients with PVT did not receive anticoagulation.   
After 2001, based on the good results of anticoagulation in non cirrhotic   
population with PVT, patients were systematically treated with anticoagulant   
on the waiting list in order to achieve recanalization and/or prevent   
extension. After LT, by contrast, none of the patients received specific   
anticoagulation. At time of surgical procedure, 54 of 91 patients still had   
partial PVT which was successfully treated by thrombectomy. In all these   
patients, portal anastomosis was performed except in 2 who had   
mesentericoportal anastomosis. Despite the absence of anticoagulation, no   
patients developed PVT in the long term. Eight of 91 had no residual portal   
vein nor mesenteric vein and only a cavernoma. In these patients, renoportal   
anastomosis has been done in 5, and caval transposition in 3. Three-month   
mortality rates were 94% in anatomic portal anastomoses and only 50% after   
renoportal anastomosis or caval transposition. Our results show that postLT   
anticoagulation should not be justified in recipients who had PVT before LT   
and in whom anatomic portal anastomosis can be perform. These data enhance   
the hypothesis suggesting that the decrease in portal flow plays a major   
role in the physiopathology of PVT in cirrhosis and that provided adequate   
portal flow is restored to the graft, the risk of rethrombosis is low even   
in the absence of specific anticoagulation.  
  
RECORD 490  
Major digestive bleeding secondary to chronic portal venous thrombosis as   
clinical onset of previously unknown hepatic cirrhosis complicated by   
hepatic carcinoma  
Arioli D. Bassi F. Pileri F. Leone M.C. Trenti C. Galimberti D. Camellini L.   
Negri E. Iori I. Casali A.  
Italian Journal of Medicine (2012) 6:1 SUPPL. 1 (5-6). Date of Publication:   
May 2012  
  
Introduction Hepatocarcinoma (HCC) is a complication of hepatic cirrhosis   
(HC) generally detected in early stage due to Ultrasonographic (US)   
surveillance in hepatopathic patients. It can be treated effectively with   
well tolerated loco-regional treatment. In advanced stages of the disease,   
HCC may be complicated by portal vein thrombosis (PVT) that represents the   
limiting element of the treatment and, therefore, the more devastating   
prognostic element. Case reports We present two cases of major digestive   
bleeding that led us to diagnose chronic PVT due to silent HC complicated by   
multifocal HCC. In both cases the diagnostic role of Doppler US study is   
emphasized such as the importance of alpha fetoprotein requested in the   
correct scenario. In both cases patients underwent endoscopic variceal   
ligation (VL) regardless to the chance of etiologic treatment of HCC and   
anticoagulant therapy was attempted. Just in the first case it was possible   
to begin Sorafenib due to the better Child Class present at the diagnosis   
(A5 versus B8). Conclusion Nowadays despite US surveillance in hepathopathic   
patients lets generally the early diagnosis of HCC in pre-clinical phase, it   
is still possible that digestive bleeding secondary to chronic PVT   
represents the clinical onset of previously unknown HC complicated by HCC.   
In this scenario anticoagulant therapy can be considered just in case of   
preserved liver function and should be individualized. Unlike the   
anticoagulation, VL is codified therapy and shouldn't be denied to patients   
since it appears to significatively prolong surveillance.  
  
RECORD 491  
Splenoportal vein thrombosis in sepsis by severe diverticulitis  
Gnocchi M. Labò P. Ratti D. Magnani L.  
Italian Journal of Medicine (2012) 6:1 SUPPL. 1 (70). Date of Publication:   
May 2012  
  
Introduction Many medical conditions can lead to portal vein thrombosis   
(PVT); extra hepatic PVT has high clinical significance and prevalence. In a   
swedish study about 10% of PVT is caused by major abdominal infection or   
inflammatory disease. Clinical case A 32 year old male came to our ED for   
fever, vomiting, diarrhoea and abdominal pain. He was hospitalized with the   
suspicious of acute gastroenteritis. Empirical antibiotic therapy   
(ciprofloxacin and amoxicillin/clavulanate) was started and then improved   
with metronidazole after blood culture results, positive for anaerobic   
bacteria. Laboratory findings showed high activated Protein C, moderate   
leukocytosis, mild hepatic function impairment. Abdominal ultrasonography   
showed unattended probable splenoportal vein thrombosis, initial   
hepatosplenomegaly, colic inflammation signs, with oedema at the sigma   
tract. CT scan confirmed wide splenoportal thrombosis and severe sigma   
diverticulitis. EPBM was started and after 1 month US showed partial   
splenoportal thrombosis resolution and hepatosplenomegaly normalization.   
Discussion PVT has a wide spectrum of clinical manifestation including liver   
function impairment, splenomegaly and abdominal pain, but the most common is   
variceal bleeding in portal hypertension. PVT has to be detected by   
efficient imaging techniques such Doppler US. anticoagulation is recommended   
in acute PVT, because spontaneous repermeation is uncommon. With our case we   
would stress to suspect PVT in severe abdominal infection and to detect it   
by accurate Doppler US.  
  
RECORD 492  
A case of septic portal vein thrombosis: the role of bacteroides fragilis  
Trenti C. Arioli D. Negri E. Galimberti D. Iori I.  
Italian Journal of Medicine (2012) 6:1 SUPPL. 1 (141-142). Date of   
Publication: May 2012  
  
Introduction portal vein thrombosis (PVT) without hepatic chyrrosis may be   
related to infective causes. Case report We report the case of an 85 years   
old man admitted to the Emergency Unit for hyperpirexia associated with   
right upper quadrant and epigastric pain. He had a clinical history of COPD   
(GOLD III) and hypertension; past gastric resection for early gastric cancer   
and right emycolectomy for cancer, pT2 N0. On physical examination the   
abdomen was tender in the upper quadrant and hepigastric regions. Clinical   
signs (polypnea, increased heart rate, temperature >38°C) of SIRS were   
present as well as laboratory findings (CRP 19 mg/dl, procalcytonin 27   
ng/ml) of sepsis. US examination showed partial PVT, the pathogenesis of   
which had to be defined. There were no laboratory nor instrumental (CT scan,   
gastric and colonscopy) signs of an underlying hepatic chyrrosis nor of a   
neoplastic disease relapse. No congenital or acquired thrombophylia was   
present on screening except for blood coltures positive for Bacteroides   
Fragilis infection. This made us formulate a diagnosis of partial PVT in   
course of Bacteroides sepsis. Anthibiotic treatment as well as   
anticoagulation was started with a favourable clinical outcome on follow-up.   
Conclusions In Literature a significant association between PVT and   
Bacteroides Fragilis bacteriemia is reported: the pathogenetic mechanism   
might be a transient hypercoagulability state. Such an association is so   
strong that some Authors recommend a systematic screening for PVT in case of   
proven Bacteroides Fragilis bacteriemia.  
  
RECORD 493  
The effect of post-operative intravenous heparin infusion on simultaneous   
kidney-pancreas transplant outcomes  
Alabbasi A. Martin P. Block M. McAlister V. Luke P. Sener A.  
American Journal of Transplantation (2012) 12 SUPPL. 3 (265). Date of   
Publication: May 2012  
  
Introduction: Graft thrombosis is the most common cause of technical failure   
in pancreas transplantation. It occur in up to 20% of patients and may be   
due to both donor and recipient factors. There is no current evidence to   
suggest that the use of anti-coagulation in the peri operative period has   
any potential benefit of reducing the rates of portal vein thrombosis in   
pancreas transplants. Objective: Retrospectively compare short and long term   
clinical outcomes and complication rates in patients underwent simultaneous   
kidney-pancreas transplants at our institution to determine if the use of   
post operative heparin played any effect. Methods: 47 SPK transplants were   
performed at our institution between 2004 and 2011. All patients since July   
2009 (n=16) received a regimen of heparin (Group 1) at 300U/h for 24h,   
followed by 400U/h until day 5, whereas patients transplanted prior to that   
date (n=31) did not (Group 2). No bolus given and heparin was started in the   
recovery unit. All patients were then placed on ASA on day 6 which they   
continued indefinitely. We assessed Donor: age, BMI, DCD/NDD, ischemic time;   
Recipient: age, previous transplant, previous thromboembolic events,   
immunosuppression, serum biochemistry (creatinine, glucose, hemoglobin,   
C-peptide, amylase, lipase, INR/PTT), complications (pancreatitis, DVT, PE,   
graft thrombosis, hemorrhage, transfusions, re-operation, graft function and   
loss). Serum parameters were measured pre-transplant and on days1, 3, 7, 14,   
30, 180 and 360. Statistical analyses were carried out using a Fisher Exact   
test and MANOVA. Results: 18% of Group 2 recipients lost their grafts to   
portal vein thromboses versus 0% in Group 1 with an overall graft function   
of 100% in Group 1 and 82% in Group 2 (p=0.11). One patient in Group 2 died   
from a fatal pulmonary embolus whereas no fatalities were observed in the   
heparinized group. Both groups had equal rates of transfusion and   
re-exploration/drain insertion for peri pancreatic fluid collections. Serum   
biochemical parameters for renal and pancreatic function were comparable   
between the groups at the time of last follow-up. Conclusion: This study   
provides novel information on the use of peri-operative intravenous   
anti-coagulative therapy in SPK transplantation. Although not statistically   
significant, there appears to be a clinically significant trend towards a   
beneficial effect of the use of post-operative intravenous heparin in this   
population. A larger cohort will be necessary to confirm these findings.  
  
RECORD 494  
Imaging and interventional radiology in congenital porto-systemic shunts  
Kanavaki A. Anooshiravani M. Wildhaber B. Mc Lin V. Hanquinet S. Terraz S.  
Pediatric Radiology (2012) 42 SUPPL. 3 (S486). Date of Publication: May 2012  
  
Purpose - Objective. To review clinical and radiological manifestations of   
five children with congenital portosystemic shunts (CPSS), as well as   
treatment and outcome. Material and methods. Between 2008 and 2011, five   
patients (age range, 12 days to 25 months) were admitted in our institution   
with a diagnosis of CPSS, established by US (5), CT (5) and MRI (4).   
Clinical presentation was hepatopulmonary syndrome (2), hepatoblastoma (1)   
and liver failure (4). Three patients underwent angiography for further   
evaluation. Results. In our series, three CPSS were extrahepatic, whereas   
two were intrahepatic. Three CPSS were closed by endovascular procedures,   
with a technical success of 100 %. One patient died 1 week after the   
procedure from brain haemorrhage. In one patient, a new small (<2 mm)   
intrahepatic shunt was detected on 24-hours US follow-up; the third patient   
developed partial portal vein thrombosis, treated by anticoagulation. Two   
patients were respectively transplanted 2 months and 1 year after diagnosis   
of CPSS with a very good outcome. One asymptomatic patient has so far been   
managed conservatively. Discussion and conclusions. CPSS is a rare condition   
and may be related to intrauterine growth retardation, galactosemia,   
cholestasis and hepatic encephalopathy. It may lead to hepatic tumours,   
hepatopulmonary syndrome and pulmonary hypertension. Ultrasonography is the   
first modality for diagnosis, with further work-up by contrast-enhanced   
CT/MRI, and angiography if necessary. Early detection and management,   
including interventional radiology, is valuable for the clinical outcome.  
  
RECORD 495  
Vascular complications after liver transplantation: A single center   
experience  
Chaman J.C. Padilla P.M. Rondon C.F. Carrasco F.A. Tan J. Bacilio W.   
Bedregal T. Mayorga R. Bobadilla F.  
Liver Transplantation (2012) 18 SUPPL. 1 (S171). Date of Publication: May   
2012  
  
Objective: To show the experience of vascular complications of liver   
transplantation at our center in more than 100 cases. Material and methods:   
A retrospective study of medical records of 109 liver transplant patients   
between adult and pediatric patients receiving liver graft with whole liver   
and segmental liver from related living donor. Results: From March 2000 to   
October 2011 were a total of 109 liver transplants, score MELD / PELD: mean   
18, range 8-40. Adults: 94 patients, all with whole liver from cadaveric   
donors (age range: 18 - 71 years, mean 47.6 y). Pediatric: 15 patients, 9   
children with segmental liver from living donor liver-related and 6 whole   
cadaveric donors (age range 8 months to 17 years, mean: 8.4 y). Hepatic   
artery (HA): Thrombosis: 6 (5.5%) (5 Whole liver, 1 LDLT). Stenosis: 1 LDLT   
Pre-anastomotic aneurism: 2 cases (1 whole liver, 1 LDLT),Portal Vein (PV):   
Thrombosis: 5 (4.58%) (3 whole liver, 1 LDLT). Stenosis: 2 (1 whole liver, 1   
LDLT). Pre-anastomotic aneurism: 2 (1 whole liver, 1 LDLT),. HV: 4 (2 whole   
liver, 2 LDLT). Retransplant: 5 cases (4.58%): 4 adults and 1 pediatric.   
Mortality: 0.91%. Conclusions: Our experience in vascular complications of   
liver transplantation shows similar results to those reported by other   
centers, with resolution by interventional radiology, with stenting and   
systemic anticoagulation, as well as immediate surgical reconstruction, and   
only in very severe cases retransplantation.  
  
RECORD 496  
anticoagulation in a cirrhotic patient with acute portal vein thrombosis   
unrelated to malignancy. A case report  
Ruiz P. Blanco S. Menénde F. Díaz A.B. Ortiz-de-Zárate J. Bravo M. Calderón   
A. Orive V.  
Revista Espanola de Enfermedades Digestivas (2012) 104:3 (152-153). Date of   
Publication: 2012  
  
RECORD 497  
Efficacy and safety of anticoagulation in patients with cirrhosis and portal   
vein thrombosis  
Seijo S. Delgado M.G. Yepes I. Achecar L. Catalina M.V. Garcia-Criado A.   
Abraldes J.G. De La Peña J. Bañares R. Albillos A. Bosch J. Garcia-Pagan   
J.C.  
Journal of Hepatology (2012) 56 SUPPL. 2 (S47). Date of Publication: April   
2012  
  
Introduction and Aim: portal vein thrombosis (PVT) is a frequent event in   
patients with cirrhosis that could worsen outcome and even prevent liver   
transplant (LT). anticoagulation has been suggested as an alternative   
therapy but there is limited data regarding safety and efficacy of this   
treatment in these patients. This study evaluates these issues in a large   
series of patients with cirrhosis and non-neoplastic PVT. Methods:   
Fifty-five patients with cirrhosis diagnosed of PVT, from June 2003 to   
September 2010, receiving anticoagulation were included. Patients with   
cavernomatous transformation were excluded. Diagnosis of thrombosis and the   
evaluation of recanalization were performed with Doppler ultrasound,   
angio-CT and/or angio-MRI. Results: The indication of anticoagulation was   
acute/subacute PVT in 31 patients and progression of previously known PVT in   
24. anticoagulation was based on low molecular weight heparins (LMWH) in 26   
patients; LMWH followed by vitamin K antagonists (VKA) in 21 and VKA alone   
in the remaining 8. Partial or complete recanalization was obtained in 33   
patients (60%) complete in 25 (45%). Precocity in starting anticoagulation   
was the only factor significantly associated with recanalization. Five of   
the 13 patients that stopped anticoagulation after achieve complete   
recanalization developed rethrombosis (38.5%) after a median follow-up of   
1.3 months. Despite similar baseline characteristics, patients achieving   
recanalization develop during follow-up less frequent portal hypertension   
related complications (portal hypertension related bleeding, ascites,   
hepatic encephalopathy), but without achieving statistical significance (p =   
0.1). Five patients developed bleeding complications probably related to   
anticoagulation. A platelet count <50×109/L was the only factor   
significantly associated with a higher risk of experiencing a bleeding   
complication. Six patients died due to liver disease but no deaths related   
to anticoagulation were observed. Conclusions: anticoagulation achieves   
complete recanalization of the portal venous axis in 45% of patients with   
cirrhosis and PVT, and this seems to be associated with a better outcome.   
anticoagulation should be maintained indefinitely to prevent rethrombosis.  
  
RECORD 498  
Bleeding risk of Endoscopic Variceal Ligation (EVL) in patients with Portal   
Vein Thrombosis (PVT) and anticoagulation: An analysis of 1235 procedures  
Christol C. Plessier A. Corbic M. Peron J.M. Vinel J.P. Valla D. Bureau C.  
Journal of Hepatology (2012) 56 SUPPL. 2 (S261). Date of Publication: April   
2012  
  
Introduction: The risk of variceal bleeding in patients with PVT is   
approximately 12% per year. As in cirrhosis, EVL is an approved procedure   
for the prevention of variceal bleeding regardless of the anticoagulation   
regimen. The risk of bleeding during EVL procedure but also due to post   
banding ulcers in these anticoagulated patients remains unknown. Our main   
objective was to assess the risk and the severity of upper gastrointestinal   
bleeding in patients treated by EVL while patients are on oral   
anticoagulants. Patients and Methods: All consecutive patients with PVT, who   
had at least one session of EVL between 2001 and 2010, were included.   
Patients were 1:1 matched with cirrhotic patients according to sex and   
severity of the disease. We collected all bleeding episodes during the EVL   
program and for each, the following data: duration of hospitalization, days   
in intensive care unit (ICU), presence of hemorrhagic shock and number of   
red blood cell transfusion. Results: There were 30 anticoagulated patients   
and 13 non anticoagulated patients in the “PVT group” (total = 43) and 43   
non anticoagulated patients in “cirrhosis group”. Bleeding occured in 9/121   
(7.4%) EVL performed in the anticoagulated PVT group, vs 6/130 (4.6%) EVL in   
the non-anticoagulated PVT group (NS). There was no difference between the 3   
groups in terms of number of hospitalizations, number of days in ICU,   
prevalence of hemorrhagic shock and number of red blood cell transfusion.   
Eradication was achieved an average 5.6 sessions in the “PVT with   
anticoagulation” group, 5.8 sessions in the “PVT without anticoagulation   
group” and 4.6 sessions in the “cirrhosis group” (NS). The eradication rates   
were similar between the 3 groups (83% vs 84% vs 84%) (NS). The mean time of   
eradication was also similar between the 3 groups. Conclusion: This study   
shows for the first time that oral anticoagulation - does not increase the   
risk of UGB in patients with PVT treated with EVL, nor the severity of   
bleeding. - does not affect the rate nor the delay of oesophagal varices   
eradication.  
  
RECORD 499  
Huge inflammatory pseudotumor of the spleen with postoperative portal vein   
thrombosis: Report of a case  
Tsutsumi N. Kawanaka H. Yamaguchi S. Sakai M. Momosaki S. Endo K. Ikejiri K.  
Surgery Today (2012) 42:4 (382-385). Date of Publication: April 2012  
  
Wereport the rare case of a splenic inflammatory pseudotumor associated with   
massive splenomegaly, diagnosed after surgery. A 51-year-old woman was   
admitted to our hospital for investigation of anemia. Physical examination   
revealed a palpable left upper quadrant mass. Computed tomography and   
magnetic resonance imaging showed a splenic mass, 20 cm in diameter. We   
performed splenectomy for both diagnosis and treatment. The spleen weighed   
2400 g, and histologic examination of the mass confirmed an inflammatory   
pseudotumor. portal vein thrombosis (PVT) developed the day after surgery,   
but resolved with anticoagulation therapy. This case highlights that there   
is a risk of PVT after splenectomy in patients with massive splenomegaly,   
and that anticoagulant therapy should be initiated promptly. © Springer   
2011.  
  
RECORD 500  
Thrombolytic therapy is effective in paroxysmal nocturnal hemoglobinuria: A   
series of nine patients and a review of the literature  
Araten D.J. Notaro R. Thaler H.T. Kernan N. Boulad F. Castro-Malaspina H.   
Small T. Scaradavou A. Magnan H. Prockop S. Chaffee S. Gonsky J. Thertulien   
R. Tarquini R. Luzzatto L.  
Haematologica (2012) 97:3 (344-352). Date of Publication: 20120301  
  
Background Thrombosis is the major risk factor for death in patients with   
paroxysmal nocturnal hemoglobinuria. Previous case reports indicate that   
venous thrombosis in patients with paroxysmal nocturnal hemoglobinuria is   
amenable to thrombolysis. Design and Methods We reviewed the outcome of   
thrombolytic therapy for patients with paroxysmal nocturnal hemoglobinuria   
who had thromboses refractory to anticoagulation at our institutions.   
Results In this study of 41 patients who had at least one thrombotic event,   
we confirmed a very high incidence of recurrence despite anticoagulation.   
Nine patients with thrombosis were regarded as eligible for administration   
of intravenous tissue plasminogen activator, which was effective in   
reversing thrombi in all of 15 occasions in which it was given. Serious   
hemorrhagic complications developed in three cases. At last follow-up visit,   
of the nine patients treated, three had died, and six were in very good to   
excellent condition in terms of clinical outcome and radiological findings.   
The only patient in whom thrombolysis may have contributed to a fatal   
outcome also had complications of "heparin induced thrombocytopenia with   
thrombosis", which we diagnosed in three additional patients. In our review   
of the literature, nine out of 15 patients treated with thrombolysis have   
had a good outcome. Conclusions Although it is associated with a significant   
but manageable risk of bleeding, systemic thrombolysis is a highly effective   
treatment for reversing venous thromboses in patients with paroxysmal   
nocturnal hemoglobinuria. © 2012 Ferrata Storti Foundation.  
  
RECORD 501  
Mesenteric vein thrombosis treated successfully with ultrasound augmented   
thrombolysis  
Agarwal A. Khan M.S. Aduli F. Li R. Culp W.C.  
Acta Gastro-Enterologica Belgica (2012) 75:1 (55-57). Date of Publication:   
March 2012  
  
Mesenteric vein thrombosis is a potentially fatal condition that is   
associated with better outcomes with early diagnosis and intervention. A   
32-year-old-man with Down syndrome presented with abdominal pain and was   
found to have extensive porto-splenomesenteric thrombosis with early bowel   
ischemia on computed tomography. He was treated successfully with ultrasound   
augmented thrombolysis. Ultrasound can improve efficiency of thrombolysis,   
decreasing the time required for thrombolysis by half, decrease thrombolytic   
dose and monitoring time and thus reduce overall costs and complications   
seen with long thrombolysis times.  
  
RECORD 502  
Acute pancreatitis with portal vein thrombosis-extremely rare complication   
of cardiac catheterisation  
Rajasurya V. Malhotra K. Rijal J.  
Journal of Hospital Medicine (2012) 7 SUPPL. 2 (S315). Date of Publication:   
March 2012  
  
Case Presentation: A 63-year-old man developed sudden onset of diffuse   
abdominal pain associated with nausea and non-bilious vomiting 12 hours   
after elective percutaneous transluminal coronary angioplasty (PTCA) with   
drug eluting stent placement in the mid left anterior descending coronary   
artery. There were no immediate post-procedure complications. Past medical   
history was significant for hypertension, angina pectoris and dyslipidemia.   
His daily medications were aspirin, clopidogrel, atorvastatin, metoprolol,   
and sublingual nitroglycerine as needed for chest pain. Physical examination   
was remarkable for tenderness in the epigastric region without rebound. Lab   
tests revealed amylase of 400 U/L, lipase of 840 U/L, and WBC of 14000 (85 %   
polymorphs). CT abdomen with contrast showed significant peripancreatic   
stranding and filling defect in the portal vein, consistent with acute   
pancreatitis and portal vein thrombosis. Patient was kept NPO, given IV   
fluids and was started on enoxaparin and coumadin. His symptoms improved and   
he was discharged home on the third day. After 3 months, follow up CT   
abdomen revealed complete resolution of pancreatitis and portal vein   
thrombosis and hence his coumadin treatment was discontinued. Discussion:   
Acute Pancreatitis is an extremely rare complication of PTCA procedure   
resulting from atheromatous embolization of the pancreatic vessels. Ischemia   
resulting from occlusion of vessels can lead to varying degrees of   
pancreatic necrosis ranging from subtle changes to necrotizing pancreatitis.   
Orvar and Johlin reported the largest series of acute pancreatitis after   
cardiac catheterization or abdominal angiographic procedures. They studied   
21,000 patients undergoing angiographic procedures and pancreatitis was   
reported in only 0.4% of the cases. Our patient had no other risk factor for   
pancreatitis such as alcoholism or cholelithiasis. Pancreatitis accounts for   
only 3-5% of cases of portal vein thrombosis via either a contiguous   
inflammatory process, direct compression of the portal vein by a pseudocyst,   
or a combination of both. In our case, the acute pancreatitis served as a   
nidus for the development of portal vein thrombosis. Management of   
pancreatis is largely supportive and anticoagulation is considered for acute   
portal vein thrombosis. Conclusions: The triad of PTCA, acute pancreatitis   
and portal vein thrombosis is extremely rare and to our knowledge this is   
the first case ever reported in the literature. Although very rare,   
atheromatous embolization and acute pancreatitis should be considered as one   
of the possible causes of acute abdomen in patients who have undergone   
angiographic procedures. Supportive care for acute pancreatitis and   
anticoagulation for portal vein thrombosis result in complete resolution of   
both of these complications.  
  
RECORD 503  
A case of portal vein thrombosis after laparoscopic low anterior resection   
of the rectum  
Naito N. Kano N.  
Surgical Endoscopy and Other Interventional Techniques (2012) 26 SUPPL. 1   
(S251). Date of Publication: March 2012  
  
Introduction: portal vein thrombosis (PVT) after laparoscopic surgery is a   
relatively uncommon but potentially lethal complication. There are several   
reports of PVT after laparoscopic surgery such as laparoscopic splenectomy.   
There are only a few reports of cases with PVT after laparoscopic colectomy   
for malignant tumors. However, its true incidence may have been   
underestimated due to difficulty in making the diagnosis. We report a case   
of PVT in a patient with no hypercoagulable states and risk factors for   
thrombosis, who underwent laparoscopy assisted low anterior resection of the   
rectum. Case: The patient is a 55 year old male with no past medical   
history. He noticed hematochezia and came to see his family physician.   
Colonoscopy revealed an early stage rectal cancer. He underwent endoscopic   
mucosal resection (EMR) for the lesion. The pathological examination showed   
the mucosal lesion with no positive margin and no vessel invasion. A   
follow-up colonoscopy showed a recurrent lesion. He undertook laparoscopy   
assisted low anterior resection for the recurrent rectal cancer. On eleventh   
day after operation, he noticed abdominal distention. Abdominal CT revealed   
thrombi in the umbilical portion of the portal vein. His general condition   
was stable and his symptom disappeared on the next day. We started systemic   
anticoagulation therapy with Warfarin. The thrombi were not detected on the   
followup CT four months after the onset. We continued anticoagulation   
therapy for six months after the follow-up CT. The patient is followed as an   
outpatient with no signs of recurrence. Result: PVT after laparoscopic   
surgery is uncommon and difficult to diagnose because of nonspecific signs   
and symptoms. Its presentation, treatment, and outcomes are poorly   
understood. Possible etiologic factors are malignant tumors, abdominal   
inflammatory diseases, alteration in coagulation during pneumoperitoneum,   
intraoperative damage to the splanchnic endothelium and systemic   
thrombophilic states. PVT should be diagnosed and treated precisely and   
promptly, because it can be lethal.  
  
RECORD 504  
Management of hepatic vascular diseases  
Plessier A. Rautou P.-E. Valla D.-C.  
Journal of Hepatology (2012) 56:SUPPL. 1 (S25-S38). Date of Publication:   
2012  
  
Primary damage to hepatic vessels is rare. (i) Hepatic arterial disorders,   
related mostly to iatrogenic injury and occasionally to systemic diseases,   
lead to ischemic cholangiopathy. (ii) Hepatic vein or inferior vena cava   
thrombosis, causing primary Budd-Chiari syndrome, is related typically to a   
combination of underlying prothrombotic conditions, particularly   
myeloproliferative neoplasms, factor V Leiden, and oral contraceptive use.   
The outcome of Budd-Chiari syndrome has markedly improved with   
anticoagulation therapy and, when needed, angioplasty, stenting, TIPS, or   
liver transplantation. (iii) Extrahepatic portal vein thrombosis is related   
to local causes (advanced cirrhosis, surgery, malignant or inflammatory   
conditions), or general prothrombotic conditions (mostly myeloproliferative   
neoplasms or factor II gene mutation), often in combination. anticoagulation   
at the early stage prevents thrombus extension and, in 40 of the cases,   
allows for recanalization. At the late stage, gastrointestinal bleeding   
related to portal hypertension can be prevented in the same way as in   
cirrhosis. (iv) Sinusoidal obstruction syndrome (or venoocclusive disease),   
caused by agents toxic to bone marrow progenitors and to sinusoidal   
endothelial cells, induces portal hypertension and liver dysfunction.   
Decreasing the intensity of myeloablative regimens reduces the incidence of   
sinusoidal toxicity. (v) Obstruction of intrahepatic portal veins   
(obliterative portal venopathy) can be associated with autoimmune diseases,   
prothrombotic conditions, or HIV infection. The disease can eventually be   
complicated with end-stage liver disease. Extrahepatic portal vein   
obstruction is common. anticoagulation should be considered. (vi) Nodular   
regenerative hyperplasia is induced by the uneven perfusion due to   
obstructed sinusoids, or portal or hepatic venules. It causes pure portal   
hypertension. © 2012 European Association for the Study of the Liver.  
  
RECORD 505  
Clinical and radiographic presentation of superior mesenteric vein   
thrombosis in Crohn's disease  
Kopylov U. Amitai M. Lubetsky A. Eliakim R. Chowers Y. Ben-Horin S.  
Journal of Crohn's and Colitis (2012) 6 SUPPL. 1 (S107). Date of   
Publication: February 2012  
  
Background: Mesenteric and portal vein thrombosis are rare and frequently   
underdiagnosed complications of Crohn's disease (CD). The data pertaining to   
the prevalence, clinicoradiologic features of these patients and the   
management approach is sparse. This study describes the clinical and   
radiological characteristics of CD patients with superior mesenteric vein   
thrombosis (MVT) diagnosed by CT or MRI. Methods: The database of Crohn's   
disease patients treated in Sheba Medical Center between 2005 2010 was   
searched for MVT diagnosis. Imaging studies of identified patients were   
retrieved and reviewed by an experienced abdominal radiologist. MVT was   
defined by superior mesenteric vein obliteration and/or thrombus in the   
vessel lumen on abdominal imaging. The clinical and radiologic data of these   
patients were collected from the medical records. Results: MVT was   
demonstrated in 6/460 CD patients in our center's database. The mean disease   
duration was 15 years. Five patients had stricturing disease, and one   
patient had a combined fistulizing and stricturing disease phenotype. All 6   
patients had small bowel disease, but 3/6 also had colonic involvement. No   
patient had a prior thromboembolic history or demonstrable   
hypercoagulability. One patient had an acute SMV thrombus demonstrable on CT   
scanning, the remaining patients showed an obliteration of superior   
mesenteric vein. Two out of six patients received anticoagulation upon   
diagnosis of thrombosis, and four patients who were diagnosed   
retrospectively did not. None of the patients developed subsequent   
thromboembolic events (mean follow-up 2.25±1.5 years). Conclusions: The   
incidence of mesenteric vein thrombosis is likely to be underestimated in   
patients with Crohn's disease. Both CT and MRI imaging demonstrate the   
extent of enteric disease and coincident SMV thrombosis. In our cohort,   
thrombosis was associated with stricturing disease of the small bowel. The   
clinical impact of SMV thrombosis and whether anticoagulation is mandatory   
for all of these patients remains to be determined.  
  
RECORD 506  
Japanese case of Budd-Chiari syndrome due to hepatic vein thrombosis   
successfully treated with liver transplantation  
Iwasaki T. Kawai H. Oseki K. Togashi T. Shioji K. Yamamoto S. Sato Y. Suzuki   
K. Toba K. Nomoto M. Hatakeyama K. Aoyagi Y.  
Hepatology Research (2012) 42:2 (213-218). Date of Publication: February   
2012  
  
A 22-year-old Japanese woman was found to have severe esophageal varices and   
then suffered from hepatic encephalopathy. She was diagnosed with   
Budd-Chiari syndrome (BCS) due to hepatic vein (HV) thrombosis accompanied   
by portal vein thrombosis without inferior vena cava (IVC) obstruction.   
Latent myeloproliferative neoplasm (MPN) lacking the JAK2-V617F mutation was   
considered to be the underlying disease. Liver transplantation was   
strikingly effective for treating the clinical symptoms attributable to   
portal hypertension. Although thrombosis of the internal jugular vein   
occurred due to thrombocythemia, which manifested after transplantation   
despite anticoagulation therapy with warfarin, the thrombus immediately   
disappeared with the addition of aspirin. Neither thrombosis nor BCS has   
recurred in more than 4years since the amelioration of the last thrombotic   
event, and post-transplant immunosuppression with tacrolimus has not   
accelerated the progression of MPN. In Japan, IVC obstruction, which was a   
predominant type of BCS, is suggested to have decreased in incidence with   
recent improvements in hygiene. The precise diagnosis of BCS and causative   
underlying diseases should be made with attention to the current trend of   
the disease spectrum, which fluctuates with environmental sanitation levels.   
Because the stepwise strategy, including liver transplantation, has been   
proven effective for patients with pure HV obstruction in Western countries,   
this strategy should also be validated for utilization in Japan and in   
developing countries where HV obstruction potentially predominates. © 2011   
The Japan Society of Hepatology.  
  
RECORD 507  
Right heart and pulmonary thromboembolism from extensive splanchnic vein   
thrombosis after splenectomy for myeloproliferative disease  
Stanziola A.A. Padula S. Carpentieri E. Rea G. Maniscalco M. Sofia M.  
Heart and Lung: Journal of Acute and Critical Care (2012) 41:2 (188-191).   
Date of Publication: March 2012  
  
Background: Splenectomy is a risk factor for both portal-vein and chronic   
thromboembolic pulmonary hypertension. The underlying mechanism is unclear,   
but may involve a hypercoagulable state. Methods: We describe 1 patient with   
polycythemia vera who developed extensive portal thrombosis of the portal,   
suprahepatic, and inferior cava veins, leading to right heart   
thromboembolism, with a resultant pulmonary embolism subsequent to   
splenectomy despite heparin prophylaxis. Results: In this patient, several   
mechanisms may have played a role, including perioperative stress,   
thrombocytosis, thrombophilia, and associated chronic liver disease.   
Nevertheless, combined treatment with intravenous heparin and thrombolysis   
and the myeloproliferative inhibitor hydroxyurea was associated with a   
favorable outcome. Conclusion: The risk of pulmonary thromboembolic   
complications and their management after splenectomies for hematologic   
disease warrant further study. © 2012 Elsevier Inc.  
  
RECORD 508  
Contrast-enhanced ultrasound for non-tumor liver diseases  
Maruyama H. Yokosuka O.  
Journal of the Nepal Medical Association (2012) 52:1 (43-48). Date of   
Publication: January-March 2012  
  
Contrast-enltanced ultrasound (CEUS) is a wimple, safe and reliable   
technique for thv clinical management at patients with various liver   
diseases. Although the major target of the technique may be focal hepatic   
lesions, it is also effective for the diagnosis of non-tumor liver diseases,   
such a grading hepatic fibrosis, charactvriiiation of chronic liver diseases   
and diagnosis of portal vein thrombosis. This review article aimed lo   
overview the necent application of CEUS in the assessment of non-tumor liver   
diseases.  
  
RECORD 509  
portal vein thrombosis after restorative proctocolectomy for familial   
adenomatous polyposis and sigmoid cancer  
Meshikhes A.-W.N. Al-Ghazal T.  
Case Reports in Gastroenterology (2012) 6:1 (124-130). Date of Publication:   
January-April 2012  
  
Postoperative portal vein thrombosis (PVT) is rare, but has been described   
after various open as well as minimal access abdominal operations,   
especially splenectomy and colorectal surgical procedures. We report the   
case of a 39-year-old female who underwent restorative proctocolectomy and   
ileal pouch-anal anastomosis for familial adenomatous polyposis with sigmoid   
cancer. She presented 14 days later with vague upper abdominal pain, nausea,   
vomiting and high output stoma. Doppler ultrasonography confirmed PVT and   
therefore anticoagulant therapy was started. Her condition improved   
dramatically and she underwent closure of ileostomy after finishing adjuvant   
chemotherapy. She remained well at 3-year follow-up with good pouch function   
and no local or distant recurrence. A high index of suspicion is essential   
for early diagnosis and prompt treatment of postoperative PVT after   
restorative proctocolectomy. Early anticoagulation is essential to avoid   
subsequent complications. Copyright © 2012 S. Karger AG, Basel.  
  
RECORD 510  
Inflammatory bowel disease-associated thromboembolism: A systematic review   
of outcomes with anticoagulation versus catheter-directed thrombolysis  
Tabibian J.H. Streiff M.B.  
Inflammatory Bowel Diseases (2012) 18:1 (161-171). Date of Publication:   
January 2012  
  
Background: Thromboembolism (TE) is a common extraintestinal complication of   
inflammatory bowel disease (IBD). Catheter-directed thrombolysis (CDT) is   
being increasingly used to treat TE but often evokes fears of hemorrhagic   
complications (HCs) in patients with IBD. We reviewed clinical outcomes with   
anticoagulation (AC) and CDT in IBD patients with TE. Methods: Published   
cases of IBD patients with TE were identified by a PubMed search. Cases were   
divided into two groups based on treatment modality: AC alone or CDT.   
Pretreatment variables and treatment-related outcomes were compared between   
treatment groups. Results: Fifty-two cases of IBD-associated TE were   
identified. Thirty-five cases were treated with AC alone and 17 with CDT.   
There were no significant differences in pretreatment variables. Patients   
treated with CDT tended to be more likely to achieve complete or partial   
symptomatic (P = 0.02) and radiologic resolution (P = 0.06).   
Gastrointestinal (GI) and non-GI HCs tended to occur more frequently with   
CDT, although these differences were not statistically significant (P = 0.44   
and 0.15, respectively). Conclusions: CDT and AC both appear to be well   
tolerated by IBD patients with TE. CDT may be used preferentially in   
patients with life-threatening TE, while AC may be preferable in patients   
with less clinically significant TE or patients at higher risk for bleeding.   
Further prospective studies are warranted to confirm these results and more   
definitively identify the best therapeutic approach for patients with   
IBD-associated TE. Copyright © 2011 Crohn's & Colitis Foundation of America,   
Inc.  
  
RECORD 511  
Porto-mesenteric thrombosis of congenital origin: An infrequent cause of   
acute abdomen  
Díaz-Roca A.B. Martínez-Garbaye S. Baranda-Martín A. Blanco-Sampascual S.   
Calderón-García A. Ruiz-Eguiluz P. Menéndez-Blazquez F. Orive-Cura V.  
Revista Espanola de Enfermedades Digestivas (2011) 103:11 (608-609). Date of   
Publication: 2011  
  
RECORD 512  
Neonatal portal vein thrombosis: Diagnosis and management  
Williams S. Chan A.K.C.  
Seminars in Fetal and Neonatal Medicine (2011) 16:6 (329-339). Date of   
Publication: December 2011  
  
Neonatal portal vein thrombosis (PVT) is an increasingly recognized event.   
Patients are generally asymptomatic in the neonatal period. The diagnosis is   
made with Doppler ultrasound. Umbilical catheterization, exchange   
transfusion and sepsis are risk factors for neonatal PVT. Thrombophilia is   
possibly a contributing risk factor. Although there are potential serious   
acute complications such as hepatic necrosis, the outcome is good in the   
majority of cases, followed up to 8 years of age. Thrombus resolution occurs   
in 30-70% in days to months. Liver lobe atrophy may occur following PVT, and   
does not appear to be associated with any impairment of liver function.   
Non-occlusive thrombosis is more likely to resolve than non-occlusive   
thrombosis. A subset of patients without resolution is at risk for   
developing portal hypertension over the next decade of life. There are no   
current defining features present during the neonatal period to enable   
identification of neonates at risk for portal hypertension. There is no   
evidence that anticoagulation therapy improves time to resolution or   
decreases the likelihood of portal hypertension. anticoagulation therapy may   
be considered. A management algorithm is proposed. © 2011 Elsevier Ltd.  
  
RECORD 513  
portal vein thrombosis is a potentially preventable complication in clinical   
islet transplantation  
Kawahara T. Kin T. Kashkoush S. Gala-Lopez B. Bigam D.L. Kneteman N.M. Koh   
A. Senior P.A. Shapiro A.M.J.  
American Journal of Transplantation (2011) 11:12 (2700-2707). Date of   
Publication: December 2011  
  
Percutaneous transhepatic portal access avoids surgery but is rarely   
associated with bleeding or portal venous thrombosis (PVT). We herein report   
our large, single-center experience of percutaneous islet implantation and   
evaluate risk factors of PVT and graft function. Prospective data were   
collected on 268 intraportal islet transplants (122 subjects). A portal   
venous Doppler ultrasound was obtained on Days 1 and 7 posttransplant.   
Therapeutic heparinization, complete ablation of the portal catheter tract   
with Avitene paste and limiting packed cell volume (PCV) to <5 mL completely   
prevented any portal thrombosis in the most recent 101 islet transplant   
procedures over the past 5 years. In the previous cumulative experience,   
partial thrombosis did not affect islet function. Standard liver volume   
correlated negatively (r =-0.257, p < 0.001) and PCV correlated positively   
with portal pressure rise (r = 0.463, p < 0.001). Overall, partial portal   
thrombosis occurred after 10 procedures (overall incidence 3.7%, most recent   
101 patient incidence 0%). There were no cases of complete thrombosis and no   
patient developed sequelae of portal hypertension. In conclusion, portal   
thrombosis is a preventable complication in clinical islet transplantation,   
provided therapeutic anticoagulation is maintained and PCV is limited to <5   
mL. © Copyright 2011 The American Society of Transplantation and the   
American Society of Transplant Surgeons.  
  
RECORD 514  
Antithrombotic therapy in non-neoplastic chronic portal venous thrombosis in   
cirrhosis: Recanalization and liver function evaluation  
Bento L. Huerta A.R. Pascual C. Rus G.P. Catalina V. Yepes I. Pérez-Corral   
A.M. Anguita J. Kwon M. Diez Martin J.L.  
Blood (2011) 118:21. Date of Publication: 18 Nov 2011  
  
INTRODUCTION: Non-neoplastic chronic portal vein thrombosis (PVT) is a   
frecuent diagnosis in the course of liver cirrhosis, with reported   
prevalences of 0.6% to 15,8%. PVT can motivate life-threatening   
complications due to worsening portal hypertension, so anticoagulation   
therapy is challenging in these patients. OBJECTIVE: To analyze the response   
to antithrombotic therapy and changes in liver function tests in 28 patients   
with chronic PVT associated with cirrhosis. PATIENTS AND METHODS: 28   
consecutive patients with liver cirrhosis and chronic PVT were treated with   
antithrombotic therapy from 2004 to 2009. Hepatocellular carcinoma and known   
thrombophilic risks were ruled out. Therapy consisted in 15 days of   
therapeutic doses of low molecular weight heparin (LMWH) (enoxaparin)   
adjusted according to baseline coagulability (Table 1), followed by either   
prophylactic doses (40mg/day) of LMWH or acenocoumarol (target INR 2-3),   
during 6 months. Response was evaluated after 6 months. If recanalization   
was complete, therapy was suspended. If recanalization was partial or no   
recanalization was observed, therapy was continued until response. [Table   
Presented] RESULTS: From the 28 patients studied, 19 (68%) were males with a   
median age of 53 years (range 35-77). cirrhosis was due to alcoholism (25%),   
virus (54%), mixed in 1 patient and other causes in 3 patients. PVT involved   
the portal trunk and/or branches in 19/28 (68%) patients, mesenteric vein in   
2 patients and portal trunk and/or branches, mesenteric and/or splenic vein   
thrombosis coexisted in 7 patients. 19/28 (68%) of the patients had moderate   
or moderate-severe hypocoagulability range. Complete and partial thrombosis   
was seen in 18 and 10 patients at diagnosis, respectively. From the 28   
patients, 18 (64%) responded to antithrombotic therapy after 6 months, with   
a complete recanalization in 13 patients 13/18 (72%) and partial in 5/18   
patients (28%). None of the 28 patients presented hemorrhagic complications   
and none showed platelets counts below baseline values. 17 from the 18   
patients who responded, showed altered liver function tests before therapy.   
After 6 months, 8/17 (47%) improved liver function (only one patient had   
received antiviral therapy). After a median follow up of 42 months (range   
7-67), 15/18 (83%) patients continued showing complete or partial response   
while 3 patients progressed. Of note, 3 patients of this group could proceed   
to further liver transplantation. CONCLUSIONS: Antithrombotic therapy in   
chronic PVT in cirrhotic patients resulted in a high response rate (64%) in   
our study, with a complete recanalization in 72% of the cases. Adjusted dose   
scheme according to level of hypocoagulability seems to be effective and   
safe, since 63% of the subgroups of moderate and moderate-severe   
hypocoagulability responded with no haemorrhagic complications.  
  
RECORD 515  
Management of acute non-cirrhotic and non-malignant portal vein thrombosis:   
a systematic review.  
Hall T.C. Garcea G. Metcalfe M. Bilku D. Dennison A.R.  
World journal of surgery (2011) 35:11 (2510-2520). Date of Publication: Nov   
2011  
  
No definitive evidence exists regarding the treatment of acute portal vein   
thrombosis (PVT). Treatment modalities described include conservative   
management, anticoagulation, thrombolysis, and thrombectomy. This review   
examines the impact of such treatment, its outcomes, and the complications   
resulting from the resultant portal hypertension. A Medline literature   
search was undertaken using the keywords portal vein thrombosis,   
anticoagulation, thrombolysis, and thrombectomy. The primary end point was   
portal vein recanalization. Secondary outcome measures were morbidity and   
the development of portal hypertension and its sequelae, including variceal   
bleeding. Data from articles relating to PVT in the context of cirrhosis,   
malignancy, or liver transplant were excluded. Early systemic   
anticoagulation results in complete portal vein recanalization in 38.3% of   
cases and partial recanalization in 14.0% of cases. Spontaneous   
recanalization without treatment can only be expected in up to 16.7% of   
patients. Frequently this is only when associated with self-limiting   
underlying pathology and/or minimal thrombus extension. Thrombolysis can be   
associated with major complications in up to 60% of patients. The natural   
history of acute PVT is poorly described. Spontaneous resolution of acute   
portal vein thrombosis is uncommon. Early anticoagulation results in a   
satisfactory rate of recanalization with minimal procedure-associated   
morbidity. Thrombolysis should be used with caution and only considered if   
the disease is progressive and signs of mesenteric ischemia are present.   
Further well-designed trials with precise outcome reporting are needed to   
improve our understanding of the disease.  
  
RECORD 516  
Medical management of chronic liver diseases (CLD) in children (part II):   
Focus on the complications of CLD, and CLD that require special   
considerations  
El-Shabrawi M.H.F. Kamal N.M.  
Pediatric Drugs (2011) 13:6 (371-383). Date of Publication: 2011  
  
Treatment of the causes of many chronic liver diseases (CLDs) may not be   
possible. In this case, complications must be anticipated, prevented or at   
least controlled by the best available therapeutic modalities. There are   
three main goals for the management of portal hypertension: (i) prevention   
of the first episode of variceal bleeding largely by non-selective   
β-adrenoceptor antagonists, which is not generally recommended in children;   
(ii) control of bleeding by using a stepwise approach from the least to most   
invasive strategies; (iii) and prevention of re-bleeding using bypass   
operations, with particular enthusiasm for the use of meso-Rex bypass in the   
pediatric population. Hepatic encephalopathy management also consists of   
three main aspects: (i) ruling out other causes of encephalopathy; (ii)   
identifying and treating precipitating factors; and (iii) starting empiric   
treatment with drugs such as lactulose, rifaximin, sodium benzoate, and   
flumazenil. Treatment of mild ascites and peripheral edema should begin with   
the restriction of sodium and water, followed by careful diuresis, then   
large-volume paracentesis associated with colloid volume expansion in severe   
cases. Empiric broad spectrum antimicrobial therapy should be used for the   
treatment of spontaneous bacterial peritonitis, bacterial and fungal sepsis,   
and cholangitis, after taking appropriate cultures, with appropriate changes   
in therapy after sensitivity testing. Empirical therapies continue to be the   
standard practice for pruritus; these consist of bile acid binding agents,   
phenobarbital (phenobarbitone), ursodeoxycholic acid, antihistamines,   
rifampin (rifampicin), and carbamazepine. Partial external biliary diversion   
can be used in refractory cases. Once hepatorenal syndrome is suspected,   
treatment should be initiated early in order to prevent the progression of   
renal failure; approaches consist of general supportive measures, management   
of concomitant complications, screening for sepsis, treatment with   
antibiotics, use of vasopressin analogs (terlipressin), and renal   
replacement therapy if needed. Hepatopulmonary syndrome and portopulmonary   
hypertension are best managed by liver transplantation. Provision of an   
adequate caloric supply, nutrition, and vitaminmineral supplements for the   
management of growth failure, required vaccinations, and special care for   
ensuring psychologic well-being should be ensured. anticoagulation might be   
attempted in acute portal vein thrombosis.Some CLDs, such as extrahepatic   
biliary atresia (EHBA), Crigler-Najjar syndrome, and Indian childhood   
cirrhosis, require special considerations. For EHBA, Kasai   
hepatoportoenterostomy is the current standard surgical approach in   
combination with nutritional therapy and supplemental fat and water soluble   
vitamins, minerals, and trace elements. In type 1 Crigler-Najjar syndrome,   
extensive phototherapy is the mainstay of treatment, in association with   
adjuvant therapy to bind photobilirubin such as calcium phosphate,   
cholestyramine, or agar, until liver transplantation can be carried out.   
Treating Indian childhood cirrhosis with penicillamine early in the course   
of the disease and at doses similar to those used to treat Wilson disease   
decreases the mortality rate by half.New hopes for the future include   
extracorporeal liver support devices (the molecular adsorbent recirculating   
system MARS® and Prometheus®), hepatocyte transplantation, liver-directed   
gene therapy, genetically engineered enzymes, and therapeutic modalities   
targeting fibrogenesis. Hepapoietin, a naturally occurring cytokine that   
promotes hepatocyte growth, is under extensive research. © 2011 Adis Data   
Information BV. All rights reserved.  
  
RECORD 517  
Coagulopathy of chronic liver disease [7]  
Qi X. Han G. Fan D.  
New England Journal of Medicine (2011) 365:15 (1452-1453). Date of   
Publication: 13 Oct 2011  
  
RECORD 518  
The authors reply  
Tripodi A. Mannucci P.M.  
New England Journal of Medicine (2011) 365:15 (1453-1454). Date of   
Publication: 13 Oct 2011  
  
RECORD 519  
Analysis of correlation factors of portal vein thrombosis in patients after   
splenctomy with portal hypertension of cirrhosis resulting from hepatitis  
Pan W.-D. Xu R.-Y.  
Journal of Gastroenterology and Hepatology (2011) 26 SUPPL. 5 (140). Date of   
Publication: October 2011  
  
Objective To analysis the correlation factors of portal vein thrombosis in   
patients after splenctomy with portal hypertension of cirrhosis resulting   
from hepatitis. Method 132 patients with portal hypertension in liver   
cirrhosis from hepatisis who had simple splenectomy, splenectomy and EVL,   
splenectomy and portaazygous devascularization were reviewed from March 1999   
to March 2005. The relationship between portal vein thrombosis and the liver   
function, average diameter of main portal vein, average diameter of spleen   
vein, splenomegaly, number of thrombocyte after operation was studied by   
Logistic Regression analysis. Results The portal vein thrombosis was related   
to the average diameter of main portal vein, splenomegaly, thickness of   
spleen, serum total bilirubin, number of thrombocyte after operation   
throught variable not in the Equation. However, through variable in the   
Equation the portal vein thrombosis was related to the average diameter of   
main portal vein, diameter of spleen vein, splenomegaly and treatment of   
anticoagulation. Conclusion The portal vein thrombosis in patients after   
splenectomy with portal hypertension of cirrhosis resulting from hepatitis   
was related to the average diameter of main portal vein, diameter of spleen   
vein, splenomegaly and treatment of anticoagulation.  
  
RECORD 520  
portal vein thrombosis (PVT) in patients with liver cirrhosis: Outcome of   
screening and anticoagulation  
Aqel B. Werner K.T. Sando S. Carey E.J. Vargas H.E. Douglas D.D.  
Hepatology (2011) 54 SUPPL. 1 (1267A). Date of Publication: October 2011  
  
Introduction: The prevalence of PVT increases with the severity of liver   
disease, being less than 1% in patients with well compensated Child's A   
cirrhosis to as high as 10% in decompensated patients awaiting liver   
transplant. In most patients with liver cirrhosis, development of PVT is   
often accompanied by increased rate of morbidity and mortality and affect   
patient candidacy for liver transplant. Furthermore, there is limited data   
regarding the role of anticoagulation therapy in patients with PVT and liver   
cirrhosis. Objectives:1.Describe the prevalence of hypercoaguable disorders   
in patients with liver cirrhosis and PVT 2. Describe outcome of   
anticoagulation in patients with liver cirrhosis and mesenteric or PVT.   
Methods: Retrospective chart review of patients with end stage liver disease   
(ESLD) awaiting liver transplant who were diagnosed with PVT or superior   
mesenteric vein thrombosis between January 2005 and April 2011. Data   
recorded include etiology of liver disease, extent of thrombus, results of   
hypercoagaulable work up, lenght of treatment, and response to treatment.   
Results:Sixty-eight patients were diagnosed with mesenteric vein thrombosis   
during the study period:48/68 had isolated PVT, 15/68 patients had combined   
mesenteric and PVT, and 5/68 patients had isolated mesenteric vein   
thrombosis. Chronic hepatitis C was the cause of liver disease in 23/68   
patients (34%), hepatocellular carcinoma was noted 25/68 (37%) of patients   
(all HCC patients met the transplant criteria). Screening for hypercoaguable   
disorder became a standard practice during the second half of the study   
period: 20 patients were screened and hypercoaguable disorder was diagnosed   
in 1/20 (5%). anticoagulation was initiated based on a strict protocol that   
included variceal eradication prior to treatment. Patients were treated for   
6-12 months . Seventeen (17/68) patients were treated during the study   
period with coumadin. PVT resolved in 6/17 (35%), showed partial resolution   
in 7/17(41%), no change in 2/17, and 2/17 patients did not have adequate   
follow up yet. One patient (5%) had significant vaginal bleeding on coumadin   
and treatment stopped after 10 months. None of the treated patients had GI   
bleeding. Conclusions: PVT is frequently seen in patients with ESLD.   
Hypercoaguable disorder was detected in 5% of the patients screened. Careful   
use of anticoagulation is safe and associated with thrombus resolution or   
partial resolution in 35% and 41% of patients respectively. Further   
prospective studies will be required to determine the safety of   
anticoagulation, prevalence of hypercoaguable disorders and the association   
between PVT and HCC.  
  
RECORD 521  
Clinical outcome of portal vein thrombosis (PVT) in cirrhotic patients:   
Observe or treat?  
Garcovich M. Zocco M.A. Ainora M.E. Annicchiarico B.E. Ponziani F.R. Cesario   
V. Campanale M. Gigante G. Siciliano M. Gasbarrini A.  
Hepatology (2011) 54 SUPPL. 1 (1261A-1262A). Date of Publication: October   
2011  
  
Background/aims: anticoagulation is considered the therapy of choice in   
patients with non-cirrhotic portal vein occlusion, while concerns regarding   
anticoagulation therapy are still present in patients with cirrhosis because   
of the high risk of bleeding related to clotting impairment and portal   
hypertension. Recently, safety and efficacy of anticoagulation therapy (AT)   
in cirrhotic patients have been shown, but little is known about long-term   
outcome and resolution of PVT with or without therapeutic intervention in   
this setting of patients. The aim of this study was to compare two   
well-matched cohorts of cirrhotic patients with PVT undergoing either   
therapy with low molecular weight heparin (LMWH) or only clinical   
observation. Methods: We retrospectively reviewed data on cirrhotic patients   
with PVT followed in our Unit and selected two cohorts of patients well   
matched for clinical and demographic characteristics: patients treated with   
LMWH (group A) and patients who didn't receive AT (group B). Exclusion   
criteria were advanced liver cirrhosis (Child-Pugh C), liver transplantation   
during follow-up, cavernomatous transformation of portal vein thrombosis,   
presence of neoplasms and active variceal bleeding or high-risk esophageal   
varices. Imaging of PVT with Doppler ultrasound or spiral CT/MRI was   
evaluated at baseline and 6 months after inclusion. Thrombosis was   
considered occludent when involving more than 75% of the vessel with minimal   
or absent blood flow; complete response was defined as whole recanalization   
or a reduction of more than 50% of the thrombus. Results: A total of 66   
cirrhotic patients with PVT were evaluated in order to select 15 patients   
with PVT (33% with occludent thrombosis) receiving LMWH for 3-6 months and   
15 patients with PVT (20% with occludent thrombosis) who didn't receive AT.   
LMWH therapy was administered for 3-6 months or until resolution of   
thrombosis, with no major side effects such as uncontrolled bleeding   
reported. Complete portal recanalization occurred in 7 out 15 patients in   
group A and in 5 out of 15 patients in group B (46% vs 33% complete   
resolution; p=0,45), suggesting no clear advantage for AT. Conclusions: As   
cirrhosis is characterized by a complex haemostasis defect including primary   
haemostasis, coagulation and fibrinolysis, clinical outcome of PVT may not   
always be easily predictable. Because evidence of a real clinical benefit   
from AT in cirrhotic patients with PVT is still lacking, more interventional   
studies evaluating the outcome predictors of PVT and benefit of AT in   
selected patient population are warranted.  
  
RECORD 522  
An interesting case of chronic idiopathic non-cirrhotic portal vein   
thrombosis  
Shah N. DePasquale J. Shah A. Shaaban H. Modi C. Spira R.  
American Journal of Gastroenterology (2011) 106 SUPPL. 2 (S287-S288). Date   
of Publication: October 2011  
  
Purpose: Introduction: portal vein thrombosis was first reported in 1868 by   
Balfour and Stewart. It is a rare condition that typically presents in   
non-cirrhotic patients. Inherited (Factor V Leiden and Prothrombin gene   
mutation G201210A, Protein C, S, Anti thrombin III deficiency) and acquired   
thrombophilias (Lupus Anticoagulant, myeloproliferative diseases,   
malignancy, surgery and trauma) account for majority of the cases of portal   
vein thrombosis. Doppler ultrasound studies are usually the initial test of   
choice. Case Report: 63 year old Hispanic Female with history of   
hypertension, Diabetes Mellitus, coronary artery disease presented with   
complaints of epigastric pain &bloody vomiting. She initially had epigastric   
discomfort &2 episodes of hematemesis. She denied alcohol use. Abdominal   
examination was unremarkable except for mild epigastric tenderness.   
Laboratory analysis revealed hemoglobin of 10.5 mg/dl with normal liver   
function tests and aminotransferases. An upper gastrointestinal endoscopic   
examination was done which revealed grade 4 esophageal varices with fresh   
blood in the distal esophagus which were subsequently ligated. She was   
started on octreotide and propranolol. She had another episode of GI bleed   
requiring emergent endoscopic intervention. She underwent a liver biopsy.   
Pathology revealed focal mild portal fibrosis with mild micro ¯o   
vesicular steatosis but no cirrhosis. A Doppler Ultrasound revealed portal   
vein thrombosis and concomitant portal hypertension. An extensive   
hypercoaguable work up was done which included protein C &S levels, anti   
thrombin III, Prothrombin gene mutation G20210A, Factor V Leiden, Lupus   
anticoagulant, Anticardiolipin antibodies, Homocysteine level &they were all   
negative. We also tested her blood for flow-cytometry for CD 55 and CD 59   
but the test was normal and in the process effectively ruled out paroxysmal   
nocturnal hemoglobinuria. She also was also tested for the JAK 2 mutation.   
It was negative. We gave her the clinical diagnosis of chronic idiopathic   
non-cirrhotic portal vein thrombosis. She subsequently had a meso-caval   
shunt done to relieve the portal hypertension. She was started on warfarin   
anticoagulation 3 days later with close monitoring for bleeding. She   
clinically got better and was then discharged home. Discussion:   
Non-cirrhotic portal vein thrombosis can be acute or chronic. Acute cases   
need at least 3 months of anticoagulation. Chronic cases needs   
porto-systemic shunting. The use of anticoagulation in chronic cases should   
be decided upon on a case by case basis weighing the risk of bleeding versus   
thrombosis. We opted to give long-term anticoagulation to our patient to   
prevent re-thrombosis of the meso-caval shunt/graft.  
  
RECORD 523  
Enoxaparin prevents portal vein thrombosis (PVT) and decompensation in   
advanced cirrhotic patients: Final report of a prospective randomized   
controlled study  
Villa E. Zecchini R. Marietta M. Bernabucci V. Lei B. Vukotic R. Ferrari A.   
De Maria N. Schepis F. Fornaciari G. Schianchi S.  
Hepatology (2011) 54 SUPPL. 1 (418A-419A). Date of Publication: October 2011  
  
PVT is a frequent complication of advanced cirrhosis, occurring in about   
8-25% of patients and leading to severe clinical deterioration,   
decompensation and death. anticoagulation has never been prospectively   
tested for its prevention. We therefore designed a prospective randomized   
trial of anticoagulant therapy in advanced cirrhotic patients with following   
endpoints: primary - evaluation of efficacy in preventing PVT; secondary -   
assessment of safety, prevention of decompensation and/or survival (Eudract   
2007-007890-22). Cirrhotic patients, Child B7- C10, were randomized to   
receive enoxaparin 4000 IU/die or placebo for 12 months followed by 12   
months observation. US was performed every 3 months and CT every 6 months to   
check for portal vein axis. PVT was considered as relevant when it was   
either complete or involved more than 50% of PV diameter and was   
symptomatic. We report the events of the 70 enrolled patients (34 randomized   
to treatment and 36 to placebo) at completion of the 24 months study. No   
relevant side effects, in particular no hemorrhagic events, were   
attributable to the active drug. Only one patient was withdrawn from active   
arm because of thrombocytopenia. During the 1-year study period, PVT (3   
complete, 3 partial) occurred in 6/36 (16.7%) patients on placebo and in   
none on enoxaparin [p=0.023 chi sq test)]. One patient with complete PVT   
died of septic shock shortly after developing PVT while the others recovered   
after acute anticoagulation. During follow-up, 6 additional thrombotic   
events occurred, 3 in the placebo group and 3 in the active arm, 2 to 6   
months after enoxaparin discontinuation (p=0.746). Decompensation occurred   
during the study period significantly more in placebo than in   
enoxaparin-treated patients [placebo 19/36 (52.7%) vs. 4/34 (11.7% ),   
p=0.0007]; this advantage was greatly attenuated but not lost during   
follow-up [placebo: 18/34 (52.9%) vs 7/29 (24.1%); p=0.02]. Survival was   
significantly better in enoxparin-treated patients (log rank 0.019). At   
logistic regression analysis, the only factor significantly associated with   
risk of developing PVT was degree of portal hypertension (OR 9.16; 95%CI   
1.1592- 52.780; p=0.013). The independent factors associated with risk of   
decompensation were bilirubin levels (OR 1.667; 95%CI 1.026-2.710, p=0.039)   
and enoxaparin treatment (OR 0.106, 95%CI 0.024-0.469, p=0.003). In this   
prospective randomized controlled study in advanced stage cirrhotics,   
enoxaparin was shown to be safe and effective in preventing PVT but, most   
importantly, was associated with greatly reduced occurrence of   
decompensation both during the active period of treatment and in the   
follow-up period. (Table Presented).  
  
RECORD 524  
Spontaneous spleno-renal shunt in patients with portal vein thrombosis is a   
predisposing factor for hepatic encephalopathy  
John B.V. Konjeti V.R. Lopez R. Carey W.D.  
Hepatology (2011) 54 SUPPL. 1 (1252A). Date of Publication: October 2011  
  
Background: Spontaneous spleno-renal shunts (SRS) are often seen in   
cirrhotics with portal hypertension. We have observed that SRS is seen more   
often in cirrhotics with portal vein thrombosis (PVT). SRS may result in   
shunting of blood away from the portal vein leading to sluggish flow and   
predispose to PVT. However, it's unclear if SRS is the cause or effect of   
PVT. Aims: The primary aim of this study is to assess if existence of SRS   
predisposes to development of new PVT in cirrhotics. Secondary aims include   
role of SRS on onset of ascites, hepatic encephalopathy and death. Methods:   
We included all cirrhotics evaluated for LT between 07/2004 and 06/2009 who   
had a minimum follow up of six months and at least one follow up imaging.   
Subjects with PVT at baseline, HCC or on anti-coagulation were excluded.   
Subjects were evaluated for SRS at baseline and prospectively followed with   
Doppler and CT or MRI of the abdomen with contrast every 6 months till LT,   
removal from transplant list, or death. Results: Of the 902 cirrhotics   
evaluated for LT, 243 met the inclusion criteria. Forty nine had SRS (group   
1) and 194 had no SRS on baseline imaging (group 2). Cirrhotics with NASH   
were more likely to have SRS than those without (27% vs. 14%; p=0.035).   
There was no difference in baseline MELD between the two groups (14.7 vs.   
13.8, p=0.23). Over a median follow up of approximately 24 months, 14% with   
SRS at baseline developed PVT compared to 8% in cirrhotics without (p=0.2).   
On multi-variate analysis, after adjusting for presence of ascites and   
creatinine, subjects with SRS were not at increased risk of developing PVT   
(Relative risk 1.5, 95% CI 0.61-3.7, p=0.37). There was no difference in the   
development of new onset ascites (32.7 vs. 36.8%, p=0.59) or encephalopathy   
(20.4 vs. 19.1%, p=0.83) between subjects with and without SRS. However,   
subjects with SRS and PVT were more likely to develop hepatic encephalopathy   
compared to those with SRS and no PVT (50% vs. 7%; p=0.022). There was no   
difference in pre or post-transplant mortality between subjects with and   
without SRS. Conclusion: Spontaneous spleno-renal shunts are seen in   
approximately 20% of cirrhotics evaluated for OLT and is more commonly seen   
in NASH cirrhosis. Subjects with spleno-renal shunt do not have an increased   
risk to develop PVT and is not associated with worsening liver disease or   
mortality. However, cirrhotics with SRS and PVT have a higher incidence of   
hepatic encephalopathy. We hypothesize that the development of PVT results   
in bypassing of blood from the portal vein to the spleno-renal shunt,   
resulting in encephalopathy.  
  
RECORD 525  
Pylephlebitis: A classically ambiguous presentation, with a rarely reported   
organism  
Lee S. Go B. Stroger J.H.  
American Journal of Gastroenterology (2011) 106 SUPPL. 2 (S281). Date of   
Publication: October 2011  
  
Purpose: A previously healthy 46-year-old-man presented to the emergency   
department with melena. He also described fevers, chills, anorexia, and   
post-prandial pain, treated with ibuprofen. Temperature was 102.3F, blood   
pressure 80/35 mmHg, heart rate 104/min, respiratory rate 20/min. Abdomen   
was mildly tender in the right upper quadrant. There was no stigmata of   
chronic liver disease. Rectal exam showed brown stool. Nasogastric lavage   
was clear. WBC count 12,600/uL (95% neutrophils); Hemoglobin 8.3 g/dL;   
platelets 147,000/uL. Sodium 127 mEq/L; BUN/Creatinine normal. Total   
bilirubin 1.8 mg/dL; direct bilirubin 1.1 mg/dL; remaining liver profile   
normal. Albumin 2.0 g/dL; total protein 4.6 g/dL; cholesterol 90 mg/dL. INR   
1.3. Abdominopelvic CT showed “dilated intrahepatic biliary ducts, mainly   
right lobe...probably cholangitis.” EGD found erosive esophagitis and   
gastritis. ERCP revealed a normal biliary system without defect or dilation.   
Repeated review of the CT suggested thrombus in the right portal vein,   
mimicking dilated biliary ducts, confirmed with abdominal ultrasound. Blood   
cultures grew Streptococcus intermedius in 4/4 bottles. Given the   
constellation of acute portal vein thrombosis (PVT), fever, abdominal   
discomfort, bacteremia/sepsis, the diagnosis of pylephlebitis was made.   
Pylephlebitis is septic thrombophlebitis of the portal vein and its   
tributaries. Intra-abdominal infection predisposes microthrombi formation,   
extension of which results in this rare cause of acute PVT. Diverticulitis   
has replaced appendicitis as the most common underlying primary infection.   
The hallmark of pylephlebitis is its remarkably vague presentation. In three   
well referenced series, 100% had fever, 74-100% abdominal pain, and 23-79%   
were bacteremic. Other features include leukocytosis and abnormal liver   
function tests; jaundice is a late finding. Enteric organisms are most   
frequently isolated, especially the uniquely thrombogenic Bacteroides   
species. S. intermedius has been reported in 2 other cases. This is the   
first English language case in which S. intermedius is the sole organism.   
Antibiotics and eradication of the primary infection are mainstays of   
therapy; the role of anticoagulation is still debated. Mortality remains   
significant at 10-50%. In this case, the presenting issue was the result of   
NSAID induced esophagitis/gastritis, itself resultant from the abdominal   
symptoms associated with pylephlebitis. Anemia and hypotension were   
initially attributed to GI bleeding, though in context of other findings,   
are entirely consistent with sepsis. Heightened awareness of this morbid   
condition, heralded only by its classically ambiguous presentation, will   
expedite recognition and treatment.  
  
RECORD 526  
Chronic portal vein thrombosis due to combined deficiency of protein C and   
protein S  
Das S.K. Ray A. Jana C.K. Banerjee N. Khaskil S.  
Journal of the Indian Medical Association (2011) 109:10 (753-754). Date of   
Publication: October 2011  
  
portal vein thrombosis (PVT) is a rare disorder that is associated with a   
variety of underlying condition of which liver cirrhosis, malignancy and   
myeloproliferative disorders are the most common. It is of two types, acute   
and chronic portal vein thrombosis. anticoagulation therapy is recommended   
for all patients with acute portal vein thrombosis. Chronic portal vein   
thrombosis is characterised by the development of portal hypertension.   
Bleeding from ruptured varices is the main complication. In the absence of   
bleeding, continuous anticoagulation therapy should be considered for   
chronic portal vein thrombosis in whom an underlying prothrombotic factor is   
to be identified. Here in this report a 13-yearold girl presented with   
haematemesis. The spleen was hugely enlarged. Her Hb was 8.38 g/dl. Grade   
III oesophageal varices were found in oesophagogastroduodenostomy. CT   
abdomen showed portal cavernoma formation with increased splenic collateral.   
Protein C activity was 45% and protein S activity was 40%. She was treated   
with β-blocker, endoscopic variceal ligation followed by low molecular   
weight heparin and warfarin.  
  
RECORD 527  
Danaparoid sodium was effective for portal vein thrombosis independent of   
plasma antithrombin III level  
Imamura J. Kimura K. Saeki S. Hayashi S.  
Hepatology (2011) 54 SUPPL. 1 (1263A). Date of Publication: October 2011  
  
Background: Antithrombin III (AT-III) has been reported to be an effective   
anticoagulant agent for portal vein thrombosis (PVT). However AT-III is very   
expensive and it costs 1049 dollars per day. On the other hand danaparoid   
sodium is inexpensive and it costs 36 dollars per day. The function of   
danaparoid sodium is dependent on AT-III. Consequently it is assumed that   
danaparoid sodium could not have sufficient anticoagulant activity under low   
plasma AT-III level such as liver cirrhosis. In this study, we assessed the   
effectiveness of danaparoid sodium for PVT. Methods: The subjects were 17   
patients (M/F=10/7, mean age 64.2 years) who were diagnosed PVT and were   
treated with danaparoid sodium at our institute from April 2006 to March   
2011. 1250 U/body of danaparoid sodium was administered twice a day   
intravenously for 14 days. Enhanced Computed Tomography (CT) was taken to   
evaluate PVT. We assessed if plasma AT-III level or the time from diagnosis   
to treatment had correlation with treatment effect by statistical analysis.   
Results: Of 17 patients, 12 patients (71%) had neoplasm and 9 patients (53%)   
had liver cirrhosis or portal hypertension. 3 patients (18%) were affected   
with PVT after laparotomy, and one patient (6%) was affected with PVT after   
transcatheter arterial chemoembolization (TACE). In 16 patients, treatment   
effect was evaluated by CT images after treatment. In 14 of 16 patients   
(88%), PVT disappeared or was markedly reduced in size. In 2 patients (12%),   
the treatment was ineffective. There was no significant correlation between   
treatment effect and the time from diagnosis to treatment. Plasma AT-III   
level was measured in 8 of 17 patients before treatment. Mean activity of   
plasma AT-III was 68.2 %. In 5 patients, plasma AT-III level was lower than   
the standard value (80 -120 %). However, in all of 8 patients, most of the   
PVT disappeared. Conclusion: Danaparoid sodium was effective for PVT even in   
the condition of low plasma AT-III level.  
  
RECORD 528  
Management of portal hypertension, Budd-Chiari syndrome and portal vein   
thrombosis  
Burroughs A.K.  
Medicine (2011) 39:10 (607-611). Date of Publication: October 2011  
  
The risk of variceal bleeding can be estimated by the size of varices, the   
presence of endoscopic red signs and the degree of liver dysfunction. All   
patients with large varices, and those with cirrhosis and severe liver   
disease, irrespective of the size of varices, should be given primary   
prophylaxis with non-selective β-blockers. Banding ligation is equivalent   
and is used if there are contraindications or intolerance to these drugs.   
Acute variceal bleeding should be managed in a gastrointestinal bleeding   
unit. Prophylactic third-generation cephalosporins and vasoactive drugs   
should always be given. Ligation or sclerotherapy should take place at   
diagnostic endoscopy. Secondary prophylaxis of variceal bleeding is   
mandatory with combined β-blockers and ligation. Hepatic outflow obstruction   
syndromes have a wide spectrum of presentation. Underlying thrombophilic   
conditions should be sought. A fulminant presentation requires liver   
transplantation. Decompression with transjugular intrahepatic stent shunt is   
effective in many cases and can also be used in cases of portal vein   
thrombosis. Hepatic and other venous webs can be treated with interventional   
radiological techniques. anticoagulation is first-line therapy and should be   
continued lifelong; it should also be used in non-cirrhotic portal vein   
thrombosis, and considered in cirrhotic portal vein thrombosis. © 2011   
Elsevier Ltd. All rights reserved.  
  
RECORD 529  
An unusual case of splancnic thrombosis  
Pacquola E. Cavallin S. Danesin C. Gherlinzoni F.  
Haematologica (2011) 96 SUPPL. 3 (214-215). Date of Publication: 1 Oct 2011  
  
A 64 year old woman was admitted to Hospital for abdominal pain and rectal   
bleeding. An emergency abdominal CT was performed which resulted in portal   
cavernoma, splenic, portal and superior mesenteric veins thrombosis with   
peripancreatic, perigastric, perisplenic hypertrofic collateral flows. The   
patient was then transferred to the Surgery Department where she underwent   
ileal resection for venous ischemia. Blood biochemistry showed normal   
parameters: HBG 11.4 g/dL, CMV 73 fl, RBC 5020 x 10∗6/mm3, WBC 5440 x   
10∗3/mm3, platelets 237 x 10∗9/mm3. Gastric endoscopy proved the presence of   
esophageal varices F1-2 and colonscopy reported sigma diverticulosis.   
Subsequently the patient was addressed to our Haematology Service for the   
assessment of thrombophilic parameters. Patient's medical history was   
investigated , giving evidence of a previous hospital admission (3 years   
earlier) for acute diverticulitis; on such occasion an abdominal CT   
evidenced a suspected portal vein thrombosis, but the patient was not   
evaluated any further nor administered anticoagulant therapy. These data   
were suggestive of Budd-Chiari Syndrome, so the JAK2 mutation was searched;   
it positivity lead us to perform bone marrow biopsy, which resulted   
comparable with polycythemia vera. Finally, based on age, previous   
thrombosis and the presence of the V617F JAK2 mutation the patient was   
treated with cytoriductive and anticoagulation therapy with good response.  
  
RECORD 530  
Idiopathic noncirrhotic portal hypertension  
Schouten J.N. Garcia-Pagan J.C. Valla D.C. Janssen H.L.  
Hepatology (2011) 54:3 (1071-1081). Date of Publication: 2 September 2011  
  
Idiopathic noncirrhotic portal hypertension (INCPH) is characterized by an   
increased portal venous pressure gradient in the absence of a known cause of   
liver disease and portal vein thrombosis. In contrast to the high prevalence   
of this disorder in India, INCPH is a rare disease in the Western world. The   
etiology of INCPH can be divided in five categories: chronic infections,   
exposure to medication or toxins, thrombophilia, immunological disorders,   
and genetic disorders. Multifactorial etiology can also be encountered.   
Chronic abdominal infection is incriminated as the most important   
etiological factor in Eastern patients and thrombophilia in Western   
patients. The majority of patients with INCPH initially present with signs   
or complications of portal hypertension (mainly variceal bleeding and   
splenomegaly). These patients usually have preserved liver function. Liver   
function impairment occurs mainly in the context of intercurrent conditions.   
Patients with INCPH are often clinically and radiologically misdiagnosed as   
liver cirrhosis, so that a liver biopsy is indispensable to discriminate   
cirrhosis from INCPH. Histopathological characteristics of INCPH are   
heterogeneous, demonstrating overlap between several pathological entities   
(e.g., hepatoportal sclerosis, nodular regenerative hyperplasia, and   
incomplete septal cirrhosis). Even though hemodynamical changes in INCPH   
patients are not comparable to those in cirrhotics, prophylaxis and   
treatment of variceal bleeding are recommended to be similar.   
anticoagulation therapy must be considered only in patients who develop   
portal vein thrombosis. INCPH has been considered a disorder with a   
relatively benign disease course. However, liver failure, hepatic   
encephalopathy, and hepatopulmonary syndrome can occur and are considered   
indications for liver transplantation. © 2011 American Association for the   
Study of Liver Diseases.  
  
RECORD 531  
Hypercoagulability in cirrhosis: Causes and consequences  
Tripodi A. Anstee Q.M. Sogaard K.K. Primignani M. Valla D.C.  
Journal of Thrombosis and Haemostasis (2011) 9:9 (1713-1723). Date of   
Publication: September 2011  
  
Decreased levels of most coagulation factors and thrombocytopenia are the   
main haemostatic abnormalities of cirrhosis. As a consequence, this   
condition was, until recently, considered as the prototype acquired   
coagulopathy responsible for bleeding. However, recent evidence suggests   
that it should, rather, be regarded as a condition associated with normal or   
even increased thrombin generation. The bleeding events that occur in these   
patients should, therefore, be explained by the superimposed conditions that   
frequently occur in this setting. Due to elevated levels of factor VIII   
(procoagulant driver) in combination with decreased protein C (anticoagulant   
driver), which are typically found in patients with cirrhosis, a   
procoagulant imbalance, defined as a partial resistance to the in vitro   
anticoagulant action of thrombomodulin, can be demonstrated. Whether this in   
vitro hypercoagulability is truly representative of what occurs in vivo   
remains to be established. However, the hypothesis that it may have clinical   
consequences is attractive and deserves attention. The possible consequences   
that we discuss herein include whether (i) cirrhosis is a condition   
associated with increased risk of venous thromboembolism or portal vein   
thrombosis; (ii) the hypercoagulability associated with cirrhosis has any   
other role outside coagulation (i.e. progression of liver fibrosis); and   
(iii) anticoagulation should be used in cirrhosis. Although apparently   
provocative, considering anticoagulation as a therapeutic option in patients   
with cirrhosis is now supported by a rationale of increasing strength. There   
may be subgroups of patients who benefit from anticoagulation to treat or   
prevent thrombosis and to slow hepatic fibrosis. Clinical studies are   
warranted to explore these therapeutic options. © 2011 International Society   
on Thrombosis and Haemostasis.  
  
RECORD 532  
TIPS in children  
Goffette P.P.  
CardioVascular and Interventional Radiology (2011) 34 SUPPL. 3 (451-453).   
Date of Publication: September 2011  
  
Learning Objectives: 1. To review the indications for TIPS in children 2. To   
describe the special technical considerations 3. To present the results and   
complications Pediatric experience with transjugular intrahepatic   
portosystemic stent-shunt (TIPSS), first described in 1992 by Kerns, is   
limited to case reports and small series and long-term results are lacking.   
Limiting factors to the wide-spread use of TIPSS in children include the   
lack of adequate pediatric device, technical difficulty because of the small   
size of the portal and hepatic veins and the presence of anatomical   
variants. Although the complications of portal hypertension in children are   
similar to those in adults, the underlying disease processes differ   
substantially. The main causes of portal hypertension in children are   
biliary atresia (40-45%), congenital hepatic fibrosis, a1-antitrypsin   
deficiency, mucovisidosis, Budd-Chiari syndrome, veno-occlusive disease and   
portal vein thrombosis. Current indications for TIPSS placement in children   
include control of acute and recurrent variceal bleeding (gastroesophageal,   
intestine, jejuno-jejunostomy or stoma) due to sinusoidal or post-sinusoidal   
portal hypertension, medically refractory ascites and hepatic hydrothorax.   
Although children requiring long-term treatment of complicated portal   
hypertension are more commonly considered for surgical portosystemic shunts   
because of an improved patency, possible long-term indications for TIPSS   
include congenital hepatic fibrosis due to polycystic kidney disease, cystic   
fibrosis and other conditions in which liver function may stabilize or   
improve with treatment, such as infectious or autoimmune hepatitis or   
cholangitis. TIPSS after liver transplantation is feasible but could be very   
challenging especially after split liver transplantation because of the risk   
of extrahepatic portal vein puncture. In this clinical setting, patency of   
the hepatic artery should be verified before TIPSS to avoid major ischemic   
complications. Special technical considerations: 1. Technical differences   
between pediatric patients and adults to perform TIPSS include, for the   
pediatric group the need for general anesthesia and for shorter size of   
metallic stent and technical modifications to access the portal vein. For   
the choice of the type of stent, anticipation of future liver growth and   
interference with liver transplantation should be kept in mind. 2. Smaller   
sheaths (<9 fr) and puncture systems (>16G) than those used for adults   
paradoxically increase the difficulty of the procedure in children because   
cirrhotic livers in children are rock-hard, especially in case of biliary   
atresia due to extensive fibrosis at the portal triad and the small size of   
the portal vein. Therefore, despite very small portal and hepatic veins in   
children, adults standard needles (Colapinto or Roesch-Uchida) are commonly   
used for TIPSS creation. Often a mismatch between needle size and vessel   
diameter necessitates ultrasound monitoring and subtle guidewire maneuvers   
to enter the portal vein. Another condition impending sometimes the portal   
vein puncture is the peripheral course of the major hepatic veins displaced   
by hyperplastic parenchymal nodules. 3. Various manoeuvers for visualizing   
the portal system could be useful in case of failed blind punctures of small   
or tortuous intrahepatic portal branches: - Transhepatic placement of a   
0.018-in wire into the portal vein under sonographic guidance. -   
Transabdominal ultrasound guidance of transjugular portal vein puncture. -   
Indirect portal venography by wedged hepatic vein injection using CO2, with   
combination of road-mapping or overlay function. - Transfemoral placement of   
a guidewire into the hepatic artery. - Transplenic portal vein targeting. 4.   
Alternative approaches in case of failed conventional jugular approach have   
been reported and include a. direct percutaneous transhepatic porto-hepatic   
connection under sonographic and/or fluoroscopic guidance, the so-called   
“Gun-sight technique” using two loop snares placed within an hepatic vein   
and a portal vein, b. direct connection between the suprahepatic IVC and the   
portal vein in BCS patient with hepatic vein and inferior vena cava   
thrombosis, c. combined direct percutaneous transhepatic and trans-femoral   
approaches. 5. Unlike adults for whom covered Viatorr stents (WL Gore) are   
usually and widely preferable, the strategy for stent selection in children   
depends on a variety of anatomic and clinical factors including the measured   
size of the main portal vein and hepatic vein on US, age less than 3 years   
and weight less than 30 kg. Because they are available in a wide range of   
size and length and could be initially under dilated and later completely   
dilate or even over dilated according to the liver growth, bare stents has   
been more commonly placed in children rather than covered stents. Both   
self-expanding (Wallstent, Boston Scientific) and balloon-expandable stents   
(Palmaz, Cordis, Johnson&Johnson) have been used, alone or in combination in   
order to combine flexibility and conformability of the first and the radial   
force of the second, especially in case of extensive portal fibrosis   
associated with biliary atresia. The combination of a covered stent placed   
within the parenchymal tract to avoid acute thrombosis and delayed stenosis   
with bare stents extending proximally and distally has been applied   
successfully in children. Appropriate stent positioning in relation to the   
portal and hepatic veins is important in potential liver transplant   
candidate. A malpositioned stent could increase the difficulty of   
transplantation by hampering vascular control or completion of the   
anastomosis to the hepatic or portal vein. There are few data on the   
clinical use of e-PTFE covered stent for the management of portal   
hypertension in children. Covered stentgraft has been used to treat   
TIPSS-biliary fistula and to revise previously malfunctioning shunt created   
with a bare stent in 2 patients. Recently, Mermuys reported a good   
medium-term patency of e-PTFE-covered stent TIPSS in 4 children. The   
advantageous use of conventional covered Viatorr stent in children by   
reducing the high rate of restenosis, reported up to 89% at 7 months, with   
bare stent is counterbalanced by the required minimal diameter of the portal   
vein and the nitinol skeleton which cannot be over dilated during the growth   
of the liver. The improved medium- and long-term patency of e-PTFE covered   
stent could avoid the need for repeated shunt revision under general   
anesthesia and, in the case of stable liver function, liver transplantation   
could be postponed or even cancelled, especially in case of acute or   
subacute Budd-Chiari syndrome. Placement of a second parallel TIPSS in order   
to accommodate the increased portal venous flow with growth may be an   
alternative technique. 6. Children submitted to TIPSS should have close   
follow-up including Doppler ultrasound at 1 day, 1 week, 3 months and then   
at every 6-month interval, so that eventual stenosis or occlusion can be   
diagnosed early. Oral anticoagulation is recommended for at least 3 months,   
and ideally maintained forever or until liver transplantation in case of   
small TIPSS with diameter less than 8 mm. Results Published procedural   
success rate in children ranges from 78 to 98% (80% after the first   
attempt), lower than in adults (95%). Complication rates in children are   
similar to those of adults except for an increase in the need for   
endovascular reintervention to maintain mid- and long-term patency. This   
fact results from small vessel size, from lower shunt diameter (6 to 9 in   
children versus 9 to 12 in adults) and the preferential use of bare stent   
instead of covered stent. Other expected complications of TIPSS in children   
may be technical problems, such as intraperitoneal bleeding, biliary   
fistula, injury to the vessels or inappropriate size during growth. Hepatic   
encephalopathy appears to be less problematic in children (15%) than in   
adults (20-30%). The reason for this difference is not clear but could be   
related to more favourable circulatory or central nervous system adaptation   
to the changes incurred by the procedure. To our knowledge, TIPSS placement   
for the treatment of complication of portal hypertension in children has   
been reported in 81 patients (7 series including 3 to 12 patients and 31   
case reports). Refractory or recurrent variceal bleeding is the primary   
indication for portal decompression in more than 90%. The reported technical   
success rate is 92, including the need for a second attempt in 11 patients.   
The clinical success rate ranges between 86 and 93% in terms of controlling   
variceal bleeding. Refractory ascites was improved in 75% of patients.   
Thrombocytopenia due to severe hypersplenism is consistently improved in   
only one-third of patients. This indication is still controversial. Early   
stenosis or occlusion need reintervention during the first month in 25%. 88%   
of children need reintervention during the first year follow-up. TIPSS   
serves as a bridge to elective liver transplantation in 49 patients (61%).   
No case of failed transplantation after TIPSS has yet been reported.   
Improvement of general condition after TIPSS in 8 children with preserved   
liver function has postponed or even obviated the need for transplantation.   
The definitive management for children with cirrhosis is liver   
transplantation. TIPSS placement provides a useful treatment bridge prior to   
transplantation, allowing for improving nutrition thereby making the patient   
a more suitable candidate for liver transplantation. However, the procedure   
is more difficult than in adults, especially in children with biliary   
atresia and/or advanced peri-portal fibrosis and the frequency of   
reinterventions is higher compared with adults. The use of covered stent,   
sometimes in combination with bare stents, seems feasible in children.  
  
RECORD 533  
Balloon-occluded retrograde transvenous obliteration (BRTO) of gastric   
varices in three patients with portal vein thrombosis  
Kim L. Kim S.K. Mani N.  
CardioVascular and Interventional Radiology (2011) 34 SUPPL. 3 (642-643).   
Date of Publication: September 2011  
  
Purpose: Transjugular intrahepatic portosystemic shunt (TIPS) is a   
well-established treatment for portal hypertension-related gastric varices.   
However, it may not be suitable for patients with portal vein thrombosis. We   
retrospectively reviewed the balloon-occluded retrograde transvenous   
obliteration (BRTO) of gastric varices in three patients with portal vein   
thrombosis. Material and Methods: We retrospectively reviewed three patients   
treated with BRTO of gastric varices from November 2009 to September 2010.   
Three patients (one man and two women; mean age, 50 years; range, 35-64   
years) were analyzed in the study. Indication of BRTO of gastric varices   
included iatrogenic complete portal vein thrombosis, complete portal vein   
thrombosis from pancreatitis and partial portal vein thrombosis from Crohn's   
disease. BRTO of gastric varices was performed in two patients due to prior   
significant hematemesis and in one patient for prophylaxis due to continuing   
anticoagulation medications. Technical and clinical success, and clinical   
outcome were analyzed. Results: BRTO of gastric varices was successfully   
performed in all patients without complications. Ethanolamine oleate was   
used as a sclerosing agent in one patient and 3% sodium tetradecyl sulfate   
(sotradecol) in two patients. Follow-up CT or MRI images (2-9 months) and   
endoscopy showed obliteration of gastric varices in all patients. There was   
no recurrent bleeding from the gastric varices during the follow-up period   
(4-11 months). Conclusion: BRTO of gastric varices can be a useful treatment   
for gastric varices in patients with portal vein thrombosis.  
  
RECORD 534  
Effect of low molecular weight heparin (LMWH) on thrombin generation (TG) in   
cirrhotic patients  
Rodriguez K.I. Rossetto V. Radu C. Gavasso S. Burra P. Simioni P. Senzolo M.  
Transplant International (2011) 24 SUPPL. 2 (143-144). Date of Publication:   
September 2011  
  
Introduction: Cirrhotics, including patients awaiting liver transplantation,   
may present thrombotic complications such as portal vein thrombosis, that   
warrant anticoagulation therapy to prevent extension into the splanchnic   
vessels, which can jeopardize transplantation. However, due to the reset   
hemostatic balance in cirrhotics, the anticoagulant effect of LMWH could   
differ from the one expected. Aim: To evaluate in vitro the effect of LMWH   
on TG in cirrhotics at different stages of liver disease with respect to   
antithrombinIII (ATIII) levels. Methods: Thirty cirrhotics (10 ChildA, 10   
ChildB, 10 ChildC) without HCC or known thrombophylic genetic defects, 10   
type1-ATIII-defect patients, and 10 healthy subjects were included in the   
study. ATIII activity was determined for every subject. TG on PPP, with   
determination of endogenous thrombin potential (ETP), was performed at basal   
conditions and with enoxaparin at 0.35UI/mL anti-Xa activity. The effect of   
LMWH was expressed in terms of ETP ratio at 0.35UI/ML (0.35ETP ratio), and   
was calculated by dividing ETP with LMWH by ETP in native plasma. Results:   
Mean±SD ATIII activity levels in cirrhotics were 75±25%, 55.3±22%, and   
41.1±13.6%, for Child A, B, and C patients, respectively, in contrast with   
51±6.8% for ATIII-defect patients. The decrease in ATIII activity was   
statistically significant in all cirrhotics compared to controls   
(104.9±8.6%,p<.001). 0,35ETP ratio was significantly lower in cirrhotic   
patients compared to controls (0.26±0.1 vs 0.48±0.1,p<.001), reduced   
parallel to increasing disease severity. There was a direct correlation   
between 0.35ETPratio and ATIII (r=.64,p=.001). Conclusions: Cirrhotic   
patients show an increasing response to LMWH parallel to increasing severity   
of liver disease, despite a decreasing level of ATIII. Clinically, LMWH dose   
adjustment should be considered in cirrhotic patients according to the Child   
class.  
  
RECORD 535  
Symptomatic and incidental thromboembolism are both associated with   
mortality in pancreatic cancer  
Menapace L.A. Peterson D.R. Berry A. Sousou T. Khorana A.A.  
Thrombosis and Haemostasis (2011) 106:2 (371-378). Date of Publication: 2011  
  
Pancreatic cancer is known to be associated with VTE, but contemporary rates   
of incidental and symptomatic VTE events and their association with   
mortality are incompletely understood. We conducted a retrospective cohort   
study of consecutive pancreatic adenocarcinoma patients at the University of   
Rochester from 2006-2009. Data were analysed using a Cox model with   
time-dependent covariates. A total of 1,151 radiologic exams of 135 patients   
were included. Forty-seven patients (34.8%) experienced VTE including 12   
pulmonary emboli (PE), 28 deep-vein thromboses (DVTs) and 47 visceral vein   
events. Incidental events comprised 33.3% of PEs, 21.4% of DVTs and 100% of   
visceral VTE. Median (95% CI) conditional survival beyond three months was   
233 (162-322) more days for those without VTE, which was significantly   
greater than 12 (3-60) days for those with DVT as first event (p<0.0001) and   
87 (14-322) days with visceral first events (p=0.022). In multivariate   
analysis, DVT (HR 25, 95% CI 10-63, p <0.0001), PE (HR 8.9, 95% CI 2.5-31.7,   
p = 0.007) and incidental visceral events (HR 2.6, 95% CI 1.6-4.2, p   
=0.0001) were all associated with mortality, though anticoagulants reduced   
these risks by 70% (26-88%, p = 0.009). In conclusion, VTE occurs in over   
one-third of contemporary pancreatic cancer patients and, whether   
symptomatic or incidental, is strongly associated with worsened mortality.   
The role of anticoagulation in treating incidental or visceral VTE warrants   
further study. © Schattauer 2011.  
  
RECORD 536  
Long-term follow-up of liver transplantation for budd-chiari syndrome with   
antithrombotic therapy based on the etiology  
Chinnakotla S. Klintmalm G.B. Kim P. Tomiyama K. Klintmalm E. Davis G.L.   
Trotter J.F. Saad R. Landaverde C. Levy M.F. Goldstein R.M. Stone M.J.  
Transplantation (2011) 92:3 (341-345). Date of Publication: 15 Aug 2011  
  
Background: Because myeloproliferative disorders (MPDs) are a frequent cause   
of Budd-Chiari syndrome (BCS), treatment directed toward altering platelet   
production and function may be more rational and effective than   
anticoagulation after liver transplantation. Methods: We reviewed data on 25   
patients who received liver transplantation for BCS at our institution from   
1987 to 2007. Posttransplant antithrombotic treatment was based on the cause   
of BCS: 17 patients with MPDs received hydroxyurea/aspirin; 5 received   
warfarin; and 3 (2 whose hypercoagulable disorder was corrected and 1 with   
sarcoidosis) received no therapy. RESULTS.: Both graft survival (88% at 5   
years) and patient survival (92% at 5 years) were superior in the BCS group   
compared with the 2609 patients who received liver transplants for other   
indications. Vascular complications included three instances of hepatic   
artery stenosis (NS compared with non-BCS liver recipients), one of portal   
vein thrombosis (nonsignificant [NS]), and one of portal vein stenosis (NS).   
All 25 patients underwent multiple liver biopsies with no bleeding   
complications. Conclusions: Using hydroxyurea and aspirin to treat patients   
with BCS caused by an MPD seems to be safe and effective and avoids the   
risks of anticoagulation with warfarin. © 2011 by Lippincott Williams &   
Wilkins.  
  
RECORD 537  
Menstrual problems and contraception in women of reproductive age receiving   
oral anticoagulation  
Huq F.Y. Tvarkova K. Arafa A. Kadir R.A.  
Contraception (2011) 84:2 (128-132). Date of Publication: August 2011  
  
Background: Oral anticoagulation is associated with increased bleeding   
complications. The aim of this study was to assess the changes in menstrual   
loss and pattern in women taking anticoagulant treatment. Study Design:   
Women on oral anticoagulant (OA) treatment at the Royal Free Hospital were   
interviewed and completed a questionnaire about their menstrual cycle before   
and after commencing oral anticoagulation treatment. They were then asked to   
complete a pictorial bleeding assessment chart (PBAC) during their next   
menstrual bleeding episode. Results: Fifty-three women between the ages of   
20 and 50 years participated in the study. Of these, 47 women completed a   
PBAC. The mean duration of menstruation increased from 5 days before   
starting OA therapy to 7 days after the commencement of treatment.   
Thirty-one (66%) of the 47 women who completed the PBAC had a score that was   
greater than 100. The number of women who experienced flooding or clots   
during menstruation and intermenstrual or postcoital bleeding also   
increased. In total, 29 (54.7%) women changed their method of contraception   
during OA treatment. Seventeen women who did not want to become pregnant   
were not using contraception, including 10 women who were on hormonal   
contraception prior to starting anticoagulant therapy. Conclusion: Women of   
reproductive age experience heavy and prolonged menstrual bleeding whilst on   
OA therapy. Women of reproductive age on OA therapy should be monitored for   
menstrual disorders to ensure that prompt and appropriate treatment is   
instituted. Advice about appropriate contraception should also be part of   
the medical care provided for these women. Barrier contraception,   
sterilization and progestin-only contraception are all suitable methods of   
contraception in this patient group. © 2011 Elsevier Inc. All rights   
reserved.  
  
RECORD 538  
Randomized controlled trial to investigate the impact of anticoagulation on   
the incidence of splenic or portal vein thrombosis after laparoscopic   
splenectomy  
Wang H. Kopac D. Brisebois R. Sample C. Shapiro A.M.J.  
Canadian Journal of Surgery (2011) 54:4 (227-231). Date of Publication:   
August 2011  
  
Background: Splenic and portal vein thrombosis (SPVT) is a potentially   
lifethreatening complication of splenectomy. There is a paucity of studies   
examining the role of prophylactic pre- and postoperative anticoagulation in   
the prevention of this complication. We designed a prospective randomized   
controlled trial (RCT) to more rigorously address the impact of prophylactic   
anticoagulation on the incidence of asymptom atic or symptomatic SPVT,   
detected on Doppler ultrasound, after lapa - roscopic splenectomy. Methods:   
This 2-centre, phase II, prospective, open-label, parallel-assignment RCT   
compared no postoperative anticoagulation to a regimen of 40 mg of   
enoxaparin subcutaneously once daily for 21 days. All patients underwent   
Doppler ultrasonography of the splenoportal system preoperatively and again   
14-28 days after surgery to screen for nonocclusive or occlusive thrombosis.   
Results: From November 2006 to November 2008, 35 patients were enrolled in   
the RCT. Four patients withdrew, 1 required conversion to an open procedure   
and 1 died at 3 months (the cause of death was not related to the study). Of   
the 29 patients remaining, 15 were randomly assigned to the anticoagulation   
group and 14 to the nonanticoagulation group. One (3.4%) patient in the   
treatment group experienced portal thrombosis. Rates of postoperative   
bleeding were similar in both groups. Conclusion: This RCT of   
anticoagulation found a low overall risk of SPVT after laparoscopic   
splenectomy; however, this is an underpowered study, and further   
multicentred clinical trials are needed. © 2011 Canadian Medical   
Association.  
  
RECORD 539  
Impact of pre-transplant liver hemodynamics and portal reconstruction   
techniques on post-transplant portal vein complications in pediatric liver   
transplantation: A retrospective analysis in 197 recipients  
De Magnée C. Bourdeaux C. De Dobbeleer F. Janssen M. Menten R. Clapuyt P.   
Reding R.  
Annals of Surgery (2011) 254:1 (55-61). Date of Publication: July 2011  
  
Background and Objective: Portal vein (PV) complications are the most   
frequent vascular complications in pediatric liver transplant (LT). We   
hypothesized that pre-LT liver hemodynamic parameters and PV reconstruction   
technique could predict the risk of PV complications post-LT. Methods: Three   
hundred seventy-three children had a primary LT. A detailed ultrasound study   
of the pre-LT native liver hemodynamics was available in 198 cases, with   
details of PV anastomosis available for 197 of these: end-to-end anastomosis   
(n = 146, 74%), interposition vein graft technique (n = 28, 14%), or   
portoplasty (latero-lateral anastomosis of vein graft and recipient PV) (n =   
23, 12%). Results: Overall 5-year patient survival rate was 90%. Among the   
198 patients with pre-LT hemodynamic data, 79 (40%) had PV hypoplasia   
(diameter ≤4 mm), 64 (32%) had a pathological portal flow (nonhepatopetal   
flow), and 47 (24%) had an arterial resistance index (ARI) ≥1. Abnormal   
hemodynamics were mostly observed in biliary atresia (BA). Among these 3   
parameters, only ARI ≥1 was significantly correlated with a higher rate of   
PV complications post-LT (P = 0.041). PV complication-free survival at 5   
years were 91% for end-to-end anastomosis, 91% for portoplasty, and 62% for   
interposition vein graft technique (P = 0.002). At multivariate analysis,   
the use of an interposition vein graft was the only factor to be   
significantly associated with a higher rate of PV complications post-LT (P =   
0.003). Conclusions: PV hypoplasia with liver hemodynamic disturbances was   
mainly observed in BA. Hepatic ARI ≥1 might be a good predictor of PV   
complications post-LT. Latero-lateral portoplasty seemed to provide the best   
results when end-to-end anastomosis is not feasible. Copyright © 2011 by   
Lippincott Williams & Wilkins.  
  
RECORD 540  
Algorithm for the management of portal vein thrombosis: A prospective study   
in patients with liver cirrhosis  
Sartori M.T. Senzolo M. Rossetto V. Burra P. Cillo U. Boccagni P. Gasparini   
P. Tsochatzis E. Simioni P. Burroughs A.K.  
Journal of Thrombosis and Haemostasis (2011) 9 SUPPL. 2 (400). Date of   
Publication: July 2011  
  
Background: There is no established management algorithm for the treatment   
of portal vein thrombosis (PVT) in patients with cirrhosis. We aimed to   
prospectively evaluate the use of anticoagulation and transjugular   
intrahepatic portosystemic shunt (TIPS), as a second line option, to treat   
PVT. Methods: Patients with cirrhosis and with non malignant PVT were   
included. anticoagulation with LWMH was considered in all; TIPS was   
indicated if there was a concomitant of portal hypertensive complication, or   
if thrombosis progressed. Patients seen in the same period, but who were not   
anticoagulated neither received TIPS, were included as controls. Results:   
Fifty-six patients were included (21 were controls). In the study group, PVT   
was occlusive in 11/35 with extension to the superior mesenteric vein or   
splenic vein in 13/35. anticoagulation was initiated in 33 patients. Mean   
follow-up ± SD was 21.6 ± 8.5 and 24.5 ± 8.2 months for study and control   
groups, respectively. Complete recanalization rate was 36% (12/33) in the   
treatment group compared to one among controls (P < 0.001). A short time   
interval between appearance of thrombosis and anticoagulation (< 6 months)   
strongly predicted chance of repermeation. During the follow-up there was   
progression of thrombosis in 15/21 who were not anticoagulated and in 5/33   
anticoagulated patients (P < 0.001). TIPS was placed in six patients. There   
were five patients with variceal bleeding and two intestinal venous infarcts   
in the control group, compared to one variceal bleeding episode in the study   
group. Conclusions: A treatment algorithm with anticoagulation and the use   
of TIPS in patients with PVT and cirrhosis achieved a good chance of   
complete repermeation, reduced portal hypertensive complications and   
decreased the rate of thrombosis progression. This should lead to improved   
survival and render liver transplantation less difficult.  
  
RECORD 541  
Health care expenditures and therapeutic outcomes of a pharmacist-managed   
anticoagulation service versus usual medical care  
Hall D. Buchanan J. Helms B. Eberts M. Mark S. Manolis C. Peele P. Docimo A.  
Pharmacotherapy (2011) 31:7 (686-694). Date of Publication: July 2011  
  
Study Objective. To evaluate the differences in health care expenditures and   
therapeutic outcomes of patients receiving warfarin therapy management by a   
pharmacist-managed anticoagulation service compared with those receiving   
warfarin management by usual medical care. Design. Retrospective,   
matched-cohort study. Data Source. University of Pittsburgh Medical Center   
(UPMC) and UPMC Health Plan. Patients. Three hundred fifty adults who   
received warfarin therapy; 175 were managed by the pharmacist-managed   
anticoagulation service for at least 2 months between October 1, 2007, and   
September 30, 2008, (case patients) and 175 received usual care (matched   
comparison group). Measurements and Main Results. Medical claims data   
compared were direct anticoagulation cost and overall medical care costs,   
anticoagulation-related adverse events, hospitalizations and emergency   
department visits, frequency of international normalized ratio (INR)   
testing, and quantity of warfarin refills. Operational costs of the   
anticoagulation service were also calculated. The INR values and time within   
therapeutic range were assessed through anticoagulation service reports and   
laboratory results. The direct anticoagulation care cost was $35,465 versus   
$111,586 and the overall medical care cost was $754,191 versus $1,480,661   
for the anticoagulation service group versus the usual care group.   
Accounting for operational and drug expenditure costs, the cost savings was   
$647,024 for the anticoagulation service group. The anticoagulation service   
group had significantly fewer anticoagulation-related adverse events (14 vs   
41, p<0.0001), hospital admissions (3 vs 14, p<0.00001), and emergency   
department visits (58 vs 134, p<0.00001). The percentage of INR values in   
range and the percentage of time the INR values were in range were   
significantly higher in the anticoagulation service group (67.2% vs 54.6%,   
p<0.0001, and 73.7% vs 61.3%, p<0.0001, respectively). Compared with the   
usual care group, the anticoagulation service group had significantly more   
INR tests performed but demonstrated no significant difference in the   
quantity of drug refills. Conclusion. After accounting for operational   
costs, pharmacist-managed anticoagulation leads to reduced health care   
expenditure while improving therapeutic outcomes compared with usual medical   
care.  
  
RECORD 542  
Acute hepatic vascular complications  
Ochs A.  
Internist (2011) 52:7 (795-803). Date of Publication: July 2011  
  
Acute hepatic vascular complications are rare. Acute portal vein thrombosis   
(PVT) and the Budd-Chiari syndrome (BSC) are the leading causes.   
Coagulopathy and local factors are present in up to 80% of cases. Diagnosis   
is established by colour-coded Doppler sonography, contrast-enhanced   
computed tomography or magnetic resonance imaging. Patients with acute PVT   
present with abdominal pain and disturbed intestinal motility. In the   
absence of cirrhosis anticoagulation with heparin is established followed by   
oral anticoagulation. In severe cases, surgical thrombectomy or transjugular   
thrombolysis with stent shunt may be necessary. Acute or fulminant BCS may   
require emergency liver transplantation or a transjugular intrahepatic   
portosystemic stent shunt, if patients present with acute liver failure.   
Milder cases receive anticoagulation for thrombolysis of occluded hepatic   
veins. Sinusoidal obstruction syndrome (SOS) is diagnosed after total body   
irradiation or chemotherapy, the term SOS replacing the former   
veno-occlusive disease. The treatment of congenital vascular malformations,   
complications in the setting of OLTX as well as patients with hepatic   
involvement of hereditary hemorrhagic telangiectasia requires significant   
expertise in a multidisciplinary approach. © 2011 Springer-Verlag.  
  
RECORD 543  
portal vein thrombosis as complication of romiplostim treatment in a   
cirrhotic patient with hepatitis C-associated immune thrombocytopenic   
purpura  
Dultz G. Kronenberger B. Azizi A. Mihm U. Vogl T.J. Sarrazin U. Sarrazin C.   
Zeuzem S. Hofmann W.-P.  
Journal of Hepatology (2011) 55:1 (229-232). Date of Publication: July 2011  
  
Background & Aims: Thrombopoietin receptor agonists are a new class of   
compounds licenced for the treatment of immune thrombocytopenic purpura.   
They are currently being studied for patients with thrombopenia in advanced   
liver disease or under therapy for hepatitis C. There are indications that   
the risk for development of portal vein thrombosis in patients with advanced   
liver cirrhosis might be increased under therapy with thrombopoietin   
receptor agonists. We report a case of a patient with Child class B liver   
cirrhosis with concurrent immune thrombocytopenic purpura that developed   
portal vein thrombosis under therapy with the thrombopoietin receptor   
agonist romiplostim. Methods: A 50-year-old woman with hepatitis C virus   
associated immune thrombocytopenic purpura and Child class B liver cirrhosis   
presented in our emergency with rapidly evolving hydropic decompensation and   
general malaise. For immune thrombocytopenic purpura, the patient was   
started on the thrombopoietin receptor agonist romiplostim nine months ago.   
Results: During hospitalization, the platelet count was measured above   
330,000/μl and partial portal vein thrombosis was diagnosed by imaging   
studies. The thrombotic event was assumed to be associated with the   
romiplostim treatment for immune thrombocytopenic purpura via excessive   
elevation of platelet count. After anticoagulation with heparin and   
cessation of romiplostim treatment, complete recanalisation of the portal   
vein was achieved. Conclusions: We conclude that romiplostim should be used   
with precaution in patients with hepatitis C-associated immune   
thrombocytopenic purpura and advanced liver cirrhosis as the risk for   
thrombotic complications may increase significantly. © 2010 European   
Association for the Study of the Liver. Published by Elsevier B.V. All   
rights reserved.  
  
RECORD 544  
Antiphospholipid syndrome in patient with portal venous thrombosis: Case   
report  
Damjanovska L. Rajcevski R.  
Macedonian Journal of Medical Sciences (2011) 4:2 (192-195). Date of   
Publication: Jun 15 2011  
  
Antiphospholipid syndrome (APS) is defined by the presence of arterial and   
venous thrombosis, recurrent fetal death, cerebrovascular accidents,   
hemolytic anemia, thrombocytopenia and various manifestations on different   
organs in the presence of anticardiolipin antibodies (aCL) and or lupus   
anticoagulant (LA). It was reported in early 1980's. This syndrome is the   
most common cause of acquired thrombophilia. There is no consensus for   
treatment among physicians. Overall there is a general agreement that   
patients with recurrent thrombotic episodes require life-long   
anticoagulation therapy and those with recurrent spontaneous abortions   
require anticoagulation therapy (low molecular weight heparin) and low dose   
aspirin during most of gestation. Immunosuppresion seems to be ineffective   
exept in patients with fulminate multiple organ failure i.e. catastrophic   
antiphospholipid syndrome where plasmapheresis can also be used. We present   
a case of 31 year old woman with primary APS and portal venous thrombosis   
(PVT), without any recognizable autoimmune disease. She has 4 spontaneous   
abortions, calf thrombosis, gangrene of one toe, refractory cutaneous ulcer   
on the heel and livedo reticularis. She is positive for aCL and LA, with   
hypergammaglobulinemia. © Damjanovska L.  
  
RECORD 545  
Impact of splenectomy at the time of liver transplantation on posttransplant   
outcome  
Onaca N. Tomiyama K. McKenna G.J. Cavaness K.M. Ruiz R.M. Asolati M. Campsen   
J. Jennings L.W. Goldstein R.M. Levy M.F. Klintmalm G.B.  
Liver Transplantation (2011) 17 SUPPL. 1 (S277-S278). Date of Publication:   
June 2011  
  
Splenectomy is performed selectively at the time of liver transplantation   
(LTX). Concerns with splenectomy include portal vein thrombosis (PVT), other   
thrombotic events, and infections including overwhelming sepsis. Our aim was   
to study the outcome of patients who underwent splenectomy at LTX. Data for   
2603 adult LTX recipients at one institution from 1985-2008 were reviewed   
retrospectively; 69 underwent splenectomy at with LTX (2.7%). Liver disease   
etiology showed that splenectomized patients had more autoimmune disease   
(11.6% vs 3.8%), less hepatitis C (26.1% vs 36.2%) and less Laennec   
cirrhosis (4,4% vs 12.4%) than non-spienectomized patients (p=0.GO74).   
Kaplan Meier patient and graft survivals were not statistically different (p   
0.1833 and p 0.0857, respectively). There was no significant difference at   
any time post LTX in the incidence oTacute cellular rejection   
(p=0.81-Q.9)andof steroid-resistant rejection (p=0.l 3-0.19) between groups.   
The overall incidence of infections/septic shock, was not significantly   
different. However, splenectomized patients had significantly more hepatic   
abscesses (10.1% vs 2.6%, p=0.0027), urinary tract infections at any time   
post LTX (30.4% vs 18.7%, p=0.OI92), and pancreatitis (11.6% vs 5%).   
Splenectomized patients had more thrombotic complications: PVT (14.5% vs   
2.4%, pO.0001), deep venous thrombosis {10.1% vs 4.1%, p=0.0258), and   
pulmonary embolism (4.4% vs 1.1%, p=0.0511). PVT occurred within 3 months   
from transplant in uon-splenectomized patients, while it occurred both   
earlier and later than 3 months in splenectomized patients Splenectomy at   
the time of LTX is relatively safe, with no significant impact in the   
patient and graft survival, the rejection rate and the overall infection   
rate. The higher incidence of thrombotic events, and in particular PVT,   
calls for systematic use of antiaggreganl treatment and consideration of   
anticoagulation in splenectomized patients.  
  
RECORD 546  
portal vein thrombosis associated with ischemic colitis  
Kobayashi A. Mizumoto H. Ando T. Matsutani S.  
Clinical Journal of Gastroenterology (2011) 4:3 (147-150). Date of   
Publication: June 2011  
  
We report the case of a 52-year-old male who was admitted for sudden   
abdominal pain and hematochezia. Colonoscopy showed erosion and edema in the   
mucosa of the descending colon, leading to a diagnosis of ischemic colitis.   
Blood tests revealed hepatic dysfunctions. Using abdominal ultrasonography   
(US), thrombus was observed in the left branch of the portal vein and a part   
of the right branch. Although the Doppler method detected blood flow in the   
right branch, no blood flow signal was observed in the left branch. Since   
coagulation examinations were almost normal, and there was no past history   
of liver cirrhosis or malignancy, it was diagnosed to be portal vein   
thrombosis (PVT) associated with ischemic colitis. anticoagulation therapy   
was initiated for PVT. According to the results of the US and abdominal   
computed tomography performed 3 months after starting the treatment,   
thrombus in the right branch had diminished but remained in the umbilical   
region of the left branch. Due to atrophy of the lateral segment of the   
liver, we terminated the treatment. Ischemic colitis is not a rare disease;   
however, when accompanying hepatic dysfunction, it is necessary to take the   
complications associated with PVT into consideration. © 2011 Springer.  
  
RECORD 547  
Splachnic vein thrombosis and myeloproliferative syndromes. The role of   
JAK2V617F mutation  
Kanellopoulou T. Alexopoulou A. Kontopidou F. Koskinas J. Pectasides D.  
Haematologica (2011) 96 SUPPL. 2 (549-550). Date of Publication: 1 Jun 2011  
  
Background. Myeloproliferative diseases(MPDs) are shown to have an increased   
risk of thrombotic complications such as splachnic vein thrombosis(   
SVT).Mutations on JAK2 pathway are thought to play key role on such   
thrombotic complications. Aims. The focus of the current work is to evaluate   
the risk of SVT in MPDs patients and its colleration with the mutation   
JAK2V617F. Methods. Patients with non-cirrhotic, non-cancer related SVT and   
with clinical or laborating findings suggesting MPD were assessed for the   
presence of JAK2V617F mutation. We suspected that normal or light increased   
platelet count might mask MPDs (portal hypertension-hypersplenism, occult   
bleeding).Assessment for hematological pro-coagulant conditions included   
factor V Leiden, antithrombin III, protein C, protein S, homocysteine, MTHFR   
mutation, prothrombin gene mutation PT20210A, anticardiolipin antibodies and   
lupus anticoagulant. Paroxysmal nocturnal hemoglobinuria was screened using   
standard flow cytometry techniques. Patients with known history of   
pylephlebitis were excluded. SVT was confirmed with computerized tomography   
and abdominal doppler ultrasound. SVT was characterized as chronic if there   
was evidence of intra-abdominal venous collaterals, carvenous transformation   
of the portal vein, or signs of portal hypertension. 4.Results:In the study   
14 patients were included. The median age at the time of diagnosis was 50.71   
years (range, 21-78) and 57% were male. All patients had chronic SVT, 64%   
had PVT and the rest were diagnosed with BCs. Every patient underwent bone   
marrow biopsy: polycythemia vera(PV) 4 patients, essential   
thrombocytosis(ET) 7 patients, primary myelofibrosis(PMF) 3 patients.   
JAK2V617F was analyzed in 12/14 patients and was positive in 100%. Inherited   
thrombophilia was not found. Acquired thrombophilia was mentioned in two   
patients. A woman with Budd-Chiari syndrome(BCs) who was provided oral   
contraceptive pills, and a man with portal vein thrombosis(PVT)   
postsplenectomy. Patients with BCs had mean age 43.2 years(range, 35-56) and   
60% were female. Three were diagnosed with PV, 1 ET and 1 PMF. One patient   
died after 17 years and one was scheluded for liver transplantation after 6   
years. The other three patients had no signs of ascites or portal   
hypertension in a six-year follow up. Patients with PVT had mean age 54.8   
years(range, 21-78) and 67% were male. Six were diagnosed with ET, 2 PMF and   
1 PV. On admission 5 patients had esophageal/gastric varices whereas 89%   
patients had splenomegaly. Five patients had also evidence of superior   
mesenteric vein thrombosis. Nobody died. All of the patients have signs of   
portal hypertension. Mean time of follow up is 1.8 years(range, 0.2-6). All   
patients were managed with routine anticoagulation therapy from diagnosis.   
Three patients had indications for decompressive procedures such as TIPS,   
all in the group of BCs. 5.Summary/Conclusions:SVT is frequent presenting   
complication of undiagnosed MPDs.In patients with SVT, portal hypertension   
is a virtually constant feature. The resulting hypersplenism and   
hemodilution decrease the accuracy of blood cell counts for MPD diagnosis.   
The atypical peripheral blood picture in the setting of SVT has led to a   
variety of denominations such as latentMPDs. In our study, all patients with   
MPD and SVT were positive for the mutation JAK2V617F.The presence of this   
mutation may predict a more aggressive phenotype with an increased risk of   
thrombosis.  
  
RECORD 548  
Antithrombotic therapy in non-neoplastic chronic portal venous thrombosis in   
cirrhosis: Recanalization and liver function evaluation  
Bento De Miguel L. Rodriguez-Huerta A. Pascual C. Pérez-Rus G.   
Gonzalez-Gascón Y Marín I. Carretero F. Infante M. Roldán A. Catalina V.   
Yepes I. Kwon M. Díez-Martín J.L.  
Haematologica (2011) 96 SUPPL. 2 (75-76). Date of Publication: 1 Jun 2011  
  
Introduction. Non-neoplastic chronic portal vein thrombosis (PVT) is a   
frecuent diagnosis in the course of liver cirrhosis, with reported   
prevalences of 0.6% to 15,8%. PVT can motivate life-threatening   
complications due to worsening portal hypertension, so anticoagulation   
therapy is challenging in these patients. OBJECTIVE: To analyze the response   
to antithrombotic therapy and changes in liver function tests in 28 patients   
with chronic PVT associated with cirrhosis. Patients and Methods. 28   
consecutive patients with liver cirrhosis and chronic PVT were treated with   
antithrombotic therapy from 2004 to 2009. Hepatocellular carcinoma and known   
thrombophilic risks were ruled out. Therapy consisted in 15 days of   
therapeutic doses of low molecular weight heparin (LMWH) (enoxaparin)   
adjusted according to baseline coagulability (Table 1), followed by either   
prophylactic doses (40mg/day) of LMWH or acenocoumarol (target INR 2-3),   
during 6 months. Response was evaluated after 6 months. (Table presented) If   
recanalization was complete, therapy was suspended. If recanalization was   
partial or no recanalization was observed, therapy was continued until   
response. RESULTS: From the 28 patients studied, 19 (68%) were males with a   
median age of 53 years (range 35-77). cirrhosis was due to alcoholism (25%),   
virus (54%), mixed in 1 patient and other causes in 3 patients. PVT involved   
the portal trunk and/or branches in 19/28 (68%) patients, mesenteric vein in   
2 patients and portal trunk and/or branches, mesenteric and/or splenic vein   
thrombosis coexisted in 7 patients. 19/28 (68%) of the patients had moderate   
or moderate-severe hypocoagulability range. Complete and partial thrombosis   
was seen in 18 and 10 patients at diagnosis, respectively. From the 28   
patients, 18 (64%) responded to antithrombotic therapy after 6 months, with   
a complete recanalization in 13 patients 13/18 (72%) and partial in 5/18   
patients (28%). None of the 28 patients presented hemorrhagic complications   
and none showed platelets counts below baseline values. 17 from the 18   
patients who responded, showed altered liver function tests before therapy.   
After 6 months, 8/17 (47%) improved liver function (only one patient had   
received antiviral therapy). After a median follow up of 42 months (range   
7-67), 15/18 (83%) patients continued showing complete or partial response   
while 3 patients progressed. Of note, 3 patients of this group could proceed   
to further liver transplantation. Conclusions: Antithrombotic therapy in   
chronic PVT in cirrhotic patients resulted in a high response rate (64%) in   
our study, with a complete recanalization in 72% of the cases. Adjusted dose   
scheme according to level of hypocoagulability seems to be effective and   
safe, since 63% of the subgroups of moderate and moderate-severe   
hypocoagulability responded with no haemorrhagic complications.  
  
RECORD 549  
Abdominal venous thrombosis following inflammatory bowel disease related   
surgeries while on dalteparin prophylaxis. Case series  
Naik A.S. Zadvornova Y. Lundeen S.J. Stein D.J. Venu N. Otterson M.F. Issa   
M. Perera L.P.  
Gastroenterology (2011) 140:5 SUPPL. 1 (S432). Date of Publication: May 2011  
  
INTRODUCTION: Patients with Inflammatory Bowel Disease (IBD), including   
Crohn's disease (CD) and Ulcerative Colitis (UC), are at increased risk for   
venous thrombosis (VT). VT occurs more often in the deep veins of leg and   
pulmonary circulation, but has been described to occur less frequently in   
other sites including: portal vein and mesenteric veins. We report a case   
series of post-operative abdominal VT following a switch in Low Molecular   
Weight Heparin (LMWH) VT prophylaxis preparations from enoxaparin (EP) to   
dalteparin (DP) on 10/11/09 in our tertiary referral center. METHODS: A   
retrospective review of consecutive IBD patients undergoing IBD related   
surgery performed by a single surgeon at our IBD center between 2008-2010   
was performed. We recorded all surgeries 1 year before and after (LMWH)   
switch. We collected surgical reports and VT prophylaxis of all cases, and   
investigated rate of abdominal VT events. Events were diagnosed on CT of   
abdomen and pelvis performed in response to a change in clinical status (i.e   
abdominal pain, fever and elevated white cell count). RESULTS: A total of   
131 IBD-related surgeries were performed, all of whom received VT   
prophylaxis with LMWH. We identified 72 surgeries (55 small bowel   
resections, 16 colectomies, 1 J pouch excision) in the EP group and 59   
surgeries (40 small bowel resections, 19 colectomies) in the DP group. There   
were 7 cases of post-operative abdominal VT in the DP group, (3 males and 4   
females) compared to 0 cases in the EP group (p< 0.02; Fischer's Exact).   
Mean disease duration was 52 mo (range 1- 126 mo). 6/ 7 (86%) patients on   
oral steroids (5 patients on prednisone [mean 40mg (range 20-60mg)] and 1   
patient on entocort (9mg) prior to surgery. One female patient had a known   
antithrombin III mutation for which DP dosing guidelines were followed. Only   
2/7 patients were actively smoking at the time of surgery. Surgery types in   
the VT group were total abdominal colectomy with end ileostomy (1   
laparoscopic, 3 open; 1 CD and 3 UC); and small bowel resections for   
stricturing CD (n=3). VT locations were 4 pts with portal vein thrombosis, 2   
patients with superior mesenteric vein thrombosis and 1 pt with both veins   
involved. In VT cases, 5 patients were on biologics, 4 patients were on   
immunomodulators, and 3 patients were on 5-ASA products. CONCLUSIONS: Our   
series shows alarming numbers of abdominal VT in the post-operative period   
following IBD-related surgeries while on appropriate VT prophylaxis dosing   
with DP. Reasons for inadequate anticoagulation may be either inappropriate   
dosing or differences in anticoagulant effect as minor differences do exist   
between DP and EP. It is unclear if this is an IBD-related phenomenon. We   
suggest further investigation of this phenomenon and caution with DP use for   
VT prophylaxis in pre-operative IBD patients. .  
  
RECORD 550  
Acute portomesenteric venous thrombosis following abdominal surgery:   
Observe, anticoagulate or operate?  
Leonardi M.J. Hollander L.L. Pitt H.A. House M.G. Zyromski N.J. Max Schmidt   
C. Nakeeb A. Howard T.J. Lillemoe K.D.  
Gastroenterology (2011) 140:5 SUPPL. 1 (S1010-S1011). Date of Publication:   
May 2011  
  
Background: Portomesenteric venous thrombosis (PMVT) is a rare, but serious,   
complication of abdominal surgery with no agreed standard of care.   
Management options include observation, anticoagulation, and thrombectomy.   
Our study aims to characterize a large series of patients with PMVT after   
abdominal surgery with a focus on management and outcomes. Methods:   
Weperformed a retrospective analysis of more than 4000 patients having   
abdominal surgery at an academic medical center between January 2007 and   
August 2010. Patients with postoperative thrombosis of the portal, superior   
mesenteric, and/or splenic veins were reviewed. Transplant patients and   
those with preexisting PMVT were excluded. The diagnosis was established by   
computed tomography (CT), magnetic resonance imaging (MRI), and/ or duplex   
ultrasound. Results: Forty-four patients had PMVT (23 isolated portal, 19   
combined portal and mesenteric and/or splenic, 1 isolated mesenteric, 1   
isolated splenic). Average patient age was 59 years, and 55% were male. Four   
patients (9%) were on preoperative anticoagulation that was held for   
surgery, and 25 patients (57%) had a malignancy. Operations performed   
included pancreas resection (21), liver resection (5), colorectal resection   
(5), pancreas debridement (4), splenectomy (3), major biliary surgery (2),   
combined pancreas and colon resection (2), palliative gastrojejunostomy (1),   
gastrectomy (1), and abdominal wall reconstruction (1). Median time from   
operation to PMVT diagnosis was 14 days, and 23 patients were diagnosed   
after discharge from the original hospitalization. Diagnostic modalities   
included computed tomography (35), duplex ultrasound (8), and magnetic   
resonance imaging (1). Treatment included observation in 15 patients,   
anticoagulation in 24 patients (20 continued as outpatients), and operative   
thrombectomy in 5 patients. All patients who underwent operative   
thrombectomy developed PMVT on postoperative day 1 after their initial   
operation, and four of these patients had a portomesenteric venous resection   
and reconstruction as part of their initial operation. PMVT-induced liver   
abscess occurred in 1 patient. Small bowel and/or colon resection for   
ischemia occurred in 2 patients. Seventeen (40%) patients were readmitted   
within 30 days of discharge from the PMVT admission. Only one patient died   
five days after a liver resection complicated by portal vein thrombosis   
requiring operative thrombectomy and enterectomy. Conclusions:   
Portomesenteric venous thrombosis (PMVT) is an uncommon complication   
following abdominal surgery which may present early postoperatively, but   
often presents after discharge. While the morbidity associated with PMVT is   
high, the mortality is low (2%). Management should be tailored to individual   
patient characteristics with respect to timing and severity of presentation.  
  
RECORD 551  
Hypercoaguable state  
Sturtevant A.  
Journal of General Internal Medicine (2011) 26 SUPPL. 1 (S377-S378). Date of   
Publication: May 2011  
  
LEARNING OBJECTIVES: 1. Identify signs andsymptoms of portal vein   
thrombosis. 2. Know when ahypercoagulable state workup is appropriate. CASE   
INFORMATION: A 63 year-old woman presentedcomplaining of a five day history   
of intermittent sharp 10/10 epigastric painthat is worse after eating.   
Herabdomen was slightly tender to palpation and there was no hepatomegaly,   
splenomegaly,distension, ascites, or lower extremity edema. She had a   
history of a cerebrovascular accident two yearsprior. No history of clotting   
ormiscarriages.A CT of the abdomen revealedocclusive thrombosis of the   
splenic vein with non occlusive thrombus in theportal vein, short left   
gastric vein and superior mesenteric vein. The spleen was moderately   
enlarged. IMPLICATIONS/DISCUSSION: General internists frequentlyencounter   
patients with thrombosis and face the dilemma of when to pursue afull   
hypercoagulability workup. There is not a firm concensus regarding which   
patients to screen, butpatients with identifiable risk factors for   
thrombosis such as SLE, prolonged periods of inactivity, malignancy, recent   
surgery, myeloproliferative disorder,Heparin-induced thrombocytopenia,   
preeclampsia should not be screened. Screening should be undertaken   
inpatients if the patient has family history of thromboses in first   
degreerelatives; if the patient is younger than 50; the patient has   
recurrentthrombosis; a history of warfarin induced skin necrosis or if the   
patient hasunusual or extensive thromboses such as the portal vein (without   
anidentifiable precipitating cause). Once a decision to test for an   
underlying disorder has been made, it isimportant to consider which tests   
can be performed in the acute phase and onanticoagulation.   
Antiphospholipidantibodies (lupus anticoagulant, anticardiolipin antibody,   
and anti-B2glycoprotein), Factor V Leiden,and Prothrombin gene mutation can   
be screened for in the acute phase prior tostarting anticoagulation.   
Lupusanticoagulant should not be measured after starting heparin therapy   
orCoumadin. Other testing such asantithrombin deficiency, Factor VIII,   
Protein C and Protein S should not bemeasured until after the patient is out   
of the acute phase (generally 6 months)and off therapy. Age   
appropriatecancer screening should also be performed. When making the   
decision to perform testing for ahypercoagulable state internists should   
consider the appropriateness, timing, and the effect a positive test would   
have on the choice and duration of therapy.  
  
RECORD 552  
portal vein thrombosis following laparoscopic cholecystectomy complicated by   
dengue viral infection: A case report  
Dan D. King K. Seetahal S. Naraynsingh V. Hariharan S.  
Journal of Medical Case Reports (2011) 5 Article Number: 126. Date of   
Publication: 2011  
  
Introduction. portal vein thrombosis is an uncommon post-operative   
complication following abdominal surgery. Although therapeutic   
anticoagulation is recommended, this treatment may be questionable when the   
patient has an associated bleeding diathesis. Case presentation. We report a   
case of a 63-year-old woman of Asian Indian ethnicity who developed portal   
vein thrombosis following an uneventful laparoscopic cholecystectomy for   
symptomatic gallstones. Her condition was further complicated by dengue   
viral infection in the post-operative period, with thrombocytopenia   
immediately preceding the diagnosis of portal vein thrombosis. The   
etiological connections between dengue viral infection with   
thrombocytopenia, laparoscopic cholecystectomy, portal vein thrombosis as   
well as the treatment dilemmas posed in treating a patient with portal vein   
thrombosis with a bleeding diathesis are discussed. Conclusion: When portal   
vein thrombosis occurs in patients with contraindications to   
anticoagulation, there is a role for initial conservative management without   
aggressive anticoagulation therapy and such patients must be approached on   
an individualized basis. © 2011 Dan et al; licensee BioMed Central Ltd.  
  
RECORD 553  
Thromboembolism in inflammatory bowel disease: An insidious association   
requiring a high degree of vigilance  
Di Fabio F. Lykoudis P. Gordon P.H.  
Seminars in Thrombosis and Hemostasis (2011) 37:3 (220-225). Date of   
Publication: 2011  
  
Venous and arterial thromboembolism are both serious extraintestinal   
manifestations of inflammatory bowel disease (IBD). Acquired risk factors   
seem to play a more prominent role than congenital in promoting thrombotic   
events. Prevention of thromboembolism is thus mainly aimed at minimizing the   
acquired/reversible risk factors (e.g., inflammation, immobility,   
hospitalization, steroid therapy, central intravenous catheters, smoking,   
oral contraceptives, and deficiency of B vitamins and folate). The diagnosis   
of venous and arterial thromboembolism is extremely challenging and requires   
a high degree of vigilance. Deep vein thrombosis and pulmonary embolism may   
be clinically silent or manifest with only few specific symptoms. Thrombosis   
of the portal vein system may occur with nonspecific symptoms such as   
abdominal pain, nausea/vomiting, abdominal tenderness, ascites, and fever.   
The diagnosis of arterial thromboembolism may also be challenging,   
particularly when the splanchnic region is involved. Indeed, arterial   
thrombosis of the splanchnic region tends to be overlooked and   
misinterpreted as a clinical exacerbation of IBD. Early diagnosis plays a   
central role in optimizing the therapeutic intervention and reducing the   
risk of short-term and long-term thrombosis-associated complications. The   
decision regarding the duration of systemic anticoagulation must take into   
account the individual risk of intestinal bleeding. Copyright © 2011 by   
Thieme Medical Publishers, Inc.  
  
RECORD 554  
Risk factors of portal vein thrombosis in clinical islet transplantation  
Kawahara T. Kin T. Kashkoush S. Bigam D.L. Kneteman N.M. Shapiro A.M.J.  
American Journal of Transplantation (2011) 11 SUPPL. 2 (176). Date of   
Publication: April 2011  
  
Introduction: Islet transplantation improves glycemic control in Type1   
diabetes complicated by refractory hypoglycemia. Percutaneous transhepatic   
portal access avoids surgery, but is rarely associated with bleeding or   
portal vein thrombosis. Herein, we evaluate factors affecting portal   
pressure and risk factors of portal vein thrombosis post islet   
transplantation. Methods: We reviewed records of 278 intraportal islet   
transplant procedures in 127 patients (mean 2.19 infusions/patient). Portal   
venous pressure (mmHg) was measured by using a pressure transducer before   
and after completion of islet infusion. A doppler ultrasound was performed   
in 24 hours post transplantation for all cases to assess the complications   
such as portal vein thrombosis, hematoma or bleeding routinely. Results: The   
mean islet mass was 407,221 IE (5,908 IE/kg) with mean packed cell volume of   
4.1 mL (range: 1.5 - 7.9). Institution of therapeutic heparinization,   
effective catheter tract ablation with Avitene paste, and limiting packed   
cell volume to <5 mL has completely prevented this complication in 101 islet   
transplant procedures over the past 4.3 years. Univariate analysis revealed   
that standard liver volume correlated negatively with portal pressure rise   
(r=-0.257, P<0.01), with a larger liver volume experiencing less   
perturbation in portal pressure. Packed cell volume correlated positively   
with elevated portal pressure (r=0.463, P<0.01). Ten patients (3.6%)   
developed partial thrombosis of the intrahepatic portal vein (none since   
August 2006). Univariate analysis revealed that both portal pressure   
elevation (r=0.256, P<0.0001) and high packed cell volume (r=0.161, P<0.01)   
were risk factors for thrombosis. Packed cell volume <5.5 mL (sensitivity   
50%, specificity 84.5%) and portal pressure rise <4.5 mmHg (sensitivity 70%,   
specificity 73.2%) were founded to be cut offs to prevent portal vein   
thrombosis. Conclusions: Portal thrombosis is a preventable complication in   
clinical islet transplantation, provided therapeutic anticoagulation is   
maintained, and packed cell volume is limited to <5 mL.  
  
RECORD 555  
Outcome for patients who undergo splenectomy at the time of liver   
transplantation  
Onaca N. Tomiyama K. McKenna G.J. Cavaness K.M. Ruiz R.M. Asolati M. Campsen   
J. Jennings L.W. Goldstein R.M. Levy M.F. Klintmalm G.B.  
American Journal of Transplantation (2011) 11 SUPPL. 2 (151-152). Date of   
Publication: April 2011  
  
Splenectomy is performed selectively at the time of liver transplantation   
for various indications. Concerns with splenectomized patients in general   
include portal vein thrombosis, other thrombotic events, and infectious   
complications including overwhelming sepsis. Our aim was to study the   
outcome of patients who underwent splenectomy at the time of liver   
transplantation. Data for 2603 adult patients who underwent liver   
transplantation at a single institution between 1985-2008 were reviewed   
retrospectively. 69 patients underwent splenectomy at the time of   
transplantation (2.7%). Liver disease etiology showed that splenectomized   
patients, when compared to non-splenectomized patients, had more autoimmune   
disease (11.6% vs. 3.8%), less hepatitis C (26.1% vs 36.2%) and less Laennec   
cirrhosis (4.4% vs 12.4%)(p=0.0074). Kaplan Meier patient and graft   
survivals were not statistically different (p=0.1833 and p=0.0857,   
respectively). There was no significant difference at all time intervals   
posttransplant in the incidence of acute cellular rejection (p=0.81-0.9) and   
of steroid-resistant rejection (p=0.13- 0.19) between groups. The overall   
incidence of infections, including septic shock, was not significantly   
different. However, splenectomized patients had significantly more hepatic   
abscesses (10.1% vs 2.6%, p=0.0027), urinary tract infections at all times   
posttransplant (30.4% vs 18.7%, p=0.0192), and pancreatitis (11.6% vs 5%)   
than patients without splenectomy. Thrombotic complications were more   
frequent in splenectomized patients: portal vein thrombosis (14.5% vs 2.4%,   
p<0.0001), deep venous thrombosis (10.1% vs 4.1%, p=0.0258), and pulmonary   
embolism (4.4% vs 1.1%, p=0.0511). portal vein thrombosis occurred within 3   
months from transplant in non-splenectomized patients, while it occurred   
both earlier and later than 3 months in splenectomized patients. Splenectomy   
performed at the time of liver transplantation is relatively safe. There is   
no significant impact in the patient and graft survival, the rejection rate   
and the overall infection rate. The higher incidence of thrombotic events,   
and in particular portal vein thrombosis, calls for systematic use of   
antiaggregant treatment and consideration of anticoagulation in   
splenectomized patients.  
  
RECORD 556  
Portal and mesenteric vein thrombosis complicated by acute mesenteric   
ischemia  
Diepenhorst G. Van Golen R. Gilijamse P. Van Gulik T.  
HPB (2011) 13 SUPPL. 2 (78). Date of Publication: April 2011  
  
Introduction: portal vein thrombosis (PVT) and mesenteric venous thrombosis   
(MVT) are uncommon disease entities in adults. In a minority of cases, PVT   
and MVT lead to acute mesenteric ischemia (AMI). Little is known about the   
clinical importance of this complication in thrombotic patients. The aim of   
this study was to describe clinical outcomes and risk factors in patients   
with PVT and/or MVT complicated by AMI. Methods: Hospital records and   
clinical data of all patients with PVT and/or MVT between 1995 and 2010 were   
reviewed. Clinical features, laboratory findings, etiologic factors, and   
treatment modalities were recorded and assessed as possible risk factors for   
mortality. All deaths within 30 days of the start of treatment   
(surgery/anticoagulation) were considered to represent mortality. Results:   
Twenty-eight patients (16 male, 12 female) were identified as having PVT   
and/or MVT using ultrasound or CT scan. Clinical outcomes of 9 out of 28   
(32%) patients were complicated by acute mesenteric ischemia. One out of   
these 9 patients underwent an explorative laparotomy, 7 underwent bowel   
resection without revascularisation, and one patient was treated   
conservatively with anticoagulation. In those patients complicated by acute   
mesenteric ischemia, none demonstrated pre-existent coagulopathies, whereas   
those patients not complicated by AMI demonstrated preexistent   
coagulopathies in 25% of cases. Patients with PVT or MVT undergoing surgery   
for mesenteric ischemia demonstrated a 30-day mortality rate of 25%, whereas   
patients without mesenteric ischemia all survived 30 days after   
anticoagulatory treatment. No etiological or clinical independent predictor   
of mortality could be identified in the small study group. Conclusions: PVT   
and MVT complicated by acute mesenteric ischemia carries a substantial   
morbidity and mortality and should be considered as the underlying etiology   
in cases of AMI.  
  
RECORD 557  
Effect of portal vein thrombosis on liver transplant outcomes  
Parajuli S. Satoskar R. Agarwal N. Shetty K. Matsumoto C. Girlanda R.   
Johnson L. Fishbein T.  
American Journal of Transplantation (2011) 11 SUPPL. 2 (334). Date of   
Publication: April 2011  
  
Background: In the past, complete PVT had been considered an absolute   
contraindication to OLT. The purpose of this report was to analyze our   
experience in the management of PVT during OLT and to evaluate its effect on   
outcomes. Methods: We conducted a retrospective chart review of all patients   
with PVT transplanted between January 2006 to October 2010.Imaging was   
reviewed and patients were determined to have either partial or complete PVT   
involving the main portal vein. Age, gender, and MELD score matched controls   
were selected for comparison.Outcome measures including operative time,   
number of units of PRBCs required during OLT, graft and patient survival   
were analyzed. The use of modified surgical technique, presence of a defined   
hypercoagulable state, and use of post transplant anticoagulation or   
antiplatelet therapy were also recorded. Results: Of 408 patients who   
underwent OLT,18 patients(4.4%) were found to have pretransplant PVT. Three   
patients were excluded due to lack of available follow-up data. Baseline   
parameters including age, gender, MELD score, were similar between the two   
groups. Median follow-up was 429 d in the PVT group and 300 d in the control   
group. 12 of 15 patients in the PVT group had partial thrombosis while the   
other 3 had complete thrombosis. Surgical technique was modified in 8   
patients(53%). Antiplatelet or anticoagulant therapy was used in 8 patients   
post transplant with aspirin as the most commonly used agent. Mean operative   
time was 509 min and 481 min in the PVT and control groups(p=0.51). The mean   
volume of PRBCs transfused intaoperatively was 9.2 units and 5.3 units in   
the 2 groups(p=0.21).When comparing all patients with PVT to controls we   
found no effect of PVT on patient or graft survival. 2 patients(13.3%) with   
portal vein thrombosis died intraoperatively while no patients in the   
control group died (p=0.14). Both of these patients had complete PVT.   
Complete PVT was associated with higher mortality when compared to partial   
PVT and controls(67% vs. 0%, p=.03,.02). Conclusions: PVT had no significant   
effect on operative time and transfusion requirements.Patients with complete   
PVT had higher mortality,although the numbers were too small to draw   
definitive conclusions.Current outcomes of OLT in the setting of PVT are   
acceptable and this should not of itself be a contraindication to listing.   
Patients with complete PVT may be at increased risk of intraoperative death.  
  
RECORD 558  
Obliterative portal venopathy: Portal hypertension is not always present at   
diagnosis  
Cazals-Hatem D. Hillaire S. Rudler M. Plessier A. Paradis V. Condat B.   
Francoz C. Denninger M.-H. Durand F. Bedossa P. Valla D.C.  
Journal of Hepatology (2011) 54:3 (455-461). Date of Publication: March 2011  
  
Background & Aims: Previous studies on obliterative portal venopathy (OPV)   
have been biased due to the selection of patients with non-cirrhotic portal   
hypertension. The aim of this study was to clarify the characteristics of   
OVP diagnosed by liver biopsy. Methods: Fifty-nine consecutive patients with   
OPV were retrospectively selected on strict histological criteria. Clinical,   
laboratory, portal vein patency, and associated disorders potentially   
involving vascular alterations were analyzed. The occurrence of   
complications was recorded during follow-up. Results: Mean age at diagnosis   
was 38.5 ± 15 years old. Initial presentation was portal hypertension (64%   
of patients) and/or extrahepatic portal vein thrombosis (EHPVT) (22%) or   
isolated abnormal laboratory tests (20%). Associated diseases found at   
diagnosis were: prothrombotic disorders (30% of patients) and   
immune-mediated disorders (17%); 53% of patients had no causal factor   
(idiopathic OPV). During follow-up (median 8.6 years, range 1-23 years),   
features of portal hypertension worsened in 46% of patients; EHPVT and   
portal hypertension were finally found in 44% and 88% of patients.   
Anti-coagulation and beta-blockers were administered in 47% and 59% of   
patients, respectively. Severe complications (liver transplantation and/or   
death) occurred in 11 (19%) patients, 8 had idiopathic OPV. Patients with   
prothrombotic disorders received earlier anticoagulation therapy; all   
survived without transplantation. Conclusions: A confident diagnosis of OPV   
can be done by biopsy and is conceivable in patients under 40 years without   
clinically significant portal hypertension. Poor outcome was noted in 19% of   
patients, most of them affected with idiopathic OPV. Patients with   
prothrombotic disorders received early anticoagulation and appeared to have   
a better outcome despite a high proportion of EHPVT. © 2010 European   
Association for the Study of the Liver. Published by Elsevier B.V. All   
rights reserved.  
  
RECORD 559  
The interaction of low standard liver volume, high packed cell volume,   
portal hypertension and risk of portal vein thrombosis in clinical islet   
transplantation  
Kawahara T. Kin T. Kashkoush S. Bigam D.L. Kneteman N.M. Shapiro J.  
HPB (2011) 13 SUPPL. 1 (29). Date of Publication: March 2011  
  
Islet transplantation improves glycemic control in type1 diabetes   
complicated by refractory hypoglycemia. Percutaneous transhepatic portal   
access avoids surgery, but is rarely associated with bleeding or portal   
venous thrombosis. Here, we evaluate factors affecting portal pressure and   
thrombosis after islet infusion. 116 patients underwent 256 percutaneous   
intraportal islet-alone transplant procedures (mean 2.16 infusions/   
patient). The mean islet mass was 407,221 IE (5,908 IE/ kg) with mean packed   
cell volume (PCV) of 4.1 ml (1.5- 7.9). Univariate analysis revealed that   
standard liver volume had a negative correlation with portal pressure   
elevation (r = -0.257, P < 0.01), and those with larger liver volume had   
less perturbation in portal venous pressure. PCV correlated positively with   
elevated portal pressure (r = 0.463, P < 0.01). 10 patients (3.9%) developed   
partial thrombosis of peripheral segmental branches of the intrahepatic   
portal vein. None had complete portal thrombosis. Multivariable logistic   
regression revealed that only elevated portal pressure was associated with   
portal thrombosis (Odds Ratio 1.16, P = 0.045), but not PCV nor actual   
number of islets infused. Of note, we have not encountered this complication   
in the past 5 years, since institution of full heparin anticoagulation   
protocols combined with effective avitene-paste plugging of the transhepatic   
tract, and limitation in PCV to less than 5 ml. We conclude that in patients   
with low standard liver volume, islet PCV should be kept below 5 ml and full   
anticoagulation given, if risk portal vein thrombosis is to be mitigated.   
Portal thrombosis is a completely avoidable complication.  
  
RECORD 560  
portal vein thrombosis in inflammatory bowel disease: A single center   
experience  
Maconi G. Dell'Era A. Ardizzone S. Bolzacchini E. De Franchis R.  
Digestive and Liver Disease (2011) 43 SUPPL. 3 (S256). Date of Publication:   
March 2011  
  
Background and aim: portal vein thrombosis (PVT) is a well recognized and   
frequent complication in advanced cirrhosis but is rare in a previously   
healthy liver. Inflammatory bowel diseases (IBDs) are characterised by a   
hypercoagulable state and by a higher incidence of systemic thromboembolic   
events than in the general population. Material and methods: We describe the   
presentation, diagnostic approaches, underlying acquired or inherited risks   
factors for hypercoagulability and clinical outcome of 7 IBD patients   
followed in our centre who developed PVT during their clinical course.   
Results: The patients (5 males; mean age: 47.7±11.1) presented with partial   
PV thrombosis (4 patients) or portal cavernoma. Five had Crohn's disease   
(CD) and two had ulcerative colitis (UC). Three CD patients had undergone   
ileocolic resection for strictures and 1 UC patient had undergone colectomy.   
One patient had HBV-positive active hepatitis and, in 2 patients, a   
primitive sclerosing cholangitis was diagnosed during diagnostic work-up of   
PVT. Mean time from diagnosis of IBD to detection of PVT was 15.7±6.4 years.   
In 4 patients, the diagnosis of PVT was made while IBD was in clinical   
remission. No patient showed specific signs or symptoms leading to diagnosis   
of PVT, which was initially made by ultrasound with colour Doppler in 6   
patients and by CT scan in one patient. Most patients showed at least 1   
potential risk factor for hypercoagulability: lupus anti-coagulant and   
protein S deficiency were detected in one patient, 2 patients had von   
Willebrand factor impairment and 2 patients increased homocysteine   
levels.None of the patients received anticoagulation following diagnosis of   
PVT and none experienced other thrombotic events during a median of 5 years   
(range 2-8 years). Conclusions: PVT may be a potential complication of IBD,   
frequently associated with underlying acquired or inherited risks factors   
for hypercoagulability, but not necessarily with active disease. The   
clinical course of PVT in IBD appears to be benign and does not require   
long-term anti-coagulation treatment.  
  
RECORD 561  
Clinical and genetic factors associated to development of portal vein   
thrombosis in cirrhotic patients without hepatocellular carcinoma  
Pellicelli A.M. D'Ambrosio C. Barbaro G. Villani R. Guarascio P. Fondacaro   
L. Cortese A. Atzori M. Regine G. Adami L. Santoro R. Ettorre G.M. Andreoli   
A.  
Journal of Hepatology (2011) 54 SUPPL. 1 (S77). Date of Publication: March   
2011  
  
Aims: The aim of this study is to identify clinical and genetic factors that   
may help to identify cirrhotic patients without hepatocellular carcinoma at   
high risk of developing portal vein thrombosis (PVT)/ Methods: 56   
consecutive cirrhotic patients were included in the study. At baseline   
clinical and genetic factors were analyzed in all the patients. The patients   
were evaluated every 6 months. Results: During a follow up of 19 months, 11   
out of 56 patients (19%) developed de novo PVT. At univariate analysis PTV   
was associated with MTHRF TT genotype, high homocysteine plasma levels, low   
mean velocity of portal vein flow and use of non selective beta blockers.   
All the data are reported in theTable. At multivariate analysis, PTV was   
associated with MTHFR TT (OR 4.1, 95% CI 3.2-7.3 p < 0.001), homocysteine   
plasmatic levels (OR 3.3 95% CI 1.4-6.8 p < 0.001), low mean velocity of   
portal vein flow (OR 4.5 95% CI 3.7-8.2 p < 0.001) and use of non selective   
b blockers (OR 3.3, 95% CI 1.4-6.8 p < 0.001). (Table presented) Conclusion:   
Cirrhotic patients with at baseline MTHFR TT genotype, high plasmatic   
homocysteine levels, low mean velocity of portal vein flow and use of non   
selective beta blockers are at risk to develop PVT. Cirrhotic patients may   
develop PVT as a result of differing combination of risk factors.   
Identification of an inherited thrombophilic mutation, in a subset of high   
risk for PVT cirrhotic patients, could have an important implication   
regarding the use of anticoagulation therapy, B-vitamins and folate as   
primary prevention of PVT.  
  
RECORD 562  
portal vein thrombosis in cirrhotic and noncirrhotic patients. Role of   
anticoagulation therapy  
Debernardi W.V. Forgia S. Ferruzzi G. Beggiato E. Martini S. Marzano A.   
Rizzetto M.  
Digestive and Liver Disease (2011) 43 SUPPL. 3 (S240). Date of Publication:   
March 2011  
  
Background and aim: portal vein thrombosis (PVT) is a complication of liver   
cirrhosis but it might be suspected when variceal bleeding appears in   
patients without liver disease. The treatment remains debatable. Aim of the   
study is to explore the efficacy of anticoagulant therapy in portal vein   
thrombosis. Material and methods: Seventy-nine patients with PVT seen from   
January 2009 to December 2010 were included. Screening for thrombophilic   
factorswas performed in 77 and all patients were evaluated for anticoagulant   
therapy. Results: In 68 patients PVT was associated with cirrhosis (86%) and   
hepatocellular carcinoma (57%). PVT was present in 26 patients (38%) despite   
standard coagulation tests showing pro-haemorrhagic state. Thrombophilic   
disorders was observed in 15 of cirrhotic patients (22%) vs 81% of   
noncirrhotics.Forthy-six patients received low molecular weight heparin   
(LMWH, 100 UI/kg/bd) or oral anticoagulant. Complete or partial   
recanalization of portal vein occurred in 68% of cirrhotics and in 75% of   
non cirrhotics. Significant side effects, as bleeding complications, were   
observed in 1.1% of treated patients. Conclusions: Anticoagulant therapy was   
effective in the treatment of PVT and safe in cirrhotic patients. The impact   
on long term outcome remains to be investigated.  
  
RECORD 563  
portal vein thrombosis after laparoscopic splenectomy:  
Vecchio R. Cacciola E. Intagliata E. Marchese S. Cacciola R. Zanghì G.   
Basile F.  
Surgical Endoscopy and Other Interventional Techniques (2011) 25 SUPPL. 1   
(S371). Date of Publication: March 2011  
  
Objective: portal vein thrombosis (PVT) can be a life-threatening   
complication of splenectomy if not diagnosed in time and treated properly.   
The actual incidence of postsplenectomy portal system thrombosis is not   
clearly determined, ranging between 0.7 and 80%. In this series, the Authors   
report their incidence in laparoscopic splenectomy and therapeutic   
strategies. Methods: Between 1998 and 2009, 102 patients were submitted to   
laparoscopic splenectomy for hematologic disease. PVT was evaluated   
clinically and diagnosed by means of abdominal computed tomography. Results:   
Clinically evident PVT has been diagnosed in 3 patients treated by   
laparoscopic splenectomy affected by lymphoma (2 cases) and b-thalassemia (1   
case). Treatment of these patients was successfully obtained by conservative   
therapy with high dose of heparin for at least 3 weeks. In one case an ileal   
resection for intestinal ischemia was needed. Conclusions: Laparoscopic   
splenectomy, expecially in patients with large spleen and/or affected by   
mielo-lymphoproliferative disorders, may be complicated by PVT. Early   
recognition and proper immediate treatment is mandatory in these cases.   
anticoagulation therapy treatment for 3 weeks after splenectomy was   
successful in all patients treated immediately. According to our experience,   
postoperative surveillance for portal vein thrombosis is mandatory in   
splenectomised patients at high risk. Perioperative thrombotic prophylaxis   
should be considered in selected patients.  
  
RECORD 564  
Transjugular intrahepatic portosystemic shunt (TIPS) after liver   
transplantation (LT): Vascular complications constitute a good indication  
Senzolo M. Magini G. Burroughs A. Agazzi R. Colledan M. Zanus G. Gaffuri G.   
Fagiuoli S.  
Journal of Hepatology (2011) 54 SUPPL. 1 (S226-S227). Date of Publication:   
March 2011  
  
Background and Aims: TIPS has demonstrated a low rate of efficacy in LT   
recipients compared to cirrhotic patients and the need for its placement   
confers very poor survival without retransplantation. However, previous   
studies have evaluated mainly patients with recurrence of primary liver   
disease. The aim of our study was to compared the efficacy and outcome of   
TIPS in LT recipients with portal hypertension due to recurrence of primary   
liver disease, with patients in whom TIPS was indicated to treat vascular   
complications. Methods: We evaluated the LT recipients in two transplant   
centers who were referred for TIPS placement between October 2006 and August   
2010. Efficacy of the procedure and outcome were analyzed with respect to   
the underlying aetiology and severity of liver disease. Results: 13 patients   
had an indication for TIPS placement (10M/3F, age 24-64 years), in 11 for   
refractory ascites (6/11 with associated hydrothorax) and in 2 for the   
presence of portal vein thrombosis (PVT) not responding to anticoagulation   
therapy. In those with refractory ascites, four patients had HCV recurrence,   
had de novo HBV-related cirrhosis associated with PVT, 5 had veno-occlusive   
disease and 1 had de novo Budd-Chiari syndrome. TIPS was successfully placed   
in all patients without complications. The time between LT and TIPS   
placement ranged from 1 to 23 months. Mean±SD MELD score before TIPS   
placement was 17±4.7 in patients with allograft dysfunction and 13±2.4 in   
those with vascular liver disorders (p = ns). During the follow up (31.5±33   
months), the latter group experienced a complete resolution of ascites and   
normalization of liver function; on the contrary, in the 5 patients with   
underlying liver disease only 3 had partial resolution of ascites. There   
were three deaths out of 5 patients in the patients with underlying liver   
disease compared to 1 death among 8 patients with vascular liver disorders.   
Conclusions: LT recipients with portal hypertension due to allograft   
dysfunction had a poor outcome in the absence of retransplantation and   
portal decompression provides only marginal clinical benefit. On the   
contrary, TIPS if indicated for the presence of vascular liver disease after   
LT seems to be effective and provide long term-benefit.  
  
RECORD 565  
Portal vein obstruction after liver transplantation in children treated by   
simultaneous minilaparotomy and transhepatic approaches: Initial experience  
Carnevale F.C. Santos A.C.B. Seda-Neto J. Zurstrassen C.E. Moreira A.M.   
Carone E. Marcelino A.S.Z. Porta G. Pugliese R. Miura I. Baggio V.D.   
Guimarães T. Cerri G.G. Chapchap P.  
Pediatric Transplantation (2011) 15:1 (47-52). Date of Publication: February   
2011  
  
portal vein thrombosis is a complication that occurs anytime after liver   
transplantation and can compromise the patient and graft survival. We   
describe a combined technique for PV recanalization in cases of PV   
obstruction after liver transplantation. Four children (1%), of 367   
subjected to liver transplantation from June 1991 to December 2008,   
underwent PV recanalization through a combined approach (transhepatic and   
minilaparotomy). All children received left lateral hepatic segments,   
developed portal vein thrombosis (n = 3) and stenosis (n = 1), and presented   
with symptoms of portal hypertension after transplantation. PV   
recanalization was tried by transhepatic retrograde access, and a   
minilaparotomy was performed when percutaneous recanalization was   
unsuccessful. Three patients underwent a successful portal recanalization   
and stent placement with the combined technique. In one patient, the   
recanalization was unsuccessful because of an extensive portomesenteric   
thrombosis. The other three children had the portal flow reestablished and   
followed with Doppler US studies. They received oral anticoagulation for   
three consecutive months after the procedure and the clinical symptoms   
subsided. In case of PV obstruction, the combined approach is technically   
feasible with good clinical and hemodynamic results. It' is a minimally   
invasive procedure and can be tried to avoid or delay surgical treatment or   
retransplantation. © 2011 John Wiley & Sons A/S.  
  
RECORD 566  
anticoagulation following pediatric liver transplantation reduces early   
thrombotic events  
McLin V.A. Rimensberger P. Belli D.C. Wildhaber B.E.  
Pediatric Transplantation (2011) 15:1 (117-118). Date of Publication:   
February 2011  
  
RECORD 567  
anticoagulation and variceal bleeding in non-cirrhotic patients with portal   
vein thrombosis  
Qi X. Han G. Bai M. Yuan S. Fan D.  
Internal and Emergency Medicine (2011) 6:1 (93-94). Date of Publication:   
February 2011  
  
RECORD 568  
A large portal vein: A rare finding of recent portal vein thrombosis  
Qi X. Han G. Yin Z. He C. Bai M. Yang Z. Guo W. Niu J. Wu K. Fan D.  
Case Reports in Gastroenterology (2011) 5:1 (33-39). Date of Publication:   
January-April 2011  
  
Acute portal vein thrombosis (PVT) is rarely encountered by clinicians. The   
most common manifestation of acute PVT is sudden onset of abdominal pain. A   
computed tomography scan without contrast often shows a high-density   
material in the portal vein. After injection of contrast agents, absence of   
luminal enhancement and enlargement of the obstructed portal vein are shown.   
In this case report, we demonstrated a rare computed tomography finding in   
which the diameter of the main portal vein was enormously distended to   
3-fold that of the aorta in a patient with recent PVT. Despite thrombolysis   
and anticoagulation were immediately given, portal venous recanalization was   
not achieved in the patient. After 5 years, variceal bleeding and ascites   
occurred and liver function had persistently deteriorated. Finally, he died   
of progressive liver failure. Considering this case, we suggest that an   
early decision for invasive interventional treatment might be necessary to   
both increase the rate of portal venous recanalization and improve   
prognosis, as anticoagulation and thrombolysis therapy failed to recanalize   
recent PVT. © 2011 S. Karger AG, Basel.  
  
RECORD 569  
Thrombosis and inflammatory bowel disease: A call for improved awareness and   
prevention  
Zitomersky N.L. Verhave M. Trenor III C.C.  
Inflammatory Bowel Diseases (2011) 17:1 (458-470). Date of Publication:   
January 2011  
  
Thrombotic complications in patients with inflammatory bowel disease (IBD)   
are common and require improved awareness and prevention. In this review the   
interface between IBD and thrombosis is discussed, with emphasis on risk   
assessment and data to aid clinical decision making. Thromboembolic   
complications are 3-fold more likely in IBD patients than controls and the   
relative risk exceeds 15 during disease flares. Improved assessment of   
thrombosis risk for an individual patient includes thorough personal and   
family history and awareness of prothrombotic medications and lifestyle   
choices. Patients with the highest risk of thrombosis are those with active   
colonic disease, personal or strong family history of thrombosis, and those   
with significant acquired risk factors. Combined risk factors or   
hospitalization should prompt mechanical thromboprophylaxis. Indications for   
prophylactic anticoagulation are not defined currently by clinical studies,   
especially in pediatric patients, although some groups now advocate   
prophylactic anticoagulation for all hospitalized IBD patients and even some   
outpatients with disease flares. Thrombosis management requires a   
multidisciplinary therapeutic approach to balance anticoagulation and   
bleeding risk. While bleeding may occur with anticoagulation in IBD, data   
and experience indicate that therapeutic heparin is safe and bleeding   
manifestations can be managed supportively in most patients. Until   
prospective trials of prophylactic anticoagulation are published, management   
of thrombotic risk and prophylaxis in IBD will remain a clinical challenge.   
© 2010 Crohn's & Colitis Foundation of America, Inc.  
  
RECORD 570  
Low-molecular-weight heparin successfully used to treat a nephrotic patient   
complicated by superior mesenteric vein thrombosis and portal vein   
thrombosis  
Wang Y.-C. Chuang F.-R. Lee W.-C. Chen T.-C. Ko S.-F. Wang I.-K. Lee C.-H.  
Medical Principles and Practice (2011) 20:2 (196-199). Date of Publication:   
January 2011  
  
Objectives: To report the success of treatment with low- molecular-weight   
heparins (LMWHs) in a case of nephrotic syndrome complicated by mesenteric   
vein thrombosis (MVT) and portal vein thrombosis (PVT). Clinical   
Presentation and Intervention: A 53-year-old man with nephrotic syndrome   
developed persistent mild abdominal pain for 3 days. Hepatic-portal venous   
system thrombosis of nephrotic syndrome was suspected due to new-onset   
superficial vein engorgement of the abdomen without liver cirrhosis.   
Abdominal computed tomography revealed MVT concomitant with PVT. He was   
successfully treated with LMWH without thrombolytic therapy. After discharge   
on day 9, he received continuous anticoagulation by LWMH on an outpatient   
basis at the nephrology clinic. Venous thromboembolic events or proteinuria   
did not recur within the 6-month follow-up. Conclusion: This report showed a   
case of MVT concomitant with PVT, a critical complication of nephrotic   
syndrome that was diagnosed in time and successfully treated with LMWH. A   
high index of clinical suspicion and timely management are crucial to tackle   
thrombotic complications in nephrotic syndrome. Copyright © 2011 S. Karger   
AG, Basel.  
  
RECORD 571  
portal vein thrombosis after laparoscopic and open splenectomy  
Vecchio R. Cacciola E. Cacciola R.R. Marchese S. Intagliata E.  
Journal of Laparoendoscopic and Advanced Surgical Techniques (2011) 21:1   
(71-75). Date of Publication: 1 Jan 2011  
  
Introduction: portal vein thrombosis (PVT) could be a life-threatening   
complication after splenectomy if not diagnosed promptly and treated   
properly. Risk factors of PVT are not completely clarified. Spleen size and   
underlying hematologic diseases are main potential risk factors for this   
complication. Laparoscopic surgery might increase the risk of developing   
PVT, as it reduces the blood flow in the portal system due to the   
pneumoperitoneum but, on the other hand, it seems to be associated with less   
postoperative modifications of coagulation parameters than open surgery,   
thus preventing PVT itself. The authors reviewed their series on open and   
laparoscopic splenectomies, pointing out their experience on PVT and   
discussing their surveillance and prophylaxis programs to prevent this   
complication. Materials and Methods: In this series, the authors report   
their experience on postsplenectomy PVT in 162 patients who have been   
splenectomised (102 operated on laparoscopically and 60 by open surgery).   
Results: PVT was clinically observed in 1 case out of 60 open splenectomies   
and in 3 cases out of 102 laparoscopic procedures. Patients were treated   
with conservative anticoagulation therapy. In one case, additional ileal   
resection was needed. Mortality was 0%. Conclusion: Low-molecular-weight   
heparin should be administered to all patients who have been splenectomised,   
especially if they are at high risk of PVT. If symptoms appear, patients   
need to be treated with high-dose heparin followed, after at least 3 weeks,   
by oral anticoagulant therapy. © Copyright 2011, Mary Ann Liebert, Inc.  
  
RECORD 572  
Transjugular intrahepatic portosystemic shunt with   
expanded-polytetrafuoroethylene-covered stents in non-cirrhotic patients   
with portal cavernoma  
Fanelli F. Angeloni S. Salvatori F.M. Marzano C. Boatta E. Merli M. Rossi P.   
Attili A.F. Ridola L. Cerini F. Riggio O.  
Digestive and Liver Disease (2011) 43:1 (78-84). Date of Publication:   
January 2011  
  
Aims: To evaluate the feasibility and efficacy of Transjugular intrahepatic   
portosystemic shunt (TIPS) in non-cirrhotic patients with symptomatic portal   
hypertension secondary to portal cavernoma. Methods: Our cohort includes 13   
consecutive patients. Eleven were considered for Transjugular intrahepatic   
portosystemic shunt placement for complications not manageable by   
medical/endoscopic treatment and two because of the need of oral   
anticoagulation in presence of high-risk varices.   
Expanded-polytetrafluoroethylene-covered stents were used in all. Results:   
One of the 13 patients was excluded because of a thrombosis of the superior   
cava and jugular veins. In 10 patients, Transjugular intrahepatic   
portosystemic shunt was successfully implanted [83.3%; 95% confidence   
interval: 52-98%]. One patient had an early shunt dysfunction with   
recurrence of variceal bleeding which required an emergency surgical shunt.   
Late shunt dysfunction occurred in two patients, successfully treated with   
angioplasty and re-stenting. Two patients experienced an episode of   
encephalopathy. Conclusions: Transjugular intrahepatic portosystemic shunt   
is feasible in most of the patients with portal cavernoma and should be   
considered in those with severe complications uncontrolled by conventional   
therapy. The use of Transjugular intrahepatic portosystemic shunt to achieve   
a lifelong anticoagulation therapy in selected patients with high-risk   
varices may be another possible indication. These patients should be   
referred to selected Units with large experience in Transjugular   
intrahepatic portosystemic shunt placement. © 2010 Editrice   
Gastroenterologica Italiana S.r.l.  
  
RECORD 573  
[Spontaneous dissolution of isolated superior mesenteric vein thrombosis in   
acute pancreatitis].  
Na B.S. John B.M. Kim K.B. Lee J.S. Jo H.W. Seock C.H. Kim D.H. Lee K.S.  
The Korean journal of gastroenterology = Taehan Sohwagi Hakhoe chi (2011)   
57:1 (38-41). Date of Publication: Jan 2011  
  
Acute pancreatitis can result in many vascular complications in both artery   
and vein. Venous complication usually occurs as a form of splenic or portal   
vein thrombosis, and also can simultaneously occur in superior mesenteric   
vein as well. Rarely, isolated superior mesenteric vein thrombosis occurs as   
a venous complication. Although it is uncommon, mesenteric vein thrombosis   
is an important clinical entity because of the possibility of mesenteric   
ischemia and infarction of small bowel. The treatments of mesenteric venous   
thrombosis include anticoagulation therapy, transcatheter therapy and   
surgical intervention. We report a case of 45-year- old man who had acute   
pancreatitis with isolated superior mesenteric vein thrombosis, which was   
spontaneously dissolved with the resolution of underlying inflammation   
without anticoagulation or surgical intervention.  
  
RECORD 574  
Pylephlebitis: An overview of non-cirrhotic cases and factors related to   
outcome  
Kanellopoulou T. Alexopoulou A. Theodossiades G. Koskinas J. Archimandritis   
A.J.  
Scandinavian Journal of Infectious Diseases (2010) 42:11-12 (804-811). Date   
of Publication: December 2010  
  
Pylephlebitis is a condition with significant morbidity and mortality. We   
review herein 100 relevant case reports published since 1971. Eighty-one   
patients were reported with acute pylephlebitis, while the remaining   
patients had chronic pylephlebitis. The most common predisposing infections   
leading to pylephlebitis were diverticulitis and appendicitis. Cultures from   
blood or other tissues were positive in 77%. The infection was polymicrobial   
in half of the patients and the most common isolates were Bacteroides spp,   
Escherichia coli and Streptococcus spp. Thrombosis was extended to the   
superior mesenteric vein (SMV), splenic vein, and intrahepatic branches of   
the portal vein (PV) in 42%, 12%, and 39%, respectively. Antibiotics were   
administered in all and anticoagulation in 35 cases. Patients who received   
anticoagulation had a favourable outcome compared to those who received   
antibiotics alone (complete recanalization 25.7% vs 14.8% (p > 0.05), no   
recanalization 5.7% vs 22.2% (p < 0.05), and death 5.7% vs 22.2% (p <   
0.01)). Cases with complete recanalization had prompt diagnosis and   
management and two-thirds were recently published. Nineteen patients died;   
the majority of these (73.7%) died over the period 1971-1990. In conclusion,   
pylephlebitis remains an entity with high morbidity and mortality, but   
modern imaging modalities have facilitated an earlier diagnosis and have   
improved the prognosis. anticoagulation has a rather beneficial effect on   
patients with pylephlebitis. © 2010 Informa Healthcare.  
  
RECORD 575  
Transcatheter local thrombolysis in patients with extensive TIPS thrombosis  
Dumortier J. Walter T. Guillaud O. Pietu F. Vallin M. Henry L. Pilleul F.  
Gastroenterologie Clinique et Biologique (2010) 34:12 (721-725). Date of   
Publication: December 2010  
  
Background: Transcatheter local thrombolytic therapy in patients with   
portosplanchnic venous thrombosis has been used in few cases. Case reports:   
Here, we present our single-center experience with transcatheter   
thrombolytic therapy in three patients with extensive refractory portal and   
transjugular intrahepatic portosystemic shunt (TIPS) thrombosis.   
Thrombolytic therapy was successful for all three patients. Two patients   
developed minor procedure-related bleeding. Conclusion: Local thrombolysis   
could be proposed in case of TIPS thrombosis for patients in whom the venous   
flow cannot be restored by using conventional anticoagulant therapy and   
stent mechanical revision. © 2010 Elsevier Masson SAS.  
  
RECORD 576  
Incidence of Cytomegalovirus-associated thrombosis and its risk factors: A   
case-control study  
Atzmony L. Halutz O. Avidor B. Finn T. Zimmerman O. Steinvil A. Zeltser D.   
Giladi M. Justo D.  
Thrombosis Research (2010) 126:6 (e439-e443). Date of Publication: December   
2010  
  
Introduction: Cytomegalovirus (CMV)-associated thrombosis has been reported   
sporadically in the medical literature until now. However, thrombosis   
incidence and its risk factors have never been studied in a cohort of   
patients with acute CMV infection. Materials and Methods: A retrospective   
case-control study. Medical charts and imaging study reports of all   
consecutive patients diagnosed with acute CMV infection during the years   
2005-2006 in a tertiary medical center were reviewed for the presence of   
arterial and/or venous thromboses, and their acquired as well as inherited   
predispositions. The control group included age-matched and sex-matched   
consecutive patients, in whom acute CMV infection was excluded. Laboratory   
tests used for acute CMV infection diagnosis/exclusion were also matched,   
including serology, antigenemia, and PCR. Results: Included were 140   
patients with acute CMV infection (study group) and 140 consecutive matched   
patients in whom acute CMV infection was excluded (control group). Among the   
control group, none of the patients had thrombosis, while among the study   
group, nine (6.4%; p = 0.003) patients had thrombosis: five (3.6%; p =   
0.025) patients had arterial thrombosis and four (2.9%; p = 0.045) patients   
had venous thrombosis. Binary logistic regression analysis showed that acute   
CMV infection was independently associated with thrombosis among the whole   
cohort (p = 0.004), while use of oral contraceptives/hormones or pregnancy   
were independently associated with thrombosis among patients with acute CMV   
infection (p = 0.043). Conclusions: Thrombosis in patients with acute CMV   
infection is not rare. Acute CMV infection is associated with thrombosis   
independent of other risk factors for thrombosis. We hope to raise   
physician's awareness to the association between acute CMV infection and   
thrombosis. © 2010 Elsevier Ltd. All rights reserved.  
  
RECORD 577  
How to manage acute mesenteric and portal vein thrombosis  
Seung M.-K. Roh Y.-N. Kim Y.-W. Kim D.-I.  
Chirurgia (2010) 23:6 (235-240). Date of Publication: December 2010  
  
Aim. Acute mesenteric vein thrombosis (MVT) and portal vein thrombosis (PVT)   
are associated with high rates of morbidity and mortality due in part to the   
difficulty to diagnostic them and the operative challenges. The initial   
treatment for MVT and PVT is controversial. Some authors have proposed a   
surgical approach, whereas others have advocated medical therapy   
(anticoagulation). In this study, we analyzed and compared the results   
obtained with surgical and medical treatments to determine the best initial   
management for this disease. Methods. We retrospectively reviewed the   
hospital records and clinical data of 10 patients who were treated for MVT   
and PVT. Each patient was assessed for the diagnosis, initial management   
(laparotomy or anticoagulation), the morbidity and mortality and the   
duration of hospitalization. Results. All of the patients were initially   
treated with unfractionated heparin. The mean hospital stay was 20 days. One   
patient underwent emergency laparotomy with bowel resection, while two   
patients developed stricture during the follow-up period that necessitated   
resection and anastomosis of the bowel. The other seven patients underwent   
anticoagulation therapy only. During the follow-up period, all the patients   
were checked by computed tomography (CT). Five patients showed improvement,   
four patients showed no change and one patient showed worsened MVT and PVT.   
Nine patients showed cavernous transformation of the venous system along the   
mesenteric vein and portal vein on CT. There was no mortality. Conclusion.   
Nonoperative management for acute MVT and PVT is feasible when the bowel   
infarction has not led to transmural necrosis and bowel perforation. The   
morbidity, mortality and long-term outcomes were similar for the cases of   
surgical and nonoperative management. A nonoperative approach, when   
indicated, avoids resection of the macroscopically infarcted small bowel in   
cases that are potentially reversible with anticoagulation alone.  
  
RECORD 578  
Thromboembolic and bleeding complications in acute leukemia  
Kwaan H.C. Huyck T.  
Expert Review of Hematology (2010) 3:6 (719-730). Date of Publication:   
December 2010  
  
The risk of both thromboembolic and bleeding complications is high in acute   
leukemia. This double hazard has a significant negative impact on the   
morbidity and mortality of patients with this disease. The clinical   
manifestations of both complications show special features specific to the   
form of acute leukemia. Recognition of these characteristics is important in   
the diagnosis and management of acute leukemia. In this article, several   
additional issues are addressed, including the features of bleeding and   
thrombosis in acute promyelocytic leukemia, the current understanding of the   
leukostasis syndrome and the iatrogenic complications including   
catheter-associated thrombosis, and the adverse effects of therapeutic   
agents used in acute leukemia. As regards the bleeding complications,   
thrombocytopenia is a major cause. Corrective measures, including recent   
guidelines on platelet transfusions, are provided. © 2010 Expert Reviews   
Ltd.  
  
RECORD 579  
Splanchnic vein thrombosis: Clinical presentation, risk factors and   
treatment  
de Stefano V. Martinelli I.  
Internal and Emergency Medicine (2010) 5:6 (487-494). Date of Publication:   
December 2010  
  
The term splanchnic vein thrombosis encompasses Budd-Chiari syndrome (BCS),   
extrahepatic portal vein obstruction (EHPVO), and mesenteric vein   
thrombosis; the simultaneous involvement of additional regions is frequent,   
and clinical presentations and risk factors may be shared. The annual   
incidence of BCS and isolated mesenteric vein thrombosis is less than one   
per million individuals, while the incidence of EHPVO is about four per   
million; autopsy studies, however, suggest higher numbers. Current advances   
in non-invasive vascular imaging allow for the identification of chronic or   
asymptomatic forms. Risk factors can be local or systemic. A local   
precipitating factor is rare in BCS, while it is common in patients with   
portal vein thrombosis. Chronic myeloproliferative neoplasms (MPN) are the   
leading systemic cause of splanchnic vein thrombosis, and are diagnosed in   
half the BCS patients and one-third of the EHPVO patients. The molecular   
marker JAK2 V617F is detectable in a large majority of patients with overt   
MPN, and up to 40% of patients without overt MPN. Inherited thrombophilia is   
present in at least one-third of the patients, and the factor V Leiden or   
the prothrombin G20210A mutations are the most common mutations found in BCS   
or EHPVO patients, respectively. Multiple factors are present in   
approximately one-third of the patients with BCS and two-thirds of the   
patients with portal vein thrombosis. Immediate anticoagulation with heparin   
is used to treat patients acutely. Upon clinical deterioration,   
catheter-directed thrombolysis or transjugular intrahepatic portosystemic   
shunt is used in conjunction with anticoagulation. Long-term oral   
anticoagulation with vitamin K-antagonists (VKA) is recommended in all BCS   
patients, and in the patients with a permanent prothrombotic state   
associated with an unprovoked EHPVO. In patients with an unprovoked EHPVO   
and no prothrombotic conditions, or in those with a provoked EHPVO,   
anticoagulant treatment is recommended for a minimum of 3-6 months. © SIMI   
2010.  
  
RECORD 580  
portal vein thrombosis after splenectomy for hypersplenism in patients with   
liver cirrhosis: An analysis of 22 cases  
Meng J. Lu S.-C. Wang M.-L. Gao F.  
World Chinese Journal of Digestology (2010) 18:33 (3584-3589). Date of   
Publication: 28 Nov 2010  
  
AIM: To determine the incidence rate of portal vein thrombosis (PVT) in   
cirrhotic patients after splenectomy for hypersplenism and to assess the   
efficacy of low molecular weight heparin (LMWH) for the treatment of PVT.   
METHODS: A total of 58 consecutive cirrhotic patients who underwent   
splenectomy for hypersplenism from January 2008 to December 2010 were   
enrolled into this study. All the patients received prophylactic   
anticoagulation therapy after the operation. Based on the presence of   
thrombus or not, the patients were divided into thrombosis group and   
non-thrombosis group. The incidence rate of PVT, thrombophilic factors, and   
thrombus location were analyzed in these patients. RESULTS: All patients   
developed thrombosis, Thrombosis of the splenic vein, superior mesenteric   
vein and multiple veins was found in 5, 1 and 13 patients, respectively.   
Above 37.93% of the patients developed PVT. Identified risk factors for the   
development of PVT included high platelet count, low blood flow and   
increased spleen weight. CONCLUSION: Blood platelet count and spleen weight   
are important risk factors for the development of PVT. Prophylactic   
treatment with low molecular weight heparin and Warfarin is likely to   
prevent the development of PVT in cirrhotic patients after splenectomy.  
  
RECORD 581  
Transsplenic endovascular therapy of portal vein stenosis and subsequent   
complete portal vein thrombosis in a 2-year-old child  
Bertram H. Pfister E.-D. Becker T. Schoof S.  
Journal of Vascular and Interventional Radiology (2010) 21:11 (1760-1764).   
Date of Publication: November 2010  
  
A complex catheter intervention for portal vein stenosis and subsequent   
complete thrombosis after split-liver transplantation was performed using   
transsplenic access to the portal vein circulation. The combination of   
intrahepatic, local thrombolysis and extrahepatic portal vein angioplasty   
performed twice on 2 consecutive days followed by anticoagulation with a   
high dose of heparin and clopidogrel completely resolved portal vein   
stenosis and thrombosis. Postinterventional angiographic and serial   
ultrasound examinations confirmed that the endovascular therapy was   
successful. In selected patients, percutaneous transsplenic access to the   
portal vein circulation may be used for diagnostic and therapeutic   
interventions even in early childhood. © 2010 SIR.  
  
RECORD 582  
Transjugular intrahepatic portosystemic shunt for the treatment of portal   
vein thrombus: Its current status  
Qi X.-S. Han G.-H. Fan D.-M.  
Journal of Interventional Radiology (2010) 19:11 (916-920). Date of   
Publication: November 2010  
  
The prevalence of portal vein thrombosis in the general population is about   
1.1%, while it is about 10% - 25% in the cirrhotic patients. The severe   
clinical complication in patients with acute portal vein thrombosis is   
ischemic intestinal infarction when the thrombus extends to the mesenteric   
venous arch. The complications include bleeding due to gastroesophageal   
varices, ascites and deterioration of live function in the patients with   
chronic portal vein thrombosis. The recently-published Practice Guidelines   
indicate that the treatment of portal vein thrombosis includes   
anticoagulation, thrombolysis, transjugular intrahepatic portosystemic shunt   
(TIPS) and surgical thrombectomy. TIPS has some advantages in treating   
portal vein thrombus. It can directly and effectively re-canalize the   
occluded portal vein. Moreover, it can accelerate portal flow and prevent   
recurrent thrombosis after the shunt is well-established. The disadvantages   
of TIPS include technical difficulties and potential complications. However,   
percutaneous transhepatic, transsplenic and transmesenteric approaches well   
facilitate the TIPS procedure. Additionally, preoperative evaluation of   
portal vein anatomy can provide a safe and effective choice in treating   
patients with portal cavernoua caver who are going to receive TIPS.   
Nevertheless, in the absence of relevant prospective studies, the   
application of TIPS for the management of portal vein thrombosis is still   
limited.  
  
RECORD 583  
Post transplantation outcomes of cirrhotics with portal vein thrombosis who   
are not anticoagulated is similar to cirrhotics without portal vein   
thrombosis  
John B.V. Konjeti V.R. Aggarwal A. Lopez R. Zein N.N. Gunasekaran G. Miller   
C.M. Carey W.D.  
Hepatology (2010) 52 SUPPL. 1 (373A). Date of Publication: October 2010  
  
Background: Most cirrhotics with portal vein thromboses (PVT) are   
asymptomatic, discovered incidentally and are not treated with   
anticoagulation. Though there are small case series suggesting that   
anticoagulation improves portal vein patency before liver transplantation   
(LT), there is little data to compare the post-transplantation outcomes   
between cirrhosis with PVT who are not anticoagulated and cirrhotics with no   
PVT. Aims: To: (1) define the technical challenges for OLT posed by partial   
or complete PVT; (2) measure the impact of PVT on 60- and 180-day mortality;   
(3) estimate the value of pre-operative anticoagulation on OLT outcome.   
Methods: A prospectively followed cohort of cirrhotics evaluated for LT at a   
large tertiary care academic medical center between 2006 and 2008 with a   
minimum 6 month follow up after evaluation were included. All patients were   
evaluated for PVT at the time of transplant evaluation by liver vascular   
ultrasound and contrast CT or MRI and every 6 months by contrast CT or MRI   
till transplantation, removal from the transplant list or death. Patients   
with hepatocellular carcinoma (HCC) at time of diagnosis of PVT were   
excluded. Results: Among 932 cirrhotics evaluated for transplant, all 294   
with follow up of at least 6 months were included. Seventy patients were   
diagnosed to have PVT after evaluation prior to LT (group 1) and 224   
developed no PVT (group2). There was no difference in age, etiology of liver   
disease and MELD scores between the groups. Forty-five percent had occlusive   
PVT. Forty-two (60%) and 150 (64%) in the two groups underwent OLT. Only 1   
of the 42 with PVT and 2 of the 150 cirrhotics without PVT (1 for venous   
thrombosis, 1 for Atrial fibrillation) were anticoagulated. Of the 42 with   
PVT who underwent OLT, 25 (60%) required thombectomy at the time of   
transplantation and 10 (25%) resolved thrombosis in the absence of   
anticoagulation. There was no difference in the 60- or 180-day post   
transplant mortality between patients with and without PVT. Conclusion: PVT   
did not increase rate 60- or 180- day post transplant mortality. The   
presence of PVT does not pose undue technical problem for transplant   
surgery. About 60% of cirrhotics with PVT who underwent OLT had thrombectomy   
while 25% had resolution of PVT without anticoagulation. Our data does not   
appear to support a post transplant benefit for anticoagulation or clot   
lysis in cirrhotics with PVT.  
  
RECORD 584  
Pregnancy in women with portal vein thrombosis (PVT): Results of a   
multicentric european study on maternal and foetal management and outcome  
Hoekstra J. Seijo S. Rautou P.-E. Ducarme G. Luton D. Alijotas-Reig J.   
Casellas-Caro M. Condat B. Garcia-Pagán J.C. Janssen H.L. Valla D.   
Casellas-Caro M.  
Hepatology (2010) 52 SUPPL. 1 (904A). Date of Publication: October 2010  
  
Background: Although many patients with primary PVT are females of   
childbearing age, data on pregnancy in this context are scarce. Aims: To   
assess maternal and fetal outcome of pregnancies in women with known PVT.   
Methods: Retrospective European multicenter analysis of pregnancy in women   
with chronic PVT seen between 1986 and 2010. Pregnancies occurred 6 months   
or more after PVT diagnosis. Results: 23 women, median age 32 years (20-45)   
had 42 pregnancies (France n=16, Netherlands n=13, Spain n=13). Median   
duration between PVT diagnosis and pregnancy was 54 months (7 to 370   
months). Twenty-one women had portal vein obstruction, including 8 with   
mesenteric vein (MV) obstruction; 2 left or right portal vein obstruction,   
including 1 with MV obstruction. Fifteen had oesophageal varices, which had   
ruptured in 9.Four had myeloproliferative disorder, 7protein S or C   
deficiency, 3 antiphospholipid syndrome, 1 factor V Leiden and 1   
prothrombine gene mutation. Low molecular weight heparin (LWMH) was started   
before gestation-week 5 in 14 pregnancies; and after week 6 in 13. No LMWH   
was given during 15 pregnancies. Median duration of gestation was 36 weeks   
(range 6-39 weeks). Eight fetuses were lost before week 20. There were 2   
very preterm birth at week 24-25; 19 moderately preterm birth (at 32-36   
weeks); and 13 term birth (after week 37). One infant underwent emergency   
surgery for a cardiac malformation; another infant had hyaline membrane   
disease, and necrotic colitis, both with favourable outcome. Two mothers had   
HELLP syndrome at week 24 and 33 respectively. There were 3/17 parietal   
bleeding after caesarean sections, and 1/17 genital bleeding after vaginal   
deliveries. Two oesophageal variceal bleedings occurred, in the absence of   
primary prophylaxis, at week 18 and 38 respectively. Three had ascites   
during pregnancy. Post-partum splenic infarction occurred once without   
anticoagulation. In univariate analysis, significant prognosis factors for   
unfavourable outcome of pregnancy were high platelets (253 +/- 140 vs   
220+/-78 (p=0.017), past history of spontaneous abortion or deep vein   
thrombosis (p=0,042) and past oestrogen contraception (p=0,008). There were   
no deaths. Conclusions: In chronic PVT patients, who become pregnant, foetal   
outcome is generally favourable and maternal outcome is good. Variceal   
bleeding is rare. HELLP syndrome seems more common than expected. Thrombotic   
events can occur postpartum in the absence of anticoagulation. Therefore,   
intensified prophylaxis for portal hypertension, close surveillance for   
HELLP, and brief anticoagulation interruption around delivery may reduce the   
rate of bleeding and thrombotic complications in these patients.  
  
RECORD 585  
portal vein thrombosis associated with primary cytomegalovirus infection in   
an immunocompetent child  
Alkhouri N. Okwu V. Elias M. Rouphail B. Alkhouri R. Carter-Kent C.  
American Journal of Gastroenterology (2010) 105 SUPPL. 1 (S384-S385). Date   
of Publication: October 2010  
  
Purpose: Cytomegalovirus (CMV) infection may occur in immunocompetent   
children and adolescents and often follows an asymptomatic course. Portal   
vein thrombosis in association with acute CMV infection is a very rare   
condition in an immunocompetent host. An 18-year-old female with no   
significant medical history presented with a one-week history of right upper   
quadrant pain and low grade fever. On examination, the upper part of the   
abdomen was tender and the spleen was palpable at 4 cm below the costal   
margin. Initial laboratory testing showed WBC of 10.1 k/uL (lymphocytes   
71%), platelet count of 156 k/uL, ALT 193 U/L, AST 134 U/L, LDH 360 U/ml   
with normal prothrombin time, alkaline phosphatase and bilirubin. An   
abdominal ultrasound showed a thrombus in the portal vein trunk and   
splenomegaly of 19.5 cm. Serologic tests were negative for hepatitis A,   
hepatitis B, hepatitis C, Epstein-Barr virus, HIV infection and antibodies   
to toxoplasmosis. Antibodies were detected against CMV with both positive   
IgG and IgM; CMV DNA was detected by PCR. Screening for thrombophilia   
revealed normal levels of protein C, protein S, antithrombin III and   
homocysteine with no prothrombin or factor V Leiden mutations and no   
antiphospholipid antibodies. A diagnosis of portal vein thrombosis secondary   
to acute CMV infection was made. The patient was started on heparin and   
transitioned to oral anticoagulant therapy. Repeated ultrasound in 3 months   
showed recanalization of the portal vein and improved splenomegaly. This   
case illustrates that in the presence of acute CMV infection with abdominal   
pain, the possibility of abdominal venous thrombosis should be always   
entertained in order to start anticoagulation as soon as possible. Acute CMV   
hepatitis should be added to the list of risk factors of acute portal vein   
thrombosis.  
  
RECORD 586  
portal vein thrombosis in cirrhotic patients is associated with advanced   
liver disease and predicts poor long term prognosis  
Ferreira C.N. Rodrigues T. Alexandrino P. Ramalho F. Velosa J.F.  
Hepatology (2010) 52 SUPPL. 1 (1072A). Date of Publication: October 2010  
  
Introduction Though the prevalence non-hepatocelular carcinoma( HCC)   
associated portal vein thrombosis (PVT) in cirrhotic patients ranges between   
0.6 - 16%, the influence on prognosis is not clear. Aims: To characterize   
non-HCC associated PVT in cirrhosis and determine it's influence on   
prognosis. Methods We studied 40 consecutive cirrhotic patients with non-HCC   
associated PVT. Patient characteristics: Age 57±14years; Males 65%(26);   
Child-Pugh (CP) score 8±3, Model for End-Stage Liver Disase (MELD) score   
15±7; CP class: A-25%(10), B-50%(20), C-25%(10); Aetiology of cirrhosis:   
alcohol-60%(20), viral-12.5%(5), alcohol+viral-10%(4), others-11.5%(7). The   
effect of PVT on short (1 year) and long term (3 years) mortality was   
analyzed by comparing with a control group of 135 patients with   
decompensated cirrhosis matched for CP and MELD scores, age and aetiology of   
liver cirrhosis. Statistical analysis was performed with SPSS 18. Results At   
diagnosis of PVT, 88%(35)patients were symptomatic. Clinical features: upper   
gastrointestinal bleeding-55%(22), abdominal pain-33%(13),   
nausea/vomiting-13%(5), intestinal infarction- 5%(2) and diarrhoea-5%(2).   
Manifestations of portal hypertension: oesophageal varices-88%(35), severe   
portal hypertensive gastropathy-65%(26), gastric varices-40% (16), ectopic   
varices-13%(5) and ascites-65%(26). At diagnosis, acute PVT of the main   
trunk and/or main branches occurred in 90%(36) and portal cavernoma in   
10%(4). There was concomitant superior mesenteric vein thrombosis in 25%(10)   
and splenic vein thrombosis in 20%(8). Median follow up was 6.5months(IQR   
3.5). Survival at the end of follow-up was 58%(23). Compared to control   
group, PVT was significantly associated with higher mortality at 3 years but   
not at 1 year (p=0.001). The association of PVT with higher mortality was   
observed in patients with CP score <10 but not in those with CP score ≥10.   
By unconditional multivariate logistic regression analysis, cirrhotic   
patients with PVT had significantly higher mortality at 3 years (Odds Ratio   
6, 95% Confidence Interval 2-18). Actuarial survival analysis showed that   
when compared to control group, mortality in patients with PVT tends to   
increase significantly six months after diagnosis (p=0.037). Kaplan- Meier   
survival analysis confirmed the poor long term prognosis in patients with   
PVT (p=0.034). Conclusions PVT occurs predominantly in advanced liver   
cirrhosis and is associated with poor long term prognosis. The effect of PVT   
on higher mortality was only observed in Child-Pugh A and B patients. Our   
results suggest that aggressive management of PVT with anticoagulation when   
possible, could improve long term prognosis.  
  
RECORD 587  
The impact of portal vein thrombosis (PVT) on cirrhotics awaiting liver   
transplantation  
John B.V. Konjeti V.R. Aggarwal A. Lopez R. Zein N.N. Atreja A. Carey W.D.  
Hepatology (2010) 52 SUPPL. 1 (888A-889A). Date of Publication: October 2010  
  
Background: Most cirrhotic portal vein thromboses (cPVT) are discovered   
incidentally and not treated with anti-coagulation. According to 2009 AASLD   
guidelines, there is little information on the impact of portal vein   
thrombosis on morbidity and mortality of cirrhosis or the value of   
anticoagulation. Aims: To: 1. Estimate incidence and prevalence of cPVT in   
subjects evaluated for liver transplantation (LT). 2. Identify risk factors   
of developing new cPVT. 3. Describe the effect of cPVT on natural history.   
4. Compare mortality on OLT list between those with and without cPVT.   
Methods: Data was collected from a prospectively followed cohort of   
cirrhotics evaluated for LT at large tertiary care academic medical center   
between 2006 and 2008. All patients were evaluated with a liver vascular   
ultrasound and contrast CT or MRI at initial LT evaluation and every 6   
months while they were on the transplant list. Patients with hepatocellular   
carcinoma at diagnosis of cPVT were excluded. Results: Among 932 cirrhotics   
evaluated for OLT, all 294 with follow up of over 6 months were included.   
Forty-eight had cPVT at baseline (group 1), 22 developed new cPVT (group 2)   
and 224 developed no cPVT (group 3). The incidence of new cPVT was 8.7 per   
100 person-years of follow up, with cumulative incidence of 7.8% and 16.8%   
at 12 and 24 months. There was no difference in age, etiology of cirrhosis   
and MELD between the groups. Forty-five percent had occlusive cPVT. Only 3   
of 70 with cPVT (group 1 and 2), and 3 of 224 cirrhotics without cPVT (2   
calf venous thrombosis, 1 cardiac) were anti-coagulated. On multivariate   
analysis, ascites and worsening renal function predicted new onset cPVT;   
prior endoscopic treatment for varices did not increase risk. Subjects who   
developed new cPVT had greater worsening of Child score. In group 1, 2 and   
3, respectively, GI bleeding occurred in 18.8%, 18.2% and 11.7%, (p=0.3) and   
spontaneous bacterial peritonitis (SBP) in 2.1%, 4.6% and 12.1% (p=0.06).   
Twelve (25%), 5(22%) and 42 (19%) in groups 1,2 and 3 respectively died   
before LT. Factors affecting mortality include MELD, ascites and baseline   
alpha-fetoprotein. There was no difference in mortality while awaiting LT   
between subjects with and without cPVT on univariable or multivariable   
analysis. (p=0.16) Conclusion: The incidence of cPVT in those evaluated for   
LT is 8.7/100 person years. Ascites at baseline and worse renal function are   
risk factors for developing new cPVT. cPVT did not increase GI bleeding, SBP   
or mortality on the transplant list. Our data does not support a role for   
anticoagulation in patients with cPVT.  
  
RECORD 588  
Surgical intervention for patent ductus venosus  
Kamimatsuse A. Onitake Y. Kamei N. Tajima G. Sakura N. Sueda T. Hiyama E.  
Pediatric Surgery International (2010) 26:10 (1025-1030). Date of   
Publication: October 2010  
  
Patent ductus venosus (PDV) is a rare condition, which usually presents   
secondary to hepatic atrophy and hepatic failure. We have treated eight   
cases of PDV, all with hypergalactosemia and hyperbilirubinemia.   
Ultrasonography and three-dimensional computed tomography demonstrated   
communication between the portal vein and the inferior vena cava. Of the   
eight PDV cases, three from the older age group (ages 9, 11, and 14 years)   
had high-density lesions in their brain nucleus, and one case (age 19 years)   
had undergone prior Kasai portoenterostomy for biliary atresia. Six PDV   
patients underwent ligation of PDV and the remaining two cases underwent   
partial banding of PDV with intraoperative monitoring to maintain portal   
vein pressure (PVP) under 30 cm H(2)O. Improvement of the intrahepatic   
portal vein flow was achieved by ligation or banding of PDV.   
Postoperatively, serum galactose and bilirubin fell to normal ranges, but   
portal thrombus occurred postoperatively in the first case. We subsequently   
administered postoperative anticoagulation in the remaining cases and   
experienced no major complications. These results suggest that PDV ligation   
and banding are effective surgical approaches for patients with PDV. Close   
postoperative monitoring to avoid portal thrombus is imperative in these   
cases. © 2010 Springer-Verlag.  
  
RECORD 589  
Portal biliopathy: An unusual cause of asymptomatic biliary ductal dilation  
Lakshminarayan S.T.K. Demetria M. Attar B.  
American Journal of Gastroenterology (2010) 105 SUPPL. 1 (S208-S209). Date   
of Publication: October 2010  
  
Purpose: Portal biliopathy, a late complication of portal hypertension   
refers to abnormalities of the entire biliary tract. A 48-year-old female   
with 2-weeks of intermittent epigastric pain unrelated to food or bowel   
movements, unremarkable physical exam and laboratory work up showed a portal   
vein thrombus with cavernous transformation on CT abdomen. Intra- and   
extrahepatic biliary dilation due to extrinsic compression by the portal   
cavernoma was seen and confirmed by MRI/MRCP. No biliary stones or   
strictures were seen. anticoagulation was initiated for the portal vein   
thrombosis. No intervention for biliary ductal dilation was planned, given   
the absence of biliary symptoms or abnormal liver function tests. Portal   
biliopathy is more common with extrahepatic portal vein obstruction   
(81-100%) than cirrhosis (0-33%). Etiology is thought to be external   
pressure from biliary collaterals and/or ischemia. Only 20% are symptomatic   
from cholestasis or cholangitis. MRCP and MR portovenography is the initial   
investigation of choice, and may show extrahepatic strictures, intrahepatic   
biliary radicle, ectasias and filling defects. EUS may be useful in cases of   
diagnostic uncertainty. Asymptomatic patients require no treatment. If   
symptomatic, treatment options are endoscopy (sphincterotomy, stone   
extraction, stricture dilation, stenting) or surgery (portosystemic   
shunting, bilioenteric anastomosis). Surgery provides long-term relief and   
is preferable in young patients with a shuntable vein. Portosystemic   
shunting is a safer and more effective than bilioenteric anastomosis.   
Hemobilia may occur with balloon sweeps and sphincterotomy due to the   
presence of varicosities around the ampulla and CBD, and with basket   
extraction due to intraluminal choledochal varices mimicking stones. TIPSS   
is a therapeutic option. Liver transplant can be life saving in certain   
cases. In conclusion, portal biliopathy is a rare cause of extrinsic biliary   
compression that may rarely cause cholestasis, choledocholithiasis or   
cholangitis. Surgical portosystemic shunting is the treatment of choice in   
the presence of symptoms. (Figure presented).  
  
RECORD 590  
Algorithm for the management of portal vein thrombosis (PVT) in patients   
with cirrhosis: A prospective case-control study  
Senzolo M. Sartori M.T. Rossetto V. Cillo U. Rodriguez K.I. Nadal E. Zanus   
G. Burroughs A.K. Burra P.  
Hepatology (2010) 52 SUPPL. 1 (903A). Date of Publication: October 2010  
  
Background: There is no established management algorithm for the treatment   
of PVT in patients with cirrhosis. The aim of our study was to prospectively   
evaluate the use of anticoagulation and TIPS, as a second line option, to   
treat PVT. Methods: patients with cirrhosis and with non malignant PVT were   
included. anticoagulation with LWMH was considered in all; TIPS was   
indicated if there was a concomitant of portal hypertensive complication, or   
if thrombosis progressed. Patients seen in the same period, but who were not   
anticoagulated neither received TIPS, were included as controls. Results: 56   
patients were included (21 were controls). In the study group, PVT was   
occlusive in 11/35 with extension to the superior mesenteric vein or splenic   
vein in 13/35. anticoagulation was initiated in 33 patients. Mean   
follow-up±SD was 21.6±8.5 and 24.5±8.2 months for study and control groups   
respectively. Complete recanalization rate was 36% (12/33) in the treatment   
group compared to 1 among controls (p<0.001). A short time interval between   
appearance of thrombosis and anticoagulation (< 6 months) strongly predicted   
chance of repermeation. During the follow-up there was progression of   
thrombosis in 15/21 who were not anticoagulated and in 5/33 anticoagulated   
patients (p<0.001). TIPS was placed in 6 patients. There were 5 patients   
with variceal bleeding and 2 intestinal venous infarcts in the control   
group, compared to 1 variceal bleeding episode in the study group.   
Conclusions: a treatment algorithm with anticoagulation and the use of TIPS   
in patients with PVT and cirrhosis achieves a good chance of complete   
repermeation, reduces portal hypertensive complications and decreases the   
rate of thrombosis progression. This should lead to improved survival and   
renders liver transplantation less difficult. (Graph presented).  
  
RECORD 591  
Clinical outcome and prognostic factors in non-cirrhotic non-neoplastic   
patients with portal vein thrombosis: A single-center experience  
Angeloni S. Cerini F. Marzano C. Riggio O.  
Hepatology (2010) 52 SUPPL. 1 (1078A). Date of Publication: October 2010  
  
Introduction. portal vein thrombosis (PVT) is a rare condition in patients   
without cancer and cirrhosis. Little information are available on its   
natural history, clinical outcome and prognostic factors. Aim. To describe   
our single-centre experience on non-cirrhotic non-neoplastic patients with   
PVT. Patients. Fifty-five consecutive patients (31 male/24 female;   
age:46.7±15.1 yrs; 10 acute and 45 chronic PVT) were enrolled from January   
1999 to December 2009 and followed-up for 30.7±37.9 months by the same   
medical team according to a prospective, protocolized, diagnostic work-up   
and surveillance strategy. Therapeutic protocol included the use of   
long-term anticoagulation when possible and the treatment of varices at risk   
with drugs, endoscopy or TIPS. Results. At entry, a portal cavernoma was   
found in 45 patients. A multisegmental thrombosis was present in 25 patients   
(splenic vein in 18, superior mesenteric vein in 19, Budd-Chiari syndrome in   
5). Chronic myeloproliferative disorders were found in 16 patients, one or   
more prothrombotic coagulation disorders in 27 and PNH in 2. At entry, 17   
patients had a past episode of variceal bleeding (in 13 as the initial   
manifestation); 17 had no or small varices and 11 large varices. Eleven out   
of 17 patients with no or small varices at entry developed large varices   
during follow-up. The rate of progression was 11% (95%CI:4-31%) at one year   
and 28% (95%CI:16-51%) at two years. By multivariate analysis, small varices   
at first endoscopy and a multisegmental involvement at entry were   
independent predictors for the variceal progression. During follow-up, 6   
patients bled and 5 rebled from varices. The cumulative bleeding rate were:   
13% (95%CI:6-28%) at one year and 20% (95%CI:11-36%) at two years. The   
finding of a multisegmental thrombosis at entry was the only independent   
predictor for variceal bleeding. Ten patients experienced new thrombotic   
episodes during follow-up (5 in splanchnic and 5 in extra-splanchnic   
vessels). The cumulative rate of new thrombotic events was 11% at one year   
(95%CI:5-25%) and 16% (95%CI:8-31%) at two years. The presence of a   
multisegmental thrombosis at entry was the only independent factor for the   
occurrence of new thrombotic events. Of the 10 patients with acute PVT, 7   
achieved a complete ricanalization while 3 developed a portal cavernoma.   
Five patients died during follow-up. There were no bleeding-related deaths,   
while two patients died because of thrombotic events. Two-year cumulative   
survival was 95% (95%CI:84-98%). Conclusions: A multisegment involvement of   
the portal system at entry seems to be an important prognostic factor for   
the clinical outcome of non-cirrhotic patients with PVT.  
  
RECORD 592  
Endoscopic evaluation and management of autoimmune pancreatitis complicated   
with pancreatic ascites: A case report  
Martinez-Vazquez M.A. Garza-Galindo A.A. Vazquez-Elizondo G. Maldonado-Garza   
H.J.  
American Journal of Gastroenterology (2010) 105 SUPPL. 1 (S212). Date of   
Publication: October 2010  
  
Purpose: Autoimmune Pancreatitis (AIP) is a unique disease characterized by   
pancreatic inflammation and elevated serum IgG4 levels. Although the disease   
usually presents as a form of chronic pancreatitis, acute manifestations   
during a first episode or exacerbations are not uncommon. We describe the   
approach and management of a patient presenting with acute pancreatitis (AP)   
who developed multiple complications. Case report: An 18-year-old male with   
a 5-year history of recurrent pancreatitis and no other known diseases was   
hospitalized with severe abdominal pain, weight loss, ascites and left lower   
extremity deep vein thrombosis. Bloodwork upon admission included   
significant hipoalbuminemia (2.2 mg/dl) and a serum IgG4 markedly elevated   
at 533 mg/dL, with high total IgG levels at 2,012 mg/dL. Ascitic fluid   
analysis reported an amylase at 27,500 UI. Abdominal ultrasound showed   
evidence of portal vein thrombosis, ascities and acute pancreatitis.   
Computed tomography imaging demonstrated a dilated main pancreatic duct (PD)   
and a pancreatic head cystic lesion. Endoscopic ultrasound showed   
alterations compatible with severe acute pancreatitis complicated with fluid   
collections and ascites. An endoscopic retrograde cholangiopancreatography   
(ERCP) was performed, detecting rupture of the PD a pancreatic stent was   
placed. after marked improvement of ascites on the following 7 days, the   
patient was discharged and placed on oral anticoagulation. On outpatient   
follow-up, patient's ascites gradually resolved, his nutritional status   
improved and the pancreatic stent was removed after 8 weeks, documenting   
resolution of the PD rupture and a focal pancreatic duct stenosis at this   
level on ERCP, which was treated with balloon dilatation to 12 Fr. A   
prednisone regimen tapered to a maintenance dose of 5 to 10 mg/day was   
continued until clinical and laboratory evaluation fully resolved (length of   
follow-up). Discussion: Severe acute AIP with pancreatic duct disruption is   
uncommon and, as occurs with most pancreatitis, the approach for this   
complication is usually challenging. As reported in this case, conventional   
management with pancreatic stent placement and treatment of the primary   
etiology with close patient follow-up to determine the time of stent removal   
and further endoscopic treatment during ERCP as needed is recommended.   
Conclusion: Transpapillary pancreatic stent placement can be useful in the   
management of pancreatic ascites due to AIP and reduce the length of   
hospitalization and the need for more aggressive interventions in these   
patients.  
  
RECORD 593  
Left ventricular thrombus in a patient with active crohn's disease: A case   
report  
Springston C. Greenspan A.  
American Journal of Gastroenterology (2010) 105 SUPPL. 1 (S349). Date of   
Publication: October 2010  
  
Purpose: Inflammatory bowel disease increases the risk of thromboembolism,   
especially during acute exacerbations. Several case-based and larger studies   
have previously reported cerebral, pulmonary, caval and portal vein thrombus   
formation. To date, the prevalence of IBD-associated cardiac   
intraventricular thrombus formation is not well established. A 42 year old,   
Caucasian female with a two year history of Crohn's disease complicated by   
recurrent flares necessitating oral corticosteroid therapy presented with   
abdominal pain, bloody diarrhea, fever and oral mucosal ulcers. A two week   
course of prednisone, ciprofloxacin and metronidazole did not improve her   
symptoms. Complete blood count revealed a hematocrit of 22% and Computerized   
Tomography showed mild colitis distal to the mid transverse colon down to   
her anus. An incidental finding of a left ventricular mass was also   
reported. Magnetic Resonance Imaging and 2D Echocardiography showed a smooth   
edged mass arising from the left ventricular apex consistent with a   
thrombus. Left ventricular ejection fraction was well preserved despite mild   
hypokinesis of the thin-walled, left ventricular apex. The patient did not   
exhibit signs and symptoms consistent with cardioembolic diseases.   
Hypercoagulability studies were unremarkable (factor V Leiden, Antithrombin   
III, protein C, protein S). The patient had no prior personal or family   
history of thrombus formation. Systemic anticoagulation with warfarin was   
initiated. Following adequate control of her Crohn's disease with infliximab   
and prednisone, the patient underwent surgical removal of the left   
ventricular mass via thoracotomy, which was confirmed as a thrombus by gross   
and microscopic evaluation. We report an extremely rare case of cardiac   
thrombus complicating refractory Crohn's disease. This rare extraintestinal   
manifestation of Crohn's disease can lead to significant morbidity including   
cerebrovascular accident and possibly death. Our patient remained   
asymptomatic but required aggressive medical management of her inflammatory   
bowel disease prior to intracardiac thrombectomy and prolonged systemic   
anticoagulation.  
  
RECORD 594  
Effect of anticoagulation therapy in patients with non cirrhotic   
extrahepatic portal vein thrombosis  
Spaander M. Hoekstra J. Hansen B.E. Janssen H.L.  
Hepatology (2010) 52 SUPPL. 1 (1068A). Date of Publication: October 2010  
  
Background: In patients with non cirrhotic extrahepatic portal vein   
thrombosis (EPVT) anticoagulation therapy should be considered if a recent   
thrombosis or prothrombotic state is present. However, data on this topic   
are scarce. Aim of this study was to assess the effect of anticoagulation   
therapy on recanalization, risk of gastrointestinal bleeding and recurrent   
thrombotic events in patient with EPVT. Methods: Consecutive patients with   
non cirrhotic EPVT, seen at our hospital from 1985 to 2009 were enrolled.   
Data were collected by systematic chart review. Results: Hundred-twenty   
patients (36% male;median age 44 years (range16-87))were included (median   
follow up 5.5 years(range 0.1-32.5)). Forty patients had acute EPVT and 71   
patients had chronic EPVT. Sixty-six patients were treated with   
anticoagulants. Recanalization was seen in 9(23%) patients with a recent   
thrombosis, six of whom used anticoagulants. Anticoagulants had a positive   
effect on recanalization (HR 2.6 p=0.14). Acute thrombosis (HR13.3 p=0.02)   
and presence of IBD (HR6.6 p=0.005) were significant predictors of   
recanalization. Failure of recanalization was significantly associated with   
presence of ascites (HR3.0 p=<0.01). In 37 patients 83 bleeding events   
occurred (variceal bleeding n= 52 and gastrointestinal non variceal bleeding   
n=31). Bleeding risk was 33% at 1-, 43% at 5- and 46% at 10- years.   
Gastrointestinal bleeding at diagnosis (HR2.1 p=0.007), ascites (HR2.0   
p=0.01) and use of anticoagulants (HR2.0 p=0.008) were significant   
predictors of (re)bleeding. Anticoagulants had no effect on the severity of   
gastrointestinal bleeding. Four fatal gastrointestinal bleedings occurred,   
two of whom used anticoagulants. Twenty-two new thrombotic events occurred   
in 19 patients (venous n=15 arterial n=7). Thrombotic risk was 3 % at 1- 8%   
at 5- and 24% at 10- years. Seventy-four percent of the thrombotic events   
occurred in patients with a prothrombotic disorder. Anticoagulants   
diminished the risk of venous thrombosis (HR 0.2 p= 0.11). Presence of a   
prothrombotic disorder (HR 3.1 p= 0.03) was the only significant predictor   
of a new thrombotic event. Bleeding or anticoagulation therapy had no   
significant effect on survival. Conclusion In patients with EPVT   
recanalization is significantly associated with acute thrombosis, and the   
presence of IBD. A new thrombotic event is mainly seen in patients with a   
prothrombotic disorder. anticoagulation therapy tended to increase   
recanalization and prevent new thrombosis. However, it significantly   
increased the risk of a gastrointestinal bleeding. These findings suggest a   
more careful role for anticoagulation therapy in EPVT patients.  
  
RECORD 595  
Transsplenic Endovascular Therapy of Portal Vein Stenosis and Subsequent   
Complete portal vein thrombosis in a 2-Year-Old Child  
Bertram H. Pfister E.-D. Becker T. Schoof S.  
Journal of Vascular and Interventional Radiology (2010)  
  
A complex catheter intervention for portal vein stenosis and subsequent   
complete thrombosis after split-liver transplantation was performed using   
transsplenic access to the portal vein circulation. The combination of   
intrahepatic, local thrombolysis and extrahepatic portal vein angioplasty   
performed twice on 2 consecutive days followed by anticoagulation with a   
high dose of heparin and clopidogrel completely resolved portal vein   
stenosis and thrombosis. Postinterventional angiographic and serial   
ultrasound examinations confirmed that the endovascular therapy was   
successful. In selected patients, percutaneous transsplenic access to the   
portal vein circulation may be used for diagnostic and therapeutic   
interventions even in early childhood. © 2010 SIR.  
  
RECORD 596  
Long-term survival after venous thromboembolism: A retrospective selected   
cohort study among young women  
Reitter S. Laczkovics C. Waldhoer T. Mayerhofer M. Vutuc C. Pabinger I.  
Haematologica (2010) 95:8 (1425-1428). Date of Publication: 2010  
  
Few data are available on long-term survival following venous   
thromboembolism. We performed a retrospective survival analysis covering the   
period January 1985 to December 2006 in 728 young women (median age 28.7   
years; interquartile range 21.6-36.3 years) with a history of venous   
thromboembolism who visited our clinic between 1985 and 1998. Mortality   
information was obtained from the Austrian Central Death Register. Survival   
of our patients was compared to the general Austrian female population after   
adjustment for age and calendar period. Overall, 23 patients (3.2%) died,   
the cumulative relative survival was 1.03 (95% CI 0.99-1.04). Site of venous   
thromboembolism or triggering factors had no significant influence. Venous   
thromboembolism does not reduce long-term survival in young women   
considering our median follow up of 14 years. The risk of fatal bleeding and   
quality of life should be assessed versus that of fatal recurrent venous   
thromboembolism when deciding on long-term anticoagulation in young women. ©   
2010 Ferrata Storti Foundation.  
  
RECORD 597  
anticoagulation for portal vein thrombosis in cirrhosis  
Qi X. Han G. Wu K. Fan D.  
American Journal of Medicine (2010) 123:9 (e19-e20). Date of Publication:   
September 2010  
  
RECORD 598  
Portal vein obstruction after liver transplantation in children treated by   
simultaneous minilaparotomy and transhepatic approaches: Initial experience  
Carnevale F.C. Santos A.C.B. Seda-Neto J. Zurstrassen C.E. Moreira A.M.   
Motta-Leal-Filho J.M. Marcelino A.Z. Cerri G.G. Chapchap P.  
CardioVascular and Interventional Radiology (2010) 33 SUPPL. 2 (299). Date   
of Publication: September 2010  
  
Purpose: portal vein thrombosis is a complication that occurs anytime after   
liver transplantation and can compromise the patient and graft survival. We   
describe a combined technique for portal vein recanalization in cases of   
portal vein obstruction after liver transplantation. Materials and Methods:   
Four children (1%), of 367 subjected to liver transplantation from June 1991   
to December 2008, underwent portal vein recanalization through a combined   
approach (transhepatic and minilaparotomy). Results: All children received   
left lateral hepatic segments, developed portal vein thrombosis (n=3) and   
stenosis (n=1), and presented with symptoms of portal hypertension after   
transplantation. Portal vein recanalization was tried by transhepatic   
retrograde access, and a minilaparotomy was performed when percutaneous   
recanalization was unsuccessful. Three patients underwent a successful   
portal recanalization and stent placement with the combined technique. In   
one patient, the recanalization was unsuccessful because of an extensive   
portomesenteric thrombosis. The other three children had the portal flow   
reestablished and followed with Doppler ultrasound studies. They received   
oral anticoagulation for 3 consecutive months after the procedure and the   
clinical symptoms subsided. Conclusion: In case of portal vein obstruction,   
the combined approach is technically feasible with good clinical and   
hemodynamic results. It is a minimally invasive procedure and can be tried   
to avoid or delay surgical treatment or retransplantation.  
  
RECORD 599  
TIPS - Where are we now?  
Bilbao J.I.  
CardioVascular and Interventional Radiology (2010) 33 SUPPL. 2 (140-141).   
Date of Publication: September 2010  
  
The idea of percutaneously establishing an intrahepatic connection between   
the hepatic veins and the portal vein dates back to 1969 when Rösch and   
Hanafee described the technique in laboratory animals (1). The intrahepatic   
tract between the portal vein and the hepatic vein was created using Teflon   
dilators and the connection was kept patent with a plastic tube. The   
introduction of the angioplasty balloon catheter allowed performing the   
dilatation in a less traumatic fashion. In 1983 Colapinto presented a group   
of patients in whom the procedure had been carried out with balloons without   
inserting any devices for stabilising the venous connection. As expected,   
the patency rate was poor (2). The experimental studies carried out by   
Palmaz, with the prosthesis designed by him (3), finally allowed to perform   
a transjugular intrahepatic porto systemic shunt (TIPS) in a safe and   
efficient manner. The first human cases were presented by Richter in 1989   
(4) and since, many articles with variable numbers of patients, randomized   
clinical trials and clinical notes have been published. This body of work   
allows for the establishment of clinical recommendations on when and in   
which patients to use TIPS in the treatment of complications of portal   
hypertension (5-8). According to the previous series of patients treated   
with TIPS, a major problem of the procedure are shunt tract stenoses which   
are the result of intimal thickening secondary to pseudointimal hyperplasia   
(proliferation of dense collagen and myofibroblasts) (9-10). On its genesis   
biliary-TIPS fistulae have been implied (11,12). Others, however, have   
demonstrated marked shunt stenoses (dense collagen and smooth muscle cells   
proliferation) without bile staining or bile duct proliferation (13). In   
draining hepatic vein stenosis intimal vein hyperplasia can also be   
observed, which is probably due to traumatic stress during the shunt   
procedure, high flow after TIPS or activation of smooth muscle cells by   
growth factors (11-13). As demonstrated in a randomized study, the use of   
stents coated with polytetrafluoroethylene (e-PTFE) improves TIPS patency   
and decreases the number of clinical relapses and reinterventions without   
increasing the risk of encephalopathy (14) compared to bare stents. At this   
moment all available RCTs used bare stents and their conclusions might be   
deeply modified by the use of e-PTFE-covered stents (15). With the use of   
these new devices, the role of TIPS in the management and treatment of the   
complications of portal hypertension continues to evolve (14, 15) and has   
been re-evaluated, updating the clinical recommendations for TIPS (8). TIPS   
and variceal bleeding: At this moment, TIPS is accepted as a second-line   
therapy and should be used when medical and endoscopic treatment have failed   
(8). In such circumstances TIPS is able to control bleeding in 89-100% of   
cases with a re-bleeding rate of 15% and a mortality (first month) of 30%   
(16-22). It is well known that there are patients with a high degree of   
rebleeding (30-60% at one year) after medical and endoscopic treatments   
(23). Therefore patients at a higher risk of re-bleeding need to be detected   
at an earlier stage. Two studies have identified which parameters, measured   
in acutely bleeding patients, are predictors of early re-bleeding:   
portosystemic gradient > 20 mmHg, advanced Child-Pugh class and systemic   
blood pressure < 100 mmHg (24, 25). According to some authors, limiting the   
use of TIPS as a rescue therapy in cases of failure of vasoactive drugs and   
endoscopic therapy needs to be revised and TIPS should be considered as   
first-line treatment in high-risk selected patients (15). Two recent studies   
comparing TIPS to surgery (distal splenorenal shunt-DSRS) have been   
published (26, 27). They have demonstrated that TIPS (with covered   
prostheses) is as effective as DSRS in preventing variceal re-bleeding and   
may be more cost effective. It has therefore been said in a recent editorial   
that “the era of surgical shunting for treatment of portal hypertension is   
over” (28). TIPS and refractory ascites: Refractory ascites represent   
another indication for TIPS (29). A review of several RCTs has shown that   
TIPS was able to significantly reduce not only the risk of recurrence of   
ascites, but also the mortality rate of patients with refractory ascites   
when compared with repeated large-volume paracentesis (30). The predictive   
factors for survival are: age, serum bilirubin and sodium levels. Younger   
patients with a less compromised liver function and systemic hemodynamics   
may benefit from TIPS as a first-line treatment for refractory ascites (15).   
TIPS and Budd-Chiari syndrome: A large study with data obtained from 6   
European institutions has collected the long-term followup of 124 patients   
with Budd-Chiari Syndrome treated with TIPS. Patients were treated with   
both, non-covered and covered stents. The OLT-free survival was 88% at one   
year and 78% at 5 years. For them, TIPS should no longer be considered as a   
bridge to liver transplantation, but the treatment of choice when   
anticoagulation has failed. Independent risk factors of mortality that have   
been identi- fied are: age, serum bilirubin levels and INR/PT values (31).   
TIPS and Portal thrombosis (PVT): Some previous reports have shown short   
series of patients with PVT in whom TIPS may be effective for the palliation   
of symptoms (32). The largest series of patients with PVT with and without   
cavernomatous transformation has recently been published by Senzolo (33).   
PVT is not a contraindication for TIPS anymore and should be considered in   
patients with severe and life-threatening complications or in whom the   
thrombus may jeopardize liver transplantation. Even in specialized centres,   
the success rate in accomplishing the procedure is 73%, which remarks the   
need to refer those patients to selected units with a large experience in   
performing TIPS. In summary, the vision of Jösef Rösch who forty years ago   
envisioned the possibility of performing percutaneous connections between   
the portal vein and the inferior vena cava is an accepted reality today.   
TIPS is continuously evolving as an established method of treatment in a   
wide variety of portal vein complications in cirrhotic and non-cirrhotic   
patients. Although implemented in many institutions throughout the world,   
some patients with expected difficulties (i.e. PVT or Budd-Chiari syndrome)   
should be referred to especially dedicated centres.  
  
RECORD 600  
Predicting thrombotic complications after liver transplantation in patients   
with Budd Chiari syndrome  
Westbrook R. Westbrook R. Orr D. Heaton N. O'Grady J. Patel R. Lea N.   
Quaglia A. Pagliuca A. Arya R. Mufti G. Heneghan M.  
Gut (2010) 59 SUPPL. 2 (A11). Date of Publication: September 2010  
  
Introduction: Myeloproliferative disorders (MPD) are the commonest cause of   
Budd Chiari syndrome (BCS). A somatic mutation of the tyrosine kinase JAK2   
gene (JAK2V617F) is present in a large proportion of patients with MPD and   
is used as a screening tool to detect occult MPD. Recently a germline 46/1   
haplotype block and mutations in the TET2 gene have also been implicated in   
the pathogenesis of MPD. We evaluate whether these underlying genetic   
abnormalities are relevant to the occurrence of thrombotic complications   
post liver transplantation (LT). Method: Samples of DNA were extracted from   
total blood or bone marrow. Real-time PCR was performed to screen for JAK2   
mutations. TET2 mutations were analysed by next generation high throughput   
DNA sequencing (Roche 454). DNA was analysed by pyrosequencing for two SNP's   
which tag the 46/1 haplotype. Histology of liver biopsies performed for   
graft dysfunction were reviewed for evidence of veno-occlusive disease   
(VOD). The INR post LT and patient outcomes were recorded. Results: 36   
patients underwent LT for BCS between 1995 and 2008. Median duration of   
follow-up after LT was 40 months (1-195 months) and 1-year survival was 84%.   
Pro-coagulant conditions were identified in 22 patients (MPD n=17, Protein C   
Deficiency n=2, Behcet's n=2 and lupus anti-coagulant n=1). The remaining 14   
patients were classed as idiopathic. Overall, 22/36 (61%) were positive for   
the JAK2 mutation, 6/27 (22%) for the TET2 mutation and 19/26 (73%) for the   
46/1 haplotype. In the idiopathic cohort, 8/13 (63%) tested positive for   
JAK2 suggesting latent MPD. All patients were treated with warfarin   
following LT. Thrombotic complications occurred in 12/36 (33%) and included   
hepatic artery thrombosis (n=3, 2/3 being late), VOD (n=7), splenic vein   
thrombosis (n=1) and portal vein thrombosis (n=1), at a median time of 40   
months post LT (range 1-164 months). Re-transplantation was more common in   
those with thrombotic complications (7/12 (58%) vs 1/24 (4%), (p=0.0006))   
and mortality was higher (4/12, (25%) vs 3/24, (13%)), but this did not   
reach statistical significance (p=0.2). The presence of a JAK2 mutation was   
associated with the development of a thrombotic complication post LT (11/12   
vs 1/24, p=0.01). Neither the 46/1 haplotype nor the TET2 mutation was   
associated with an increase in post LT thrombotic complications or   
morbidity. Mean INR was not significantly different in those patients who   
developed a thrombotic complication (2.73 vs 2.70, p=NS). Conclusion: A JAK2   
mutation appears to be associated with an increased risk of recurrent BCS   
and other thrombotic complications post LT. Thrombotic complications   
following LT are associated with an increase in morbidity and mortality. In   
patients with a JAK2 mutation, the role of additional anticoagulation or   
JAK2 inhibitor therapy needs to be investigated to try and prevent   
thrombotic complications.  
  
RECORD 601  
Feasibility and long-term evolution of TIPS in cirrhotic patients with   
portal thrombosis  
Perarnau J.-M. Baju A. D'Aalteroche L. Viguier J. Ayoub J.  
European Journal of Gastroenterology and Hepatology (2010) 22:9 (1093-1098).   
Date of Publication: September 2010  
  
AIM: Many researchers consider portal thrombosis (PT) as a contraindication   
to transjugular intrahepatic portosystemic shunt (TIPS). The aim of this   
retrospective study was to compare the feasibility and long-term prognosis   
of TIPS in cirrhotic patients, with and without, complete PT. PATIENTS AND   
Methods: Four hundred and thirty-six consecutive cirrhotic patients with   
portal hypertension were referred for TIPS, between 1990 and 2004. These   
patients were divided into two groups according to their portal patency.   
PT+: 34 patients with complete PT with cavernoma (19) or without (15)   
cavernoma versus PT-: 402 patients with normal portal patency (308) and   
partial PT (94). Epidemiological data were compared using the χ and   
Student's t-tests, and comparative evolution was made from actuarial data   
using the log-rank test. Results: PT+ patients were more frequently women   
with viral hepatitis, and TIPS was performed more often for bleeding   
indications. The TIPS success rate was significantly lower in the PT+ group   
(79%) than in the PT- group (99.5%) (P<10). Presence of a cavernoma   
decreased the success rate to 63%. TIPS was always feasible in cases of   
recent PT and portal cavernoma with an accessible intrahepatic patent portal   
branch. Early and late outcome and complications were not significantly   
different between the two groups. Conclusion: Complete PT does not modify   
TIPS' long-term outcome. Rather than a contraindication, PT should be   
considered as an indication for TIPS in cirrhotic patients with accessible   
intrahepatic portal vein. Further randomized studies should be planned in   
cirrhotic patients with recent PT to better qualify TIPS and anticoagulation   
indications, respectively. Copyright © 2010 Lippincott Williams & Wilkins.  
  
RECORD 602  
New oral anticoagulants essentially required for long-term secondary   
prophylaxis in four different cases  
Seidel H. Pötzsch B. Hertfelder H.-J. Harbrecht U. Oldenburg J.  
Hamostaseologie (2010) 30:1 (A109). Date of Publication: 2010  
  
Background: Vitamin K antagonists (VKA) represent the most commonly   
prescribed drugs for the therapy and long-term prevention of thromboembolic   
conditions. Drug intolerance, instability of VKA therapy and bleeding   
complications may limit the use of VKA therapy. A change of VKA agents (e.   
g. phenprocoumon to warfarin) usually assists to avoid such adverse events.   
For short or intermediate-term anticoagulation a switch to LMWH or   
fondaparinux is often a suitable alternative. However, more switching   
options may be required for few patients with further contraindications.   
Methods: (1) We report on a 15-year-old boy with compound heterozygosity for   
the FVLeiden- and prothrombin-G20210A mutation who developed spontaneous   
portal vein thrombosis. Due to an inherited deficiency of factor VII the   
patient showed an unstable response to VKA. Alternative treatment with   
dabigatran, 2 x 75 mg daily was initiated. (2) A 68-year-old male patient   
with atrial fibrillation (AF) required VKA therapy because of developing   
pulmonary embolism (PE) in presence of inhibitors interfering with   
VK-dependent clotting factors and most likely antiphospholipid antibodies.   
We suggested therapy with dabigatran with a daily dose of 2 x 110 mg. (3) A   
52-year-old male with AF, received 10 mg rivaroxaban daily because of   
coumarin-induced hepatitis. (4) A 42-year-old female with postthrombotic   
syndrome, recurrent PE and a history of heparin-induced thrombocytopenia and   
cross-reactivity to danaparoid had been treated with fondaparinux for   
intolerance of phenprocoumon and warfarin. When developing intolerance of   
also fondaparinux we switched her to rivaroxaban, 10 mg daily. Results: No   
further adverse event occur in any of the four patients. Normal D-Dimer in   
the follow-up visits indicate efficacy of anticoagulant treatment.   
Conclusion: In patients with contraindications against VKA respectively LMWH   
or fondaparinux the new oral anticoagulants rivaroxaban and dabigatran   
appear suitable alternatives.  
  
RECORD 603  
The reply  
Parikh S. Shah R. Kapoor P.  
American Journal of Medicine (2010) 123:9 (e21). Date of Publication:   
September 2010  
  
RECORD 604  
Pylephlebitis in the child: A challenging diagnosis  
Gatibelza M.-E. Gaudin J. Mcheik J. Levard G.  
Archives de Pediatrie (2010) 17:9 (1320-1324). Date of Publication:   
September 2010  
  
Pylephlebitis or septic thrombophlebitis of the portomesenteric veins is a   
complication of intra-abdominal infections. The disease is rare in children   
and the diagnosis is often delayed. The morbidity of pylephlebitis is   
relatively low, although there is a risk of residual thrombosis. We report   
on 2 cases of pylephlebitis in a 12-year-old girl and a 13-year-old boy,   
following undiagnosed appendicitis. In the 1st case, the young girl had been   
misdiagnosed with Salmonella infection and was given antibiotics; in the 2nd   
case, the boy had retrocecal appendicitis that was clinically subacute. An   
accurate diagnosis was finally made in both cases by CT scan. Both children   
evolved satisfactorily following appendectomy, long-term antibiotics, and   
anticoagulation. Clinically, the severe sepsis associated with pylephlebitis   
is at the forefront. Physical examination is often normal and therefore of   
little help; the knowledge of a preceding abdominal infection leads to   
further radiological investigations. Biologically, there are pronounced   
signs of infection. CT is the preferred exam for diagnosing pylephlebitis,   
as it can also show the underlying cause of the intra-abdominal sepsis or   
possible complications. Doppler sonography is recommended more for follow-up   
of the portal vein thrombosis. Treatment of pylephlebitis associated with   
appendicitis always includes long-term antibiotics. An appendectomy is   
always performed either at the time of diagnosis or later. The need for   
anticoagulation therapy in children is controversial. However, most   
pediatricians recommend its use, beginning as soon as possible, to be   
continued until normalization of portal vein flow. © 2010 Elsevier Masson   
SAS.  
  
RECORD 605  
Duet TRSTM reload is a preloaded absorbable reinforcement material on an   
endoscopic stapler that is straightforward and effective for gastric pouch   
creation  
Pryor A. Jiang N. DeMaria E.J. Portenier D.D. Sudan R. Torquati A.  
Obesity Surgery (2010) 20:8 (984). Date of Publication: August 2010  
  
Background: Preloaded absorbable reinforcement material (PARM) (Duet TRSTM,   
Covidien, North Haven, CT) is theorized to facilitate hemostatic staple   
lines without added time or user error. We undertook a prospective trial of   
PARM in gastric pouch creation during Roux-en-Y gastric bypass (RYGB).   
Methods: Consecutive patients presenting for non-revisional RYGB with BMI<60   
were approached for enrollment. Demographics and pre-operative labs were   
recorded. Patients underwent laparoscopic RYGB using the surgeon's standard   
technique. All gastric pouch transections were created using linear staplers   
with PARM. OR time, peri-operative and post-operative complications were   
recorded. Patients were assessed at up to 1 month after surgery. Results:   
Thirty patients underwent surgery in the study. Twenty-four have completed   
their post-operative visit. 5 Men and 25 Women were enrolled. Pre-operative   
BMI averaged 46 (38-62). Post-operative BMI averaged 42 (34-56) at 3 weeks   
post-operative. Eighty-eight PARM reloads were used in the study.   
Eighty-five (97%) of the PARM staple fires (97%) appeared ideal and did not   
require supplemental clips or suture reinforcement. Only one firing (1%) was   
felt by the surgeon to require over-sewing. One cartridge had the PARM   
dislodge during placement and one failed to open. OR time averaged 99   
minutes (62-165). Average length of stay was 1.3 days (1-2). Three adverse   
events were seen. Two were anastomotic strictures not involving the PARM and   
one was a portal vein thrombosis treated with anticoagulation. There was no   
leak and no transfusion requirement in the study population. Conclusion:   
PARM can be used safely and efficiently with a low complication rate for   
gastric pouch creation.  
  
RECORD 606  
portal vein thrombosis postlaparoscopic splenectomy presenting with   
infarction of gut: Review of risk factors, investigations, postoperative   
surveillance, and management  
MacHado N.O. Chopra P.J. Sankhla D.  
Surgical Laparoscopy, Endoscopy and Percutaneous Techniques (2010) 20:4   
(273-277). Date of Publication: August 2010  
  
portal vein thrombosis after laparoscopic splenectomy is a known   
complication even though it is underappreciated. Presenting symptoms are   
usually mild and nonspecific. Progression to intestinal infarction and   
portal hypertension are potentially life threatening complications. The   
short hospital stay associated with laparoscopic approach could delay early   
diagnosis, unless routine imaging studies is planned after discharge. We   
present a patient who after laparoscopic splenectomy for idiopathic   
thrombocytopenic purpura developed portal vein thrombosis leading to   
infarction of small intestine 9 days after the surgery. She made uneventful   
recovery after resection and anticoagulation. Literature is reviewed to   
assess the risk factors and discuss the present status regarding   
investigations, surveillance in postoperative period, management of   
established case, and role of prophylactic anticoagulation. Copyright © 2010   
by Lippincott Williams & Wilkins.  
  
RECORD 607  
Liver transplantation in patients with portal vein thrombosis is associated   
with increased risk of mortality according to thrombosis extension  
Figueroa E. Pérez R.M. Arrese M. Soza A. Domínguez P. Torres J. Guerra J.F.   
Jarufe N. Martínez J.  
Liver Transplantation (2010) 16 SUPPL. 1 (S212)  
  
portal vein thrombosis (PVT) is a serious complication of end-stage liver   
disease and represents a challenge for most liver transplantation (LT)   
teams. It has been considered a LT contraindication in many centers. With   
innovative surgical techniques, such as thrombectomy and portal vein   
reconstruction using vein grafts, current PVT patients can be successfully   
submitted to LT. The aim of this study was to analyze the results of PVT   
treatment in patients undergoing LT in our center. Patients and methods:   
Between January 1994 and September 2009, 115 LT were performed over 105   
patients; on 12 (10.9%) PVT was found. In 6 (50%), thrombosis was disclosed   
by pre-transplant routine imaging on waiting list. In 3 of them systemic   
anticoagulation treatment was indicated. PVT was classified in four grades   
described by Yerdel and Mc Master (2000). Results: On transplant procedure,   
only 9 patients had a PVT, 7 (58%) males; mean age 55 ± 10 [29 - 64]   
years-old. PVT was detected during surgery in 75%. PVT grade I; II; III and   
IV was found on 2, 3, 3 and 1 patients respectively. The 3 patients with PVT   
on pre-transplant imagery none found at surgery had grade I (1) and grade II   
(2) PVT. Techniques used during procedure were an eversion thrombectomy in 6   
(50%) patients, simple thrombectomy in one and an extra-anatomical   
mesenteric vein graft in 2 patients with grade III and IV PVT respectively.   
Morbidity was 50%. No recurrence of PVT was detected on doppler/Imagery   
follow-up, and in-hospital mortality occurred on 2 patients with PTV grade   
III and IV due to hemorrhagic strokes in one case and to invasive   
aspergillosis in the other. One-year survival was 67%. Conclusion: Liver   
transplantation in PVT patients was associated with increased risk of   
mortality, according to PVT extension.  
  
RECORD 608  
portal vein thrombosis in pregnancy - A case series  
Anbazhagan A. Harper A. Bailie C.  
Archives of Disease in Childhood: Fetal and Neonatal Edition (2010) 95   
SUPPL. 1 (Fa60). Date of Publication: June 2010  
  
portal vein thrombosis is rare in pregnancy. It is associated with   
thrombophilias, liver cirrhosis, abdominal infections and myeloproliferative   
disorders. The hypercoagulable state of pregnancy itself can precipitate   
this condition. The incidence of spontaneous abortion, prematurity, SGA   
babies and perinatal death are high. Long term anticoagulation and timely   
recognition and management of complications is the key to management. Here   
the authors present a case series of 3 pregnant women with portal vein   
thrombosis, and its associated complications namely portal hypertension,   
hypersplenism, thrombocytopenia, oesophageal varices and ascites that were   
successfully managed by a multidisciplinary team of specialists. All three   
women presented with the condition diagnosed prior to pregnancy. In one of   
them the cause was idiopathic and the other two resulted from thrombophilia   
(protein S deficiency and Factor V Leiden mutation). They had fortnightly   
antenatal checks, serial fetal well-being scans, prophylactic betamethasone   
for fetal lung maturity and treatment with propranolol, spironolactone and   
clexane. Two of them needed oesophageal banding during pregnancy. The three   
women were delivered by uncomplicated elective caesarean section at 38, 32   
and 31 weeks of gestation respectively. The mothers and babies were   
discharged home in good health following an uneventful puerperium.  
  
RECORD 609  
Mesenterico-portal bypass (“Rex” Shunt) for portal vein thrombosis after   
adult living donor liver transplantation  
Soejima Y. Shirabe K. Taketomi A. Uchiyama H. Maehara Y.  
Liver Transplantation (2010) 16 SUPPL. 1 (S188)  
  
(Purpose) portal vein thrombosis (PVT) after liver transplantation (LT) is a   
relatively common but serious complication which could lead to portal   
hypertension or a direct graft loss. A “Rex” shunt created between the   
superior mesenteric vein (SMV) and the umbilical portion (UP) of the liver   
can be a useful option to treat PVT after living donor liver transplantation   
(LDLT) but few have been reported so far. We present a case of PVT after   
LDLT who underwent the procedures using the own inferior jugular vein (IJV)   
and the gonadal vein (GV) as a shunt graft. (Methods) The patient was a   
46-year-old female who developed PVT at 2 months after ABO-incompatible,   
left lobe LDLT for giant hemangioma of the liver. An 8cm-long left IJV and a   
5cm-long, enlarged GV were procured for venous grafts. The IJV and GV were   
independently anastomosed endto- side to the infrapancreatic SMV and the   
U-portion of the left lobe graft, respectively. The two venous grafts were   
then anastomosed end-to-end and reperfused, which resulted in resumed   
hepatopetal portal fl ow in the liver. (Results) The operative time was   
8.5hrs and the blood loss was 482ml. The shunt was patent immediately after   
the procedures but was thrombosed 2 days after the procedures probably due   
to the insufficient infl ow from the SMV and the absence of anticoagulation   
therapy, for which emergent thrombectomy and ligation of the additional   
collateral veins followed by full anti-coagulation therapy were performed.   
The shunt remains open at 3 month after the procedure with a normal anmonia   
level and liver function. (Conclusion) “Rex” shunt using a donor's own vein   
graft is a feasible and valuable option to treat PVT after adult LDLT.  
  
RECORD 610  
Predicting thrombotic complications after liver transplantation in patients   
with Budd Chiari Syndrome  
Westbrook R. Orr D. Heaton N. O'Grady J. Patel R. Lea N. Smith A. Quaglia A.   
Mufti G. Heneghan M.  
Liver Transplantation (2010) 16 SUPPL. 1 (S72)  
  
Background: Myeloproliferative disorders (MPD) are the commonest cause of   
Budd Chiari Syndrome (BCS). The presence of a JAK2 mutation in “idiopathic”   
BCS is used as a screening tool to identify latent MPD. Recently a germline   
46/1 haplotype block and mutations in the TET2 gene have been implicated in   
the pathogenesis of MPD. We evaluate the incidence and clinical relevance of   
these genetic abnormalities in patients transplanted for BCS. Real time PCR   
was performed to screen for JAK2 mutations. TET2 mutations were analysed by   
next generation high throughput DNA sequencing (Roche 454). DNA was analysed   
by pyrosequencing for 2 SNP's which tag the 46/1 haplotype. Histology of   
liver biopsies performed for graft dysfunction to identify venoocclusive   
disease (VOD), mean INR and patient outcome were recorded. Results: Thirty   
six patients underwent LT for BCS between 1995 and 2008. Median duration of   
follow-up after LT was 40 months (1-195 months) and 1 year survival was 84%.   
Pro-coagulant conditions were identified in 22 patients (MPD n=17, Protein C   
Deficiency n=2, Behcet's n=2 and lupus anticoagulant n=1), 14 patients were   
labelled idiopathic. Overall, 22/36 (61%) had the JAK2 mutation (8/13 in the   
idiopathic cohort), 6/27 (22%) the TET2 mutation and 19/26 (73%) the 46/1   
haplotype. All patients received warfarin following LT. Thrombotic   
complications occurred in 12/36 (33%) and included hepatic artery thrombosis   
(n=3, 2/3 late), VOD (n=7), splenic and portal vein thrombosis (n=2), at a   
median time of 40 months post LT (range 1-164 months). The JAK2 mutation was   
associated with thrombotic complications post LT (11/12 vs. 1/24, p=0.01),   
but the 46/1 haplotype and the TET2 mutation wern't. Thrombotic   
complications were associated with re-transplantation (7/12 (58%) vs. 1/24   
(4%), p=0.0006) and increased mortality (4/12, (25%) vs. 3/24, (13%),   
p=0.2). Mean INR was not significantly different in patients who developed a   
thrombotic complication (2.73 vs. 2.70, p=NS). Conclusions: A JAK2 mutation   
is associated with an increased risk of thrombotic complications post LT. In   
patients with a JAK2 mutation the role of additional anticoagulation or JAK2   
inhibitor therapy should be investigated to try and prevent thrombotic   
complications.  
  
RECORD 611  
Venous thrombotic emergencies  
DeLoughery T.G.  
Hematology/Oncology Clinics of North America (2010) 24:3 (487-500). Date of   
Publication: June 2010  
  
Thrombosis is a common complication of cancer, occurring in up to 15% of   
patients. This article reviews the diagnosis and management of the most   
common cancer-related thrombotic problems; deep venous thrombosis, pulmonary   
embolism, and catheter-related thrombosis. Rarer entities, such as cerebral   
vein thrombosis and Budd-Chiari syndrome, are also reviewed. © 2010 Elsevier   
Inc.  
  
RECORD 612  
Adverse drug events associated with disorders of coagulation  
Barletta J.F. Cooper B. Ohlinger M.J.  
Critical Care Medicine (2010) 38:6 SUPPL. (S198-S218). Date of Publication:   
June 2010  
  
Disorders of coagulation are common adverse drug events encountered in   
critically ill patients and present a serious concern for intensive care   
unit (ICU) clinicians. Dosing strategies for medications used in the ICU are   
typically developed for use in noncritically ill patients and, therefore, do   
not account for the altered pharmacokinetic and pharmacodynamic properties   
encountered in the critically ill as well as the increased potential for   
drug-drug interactions, given the far greater number of medications ordered.   
This substantially increases the risk for coagulation-related adverse   
reactions, such as a bleeding or prothrombotic events. Although many   
medications used in the ICU have the potential to cause coagulation   
disorders, the exact incidence will vary based on the specific medication,   
dose, concomitant drug therapy, ICU setting, and patient-specific   
comorbidities. Clinicians must strongly consider these factors when   
evaluating the risk/benefit ratio for a particular therapy. This review   
surveys recent literature documenting the risk for adverse drug reactions   
specific to bleeding and/or clotting with commonly used medications in the   
ICU. Copyright © 2010 by the Society of Critical Care Medicine and   
Lippincott Williams & Wilkins.  
  
RECORD 613  
Asparaginase-related venous thrombosis in UKALL 2003- re-exposure to   
asparaginase is feasible and safe  
Qureshi A. Mitchell C. Richards S. Vora A. Goulden N.  
British Journal of Haematology (2010) 149:3 (410-413). Date of Publication:   
May 2010  
  
We report the incidence and outcome of venous thrombosis (VT) in the UK   
acute lymphoblastic leukaemia (ALL) 2003 trial. VT occurred in 59/1824   
(3·2%) patients recruited over 5 years with 90% occurring during a period of   
Asparagine depletion. Pegylated Escherichia Coli Asparaginase (Peg-ASP) 1000   
units/m(2) was used throughout. Thirty-four children received further   
Peg-ASP, most with concurrent heparin prophylaxis. There were no episodes of   
bleeding or recurrent thrombosis. Optimal Asparagine depletion is central to   
success of modern regimes for treatment of ALL. This report confirms a   
significant risk of thrombosis with such therapy, but demonstrates that   
re-exposure to Asparaginase is feasible and safe. © 2010 Blackwell   
Publishing Ltd.  
  
RECORD 614  
A case of diabetic ketoacidosis presenting with acute pancreatitis and   
visceral vein thrombosis  
Kadaria D. Bergeron J. Pant N. Patel N. Nasser W.  
American Journal of Respiratory and Critical Care Medicine (2010) 181:1   
MeetingAbstracts. Date of Publication: 1 May 2010  
  
Introduction: Diabetic Ketoacidosis (DKA) is one of the frequent reasons for   
admission, so is acute pancreatitis (AP). AP also is one of the   
manifestations of antiphospholipid syndrome (APS) whereas visceral vein   
thrombosis can be a complication of AP. We report a case of 19-yr old female   
who presented with DKA and was found to have AP along with portal, splenic   
and mesenteric vein thrombosis. Case Report: A 19 year old AAF, known case   
of Multiple sclerosis, Diabetes Mellitus presented with complains of nausea,   
vomiting and abdominal pain for three days. She also gave history of   
constipation for two days. Past medical history was negative for   
pancreatitis, venous or arterial thrombosis. Physical examination revealed   
dehydration, tender abdomen and absent bowel sounds. Initial labs showed   
blood sugar of 560 mg/dl, anion gap of 26 and urine and serum positive for   
ketone. Patient also had elevated lipase (534) and amylase (224). Patient   
was started on treatment for DKA and Pancreatitis. CT abdomen was done which   
showed pancreatic necrosis, hepatic infarction along with portal, splenic   
and superior mesenteric vein thrombosis. CT was negative for gall stones.   
Patient was started on anticoagulation for her portal vein thrombosis.   
Causes for hypercoagulability were sought and she was found to be positive   
for lupus anticoagulant. Anticardiolipin antibody, Antiglycoprotein   
antibody, ANA and RA were negative. Patient improved with treatment. Repeat   
CT scans showed patency of her visceral veins. She was discharged home in   
stable condition. Discussion: The relationship between DKA and AP has been   
sought. AP coexisting with DKA as a cause or result has been reported   
previously. As per literature DKA may mask a coexisting AP, which occurs in   
nearly 10-15 % of cases. This case underlines the importance of careful   
consideration of AP in cases of DKA especially if initial presentation   
includes severe abdominal pain. One of the rare causes for AP is APS. On   
other hand AP is also considered a cause for visceral vein thrombosis   
especially portal vein thrombosis. Our lady had AP in her presentation and   
CT scan showed visceral vein thrombosis. She was later found to be positive   
for lupus anticoagulant. We don't know which one among these two was   
precipitating cause, but this case suggests consideration of APS and   
visceral vein thrombosis in case of AP as early initiation of   
anticoagulation in visceral vein thrombosis has shown to improve outcome. No   
relationship between DKA and APS was found in literature.  
  
RECORD 615  
The management of pregnancy in paroxysmal nocturnal haemoglobinuria on long   
term eculizumab  
Kelly R. Arnold L. Richards S. Hill A. Bomken C. Hanley J. Loughney A.   
Beauchamp J. Khursigara G. Rother R.P. Chalmers E. Fyfe A. Fitzsimons E.   
Nakamura R. Gaya A. Risitano A.M. Schubert J. Norfolk D. Simpson N. Hillmen   
P.  
British Journal of Haematology (2010) 149:3 (446-450). Date of Publication:   
May 2010  
  
In Paroxysmal nocturnal haemoglobinuria (PNH), pregnancy is associated with   
increased maternal and foetal complications to such an extent that the   
condition has been considered relatively contra-indicated in PNH. Eculizumab   
has revolutionized the treatment of PNH. We evaluate its use in pregnancy to   
date. We report on seven patients exposed to eculizumab at different stages   
of pregnancy including the first two patients to receive the drug from   
conception to delivery. There was no evidence of complement blockade from   
cord blood samples taken at delivery. Eculizumab appears safe to use in this   
setting and is likely to prevent many of the complications usually observed.   
© 2010 Blackwell Publishing Ltd.  
  
RECORD 616  
Acute mesenteric, portal and inferior vena cava (IVC) venous thrombosis:   
Optimal outcome achieved with anticoagulation  
Alvi A.R. Bibi S. Rehman Z. Niazi S.K.  
Journal of the Pakistan Medical Association (2010) 60:5 (397-399). Date of   
Publication: May 2010  
  
The prevalence and clinical spectrum of acute mesenteric venous thrombosis   
(AMVT) in Pakistan is largely unknown. The authors report two patients with   
acute mesenteric, portal and inferior vena cava venous thrombosis confirmed   
on CT imaging. The diagnoses were established within 24 hours of   
presentation and both patients were successfully treated with therapeutic   
heparin during hospital admission and continued on oral warfarin because of   
hypercoagulable state. The protocol that we currently use is evidence based   
and is leading to optimal outcome.  
  
RECORD 617  
Portal vein thrombus after pediatric proctocolectomy with ileoanal   
anastomosis  
Ibele A.R. Kennedy G.D. Lund D.P. Nichol P.F.  
Journal of Pediatric Surgery (2010) 45:5 (1026-1029). Date of Publication:   
May 2010  
  
In adults, mesenteric venous thrombosis with extension into the portal   
system is a known complication of total proctocolectomy with pouch ileoanal   
anastomosis. Although frequently reported in adults, this complication is   
rare in pediatric patients undergoing this operation. We report 2 cases of   
adolescent patients with ulcerative colitis who experienced portal vein   
thrombosis after this procedure. Both were treated with systemic   
anticoagulation therapy with complete resolution of their clots. We   
recommend that mesenteric/portal venous thrombosis be considered in the   
differential diagnosis in any child presenting with fever, abdominal pain,   
and leukocytosis after restorative proctocolectomy with ileal pouch   
anastomosis and that imaging obtained to evaluate abdominal complaints in   
this population be directed toward ruling out this complication. © 2010   
Elsevier Inc. All rights reserved.  
  
RECORD 618  
Anticoagulant therapy is safe and effective in preventing portal vein   
thrombosis (PVT) in advanced cirrhotic patients: A prospective randomized   
controlled study  
Zecchini R. Ferrari A. Bernabucci V. Lei B. Vukotic R. De Maria N. Schepis   
F. Marietta M. Fornaciari G. Schianchi S. Villa E.  
Journal of Hepatology (2010) 52 SUPPL. 1 (S460). Date of Publication: April   
2010  
  
Background: PVT is a frequent complication of advanced cirrhosis, the   
reported prevalence being 8-25%. PVT leads to severe deterioration of   
clinical course and death and may also strongly compromise post-transplant   
prognosis. Safety and efficacy of anticoagulation have never been   
prospectively tested for its prevention. We therefore designed a prospective   
randomized trial of anticoagulant therapy in advanced cirrhotic patients to   
verify whether it can prevent PVT and improve course of disease and survival   
(Eudract 2007-007890-22). Methods: Cirrhotic patients, Child B7-C10, were   
randomized to receive enoxaparin 4000 IU/die or placebo for 12 months   
followed by 12 months observation. US was performed every 3 months and CT   
every 6 months to check for portal vein axis. PVT was considered as relevant   
event when it was either complete or involved more than 50% of portal vein   
diameter and was symptomatic (partial PVT). Primary aim of the study was   
evaluation of efficacy in preventing PVT; secondary aims were assessment of   
safety of anticoagulation, effect on occurrence of decompensation and/or   
survival. Results: We report the events of the 51 patients (26 randomized to   
treatment and 25 to placebo) who completed 24 months of observation. No   
relevant side effects, in particular no hemorrhagic events, were   
attributable to the active drug. During the 1-year study period, PVT (2   
complete, 3 partial) occurred in 5 patients on placebo and in none on   
enoxaparin [Kaplan-Meier (KM) p=0.045 log rank test)]. One patient with   
complete PVT died of septic shock shortly after developing PVT while the   
other recovered. During follow-up 2 additional events occurred, one in the   
placebo group and the other in 1 patient in the active arm, 4 months after   
enoxaparin discontinuation. In treated patients, fewer episodes of   
decompensation occurred during the study period (KM p = 0.034); this   
advantage was lost during followup (KM p = 0.474). At logistic regression   
analysis, the only factor significantly associated with risk of developing   
PVT was degree of portal hypertension (OR 7.028; 95%CI 1.155-42.780;   
p=0.034). Conclusions: In this prospective randomized controlled study,   
enoxaparin was shown to be safe and effective in preventing both occurrence   
of PVT and decompensation in cirrhotics with advanced stage of disease.  
  
RECORD 619  
Peroral transhepatic cholangioscopy and lithotripsy after biliopancreatic   
diversion  
Perez-Miranda M. De La Serna C.  
Gastrointestinal Endoscopy (2010) 71:5 (AB101). Date of Publication: April   
2010  
  
Background: Bariatric surgery makes ERCP more difficult. Peroral ERCP is   
feasible using enteroscopy after Rox-en-Y, and intraoperative ERCP through   
the excluded gastric antrum. However, after BPD with distal gastrectomy   
patients with CBD stones require either repeat surgery or PTC, the former   
particularly inconvenient after cholecystectomy. We offer a novel endoscopic   
approach in this setting, illustrated by the case of a 72 y.o. female with   
symptomatic proven residual CBD stones after BPD. Endoscopic methods: A   
3-step endoscopic treatment was carried out: 1) EUS-guided   
hepatico-gastrostomy using a 10mm biliary covered SEMS; 2) Peroral   
cholangioscopy & lithotripsy (EHL) with a 5.2mm pediatric gastroscope thru   
the transhepatic fistula 4 weeks later, after removal of the c-SEMS. Stone   
fragment evacuation was achieved via another temporary transpapillary   
biliary c-SEMS, while mantaining the fistula patent with a second   
transhepatic c-SEMS; and 3) Transhepatic fluoroscopy-guided final stone   
clearance & stent removal. Treatment goal was acomplished. Acute portal vein   
thrombosis developed after EUS-guided H-G, for which 6 month oral   
anticoagulation indicated. Asymptomatic since then. Clinical implications:   
Temporary transmural c-SEMS by EUS afford minimally invasive solution to   
complex benign biliary disease not otherwise amenable to endotherapy.   
Careful risk/benefit evaluation needed for highly selected cases.  
  
RECORD 620  
portal vein thrombosis, revisited  
Primignani M.  
Digestive and Liver Disease (2010) 42:3 (163-170). Date of Publication:   
March 2010  
  
This review article aims to discuss the aetiology, pathophysiology, clinical   
presentation, diagnostic workup and management of portal vein thrombosis,   
either as a primary vascular liver disease in adults and children, or as a   
complication of liver cirrhosis. In addition, indications and limits of   
anticoagulant therapy are discussed in detail. © 2009 Editrice   
Gastroenterologica Italiana S.r.l.  
  
RECORD 621  
portal vein thrombosis  
Seijo-Ríos S. García-Pagán J.C.  
Gastroenterologia y Hepatologia (2010) 33:3 (179-190). Date of Publication:   
March 2010  
  
Thrombosis of the splenoportal axis not associated with liver cirrhosis or   
tumoral disease is the second cause of portal hypertension in the western   
world. In up to 60% of cases, an underlying systemic prothrombotic disorder   
can be identified as an etiological factor. One third of cases are caused by   
local factors and the coexistence of several entities is not unusual.   
Therefore, an etiologic diagnosis is essential in these patients. Early   
anticoagulation therapy in the acute phase of thrombosis of the splenoportal   
axis significantly affects the probability of recanalization and   
consequently the prognosis of these patients. In the chronic phase of   
splenoportal thrombosis (or portal cavernoma), the symptoms are caused by   
the complications of established portal hypertension. To date,   
anticoagulation therapy is limited to patients in whom an underlying   
prothrombotic disorder has been demonstrated. © 2009 Elsevier España, S.L.   
All rights reserved.  
  
RECORD 622  
Degree of portal vein thrombosis  
Qi X. Han G. Jianhong Wang Wu K. Fan D.  
Hepatology (2010) 51:3 (1089-1090). Date of Publication: March 2010  
  
RECORD 623  
Palliative care from the beginning of treatment for advanced pancreatic   
cancer  
Lazenby J.M. Saif M.W.  
Journal of the Pancreas (2010) 11:2 (154-157). Date of Publication: March   
2010  
  
Palliative care ought to be offered at the initiation of treatment for   
people who are diagnosed with pancreatic cancer, given the poor relative   
survival rate and the intractable symptom profile of those who have this   
life-limiting disease. In this article, we argue that palliative treatment   
of people with pancreatic cancer is not found in extending survival, but   
rather, in promoting quality of life. This argument is made by reviewing the   
literature on the state of palliative care in pancreatic cancer and by   
summarizing key studies presented at the "2010 ASCO Gastrointestinal Cancers   
Symposium" held in Orlando, FL, USA on January 22-24, 2010. The studies   
discussed here include: i) a study of a random sample of 564 patients with   
pancreatic cancer that found that the symptom cluster of fatigue and pain   
predicted survival (Abstract #265); ii) a retrospective study of 108   
patients that identified anticoagulation therapy in those who developed   
portal vein thrombosis prolonged survival (Abstract #143); iii) a   
double-blind randomized control trial of 50 patients with gastrointestinal   
cancers who were cachexic in which a thalidomide-olanzapine-megasterol   
acetate combination attenuated the effects of cancer-anorexia-cachexia   
syndrome (Abstract #209); iv) a retrospective study on the role of adjuvant   
chemoradiation and chemotherapy in the treatment of advanced pancreatic   
cancer (Abstract #230); and v) the benefit of chemotherapy in patients with   
metastatic pancreatic cancer 80-year-old or more (Abstract #232). Based on   
the results presented at the meeting, we believe that the discussion of   
palliative care in the treatment of advanced pancreatic cancer must not   
conflate the notion of increased survival with increased quality of life,   
the latter of which is part and parcel of the goal of palliative care. We   
believe that future study on the effect on quality of life of early   
palliative-care interventions among people with pancreatic cancer is   
necessary.  
  
RECORD 624  
Mutation in BCP and precore region of HBV genome was in connection with the   
progression of the chronic hepatitis B  
Zhang X. Zhang D.-K. Han Y. Fan C.-L. Dong P.-L. Zhang B. Zeng C.-Q. Ding   
H.-G.  
Hepatology International (2010) 4:1 (123). Date of Publication: March 2010  
  
Objectives: To study the features and rules of mutation in BCP, precore and   
preS region of HBV genome with different status of liver disease after HBV   
infection, and to analyze the clinical significance of the mutation.   
Methods: Blood samples were retrieved from China northern patients with HBV   
infection. Serum HBV-DNA was acquired from samples and amplified with   
routine PCR. Sequences of the cloning products were got and analyzed.   
Results: 201 patients were enrolled in the study, which included acute   
hepatitis B (7 cases), asyptomatic hepatits B virus carriers (13 cases),   
chronic hepatitis B (89 cases), liver cirrohsis (62 cases) and HBV related   
carcinoma (30 cases). Nucleotide sites such as nt C1726, T1727, G1730, G1752   
and G1799 in BCP and precore region of HBV genome have genotype specificity.   
The result shows that G1776A (OR = 10.7,95% CI:2.4-48.1, P = 0.002), A1846T   
(OR = 3.8,95%CI:1.2-11, 7, P = 0.02), G1896A (OR = 3.4, 95% CI: 1.4-8.3, P =   
0.01) and mutation sites ≤3 (OR = 2.7, 95% CI: 1.1-6.4, P = 0.027) may be   
associated with HBeAg negativity. G1896A mutation may be correlated with   
disease progression (P = 0.004) and be essential in many mutation   
combinations. In mutants bearing more than three substitutions, 53% had   
G1896A (35/66), and only 8% (8/98) in strains with point mutations less than   
3 (P < 0.05). The experiment also hints various substitutions could coexist   
in one strain after long history of HBV infection. Our experiment shows HBV   
preS1 and preS2 region deletion could coexist in the same patient and often   
occur in liver cirrhosis patients. Conclusion: A1727G mutation is a common   
mutation in northern China. A novel G1776A mutation is identified to be   
statistically responsible for HBeAg negativity. G1896A mutation may be the   
risk factor in liver disease progression and prognosis independent of age.   
HBV preS gene deletion may be associated with progression of liver disease.  
  
RECORD 625  
Algorithm for the treatment of portal (PVT) and splancninc veins thrombosis   
(SVT) in patients with liver cirrhosis  
Senzolo M. Sartori M.T. Gasparini D. Boccagni P. Cillo U. Zanus G. Burroughs   
A.K. Burra P.  
Digestive and Liver Disease (2010) 42 SUPPL. 1 (S38). Date of Publication:   
February 2010  
  
Background and Aims: PVT can lead to worsening of liver function, portal   
hypertension and contraindicate liver transplantation in patients with liver   
cirrhosis. There is no established management algorithm for the treatment of   
PVT and SVT in patients with liver cirrhosis. The aim of the study was to   
evaluate prospectically an integrated algorithm for the treatment of this   
condition, integrating the use of radiological treatment when failure of   
anticoagulation occurred. Methods: All patients who were referred to our   
department with PVT or SVT from February 2006 were included in the study.   
Patients with history of portal vein or splancnic vein thrombosis in the   
same period but not included in the protocol were used as control group.   
Screening for prothrombotic disordes and local risk factors was performed in   
all patients. anticoagulation was considered in all and contraindicated when   
high risk of bleeding from varices persist and stabilized presence of   
cavernous transformation of portal vein with recanalization of intrhepatic   
portal vein branches. All patients who were at risk of bleeding from varices   
were banded first. Transjugulat intrahepatic portosystemic shunt (TIPS) was   
indicated when there were concomitant presence of portal hypertensive   
complications or there was progression of thrombosis, or anticoagulation was   
contraindicated. Results: 45 cirrhotics were included in the protocol, 34   
males, mean age 55.5±5, Chils score was distributed as follows: 14 Child C,   
22 Child B, 9 Child A. At the moment of inclusion 20 had total portal vein   
thrombosis (9 cavernoma) and 22 partial PVT; amongst them 2 splenic vein   
thrombosis, 6 SMV thrombosis and 5 total splancninc thrombosis were   
associated; 3 isolated SMV Prothrombotic systemic conditions or local   
factors were identified in 7 patients (15%): 5 prothrombotic genetic   
defects. anticoagulation was initiated in 39 patients, 4 with intraveous   
infusion of LWMH for acute PVT. Twenty patients, 14 males, 9 with complete   
PVT (4 cavernoma) were used as controls. Mean follow-up ±SD was 17±3 months   
for the study group and 20±4 months for the control group. Sixteen over 39   
(38%) who were anticoagulated had ricanalization of the thrombosed vessels,   
compared to none of the controls and the non coagulated patients (p <   
0.001). During the follow-up there were progression of thrombosis in 15/26   
patients non anticoagulated and control group and in 1 amongst   
anticoagulated patients (p < 0.001). Only age of thrombus was correlated   
with occurrence of repermeation. TIPS was indicated in 11 patients, and   
successfully placed in 10 without complications. There were 7 variceal   
bleeding and 4 intestinal venous infarct in the control group, compared to 1   
bleeding episode in the study group (p < 0.001). 2 patients died in the   
control group, compare to 1 in the study group who failed TIPS. Conclusions:   
Integrated algorithm with anticoagulation and TIPS for the treatment of PVT   
with and without splancnic vein thrombosis in cirrhotic and non cirrhotic   
patients is safe and seems useful to prevent thrombosis progression and to   
treat portal hypertensive complications.  
  
RECORD 626  
Anticoagulant treatment for not neoplastic portal vein thrombosis in   
patients with liver cirrhosis and esophageal varices  
Butera G. Simone F. Iacò A. Calvaruso V. Di Marco V. Craxì A.  
Digestive and Liver Disease (2010) 42 SUPPL. 1 (S37). Date of Publication:   
February 2010  
  
Background and Aim: To assess the efficacy and the safety of anticoagulant   
therapy in patients with cirrhosis and esophageal varices with recent not   
malignant portal vein thrombosis. Methods: From May 2007 to April 2009 we   
recorded all cirrhotic patients developing a nonneoplastic portal   
thrombosis. Presence and size of esophageal or gastric varices were assessed   
in all patients by upper gastrointestinal endoscopy (UGE). Anticoagulant   
treatment was initiated and patients followed with US to assess the response   
to therapy. Results: Sixteen patients with cirrhosis and not neoplastic   
portal thrombosis were observed. cirrhosis was due to HCV in 6, HBV in 2,   
ALD in 3 and was cryptogenetic in 5. UGE found F1 varices in 6, F2 in 6 and   
F3 in 4. All patients with large varices were treated prophylactically with   
beta-blockers or variceal band ligation. Anticoagulant drugs used were low   
weight molecular heparin (LWMH) in 7, warfarin in 4 and LMWH later converted   
to warfarin in 5. Extension of thrombus after starting anticoagulation was   
never observed, and a significant reduction in its size was observed in 15   
out of 16. Five patients achieved complete recanalization of the portal   
vessels after a mean treatment of 12 months (range 6-16). No patients   
experienced adverse events linked to anticoagulants and no instances of GI   
bleeding occurred. There were no significant differences in the varices size   
or anticoagulant treatment between patients with partial or complete   
recanalization of portal vein. Conclusions: Patients with cirrhosis, portal   
hypertension and nonneoplastic portal thrombosis may benefit of   
anticoagulants for the treatment of the thrombosis without development of   
SAE. A larger cohort of patients need to be studied to confirm and validate   
these data.  
  
RECORD 627  
The preferable treatment for cirrhotic portal vein thrombosis:   
anticoagulation or transjugular intrahepatic portosystemic shunt?  
Qi X. Han G. Fan D.  
Hepatology (2010) 51:2 (713-714). Date of Publication: February 2010  
  
RECORD 628  
Suspected allergy to warfarin: A management approach based on experience  
Lopez L. Hepner M. Doshi D. Lauter C.  
Journal of Allergy and Clinical Immunology (2010) 125:2 SUPPL. 1 (AB153).   
Date of Publication: February 2010  
  
RATIONALE: There are four types of cutaneous reactions with warfarin:   
purpura, hemorrhagic necrosis, purple toe syndrome, and urticaria.   
Urticarial eruptions are rarest. A previous study noted evidence of   
immunologic involvement. The role of IgE remains uncertain; skin testing is   
unavailable. No desensitization procedure has been published. METHODS: Five   
patients were evaluated. Based on history, examination and positive   
challenge, two patients underwent oral desensitization. Desensitization   
utilized dye-free Coumadin® starting with 0.005mg dose. At 15-minute   
intervals, the dose was advanced to 1mg. Three patients with less well   
documented reactions underwent graded drug challenge with dye-free   
Coumadin®. RESULTS: A 66 year-old female developed pruritic, generalized   
urticaria after starting on warfarin for atrial fibrillation (AF). The   
symptoms partially improved with Medrol®, but not with antihistamines. She   
had persistent hives for over two years with continued use. After warfarin   
was discontinued, her urticaria resolved. Subsequently, she needed AF   
ablation therapy and underwent successful desensitization with Coumadin®.A   
30 year-old female with portal vein thrombosis received warfarin. Within   
hours, she developed pruritic urticaria which resolved with diphenhydramine.   
Warfarin intake was interrupted for INR elevation. Reintroduction resulted   
in diffuse urticaria and lip angioedema. She was successfully desensitized   
with Coumadin®. Three patients underwent graded drug challenge with dye-free   
(10mg) Coumadin®, starting with 0.5mg with a target dose of 5mg. They   
tolerated the challenge and subsequent therapy with the same product.   
CONCLUSIONS: Patients with warfarin allergy who require oral anticoagulation   
may be managed in a supervised graded drug challenge with dyefree Coumadin®   
or desensitization in a controlled setting if drug challenge is positive.  
  
RECORD 629  
portal vein thrombosis  
Parikh S. Shah R. Kapoor P.  
American Journal of Medicine (2010) 123:2 (111-119). Date of Publication:   
February 2010  
  
portal vein thrombosis is a condition not infrequently encountered by   
clinicians. It results from a combination of local and systemic   
prothrombotic risk factors. The presentation of acute thrombosis varies   
widely from an asymptomatic state to presence of life-threatening intestinal   
ischemia and infarction. In the chronic stage, patients typically present   
with variceal bleeding or other complications of portal hypertension.   
Abdominal ultrasound color Doppler imaging has a 98% negative predictive   
value, and is considered the imaging modality of choice in diagnosing portal   
vein thrombosis. Controlled clinical trials to assist with clinical   
decision-making are lacking in both acute and chronic portal vein   
thrombosis. Oral anticoagulant therapy is initiated if the risks of bleeding   
are low, but long-term anticoagulation is generally not recommended in   
patients with concomitant hepatic cirrhosis. The roles of invasive   
therapeutic approaches such as thrombolysis and transjugular intrahepatic   
portosystemic shunt continue to evolve. This review conflates dissenting   
views into a rational approach of managing patients with portal vein   
thrombosis for the general internist. © 2010 Elsevier Inc. All rights   
reserved.  
  
RECORD 630  
Transradial approach for transcatheter selective superior mesenteric artery   
urokinase infusion therapy in patients with acute extensive portal and   
superior mesenteric vein thrombosis  
Wang M.Q. Guo L.P. Lin H.Y. Liu F.Y. Duan F. Wang Z.J.  
CardioVascular and Interventional Radiology (2010) 33:1 (80-89). Date of   
Publication: February 2010  
  
The purpose of this investigation was to assess the feasibility and   
effectiveness of transradial approach for transcatheter superior mesenteric   
artery (SMA) urokinase infusion therapy in patients with acute extensive   
portal and superior mesenteric venous thrombosis. During a period of 7   
years, 16 patients with acute extensive thrombosis of the portal (PV) and   
superior mesenteric veins (SMV) were treated by transcatheter selective SMA   
urokinase infusion therapy by way of the radial artery. The mean age of the   
patients was 39.5 years. Through the radial sheath, a 5F Cobra catheter was   
inserted into the SMA, and continuous infusion of urokinase was performed   
for 5-11 days (7.1 ± 2.5 days). Adequate anticoagulation was given during   
treatment, throughout hospitalization, and after discharge. Technical   
success was achieved in all 16 patients. Substantial clinical improvement   
was seen in these 16 patients after the procedure. Minor complications at   
the radial puncture site were observed in 5 patients, but trans-SMA infusion   
therapy was not interrupted. Follow-up computed tomography scan before   
discharge demonstrated nearly complete disappearance of PV-SMV thrombosis in   
9 patients and partial recanalization of PV-SMV thrombosis in 7 patients.   
The 16 patients were discharged 9-19 days (12 ± 6.0 days) after admission.   
Mean duration of follow-up after hospital discharge was 44 ± 18.5 months,   
and no recurrent episodes of PV-SMV thrombosis developed during that time   
period. Transradial approach for transcatheter selective SMA urokinase   
infusion therapy in addition to anticoagulation is a safe and effective   
therapy for the management of patients with acute extensive PV-SMV   
thrombosis. © 2009 Springer Science+Business Media, LLC and the   
Cardiovascular and Interventional Radiological Society of Europe (CIRSE).  
  
RECORD 631  
Systematic review: portal vein thrombosis in cirrhosis  
Tsochatzis E.A. Senzolo M. Germani G. Gatt A. Burroughs A.K.  
Alimentary Pharmacology and Therapeutics (2010) 31:3 (366-374). Date of   
Publication: February 2010  
  
Aliment Pharmacol Ther 31, 366-374 SummaryBackground As current imaging   
techniques in cirrhosis allow detection of asymptomatic portal vein   
thrombosis during routine ultrasonography, more patients with cirrhosis are   
diagnosed with portal vein thrombosis. Although a consensus on noncirrhotic   
extra-hepatic portal vein thrombosis has been published, no such consensus   
exists for portal vein thrombosis with cirrhosis. Aim To perform a   
systematic review of nonmalignant portal vein thrombosis in cirrhosis in   
terms of prevalence, pathogenesis, diagnosis, clinical course and   
management. Methods Studies were identified by a search strategy using   
MEDLINE and EMBASE. Results portal vein thrombosis is encountered in 10-25%   
of cirrhotics. In terms of pathophysiology, cirrhosis is no longer   
considered a hypocoagulable state; rather than a bleeding risk in cirrhosis,   
various clinical studies support a thrombotic potential. Clinical findings   
of portal vein thrombosis in cirrhosis vary from asymptomatic disease to a   
life-threatening condition at first presentation. Optimal management of   
portal vein thrombosis in cirrhosis is currently not addressed in any   
consensus publication. Treatment strategies most often include the use of   
anticoagulation, while thrombectomy and transjugular intrahepatic   
portosystemic shunts are considered second-line options. Conclusions Portal   
vein thrombosis in cirrhosis has many unresolved issues, which are often the   
critical problems clinicians encounter in their everyday practice. We   
propose a possible research agenda to address these unresolved issues. ©   
2010 Blackwell Publishing Ltd.  
  
RECORD 632  
Venous Thromboembolism in Children  
Goldenberg N.A. Bernard T.J.  
Hematology/Oncology Clinics of North America (2010) 24:1 (151-166). Date of   
Publication: February 2010  
  
With improved pediatric survival from serious underlying illnesses, greater   
use of invasive vascular procedures and devices, and a growing awareness   
that vascular events occur among the young, venous thromboembolism (VTE)   
increasingly is recognized as a critical pediatric concern. This review   
provides background on etiology and epidemiology in this disorder, followed   
by an indepth discussion of approaches to the clinical characterization,   
diagnostic evaluation, and management of pediatric VTE. Prognostic   
indicators and long-term outcomes are considered, with emphasis on available   
evidence underlying current knowledge and key questions for further   
investigation. © 2010 Elsevier Inc. All rights reserved.  
  
RECORD 633  
portal vein thrombosis (PVT) after splenectomy in a liver transplant (LT)   
patient  
Abbass A.A. Abouljoud M. Getzen T. Yoshida A. Hundley J. Kazimi M. Slater R.   
Patil V. Kim D.Y.  
American Journal of Transplantation (2010) 10 SUPPL. 1 (66). Date of   
Publication: January 2010  
  
BACKGROUND: portal vein thrombosis (PVT) is a complication following   
splenectomy, but not reported post liver transplantation (LT). We describe a   
case of acute post-splenectomy PVT in a LT recipient which was successfully   
treated with pharmacomechanical thrombolysis using the Trellis device. CASE   
REPORT: A 54 year old LT recipient presented two years later with   
symptomatic splenomegaly and underwent an uncomplicated splenectomy. He   
developed complete PVT within 24 hours postoperatively, sparing the superior   
mesenteric vein. anticoagulation was initiated. Complete thrombosis of the   
intrahepatic portal vein branches precluded percutaneous cannulation of the   
portal system. Hence the portal vein (PV) was cannulated intraoperatively   
through a jejunal branch. Tissue Plasminogen Activator (t-PA) was injected   
and partial recanalization of the main PV was achieved which was   
subsequently accessed trans-hepatically via right internal jugular vein   
puncture. A Trellis 8 (Bacchus Vascular, Santa Clara, CA) thrombolysis   
device was used and resulted in significant improvement of PV flow. This was   
followed by 24 hour continuous direct infusion of tPA and systemic   
anticoagulation. Repeat imaging studies demonstrated re-canalization of the   
PV system. CONCLUSIONS: PVT after splenectomy has been reported in the   
literature, but not in the setting of LT. Using the Trellis thrombectomy   
device may be safe and effective in restoring portal vein patency.  
  
RECORD 634  
Acute portal vein thrombosis unrelated to cirrhosis: A prospective   
multicenter follow-up study  
Plessier A. Darwish-Murad S. Hernandez-Guerra M. Consigny Y. Fabris F.   
Trebicka J. Heller J. Morard I. Lasser L. Langlet P. Denninger M.-H. Vidaud   
D. Condat B. Hadengue A. Primignani M. Garcia-Pagan J.-C. Janssen H.L.A.   
Valla D.  
Hepatology (2010) 51:1 (210-218). Date of Publication: January 2010  
  
Current recommendations for early anticoagulation in acute portal vein   
thrombosis unrelated to cirrhosis or malignancy are based on limited   
evidence. The aim of this study was to prospectively assess the risk   
factors, outcome, and prognosis in patients managed according to these   
recommendations. We enrolled 102 patients with acute thrombosis of the   
portal vein, or its left or right branch. Laboratory investigations for   
prothrombotic factors were centralized. Thrombus extension and   
recanalization were assessed by expert radiologists. A local risk factor was   
identified in 21% of patients, and one or several general prothrombotic   
conditions in 52%. anticoagulation was given to 95 patients. After a median   
of 234 days, the portal vein and its left or right branch were patent in 39%   
of anticoagulated patients (versus 13% initially), the splenic vein in 80%   
(versus 57% initially), and the superior mesenteric vein in 73% (versus 42%   
initially). Failure to recanalize the portal vein was independently related   
to the presence of ascites (hazard ratio 3.8, 95% confidence interval   
1.3-11.1) and an occluded splenic vein (hazard ratio 3.5, 95% confidence   
interval 1.4-8.9). Gastrointestinal bleeding and intestinal infarction   
occurred in nine and two patients, respectively. Two patients died from   
causes unrelated to thrombosis or anticoagulation therapy. Conclusion:   
Recanalization occurs in one-third of patients receiving early   
anticoagulation for acute portal vein thrombosis, whereas thrombus   
extension, intestinal infarction, severe bleeding, and death are rare.   
Alternative therapy should be considered when ascites and splenic vein   
obstruction are present. Copyright © 2009 by the American Association for   
the Study of Liver Diseases.  
  
RECORD 635  
Protein C and D-dimer are related to portal vein thrombosis in patients with   
liver cirrhosis  
Zhang D. Hao J. Yang N.  
Journal of Gastroenterology and Hepatology (Australia) (2010) 25:1   
(116-121). Date of Publication: January 2010  
  
Background and Aim: To profile changes of coagulation, anticoagulation and   
fibrolytic factors associated with liver function failure and portal vein   
thrombosis (PVT) formation in chronic liver cirrhosis patients. Methods: A   
total of 116 cirrhotic patients admitted to our hospital from June 2006 to   
October 2008 were included in our study. All patients were classified into   
two groups: PVT group (31 patients), composed of patients with PVT and a   
control group (85 patients), including patients without PVT. Platelet,   
prothrombin time (PT), activated partial prothrombin time (APTT) and   
fibrinogen were measured. Also, plasma samples from the patients were   
analyzed for the levels of antithrombin III (AT-III), protein C (PC),   
protein S (PS), D-dimer, tissue-type plasminogen activator as well as   
plasminogen activator inhibitor-1. Statistical analyses were carried out to   
evaluate the correlation of specific variations with the disease status.   
Results: In general, the higher Child-Pugh scores, indicating the   
aggravation of hepatic impairment of the patients, correlated well with the   
prolonged PT/APTT and increased D-dimer, as well as decreased platelet,   
fibrinogen, PC and AT-III levels in the serum. Furthermore, we found that   
the PC, PS and D-dimer levels in PVT patients were 2.32 ± 0.72 mg/L, 17.14 ±   
3.62 mg/L and 0.99 ± 0.36 mg/L, respectively, both representing a   
significant difference compared with those in the control group without PVT.   
Logistic regression model shows that the odds ratio value of one unit of   
increase of PC and D-dimer were 0.48 and 15.57. Conclusions: Cirrhotic   
patients displayed dysfunctions in the coagulation, anti-coagulation and   
fibrolytic systems. The development of PVT in these patients may be   
independently associated with the decrease of PC, PS and D-dimer.   
Furthermore, decreasing PC and increasing D-dimer may be risk factors   
inducing PVT in cirrhotic patients. © 2009 Journal of Gastroenterology and   
Hepatology Foundation and Blackwell Publishing Asia Pty Ltd.  
  
RECORD 636  
Rare thromboses of cerebral, splanchnic and upper-extremity veins: A   
narrative review  
Martinelli I. De Stefano V.  
Thrombosis and Haemostasis (2010) 103:6 (1136-1144). Date of Publication:   
June 2010  
  
Venous thrombosis typically involves the lower extremity circulation.   
Rarely, it can occur in the cerebral or splanchnic veins and these are the   
most frightening manifestations because of their high mortality rate. A   
third site of rare venous thrombosis is the deep system of the upper   
extremities that, as for the lower extremity, can be complicated by   
pulmonary embolism and post-thrombotic syndrome. The authors conducted a   
narrative review focused on clinical manifestations, risk factors, and   
treatment of rare venous thromboses. Local risk factors such as infections   
or cancer are frequent in thrombosis of cerebral or portal veins. Upper   
extremity deep-vein thrombosis is mostly due to local risk factors   
(catheter- or effort-related). Common systemic risk factors for rare venous   
thromboses are inherited thrombophilia and oral contraceptive use; chronic   
myeloproliferative neoplasms are closely associated with splanchnic vein   
thrombosis. In the acute phase rare venous thromboses should be treated   
conventionally with low-molecular-weight heparin. Use of local or systemic   
fibrinolysis should be considered in the case of clinical deterioration in   
spite of adequate anticoagulation. anticoagulation with vitamin   
K-antagonists is recommended for 3-6 months after a first episode of rare   
venous thrombosis. Indefinite anticoagulation is recommended for Budd-Chiari   
syndrome, recurrent thrombosis or unprovoked thrombosis and permanent risk   
factors. In conclusion, the progresses made in the last couple of decades in   
diagnostic imaging and the broadened knowledge of thrombophilic   
abnormalities improved the recognition of rare venous thromboses and the   
understanding of pathogenic mechanisms. However, the recommendations for   
treatment mainly derive from observational studies. © Schattauer 2010.  
  
RECORD 637  
Esophageal Varices in Chronic Intestinal Insufficiency in Absence of Portal   
Hypertension or liver cirrhosis: Case Report  
Yandza T. Schneider S.M. Novellas S. Badan L. Saint-Paul M.C. Bounin P.A.   
Rahili A. Zeanandin G. Benchimol D. Gugenheim J. Hébuterne X.  
Transplantation Proceedings (2010) 42:1 (103-105). Date of Publication:   
January 2010/February 2010  
  
We report the case of a 62-year-old man with short-bowel syndrome, referred   
for intestinal transplantation, who had esophageal varices (EV) due to   
superior vena cava (SVC) thrombosis. Pretransplantation work-up revealed   
protein S deficiency. Results of liver function tests were normal. Upper   
endoscopy showed grade II to III EV in the upper and middle segments of the   
esophagus. Computed tomography demonstrated thrombosis of the jugular,   
subclavian, and SVC veins and marked collateral vessels in the chest.   
Transient elastography yielded normal findings. A liver biopsy specimen   
showed a normal aspect of the liver, without fibrosis or liver cirrhosis.   
Presence of EV in a patient with chronic intestinal insufficiency may be   
related to collateral venous circulation associated with SVC thrombosis in   
the absence of portal hypertension. In this situation, an isolated   
intestinal graft is indicated. © 2010 Elsevier Inc. All rights reserved.  
  
RECORD 638  
Portomesenteric venous thrombosis associated with rectal venous   
malformations  
Kulungowski A.M. Fox V.L. Burrows P.E. Alomari A.I. Fishman S.J.  
Journal of Pediatric Surgery (2010) 45:6 (1221-1227). Date of Publication:   
June 2010  
  
Purpose: We report thrombosis of portal and mesenteric veins in patients   
with a pattern of rectal venous malformations (VMs) and ectatic major   
mesenteric veins. Methods: Eight patients having rectal VMs with either   
ectatic mesenteric veins and/or evidence of portomesenteric venous   
thrombosis (PVT), evaluated from 1995-2009, were reviewed. Results:   
Portomesenteric venous thrombosis was evident in 5 patients at presentation.   
Three had patent ectatic mesenteric veins, 2 with demonstrated reversal of   
flow, and 2 of whom went on to thrombosis during observation. Six patients   
developed portal hypertension. Five remain on long-term anticoagulation.   
After recognizing this pattern, one patient underwent preemptive proximal   
ligation of the inferior mesenteric vein (IMV) to enhance antegrade portal   
vein flow and prevent propagation or embolization of venous thrombus from   
the IMV to the portal vein. Conclusion: Rectal VMs should be evaluated for   
associated ectatic mesenteric veins. The ectatic vein siphons flow from the   
portal vein down to the rectal VM, leading to stagnation of blood in the   
portal vein and resultant thrombosis. Primary thrombosis in the stagnant   
rectal VM and/or mesenteric vein can also predispose to embolization up into   
the portal vein. This pattern of rectal VM and ectatic mesenteric vein   
should be considered a risk factor for devastating PVT. © 2010 Elsevier Inc.   
All rights reserved.  
  
RECORD 639  
Safety and efficacy of anticoagulation therapy with low molecular weight   
heparin for portal vein thrombosis in patients with liver cirrhosis  
Amitrano L. Guardascione M.A. Menchise A. Martino R. Scaglione M. Giovine S.   
Romano L. Balzano A.  
Journal of Clinical Gastroenterology (2010) 44:6 (448-451). Date of   
Publication: July 2010  
  
Background: Treatment of portal vein thrombosis (PVT) in patients with liver   
cirrhosis is not well established. AIM: We intended to assess the safety and   
efficacy of low molecular weight heparin (LMWH) to treat PVT in cirrhotic   
patients. Study: All 39 patients diagnosed with non-neoplastic PVT and   
cirrhosis from June 2005 to December 2006 were evaluated for anticoagulation   
therapy (AT). PVT was occludent in 15.4%, partial in 64.1%, and portal   
cavernoma presented in 20.5%. Twenty-eight patients received 200 U/kg/d of   
enoxaparin for at least 6 months. In 39.3% of patients PVT was an occasional   
finding, in 10.7% presented with acute abdominal pain, in 50% with bleeding   
from gastroesophageal varices. In this last group LMWH was started after   
endoscopic eradication of varices by band ligation. Results: Complete   
recanalization of portal vein occurred in 33.3%, partial recanalization in   
50% and no response in 16.7% of patients. Further 12 patients who continued   
AT obtained complete recanalization at a median time of 11 months (range 7   
to 17 mo). Overall, a complete response was obtained in 75% of patients. No   
significant side effects, particularly bleeding complications, were observed   
during the treatment. Conclusions: LMWH demonstrated safe and effective in   
the treatment of PVT in patients with liver cirrhosis. Copyright © 2010 by   
Lippincott Williams & Wilkins.  
  
RECORD 640  
Impact of antithrombin III concentrates on portal vein thrombosis after   
splenectomy in patients with liver cirrhosis and hypersplenism  
Kawanaka H. Akahoshi T. Kinjo N. Konishi K. Yoshida D. Anegawa G. Yamaguchi   
S. Uehara H. Hashimoto N. Tsutsumi N. Tomikawa M. Maehara Y.  
Annals of Surgery (2010) 251:1 (76-83). Date of Publication: January 2010  
  
OBJECTIVE: The aim of this study was to determine the role of antithrombin   
III (AT-III) in portal vein thrombosis (PVT) after splenectomy in cirrhotic   
patients. SUMMARY BACKGROUND DATA: There is no standard treatment for PVT   
after splenectomy in liver cirrhosis. METHODS: A total of 50 consecutive   
cirrhotic patients who underwent laparoscopic splenectomy for hypersplenism   
were enrolled into this study. From January 2005 to December 2005, 25   
cirrhotic patients received no prophylactic anticoagulation therapy after   
the operation (AT-III group). From January 2006 to July 2006, 25 cirrhotic   
patients received prophylactic administration of AT-III concentrates (1500   
U/d) on postoperative day (POD) 1, 2, and 3 (AT-III [+] group). RESULTS: In   
AT-III (-) group, 9 (36.0%) patients developed PVT up to POD 7, and risk   
factors for PVT were identified as: low platelet counts, low AT-III   
activity, and increased spleen weight. Although there were no significant   
differences in the clinical characteristics, including the above risk   
factors, between the 2 groups, only 1 (4.0%) patient developed PVT on POD 30   
in AT-III (+) group, and the incidence of PVT was significantly lower than   
in AT-III (-) group (P = 0.01). In AT-III (-) group, AT-III activity was   
significantly decreased from POD 1 to POD 7, as compared with the   
preoperative level, whereas AT-III concentrates prevented the postoperative   
decrease in AT-III activity. CONCLUSIONS: These results demonstrate that low   
AT-III activity and further decreases in this activity are associated with   
PVT after splenectomy in cirrhotic patients, and that treatment with AT-III   
concentrates is likely to prevent the development of PVT in these patients.   
Copyright © 2009 by Lippincott Williams & Wilkins.  
  
RECORD 641  
Recommended timing for surveillance ultrasonography to diagnose portal   
splenic vein thrombosis after laparoscopic splenectomy  
Tran T. Demyttenaere S.V. Polyhronopoulos G. Séguin C. Artho G.P. Kaneva P.   
Fried G.M. Feldman L.S.  
Surgical Endoscopy and Other Interventional Techniques (2010) 24:7   
(1670-1678). Date of Publication: July 2010  
  
Background Symptomatic portal or splenic vein thrombosis (PSVT) is a rare   
but potentially lethal complication of laparoscopic splenectomy (LS). While   
routine postoperative duplex ultrasound surveillance can be used for early   
detection, the optimal timing is unknown. The aim of this study is to   
investigate the incidence and progression of asymptomatic PSVT 1 week and 1   
month after LS. Methods Consecutive patients scheduled for LS for   
hematologic disease participated in this study. Patients underwent   
surveillance for PSVT using duplex ultrasonography 1 week and 1 month   
postoperatively. Results 43 of 48 patients planning to undergo LS in the   
study period were enrolled, with 3 subsequently excluded, leaving 40 for   
further analysis. The indications for LS were benign disease in 31 [19 had   
immune thrombocytopenia purpura (ITP)] and malignant disease in 9. A   
hand-assisted technique was used in 12 cases. PSVT was diagnosed in 9/40   
patients (22.5%). Seven (77.8%) were diagnosed by 1 week with ultrasound, of   
whom one had mild symptoms (fever and diarrhea). After anticoagulation,   
subsequent ultrasounds showed resolution or improvement in all seven   
patients. Thirty-three patients had a normal ultrasound result at 1 week.   
One of these patients also had a computed tomography (CT) scan that found a   
PSVT not seen on ultrasound. Twenty-seven patients returned for follow-up   
after normal 1-week imaging: 26 patients had an ultrasound at 1 month, with   
no new PSVT found. One additional patient did not return for subsequent   
ultrasound until 2 months later, when a new distal SVT was found; ultrasound   
at 6 months showed complete resolution without treatment. Conclusion The   
1-week incidence of PSVT after LS was 8/40 (20%). The high incidence   
justifies ultrasonographic screening on postoperative day 7. If asymptomatic   
PSVT has not developed at this time, it is unlikely to develop by 1 month,   
and subsequent screening ultrasound at 1 month is not required. © Springer   
Science+Business Media, LLC 2009.  
  
RECORD 642  
Outcome of patients with primary hepatic venous obstruction treated with   
anticoagulants alone.  
Shukla A. Bhatia S.J.  
Indian journal of gastroenterology : official journal of the Indian Society   
of Gastroenterology (2010) 29:1 (8-11). Date of Publication: Jan 2010  
  
BACKGROUND: Outcome of patients with hepatic venous outflow tract   
obstruction (HVOTO) has improved with newer treatments, including   
anticoagulants, radiological interventions and liver transplant. In India,   
however, liver transplant and radiological interventions are costly and have   
limited availability. Hence, patients often opt for anticoagulation alone.   
We followed up a group of such patients to determine the clinical outcome   
with such treatment. METHODS: Consecutive patients with HVOTO, treated with   
oral anticoagulation and supportive medical therapy but no radiological or   
surgical intervention, were followed up for at least 12 months. Diagnosis of   
HVOTO was based on color Doppler, and either angiography or magnetic   
resonance venography. Warfarin dose was adjusted to maintain international   
normalized ratio (INR) between 2.0 and 3.0. Patients with secondary HVOTO   
and those with baseline INR > or = 2.0 were excluded. Response was defined   
as absence of ascites and/or encephalopathy, normal AST/ALT, bilirubin <1.5   
mg/dL, and no portal hypertension related bleed after starting therapy.   
RESULTS: Of 43 patients (mean [SD] age=28.7 [8.4] years; 20 men), 26 (61%)   
had a response during a median follow up of 23 (range 15-33) months. The   
response first appeared within 2 months of the start of treatment in 18   
patients and between 2 and 5 months from the start of treatment in eight   
patients. Seven patients died of progressive liver failure (6 patients) or   
GI bleed (1 patient). Nine patients had anticoagulation-related   
complications. On univariate analysis, short duration of symptoms, high   
serum albumin, low baseline INR, and low baseline Child-Pugh's (CP) or   
Clichy scores predicted response. Presence of hepatic encephalopathy, portal   
vein thrombosis, obstruction of all hepatic veins, low albumin, high INR,   
high serum bilirubin, high baseline CP score, Murad score and adverse Clichy   
index were associated with higher mortality rate. However, on multivariate   
analysis, only low CP score was associated with response, and no factor was   
found to predict death. CONCLUSIONS: More than half of patients with HVOTO   
show response with only supportive medical therapy and anticoagulants. This   
occurs more often in patients with low CP score. Some patients may have   
delayed response.  
  
RECORD 643  
Portal, splenic and mesenteric vein thrombosis in a patient double   
heterozygous for factor v Leiden and prothrombin G20210A mutation  
Grouzi E. Politou M. Douramani P. Merkouri E. Gialeraki A. Brountzos H.   
Perros G. Travlou A.  
Blood Coagulation and Fibrinolysis (2009) 20:8 (722-725). Date of   
Publication: December 2009  
  
We herein report a 56-year-old man who presented with abdominal pain,   
diarrhea and a 22-kg-weight loss over 4 months. He was on acenocoumarol   
treatment because of portal, splenic and mesenteric vein thrombosis (PSMVT)   
3 months before, with admission international normalized ratio (INR):1.6.   
Doppler ultrasonography and helical computerized tomographic scan of the   
abdomen showed complete thrombosis of the extrahepatic portal vein extending   
into the superior mesenteric vein and splenic vein. The manifestation of   
thrombosis was in the absence of provocative stimuli or local cause. The   
patient had a negative history of venous thromboembolism. Thrombophilia   
workup revealed double heterozygosity for factor V Leiden and prothrombin   
G20210A mutation. He was immediately started with intravenous unfractionated   
heparin, followed by oral anticoagulation with target INR 2-3. Five days   
after a Doppler examination showed significant improvement in the flow   
within the portal vein, and a computerized tomographic scan of the abdomen 1   
month later showed extensive recanalization of the portal venous system. The   
patient is now 36 months out from the second PSMVT episode and is doing well   
although maintaining oral lifelong anticoagulation. The case is of   
particular interest in that PSMVT was the first manifestation of this   
combined disorder. We conclude that all patients presenting with unexplained   
PSMVT should be investigated for the presence of a hypercoagulable state.   
anticoagulation should be considered in all patients with this diagnosis and   
should be a lifelong therapy in those with an underlying thrombophilia. ©   
2009 Wolters Kluwer Health | Lippincott Williams & Wilkins.  
  
RECORD 644  
Multiple liver abscesses due to portal vein thrombophlebitis  
Kajzrliková I. Vítek P. Chalupa J. Talafa V. Bolek K. Chrostek M.  
Ceska a Slovenska Gastroenterologie a Hepatologie (2009) 63:6 (259-264).   
Date of Publication: 2009  
  
Introduction. Extrahepatal portal vein obstruction occurs mainly as a   
complication of intraabdominal infection, malignancy and hypercoagulable   
syndromes. Pyogenic thrombophlebitis with subsequent thrombus embolisation   
into intrahepatic branches of the portal vein may cause multiple liver   
abscesses. Case report. A 67-year-old man was admitted for clinical   
presentation of acute abdomen and the diagnosis of portal vein thrombosis   
and multiple liver abscesses was made. The patient was treated succesfully   
in the intensive care unit with anticoagulants and antibiotics together with   
CT guided drainage. Surgery was avoided. This therapy resulted in complete   
regression of liver abscesses. Discussion. We have found a sigmoid   
diverticulosis on the abdominal CT in our patient, and we pressume possible   
occult diverticulitis with pylephlebitis as the aetiologic factor. The   
malignancy or hypercoagulable state were excluded. Pylephlebitis is an   
ascendent septic portal vein infection in the course of the abdominal   
sepsis. The subsequent portal vein thrombosis is rare but it is accompanied   
with high morbidity and mortality. The treatment modality is the drainage of   
liver abscesses with antibiotics together with anticoagulants. The benefit   
of anticoagulation therapy outweighs the risk of possible bleeding from   
esophageal or gastric varices. Conclusions. This serious condition   
demonstrated as an acute abdomen was successfully treated conservatively due   
to early use of proper imaging methods and appropriate therapy.  
  
RECORD 645  
Idiopathic suppurative pylephlebitis: Interventional radiological diagnosis   
and management  
Bogue C.O. Leahy T.R. Rea D.J. Bitnun A. Brandao L.R. Kahr W.H.A. Jacobson   
S. Amaral J.G. Connolly B.L.  
CardioVascular and Interventional Radiology (2009) 32:6 (1304-1307). Date of   
Publication: November 2009  
  
We report the imaging findings and management of a case of suppurative   
pylephlebitis of unknown cause in a 10-year-old girl. Percutaneous   
aspiration of frank pus from the portal vein confirmed the diagnosis and   
contributed to therapy. Percutaneous transhepatic thrombolysis was attempted   
but was unsuccessful. Because of the nonspecific presentation of this   
condition and the lack of familiarity of physicians with this entity, the   
diagnosis is often delayed. Our aim is to increase the awareness of this   
entity and stress the importance of early diagnosis and appropriate therapy.   
© 2009 Springer Science+Business Media, LLC and the Cardiovascular and   
Interventional Radiological Society of Europe (CIRSE).  
  
RECORD 646  
Review article: The modern management of portal vein thrombosis  
Chawla Y. Duseja A. Dhiman R.K.  
Alimentary Pharmacology and Therapeutics (2009) 30:9 (881-894). Date of   
Publication: November 2009  
  
portal vein thrombosis (PVT) is an important cause of portal hypertension.   
It may occur as such with or without associated cirrhosis and hepatocellular   
carcinoma. Information on its management is scanty. Aim To provide an update   
on the modern management of portal vein thrombosis. Information on portal   
vein thrombosis in patients with and without cirrhosis and hepatocellular   
carcinoma is also updated. Methods A pubmed search was performed to identify   
the literature using search items portal vein thrombosis-aetiology and   
treatment and portal vein thrombosis in cirrhosis and hepatocellular   
carcinoma. Results portal vein thrombosis occurs because of local   
inflammatory conditions in the abdomen and prothrombotic factors. Acute   
portal vein thrombosis is usually symptomatic when associated with cirrhosis   
and/or superior mesenteric vein thrombosis. anticoagulation should be given   
for 3-6 months if detected early. If prothrombotic factors are identified,   
anticoagulation should be given lifelong. Chronic portal vein thrombosis   
usually presents with well tolerated upper gastrointestinal bleed. It is   
diagnosed by imaging, which demonstrates a portal cavernoma in place of a   
portal vein. anticoagulation does not have a definite role, but bleeds can   
be treated with endotherapy or shunt surgery. Rarely liver transplantation   
may be considered. Conclusion Role of anticoagulation in chronic portal vein   
thrombosis needs to be further studied. © 2009 Blackwell Publishing Ltd.  
  
RECORD 647  
Administration of Dalteparin Based on the Activated Clotting Time for   
Prophylaxis of Hepatic Vessel Thrombosis in Living Donor Liver   
Transplantation  
Uchikawa Y. Ikegami T. Masuda Y. Ohno Y. Mita A. Urata K. Nakazawa Y. Terada   
M. Miyagawa S.  
Transplantation Proceedings (2009) 41:9 (3784-3790). Date of Publication:   
November 2009  
  
Beginning in 2004, dalteparin doses based on activated clotting time (ACT)   
were administered for hepatic vessel thrombosis prophylaxis in living donor   
liver transplantation (LDLT). We verified the feasibility of this new   
therapy by comparing it with the previous one. From 1993 through 2008, 42   
metabolic liver patients who underwent LDLT were divided into two groups.   
Group A (1993-2003, n = 32) was administered a fixed dalteparin dose and a   
large amount of fresh frozen plasma (FFP); Group B (2004-2008, n = 10) was   
administered an appropriate dosage of dalteparin to maintain the ACT levels   
from 140 to 150 seconds and a small amount of FFP. Group B was administered   
a lesser amount of FFP and more dalteparin. This resulted in longer   
activated partial thromboplastin time, lower fibrinogen degradation products   
D-dimer, and lower aspartate aminotransferase levels compared to group A;   
all differences were significant. Group B showed neither thrombotic nor   
hemorrhagic complications. anticoagulation therapy comprising adjustment of   
the dalteparin dose based on ACT reduces thrombotic complications without   
increasing hemorrhagic complications. ACT measurement is a simple, reliable   
method for bedside monitoring of dalteparin anticoagulant effects for LDLT.   
© 2009 Elsevier Inc. All rights reserved.  
  
RECORD 648  
Modified technique of meso-Rex shunt in case of insufficient length of the   
jugular vein graft  
Chardot C. Darani A. Dubois R. Mure P.-Y. Pracros J.-P. Lachaux A.  
Journal of Pediatric Surgery (2009) 44:11 (e9-e12). Date of Publication:   
November 2009  
  
Meso-Rex shunt (MRS) can relieve portal hypertension and restore a   
physiological portal flow in patients with portal vein thrombosis. We   
describe a technical variant where the autologous internal jugular vein   
(IJV) was too short to bridge the superior mesenteric vein (SMV) and the Rex   
recessus. Patient: A 15-year-old boy with portal cavernoma had several   
episodes of gastrointestinal bleeding despite repeated sclerotherapy.   
Preoperative assessment, including retrograde transjugular portography,   
showed persistent esophageal and gastric varices, severe hypertensive   
gastropathy, obstructed portal vein, patent SMV and splenomesenteric   
confluence, patent intrahepatic portal branches, and normal transhepatic   
pressure gradient. An MRS was planned. The left IJV was retrieved from its   
infracranial part to its confluence with subclavian vein. After performing   
the Rex recessus to IJV graft anastomosis, the IJV graft proved to be too   
short for classical end-to-side anastomosis onto the SMV. After clamp   
testing showing good tolerance of the small bowel, the proximal jejunal   
branches of the SMV were tied, the proximal SMV was mobilized and   
transsected 4 cm below the pancreas, and an end-to-end anastomosis between   
SMV and IJV was performed. Portal pressure decreased from 23 to 13 mm Hg,   
and intraoperative Ultra Sound Doppler (US Doppler) showed good flows in the   
shunt. Postoperative course was uneventful, and 1 year after surgery, the   
child is clinically well, off medication, with a patent shunt, and no portal   
hypertension. Conclusion: This modified MRS technique may be useful when the   
autologous IJV graft is too short, avoiding the need for prosthetic conduits   
and prolonged postoperative anticoagulation. © 2009 Elsevier Inc. All rights   
reserved.  
  
RECORD 649  
Adult to pediatric living donor liver transplantation for portal cavernoma  
Zhang M. Guo C. Pu C. Ren Z. Li Y. Kang Q. Jin X. Yan L.  
Hepatology Research (2009) 39:9 (888-897). Date of Publication: 2009  
  
Aim: Portal cavernoma (PC) is an important cause of non-cirrhotic portal   
hypertension with severe complications, such as variceal hemorrhage in   
pediatric patients. With the development of new surgical techniques, living   
donor liver transplantation (LDLT) has recently been recognized as a viable   
but challenging treatment option for PC. The purpose of the present study   
was to summarize the efficacy of LDLT in PC patients and to carry out a   
follow-up study of pediatric recipients. Methods: The primary indication for   
LDLT in our research was PC with severe variceal bleeding and liver function   
decompensation. Three patients were diagnosed with PC following evaluation   
with computed tomography angiography and abdominal color Doppler   
ultrasonography (CDU). Results: Various surgical techniques, including jump   
bypass grafting for portal vein anastomosis, were carried out according to   
the range and degree of cavernous transformation within the splenic vein and   
superior mesenteric vein. Postoperative CDU confirmed the early integrity of   
the portal vein (PV) in each patient. PV rethrombosis occurred in one   
patient 7days after LDLT, despite anticoagulation therapy with coumadin. Two   
of the three patients had no further episodes of variceal hemorrhage during   
the 2-year follow-up period. Conclusions: The present study is the first   
report of the successful use of LDLT to treat pediatric PC patients. We   
conclude that LDLT is effective for the majority of pediatric patients with   
PC. © 2009 The Japan Society of Hepatology.  
  
RECORD 650  
Late acute celiac and hepatic artery thrombosis with portal vein thrombosis   
resulting in hepatic infarction 12 years post orthotopic liver   
transplantation  
Haque M. Schumacher P.A. Harris A. Scudamore C.H. Steinbrecher U.P. Chung   
S.W. Buczkowski A.K. Erb S.R. Yoshida E.M.  
Annals of Hepatology (2009) 8:4 (396-399). Date of Publication:   
October-December 2009  
  
Hepatic artery thrombosis (HAT) is relatively infrequent, but possibly a   
devastating complication of orthotopic liver transplantation (OLT). It often   
requires urgent retransplantation. Two main forms of HAT are recognized as   
early and late HAT (diagnosis within or after 30 days following LT). Early   
HAT typically results in graft failure. Late HAT features biliary   
obstruction, cholangitis, and hepatic abscess formation. We report here the   
case of a patient of Wilson's disease who presented twelve years post-liver   
transplant symptoms typical of acute HAT and hepatic infarction. On   
diagnostic imaging, celiac axis and hepatic artery were thrombosed,   
resulting in ischemic necrosis of the left hepatic lobe. The resulting   
sepsis and transient hepatic insufficiency were managed conservatively, and   
repeat OLT was avoided. The patient remains stable more than one year later.   
To the best of our knowledge this case report is unique in the literature   
for the unusually long interval between OLT and late acute HAT, as well as   
celiac and portal vein occlusion. The acute presentation of sub massive   
hepatic necrosis is also uncharacteristic of late HAT and more typical of   
acute HAT. This report describes our experience in managing this and a   
literature review of the topic.  
  
RECORD 651  
Clinical study of portal thrombosis with acute biliary infections  
Kamata I. Igarashi Y. Hara S. Takuma K. Kishimoto Y. Suzuki T. Mimura T. Ito   
K. Okano N. Miura T. Sumino Y.  
Journal of Gastroenterology and Hepatology (2009) 24 SUPPL. 1 (A152). Date   
of Publication: October 2009  
  
Introduction: Portal thrombosis is a rare disease, but early treatments are   
important. When the thrombus obstructs the main portal vein rapidly, it   
causes liver failures. We studied the patients with portal thrombosis of   
acute biliary infection in our hospital. Materials and Methods: We studied   
four cases, three cases were cho-ledocholithiasis and one was   
pancreaticobiliary maljunction. All cases were immediately treated   
transpapillary or percutaneously, and used anticoagulant drugs. Results:   
Four cases were recognized moderate or severe acute cholangi-tis, and   
moderate acute cholecystitis. Thrombus was recognized at right branch of   
portal vein in four cases. In one case, thrombus was recognized at bilateral   
branch. After anticoagulation therapy, thrombus was reduced in two cases.   
Collateral vessels developed slightly in one of the unchanged case at right   
branch of portal vein. The mean number of WBC and CRP were 13750 and 40.6   
mg/dL in the unchanged cases on admission. But in the recovered cases were   
8000 and 4.7 mg/dL. Conclusion: Direct inflammation of acute biliary   
infections made severe thrombus at right branch of portal vein. Blood fows   
of left branch were continued. Then collateral vessels developed slightly in   
the unchanged cases. In the thrombus cases, the inflammations on admission   
were severe. Therefore, early treatments are very important.  
  
RECORD 652  
Pancreatic islet cell transplantation  
Greget M. Kessler L.  
CardioVascular and Interventional Radiology (2009) 32 SUPPL. 2 (261-262).   
Date of Publication: September 2009  
  
Type 1 dependent diabetes mellitus (T1D) is consecutive to loss of insulin   
production due to autoimmune destruction of β pancreatic cells (islets of   
Langerhans). Classical treatment is based on insulin therapy to normalize   
blood glucose levels and prevent acute and chronic complications of type 1   
diabetes. Transplantation of human islets began in the 1970s but it was not   
until 1989 that the first patient was able to stop exogenous insulin. The   
success rate improved dramatically in 2000 with the “Edmoton Protocol” based   
on the need to transplant high quality islets in sufficient number and the   
use of steroid-free immunosuppressive therapy. General admitted criteria for   
allogenic islet cells transplantation are C-peptide negative type 1 diabetes   
for more than 5 years with previous kidney transplantation or T1D with poor   
diabetes control including episodes of severe hypoglycemia, hypoglycemia   
unawareness, wide swings of blood glucose levels or consistently high HbA1c   
levels (>8%). Islets isolation: islets are processed from pancreas procured   
from cadaveric heart-beating donors. The procedure of islets isolation   
consists in placing the harvested pancreas in a digestion chamber after   
injection of an enzyme (collagenase or liberase) in the main pancreatic   
duct. Islets are purified from the obtained preparation by gradient in a   
cell separator. Islets are then cultured in adapted solution. All the   
processing is done under sterile conditions. To be suitable for   
transplantation, the islet preparation isolated from a donor must contain   
more than 250,000 islet equivalents and viability up to 80%. The goal is to   
infuse 10,000 islets equivalent/kg of body mass of the recipient, though it   
is frequently necessary to perform one or two subsequent grafts. Procedure   
of transplantation: the transplantation of islets is performed in a   
heterotopic location in the liver via the portal vein. The access to the   
portal vein is obtained by either trans-hepatic venous catheterization or   
through a mesenteric vein during a minilaparotomy. The percutaneous   
image-guided trans-hepatic route is mainly used. This procedure can be done   
under local anesthesia and conscious sedation. An intra-hepatic portal   
branch is punctured generally in the right lobe of the liver. Ultrasonic   
guidance allows succeeding and securing the puncture. The remaining   
procedure is performed under fluoroscopic control. A guide wire is placed   
through the needle in the portal vein and a 4 to 6 French catheter is then   
pushed up to the portal trunk. Prior to islets infusion, an angiogram is   
performed to check the position of the catheter, the distribution and the   
patency of the portal tree. The pancreatic islets (size about 150 ?m)   
suspended in albumin solution are infused by gravity, along with heparin to   
embolize in the whole liver parenchyma. Portal pressure monitoring shows   
usually a slight elevation during infusion of cells. At the end of the   
delivery, as the catheter is withdrawn, the transhepatic tract is usually   
occluded by embolic agent. A prophylactic anticoagulation is continued for   
several days to reduce the likelihood of an instant blood mediated   
inflammatory reaction. Exogenous insulin is given in the early post   
transplant period to prevent islet damage caused by hyperglycemia. The   
majority of serious adverse events related to the infusion procedure consist   
in bleeding complications mainly (13% of procedures) and portal vein   
thrombosis more rarely (4% partial or complete). The use of heparin has been   
shown to limit the incidence of thrombosis but to increase the rate of   
procedural bleeding. Sealing intra-hepatic tract has demonstrated a   
reduction of the incidence of post-procedural bleeding. The most frequently   
administered immunosuppressive protocol uses Sirolimus and Tacrolimus in   
combination as maintenance therapy and one or more induction agents (i.e.,   
anti IL-2 receptor) at the time of the first islet infusion. Results: the   
report published by the Collaborative Islet Transplant Registry (CITR) in   
2008 about 325 recipients of 624 islet infusions shows at three years 23%   
insulin independence, 29% insulin dependence with detectable C-peptide, 26%   
loss of graft function and 22% missing data. Severe hypoglycemic events   
decrease dramatically from 85% of patients before transplantation to less   
than 5% in the first year. High numbers of infusion, greater number of islet   
equivalents infused, lower pre-transplant HbA1c, processing center related   
to the transplant center and larger islet size are factors that favor the   
primary outcomes. In our Swiss-French multicenter study GRAGIL 2 concerning   
18 T1D patients with poor glucose control (34 infusions), we report   
significant decrease of HbA1c levels (≤7%) in 67% of recipients, decrease of   
insulin requirement ≥30% in 89%, C-peptide ≥ 0.5ng/ml in 83% and no severe   
hypoglycemia in 67% at one year after transplantation. Conclusion:   
transplantation of isolated pancreatic islet has presently become a clinical   
option to be considered in the treatment of T1D after kidney transplantation   
or in case of unstable T1D despite optimal insulin therapy.  
  
RECORD 653  
The evaluation and management of postnatal thromboses  
Saxonhouse M.A. Burchfield D.J.  
Journal of Perinatology (2009) 29:7 (467-478). Date of Publication: 2009  
  
In the pediatric population, neonates have the highest risk for   
thromboembolism (TE), most likely due to the frequent use of intravascular   
catheters. This increased risk is attributed to multiple risk factors.   
Randomized clinical trials dealing with management of postnatal thromboses   
do not exist, thus, opinions differ regarding optimal diagnostic and   
therapeutic interventions. This review begins with an actual case study   
illustrating the complexity and severity of these types of cases, and then   
evaluates the neonatal hemostatic system with discussion of the common sites   
of postnatal thrombosis, perinatal and prothrombotic risk factors, and   
potential treatment options. A proposed step-wise evaluation of neonates   
with symptomatic postnatal thromboses will be suggested, as well as future   
research and registry directions. Owing to the complexity of ischemic   
perinatal stroke, this topic will not be reviewed.  
  
RECORD 654  
A large thrombus in the right atrium and in the inferior vena cava   
associated with a portal vein thrombosis in a patient with hepatic cirrhosis   
- A case report  
Wozakowska-Kapłon B. Sosnowska-Pasiarska B.  
Kardiologia Polska (2009) 67:4 (415-419). Date of Publication: 2009  
  
A case of a 54-year-old female with hepatic cirrhosis, who developed a large   
thrombus in the inferior vena cava that extended up to the right atrium and   
was associated with a portal vein thrombosis. She was admitted to our   
hospital because of symptoms of overt heart failure. A two-dimensional   
echocardiogram demonstrated a large mass in the right atrium originated from   
the inferior vena cava system. Computed tomography scans revealed tumor of   
the liver and a portal vein thrombosis. The patient was discharged on oral   
anticoagulation. Her remaining 1-year course has been uncomplicated.  
  
RECORD 655  
Clinical efficacy of interventional therapy via TIPS approach for the   
treatment of acute or subacute portal venous thrombosis  
Li S. Yan Z.-P. Luo J.-J. Liu Q.-X. Zhu L. Wang Y.-G. Wang J.-H.  
Journal of Interventional Radiology (2009) 18:8 (581-583). Date of   
Publication: August 2009  
  
Objective: To evaluate the clinical efficacy of interventional therapy via   
TIPS approach for the treatment of acute or subacute portal venous   
thrombosis (PVT). Methods: Twelve patients with acute or subacute PVT were   
treated with interventional managements via TIPS approach, including   
balloon-catheter dilating, PTD pulverizing, catheter-directed aspirating and   
continuously urokinase infusing. Reopen of the portal vein was observed   
after the procedure. The stent patency and the relief of the symptoms were   
followed up for (8 - 42) months. Results: One patient died of massive   
bleeding in abdominal cavity at the second day after therapy. Reopen of main   
portal vein was obtained in eleven patients after thrombolysis. Three months   
after the procedure, PVT recurred and the stent was obstructed in one   
patient, perhaps due to the discontinuation of anticoagulation. In the   
remaining 10 patients, the main portal vein and the shunt remained patency   
during a fellow-up period of (8 - 42) months. No bleeding caused by   
varicosity or symptoms related to PVT occurred in all patients. Conclusion:   
Interventional thrombolysis via TIPS approach is an effective therapy for   
treating patients with acute or subacute portal venous thrombosis.  
  
RECORD 656  
Catheter-directed tissue plasminogen activator infusion and concurrent   
systemic anticoagulation with heparin to treat portal vein thrombosis post   
orthoptic liver transplantation  
Gill P. Oniscu G.C. Mayer D.A. Mirza D.F. Olliff S.  
Transplantation (2009) 88:4 (595-596). Date of Publication: August/2009  
  
RECORD 657  
Partial splenic embolization versus splenectomy for the management of   
hypersplenism in cirrhotic patients  
Amin M.A. El Gendy M.M. Dawoud I.E. Shoma A. Negm A.M. Amer T.A.  
World Journal of Surgery (2009) 33:8 (1702-1710). Date of Publication:   
August 2009  
  
Background: Hypersplenism occurs in patients with chronic liver disease, and   
splenectomy is the definitive treatment. However, the operation may be   
hazardous in patients with poor liver function. In recent years, partial   
splenic embolization (PSE) has been widely used in patients with   
hypersplenism and cirrhosis. This study was conducted to assess the safety   
and efficacy of PSE compared to splenectomy in the management of   
hypersplenism in cirrhotic patients. Methods: This study comprised 40   
patients with hypersplenism secondary to cirrhosis. They were divided into   
two groups, each including 20 patients. The first group of patients were   
treated by PSE using polyvinyl alcohol particles to achieve embolization of   
at least 50% of the distal branches of the splenic artery. Postembolization   
arteriography and computed tomography were performed to document the extent   
of devascularization. Patients in the second group were treated by   
splenectomy with or without devascularization and left gastric ligation   
according to the presence or absence of esophageal varices. Results: There   
was marked improvement in platelet and leukocytic counts in both groups, and   
the counts remained at appropriate levels during the follow-up period. All   
patients in the first group had problems related to postembolization   
syndrome that abated by the first week. One patient in the first group died   
from myocardial infarction. No deaths occurred in the second group.   
Asymptomatic portal vein thrombosis developed in one patient in the first   
group that was treated with anticoagulation, and another patient developed   
splenic abscess treated by splenectomy with a good outcome. In the second   
group, three patients developed portal vein thrombosis, one of them being   
readmitted 4 months postoperatively with mesenteric vascular occlusion; that   
patient underwent a resection anastomosis with good outcome. Conclusions:   
Partial splenic embolization is an effective therapeutic modality for the   
treatment of hypersplenism secondary to chronic liver disease. It is a   
simple, rapid procedure that is easily performed under local anesthesia; and   
it allows preservation of adequate splenic tissue to safeguard against   
overwhelming infection. © 2009 Société Internationale de Chirurgie.  
  
RECORD 658  
portal vein thrombosis management in candidates for liver transplantation   
(LT)  
Sartori M.T. Ferronato C. Boccagni P. Gasparini D. Cillo U. Senzolo M.  
Journal of Thrombosis and Haemostasis (2009) 7:S2 (768). Date of   
Publication: July 2009  
  
Background: portal vein thrombosis (PVT) occurs in about 8% of candidates   
for LT and its extension may jeopardize the outcome or contraindicate LT.   
Our aim was to prospectively evaluate an integrated treatment algorithm,   
comprehending anticoagulation and the use of transjugular intrahepatic   
portosystemic shunt (TIPS), to treat PVT in candidates for LT. Methods:   
Patients with previous or occurring PVT and/or splancnic vein thrombosis   
(SVT) while in the waiting list for LT since February 2007 were included.   
All patients underwent screening for systemic and local prothrombotic   
factors. anticoagulation was always considered and contraindicated when high   
risk varices despite prophylaxis or stabilized presence of cavernous   
transformation were present. TIPS was indicated in presence of severe portal   
hypertensive complications, progression of thrombosis or contraindication to   
anticoagulant use. Results: 12 patients with PVT and/or SVT were included:   
eight males, mean age 56.25 ± 5 year; 11 liver cirrhosis, one Budd Chiari   
syndrome (BCS). Eight had PVT, one isolated superior mesenteric vein   
thrombosis at the time of listing for LT; three patients developed de novo   
PVT while awaiting LT. Systemic or local prothrom- botic risk factors were   
identified in 5/12 patients (40%). anticoagulation was initiated in eight   
patients and contraindicated in 4. 4/8 (50%) anticoagulated patients had   
recanalization, whereas two of the untreated patients had thrombosis   
progression in all splancnic veins requiring evaluation for liver and   
intestine transplantation. TIPS was placed in six patients: in three for   
contraindicated anticoagulation, in two for thrombosis progression and in   
one with BCS for acute PVT. LT was performed in four patients without   
complications. Conclusions: An aggressive algorithm to treat PVT in   
candidates for LT, including anticoagulation and TIPS, seems effective to   
avoid PVT progression and allows LT without complications.  
  
RECORD 659  
Management of portal and splancnic vein thrombosis in patients awaiting   
Liver Transplantation (LT)  
Senzolo M. Ferronato C. Boccagni P. Gasparini D. Miotto D. Sartori M.T.   
Cillo U. Burra P.  
Liver Transplantation (2009) 15 SUPPL. 7 (S201-S202). Date of Publication:   
July 2009  
  
Background/aims: portal vein thrombosis (PVT) is reported in about 8% of   
candidates for LT and its extension may jeopardize the outcome of surgery or   
contraindicate LT. The aim of the study was to prospectively evaluate an   
integrated treatment algorithm comprehending the use of transjugular   
intrahepatic portosystemic shunt (TIPS) when anticoagulation was   
contraindicated or failed to treat PVT in candidates for LT. Methods:   
patients with previous or occurring PVT and/or splancnic vein thrombosis   
while awaiting LT since February 2007 were included. All patients underwent   
screening for prothrombotic disordes. anticoagulation was always considered   
and contraindicated when high risk varices despite prophylaxis or stabilized   
presence of cavernous transformation with recanalization were present. TIPS   
was indicated when there was concomitant presence of severe portal   
hypertensive complications, progression of thrombosis or anticoagulation was   
contraindicated. Results: 12 patients with PVT and/or splancnic vein   
thrombosis were included: 8 males, mean age±SD 56,25±5 yrs; 11 cirrhosis, 1   
Budd Chiari syndrome (BCS).8 had partial PVT, 1 isolated superior mesenteric   
vein thrombosis at the time of listing for LT ; 3 patients developed de novo   
PVT while awaiting LT. Prothrombotic systemic disorders or local risk   
factors were identified in 5/12 patients (40%). anticoagulation was   
initiated in 8 patients whereas in 4 it was contraindicated. 4/8 (50%)   
amongst anticoagulated patients had ricanalization (2 partial, 2 total),   
whereas 2 of the non-anticoagulated patients had thrombosis progression in   
all splancnic vein requiring evaluation for liver and intestine   
transplantation. TIPS was performed in 6 patients: 3 in whom anticoagulation   
was contraindicated and 2 who had thrombosis progression and 1 with BCS and   
acute PVT . LT was performed in 4 patients without complications.   
Conclusions: an aggressive algorithm to treat PV/splancnic vein thrombosis   
in candidates for LT, including anticoagulation and TIPS seems effective to   
avoid PVT progression and allow LT without complications.  
  
RECORD 660  
Portal vein recanalization after pediatric transplantation  
Seda-Neto J. Carnevale F. Carone E. Moreira A. Zurstrassen C. Pugliese V.   
Godoy A. Porta G. Miura I. Baggio V. Pugliese R. Fonseca E. Chapchap P.  
Liver Transplantation (2009) 15 SUPPL. 7 (S275). Date of Publication: July   
2009  
  
Background: portal vein thrombosis is an important complication after   
pediatric liver transplantation and occurs at anytime following the   
operation. Early post-operatively, it can lead to graft dysfunction,   
especially in patients receiving large grafts (GRWR>4%). Later, patients can   
develop thrombocytopenia, ascites, splenomegaly, upper GI bleeding, and   
other clinical manifestations. Aim: To describe a combined technique to   
reestablish portal flow in cases of portal vein thrombosis/stenosis after   
the transplant. Methods: 1% (4 patients) of 367 children submitted to liver   
transplantation from Jun/1991 to Dec/2008 underwent portal vein   
recanalization through a combined approach, (transhepatic + laparotomy). All   
patients had biliary atresia with a median body weight of 6.9kg (range   
5.3-10.4Kg) at the time of transplantation. They developed portal vein   
thrombosis (n=3) and stenosis (n=1). The laparotomy was used to give the   
intervention radiology team a tributary of the superior mesenteric vein in   
order to guide the portal vein dilatation and stenting. All patients   
received a left lateral segment (3 LDLT, 1 SPLIT), with a median GRWR=3.3%   
(range 2.5-6%). These patients presented with ascites, splenomegaly,   
thrombocytopenia, and GI bleeding 1.3 to 40 months after the transplant. At   
the time of the procedure, these patients median age and body weight were 28   
months (range 8-43 mo) and 13.2Kg (range 5.5-20.9Kg), respectively. Results:   
Three patients underwent a successful balloon dilatation and stenting with   
the combined technique. One patient had an extensive thrombosis of the   
portomesenteric venous system and the guidewire could not be advanced to the   
emergence of the portal vein. All other patients had the portal flow   
reestablished as observed with serial Doppler ultrasound studies. They   
received oral anticoagulation for three consecutive months after the   
procedure and the clinical symptoms subsided. Conclusion: The combined   
technique to reestablish the portal flow in cases of portal vein   
thrombosis/stenosis is effective and safe. Continuous follow-up is needed to   
access the long-term consequences of the procedure.  
  
RECORD 661  
Incidental splanchnic thrombosis in cancer patients  
Ramasamy S.M. Bozas G. Avery G. Maraveyas A.  
Journal of Thrombosis and Haemostasis (2009) 7:S2 (797). Date of   
Publication: July 2009  
  
Incidental Splanchnic Venous Thrombosis in Cancer Patients. Introduction:   
With advances in CT-imaging, an increasing number of 'incidental' venous   
thromboembolism is being diagnosed. Among these we find a subset with   
incidental splanchnic thromboembolism (SVT). The natural history, morbidity   
and mortality of this phenomenon is largely unknown. This is a descriptive   
retrospective review of cancer patients with incidental diagnosis of SVT   
identified on routine CT. Method: A search of the radiology database at   
Castle Hill Hospital was used to identify oncology patients with incidental   
SVT over a 24 months period using the keywords emboli, embolus, and   
thrombus. Information on patient demographics, malignancy, treatment and   
progress was recorded from review of the clinical notes. Results: Fourteen   
patients (8 males, 6 females) with incidental SVT were identified. The   
prevalence of pancreatic cancer was 50% (7), colorectal cancer 29% (4),   
gallbladder cancer, neuroendocrine small bowel cancer and retroperitoneal   
leiosarcoma 7% (1) each. Eight had portal vein thrombus (PVT), five superior   
mesenteric vein thrombus (SMVT) and one hepatic vein thrombus (HVT). One   
developed SVT while on treament for a previous pulmonary embolus (PE), four   
had concurrent thrombus in other branches of the splanchnic circulation and   
two had further thromboses (deep vein thrombosis, and PE). Eight patients   
were treated with low molecular weight heparin (LMWH), 2 with warfarin, 1   
had an inferior vena cava filter. Data on anticoagulation was unavailable   
for 3 patients. Previous diagnostic/follow-up CT scans free of thromboses   
were found for 12 patients. Mean time to SVT event 4.8 months. Nine patients   
have died and mean survival from the SVT event was 4 months.Conclusion: This   
series is the first to our knowledge to demonstrate a natural history of   
this condition in cancer patients. SVT in cancer patients is not as rare as   
generally thought and needs further study vis a vis impact on survival and   
contribution to morbidity.  
  
RECORD 662  
JAK2-positive latent essential thrombocythemia and splanchnic vein   
thrombosis: The role of bone marrow biopsy for the diagnosis of   
myeloproliferative disease  
Allegra A. Alonci A. Penna G. D'Angelo A. Rizzotti P. Granata A. Musolino C.  
Acta Haematologica (2009) 121:4 (218-220). Date of Publication: July 2009  
  
Background: Splanchnic vein thrombosis (SVT) is a severe complication of   
essential thrombocythemia (ET). No clear explanation has been given for the   
occurrence of thrombosis in this unusual site in patients with ET, but the   
existence of a specific association between unexplained SVT and the JAK2   
mutation has been reported. Methods and Results: The present study describes   
SVT (portal and splenic vein thrombosis) in a young woman as the first   
presenting symptom of latent ET. Extensive screening for thrombophilia was   
negative. Our patient in fact did not fulfill the WHO diagnostic criteria   
for myeloproliferative disease (MPD), while she had splenomegaly and   
developed features suggestive of latent ET during follow-up. Conclusions: In   
these patients with SVT, the detection of JAK2(V617F) mutation is diagnostic   
for masked MPD as could be documented by bone marrow histopathology. The   
presence of JAK2(V617F) mutation should be considered per se a prothrombotic   
state for cerebral, coronary and peripheral microvascular disturbances and   
for SVT but not for deep vein thrombosis. anticoagulation is the treatment   
of choice for all SVT and proper treatment of the MPD is recommended in   
patients with SVT associated with the JAK2(V617F) mutation. Copyright © 2009   
S. Karger AG.  
  
RECORD 663  
Risk factors and clinical course of portal and/or splenic vein thrombosis   
after partial splenic embolization.  
Matsumoto T. Yamagami T. Terayama K. Kato T. Hirota T. Yoshimatsu R. Miura   
H. Ito H. Okanoue T. Nishimura T.  
Acta radiologica (Stockholm, Sweden : 1987) (2009) 50:6 (617-623). Date of   
Publication: Jul 2009  
  
BACKGROUND: Although portal and/or splenic vein thrombosis after partial   
splenic embolization (PSE) is a well-known complication, few reports   
evaluating risk factors have been published. PURPOSE: To investigate risk   
factors and clinical course of portal and/or splenic vein thrombosis after   
PSE. MATERIAL AND METHODS: Sixteen patients with severe hypersplenism   
underwent PSE between March 2005 and April 2008. The correlation between   
portal and/or splenic vein thrombosis after PSE detected on multidetector   
row CT (MDCT) and various factors were retrospectively reviewed. Further,   
the clinical course of portal and/or splenic vein thrombosis after PSE was   
observed on follow-up MDCT. RESULTS: Splenic vein thrombosis was detected in   
eight patients (50%) on MDCT images taken within 9 days after PSE. In one,   
the thrombosis also involved the portal vein. The infarct volume was   
identified as a significant risk factor for portal and/or splenic vein   
thrombosis (P=0.046). In all but one patient, splenic vein thrombosis   
resolved completely or improved without anticoagulation therapy. In this   
patient, both portal and splenic vein thrombosis developed after PSE, and   
anticoagulation therapy was necessary. CONCLUSION: It is suggested that a   
large splenic infarct volume is a risk factor for portal and/or splenic vein   
thrombosis after PSE. Indications for treatment of thrombosis of the portal   
vein system after PSE may be limited to patients with portal vein   
thrombosis.  
  
RECORD 664  
Acute extensive portal and mesenteric venous thrombosis after splenectomy:   
Treated by interventional thrombolysis with transjugular approach  
Wang M.-Q. Lin H.-Y. Guo L.-P. Liu F.-Y. Duan F. Wang Z.-J.  
World Journal of Gastroenterology (2009) 15:24 (3038-3045). Date of   
Publication: 28 Jun 2009  
  
Aim: To present a series of cases with symptomatic acute extensive portal   
vein (PV) and superior mesenteric vein (SMV) thrombosis after splenectomy   
treated by transjugular intrahepatic approach catheter-directed   
thrombolysis. Methods: A total of 6 patients with acute extensive PV-SMV   
thrombosis after splenectomy were treated by transjugular approach   
catheter-directed thrombolysis. The mean age of the patients was 41.2 years.   
After access to the portal system via the transjugular approach, pigtail   
catheter fragmentation of clots, local urokinase injection, and manual   
aspiration thrombectomy were used for the initial treatment of PV-SMV   
thrombosis, followed by continuous thrombolytic therapy via an indwelling   
infusion catheter in the SMV, which was performed for three to six days.   
Adequate anticoagulation was given during treatment, throughout   
hospitalization, and after discharge. Results: Technical success was   
achieved in all 6 patients. Clinical improvement was seen in these patients   
within 12-24 h of the procedure. No complications were observed. The 6   
patients were discharged 6-14 d (8 ± 2.5 d) after admission. The mean   
duration of follow-up after hospital discharge was 40 ± 16.5 mo. Ultrasound   
and contrast-enhanced computed tomography confirmed patency of the PV and   
SMV, and no recurrent episodes of PV-SMV thrombosis developed during the   
follow-up period. Conclusion: Catheter-directed thrombolysis via   
transjugular intrahepatic access is a safe and effective therapy for the   
management of patients with symptomatic acute extensive PV-SMV thrombosis. ©   
2009 The WJG Press and Baishideng. All rights reserved.  
  
RECORD 665  
Liver abscesses with portal and mesenteric vein thrombosis: A rare   
complication of appendicular peritonitis  
Jaouadi S. Boulifi F. Barhoumi H. Sindi S. Rekk S. Mbarek S. Selmi M.   
Kharrat M. Morjane A.  
Jamahiriya Medical Journal (2009) 9:2 (152-154). Date of Publication: Summer   
2009  
  
Pyogenic liver abscesses are caused by appendicitis in less than 10%. Also   
the ascending septic inflammation of portal vein (pylephlebitis) could be a   
serious complication of intra-abdominal infection. Although pylephlebitis is   
not frequent today, its mortality and morbidity rates remain high. We   
describe a case of pylephlebitis, portal-mesenteric thrombosis, and multiple   
liver abscesses, caused by appendicular peritonitis. The patient was   
admitted three weeks after appendectomy with the chief complaints of high   
fever and chills. He was diagnosed of pylephlebitis, portal-mesenteric   
thrombosis, and multiple liver abscesses by CT-scan. He was treated with   
long-term antibiotics and anticoagulation.  
  
RECORD 666  
Portal vein thrombus and liver failure in a patient with pheochromocytoma   
crisis  
Brauchlin A.E. Rudiger A. Bächli E.B. Schmid C. Maggiorini M.  
American Journal of Emergency Medicine (2009) 27:5 (630.e3-630.e5). Date of   
Publication: June 2009  
  
A 51-year-old man with known pheochromocytoma refused surgical treatment   
over several years and subsequently presented in catecholamine crisis with   
shock and multiple organ failure. Laboratory testing revealed liver failure   
with elevated liver enzymes and coagulation abnormalities, and imaging   
showed ischemia of extended parts of the right liver lobe. It also revealed   
a large thrombus in the right portal vein, which together with severe   
arterial vasoconstriction impaired the dual blood supply of the liver. The   
patient recovered after effective medical treatment and finally underwent   
surgical tumor resection. Thereafter, antihypertensive treatment could be   
stopped. We present this exceptional case of adrenal crisis and discuss the   
mechanisms leading to liver failure in general and portal vein thrombosis in   
particular. We present the case of a 51-year-old man who had hypertensive   
spells up to 280/150 mm Hg 40 years ago. At 11 years of age, a   
pheochromocytoma (24 g) was removed after explorative laparatomy. Follow-up   
for 12 years confirmed normal blood pressures, and the patient was   
considered cured. After years of remission, recurrent disease was diagnosed   
4 years ago, as the patient had fatigue and hypertensive spells again. A   
computed tomographic scan revealed a tumor in the lower pelvis, and a biopsy   
confirmed recurrent pheochromocytoma. The patient refused surgical resection   
and he was treated with phenoxybenzamine, an α-receptor blocker. In   
addition, the patient sought relief with alternative medicine. As fatigue   
progressed and jaundice occurred, he was seen by a general practitioner. The   
patient was normotensive and laboratory testing showed normal creatinine   
levels (64 μmol/L) and liver enzymes (aspartate aminotransferase, 25 U/L;   
alanine aminotransferase, 14 U/L). Two weeks later, the patient became   
confused and somnolent and he was brought to a local hospital. The initial   
blood pressure was 151/110 mm Hg. Examination revealed acute renal and liver   
failure. Echocardiography showed a severely depressed left ventricular   
ejection fraction (LVEF) of less than 20%. Antihypertensive treatment with   
labetalol was started and the patient was transferred to our tertiary   
intensive care unit. The somnolent patient presented with clammy skin and   
severe peripheral cyanosis. He was afebrile. Blood pressure values were   
normal at that time, despite grossly elevated norepinephrine levels of 70   
times the upper limits of normal. Intubation for airway protection was   
performed. Laboratory testing was remarkable for high liver enzymes and   
creatinine values, coagulation abnormalities, and elevated lactate levels   
(Table 1). The patient was anuric, and severely impaired kidney perfusion   
was seen by ultrasound. A computed tomographic scan confirmed a large   
abdominal tumor with central necrosis (Fig. 1). A portal vein thrombus with   
ischemia of extended parts of the right liver lobe was detected (Fig. 2).   
Labetalol was stopped and replaced by IV phentolamine, a pure α-blocker.   
Continuous renal replacement therapy was initiated. With normalization of   
the peripheral circulation, cardiac index increased substantially and   
lactate normalized. After 12 hours of treatment, echocardiography showed an   
improvement of the LVEF to 37%. As liver function ameliorated,   
anticoagulation was initiated for the portal vein thrombosis. Four days   
later, the patient was extubated and oral medication begun. Efficient blood   
pressure control was achieved by doxazosine (10 mg BID), carvedilol (25 mg   
BID), lisinopril (30 mg OD), and nifedipine (60 mg OD). After 15 days in the   
intensive care unit and 22 days on the ward, the patient was discharged. Six   
weeks after the initial admission, he was scheduled for surgery.   
Preoperatively, an (18)F-DOPA-PET scan did not show metastasis, and an   
embolization of the lesion was performed. The tumor was removed, and   
histology confirmed the diagnosis of a pheochromocytoma/paraganglioma.   
Postoperatively, LVEF recovered to more than 50% and the antihypertensive   
medication could be stopped, while the patient still required intermittent   
hemodialysis. Pheochromocytomas are rare tumors, occurring in 0.05% of the   
population [1]. They are the cause of secondary hypertension in less than 1%   
[2]. About 25% of the tumors are associated with genetic disorders [3].   
Although our patients' family history was negative, the presentation in   
childhood (classic pheochromocytoma) and the extraadrenal manifestation 40   
years later would be consistent with a germline mutation in the gene   
encoding the B subunit of mitochondrial succinate dehydrogenase (SDH) [3].   
However, genetic testing was negative (Table 2). Pheochromocytoma   
classically presents with recurrent headache, sweating, and tachycardia [4].   
However, it can present with a wide variety of symptoms including shock [3].   
Our case is not only remarkable for its initial presentation and recurrence   
4 decades later but also for its most recent presentation with portal vein   
thrombosis. We hypothesize that low blood flow as a result of the impaired   
LV function as well as volume depletion secondary to vasoconstriction led to   
this complication. Case reports of venous thromboses in pheochromocytoma   
exist. However, they are associated with tumor invasion into blood vessels   
[5,6]. Paraneoplastic thrombophilia is uncommon in pheochromoytomas.   
Ischemia of parts of the right liver lobe with subsequent liver failure is   
explained by interruption of the dual blood supply: the thrombus obstructed   
venous inflow whereas vasoconstriction impaired arterial liver perfusion.   
Liver function recovered remarkable quickly after restoration of arterial   
perfusion. Low cardiac output was caused by several consequences of the   
massive catecholamine release [7]. First, a persistent overstimulation with   
epinephrine leads to down-regulation of cardiac β(1) receptors [8,9] and to   
a switch from β(1) to β(2) signaling, resulting in negative inotropy [10].   
Second, vasoconstriction increased afterload, which further impaired LV   
function. Finally, tachycardia can impair diastolic ventricular filling. The   
case highlights that pheochromocytoma can induce multiple organ failure.   
Liver infarction developed because venous inflow was obstructed by a portal   
vein thrombus and arterial perfusion was severely impaired due to   
vasoconstriction. Effective α-blocking treatment improved the macro- and   
microcirculation resulting in an improvement of organ dysfunction in general   
and liver failure in particular. © 2009 Elsevier Inc. All rights reserved.  
  
RECORD 667  
Vascular Complications of Orthotopic Liver Transplantation: Experience in   
More than 4,200 Patients  
Duffy J.P. Hong J.C. Farmer D.G. Ghobrial R.M. Yersiz H. Hiatt J.R. Busuttil   
R.W.  
Journal of the American College of Surgeons (2009) 208:5 (896-903). Date of   
Publication: May 2009  
  
Background: Thromboses of the hepatic artery (HAT) and portal vein (PVT) may   
complicate orthotopic liver transplantation (OLT) and result in graft loss   
and mortality. Revision and retransplantation are treatment options, but   
their longterm outcomes remain undefined. This study was undertaken to   
evaluate the incidence of major vascular complications after OLT, determine   
efficacy of therapies, and identify factors influencing longterm outcomes.   
Study Design: All patients undergoing OLT from 1984 to 2007 were evaluated.   
Kaplan-Meier analysis was performed to define the effects of vascular   
complications on posttransplant survival. Anastomotic revision and arterial   
thrombolysis were compared with retransplantation as treatment for HAT.   
After 2002, porta hepatis dissection was initiated with early occlusion of   
common hepatic artery (CHA) inflow; its impact on HAT incidence was   
determined. Results: From 1984 to 2007, 4,234 OLTs were performed. HAT   
occurred in 203 patients (5%) and PVT in 84 (2%). Graft survival was   
significantly reduced by HAT or PVT; patient survival was reduced only by   
PVT. Retransplantation for HAT improved patient survival over revision or   
thrombolysis in the first year but did not provide longterm survival   
advantage (56% versus 56% at 5 years; p = 0.53). Patients with HAT had only   
10% graft salvage with anastomotic revision or thrombolysis. HAT was   
significantly reduced with early CHA inflow occlusion (1.1% versus 3.7%; p =   
0.002). Factors increasing risk of HAT included pediatric recipients, liver   
cancer, and aberrant arterial anatomy requiring complex reconstruction.   
Conclusions: Both HAT and PVT significantly reduce graft survival after OLT;   
PVT more adversely affects patient survival. Revision and thrombolysis   
rarely salvage grafts after HAT; retransplantation provides superior   
short-term, but not longterm, survival. Avoidance of vascular complications   
in OLT is critical, especially with today's scarcity of donor livers. Early   
atraumatic CHA occlusion significantly reduces the incidence of HAT. © 2009   
American College of Surgeons.  
  
RECORD 668  
Portal and splenic vein thrombosis caused by acute pancreatitis  
De Cicco I. Varon J.  
Critical Care and Shock (2009) 12:2 (52). Date of Publication: May 2009  
  
RECORD 669  
Diagnostic and therapeutic direct selective portal vein angiography  
Bertram H. Pfister E. Ulrich B. Thomas B. Stephan S.  
Journal of Pediatric Gastroenterology and Nutrition (2009) 48 SUPPL. 3   
(E118). Date of Publication: May 2009  
  
Background and Aim: Selective angiography is the gold standard for detailed   
evaluation of vascular structures. Modern catheter technology enables   
endovascular interventions during the same procedure. We present our   
preliminary experience in pre- and postoperative diagnostic as well as   
therapeutic catheterizations of the portal vein circulation. Methods: Direct   
portal vein catheterizations were performed percutaneously in general   
anaesthesia using the transhepatic or transsplenic approach, respectively,   
with ultrasound as well as fluoroscopic guidance. The track in the spleen   
left by the sheath was closed with coils in all but one patient, we only   
used coils after transhepatic catheterization once. Results: Between 2006   
and 2008, 17 direct portal vein angiographies were performed in 11 patients   
[age: 2-16 years, body weight: 12-68 kg]. Transsplenic direct selective   
angiography (n = 8) of the extrahepatic portal vein was predominantly used   
to display splenic and mesenteric veins as well as the extent and the   
distribution of collateral vessels in patients with portal vein thrombosis   
to define surgical [shunt procedure vs liver transplantation] or   
endovascular [recanalization and embolization of collateral vessels,   
respectively] options. Transsplenic stent angioplasty of portal vein   
stenosis and transsplenic recanalization of complete portal vein thrombosis   
was successfully performed in one patient each. Transhepatic catheterization   
of the portal vein was chosen for balloon (n = 2) or stent angioplasty (n =   
5) of portal vein stenosis after liver transplantation. In general, these   
are time-consuming procedures requiring sophisticated equipment and   
experienced investigators. Most patients needed multiple punctures, before a   
wire could be advanced into the portal vein. In 3 patients we were not able   
to enter the portal vein percutaneously by the transhepatic (n = 2) or the   
transsplenic route, respectively. All 3 were catheterized successfully   
during a second procedure using transsplenic access. We faced an oozing   
bleeding into the abdominal cavity along the sheath requiring blood   
transfusion in one patient. A thrombus without compromise of blood flow was   
noticed after stent angioplasty of portal vein stenosis which resolved   
during anticoagulation therapy. Transitory mild fever and moderate abdominal   
pain within 48 h after intervention occurred in 60% of patients. Fever with   
positive blood culture was noticed in one patient after transhepatic stent   
angioplasty of portal vein stenosis. Conclusions: Direct selective portal   
vein angiography is a technically demanding procedure. It uniquely displays   
the extrahepatic portal venous system including collateral vessels and gives   
haemodynamic data that may help to take therapeutic decisions. Endovascular   
catheter interventions may successfully be performed using the transhepatic   
or transsplenic approach.  
  
RECORD 670  
Pylephlebitis due to perforated appendicitis in a teenager  
Levin C. Koren A. Miron D. Lumelsky D. Nussinson E. Siplovich L. Horovitz Y.  
European Journal of Pediatrics (2009) 168:5 (633-635). Date of Publication:   
May 2009  
  
Pylephlebitis, a septic thrombophlebitis of the portal vein, is a   
life-threatening complication of intraabdominal infections, commonly   
associated with acute appendicitis in children, and diverticulitis in   
adults. A 13-year-old boy was admitted for high fever and jaundice. On the   
fifth day of hospitalization, ultrasound Doppler flow and Computer   
Tomography scan studies showed thrombosis of the portal vein and acute   
appendicitis. The patient was treated with antibiotics, anticoagulation and   
laparotomy with appendectomy. No thrombophilic risk factors were diagnosed.   
Our aim is to improve physicians' awareness of this complication and   
emphasize the importance of early diagnosis and appropriate therapy in   
children in order to reduce serious complications and long-term sequels. ©   
2008 Springer-Verlag.  
  
RECORD 671  
Cytomegalovirus-associated superior mesenteric vein thrombosis treated with   
systemic and in-situ thrombolysis  
Moerkercke W.V. Pauwelyn K. Brugman E. Verhamme M.  
European Journal of Gastroenterology and Hepatology (2009) 21:5 (587-592).   
Date of Publication: May 2009  
  
A 56-year-old patient, first diagnosed with an acute cytomegalovirus   
infection, presented with progressive abdominal pain because of a superior   
mesenteric vein thrombosis for which he was treated with systemic   
thrombolysis and heparin in continuous infusion. As this therapy did not   
have the intended success after 5 days, an interventional radiological   
procedure was performed with local thrombolysis in the superior mesenteric   
artery resulting in recanalisation of the vein. Oral anticoagulation was   
initiated and continued for a period of 6 months. Mesenteric venous   
thrombosis is a relatively uncommon cause of mesenteric ischemia that can be   
associated with severe morbidity and significant mortality. With noninvasive   
techniques, it is possible to establish a diagnosis in the majority of the   
cases. The importance of an early diagnosis and therapy - not only with   
anticoagulation, but also thrombolysis in selected cases - is shown with   
this case and review of the literature. © 2009 Lippincott Williams &   
Wilkins, Inc.  
  
RECORD 672  
Non-alcoholic fatty liver: Its diagnosis at present. Part Three  
Pluma V.H.C. Morales A.C. Arroyo J.L.A. Sánchez M.D.C.D.L.T. González M.V.  
Medicina Interna de Mexico (2009) 25:3 (217-228). Date of Publication:   
May-June 2009  
  
The non-alcoholic fatty liver is a little diagnosed but more and more   
frequent because of their association with metabolic syndrome, predominantly   
with obesity and diabetes mellitus. In our country every day we find more   
patients facing liver cirrhosis, that were not timely diagnosed with this   
disease, situation capable of transforming its natural history. The   
following paper attempts to define the disease, revealing their causes,   
describing pathophysiologic mechanisms that generate it, to establish routes   
diagnostic, therapeutic and offer different approaches to mention the   
complications in the adult population, through an extensive analysis of   
selected bibliographies by a group of doctors trained in clinical research,   
with the aim of offering a final document that provides the most relevant   
topic in recent years, due to its extension has been divided into several   
chapters. More than nine hundred citations and electronic spaces were   
studied to eventually include the material contained only five hundred and   
seven references. In his last chapter, presented exclusively works by   
Mexican researchers and annexed various suggestions of authors in terms of   
dietary management and drug therapy, in an experiment on 97 patients treated   
over a period of four years.  
  
RECORD 673  
Liver transplantation in end-stage liver disease with portal vein thrombosis  
Wu G. Liu Y.F. Liu S.R. Zhang J.L. Chen X.C. Cheng D.H.  
Zhonghua wai ke za zhi [Chinese journal of surgery] (2009) 47:8 (590-593).   
Date of Publication: 15 Apr 2009  
  
OBJECTIVE: To summarize the experience in the managements of portal vein   
thrombosis (PVT) and to evaluate the impact of PVT on intraoperative course   
and postoperative outcome in liver transplantation. METHODS: Between May   
1995 and September 2007, 194 orthotopic liver transplantations were   
performed, of which 24 cases presented portal vein thrombosis. There were 12   
patients with grade I, 9 with grade II, 2 with grade III and 1 with grade   
IV. The management of PVT depended mainly on its extent. Ligation of the   
collateral circulation, especially spontaneous or surgical splenorenal   
shunt, was made as approaches to improve portal flow.Heparin or   
low-molecule-weight heparin as a prophylactic anticoagulation therapy was   
maintained during and after operation if prothrombin time is less than   
eighteen seconds. Follow-up Doppler ultrasonography was used daily in the   
early postoperative period. Risk factors and variables associated with the   
transplant and the post-transplant period were analyzed and compared with   
170 patients transplanted without PVT. RESULTS: Surgical techniques were   
eversion thromboendovenectomy in 21 patients with PVT grades I and II,   
extra-anatomic mesenteric graft in 2 with grade III, and anastomosis to a   
collateral vein in 1 with grade IV. The study demonstrated more RBC   
transfusions [(15.2 +/- 11.8) U vs. (8.6 +/- 6.6) U, P = 0.006], longer   
surgery procedures [(492 +/- 89) min vs. (403 +/- 105) min, P = 0.001] and   
hospital stay [(32.4 +/- 13.5) d vs. (22.1 +/- 9.1) d, P = 0.001] in the PVT   
group. However, there were no differences in overall morbidity (58.3% vs.   
50.6%, P = 0.478), hospital mortality (8.3% vs.6.5%, P = 0.73) and 1-year   
survival (87.5% vs. 89.4%, P = 0.778). The incidence of rethrombosis was   
higher in the PVT group (8.3% vs.1.2%, P = 0.021). Two cases rethrombosis   
were successfully cured by percutaneous thrombolysis, balloon angioplasty,   
and stent placement. CONCLUSION: Portal thrombosis is associated with   
greater operative complexity and rethrombosis, but has no influence on   
overall morbidity and mortality in liver transplantation.  
  
RECORD 674  
Obliterative portal venopathy (OPV): A retrospective study of 59 patients  
Hillaire S. Cazals-Hatem D. Rudler M. Denninger M.-H. Plessier A. Francoz C.   
Durand F. Bedossa P. Valla D.  
Journal of Hepatology (2009) 50 SUPPL. 1 (S79-S80). Date of Publication:   
April 2009  
  
Aims: Obliterative portal venopathy (OPV) is a rare condition characterized   
by a primary occlusion of intrahepatic portal vein branches. Clinical   
features, course, and causes remain unclear. The aim of this work was to   
clarify the clinical and histological presentations, the associated diseases   
and the outcome of patients in whom a diagnosis of OPV was made at biopsy.   
Methods: A retrospective study of 59 patients diagnosed with OPV between   
1987 to 2008, in a single hospital. Diagnosis was established when, in a   
liver biopsy sample (>1 cm in length and containing >5 portal tracts), >60%   
of portal venules were obstructed, in the absence of cirrhosis. Results: Age   
at diagnosis was 7-77 yr. Median follow-up was 8 years (±7 years). Diagnosis   
required repeated histologies in 24 (41%) patients: OPV was combined with   
regenerative hepatocellular changes (71%), sinusoidal fibrosis (57%),   
sinusoidal dilatation (41%), and aberrant vessels (35%); septal fibrosis   
(Hepatoportal sclerosis 29%). Prothrombotic and/or systemic diseases were   
associated with OPV in only 48% of patients. 19% of patients had severe   
complications, leading to transplantation or death at a median age of 47.5   
yrs. Comparisons according to associated diseases are presented in the table   
(statistically significant differences in bold). Patients with prothrombotic   
disorders had earlier and more frequent anticoagulation therapy, and   
survived without transplantation. Conclusion: OVP is a heterogeneous,   
potentially lethal condition, frequently complicated by portal thrombosis.   
Patients with underlying pro- thrombotic conditions and receiving early   
anticoagulation appear to have a better outcome despite a high proportion of   
portal thrombosis. A trial of anticoagulation therapy in patients with OPV   
is warranted.  
  
RECORD 675  
Portal hypertension resulted from paroxysmal nocturnal hemoglobinuria: A   
case report and review of literature  
Yin D.-L. Liu L.-X. Zhang S.-G. Tian L.-T. Lu Z.-Y. Jiang H.-C.  
International Journal of Hematology (2009) 89:3 (302-304). Date of   
Publication: April 2009  
  
Paroxysmal nocturnal hemoglobinuria is a rare intravascular hemolytic   
anemia, and thrombosis is the leading cause of mortality rate. The hepatic   
veins is the common site where Budd-Chiari syndrome usually occurs. We   
confronted a patient who simultaneously happened to have portal vein and   
superior mesenteric vein thrombosis leading to prehepatic portal   
hypertension and upper gastrointestinal bleeding. Percutaneous thrombolysis   
is an efficacious treatment. © 2009 The Japanese Society of Hematology.  
  
RECORD 676  
Combined portal and hepatic vein thrombosis defined in a case with chronic   
constrictive pericarditis of tuberculosis etiology  
Yetkin U. Ilhan G. Calli A.O. Yesil M. Gurbuz A.  
Interactive Cardiovascular and Thoracic Surgery (2009) 8 SUPPL. 1   
(S118-S119). Date of Publication: April 2009  
  
Objective: Although decrease in incidence of pulmonary tuberculosis recently   
reduced the incidence of chronic constrictive pericarditis developing due to   
tuberculosis, tuberculosis still plays a role of as much as 10% in the   
etiology of pericarditis. It also reflects many complications developing   
secondary to constriction. Methods: Our case was a 38-year-old male. He was   
suffering from dyspnea, fatigue, chest pain, ascites and palpitation   
increasing in intensity for the last two months. He was still receiving a   
combined antibiotic regimen against pulmonary tuberculosis which was   
diagnosed eight months ago. Results: Two dimensional colored Doppler   
echocardiography revealed diffuse pericardial calcification and fresh   
thrombus within dilated hepatic veins. Abdominal ultrasound showed   
hepatomegaly beside massive portal vein thrombosis. He was consulted by the   
Department of Gastroenterology and an anticoagulation therapy with low   
molecular weight heparin was initiated at an optimal dosage aimed at the   
portal vein thrombosis. Our surgical approach was a successful   
pericardiectomy. He was discharged after complete recovery. Conclusions:   
Constrictive pericarditis typically demonstrates itself with long-lasting   
and insidious symptoms and signs secondary to systemic venous congestion.   
Recently, it is observed obviously that number of cases proceeding severely   
due to secondary complications reduced with the early diagnosis and   
appropriately planned surgical therapy.  
  
RECORD 677  
anticoagulation for portal vein thrombosis in cirrhotic patients should be   
always considered  
Senzolo M. Ferronato C. Burra P. Sartori M.T.  
Internal and Emergency Medicine (2009) 4:2 (161-162). Date of Publication:   
April 2009  
  
RECORD 678  
Unenhanced ct scan in acute portal vein thrombosis (PVT): An easy, accurate   
and useful radiological finding for diagnosis and thrombosis dating  
Bruno O. Plessier A. Bureau C. Chagneau-Derrode C. Condat B. Valla D.   
Vilgrain V.  
Hepatology (2009) 50 SUPPL. 4 (474A). Date of Publication: 2009  
  
Background: Portal hypertension and intestinal infarction are potentially   
lethal complications of acute PVT. Recent venous thrombi may be   
hyperattenuated on unenhanced CT scans, caused by high protein content of   
concentrated blood clots. Early accurate diagnosis of PVT is increasingly   
important for specific anticoagulant treatment. Aim: To determine the   
frequency of hyperattenuation within the portal vein on unenhanced CT scans   
in patients with proved acute portal vein thrombosis. Patients and Methods:   
Twenty six patients with acute portal vein thrombosis or its main   
tributaries were included over a four-year period. All patients had   
unenhanced and contrast-enhanced CT scan at diagnosis. CT scans were   
retrospectively analyzed by two expert radiologists on PACS. Attenuation   
characteristics of the portal vein and its main tributaries were assessed on   
unenhanced CT scans. portal vein thrombosis was diagnosed by the presence of   
low-attenuation intra-luminal filling defect, on portal venous phase.   
Results: 26 patients (16 men, median age 46.6 years, 25-84 years) with acute   
PVT were included. Clinical symptoms at onset were abdominal pain in 25/26   
(96%), and systemic inflammatory response syndrome (SIRS) in 13/26 (50%) of   
patients Inherited thrombophilia was present in 6/26 patients, acquired   
thrombophilia in12/26, oral contraceptive use in 5/10, while 9/26 had a   
local factor. Morphological changes of the liver, splenomegaly, ischemic   
bowel signs and radiological ascites were present respectively in 8, 13, 4   
and 6/26 patients At diagnosis, 6/26 patients had isolated portal vein   
thrombosis, and 20/26 had associated mesenteric and splenic vein thrombosis.   
Hyperattenuation within the portal vein and/or its main tributaries on   
unenhanced CT was found in 21/26 (81%) patients (5 portal vein alone, 6 main   
tributaries alone, 10 portal vein and tributaries). Median time between   
onset of symptoms and CT was 5 days (range: 1-60). Hyperattenuation was seen   
in 15/16, (93%), in6/8 (75%) and in0/2 (0%) patients when CT was performed   
within 0-7, 7-30 or 30-60 days from onset of symptoms respectively.   
Conclusion: Hyperattenuation within the portal vein or its main tributaries   
on unenhanced CT is frequent in acute portal vein thrombosis. It gives   
relevant information concerning PVT dating. In patients with acute abdominal   
pain, hyperattenuation within the portal vein on unenhanced CT is   
interesting for early facilitated acute PVT diagnosis. Early diagnosis may   
improve early anticoagulation and recanalization.  
  
RECORD 679  
Long-term follow-up of patients with portal vein thrombosis and   
myeloproliferative diease  
Bresser E.L. Hoekstra J. Smalberg J. Spaander M.C. Leebeek F.W. Janssen H.L.  
Hepatology (2009) 50 SUPPL. 4 (473A-474A). Date of Publication: 2009  
  
Background: In patients with non-malignant non-cirrhotic portal vein   
thrombosis (PVT), myeloproliferative disorders (MPD) are the most frequent   
underlying cause, occurring in approximately one third of the cases. The aim   
of this study was to describe the long-term outcome of this specific patient   
group. Methods: A retrospective cohort study was performed including all   
patients referred to our hospital between January 1980 and December 2008   
with non-malignant non-cirrhotic PVT and confirmed MPD. Results: We included   
47 patients (72% female) with a median age at diagnosis of PVT of 47 years   
(range 11-79). Thrombosis was either confined to the portal vein (n=24) or   
included an extension into the splenic and/or superior mesenteric vein   
(n=23). In 34 patients (72%) PVT was the first manifestation of MPD. Type of   
MPD was defined as polycythemia vera (n=14), essential thrombocytosis   
(n=12), myelofibrosis (n=6) or unclassified MPD (n=15). The JAK2 V617F   
mutation was present in 28 of 30 tested patients. Additional prothrombotic   
factors were present in 32% of the cases (n=15). Median follow-up time after   
diagnosis of PVT was 5.8 years (range 0.4-22). During follow-up 26 patients   
(55%) were treated with anticoagulation. Treatment for MPD was given in 39   
patients (83%), most frequently consisting of acetylsalicylic acid,   
hydroxyurea and/or phlebotomy. During follow-up 31 patients (66%) developed   
esophageal varices, of whom 18 (38%) experienced at least one bleeding   
episode. The occurrence of variceal bleeding was not significantly related   
to long-term use of anticoagulation (p=0.26). In 12 patients (26%) at least   
one additional thrombotic event occurred, of whom 3 were using   
anticoagulants at the time of first new event. In 3 patients recurrent   
thrombosis developed after previous anticoagulation had been discontinued.   
Eighteen patients (38%) died during followup at a median age of 64.4 years   
(range 30-88). Overall survival rate was 97% and 88% at 1 and 5 years,   
respectively. In 11 cases (61%) death was directly related to a new   
thrombotic event (cerebral infarction (n=2) or mesenteric vein thrombosis   
(n=1)) or end-stage MPD (end-stage myelofibrosis (n=5) or acute myeloid   
leukemia (n=3)). One patient died due to variceal bleeding. Conclusions: PVT   
is often the presenting symptom of an underlying MPD, highlighting the   
necessity for extensive screening. Treatment with anticoagulation was not   
associated with an increased risk of variceal bleeding during follow-up.   
Recurrent thrombosis is a frequent complication in patients with PVT and   
MPD. Mortality is primarily related to the underlying MPD and not to   
complications of portal hypertension.  
  
RECORD 680  
Prevalence of portal vein thrombosis in patients with obstructive portal   
venopathy (OPV) during HIV infection: Impact of anticoagulation on   
recanalization  
Plessier A. Gervais A. Rautou P.-E. Lemoine M. Cazals-Hatem D. Francoz C.   
Labadie H. De Gennes C. Campa P. Piketty C. Condat B. Hillaire S. Ozenne V.   
Aumaitre H. Durand F. Bedossa P. Valla D.  
Hepatology (2009) 50 SUPPL. 4 (441A-442A). Date of Publication: 2009  
  
Background: Course, outcome and prognosis of portal vein thrombosis in   
patients with obstructive portal venopathy (OPV) and HIV infection need to   
be clarified. Methods: Multicentric retrospective analyses of 20 patients   
with HIV and OPV seen in 2003 and 2009. Results: 20 patients (12 males),   
median age 51 years (range 32-69), median CD4 227/mm3 (100-355), median HIV   
viral load 25 copies/mL (0-26000) were followedup for 3-69 months (median 14   
months) after histological diagnosis of OPV. Liver biopsy showed associated   
nodular regenerative hyperplasia in 13 patients. OPV was diagnosed 4-19   
years (median 13 years) after HIV infection diagnosis. Identified   
prothrombotic factors were protein S deficiency in 10 patients, protein C   
deficiency in 1 and antiphospholipid syndrome in 1. All patients had   
received didanoside for a median duration of 120 months (48-1156). At   
diagnosis, median AST level was 55 IU (32-131). Oesophageal varices and   
splenomegaly were present in 16/20 and 13/20 patients respectively. Median   
platelets count was 132 ×109/L (58-226). During follow up, among 17 patients   
with oesophageal varices, 9 had variceal bleeding, 2 had bleeding recurrence   
despite beta blockers. Severe related bleeding complications were: ascites   
in 5 patients responsible for severe undernutrition in 3, ascites infection   
in 2; and liver related death in 2. One patient with refractory hydrothorax   
had liver transplantation with a good outcome. Eleven patients had   
thrombosis of the portal vein or its branches, extending to the mesenteric   
vein in 1 patient. In 4 of these 11 patients, portal vein thrombosis   
occurred during follow-up. All these 11 patients were subsequently given   
anticoagulation therapy. Recanalization rate was 40% at one year. Two   
patients had extension of thrombosis despite anticoagulation with Warfarin.   
One year transplantfree and overall survival rates were 81% and 86%,   
respectively. Ascites and lower body mass index were significantly   
associated to an unfavourable outcome (transplantation or death).   
Conclusion: OPV is a potentially fatal disease occurring late in HIV   
infection course. Survival is poor in patients with ascites, or   
undernutrition, suggesting that liver transplantation should be considered   
in these patients. portal vein thrombosis is a frequent complication and may   
in case of extension preclude liver transplantation. Recanalization is   
obtained in 40% of patients treated with anticoagulation. Cautious   
surveillance for early detection of PVT and close INR monitoring is   
necessary to avoid thrombosis extension.  
  
RECORD 681  
Splanchnic venous thrombosis  
Plessier A.  
Sang Thrombose Vaisseaux (2009) 21:3 (140-150). Date of Publication: March   
2009  
  
The management of splanchnic venous thrombosis, extra-hepatic portal vein   
obstruction and the Budd-Chiari syndrome has been transformed in the last   
ten years. The diagnostic methods are less invasive due to advances in   
imaging techniques. Myeloproliferatve disease, observed in 20 to 50% of   
cases, may be identified by the JAK2 V817B mutation. Therapeutic strategies   
are planned by stages according to response to previous treatment. The   
common findings of a prothrombotic state such as myeloproliferative   
syndromes, deficits in protein S and C, mutation of factor V Leiden or   
factor II and the antiphospholipid syndrome, require complete investigation   
including the search for systemic diseases or more rare associated   
aetiologies. Doppler ultrasonography should be performed by a trained   
operator informed of the suspected diagnosis so that the obstruction and/or   
collateral circulation can be documented. The presenting signs of acute   
portal vein thrombosis are epigastric pain in 80% of cases, associated with   
a marked inflammatory syndrome.When the diagnosis is missed, portal   
cavernoma may develop. The clinical signs are then those of portal   
hypertension and its complications, biliary symptoms secondary to   
compression of the bile ducts by the veins of the cavernoma or pain and an   
inflammatory syndrome in cases of thrombosis of a cavernoma vein. In   
patients seen at an early stage of thrombosis, anticoagulant therapy is   
recommended and investigation for a prothrombotic state. anticoagulation   
favours the recanalisation of the portal vein (30% of cases) and prevents   
mesenteric necrosis in many patients. In the Budd-Chiari sundrome, the five   
year survival rate of patients treated by this step-wise therapeutic   
strategy (anticoagulation, treatment of cause, recanalisation, trans-jugular   
intra-hepatic porto-systemic shunt (TIPS), liver transplantation) is over   
80%.  
  
RECORD 682  
Budd-Chiari syndrome in Sweden: Epidemiology, clinical characteristics and   
survival - An 18-year experience  
Rajani R. Melin T. Björnsson E. Broomé U. Sangfelt P. Danielsson Å.   
Gustavsson A. Grip O. Svensson H. Lööf L. Wallerstedt S. Almer S.H.C.  
Liver International (2009) 29:2 (253-259). Date of Publication: 2009  
  
Background: The exact incidence and prevalence of Budd-Chiari syndrome (BCS)   
is unknown in the general population. Published reports differ in terms of   
the clinical characteristics, effects of therapy and survival. Aims: To   
investigate the epidemiology, clinical presentation and survival in patients   
with BCS. Methods: Retrospective multicentre studyin Sweden reviewing the   
medical records of all patients with BCS 1986-2003, identified from the   
computerised diagnosis database of 11 hospitals, including all university   
hospitals and liver transplantation centres. Results: Forty-three patients   
with BCS were identified, of whom nine (21%) had concomitant portal vein   
thrombosis. The mean age-standardised incidence and prevalence rates in   
1990-2001 were calculated to be 0.8 per million per year and 1.4 per million   
inhabitants respectively. Myeloproliferative disorders (38%), thrombophilic   
factors (31%) and oral contraceptives (30%) were common aetiological   
factors. Two or more risk factors were present in 44%. In 23%, no risk   
factor was evident. The median follow-up time was 2.7 years. Seventy-two   
percent were on anticoagulant therapy during follow-up. Transjugular   
intrahepatic portosystemic shunting, surgical shunting procedures and liver   
transplantation were performed in 4, 6 and 18 patients respectively.   
Nineteen patients died. The overall transplantation-free survival at 1, 5   
and 10 years was 47, 28 and 17% respectively. Conclusions: Budd-Chiari   
syndrome is a rare disorder; the mean age-standardised incidence and   
prevalence rates in Sweden in 1990-2001 were calculated to be 0.8 per   
million per year and 1.4 per million inhabitants respectively. The presence   
of a myeloproliferative disorder was a common aetiological factor in our   
cohort and about half of the patients had a multifactorial aetiology. The   
transplantation-free survival was poor. © 2009 The Authors. Journal   
compilation © 2009 Blackwell Munksgaard.  
  
RECORD 683  
Vascular liver disorders (II): portal vein thrombosis  
Hoekstra J. Janssen H.L.A.  
Netherlands Journal of Medicine (2009) 67:2 (46-53). Date of Publication:   
February 2009  
  
portal vein thrombosis (PVT) is a rare disorder that is associated with a   
variety of underlying conditions, of which liver cirrhosis, malignancy and   
myeloproliferative disorders are the most common. Based on clinical   
presentation and results of imaging, two different entities can be   
identified, acute and chronic PVT. anticoagulation therapy is recommended   
for all patients with acute PVT in an attempt to prevent further thrombosis   
and to promote recanalisation of the obstructed veins. Chronic PVT is   
characterised by the presence of a portal cavernoma and development of   
portal hypertension. Bleeding from ruptured oesophageal or gastric varices   
is the main complication of portal hypertension in these patients. Both   
endoscopic therapy and β-adrenergic blockade are used for the prevention and   
treatment of gastrointestinal bleeding. In the absence of bleeding,   
continuous anticoagulant therapy should be considered for the group of   
chronic PVT patients in whom an underlying prothrombotic factor can be   
identified. With adequate management of complications and concurrent   
diseases, prognosis of PVT is good in patients without underlying cirrhosis   
or malignancies. ©2009 Van Zuiden Communications B.V. All rights reserved.  
  
RECORD 684  
Role of citrate and other methods of anticoagulation in patients with severe   
liver failure requiring continuous renal replacement therapy  
Bouchard J. Madore F.  
NDT Plus (2009) 2:1 (11-19). Date of Publication: February 2009  
  
anticoagulation is required during continuous renal replacement therapy to   
prevent filter clotting and optimize filter performance. However,   
anticoagulation may also be associated with serious bleeding complications.   
Patients with liver failure often suffer from underlying coagulopathy and   
are especially prone to anticoagulation complications. The aim of this   
review is to present the unique features of patients with hepatic injury in   
terms of anticoagulation disorders and to analyze data on safety and   
efficacy of the different anticoagulation methods for liver failure patients   
undergoing continuous renal replacement therapy. © The Author [2008].   
Published by Oxford University Press on behalf of ERA-EDTA. All rights   
reserved.  
  
RECORD 685  
The Evaluation and Management of Neonatal Coagulation Disorders  
Saxonhouse M.A. Manco-Johnson M.J.  
Seminars in Perinatology (2009) 33:1 (52-65). Date of Publication: February   
2009  
  
Neonatal hemostatic abnormalities can present diagnostic and therapeutic   
challenges to the physician. Developmental deficiencies and/or increases of   
certain coagulation proteins, coupled with acquired or genetic risk factors,   
can result in a hemorrhagic or thromboembolic emergency. The timely   
diagnosis of a congenital hemorrhagic or thrombotic disorder can avoid   
significant long-term sequelae. However, due to the lack of randomized   
clinical trials addressing the management of neonatal coagulation disorders,   
treatment strategies are usually empiric and not evidence-based. In this   
chapter, we will review the neonatal hemostatic system and will discuss the   
most common types of hemorrhagic and thrombotic disorders. Congenital and   
acquired risk factors for hemorrhagic and thromboembolic disorders will be   
presented, as well as current treatment options. Finally, suggested   
evaluations for neonates with either hemorrhagic or thromboembolic problems   
will be reviewed. © 2009 Elsevier Inc. All rights reserved.  
  
RECORD 686  
A clinical analysis of 110 patients with sporadic viral hepatitis E  
Huang S.M. Tang Y.H. Chen Y.P.  
Hepatology International (2009) 3:1 (205). Date of Publication: 2009  
  
Objective: To investigate the clinical features of sporadic hepatitis E.   
Methods: To analyze 110 patients with hepatitis E retrospectively. Results:   
The hepatitis E was predominantly sporadic, some patients superinfected with   
other viral hepatitis, especially hepatitis B. In the old patients, jaundice   
lasted longer and the length of stay was longer, the incidence of   
complication was higher than the young men. The incidence of complication in   
the superinfected group was higher than the simple infection. The   
transaminase in the simple infection group was obviously raise than   
superinfected with liver cirrohsis. Conclusion: The patients infected with   
hepatitis E of young men were frequently. Jaundice lasted long in the old   
patients, the incidence of complication was higher in the superinfected men   
and the old men.  
  
RECORD 687  
Venous Thrombotic Emergencies  
DeLoughery T.G.  
Emergency Medicine Clinics of North America (2009) 27:3 (445-458). Date of   
Publication: August 2009  
Cancer Emergencies: Part II, Book Series Title:  
  
Thrombosis is a common complication of cancer, occurring in up to 15% of   
patients. This article reviews the diagnosis and management of the most   
common cancer-related thrombotic problem; deep venous thrombosis, pulmonary   
embolism, and catheterrelated thrombosis. Rarer entities, such as cerebral   
vein thrombosis and Budd-Chiari syndrome, are also reviewed. © 2009 Elsevier   
Inc. All rights reserved.  
  
RECORD 688  
Antithrombotic therapy in children with venous thromboembolism  
Yang J. Paredes N. Chan A.K.C.  
Hamostaseologie (2009) 29:1 (80-87). Date of Publication: January 2009  
  
Antithrombotic therapy has recently become more frequent for the treatment   
of venous thromboembolism (VTE) in the paediatric population. This can be   
explained by the increased awareness of morbidities and mortalities of VTE   
in children, as well as the improved survival rate of children with various   
kinds of serious illnesses. Considering the large number of years a child is   
expected to survive, associated morbidities such as postthrombotic syndrome   
and risk of recurrence can significantly impact on the quality of life in   
children. Therefore, timely diagnosis, evidence-based treatment and   
prophylaxis strategies are critical to avoid such complications. This review   
summarizes the current literature about the antithrombotic treatment for VTE   
in infants and children. It guides the paediatric medical care provider for   
making a logical and justifiable decision. © 2009 Schattauer GmbH.  
  
RECORD 689  
Portal Hypertension-Related Complications After Acute Portal Vein   
Thrombosis: Impact of Early anticoagulation  
Turnes J. García-Pagán J.C. González M. Aracil C. Calleja J.L. Ripoll C.   
Abraldes J.G. Bañares R. Villanueva C. Albillos A. Ayuso J.R. Gilabert R.   
Bosch J.  
Clinical Gastroenterology and Hepatology (2008) 6:12 (1412-1417). Date of   
Publication: December 2008  
  
Background & Aims: Acute portal vein thrombosis (APVT) is a rare disorder   
that causes chronic portal hypertension if recanalization is not obtained.   
However, response to anticoagulation and long-term prognosis of APVT are not   
well-defined. Methods: Thirty-eight patients diagnosed with APVT between   
1995 and 2003 from 5 Spanish referral hospitals, in whom cirrhosis and   
malignancy were specifically excluded, were included in this retrospective   
study. The response to anticoagulation therapy and development of portal   
hypertension-related complications during follow-up were evaluated. Results:   
Mean follow-up was 43 months (range, 6-112 months). Recanalization occurred   
in 12 of 27 patients receiving anticoagulation versus 0 of 11 patients who   
did not receive anticoagulation (P = .008). Rates of recanalization were   
influenced by the precocity of heparin administration and the number of   
underlying prothrombotic conditions. Follow-up upper endoscopy performed in   
29 patients disclosed gastroesophageal varices in 16 (55%). Varices appeared   
as early as 1 month after APVT. However, in most patients varices were   
detected in successive endoscopies, mainly during the first year. Two-year   
actuarial probability of variceal bleeding was 12% and for ascites 16%.   
Five-year survival was 87%. Mortality was related to the APVT episode in 2   
cases and to an underlying hematologic disorder in one. Conclusions:   
anticoagulation achieved recanalization in about 40% of patients. Most   
patients not achieving recanalization will develop gastroesophageal varices   
during follow-up. However, development of variceal bleeding and ascites is   
infrequent, and survival is satisfactory. © 2008 AGA Institute.  
  
RECORD 690  
portal vein thrombosis after laparoscopic splenectomy: The size of the risk  
Targarona E.M.  
Surgical Innovation (2008) 15:4 (266-270). Date of Publication: 2008  
  
portal vein thrombosis (PVT) after splenectomy is a potentially   
life-threatening complication. Clinical symptoms may be insidious, and   
progression can lead to intestinal infarction and portal hypertension.   
Interest in PVT has increased as a high incidence has been found in the   
laparoscopic setting. The higher incidence of PVT found in recent   
prospective studies of laparoscopically operated patients compared with   
retrospective reports from the 1990s suggests that PVT may have been   
underreported. Clinical outcome depends on the extension of the thrombus and   
the underlying disease. Main risk factors may be myeloproliferative diseases   
requiring splenectomy and splenomegaly, but PVT may occur after splenectomy   
for any clinical indication. The extent to which laparoscopy is responsible   
for PVT remains unclear. Laparoscopic surgeons should be aware of the risk   
of PVT, and it should be suspected in cases with an atypical outcome after   
laparoscopic splenectomy. Once diagnosed, prompt anticoagulation therapy may   
resolve the thrombotic event. © 2008 SAGE Publications.  
  
RECORD 691  
Budd-Chiari syndrome - From diagnosis to treatment - case reports  
Kozielewicz D. Smukalska E. Dybowska D.  
Polski Merkuriusz Lekarski (2008) 24:141 (260-264). Date of Publication:   
2008  
  
Budd-Chiari syndrome is a rare disease, caused by obstruction of the hepatic   
venous outflow, at the level of either the large hepatic veins or (and) the   
subdiaphragm segment of the inferior vena cava. The hematological disorders   
(myeloproliferative disorders, factor V Leiden deficiency), tumor and   
chronic inflammatory diseases are the most frequent causes of BCS in Europe   
and North America. Two cases of BCS, recognized in 24 and 43 years old   
females with subacute and chronic forms of the disease are presented in this   
article. The underlying cause was polycythemia rubra vera and   
osteomyelofibrosis. In first case, except causal and anticoagulation   
therapy, a transjugular intrahepatic portosystemic stent has been performed.   
In the second one low - sodium diet and diuretic for the control of ascites   
and oedemas were used. Hydroxycarbamid was the first choice line medication   
in treatment hematological disorders and acenocumarol in the prevention of   
the trombotic complications.  
  
RECORD 692  
Management of portal vein thrombosis  
Boyer T.D.  
Gastroenterology and Hepatology (2008) 4:10 (699-700). Date of Publication:   
October 2008  
  
RECORD 693  
Amputation of Digits or Limbs in Patients with Antiphospholipid Syndrome  
Asherson R.A. Cervera R. Klumb E. Stojanovic L. Sarzi-Puttini P. Yinh J.   
Bucciarelli S. Espinosa G. Levy R. Shoenfeld Y.  
Seminars in Arthritis and Rheumatism (2008) 38:2 (124-131). Date of   
Publication: October 2008  
  
Objective: To describe the characteristics of patients with peripheral   
vascular disease leading to amputation of digits or limbs encountered in   
patients with the antiphospholipid syndrome (APS). Methods: Twenty-one cases   
derived from several geographical centers (Brazil, Serbia, Italy, Israel,   
United Kingdom, and South Africa) are presented. The major clinical,   
serological, and histopathological data (where available) of this cohort are   
described, documented, and analyzed. Results: Patients were suffering mainly   
from systemic lupus erythematosus (9 patients) or primary APS (8 patients).   
Peripheral vascular occlusions occurred during the course of the   
catastrophic APS in 5 patients. The vascular occlusions occurred both early   
and very late in the course of the disease (time after APS diagnosis, 0-38   
years). Vasculitis was present in 7 patients and 5 demonstrated the typical   
antiphospholipid antibody (aPL)-vasculopathy with complicating bland   
thrombosis. Myocardial infarctions had occurred in 4 patients but it was not   
possible to determine whether they suffered from premature atherosclerotic   
disease or whether the infarctions were aPL-related. The appearance of   
livedo reticularis preceding the arterial thrombosis was noted in 9   
patients. Cryoglobulinemia was detected in only 1 patient. Conclusions:   
Peripheral vascular disease leading to amputation of digits or limbs is a   
severe complication encountered in patients with APS. In the absence of   
histopathology, it may be difficult to distinguish whether concomitant   
atherosclerotic occlusions, vasculitis, or aPL-related thrombosis of   
peripheral vessels is the main cause of the vascular ischemia. Treatment   
should, therefore, include full anticoagulation as well as corticosteroids   
and immunosuppression in these patients. © 2008 Elsevier Inc. All rights   
reserved.  
  
RECORD 694  
Portal and mesenteric vein thromboses in a patient with prothrombin G20210   
mutation, elevated lipoprotein (a), and high factor VIII  
Jana H. Vaclav L. Hynek M. Zdenek C. Vladislav T.  
Clinical and Applied Thrombosis/Hemostasis (2008) 14:4 (481-485). Date of   
Publication: October 2008  
  
A 65-year-old man was examined for abdominal pain. Portal and mesenteric   
vein thromboses were described by ultrasound and computed tomography. No   
local cause was found. The patient had a positive history of venous   
thromboembolism. Thrombophilia workup revealed prothrombin G20210A mutation   
(heterozygous), C677T mutation of methylenetetrahydrofolate reductase gene   
(homozygous), elevated level of lipoprotein (a), and high level of   
coagulation factor VIII. anticoagulation was started and planned for a   
long-term duration. The etiology of portal vein thrombosis is often   
multifactorial, with various combinations of systemic factors (inherited or   
acquired prothrombotic conditions) and local precipitating factors   
(inflammation, injury to the portal venous system, cancer of the abdominal   
organs, cirrhosis). The reported prevalence of hypercoagulable states in   
patients with portal vein thrombosis has been very heterogeneous so far.   
Some authors support a role of the prothrombin G20210A mutation. In the   
reported patient, this mutation was revealed in a combination with other   
hypercoagulable states. © 2008 Sage Publications.  
  
RECORD 695  
portal vein thrombosis following laparoscopic total mesorectal excision:   
Case report  
Vadalà S. Cinardi N. Li Volti G. Foresta G. Giannone G.  
Techniques in Coloproctology (2008) 12:3 (259-261). Date of Publication:   
September 2008  
  
Data continue to grow regarding the safety and technical feasibility of   
laparoscopically assisted total mesorectal excision (TME). As this minimally   
invasive alternative to open colonic resection becomes more popular, it is   
inevitable that information on the benefits and complications associated   
with it will continue to expand. portal vein thrombosis (PVT) has been   
reported after a variety of laparoscopic procedures. We report a case of   
superior mesenteric, splenic and portal vein thrombosis following   
laparoscopically assisted TME. To our knowledge, this complication of   
laparoscopic TME has not been previously reported in the literature. PVT   
should be ruled out in patients who present with vague abdominal symptoms.   
The course of this complication, while potentially devastating, is usually   
benign and responds well to lysis and/or anticoagulation. © 2008   
Springer-Verlag.  
  
RECORD 696  
Recent portal and mesenteric venous thrombosis associated with Fusobacterium   
bacteremia  
Hamidi K. Pauwels A. Bingen M. Simo A.C. Medini A. Jarjous N. Delafolie A.   
Barraud D.  
Gastroenterologie Clinique et Biologique (2008) 32:8-9 (734-739). Date of   
Publication: August/September 2008  
  
Septic pylephlebitis is usually a complication of intraabdominal infection   
in the region drained by the portal venous system. We report two cases of   
portal and mesenteric venous thrombosis associated with   
Fusobacterium necrophorum bacteremia, which did not show any obvious   
intra-abdominal source of infection with noninvasive imaging procedures. In   
one case, early anticoagulation treatment was associated with repermeation   
of the portal vein and its right branch. As in Bacteroides bacteremia,   
portal and/or mesenteric venous thrombosis should be searched for in case of   
Fusobacterium bacteremia of unknown origin. Repermeation of the portal vein   
and relief of extrahepatic portal hypertension can be achieved in these   
cases with early anticoagulation. © 2008 Elsevier Masson SAS. All rights   
reserved.  
  
RECORD 697  
Extrahepatic portal vein thrombosis  
Garcia-Pagán J.C. Hernández-Guerra M. Bosch J.  
Seminars in Liver Disease (2008) 28:3 (282-292). Date of Publication: August   
2008  
Vascular Diseases of the Liver, Book Series Title:  
  
Noncirrhotic, nontumoral portal vein thrombosis (PVT) is the second   
most-frequent cause of portal hypertension in the world. General   
thrombophilic factors can be identified in approximately 60% of patients.   
PVT may manifest as an acute process. However, the acute episode more   
frequently is asymptomatic or paucisymptomatic and portal vein thrombosis is   
misdiagnosed until the development of complications secondary to portal   
hypertension, such as variceal bleeding or portal biliopathy. Although no   
randomized controlled trials have been performed, after the diagnosis of   
acute PVT early initiation of anticoagulation (within 30 days of the onset   
of symptoms) is recommended to achieve recanalization. In patients with   
portal cavernoma, anticoagulation is aimed to prevent the progression and   
recurrence of thrombosis. Because of the lack of data in this specific   
population, variceal bleeding is managed as in cirrhotic patients.   
Ursodeoxycholic acid has been proposed empirically for the treatment of   
patients with symptomatic portal biliopathy. Choledocholithiasis might be   
present, complicating a bile duct stenosis. Accordingly, an endoscopic   
retrograde cholangiopancreatography with sphincterotomy, extraction with   
balloon catheter, and stent placement is indicated. Mortality among patients   
with PVT is low (5-year mortality rate of 5 to 10%) and is mainly related to   
associated diseases rather than to complications of portal hypertension.   
Copyright © 2008 by Thieme Medical Publishers, Inc.  
  
RECORD 698  
Endoscopic band ligation of esophageal varices in patients on   
anticoagulation  
Bajaj J.S. Franco J.  
Journal of Clinical Gastroenterology (2008) 42:7 (782-785). Date of   
Publication: August 2008  
  
Endoscopic band ligation is an effective technique for primary and secondary   
prevention of gastro-esophageal variceal bleeding (GEVB), but can also   
result in rebleeding from postbanding ulcers. Its use in primary and   
secondary prevention of GEVB in anticoagulated patients has not been   
systematically studied. The aim of the study was to evaluate the feasibility   
of band ligation in primary and secondary prevention of GEVB in   
anticoagulated patients. Five patients (age 60.2±7.3 SD years: 3 males, 2   
females) with esophageal varices on anticoagulation were studied using a   
retrospective chart review in a tertiary hospital setting. Patients were on   
mandatory anticoagulation with warfarin (international normalized ratio >2),   
on nonselective β-blocker therapy if tolerated and were not transvenous   
intrahepatic porto-systemic shunting candidates. One patient had   
polycythemia vera (noncirrhotic), the rest were cirrhotics Child class B/C   
(1 cardiogenic, 1 primary sclerosing cholangitis, 1 Budd-Chiari, and 1   
cryptogenic cirrhosis). Two patients had experienced prior acute GEVB; band   
ligation performed during acute bleeding was not included in the study. All   
patients had at least grade III-IV esophageal varices on outpatient   
follow-up for band ligation. Three bands were placed/patient and study   
patients underwent 3 banding sessions on an average. None of the patients   
developed GEVB after band ligation. In 3 patients banding resulted in   
complete variceal eradication, the remaining 2 are still being followed-up   
for outpatient band ligation. In conclusion, this case series suggests that   
endoscopic band ligation can potentially be used in anticoagulated patients   
without alternatives for prevention of acute GEVB. © 2008 by Lippincott   
Williams & Wilkins.  
  
RECORD 699  
Hepatic vascular involvement related to pregnancy, oral contraceptives, and   
estrogen replacement therapy  
Perarnau J.-M. Bacq Y.  
Seminars in Liver Disease (2008) 28:3 (315-327). Date of Publication: August   
2008  
Vascular Diseases of the Liver, Book Series Title:  
  
Both pregnancy and oral contraception (mainly when estrogen is included) may   
precipitate the development of Budd-Chiari syndrome in patients with   
underlying thrombophilia. By contrast, there is little evidence for such a   
role of pregnancy and oral contraception in women with portal vein   
thrombosis. In pregnant women, special modalities for anticoagulation are   
required, whereas the management of portal hypertension can be similar to   
that recommended in other diseases and settings. Hereditary hemorrhagic   
telangiectasia may deteriorate during pregnancy and improve after delivery.   
Hepatic sinusoidal dilatation and hepatic peliosis are classic complications   
of long-term use of oral contraceptives. The impact of pregnancy or oral   
contraceptives on the natural history on hemangioma and focal nodular   
hyperplasia appears to be limited. Preeclampsia, a liver disease unique to   
pregnancy, may be complicated by life-threatening liver vascular   
involvement, especially when the syndrome of hemolysis, elevated liver   
enzymes, and low platelet count (HELLP syndrome) is present. Copyright ©   
2008 by Thieme Medical Publishers, Inc.  
  
RECORD 700  
Percutaneous treatment of portal vein thrombosis in a child who has   
undergone splenectomy  
Oǧuzkurt P. Tercan F. Ince E. Ezer S.S. Hiçsönmez A.  
Journal of Pediatric Surgery (2008) 43:8 (e29-e32). Date of Publication:   
August 2008  
  
Thrombosis of the portal venous system is a well-recognized and potentially   
lethal complication after open or laparoscopic splenectomy. A 7-year-old   
girl with idiopathic thrombocytopenic purpura developed a portal vein   
thrombosis after open splenectomy. The portal vein thrombosis was diagnosed   
by color Doppler sonography. A percutaneous transhepatic thromboaspiration   
of the acute thrombus was done on the third postoperative day.   
anticoagulation was continued for 6 months. The presented patient is the   
youngest patient to undergo percutaneous thromboaspiration of an acute   
thrombus via the transhepatic route. Percutaneous thromboaspiration via the   
transhepatic route is an effective means of treating a portal vein   
thrombosis. © 2008 Elsevier Inc. All rights reserved.  
  
RECORD 701  
Septic thrombophlebitis of the porto-mesenteric veins as a complication of   
acute appendicitis  
Chang Y.S. Min S.Y. Joo S.H. Lee S.-H.  
World Journal of Gastroenterology (2008) 14:28 (4580-4582). Date of   
Publication: 28 Jul 2008  
  
Pylephlebitis, a rare complication of acute appendicitis, is defined as   
thrombophlebitis of the portal venous system. Pylephlebitis usually occurs   
due to secondary infection in the region drained into the portal system. We   
report a case of pylephlebitis caused by acute appendicitis. The patient was   
transferred from a private clinic 1 wk after appendectomy with the chief   
complaints of high fever and abdominal pain. He was diagnosed with   
pylephlebitis of the portal vein and superior mesenteric vein by CT-scan.   
The patient was treated with antibiotics and anticoagulation therapy, and   
discharged on the 25th day and follow-up CT scan showed a cavernous   
transformation of portal thrombosis. © 2008 The WJG Press. All rights   
reserved.  
  
RECORD 702  
Extensive portocava thrombosis revealing a primary antiphospholipid   
syndrome: A case report  
Belkahla N. Maamouri N. Ouerghi H. Cheikh I. Hamida S.B. Bouzid H. Ammar   
A.B.  
Revue de Medecine Interne (2008) 29:6 (504-507). Date of Publication: June   
2008  
  
We report a 20-year-old woman who presented with a massive portal thrombosis   
that rapidly extended to the superior and inferior vein cava system causing   
an acute Budd-Chiari syndrome. The investigations concluded to a primary   
antiphospholipid syndrome without any other prothrombotic factors. The   
outcome was fatal, 18 months later, despite anticoagulation, with   
hepatorenal syndrome and severe liver failure. © 2008 Elsevier Masson SAS.   
All rights reserved.  
  
RECORD 703  
Etiology and portal vein thrombosis in Budd-Chiari syndrome  
Uskudar O. Akdogan M. Sasmaz N. Yilmaz S. Tola M. Sahin B.  
World Journal of Gastroenterology (2008) 14:18 (2858-2862). Date of   
Publication: 14 May 2008  
  
Aim: To research the etiology, portal vein thrombosis and other features of   
Budd-Chiari syndrome (BCS) patients prospectively. Methods: A total of 75   
patients (40 female, 35 male) who were diagnosed between January 2002 and   
July 2004 as having BCS were studied prospectively. Findings from on   
physical examination, ultrasonography, duplex ultrasonography and venography   
were analyzed. Hemogram and blood chemistry were studied at the time of   
diagnosis and on each hospital visit. Bone marrow examination and immune   
phenotyping were performed by a hematologist when necessary. Protein C, S,   
antithrombin III, activated protein C resistance, and anticardiolipin   
antibodies, antinuclear antibodies, and anti ds-DNA were studied twice. The   
presence of ascite, esophageal varices, and portal thrombosis were evaluated   
at admission and on every visit. Results: At least one etiological factor   
was determined in 54 (72%) of the patients. The etiology could not be   
defined in 21 (28%) patients. One etiological factor was found in 39, 2   
factors in 14 and 3 factors in 1 patient. The most common cause was the web   
(16%), the second was Hydatid disease (11%), the third was Behcet's disease   
(9%). portal vein thrombosis was present in 11 patients and at least one   
etiology was identified in 9 of them (82%). Conclusion: Behcet's disease and   
hydatid disease are more prominent etiological factors in Turkey than in   
other countries. Patients with web have an excellent response to treatment   
without signs of portal vein thrombosis while patients having thrombofilic   
factors more than one are prone to develop portal vein thrombosis with worse   
clinical outcome. © 2008 WJG. All rights reserved.  
  
RECORD 704  
portal vein thrombosis: An unexpected finding in a 28-year-old male with   
abdominal pain  
Ferguson J.L. Hennion D.R.  
Journal of the American Board of Family Medicine (2008) 21:3 (237-243). Date   
of Publication: May/June 2008  
  
Background: Abdominal pain is a common primary care complaint. Portal vein   
thrombosis (PVT) is a rare cause of abdominal pain, typically associated   
with cirrhosis or thrombophilia. The following describes the presentation of   
PVT in a young male, the search for risk factors and underlying etiology,   
and the debate of anticoagulation therapy. Case: A 28-year-old male   
presented with periumbilical pain, post-prandial nausea, and sporadic   
hematemesis for 3 weeks. The diagnosis was confirmed with a triphasic liver   
computerized tomography after obtaining an abnormal right upper quadrant   
ultrasound. This unexpected finding prompted investigation for intrinsic   
hepatic disease and potential hypercoagulable disorders. Laboratory analysis   
revealed a heterozygous genotype for the prothrombin 20210G/A mutation, an   
identified risk factor for venous thrombosis. Discussion: Recommendations   
concerning anticoagulation for PVT in the absence of cirrhosis are not   
clearly defined. Current literature describes the following factors as   
indications for anticoagulation: acute thrombus, lack of cavernous   
transformation, absence of esophageal varices, and mesenteric venous   
thrombosis. This patient had clinical indications both for and against   
anticoagulation. Weighing this individual's clinical circumstances, we   
concluded the risk of thrombus in the setting of a hypercoagulable disorder   
outweighed the risk of variceal bleeding. A minimum of 6 months of   
anticoagulation was initiated. Conclusion: PVT is an uncommon cause of   
abdominal pain, and the absence of hepatic disease should raise the index of   
suspicion for an underlying thrombophilia. Specific recommendations for   
anticoagulation are not well defined, demonstrating the importance of   
weighing the individual risks and benefits in treatment with anticoagulation   
for young persons with thrombophilia.  
  
RECORD 705  
A rare incidental finding in a case of painless jaundice  
Patel S.N. Baumann B.M. Farmer M.C.  
American Journal of Emergency Medicine (2008) 26:4 (516.e1-516.e2). Date of   
Publication: May 2008  
  
RECORD 706  
portal vein thrombosis after laparoscopic splenectomy for systemic   
mastocytosis: A case report and review of the literature  
Maalouf M. Papasavas P. Goitein D. Caushaj P.F. Gagne D.  
Surgical Laparoscopy, Endoscopy and Percutaneous Techniques (2008) 18:2   
(219-221). Date of Publication: April 2008  
  
INTRODUCTION: Laparoscopic splenectomy has become the surgical procedure of   
choice for various diseases of the spleen. portal vein thrombosis (PVT)   
after splenectomy occurs in 0.5% to 22% of patients. Symptoms are   
nonspecific and include fever, abdominal pain, and epigastric distress. Risk   
factors for PVT after splenectomy include underlying hematologic disorders,   
massive splenectomy, and other hypercoagulable states. METHODS: We describe   
a case of PVT in a woman who underwent laparoscopic splenectomy for   
symptomatic splenomegaly secondary to systemic mastocytosis. The patient was   
discharged from the hospital without anticoagulation and experienced   
nonspecific symptoms beginning 10 days postoperatively. Diagnosis of PVT was   
made by contrast-enhanced abdominal computed tomography. The patient had no   
underlying risk factors. anticoagulation treatment facilitated   
recanalization of the portal vein and this was verified by Doppler   
ultrasound at follow-up. CONCLUSIONS: PVT after laparoscopic splenectomy is   
not uncommon. Signs and symptoms are vague and require a high index of   
suspicion for timely diagnosis. anticoagulation is the treatment of choice   
and allows recanalization of the portal system in the majority of cases. ©   
2008 Lippincott Williams & Wilkins, Inc.  
  
RECORD 707  
Thrombosis and anticoagulation in liver disease  
Valla D.C.  
Hepatology (2008) 47:4 (1384-1393). Date of Publication: April 2008  
  
RECORD 708  
A 17-year-old girl with fever, chills, rib, and pelvic pain  
Listernick R. Shulman S.T. Brown J. Strople J. Donaldson J. Mack K. Chin T.   
Klein-Gitelman M.  
Pediatric Annals (2008) 37:3 (123-126). Date of Publication: March 2008  
  
RECORD 709  
Changing spectrum of Budd-Chiari syndrome in India with special reference to   
non-surgical treatment  
Amarapurkar D.N. Punamiya S.J. Patel N.D.  
World Journal of Gastroenterology (2008) 14:2 (278-285). Date of   
Publication: 14 Jan 2008  
  
Aim: To evaluate patterns of obstruction, etiological spectrum and   
non-surgical treatment in patients with Budd-Chiari syndrome in India.   
Methods: Forty-nine consecutive cases of Budd-Chiari syndrome (BCS) were   
prospectively evaluated. All patients with refractory ascites or   
deteriorating liver function were, depending on morphology of inferior vena   
cava (IVC) and/or hepatic vein (HV) obstruction, triaged for radiological   
intervention, in addition to anticoagulation therapy. Asymptomatic patients,   
patients with diuretic-responsive ascites and stable liver function, and   
patients unwilling for surgical intervention were treated symptomatically   
with anticoagulation. Results: Mean duration of symptoms was 41.5 ± 11.2   
(range = 1-240) mo. HV thrombosis (HVT) was present in 29 (59.1%), IVC   
thrombosis in eight (16.3%), membranous obstruction of IVC in two (4%) and   
both IVC-HV thrombosis in 10 (20.4%) cases. Of 35 cases tested for   
hypercoagulability, 27 (77.1%) were positive for one or more hypercoagulable   
states. Radiological intervention was technically successful in 37/38   
(97.3%): IVC stenting in seven (18.9%), IVC balloon angioplasty in two   
(5.4%), combined IVC-HV stenting in two (5.4%), HV stenting in 11 (29.7%),   
transjugular intrahepatic portosystemic shunt (TIPS) in 13 (35.1%) and   
combined TIPS-IVC stenting in two (5.4%). Complications encountered in   
follow-up: death in five, re-stenosis of the stent in five (17.1%), hepatic   
encephalopathy in two and hepatocellular carcinoma in one patient. Of nine   
patients treated medically, two showed complete resolution of HVT.   
Conclusion: In our series, HVT was the predominant cause of BCS. In the last   
five years with the availability of sophisticated tests for   
hypercoagulability, etiologies were defined in 85.7% of cases. Non-surgical   
management was successful in most cases. © 2008 WJG. All rights reserved.  
  
RECORD 710  
Review article: The management of non-cirrhotic non-malignant portal vein   
thrombosis and concurrent portal hypertension in adults  
Spaander V.M.C.W. Van Buuren H.R. Janssen H.L.A.  
Alimentary Pharmacology and Therapeutics (2007) 26:SUPPL. 2 (203-209). Date   
of Publication: December 2007  
  
Background: Extrahepatic portal vein thrombosis is an important cause of   
non-cirrhotic portal hypertension. Aim: To provide an update on recent   
advances in the aetiology and management of acute and chronic non-cirrhotic   
non-malignant extrahepatic portal vein thrombosis. Method: A PubMed search   
was performed to identify relevant literature using search terms including   
'portal vein thrombosis', 'variceal bleeding' and 'portal biliopathy'.   
Results: Myeloproliferative disease is the most common risk factor in   
patients with non-cirrhotic non-malignant extrahepatic portal vein   
thrombosis. anticoagulation therapy for at least 3 months is indicated in   
patients with acute extrahepatic portal vein thrombosis. However, in   
patients with extrahepatic portal vein thrombosis due to a prothrombotic   
disorder, permanent anticoagulation therapy can be considered. The most   
important complication of extrahepatic portal vein thrombosis is   
oesophagogastric variceal bleeding. Endoscopic treatment is the first-line   
treatment for variceal bleeding. In several of the patients with   
extrahepatic portal vein thrombosis biliopathy changes on endoscopic   
retrograde cholangiography (ERCP) have been reported. Dependent on the   
persistence of the biliary obstruction, treatment can vary from ERCP to   
hepaticojejunostomy. Conclusion: Prothrombotic disorders are the major   
causes of non-cirrhotic, non-malignant extrahepatic portal vein thrombosis   
and anticoagulation therapy is warranted in these patients. The prognosis of   
patients with non-cirrhotic, non-malignant extrahepatic portal vein   
thrombosis is good, and is not determined by portal hypertension   
complications but mainly by the underlying cause of thrombosis. © 2007 The   
Authors.  
  
RECORD 711  
Coagulation abnormalities in cirrhotic patients with portal vein thrombosis  
Amitrano L. Guardascione M.A. Ames P.R.J.  
Clinical Laboratory (2007) 53:11-12 (583-589). Date of Publication: 2007  
  
The liver has a central role in the clotting process and an altered   
haemostasis is common in advanced liver disease. Nevertheless, recent   
studies have questioned the historical belief that impaired haemostasis in   
liver disease means an increased risk of bleeding. Coagulation and   
anticoagulation mechanisms are still balanced but are set at a lower level.   
Platelet function and number also play a role. The prevalence of thrombotic   
events is similar in both cirrhotic patients and in the general population   
but the cirrhotic patients have an increased risk for thrombosis in the   
splanchnic area. Portal blood flow stasis is the main underlying change   
favouring thrombosis even if other local, systemic, congenital and acquired   
factors are present. The onset of portal vein thrombosis strongly affects   
the prognosis of liver cirrhosis, worsening both portal hypertension and   
liver function. Some of the known risk factors for venous thrombosis -   
G20210A mutation of prothrombin, factor V Leiden, endoscopic treatment of   
esophageal varices and abdominal surgery - have a specific role in the   
development of splanchnic thrombosis in cirrhotic patients. The knowledge of   
the pathophysiological aspects of portal vein thrombosis and clotting   
alterations in liver disease will allow determination of the indication,   
duration and timing of anticoagulation therapy.  
  
RECORD 712  
portal vein thrombosis  
Rodriguez-Luna H. Vargas H.E.  
Current Treatment Options in Gastroenterology (2007) 10:6 (435-443). Date of   
Publication: December 2007  
  
portal vein thrombosis (PVT) can be a difficult clinical problem to assess   
and manage. A high index of suspicion is needed for a PVT diagnosis given   
the subtle presentation and potentially serious long-term complications. It   
should be considered a clue to the presence of one or several underlying   
disorders, including prothrombotic disorders, whether or not a local   
precipitating factor is identified. The accruing evidence shows that acute   
PVT can and probably should be treated with anticoagulation or thrombolytic   
agents in an effort to prevent extension of thrombus, mesenteric vessel   
occlusion, and portal hypertension. However, chronic PVT should be treated   
conservatively with measures to control major consequences related to portal   
hypertension. anticoagulation therapy duration should be tailored to the   
identified predisposing factors. Copyright © 2007 by Current Medicine Group   
LLC.  
  
RECORD 713  
Use of splenic artery embolization to relieve tense ascites following liver   
transplantation in a patient with paroxysmal nocturnal hemoglobinuria  
Chang C.Y. Singal A.K. Ganeshan S.V. Schiano T.D. Lookstein R. Emre S.  
Liver Transplantation (2007) 13:11 (1532-1537). Date of Publication:   
November 2007  
  
Recurrent venous thrombosis following liver transplantation for Budd-Chiari   
syndrome is common, particularly in the setting of an underlying   
myeloproliferative disorder. We describe a patient who developed refractory   
ascites due to portal vein thrombosis following liver transplantation for   
Budd-Chiari syndrome in the setting of paroxysmal nocturnal hemoglobinuria.   
Extensive portal vein thrombosis, dense abdominal adhesions, and   
portosystemic collaterals precluded the use of a transjugular intrahepatic   
portosystemic shunt or surgical portosystemic shunt to manage the patient's   
ascites. Splenic artery embolization to decrease portal hypertension was   
performed, and this resulted in complete resolution of ascites. This case   
demonstrates the successful use of splenic artery embolization to manage   
ascites due to portal vein thrombosis following liver transplantation.   
Splenic artery embolization may be considered as an alternative option for   
the management of refractory ascites due to portal hypertension in patients   
who are unable to undergo safe transjugular intrahepatic portosystemic shunt   
or surgical shunt placement. © 2007 AASLD.  
  
RECORD 714  
JAK2(V617F) positive early stage myeloproliferative disease (essential   
thrombocythemia) as the cause of portal vein thrombosis in two middle-aged   
women: Therapeutic implications in view of the literature  
Michiels J.J. Commandeur S. Hoogenboom G.J. Wegman J.J. Scholten L. Rijssel   
R.H. De Raeve H.  
Annals of Hematology (2007) 86:11 (793-800). Date of Publication: November   
2007  
  
The present study describes portal vein thrombosis (PVT) in two women as the   
first and single presenting symptom of latent or masked myeloproliferative   
disease (MPD). Essential thrombocythemia (ET) was suspected by a sustained   
increase in platelet count (>400×10(9)/l) and slight splenomegaly on   
echogram. ET could be diagnosed by the presence of large platelet in   
peripheral blood smear, an increase in clustered large megakaryocytes in   
bone marrow smear and the presence of the JAK2(V617F) mutation. A subsequent   
biopsy specimen was consistent with the diagnosis of true ET. In patients   
with a first episode of splanchnic vein thrombosis (SVT), analysis of any   
venous thrombophilic risk factors as well as a JAK2(V617F) mutation status   
indicative for MPD is warranted. Administration of heparin followed by oral   
anticoagulation with vitamin K antagonists is the treatment of choice in   
patients with SVT. anticoagulation therapy combined with low-dose aspirin   
and proper treatment of the MPD is recommended in patients with SVT   
associated with the JAK2(V617F) mutation. © Springer-Verlag 2007.  
  
RECORD 715  
Prognostic factors in noncirrhotic patients with splanchnic vein thromboses  
Amitrano L. Guardascione M.A. Scaglione M. Pezzullo L. Sangiuliano N.   
Armellino M.F. Manguso F. Margaglione M. Ames P.R.J. Iannaccone L. Grandone   
E. Romano L. Balzano A.  
American Journal of Gastroenterology (2007) 102:11 (2464-2470). Date of   
Publication: November 2007  
  
OBJECTIVES AND METHODS: Splanchnic vein thrombosis (SVT), not associated   
with cancer or liver cirrhosis, is a rare event and scanty data are   
available on its natural history, long-term prognosis, and treatment. In   
this study 121 SVT patients consecutively seen from January 1998 to December   
2005 were included and 95 of them were followed up for a median time of 41   
months. Screening for thrombophilic factors was performed in 104 patients.   
New thrombotic or bleeding episodes were registered and anticoagulant   
therapy was performed according to preestablished criteria. RESULTS: SVT was   
an incidental finding in 34 (28.1%) patients; 34 (28.1%) presented with   
abdominal infarction; 39 (32.2%) had bowel ischemia or acute portal vein   
thrombosis; 14 (11.6%) had bleeding from portal hypertensive sources.   
Survival rates at 1, 3, and 7 yr were 95%, 93.3%, and 89.6%, respectively;   
87.5% of deaths occurred at onset of SVT as complications of intestinal   
infarction. Patients with isolated portal vein thromboses had symptoms and   
intestinal infarction in 16/41 (39%) and 0/41 (0%) of the cases,   
respectively, whereas superior mesenteric vein thromboses, isolated or not,   
were associated with symptoms and intestinal infarction in 69/75 (92%) and   
34/75 (45%), respectively. During the follow-up 14 (14.7%) suffered from 39   
episodes of gastrointestinal bleeding with no deaths. A previous   
gastrointestinal bleed was associated with new hemorrhagic events during   
follow-up. New venous thrombotic episodes occurred in 10 of 95 patients   
(10.5%), of which 73% were in the splanchnic area. Seven out of these 10   
patients had a chronic myeloproliferative disease (MPD) and none was on   
anticoagulation. CONCLUSIONS: Anticoagulant therapy was effective to obtain   
recanalization of acute SVT in 45.4% of patients and preserved patients from   
recurrent thrombosis when given lifelong. © 2007 by Am. Coll. of   
Gastroenterology.  
  
RECORD 716  
Portal vein thrombi after restorative proctocolectomy: Serious complication   
without long-term sequelae  
Millan M. Hull T.L. Hammel J. Remzi F.  
Diseases of the Colon and Rectum (2007) 50:10 (1540-1544). Date of   
Publication: October 2007  
  
PURPOSE: Portal vein thrombi have been observed after restorative   
proctocolectomy and ileal pouch-anal anastomosis, and present as a clinical   
spectrum of abdominal pain, fever, and leukocytosis. anticoagulation   
treatment is usually associated with resolution of symptoms. However, the   
long-term consequences and effect on pouch function are not known. The   
purpose of this study was to analyze the long-term functional outcome of   
patients with confirmed portal vein thrombi after restorative   
proctocolectomy. METHODS: A retrospective study of all patients undergoing   
restorative proctocolectomy from January 1997 to 2000 was performed. A   
case-control study was designed that matched 37 patients with confirmed   
portal vein thrombi in this period with 133 patients without portal vein   
thrombi; the groups were compared with respect to pouch function and quality   
of life by using the Global Cleveland Clinic Quality of Life Questionnaire   
for pelvic pouch patients. RESULTS: The mean follow-up was 4.73 (range,   
4.21-7.28) years. The percentage of male patients was 58.8. The most common   
diagnosis was ulcerative colitis (62.4 percent). There were no significant   
differences between portal vein thrombi patients and controls with respect   
to pouch function (number of bowel movements, urgency, incontinence),   
episodes of pouchitis, or quality of life. CONCLUSIONS: Portal vein thrombi   
can be a serious complication after restorative proctocolectomy that usually   
resolves with anticoagulation therapy. Long-term pouch function and quality   
of life are not affected. © 2007 The American Society of Colon and Rectal   
Surgeons.  
  
RECORD 717  
Postoperative complications in patients with portal vein thrombosis after   
liver transplantation: Evaluation with Doppler ultrasonography  
Jia Y.-P. Lu Q. Gong S. Ma B.-Y. Wen X.-R. Peng Y.-L. Lin L. Chen H.-Y. Qiu   
L. Luo Y.  
World Journal of Gastroenterology (2007) 13:34 (4636-4640). Date of   
Publication: 14 Sep 2007  
  
Aim: To study the postoperative complications in patients with preoperative   
portal vein thrombosis (PVT) undergoing liver transplantation (LT) and to   
evaluate the complications with Doppler ultrasonography. Methods:   
Petrospective studies were performed on 284 patients undergoing LT (286 LT)   
with respect to pre- and postoperative clinical data and Doppler   
ultrasonography. According to the presence and grade of preoperative PVT,   
286 LTs were divided into three groups: complete PVT (c-PVT), partial PVT   
(p-PVT) and non-PVT, with 22, 30 and 234 LTs, respectively. Analyses were   
carried out to compare the incidence of early postoperative complications.   
Results: PVT, inferior vena cava (IVC) thrombosis, hepatic artery thrombosis   
(HAT) and biliary complications were found postoperatively. All   
complications were detected by routine Doppler ultrasonography and diagnoses   
made by ultrasound were confirmed by clinical data or/and other imaging   
studies. Nine out of 286 LTs had postoperative PVT. The incidence of the   
c-PVT group was 22.7%, which was higher than that of the p-PVT group (3.3%,   
P < 0.05) and non-PVT group (1.3%, P < 0.005). No difference was found   
between the p-PVT and non-PVT groups (P > 0.25). Of the 9 cases with   
postoperative PVT, recanalizations were achieved in 7 cases after   
anticoagulation under the guidance of ultrasound, 1 case received portal   
vein thrombectomy and 1 case died of acute injection. Ten LTs had   
postoperative IVC thrombosis. The c-PVT group had a higher incidence of IVC   
thrombosis than the non-PVT group (9.1% vs 2.6%, P < 0.05); no significant   
difference was found between either the c-PVT and p-PVT groups (9.1% vs   
6.7%, P > 0.5) or between the p-PVT and non-PVT groups (P > 0.25). Nine   
cases with IVC thrombosis were cured by anticoagulation under the guidance   
of ultrasound, and 1 case gained natural cure without any medical treatment   
after 2 mo. HAT was found in 2 non-PVT cases, giving a rate of 0.7% among   
286 LTs. Biliary complications were seen in 12 LTs. The incidence of biliary   
complications in the c-PVT, p-PVT and non-PVT groups was 9.1%, 3.3% and   
4.3%, respectively (P > 0.25 for all), among which 2 stenosis led   
retransplantations and others were controlled by relative therapy.   
Conclusion: C-PVT patients tend to have a higher incidence of PVT and IVC   
thrombosis than non-PVT patients after LT. The incidence of postoperative   
complications in p-PVT patients does not differ from that of non-PVT   
patients. A relatively low incidence of HAT was seen in our study. Doppler   
ultrasonography is a convenient and efficient method for detecting   
posttransplant complications and plays an important role in guiding   
treatment. © 2007 WJG. All rights reserved.  
  
RECORD 718  
portal vein thrombosis; risk factors, clinical presentation and treatment  
Sogaard K.K. Astrup L.B. Vilstrup H. Gronbaek H.  
BMC Gastroenterology (2007) 7 Article Number: 34. Date of Publication: 15   
Aug 2007  
  
Background: portal vein thrombosis (PVT) is increasingly frequently being   
diagnosed, but systematic descriptions of the natural history and clinical   
handling of the condition are sparse. The aim of this retrospective study   
was to describe risk factors, clinical presentation, complications and   
treatment of portal vein thrombosis in a single-centre. Methods: Sixty-seven   
patients were identified in the electronic records from 1992 to 2005. All   
data were obtained from the patient records. Results: One or more risk   
factors (e.g. prothrombotic disorder or abdominal inflammation) were present   
in 87%. Symptoms were abdominalia, splenomegaly, fever, ascites,   
haematemesis, and weight loss. Abdominalia and fever occurred more   
frequently in patients with acute PVT. Frequent complications were   
splenomegaly, oesophageal- and gastric varices with or without bleeding,   
portal hypertensive gastropathy and ascites. Varices and bleeding were more   
frequent in patients with chronic PVT. Patients who received anticoagulant   
therapy more frequently achieved partial/complete recanalization. Patients   
with varices who were treated endoscopically in combination with β-blockade   
had regression of the varices. The overall mortality was 13% in one year,   
and was dependent on underlying causes. Conclusion: Most patients had a   
combination of local and systemic risk factors for PVT. We observed that   
partial/complete recanalization was more frequent in patients treated with   
anticoagulation therapy, and that regression of varices was more pronounced   
in patients who where treated with active endoscopy combined with   
pharmacological treatment. © 2007 Sogaard et al; licensee BioMed Central   
Ltd.  
  
RECORD 719  
portal vein thrombosis after laparoscopic splenectomy in benign hematologic   
diseases  
Ruiz-Tovar J. De Oteyza J.P. Sánchez J.B. Velardo A.A. Blanco R.R. Guirao   
M.V.C. Villanueva A.G.  
Journal of Laparoendoscopic and Advanced Surgical Techniques (2007) 17:4   
(448-454). Date of Publication: August 2007  
  
Introduction: portal vein thrombosis is an unfrequent, but potentially   
deadly, complication of the laparoscopic splenectomy procedure. The   
laparoscopic approach has shortened the duration of hospital stay; portal   
vein thrombosis may appear after the patient has left the hospital,   
determining a later diagnosis. Because of the mild, nonspecific symptoms,   
the diagnosis can even be missed and only achieved when chronic   
complications take place. Objectives: In this study, we aimed to determine   
the appearance of portal vein thrombosis in a consecutive series of patients   
who underwent laparoscopic splenectomy by performing a contrast-enhanced   
computed tomography (CT) scan postoperatively. Materials and Methods: A   
transversal study was established, performing in 2005 a contrast-enhanced CT   
scan on 20 patients who underwent laparoscopic splenectomy between 1999 and   
2005 at Ramón y Cajal University Hospital (Madrid, Spain). The presence of   
thrombosis in the splenoportomesenteric axis was investigated. Results: Two   
(2) cases (10%) of portal vein thrombosis were detected: 1 symptomatic case,   
7 days after surgery, was treated with anticoagulation, resulting in the   
disappearance of the thrombus in a new CT scan 6 months later; the second   
case was asymptomatic and was discovered during the performance of this   
study. Conclusions: The contrast-enhanced CT scan shows the best accuracy   
for the diagnosis of portal vein thrombosis, and it must be performed when   
any clinical manifestation appear; also, it must still be determined if a   
contrast-enhanced CT scan should be systematically performed in high-risk   
thromboembolic patients. An ultrasound Doppler may present many diagnostic   
errors. It is probably advisable to prolong the antithromboembolic   
prophylaxis. © Mary Ann Liebert, Inc.  
  
RECORD 720  
Superior mesenteric and portal vein thrombosis in a polycythemia vera   
patient with JAK2 mutation  
Araki N. Takimoto R. Chiba H. Araki H. Sato T. Iyama S. Hirakawa M. Ono K.   
Kawano Y. Takada K. Miyanishi K. Kobune M. Matsunaga T. Kato J. Nakamura T.   
Niitsu Y.  
[Rinshō ketsueki] The Japanese journal of clinical hematology (2007) 48:7   
(554-558). Date of Publication: Jul 2007  
  
A 47-year-old woman was admitted to our hospital in December 1994 with   
polycythemia. The patient's red blood cell volume was 33 ml/kg and bone   
marrow cytology was able to rule out other myeloproliferative diseases such   
as chronic myelogenous leukemia, essential thrombocytosis and myelofibrosis.   
The patient was diagnosed as having polycythemia vera. She had undergone   
only phlebotomy until 1999 when the thrombocytosis appeared, subsequent to   
which she was treated with oral hydroxyurea. However, in March 2006, she   
developed upper abdominal pain and was admitted to our hospital on March   
14th, 2006. Computed tomography scan revealed thromboses in the portal and   
superior mesenteric veins. anticoagulation therapy delivered intravenously   
via the superior mesenteric vein dramatically improved her symptoms and   
liver function. She is currently on anticoagulation therapy in our   
outpatient clinic.  
  
RECORD 721  
A case of portal vein thrombosis associated with congenital protein S   
deficiency  
Ishikawa A. Ito H. Hotta S. Ono S. Kakinoki N. Kishimoto Y. Kamoshida T.   
Hirai S. Oka Y.  
Japanese Journal of Gastroenterology (2007) 104:6 (822-828). Date of   
Publication: June 2007  
  
A 25-year-old man was admitted to our hospital because of abdominal pain,   
nausea and low-grade fever. An abdominal CT showed remarkable thickening of   
the wall of the small intestine and extensive thrombosis of the mesenteric,   
portal and splenic veins. Because neither intestinal infarction nor   
peritonitis was seen, anticoagulation therapy was chosen. Heparin was   
administered intravenously and was used alternatively with warfarin later.   
The patient's symptoms and clinical data improved gradually. Concerning the   
etiological factors of the thrombosis, only protein S activity definitely   
decreased. Genetic analysis indicated a variant of protein S, protein S   
Tokushima.  
  
RECORD 722  
Portal hypertension due to portal venous thrombosis: Etiology, clinical   
outcomes  
Harmanci O. Bayraktar Y.  
World Journal of Gastroenterology (2007) 13:18 (2535-2540). Date of   
Publication: 14 May 2007  
  
The thrombophilia in adult life has major implications in the hepatic   
vessels. The resulting portal vein thrombosis has various outcomes and   
complications. Esophageal varices, portal gastropathy, ascites, severe   
hypersplenism and liver failure needing liver transplantation are known   
well. The newly formed collateral venous circulation showing itself as   
pseudocholangicarcinoma sign and its possible clinical reflection as   
cholestasis are also known from a long time. The management strategies for   
these complications of portal vein thrombosis are not different from their   
counterpart which is cirrhotic portal hypertension, but the prognosis is   
unquestionably better in former cases. In this review we present and discuss   
the portal vein thrombosis, etiology and the resulting clinical pictures.   
There are controversial issues in nomenclature, management (including   
anticoagulation problems), follow up strategies and liver transplantation.   
In the light of the current knowledge, we discuss some controversial issues   
in literature and present our experience and our proposals about this group   
of patients. © 2007 The WJG Press. All rights reserved.  
  
RECORD 723  
Antiphospholipid syndrome with lupus erythematosus presenting with   
myocardial infarction  
Kaynar K. Ulusoy S. Gul S. Kilicarslan F. Oztuna F. Ahmetoglu A. Omay S.B.  
Scottish Medical Journal (2007) 52:2. Date of Publication: May 2007  
  
Arterial and venous thrombosis is a prominent feature of antiphospholipid   
syndrome together with antiphospholipid antibodies. We report generalised   
thrombosis in a 28 years old male patient with antiphospholipid syndrome   
associated with lupus erythematosis. Firstly the patient had myocardial   
infarction. Eight months later he started to complain about oedema which was   
found to be secondary to nephrotic syndrome. In his third hospital day he   
developed pulmonary emboli. Tomographic angiography revealed left renal vein   
thrombosis, portal vein thrombosis, right renal infarct, pulmonary emboli.   
His tests for antinuclear antibody, anti dsDNA antibody and antiphospholipid   
antibody IgM were positive. After anticoagulation therapy and   
immunosupressive therapy he could have been disconnected from mechanical   
ventilator and his oedema nearly disappeared. This case clearly highlights   
the fact that antiphospholipid syndrome with systemic lupus erythematosus   
can present with myocardial infarction in young patients.  
  
RECORD 724  
Superior mesenteric and portal vein thrombosis caused by congenital   
antithrombin III deficiency: Report of a case  
Shibahara K. Tatsuta K. Orita H. Yonemura T. Kohno H.  
Surgery Today (2007) 37:4 (308-310). Date of Publication: April 2007  
  
A 50-year-old man presented with a 24-h history of gradually worsening   
abdominal pain. Enhanced computed tomography showed segmental dilation of   
the small intestine, wall thickening, and ascites, as well as thrombosis of   
the superior mesenteric vein (SMV) and portal vein. Thus, an emergency   
laparotomy was performed, which revealed segmental intestinal infarction   
caused by the thrombosis in the SMV and portal vein. We resected the   
necrosed intestine and performed anastomosis. The patient was given   
intravenous heparin and nafamostat mesilate as anticoagulation therapy. The   
abdominal pain again recurred 4 days after this operation, necessitating a   
second laparotomy. Segmental congestion of the intestine was found and   
another resection was done, after which he recovered rapidly. Blood   
chemistry subsequently revealed an antithrombin III deficiency, which was   
confirmed to be inherent, after screening his family. Thus, laboratory   
testing for these proteins may help define the cause of mesenteric venous   
thrombosis. © 2007 Springer-Verlag.  
  
RECORD 725  
Prothrombin 20210 G/A defect as a cause of mesenteric venous infarction:   
Report of a case  
Karagulle E. Turk E. Gokturk H.S. Yildirim E. Moray G.  
Surgery Today (2007) 37:3 (251-253). Date of Publication: March 2007  
  
A 50-year-old man with abdominal pain, nausea, and vomiting presented at our   
emergency department. Physical examination revealed diffuse abdominal   
tenderness and absent bowel sounds. Computed tomography showed partial   
portal vein thrombosis extending to the right portal vein and the superior   
mesenteric vein, perfusion defects in the liver, and nonopacified intestinal   
segment after contrast injection. An emergency laparotomy was performed. The   
wall of the distal jejunum was edematous, congested, and a 10-cm jejunal   
segment was necrotic. A partial intestinal resection and a primary   
anastomosis were performed. Screening for thrombophilia revealed a   
heterozygote 20210 G/A mutation of the prothrombin gene. anticoagulation was   
initiated. Computed tomography 45 days after surgery showed a complete   
dissolution of the thrombi and cavernous transformation in the main portal   
vein. His subsequent clinical course was uneventful. Mesenteric venous   
thrombosis which causes an intestinal infarction is rare, and also difficult   
to diagnose. However, a prothrombin 20210 defect should be considered in the   
differential diagnosis of patients with unexplained thrombosis. © 2007   
Springer-Verlag.  
  
RECORD 726  
Phylephebitis due to diverticulitis  
Casallo Blanco S. Muñoz Ruiz A.I. Marcos Sánchez F. De Matías Salces L.   
Blanco González J. Castañeda Bergamín C.  
Anales de Medicina Interna (2006) 23:12 (593-595). Date of Publication:   
December 2006  
  
A case of a 52 year-old-male, with past medical history of renoureteral   
crisis and recurrent episodes of abdominal pain, is presented. The patient   
presented to the Emergency Department with abdominal pain (similar to   
previous episodes), fever and abnormal liver function test (marked elevation   
of gammaglutamyltranspeptidase and alkaline phosphatase). An abdominal   
ultrasound was performed showing hepatomegaly, and enlarged spleen and an   
echogenic material that suggested a thrombosis. A CT scan confirmed the   
thrombosis of the inferior mesenteric vein extending up to the splenic vein   
and the portal vein. It also showed a large number of diverticulum. Surgery   
was performed in order to rule out an acute diverticulitis. A   
phylephlebitis, infective suppurative thrombosis of the portal vein and its   
branches (inferior mesenteric vein and splenic vein) was found due to an   
acute diverticulitis with neither perforation nor abscess. A ligature of the   
inferior mesenteric vein and a Hartmann procedure with resection of the   
diseased colon, and end colostomy and creation of a rectal stump, were   
performed. A favourable outcome was obtained with antibiotics and   
anticoagulation. Some aspects of the aetiology, symptoms, diagnosis and   
treatment of this unusual complication of diverticulitis are also presented.   
Copyright © 2006 Aran Ediciones, S.L.  
  
RECORD 727  
Acute partial Budd-Chiari syndrome and portal vein thrombosis in   
cytomegalovirus primary infection: a case report.  
Spahr L. Cerny A. Morard I. Rubbia-Brandt L. Schrenzel J.  
BMC gastroenterology (2006) 6 Article Number: 10. Date of Publication: 2006  
  
Splanchnic vein thrombosis may complicate inherited thrombotic disorders.   
Acute cytomegalovirus infection is a rare cause of acquired venous   
thrombosis in the portal or mesenteric territory, but has never been   
described extending into a main hepatic vein. A 36-year-old immunocompetent   
woman presented with acute primary cytomegalovirus infection in association   
with extensive thrombosis in the portal and splenic vein. In addition, a   
fresh thrombus was evident in the right hepatic vein. A thorough evaluation   
for a hypercoagulable state was negative. The clinical course, biological   
evolution, radiological and histological findings were consistent with   
cytomegalovirus hepatitis complicated by a partial acute Budd-Chiari   
syndrome and portal thrombosis. Therapeutic anticoagulation was associated   
with a slow clinical improvement and partial vascular recanalization. We   
described in details a new association between cytomegalovirus infection and   
acute venous thrombosis both in the portal vein and in the right hepatic   
vein, realizing a partial Budd-Chiari syndrome. One should be aware that   
this rare thrombotic event may be complicated by partial venous outflow   
block.  
  
RECORD 728  
An unusual cause of ascites  
Bhattacharyya R. Mrikaria S. Abdelhafiz A.  
CME Journal Geriatric Medicine (2006) 8:1 (44-45). Date of Publication: 2006  
  
We present a case of idiopathic portal vein thrombosis in a 78 year old man   
noted to have ascites when he presented to Accident and Emergency (A&E)   
after af all. Ascites is an uncommon presentation in these cases. Other   
common signs of portal vein thrombosis (gastrointestinal haemorrhage,   
oesophageal varices, and splenomegaly) were absent probably due to early   
diagnosis. He responded fully to long term oral anticoagulation treatment.   
That we were able to detect and treat this at an early stage illustrates the   
value of comprehensive geriatric assessment (CGA).  
  
RECORD 729  
Transjugular Intrahepatic Portosystemic Shunt (TIPS), the preferred   
therapeutic option for Budd Chiari syndrome associated with portal vein   
thrombosis [3]  
Senzolo M. Cholongitas E. Davies N. Marelli L. Shusang V. Patch D. Burroughs   
A.K.  
American Journal of Gastroenterology (2006) 101:9 (2163-2164). Date of   
Publication: September 2006  
  
RECORD 730  
Nonmalignant portal vein thrombosis in adults  
Condat B. Valla D.  
Nature Clinical Practice Gastroenterology and Hepatology (2006) 3:9   
(505-515). Date of Publication: September 2006  
  
portal vein thrombosis (PVT) consists of two different entities: acute PVT   
and chronic PVT. Acute PVT usually presents as abdominal pain. When the   
thrombus extends to the mesenteric venous arches, intestinal infarction can   
occur. Chronic PVT is usually recognized after a fortuitous diagnosis of   
hypersplenism or portal hypertension, or when there are biliary symptoms   
related to portal cholangiopathy. Local risk factors for PVT, such as an   
abdominal inflammatory focus, can be identified in 30% of patients with   
acute PVT; 70% of patients with acute and chronic PVT have a general risk   
factor for PVT, most commonly myeloproliferative disease. Early initiation   
of anticoagulation therapy for acute PVT is associated with complete and   
partial success in 50% and 40% of patients, respectively. A minimum of 6   
months' anticoagulation therapy is recommended for the treatment of acute   
PVT. For patients with either form of PVT, permanent anticoagulation therapy   
should be considered if they have a permanent risk factor. In patients with   
large varices, β-adrenergic blockade or endoscopic therapy seems to prevent   
bleeding as a result of portal hypertension, even in patients on   
anticoagulation therapy. In patients with jaundice or recurrent biliary   
symptoms caused by cholangiopathy, insertion of a biliary endoprosthesis is   
the first treatment option. Overall, the long-term outcome for patients with   
PVT is good, but is jeopardized by cholangiopathy and transformation of   
underlying myeloproliferative disease into myelofibrosis or acute leukemia.  
  
RECORD 731  
Thrombolysis via an operatively placed mesenteric catheter for portal and   
superior mesenteric vein thrombosis: Report of a case  
Ozdogan M. Gurer A. Gokakin A.K. Kulacoglu H. Aydin R.  
Surgery Today (2006) 36:9 (846-848). Date of Publication: September 2006  
  
Mesenteric venous thrombosis (MVT) is a catastrophic form of mesenteric   
vascular occlusion. In the absence of peritoneal signs, anticoagulation   
therapy should be started immediately. For selected patients, thrombolysis   
through the superior mesenteric artery (SMA), jugular vein, or portal vein   
via a transhepatic route might be successful; however, exploratory   
laparotomy is mandatory when peritoneal signs develop. We report a case of   
acute MVT associated with protein C and S deficiency, treated successfully   
by limited bowel resection and simultaneous thrombolytic infusion, given via   
an operatively placed mesenteric vein catheter. © Springer 2006.  
  
RECORD 732  
Idiopathic eosinophilia associated with portal vein and massive thrombosis:   
Successful thrombolysis with streptokinase  
Villar J.M. López A.C. Macayo Sánchez A.J.  
Medical Science Monitor (2006) 12:6 (CS53-CS56). Date of Publication: 2006  
  
Background: portal vein thrombosis in adults is usually related to   
cirrhosis. There are several possible therapies. including anticoagulation,   
transjugular intrahepatic portosystemic shunt, ballon dilatation, local and   
systemic fibrinolytics agents. Hypercoagulable states are also reported in   
association with this disease entity. Eosinophilia may activate platelets   
and promote thrombosis due to proteins contained in intracytoplasmic   
granules, such as eosinophil cationic protein and major basic protein. There   
is only one paper in the medical literature linking eosinophilia and portal   
vein thrombosis. Case Report: We present here the case of a middle-age woman   
with idiopathic eosinophilia and acute portal vein thrombosis with massive   
venous thrombosis, involving the mesenteric, splenic, inferior cava, iliac   
and femoral veins, successfully treated with systemic streptokinase.   
Conclusions: Acute portal vein thrombosis with associated mesenteric and   
splenic vein thrombosis is a potentially lethal coagulation disorder that   
can be treated successfully with systemic streptokinase. © Med Sci Monit,   
2006.  
  
RECORD 733  
Subcutaneous administration of hepatitis B immune globulin in combination   
with lamivudine following orthotopic liver transplantation: Effective   
prophylaxis against recurrence  
Powell J.J. Apiratpracha W. Partovi N. Erb S.R. Scudamore C.H. Steinbrecher   
U.P. Buczkowski A.K. Chung S.W. Yoshida E.M.  
Clinical Transplantation (2006) 20:4 (524-525). Date of Publication:   
July/August 2006  
  
Prophylaxis against recurrent hepatitis B virus (HBV) infection with   
hepatitis B immune globulin (HBIG), in combination with antiviral agents   
such as lamivudine, has allowed transplantation for this condition to become   
feasible and accepted. Current protocols allow for HBIG administration   
either intravenously or intramuscularly. To date, there has been no reported   
experience with the subcutaneous route of post-transplant HBIG delivery. We   
report our experience of a 60-yr-old man for whom liver transplantation was   
performed for chronic HBV. HBIG was administered intramuscularly during the   
anhepatic phase of surgery. The finding of a portal vein thrombosis   
requiring repeated thrombectomy necessitated chronic anticoagulation.   
Post-operatively, HBIG was administered subcutaneously, in four separate   
injections, for a daily dose of 2170 IU along with continued lamivudine   
dosing. Hepatitis B surface antibody (anti-HBs) titres reached a serum   
concentration of >500 IU/L by seven d post-transplant and approximately 1000   
IU/L by nine d post-transplant. Five months post-transplant, with continued   
combination of subcutaneous HBIG and lamivudine, there has been no   
recurrentHBVinfection and anti-HBs titres have been at target levels. Our   
experience suggests that subcutaneous delivery of HBIG may be a feasible   
consideration when intramuscular/intravenous dosing is not possible. ©   
Blackwell Munksgaard, 2006.  
  
RECORD 734  
Neonatal thromboembolic emergencies  
Thornburg C. Pipe S.  
Seminars in Fetal and Neonatal Medicine (2006) 11:3 (198-206). Date of   
Publication: June 2006  
  
Thrombosis risk is multifactorial, with interaction of hereditary risk   
factors and acquired environmental and clinical conditions. Newborns are at   
particular risk for thrombotic emergencies secondary to the unique   
properties of their hemostatic system, influences of the maternal-fetal   
environment, and perinatal complications and interventions. Thrombotic   
complications range from arterial and venous catheter thrombosis to purpura   
fulminans. Prompt identification and appropriate management of thrombotic   
emergencies is critical in avoiding limb-, organ-, and life-threatening   
complications. Treatment strategies have been extrapolated from adult   
literature but clinical experience from small-scale neonatal studies has   
resulted in therapeutic guidelines, which should be individualized for each   
neonate, taking into consideration age and clinical status. © 2006 Elsevier   
Ltd. All rights reserved.  
  
RECORD 735  
portal vein thrombosis in the neonate: Risk factors, course, and outcome  
Morag I. Epelman M. Daneman A. Moineddin R. Parvez B. Shechter T. Hellmann   
J.  
Journal of Pediatrics (2006) 148:6 (735-739). Date of Publication: June 2006  
  
Objective: To determine the risk factors, clinical features, and outcome of   
infants diagnosed with portal vein thrombosis (PVT). Study design: A   
retrospective chart review was conducted of all consecutive infants admitted   
to the Hospital for Sick Children, Toronto, between January 1999 and   
December 2003 diagnosed with PVT. Results: PVT was diagnosed in 133 infants,   
all but 5 of whom were neonates, with a median age at time of diagnosis of 7   
days. An umbilical venous catheter (UVC) was inserted in 73% of the infants   
and was in an appropriate position in 46% of them. Poor outcome, defined as   
portal hypertension or lobar atrophy, was diagnosed in 27% of the infants   
and was significantly more common in those with an initial diagnosis of   
grade 3 PVT and in those with a low or intrahepatically placed UVC.   
anticoagulation treatment did not appear to have a significant effect on   
outcome. Conclusions: PVT occurs early in life; major risk factors in   
addition to the neonatal period are placement of UVC and severe neonatal   
sickness. Poor outcome is associated with an improperly placed UVC and with   
grade 3 thrombus. © 2006 Elsevier Inc. All rights reserved.  
  
RECORD 736  
Acute and chronic thromboses of the portal system  
Wolff M. Schäfer N. Schepke M. Hirner A.  
Gefasschirurgie (2006) 11:3 (188-194). Date of Publication: Jun 2006  
  
The cause of thrombosis of the portal system is multifactorial, often   
exhibiting a combined etiology of systemic thrombophilia (deficiency of   
inhibitory coagulation factors, myeloproliferative disease) and local   
factors (trauma, compression, decreased portal flow, inflammation). The   
prognosis of acute venous mesenteric thrombosis (AMT) has improved during   
the last decade due to better imaging by CT and Doppler ultrasound,   
understanding of the pathophysiology of genetic and acquired coagulation   
disorders, and more aggressive interventions to restore the patency of the   
mesenteric veins. In AMT, the extent of thrombosis and clinical conditions   
dictate whether anticoagulation alone, transhepatic lysis, interventional or   
surgical thrombectomy, or bowel resection with second-look procedures are   
appropriate treatment modalities. Chronic thrombosis of the portal system is   
characterized by a long asymptomatic latency and sequelae of portal   
hypertension, such as variceal hemorrhage, hypersplenism, pseudosclerosing   
cholangitis, or growth retardation in children. If endoscopic therapy fails   
to control variceal bleeding portosystemic shunt surgery offers an effective   
therapy which leads to freedom from recurrent bleeding and repeated   
endoscopies for many years and improves hypersplenism without deteriorating   
liver function or encephalopathy. Gastroesophageal devascularization and   
other direct variceal ablative procedures should be restricted to treat   
endoscopic therapy failures without shuntable portal tributaries. © Springer   
Medizin Verlag 2006.  
  
RECORD 737  
portal vein thrombosis and recurrent bacteremia complicating a gastric   
variceal sclerosis by embucrylate  
Trabut J.-B.  
Hepato-Gastro (2006) 13:3 (236-237). Date of Publication: May 2006  
  
RECORD 738  
Treatment of Cancer-Associated Thrombosis: Distinguishing Among   
Antithrombotic Agents  
Pruemer J.  
Seminars in Oncology (2006) 33:SUPPL. 4 (26-39). Date of Publication: April   
2006  
  
The risk of cancer-associated thrombosis can be substantial, depending on   
tumor type, extent of cancer, and type of treatment. Unfractionated heparin   
and warfarin have been used in the prevention of cancer-associated   
thrombosis, but low-molecular-weight heparin (LMWH) is widely used for the   
prevention of venous thromboembolism in high-risk patients. Long-term   
management with warfarin is associated with close monitoring, an increased   
risk of drug interactions, and bleeding. LMWHs may offer an alternative   
outpatient treatment strategy for prophylactic treatment because of their   
simpler dosing, more predictable anticoagulant activity, and improved safety   
profile. Clinical trials examining the treatment of venous thromboembolism   
with LMWH in patients with cancer suggest a survival advantage for the   
treated groups. Subtle differences in the pharmacokinetics of available   
LMWHs exist, and each LMWH should be regarded as a distinct drug.   
Pharmacists should be aware of the US Food and Drug Administration-approved   
uses for each LMWH, dosing options, and the advantages and disadvantages of   
available delivery systems for various patient populations. Pharmacists can   
play a major role in educating patients and other health care professionals   
on risk factor recognition, patient risk stratification, and proper agent   
selection for prevention and treatment of cancer-associated thrombosis. ©   
2006 Elsevier Inc. All rights reserved.  
  
RECORD 739  
A case of primary superior mesenteric and portal venous thrombosis performed   
a second-look operation  
Makino S. Kawachi Y. Shimizu T. Nishimura A. Nikkuni K. Shimizu T.   
Hatakeyama K.  
Japanese Journal of Gastroenterological Surgery (2006) 39:4 (492-497). Date   
of Publication: Apr 2006  
  
A 49-year-old man referred for severe abdominal pain after about 10 days   
from the onsets of slight abdominal pain to have superior mesenteric and   
portal venous thrombosis was found in abdominal computed tomography (CT)   
necessitating emergency laparotomy. Despite partial resection of the jejunum   
of about 200cm long including the necrotic part and thrombectomy for the   
superior mesenteric and portal vein was performed, a thrombus remained in   
the superior mesenteric vein (SMV) and portal vein (PV) and extended to the   
peripheral veins of the surrounding mesenteruim. We completed surgery   
without closing the abdominal wound and started anticoagulation therapy with   
continuous heparin administration. About 12 hours after primary surgery, a   
second-look operation showed no appearance of new necrotic lesions. Six   
weeks after surgery CT showed the thrombus in SMV and PV had disappeared.   
The patient in now being followed up by administration of oral   
anticoagulation therapy for warfarin and has had no recurrence of SMV or PV   
thrombosis. In such cases, a second-look operation may help to minimize the   
amount of the segment resected in the small intestine. ©2006 The Japanese   
Society of Gastroenterological Surgery.  
  
RECORD 740  
Acute portal and mesenteric thrombosis: Unusual presentation of   
cytomegalovirus infection  
Amitrano L. Guardascione M.A. Scaglione M. Menchise A. Romano L. Balzano A.  
European Journal of Gastroenterology and Hepatology (2006) 18:4 (443-445).   
Date of Publication: April 2006  
  
Cytomegalovirus infection is a benign disease in immunocompetent patients.   
In-vitro and in-vivo studies show that cytomegalovirus may cause arterial   
and venous thrombosis through different mechanisms. We describe two cases of   
acute cytomegalovirus infection complicated by portal and mesenteric vein   
thrombosis leading to intestinal ischemia. Both patients carried the   
heterozygous prothrombin G20210A mutation. The presence of this unusual   
complication should be searched for in patients with acute cytomegalovirus   
infection and abdominal symptoms in order to start early anticoagulation.   
The necessity for full thrombophilic screening is also pointed out. © 2006   
Lippincott Williams & Wilkins.  
  
RECORD 741  
Acute partial Budd-Chiari syndrome and portal vein thrombosis in   
cytomegalovirus primary infection: A case report  
Spahr L. Cerny A. Morard I. Rubbia-Brandt L. Schrenzel J.  
BMC Gastroenterology (2006) 6 Article Number: 10. Date of Publication: 10   
Mar 2006  
  
Background: Splanchnic vein thrombosis may complicate inherited thrombotic   
disorders. Acute cytomegalovirus infection is a rare cause of acquired   
venous thrombosis in the portal or mesenteric territory, but has never been   
described extending into a main hepatic vein. Case presentation: A   
36-year-old immunocompetent woman presented with acute primary   
cytomegalovirus infection in association with extensive thrombosis in the   
portal and splenic vein. In addition, a fresh thrombus was evident in the   
right hepatic vein. A thorough evaluation for a hypercoagulable state was   
negative. The clinical course, biological evolution, radiological and   
histological findings were consistent with cytomegalovirus hepatitis   
complicated by a partial acute Budd-Chiari syndrome and portal thrombosis.   
Therapeutic anticoagulation was associated with a slow clinical improvement   
and partial vascular recanalization. Conclusion: We described in details a   
new association between cytomegalovirus infection and acute venous   
thrombosis both in the portal vein and in the right hepatic vein, realizing   
a partial Budd-Chiari syndrome. One should be aware that this rare   
thrombotic event may be complicated by partial venous outflow block. © 2006   
Spahr et al; licensee BioMed Central Ltd.  
  
RECORD 742  
Budd-Chiari syndrome and acute portal vein thrombosis: management by a   
transjugular intrahepatic portosystemic shunt (TIPS) and portal vein   
interventions via a TIPS  
Kori I. Bar-Zohar D. Carmiel-Haggai M. Samuels D. Nakache R. Oren R. Kessler   
A. Szold O. Ben-Haim M.  
Journal of Gastrointestinal Surgery (2006) 10:3 (417-421). Date of   
Publication: March 2006  
  
Acute portal vein thrombosis (PVT) is a devastating complication of   
Budd-Chiari syndrome (BCS). Conservative approach, anticoagulation, systemic   
or transarterial thrombolysis, and urgent liver transplantation were applied   
in this scenario but with poor results. We present and discuss an approach   
to treat BCS complicated by acute PVT. Two young female patients presented   
with acute liver failure, rapidly progressive tense ascites, renal- and   
respiratory failure. The diagnosis of chronic BCS complicated by acute PVT   
was confirmed with ultrasound Doppler. Initial treatment was supportive.   
Right portal vein localization was by transarterial portogram or by computed   
tomography-guided microcoil placement. Transjugular intrahepatic   
portosystemic shunt (TIPS) was performed and included Wallstents and a   
Jograft in one case and Viatorr stentgraft that was extended later with a   
Hemobahn stentgraft in another. Mechanical clot removal from the portal   
system was performed in the primary procedure and in a revision procedure in   
the following few days. Stents were placed precisely with no extension into   
the inferior vena cava or deeply into the main portal vein. Patients were   
fully anticoagulated and patency was assessed by ultrasound Doppler. The   
procedures were performed on days 5 and 10 following admission. In both   
cases, successful thrombectomies were reveised and maintained. Partial   
occlusion of the TIPS and reaccumulation of ascites were reversed with   
repeated procedure. Both patients were discharged without ascites and normal   
liver function. In conclusion, urgent TIPS and portal vein thrombectomy via   
TIPS are emerging therapeutic options that offer a safe and effective   
treatment to patients with BCS complicated by acute portal vein thrombosis.   
© 2006 The Society for Surgery of the Alimentary Tract.  
  
RECORD 743  
Etiology and consequences of thrombosis in abdominal vessels  
Bayraktar Y. Harmanci O.  
World Journal of Gastroenterology (2006) 12:8 (1165-1174). Date of   
Publication: 28 Feb 2006  
  
The thrombophilia which can be either congenital or acquired in adult life   
has major implications in the abdominal vessels. The resulting portal vein   
thrombosis, Budd-Chiari syndrome and mesenteric vein thrombosis have a   
variety of consequences ranging from acute abdomen to chronic hepatomegaly   
and even totally asymptomatic patient in whom the only finding is   
pancytopenia. The complications like esophageal varices, portal gastropathy,   
ascites, severe hypersplenism, liver failure requiring liver transplantation   
are well known. Interesting features of collateral venous circulation   
showing itself as pseudocholangiocarcinoma sign and its possible clinical   
reflection as cholestasis are also known from a long time. The management   
strategies for these complications of intraabdominal vessel thrombosis are   
not different from their counterpart which is cirrhotic portal hypertension,   
but the prognosis is unquestionably better in former cases. In this review   
we presented and discussed the abdominal venous thrombosis, etiology and the   
resulting clinical pictures. There are controversial issues both in   
nomenclature, and management including anticoagulation problems and follow   
up strategies. In light of the current knowledge, we discussed some   
controversial issues in literature and presented our experience and our   
proposals about this group of patients. © 2006 The WJG Press. All rights   
reserved.  
  
RECORD 744  
Elective laparoscopic splenectomy and thrombosis of the spleno-portal axis:   
A prospective study with ecocolordoppler ultrasound  
Romano F. Caprotti R. Scaini A. Conti M. Scotti M. Colombo G. Uggeri F.  
Surgical Laparoscopy, Endoscopy and Percutaneous Techniques (2006) 16:1   
(4-7). Date of Publication: February 2006  
  
Thrombosis of the portal system is a potentially life-threatening but   
otherwise underappreciated complication after splenectomy. Nonspecific and   
mild onset symptoms are the cause of delay in diagnosis, and the short   
hospital stay after laparoscopic approach could even contribute to the   
difficulty of early detection of this condition. The aim of this study was   
to verify if planned imaging controls are able to discover this complication   
leading to a prompt treatment. Thirty-eight patients (19 males and 19   
females with a mean age of 24 years) who underwent laparoscopic splenectomy   
at our institution were studied to identify clinical signs of thrombosis of   
the portal venous system and eventually associated factors. All the patients   
were enrolled in a protocol of imaging surveillance using a doppler   
ultrasound method. Postoperative thrombosis of the spleno-portal axis   
occurred in 7 patients (18.9%) of the series. In 3 cases (8.1%) the thrombus   
extended from the splenic vein to occlude the portal axis. The complication   
was symptomatic in 4 cases (10.8%), whereas in 3 cases, the thrombosis was   
an ultrasonographic surprise in totally asymptomatic patients. Thrombosis   
occurred even as late as 2 months after splenectomy. Splenomegaly was the   
only significant factor predictive of thrombosis. Only those patients who   
had an early detection of portal or splenic vein thrombosis had a   
recanalization of the veins with anticoagulant therapy. Patients with   
splenomegaly who underwent laparoscopic splenectomy are at risk of   
thrombosis of the portal system and should undergo strict imaging   
surveillance and aggressive anticoagulation therapy. Copyright © 2006 by   
Lippincott Williams & Wilkins.  
  
RECORD 745  
Portal and mesenteric venous thrombosis in inflammatory bowel disease   
treated by fibrinogen-guided thrombolysis with urokinase  
Brueck M. Runde T. Rauber K. Kramer W.  
Deutsche Medizinische Wochenschrift (2006) 131:3 (84-88). Date of   
Publication: 20 Jan 2006  
  
History and admission findings: A 23-year-old woman with a 5-year history of   
ulcerative colitis was admitted to our hospital because of bloody diarrhea.   
Two years previously she had undergone a hemicolectomy for a right colonic   
stricture. A recurrence of inflammatory bowel disease was suspected and   
treatment with prednisolone begun. The symptoms improved gradually, but 7   
days later she complained of lower abdominal pain. Physical examination   
revealed a soft abdomen, but bowel sounds were reduced. Investigations: The   
abdominal X-ray was unremarkable, but ultrasonography revealed moderate   
ascites and no blood flow in the portal vein on Doppler examination. The   
spleen was slightly enlarged. Contrast-enhanced abdominal magnetic resonance   
imaging (MRI) was performed immediately, revealing thrombosis of the portal   
and mesenteric veins. Treatment and course: As there was no suggestion of   
intestinal necrosis, laparatomy was not considered necessary. Intravenous   
thrombolytic treatment with urokinase was given continuously (bolus of   
250000 units, followed by 200 000 units per hour), in order to lower the   
fibrinogen level to 100 - 150 mg/dl, together with unfractionated heparin,   
maintaining the activated partial thromboplastin time between 60 and 85   
seconds. The thrombolytic treatment had to be stopped several times because   
of bloody diarrhea, but no transfusion was necessary. Two days after the   
start of thrombolytic treatment the abdominal pain and ascites ceased.   
Doppler sonography now demonstrated hepatopetal flow in the previously   
occluded portal vein. 4 days later, MRI revealed that the thrombus in the   
portal vein had dissolved and the portal vein was fully patent. The   
mesenteric vein was partially perfused, a residual thrombus extending into   
the portal vein. Tests for thrombophilia were negative. The thrombolytic   
therapy was stopped after 112 hours and the patient was treated with oral   
anticoagulation for 6 months. The patient recovered completely, with no   
evidence of portal hypertension during the following 6 months. Conclusions:   
Thrombolysis with urokinase, guided by the level of fibrinogen, may be an   
alternative, semi-invasive treatment option in acute thrombosis of the   
portal and mesenteric veins. © Georg Thieme Verlag Stuttgart.  
  
RECORD 746  
Pathogenesis and treatment of Budd-Chiari syndrome combined with portal vein   
thrombosis  
Murad S.D. Valla D.-C. De Groen P.C. Zeitoun G. Haagsma E.B. Kuipers E.J.   
Janssen H.L.A.  
American Journal of Gastroenterology (2006) 101:1 (83-90). Date of   
Publication: January 2006  
  
OBJECTIVES: Combined Budd-Chiari syndrome and portal vein thrombosis   
(BCS-PVT) is a challenging clinical condition with as yet unknown outcome.   
The aim of the present study was to investigate etiology, treatment options,   
and prognosis of patients with BCS-PVT. METHODS: Patients diagnosed with   
nonmalignant BCS between 1984 and 2001 were identified in a large   
international study and classified into isolated BCS (n = 204), BCS-PVT   
without spleno-mesenteric vein thrombosis (SMVT; n = 15), and BCS-PVT with   
SMVT (n = 18). RESULTS: Multifactorial etiology was present in 58% of   
patients with combined BCS-PVT. Number of etiological factors increased   
significantly with the extent of thrombosis (p= 0.002). Main treatment   
options included anticoagulation and portosystemic shunting, of which   
extended TIPS showed the most beneficial results. Five-year survival was 59%   
(95% CI 39-80%) in BCS-PVT versus 85% (95% CI 76-88%) in isolated BCS (p=   
0.11). Survival tended to be worse in BCS-PVT patients with SMVT as compared   
to patients without SMVT (RR = 3.47, p= 0.11). CONCLUSIONS: In BCS,   
extension of thrombosis into the splanchnic venous bed was significantly   
related to the number of etiological factors, and was associated with poor   
outcome. These results strongly support a liberal use of anticoagulants,   
which so far had been widely debated. Alternatively, derivative shunt   
procedures appear difficult, yet not impossible. © 2006 by Am. Coll. of   
Gastroenterology Published by Blackwell Publishing.  
  
RECORD 747  
Portal thrombosis complicating an acute cytomegalovirus infection in an   
immunocompetent patient  
Chelbi F. Boutin-Le Thi Huong D. Frigui M. Asli B. Hausfater P. Piette J.-C.  
Revue de Medecine Interne (2006) 27:1 (54-58). Date of Publication: January   
2006  
  
Introduction. - The cytomegalovirus (CMV) infection is most often   
asymptomatic. The grave forms concern the immunocompromised patients. We   
report a new case pf acute CMV hepatitis complicated with portal thrombosis   
in an immunocompetent patient. Exegesis. - A 29 year old man has presented a   
CMV hepatitis proved by the presence of pp65 protein and the viral DNA in   
serum. This infection was complicated by a portal thrombosis and the   
evolution was rapidly favourable under anticoagulant treatment. Eleven cases   
of major thrombosis complicating acute CMV infection in immunocompetent   
patients were previously reported in the English and French literature. The   
absence of local and general cause, the remission without anticoagulation,   
the elevated risk of thrombosis in both HIV and CMV seropositive patients,   
and in CMV seropositive renal transplant patients suggest a causal relation.   
Various pathogenic hypotheses were raised: presence of antiphospholipid   
antibodies, absent in our case, procoagulant phenotype induction of infected   
endothelial cells, proliferation induction of smooth cells. Conclusion. -   
The acute CMV infection can be considered such as a possible cause of major   
thrombosis. © 2005 Elsevier SAS. Tous droits réservés.  
  
RECORD 748  
anticoagulation therapy may reverse biliary abnormalities due to acute   
portal thrombosis  
Louvet A. Texier F. Dharancy S. Pruvot F.-R. Sergent G. Deltenre P. Ernst O.   
Paris J.-C. Mathurin P.  
Digestive Diseases and Sciences (2006) 51:1 (11-17). Date of Publication:   
January 2006  
  
RECORD 749  
Liver transplant in Hawaii: The survival of a small centre  
Wong L.L. Limm W. Cheung A. Noguchi H.  
Clinical Transplantation (2006) 20:1 (55-61). Date of Publication:   
January/February 2006  
  
Although many report the importance of case volume in complex cases, liver   
transplantation (LT) can be carried out successfully in a small centre.   
During a 11.5-yr period, 88 patients underwent LT in a single transplant   
centre in Hawaii. Indications for LT were primarily hepatitis C (n = 49) and   
hepatitis B (n = 13) and 22 patients (25%) had hepatocellular cancer (HCC)   
on explanted liver. There was no primary graft nonfunction, one retransplant   
for recurrent hepatitis C and two late hepatic artery thromboses, which did   
not require a retransplant. One patient developed partial portal vein   
thrombosis related to a hypercoagulable state and was rescued with   
anticoagulation. Of the 22 patients with HCC, 18 are alive, two died from   
recurrent disease (253 and 1428 d post-LT, respectively), one died because   
of a ruptured hepatic artery aneurysm (151 d) and one from complications   
caused by noncompliance (723 d). One-, 3- and 5-yr survival rates were 89%,   
82% and 71%, respectively. Mean survival was 3034.9 d. During this time   
period, 142 liver resections, 77 pancreatic resections and 43 splenorenal   
shunts were performed by this group of surgeons. Because of the recent   
explosion of information on case volumes and centres of excellence, LT can   
be performed successfully at a small centre. Other major   
hepatobiliary/transplant procedures can help the surgeons maintain their   
operative skills. A smaller LT program may require a longer period of   
evolution, but it can provide a service for a geographically isolated   
population that would otherwise have limited opportunity for LT. © Blackwell   
Munksgaard, 2005.  
  
RECORD 750  
Intrahepatic cholangiocarcinoma presenting as Budd-Chiari syndrome: A case   
report and literature review  
Law J.K. Davis J. Buckley A. Salh B.  
Canadian Journal of Gastroenterology (2005) 19:12 (723-728). Date of   
Publication: December 2005  
  
Intrahepatic cholangiocarcinoma, an increasingly recognized primary tumour   
of the liver, is associated with a very poor prognosis. A patient with this   
tumour who presented with Budd-Chiari syndrome (the first to the authors'   
knowledge in Western literature and only the third patient overall)   
secondary to extensive thrombosis in his inferior vena cava extending from   
the right atrium down to his iliac vessels is described. Neither curative   
nor palliative intervention was deemed to be an option in this patient, who   
deteriorated rapidly while on anti-coagulants. Postmortem examination   
confirmed the radiological findings, and histological analysis revealed   
characteristic appearances of this tumour within the biliary tree and   
invasion into the inferior vena cava. Furthermore, biliary dysplasia, which   
can be a precursor to this cancer, was also noted within some of the bile   
ducts. ©2005 Pulsus Group Inc. All rights reserved.  
  
RECORD 751  
Thrombophilic conditions in non-cirrhotic portal vein thrombosis  
Shah S.R. DasGupta A. Sharma A. Joshi A. Desai D. Abraham P. Rathi P. Bapat   
M.  
Indian Journal of Gastroenterology (2005) 24:5 (205-210). Date of   
Publication: 2005  
  
Objective: To study the prevalence of thrombophilic conditions in patients   
with acute and chronic portal vein thrombosis (PVT) and to compare it with   
those in patients suffering from deep vein thrombosis (DVT) after lower limb   
arthroplasty and in healthy subjects. Methods : Twenty-six patients with   
spontaneous PVT (20 chronic, 6 acute) with normal liver function and not   
receiving anticoagulants were evaluated for thrombophilic conditions. Levels   
of protein C, protein S and antithrombin were compared with those in 50   
healthy controls. Factor V gene 'Leiden' mutation (FVL) and high   
homocysteine levels were looked for in patients with PVT and in 18 patients   
developing post-arthroplasty lower limb DVT despite anticoagulation.   
Results: Of 26 patients with PVT, 19 had at least one thrombotic condition   
(acute PVT 5/6, chronic PVT 14/20) and 12 had more than one such condition;   
in comparison, of 18 patients with DVT, eight had one thrombophilic   
condition and one had two such conditions (p=0.03). Patients with PVT had   
significantly lower levels of protein C, protein S and antithrombin than   
healthy subjects and those with DVT. Six patients had Factor VIII levels   
above 150%; four had elevated homocysteine levels and three had detectable   
anti-cardiolipin antibodies. Three patients with PVT (acute 2, chronic 1)   
were heterozygous for FVL mutation. Conclusions: Underlying thrombophilic   
conditions are common in Indian patients with spontaneous PVT. In many   
patients, multiple thrombophilic conditions are present and these may play a   
role in the pathogenesis of PVT. © 2004 Indian Journal of Gastroenterology.  
  
RECORD 752  
Letter to the editor [1] (multiple letters)  
Winslow E.R. Klingensmith M.E. Brunt L.M. Ikeda M. Sekimoto M. Takiguchi S.   
Takemasa I. Yamamoto H. Monden M.  
Annals of Surgery (2005) 242:5 (745-746). Date of Publication: Nov 2005  
  
RECORD 753  
Deciphering mesenteric venous thrombosis: Imaging and treatment  
Grisham A. Lohr J. Guenther J.M. Engel A.M.  
Vascular and Endovascular Surgery (2005) 39:6 (473-479). Date of   
Publication: November/December 2005  
  
The principal cause of a high mortality rate in mesenteric vein thrombosis   
(MVT) is a delay in diagnosis. Recent data indicate that the mortality rate   
is decreasing owing to earlier diagnosis and anticoagulation. The authors   
examined the treatment profile of MVT to see how the increased use of   
imaging and early anticoagulation has impacted this process. They   
retrospectively analyzed the treatment paradigm with acute MVT at one   
institution over a 10-year period. Twenty-three patients were identified.   
Data were analyzed using chi-squares and Student's t tests. Twenty-three   
patients (11 men and 12 women with an average age of 51.74 ± 14.8 years)   
were identified with acute MVT between the years of 1993 and 2003. Five   
patients had splenic vein thrombosis, 17 had superior mesenteric vein   
thrombosis, 1 had inferior mesenteric vein thrombosis, and 12 had portal   
vein thrombosis. Nine patients had combination mesenteric vein segment   
thrombosis. Thrombolytics were utilized in a total of 6 patients. Four of   
the 6 patients in whom lytics were utilized had combined mesenteric vein   
thrombosis; however, these 4 patients did not require surgical intervention.   
There was no significant difference in length of hospital stay between   
patients taking lytics versus patients treated with traditional   
anticoagulation with heparin (p = 0.291). A hypercoagulable state was   
identified in 66.7% of the patients. Four patients required surgical   
intervention. The overall mortality rate was 8.7% (2 of 23). The use of   
thrombolytics was associated with a significant mortality (p = 0.04). The   
use of antibiotics made no difference in mortality (p = 0.235), nor did   
antibiotic use influence length of hospitalization (p = 0.192). MVT is   
relatively rare, and often the delay in diagnosis increases the mortality   
rate. In the majority of cases prompt anticoagulation will preserve bowel   
viability and decrease mortality and morbidity rates. The majority of   
patients do not need surgery. There is a marked increase in mortality rate   
when these patients progress to surgical intervention. An increased   
awareness and early diagnosis has led to decreased morbidity and mortality   
rates. ©2005 Westminster Publications, Inc.  
  
RECORD 754  
Mesogonadal shunts for extrahepatic portal vein thrombosis and variceal   
hemorrhage  
Kim H.B. Pomposelli J.J. Lillehei C.W. Jenkins R.L. Jonas M.M. Krawczuk L.E.   
Fishman S.J.  
Liver Transplantation (2005) 11:11 (1389-1394). Date of Publication:   
November 2005  
  
Extrahepatic portal vein thrombosis (EHPVT) may occur in children or adults   
and usually comes to clinical attention due to complications of portal   
hypertension such as variceal hemorrhage. A variety of standard surgical   
techniques exist to manage these patients, but when these fail surgical   
options are limited. We describe two novel portosystemic shunts that utilize   
the gonadal vein as an autologous conduit. Four patients were evaluated for   
EHPVT with variceal bleeding. None of the patients were candidates for a   
standard splenorenal shunt due to prior surgical procedures. The first   
patient underwent a left mesogonadal shunt and the remaining 3 patients   
underwent a right mesogonadal shunt. Postoperative ultrasound or computed   
tomography (CT) scan confirmed early patency of the shunt in each patient.   
There have been no further episodes of variceal hemorrhage with follow-up of   
3.5 years in the child who underwent the left mesogonadal shunt, and 17, 19,   
and 20 months in the patients who underwent the right mesogonadal shunt.   
Three of the 4 shunts remain patent. One shunt thrombosis occurred in a   
patient homozygous for the Factor V Leiden mutation despite anticoagulation   
with coumadin. This is the first report of the successful use of the gonadal   
vein as an in situ conduit for constructing a portosystemic shunt. In   
conclusion, the right and left mesogonadal shunts may be useful as salvage   
operations for patients with EHPVT who have failed standard surgical shunt   
procedures.r Copyright © 2005 by the American Association for the Study of   
Liver Diseases.  
  
RECORD 755  
portal vein thrombosis after laparoscopic colectomy: thrombolytic therapy   
via the superior mesenteric vein.  
Poultsides G.A. Lewis W.C. Feld R. Walters D.L. Cherry D.A. Ruby S.T.  
The American surgeon (2005) 71:10 (856-860). Date of Publication: Oct 2005  
  
portal vein thrombosis is a rare but well-reported complication after   
laparoscopic surgery. We present a case of portomesenteric venous thrombosis   
that occurred 8 days after a laparoscopic-assisted right hemicolectomy.   
Systemic anticoagulation failed to improve symptoms. The early postoperative   
state precluded the use of transarterial thrombolytic therapy. Transjugular   
intrahepatic catheter-directed infusion of urokinase into the superior   
mesenteric vein resulted in clearance of thrombus and resolution of   
symptoms. The published data on laparoscopy-induced splanchnic venous   
thrombosis and transjugular intrahepatic intramesenteric thrombolysis are   
discussed.  
  
RECORD 756  
Long-term outcomes of venous thrombosis in children  
Goldenberg N.A.  
Current Opinion in Hematology (2005) 12:5 (370-376). Date of Publication:   
September 2005  
  
Purpose of review: Venous thromboembolism has become an increasingly   
recognized clinical entity in children over the past decade. Recently,   
important efforts have been made to track post thrombotic outcomes. The   
present review discusses the knowledge gained from seminal studies in the   
field over the past decade, with particular emphasis on the findings of   
numerous published reports on outcomes of various types of venous   
thromboembolism in the pediatric literature from January 2003 to January   
2005. Recent findings: Large cohort studies involving acute venous   
thromboembolism of all types in children have recently confirmed a rather   
low frequency of recurrent venous thromboembolism, but have shown a lack of   
thrombus resolution following standard-duration anticoagulant therapy in as   
many as 50% of patients. In addition, the development of the post thrombotic   
syndrome has been demonstrated in greater than one third of children with   
venous thromboembolism involving the extremities. Persistent thrombosis   
despite adequate anticoagulation has been independently associated with   
complete vaso-occlusion at diagnosis. Furthermore, the presence of elevated   
levels of factor VIII and D-dimer either at diagnosis or following three to   
six months anticoagulation has now been defined as a predictor of adverse   
long-term outcomes of pediatric thrombosis. Summary: This body of work   
indicates that the various forms of venous thromboembolism in children are   
in general associated with a considerable degree of adverse outcomes,   
particularly in the form of venous thromboembolism-related mortality,   
recurrent thromboembolism, and development of post thrombotic syndrome.   
Advances in the prediction of post thrombotic outcomes have begun, and must   
continue to facilitate a risk-stratified approach to antithrombotic   
management in children, and to ultimately achieve meaningful improvements in   
long-term outcomes. © 2005 Lippincott Williams & Wilkins.  
  
RECORD 757  
High incidence of recurrence and hematologic events following liver   
transplantation for Budd-Chiari syndrome  
Cruz E. Ascher N.L. Roberts J.P. Bass N.M. Yao F.Y.  
Clinical Transplantation (2005) 19:4 (501-506). Date of Publication: August   
2005  
  
Background: Most cases of Budd-Chiari syndrome (BCS) in Western countries   
are related to underlying hematologic diseases with inherent thrombogenic   
propensity. We evaluated the long-term outcome, risks for recurrent disease,   
and other hematologic complications following orthotopic liver   
transplantation (OLT) for BCS. Methods: Clinical data from 11 consecutive   
patients with BCS who underwent OLT were retrospectively reviewed. Four   
patients had a prior transjugular intrahepatic portosystemic shunt and one   
had a surgical shunt procedure. All patients were started on intravenous   
heparin within the first 24 h following OLT. All except one patient who had   
protein C deficiency were maintained on long-term oral anticoagulation.   
Results: The Kaplan-Meier survival rates at 1, 5 and 10 yr were 81, 65 and   
65%, respectively. Three patients developed BCS recurrence, including two   
who died as a consequence of rapid graft failure within days after OLT.   
Three patients developed other thrombotic events, including splenic vein   
thrombosis associated with gastric variceal hemorrhage requiring   
splenectomy, portal vein thrombosis and pulmonary embolism. Four patients   
experienced severe bleeding complications within 7 d after OLT requiring   
exploratory laparotomy. One patient died after transformation of   
polycythemia vera to acute myelogenous leukemia at 2.1 yr after OLT.   
Conclusion: We observed a high incidence of recurrent BCS and complications   
related to the underlying hematologic disorder or anticoagulation after OLT   
for BCS. The present series also included the first two cases of rapid   
recurrence of BCS and graft failure within days after OLT. © Blackwell   
Munksgaard, 2005.  
  
RECORD 758  
Transjugular intrahepatic portosystemic shunt  
Ochs A.  
Digestive Diseases (2005) 23:1 (56-64). Date of Publication: 2005  
  
The transjugular intrahepatic portosystemic shunt (TIPS) is an   
interventional treatment resulting in decompression of the portal system by   
creation of a side-to-side portosystemic anastomosis. Since its introduction   
16 years ago, more than 1,000 publications have appeared demonstrating broad   
acceptance and increasing clinical use. This review summarizes our present   
knowledge about technical aspects and complications, follow-up of patients   
and indications. A technical success rate near 100% and a low occurrence of   
complications clearly depend on the skills of the operator. The follow-up of   
the TIPS patient has to assess shunt patency, liver function, hepatic   
encephalopathy and the possible development of hepatocellular carcinoma.   
Shunt patency can best be monitored by duplex sonography and can avoid   
routine radiological revision. Short-term patency may be improved by   
anticoagulation, while such a treatment does not influence long-term   
patency. Stent grafts covered with expanded polytetrafluoroethylene show   
promising long-term patency comparable with that of surgical shunts. With   
respect to the indications of TIPS, much is known about treatment of   
variceal bleeding and refractory ascites. The thirteen randomized studies   
that are available to date show that survival is comparable in pa-tients   
receiving TIPS or endoscopic treatment for acute or recurrent variceal   
bleeding. Another group comprises patients with refractory ascites and   
related complications, such as hepatorenal syndrome and hepatic hydrothorax.   
It has been demonstrated that TIPS improves these complications. Five   
randomized studies comparing TIPS with paracentesis and one study comparing   
TIPS with the peritoneo-venous shunt showed good response of ascites but   
controversial results on survival. In addition, TIPS has been successfully   
applied to patients with Budd-Chiari syndrome, portal vein thrombosis,   
before liver transplantation, and for the treatment of ectopic variceal   
bleeding. Copyright © 2005 S. Karger AG.  
  
RECORD 759  
portal vein thrombosis: What is the role of genetics?  
Walker A.P.  
European Journal of Gastroenterology and Hepatology (2005) 17:7 (705-707).   
Date of Publication: July 2005  
  
The aetiology of portal vein thrombosis (PVT) in adults is complex. Risk   
factors include local precipitating factors and acquired and inherited   
factors, an area in which there has been much recent progress. Although PVT   
in the absence of cirrhosis may be regarded as a somewhat different disorder   
to PVT in the presence of cirrhosis, in both cases most studies support a   
role of the prothrombin G20210A mutation. Some differences in risk factors   
observed between different studies may relate partly to referral patterns or   
study design, although individual patients may develop PVT as a result of   
differing combinations of risk factors. The demonstration of an inherited   
thrombophilic mutation in a subset of PVT may ultimately inform clinical   
management regarding the use and duration of anticoagulation therapy,   
although there is a need for evidence from randomized-controlled clinical   
trial data. © 2005 Lippincott Williams & Wilkins.  
  
RECORD 760  
A case of portal vein thrombosis associated with acute pancreatitis and   
cholangitis  
Cheung D.Y. Kim J.K. Jo D.H. Oh H.J. Kim T.H. Lee S.Y. Park S.H. Han J.Y.   
Chung K.W. Sun H.S.  
The Korean journal of gastroenterology = Taehan Sohwagi Hakhoe chi (2005)   
46:1 (60-65). Date of Publication: Jul 2005  
  
portal vein thrombosis is a rare complication accompanied with acute   
pancreatitis or cholangitis/cholecystitis. The main pathogenesis of portal   
vein thrombosis in pancreatitis or cholangitis/cholecystitis are suggested   
to be venous compression by pseudocyst and an imbalance between the blood   
coagulation and fibrinolysis. In this case report, we experienced a 63 year   
old male who developed portal vein thrombosis later in the course of the   
treatment of acute gallstone pancreatitis with cholangitis/cholecystitis   
without any symptom or sign. The diagnosis of portal vein thrombosis was   
given on follow up CT scan and serum protein S activity was decreased to 27%   
in laboratory study. Immediate anticoagulation therapy with heparin and   
thrombolytic therapy with urokinase and balloon dilatation were performed.   
Despite the aggressive treatment, complete reperfusion could not be   
obtained. With oral warfarin anticoagulation, the patient showed no disease   
progression and was discharged. We report a case of portal vein thrombosis   
as a complication of acute pancreatitis and cholangitis/cholecystitis with a   
review of literatures.  
  
RECORD 761  
Imaging and radiological interventions of portal vein thrombosis.  
Hidajat N. Stobbe H. Griesshaber V. Felix R. Schroder R.J.  
Acta radiologica (Stockholm, Sweden : 1987) (2005) 46:4 (336-343). Date of   
Publication: Jul 2005  
  
portal vein thrombosis (PVT) is diagnosed by imaging methods. Once diagnosed   
by means of ultrasound, Doppler ultrasound can be performed to distinguish   
between a benign and malignant thrombus. If further information is required,   
magnetic resonance angiography or contrast-enhanced computed tomography is   
the next step, and if these tests are unsatisfactory, digital subtraction   
angiography should be performed. Many papers have been published dealing   
with alternative methods of treating PVT, but the material is fairly   
heterogeneous. In symptomatic non-cavernomatous PVT, recanalization using   
local methods is recommended by many authors. Implantation of transjugular   
intrahepatic portosystemic shunt is helpful in cirrhotic patients with   
non-cavernomatous PVT in reducing portal pressure and in diminishing the   
risk of re-thrombosis. In noncirrhotic patients with recent PVT, some   
authors recommend anticoagulation alone. In chronic thrombotic occlusion of   
the portal vein, local measures may be implemented if refractory symptoms of   
portal hypertension are evident.  
  
RECORD 762  
portal vein thrombosis after laparoscopic splenectomy: an ongoing clinical   
challenge.  
Miniati D.N. Padidar A.M. Kee S.T. Krummel T.M. Mallory B.  
JSLS : Journal of the Society of Laparoendoscopic Surgeons / Society of   
Laparoendoscopic Surgeons (2005) 9:3 (335-338). Date of Publication: 2005   
Jul-Sep  
  
OBJECTIVES: portal vein thrombosis (PVT) following open splenectomy is a   
potentially lethal complication with an incidence of up to 6%. The objective   
of this report is to describe our management of a recent laparoscopic case,   
discuss current therapies, and consider antiplatelet therapy for   
prophylaxis. METHODS: Medical records, laboratory studies, and imaging   
studies pertaining to a recent case of a laparoscopic splenectomy were   
examined. Current literature related to this topic was reviewed. RESULTS: A   
16-year-old girl underwent laparoscopic splenectomy for idiopathic   
thrombocytopenic purpura. Her preoperative platelet count was 96K. She was   
discharged on postoperative day 1 after an uneventful operation including   
division of the splenic hilum with an endoscopic linear stapler. On   
postoperative day 20, she presented with a 5-day history of epigastric pain,   
nausea, and low-grade fevers without peritoneal signs. Her white blood cell   
count was 17.3; her platelets were 476K. Computed tomography demonstrated   
thrombosis of the splenic, superior mesenteric, and portal veins propagating   
into the liver. Heparinization was begun followed by an unsuccessful attempt   
at pharmacologic and mechanical thrombolysis by interventional radiology.   
Over the next 5 days, her pain resolved, she tolerated a full diet, was   
converted to oral anticoagulation and sent home. Follow-up radiographic   
studies demonstrated the development of venous collaterals and cavernous   
transformation of the portal vein. DISCUSSION: No standard therapy for PVT   
exists; several approaches have been described. These include systemic   
anticoagulation, systemic or regional medical thrombolysis, mechanical   
thrombolysis, and surgical thrombectomy. Unanswered questions exist about   
the most effective acute therapy, duration of anticoagulation, and the   
potential efficacy of routine prophylaxis with perioperative antiplatelet   
agents. PVT following splenectomy occurs with both the open and laparoscopic   
approach.  
  
RECORD 763  
portal vein thrombosis (PVT): A study of 20 non-cirrhotic cases  
Kocher G. Himmelmann A.  
Swiss Medical Weekly (2005) 135:25-26 (372-376). Date of Publication: 25 Jun   
2005  
  
Background: Portal and mesenteric venous thrombosis (PVT) is an uncommon   
disease with serious consequences if not discovered early in order to   
prevent complications such as variceal bleeding and intestinal ischaemia.   
The objective of this study was to describe the clinical presentation and   
outcome of patients with PVT with a view to early diagnosis and treatment of   
this disease. The study was restricted to patients with PVT not caused by   
underlying liver cirrhosis. Patients and methods: To analyse important   
clinical characteristics of this entity we performed a retrospective study   
of 20 non-cirrhotic patients seen in our hospital from February 1998 to   
March 2003. Results: The main clinical symptom was abdominal pain (13   
patients, 86%), sometimes in combination with diarrhoea and vomiting (5   
patients, 33%), nausea and anorexia (3 patients). Laboratory signs were   
non-specific and diagnosis was usually by computed tomography (19 patients,   
95%). Causative factors included prothrombotic states (9 patients, 45%)   
and/or local factors (5 patients, 25%). Complications must be expected from   
portal hypertension (15 patients, 75%), which was associated with variceal   
bleeding in 6 patients (30%). Bowel ischaemia (5 patients, 25%) and bowel   
infarction (2 patients) were less frequent. Treatment consisted of immediate   
anticoagulation in almost all cases (18 patients, 90%), while invasive   
approaches were followed in selected patients. The prognosis of PVT was good   
in patients without a severe underlying disease (median followup 21 months).   
Conclusions: In agreement with other studies our results suggest that early   
diagnosis and treatment by immediate anticoagulation are important in   
preventing the serious consequences of portal and mesenteric vein occlusion.   
The role of more invasive approaches is less well defined. Since in 18   
patients (90%) of the non-cirrhotic cases in the present series causative   
factors were found which may have therapeutic implications, aetiological   
screening seems worthwhile in every case with PVT.  
  
RECORD 764  
Portal-splenic-mesenteric venous thrombosis secondary to a mutation of the   
prothrombin gene  
Frutos Bernal M.D. Fernández Hernández J.A. Carrasco Prats M. Soria Cogollos   
T. Luján Mompeán J.A. Hernández Agüera Q. Parrilla Paricio P.  
Gastroenterologia y Hepatologia (2005) 28:6 (329-332) Article Number:   
100.647. Date of Publication: June/July 2005  
  
Thrombosis of the portal-mesenteric axis is an infrequent cause of   
intestinal ischemia or infarction. In addition to the multiple acquired   
factors that contribute to the development of this entity, hereditary risk   
factors, especially the factor V Leiden mutation and the G20210A mutation of   
the prothrombin gene, have been implicated. The G20210A mutation of the   
prothrombin gene is found in up to 40% of patients with   
splenic-portal-mesenteric thrombosis. The present case illustrates the   
unusual and nonspecific presentation of this mutation in the form of   
diarrhea and images of thrombosis of the superior mesenteric-portal vein and   
cavernous transformation of the portal vein. Delayed diagnosis is highly   
frequent since the clinical signs, laboratory investigations and   
radiological tests do not suggest the diagnosis. The patient received   
anticoagulant treatment and showed clinical improvement with complete   
portal-mesenteric recanalization. Currently the diagnostic technique of   
choice is magnetic resonance angiography or computerized tomography   
angiography and treatment consists of indefinite anticoagulation. This case   
illustrates that an unusual or atypical localization of venous thrombosis   
may be a manifestation of thrombophilia, emphasizing the importance of   
genetic screening in these cases.  
  
RECORD 765  
Splanchnic vein thrombosis in candidates for liver transplantation:   
Usefulness of screening and anticoagulation  
Francoz C. Belghiti J. Vilgrain V. Sommacale D. Paradis V. Condat B. H   
Denninger M. Sauvanet A. Valla D. Durand F.  
Gut (2005) 54:5 (691-697). Date of Publication: May 2005  
  
Background and aims: Splanchnic vein thrombosis is a significant source of   
complications in candidates for liver transplantation. The aims of this   
study were: (a) to determine the prevalence of and risk factors for   
splanchnic vein thrombosis in cirrhotic patients awaiting transplantation   
and (b) to assess the usefulness of anticoagulation. Methods: A total of 251   
cirrhotic patients listed for transplantation were analysed. All underwent   
systematic screening for thrombosis with Doppler ultrasonography. During the   
second period of the study, all patients with thrombosis received   
anticoagulation up to transplantation while during the first period none had   
received anticoagulation. Results: The incidence of splanchnic vein   
thrombosis at evaluation was 8.4%. Seventeen additional patients (7.4%)   
developed de novo thrombosis after evaluation. Independent risk factors for   
thrombosis were low platelet count (77.4 (36.3) v 111.6 (69.2) 10(9)/l; P =   
0.001), a past history of variceal bleeding (47.4% v 29.1%; p = 0.003), and   
a prolonged interval from listing to transplantation (8.5 (6.8) v 4.8 (4.4)   
months; p = 0.002). The proportion of partial or complete recanalisation was   
significantly higher in those who received (8/19) than in those who did not   
receive (0/10, p = 0.002) anticoagulation. Survival was significantly lower   
in those who had complete portal vein thrombosis at the time of surgery (p =   
0.04). Conclusion: These results support a systematic screening for   
splanchnic vein thrombosis in patients awaiting transplantation. They   
suggest that in these patients, anticoagulation is safe and has a   
significant impact on recanalisation as well as prevention of extension of   
thrombosis.  
  
RECORD 766  
Transcatheter thrombolytic therapy for acute mesenteric and portal vein   
thrombosis  
Hollingshead M. Burke C.T. Mauro M.A. Weeks S.M. Dixon R.G. Jaques P.F.  
Journal of Vascular and Interventional Radiology (2005) 16:5 (651-661). Date   
of Publication: May 2005  
  
PURPOSE: The purpose of this study was to evaluate the utility of   
transcatheter thrombolytic therapy in 20 patients with acute or subacute   
(symptoms <40 days) portal and/or mesenteric vein thrombosis with severe   
symptoms, deteriorating clinical condition, and/or persistent symptoms   
despite anticoagulation. MATERIALS AND METHODS: This retrospective study   
examined 12 male patients and eight female patients seen over a period of 11   
years. The average age was 37.6 years. Four of the patients had previously   
undergone liver transplantation. An anatomic classification system was   
established to describe the extent of thrombus at the time of diagnosis.   
Patients were treated with thrombolytic therapy via the transhepatic route,   
common femoral vein route, and/or superior mesenteric artery route.   
Improvement in symptoms, avoidance of bowel resection, complications, and   
radiographic evidence of clot resolution were the main clinical outcomes.   
RESULTS: Fifteen of the 20 patients exhibited some degree of lysis of the   
thrombus. Three patients had complete resolution, 12 had partial resolution,   
and five had no resolution. Eighty-five percent of patients (n = 17) had   
resolution of symptoms. Sixty percent of patients (n = 12) developed a major   
complication. No patients required bowel resection after thrombolytic   
therapy. One patient died with gastrointestinal hemorrhage and septic shock   
2 weeks after thrombolytic therapy. Other major complications included   
bleeding and conditions requiring transfusion. No patients developed new   
portal or mesenteric thromboses. Two of the patients who received   
transplants eventually required repeat transplantation. CONCLUSIONS:   
Transcatheter thrombolysis was beneficial in avoiding patient death,   
resolving thrombus, improving symptoms, and avoiding bowel resection.   
However, there was a high complication rate, indicating that this therapy   
should be reserved for patients with severe disease. Further evaluation of   
these techniques and outcomes should continue to be pursued. © SIR, 2005.  
  
RECORD 767  
portal vein thrombosis: Etiology, diagnostic strategy, therapy and   
management  
Hidajat N. Stobbe H. Griesshaber V. Schroder R.-J. Felix R.  
Vasa - Journal of Vascular Diseases (2005) 34:2 (81-92). Date of   
Publication: May 2005  
  
Myeloproliferative disorder, liver cirrhosis with portal hypertension,   
deficiency of natural anticoagulant proteins, gene mutation and   
hepatocellular carcinoma are the most frequent causes of portal vein   
thrombosis (PVT). Higher accuracy of the diagnostic methods is the reason   
why today the cause of PVT can be found more frequently. With imaging   
methods, PVT with or without cavernous transformation can be diagnosed.   
Fresh thrombus can be undetected in sonography due to the low echogenity but   
can be recognized in color Doppler sonography, especially with   
contrast-enhancing agent. Contrast-enhanced 3D MR angiography allows a   
comparable accuracy in the detection of PVT as digital subtraction   
angiography. Therapeutical options of PVT consist of mechanical   
recanalization of the portal vein, local fibrinolysis with or without   
placement of transjugular intrahepatic portosystemic stent shunt (TIPS),   
combination of mechanical recanalization and local fibrinolysis, systemic   
thrombolytic therapy, anticoagulation alone and surgical thrombectomy. Once   
PVT is found in sonography, Doppler sonography may be performed in order to   
distinguish benign from malignant thrombus. If further information is   
needed, MR angiography or contrast enhanced CT is the next step. If these   
tests are unsatisfactory, digital subtraction angiography should be   
performed. Until the early nineties, shunt surgery was recommended in   
patients with PVT who bled despite endoscopic treatment. Today, in   
symptomatic noncavernomatous PVT, recanalization with local methods is   
recommended. Additional implantation of TIPS should be performed when the   
patient is cirrhotic. In recent PVT in non-cirrhotic patients   
anticoagulation alone is recommended. It is expected that in old PVT   
anticoagulation can prevent further extension of the thrombus. © by Verlag   
Hans Huber, Hogrefe AG, Bern 2005.  
  
RECORD 768  
Transhepatic fibrinolysis of mesenteric and portal vein thrombosis in a   
patient with ulcerative colitis: A case report  
Guglielmi A. Fior F. Halmos O. Veraldi G.F. Rossaro L. Ruzzenente A.   
Cordiano C.  
World Journal of Gastroenterology (2005) 11:13 (2035-2038). Date of   
Publication: 7 Apr 2005  
  
Aim: To present a case of acute mesenteric and portal vein thrombosis   
treated with thrombolytic therapy in a patient with ulcerative colitis in   
acute phase and to review the literature on thrombolytic therapy of   
mesenteric-portal system. Treatment of acute portal vein thrombosis has   
ranged from conservative treatment with thrombolysis and anticoagulation   
therapy to surgical treatment with thrombectomy and/or intestinal resection.   
Methods: We treated our patient with intraportal infusion of plasminogen   
activator and then heparin through a percutaneous transhepatic catheter.   
Results: Thrombus resolved despite premature interruption of the   
thrombolytic treatment for neurological complications, which subsequently   
resolved. Conclusion: Conservative management with plasminogen activator,   
could be considered as a good treatment for patients with acute   
porto-mesenteric thrombosis. © 2005 The WJG Press and Elsevier Inc. All   
rights reserved.  
  
RECORD 769  
portal vein thrombosis despite anticoagulation in a person with diabetes  
Schweigart J.H. Klotsas A. Schelenz S. Dhatariya K.  
Journal of the Royal Society of Medicine (2005) 98:4 (161-163). Date of   
Publication: April 2005  
  
RECORD 770  
Update on the classification, assessment of prognosis and therapy of   
Budd-Chiari syndrome  
Senzolo M. Cholongitas E.C. Patch D. Burroughs A.K.  
Nature Clinical Practice Gastroenterology and Hepatology (2005) 2:4   
(182-190). Date of Publication: April 2005  
  
Budd-Chiari syndrome (BCS) occurs as a result of obstruction of hepatic   
venous outflows at any level from the small hepatic veins to the junction of   
the inferior vena cava with the right atrium. Diagnosis can be difficult   
because of the wide spectrum of presentation of the disease and the varying   
severity of liver damage. The traditional classification of BCS - as   
fulminant, acute or chronic - is not prognostically useful. This makes   
assessing the benefit of therapy difficult, especially as there is no   
evidence from randomized studies. This article highlights advances in the   
prognosis and therapy of BCS. Identification of the site of venous   
obstruction has a major effect on prognosis. Portal-vein thrombosis occurs   
in 20-30% of cases, and acute presentation of BCS reflects an acute or   
chronic syndrome in 60% of BCS cases. BCS can be diagnosed and treated on a   
single occasion in the setting of the radiology department, with hepatic   
venography, transjugular liver biopsy, retrograde CO2 portography and   
inferior vena cava pressure measurements performed simultaneously with   
therapies such as dilation or stenting of webs in the inferior vena cava or   
hepatic veins, and placement of transjugular intrahepatic portosystemic   
shunts. Disruption of a portal vein thrombus can also be done during the   
same session. Surgical shunts have been superseded by the use of   
transjugular intrahepatic portosystemic shunts. Liver transplantation is   
reserved for ftihninant and progressive chronic forms of BCS.   
anticoagulation therapy must be used routinely, before and after specific   
therapy, regardless of whether a thrombophilic disorder is diagnosed.  
  
RECORD 771  
Portal hypertension due to cavernomatosis of the portal vein [4]  
Varona Arche J.F. Aranda Arcas J.L.  
Anales de Medicina Interna (2005) 22:2 (93-94). Date of Publication:   
February 2005  
  
RECORD 772  
Mesenteric and portal vein thrombosis: Treated with early initiation of   
anticoagulation  
Joh J.-H. Kim D.-I.  
European Journal of Vascular and Endovascular Surgery (2005) 29:2 (204-208).   
Date of Publication: February 2005  
  
Objective: Superior mesenteric vein thrombosis (SMVT) is generally difficult   
to diagnose and can be fatal. Mesenteric and portal vein thrombosis is rare   
and can be presented as more serious conditions than that of SMVT. We report   
patients with combined SMVT and portal vein thrombosis (PVT) who were   
treated successfully with early initiation of anticoagulation. Methods: The   
medical records of six patients (five male, one female) who presented with   
combined SMVT and PVT in our institute between January 1994 and September   
2003 were reviewed retrospectively. All of the patients were treated with   
early initiation of anticoagulation using unfractionated heparin or low   
molecular weight heparin. Results: The mean hospital stay was 31 days and   
the mean follow-up period was 32 months. Three patients had an antithrombin   
III deficiency. The most common symptom was diffuse abdominal pain and signs   
included abdominal distension and tenderness. During the follow-up period,   
there were two patients who developed stricture of the small bowel   
necessitating resection and anastomosis of the small bowel. There was no   
case of peritonitis due to bowel necrosis or mortality. Conclusion: The   
early initiation of anticoagulation in patients of SMVT combined with PVT   
could minimise the serious complication such as peritonitis due to bowel   
necrosis required immediate exploratory laparotomy. © 2004 Elsevier Ltd. All   
rights reserved.  
  
RECORD 773  
Partial splenic embolization in patients with cirrhosis: Efficacy, tolerance   
and long-term outcome in 32 patients  
N'Kontchou G. Seror O. Bourcier V. Mohand D. Ajavon Y. Castera L.   
Grando-Lemaire V. Ganne-Carrie N. Sellier N. Trinchet J.-C. Beaugrand M.  
European Journal of Gastroenterology and Hepatology (2005) 17:2 (179-184).   
Date of Publication: February 2005  
  
Background: Although partial splenic embolization (PSE) has been proposed in   
patients with cirrhosis in cases when thrombocytopenia or neutropenia may   
cause clinical manifestations or if there are contra-indications to other   
therapeutic procedures, there are limited data on long-term outcome. We   
provide a retrospective review of results and the tolerance of all PSE   
procedures in patients with cirrhosis in our department. Patients and   
methods: Thirty-two consecutive patients with cirrhosis were included over a   
6 year period. Indications for PSE were as follows: (1) severe cytopenia   
preventing necessary antiviral treatment (n=14), percutaneous destruction of   
hepatocellular carcinoma (n=8) or major surgery (n=3), severe purpura (n=3);   
(2) painful splenomegaly (n=4). After superselective catheterization,   
embolization was performed with up to 50% reduction of splenic blood flow.   
Results: Thrombocyte and leucocyte counts increased markedly (185% and 51%   
at 1 month; 95% and 30% at 6 months). Thirty-one and 20 patients had   
platelet count >80 000/mm(3) at months 1 and 6 vs only one before PSE.   
Overall, the aim of PSE was achieved in 27 patients (84%) (planned   
treatment: 20/25; disappearance of purpura and splenic pain: 7/7). Severe   
complications occurred in five patients (16%): transient ascites (n=2),   
splenic and/or portal vein thrombosis (n=2) that resolved after   
anticoagulation therapy, and splenic abscess (n=2) leading to death. These   
two patients had splenic necrosis >70%. Conclusion: In patients with   
cirrhosis, PSE may resolve cytopenia and the clinical complications related   
to hypersplenism or splenomegaly. However, due to a high risk of severe   
complications, particularly splenic abscess, the indications of PSE should   
be very limited and the extent of necrosis should be strictly controlled   
during the PSE procedure. © 2005 Lippincott Williams & Wilkins.  
  
RECORD 774  
Unrecognized pylephlebitis causing life-threatening septic shock: A case   
report  
Wireko M. Berry P.A. Brennan J. Aga R.  
World Journal of Gastroenterology (2005) 11:4 (614-615). Date of   
Publication: 28 Jan 2005  
  
A man who developed profound septic shock was treated for Escherichia coli   
sepsis of unknown origin. Following stabilisation, a diagnosis of   
pylephlebitis (infection and thrombosis in the portal vein) was made at   
computed tomography. A review of the condition, its primary causes, typical   
features, investigation and management was presented. © 2005 The WJG Press   
and Elsevier Inc. All rights reserved.  
  
RECORD 775  
Review article: portal vein thrombosis - New insights into aetiology and   
management  
Webster G.J.M. Burroughs A.K. Riordan S.M.  
Alimentary Pharmacology and Therapeutics (2005) 21:1 (1-9). Date of   
Publication: 1 Jan 2005  
  
portal vein thrombosis may occur in the presence or absence of underlying   
liver disease, and a combination of local and systemic factors are   
increasingly recognized to be important in its development. Acute and   
chronic portal vein thrombosis have traditionally been considered   
separately, although a clear clinical distinction may be difficult.   
Gastrooesophageal varices are an important complication of portal vein   
thrombosis, but they follow a different natural history to those with portal   
hypertension related to cirrhosis. Consensus on optimal treatment continues   
to be hampered by a lack of randomized trials, but recent studies   
demonstrate the efficacy of thrombolytic therapy in acute thrombosis, and   
the apparent safety and benefit of anticoagulation in patients with chronic   
portal vein thrombosis.  
  
RECORD 776  
portal vein thrombosis: an unusual complication of laparoscopic   
cholecystectomy.  
Preventza O.A. Habib F.A. Young S.C. Penney D. Oppat W. Mittal V.K.  
JSLS : Journal of the Society of Laparoendoscopic Surgeons / Society of   
Laparoendoscopic Surgeons (2005) 9:1 (87-90). Date of Publication: 2005   
Jan-Mar  
  
BACKGROUND: Complications following laparoscopic cholecystectomy are   
encountered infrequently due to increasing proficiency in laparoscopic   
surgery. The occurrence of portal venous thrombosis following laparoscopic   
cholecystectomy has not been previously described and forms the basis of   
this report. METHODS: A healthy, 32-year-old, female on oral contraceptives   
underwent an uneventful laparoscopic cholecystectomy for symptomatic   
gallbladder disease. Sequential compression devices and mini-dose   
unfractionated heparin were used before the procedure. The patient was   
discharged home on the first postoperative day without complaints. She   
returned 1 week later with nausea, bloating, and diffuse abdominal pain.   
RESULTS: Ultrasonography of the abdomen revealed thrombosis of the portal   
vein not seen in the preoperative ultrasound and the superior mesenteric   
vein. Computer tomography of the abdomen and pelvis on the same day   
confirmed this finding and showed a wedge-shaped infarction of the right   
lobe of the liver. The patient was anticoagulated with intravenous heparin.   
An extensive coagulation workup revealed elevation of the Immunoglobulin G   
anticardiolipin antibody. A percutaneous transhepatic portal vein   
thrombectomy was performed. A postprocedure duplex ultrasound of the abdomen   
demonstrated recannalization of the portal venous system with no flow voids.   
anticoagulation therapy was continued, and the patient was discharged home   
with resolution of her ileus. She was maintained on a therapeutic dose of   
warfarin. CONCLUSIONS: This case demonstrates an unusual complication of   
laparoscopic cholecystectomy. It may have resulted from the use of oral   
contraceptives, elevation of the Immunoglobulin G anticardiolipin antibody,   
unrecognized trauma, and was accentuated by the pneumoperitoneum generated   
for the performance of the laparoscopic cholecystectomy. Our case report   
provides insight and poses questions regarding necessary perioperative   
measures for thromboprophylaxis in young females on oral contraceptives   
undergoing elective laparoscopic abdominal surgery.