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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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

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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.

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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

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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

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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

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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

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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

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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

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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.

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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

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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

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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

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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.

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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

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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

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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

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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

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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

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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

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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

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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

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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.

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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

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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

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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

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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

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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

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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)

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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

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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

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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

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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

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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

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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

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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

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'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

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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

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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

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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

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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

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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

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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

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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

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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, &#60;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

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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. ©

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RECORD 350

Usefulness of conventional mri sequences and diffusion-weighted imaging in

differentiating malignant from benign portal vein thrombus in cirrhotic

patients

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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?

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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

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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

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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

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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

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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

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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.

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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

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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

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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

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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

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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

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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

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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

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RECORD 389

Intrahepatic portal vein thrombosis: Is gastric surgery a risk factor?

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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

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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

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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

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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.

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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

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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.

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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. ©

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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

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(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,

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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.

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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

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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 &macro

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

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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

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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.

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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

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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?

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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

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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

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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

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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

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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

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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

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Casellas-Caro M. Condat B. Garcia-Pagán J.C. Janssen H.L. Valla D.

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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

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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

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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

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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

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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

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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.

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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

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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.

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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.

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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.

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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. ©

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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.

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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.

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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

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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. ©

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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

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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.

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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].

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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

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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

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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.

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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.

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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.

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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

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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

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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.