CIRRHOSIS OF LIVER

Cirrhosis is a chronic, degenerative disease in which normal liver cells are damaged and are then replaced by fibrous (scar) tissue. Scar tissue replaces healthy liver tissue, partially blocking the flow of blood through the liver. Thus, the liver is unable to function properly resulting in liver failure. The liver itself becomes distorted, hardened and lumpy.

Cirrhosis refers to chronic progressive liver disease It occurs when the normal flow of blood, bile and hepatic metabolities is altered by the following.

1. Fibrosis and changes in the hepatocytes

2. Bile ductules

3. Vascular channels

4. Reticular cells

Usually, cirrhosis develops insidiously with a prolonged, destructive course. As an end stage process, it is essentially an irreversible reaction to hepatic inflammation and necrosis.

Classification

1 **Laennec's cirrhosis/ Alcoholic Cirrhosis:** Also called Laennec's cirrhosis is the most common type and primarily caused by hepatitis C and chronic alcohol use. Liver damage results from malnutrition and chronic alcohol consumption. Fibrous tissue forms in portal area and around central veins. Most common type of cirrhosis. It is a massive loss of liver cells with irregular patterns of regenerating cells.

Etiology

- Alcoholism
- Malnutrition

Description

- Also known as alcohol induced, nutritional and portal cirrhosis
- Caused by long term use of alcohol
- Direct taxic effect on liver cells causing liver inflammation.
- Massive collagen formation occurs

2 **Post necrotic cirrhosis:** It comes from various types of hepatitis.

Etiology

- Post acute viral (types B and C hepatitis)

Description

- Occurs after massive liver cell necrosis
- Liver decreased in size with nodules and fibrous tissue

3**. Biliary cirrhosis:** Bile flow is decreased with concurrent cell damage to hepatocytes around bile ductules.

Etiology

- Chronic biliary obstruction
- Bile stasis
- Inflammation

Description

- Primary biliary cirrhosis. It results from intrahepatic bile stasis
- Secondary biliary cirrhosis It is caused by obstruction of the hepatic or common bile ducts.

4. **Cardiac cirrhosis:** It is caused by right sided congestive heart failure

Etiology

- Atrioventricular valve disease
- Prolonged constrictive pericarditis
- Decompensated or pulmonale

Description

- Caused by severe or chronic heart failure
- Liver becomes enlarged and congested with venous blood resulting in cell necrosis from anoxia

Etiology

The exact causes of cirrhosis has not been clearly identified.

1. There is a genetic component with a familial tendency to develop cirrhosis as well as familial, hypersensitivity to alcohol in some people

2. Alcoholism and malnutrition are two major pre- disposing factors for the development of Laennec's cirrhosis.

3. Different types of hepatitis can cause post necrotic cirrhosis.

4. Less common causes of cirrhosis are:

a. Right sided congestive heart failure.

b. Hemochromatosis

c. Cystic fibrosis

d. Glycogen storage disease

Risk Factors

1. Alcohol is the primary risk factor for cirrhosis, especially in the absence of proper nutrition.

2. Viral hepatitis

3. Obesity

Pathophysiology

Due to etiological factors

⬇

Diffuse destruction and regeneration of liver cells

⬇

Formation of fibrous tissue

⬇

Destroyed liver cells are replaced gradually by scar tissues

⬇

As necrotic tissue yields fibrosis, cirrhosis damage liver tissue

⬇

Obstruction in blood, lymph and bile flow

⬇

Hepatic Insufficiency

Clinical Manifestations

1. Gastrointestinal system:

- Nausea and vomiting.
- Anorexia
- Diarrhea
- Indigestion
- Dyspepsia
- Change in bowel habits
- Dull abdominal pain
- Fetor hepaticus - Fetor hepaticus means “fetid liver” or “liver stench”. It's a type of chronic bad breath that's actually a symptom of liver disease. It has a distinctive smell — some say, like rotten eggs and garlic
- Esophageal and gastric varices
- Hematemesis
- Hemorrhoidal varices
- Congestive gastritis

2. Hematological:

- Bleeding
- Anemia
- Thrombocytopenia
- Leukopenia
- Coagulation disorders
- Splenomegaly

3. Neurological:

- Asterixis - Asterixis is a clinical sign that describes the inability to maintain sustained posture with subsequent brief, shock-like, involuntary movements.
- Hepatic encephalopathy
- Peripheral neuropathy

4. Reproductive system:

- Impotence
- Amenorrhea
- Testicular atrophy

5. Cardiovascular system:

- Ascites
- Peripheral edema
- Fluid retention

Integumentary

- Jaundice
- Spider angioma- is a vascular lesion characterized by anomalous dilatation of end vasculature found just beneath the skin surface.
- Palmar erythema - Palmar erythema is a rare ailment that causes the hands' palms to turn red.
- Purpura
- Petechiae
- Caput medusae - Caput medusae is the appearance of distended and engorged superficial epigastric veins, which are seen radiating from the umbilicus across the abdomen.

Metabolic

- Hypokalemia
- Hyponatremia
- Hypoalbuminemia

Clinical manifestations in Early Stages

- ✓ Loss of appetite
- ✓ Weight loss
- ✓ Enlarged liver
- ✓ Vascular spider
- ✓ Palmer Erythema
- ✓ Ankle edema
- ✓ Nail changes
- ✓ Vitamin deficiency
- ✓ Nausea and vomiting
- ✓ Mild fever
- ✓ Abdominal pain
- ✓ Abnormal pigmentation
- ✓ Intense itching (pruritus)
- ✓ Morning indigestion
- ✓ Lack of energy and tiredness
- ✓ Anemia

Clinical manifestations in Late Stages

- ✓ Ascites
- ✓ Bruising and bleeding
- ✓ Reddening of the palms of the hands
- ✓ Gallstones
- ✓ Frequent attacks of indigestion
- ✓ Oesophagus Varices
- ✓ Asterixis
- ✓ Hepatic encephalopathy
- ✓ Menstrual irregularities
- ✓ Personality change
- ✓ Jaundice
- ✓ Spider naevi: spiderlike blood vessels on skin
- ✓ Distended veins in the abdomen
- ✓ Enlarged breast tissue in men (gynaecomastia)
- ✓ Abdominal pain and bloating
- ✓ Mental disturbances
- ✓ Slurred speech

- ✓ Dark tea colored urine
- ✓ Gonadal atrophy
- ✓ Poor concentration

Diagnostic Evaluation

1. **Radioisotope liver scan:** It helps to identify abnormal thickening and a liver mass.

2. **Abdominal X-rays:** It may reveal the following:

a. Enlarged liver.

b. Gas or cysts within the liver and biliary tract.

c. Massive ascites.

d. Calcification of liver

3. **CT scan:** CT scan is helpful in detecting minimal ascites and provides information about the volume and character of fluid collection.

4. **Ultrasound and MRI:** It helps to confirm the diagnosis of cirrhosis through visualization of masses, abnormal growth and metastasis.

5. **Esophagogastroduodenoscopy (EGD):** Performed to directly visualize the upper gastrointestinal tract and also to detect the presence of bleeding, stomach irritation and ulceration, duodenal ulceration and bleeding.

6. A physician uses liver biopsy as the definitive test for cirrhosis. It is performed to identify destruction and fibrosis hepatic tissue.

7. **Complete Blood Count (CBC):** CBC will show decreased white blood cell, hemoglobin level, hematocrit, albumin and platelets.

Management

Medical Management

There are three goals which guide the medical management of a patient with cirrhosis.

1. Maximize liver function.

2. Prevent infection.

3. Control disabling complications.

Non-pharmacologic Interventions

1. Bed rest.

2. A high carbohydrate, low sodium, low fat diet with vitamin supplement and with protein content ad- justed according to blood urea nitrogen and serum ammonia levels.

3. Monitoring of serum ammonia levels, prothrombin time and bilirubin.

Pharmacologic Interventions

1. **Diuretics such as chlorothiazide (Diuril**): It is prescribed to increase urinary sodium and water excretion which helps to reduce ascites.

2. **More potent diuretics such as furosemide (Lasix and ethacrynic acid (Edecrin):** Sometimes necessary to help rid the body of excessive abdominal ascetic fluid.

3. **Antihistamines:** They are recommended to alleviate pruritus.

4. **Potassium supplements:** To correct hypokalemia.

5. **Corticosteroids:** It may be used for post necrotic cirrhosis. It helps to reduce signs and symptoms of cirrhosis and improve liver function.

6. **Octreotide:** It is prescribed for esophageal varices.

7. **Antibiotics:** Recommended to decrease intestinal bacteria and reduce ammonia production.

8. **Vitamin B and fat soluble vitamins, such as A, D, E, K: They** are commonly given to patient with Laennec's cirrhosis.

9. Patients with cirrhosis often require antacid therapy for gastrointestinal symptoms because most antacids are high in sodium. A physician prescribed low sodium antacid such as magaldrate (Riopan) and aluminium hydroxide gel (Amphojel) to patient.

Surgical Management

Liver Transplant

1. When the complications cannot controlled by treatment, liver transplant is required.

2. It is a major surgery in which patient's diseased liver is removed and replaced with a healthy one from an organ donor

3. In certain cases, portion of the liver of a living related donor may be used.

Transjugular Intrahepatic Portosystemic Shunt (TIPS)

1 It is performed under two conditions:

a. Abdominal fluid which does not respond to standard medical management.

b. Acute bleeding from esophageal varices unresponsive to standard medical or endoscopic treatment.

2. During this procedure, a flexible tube, 1.e. catheter is inserted through the skin into a vein in patient's neck. With the help of X-ray machine, doctor guides the catheter into a vein in patient's liver. Dye is injected into vein to look inside clearly Balloon is inflated to place the stent. Stent is used to connect portal vein to one of hepatic vein. At the end, portal vein is measured to ensure that it has gone down. Then, the catheter with balloon is removed. After procedure, a small bandage is placed over the patient's neck area.

Paracentesis

1. Paracentesis is indicated if dietary restriction and the administration of drugs fail to control ascites.

2. This procedure simply takes fluid out from the abdominal cavity

3. During this surgical procedure, a soft catheter is inserted into patient's abdomen to remove and drain ascetic fluid in the patient's peritoneal cavity

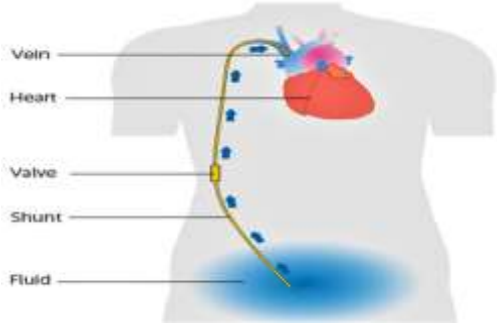
4. Repeated paracentesis procedures are contra- indicated because of the increased incidence of protein, depletion, hypovolemia and electrolyte imbalance.

5. **Peritoneovenous shunt:**

a. Insertion of a peritoneovenous shunt to redirect ascetic fluid from the peritoneal cavity into the systemic circulation is a treatment modality for ascites, but this procedure is not used because of high complications rate and high incidence of shunt failure.

Peritoneovenous shunt is a device that relieves ascites by transferring fluid through a one-way valve from the peritoneal cavity into the superior vena cava

b. Shunt is reserved for those who are resistant to diuretic therapy, are not candidates for liver transplantation, have abdominal adhesions or are ineligible for other procedures because of severe medical condition, such as cardiac disease.



Nursing Management

1. Obtain patient's complete medical history

2. Determine whether there is history of alcoholism in the family or not.

3. Assess patient for the presence of any of the early symptoms such as hepatomegaly and carefully observe the laboratory data.

4. Inspect the extremities and the sacrum for the presence of peripheral, dependent edema.

5. Position bed for maximal respiratory efficiency, provide oxygen, if needed. 6. Observe and document for bleeding gums, jaundice, epistaxis, petechiae, ecchymosis, etc.

7. Inspect stool for amount, color and consistency.

8. Encourage patient to eat well balanced, high calorie, high carbohydrate diet with adequate vitamins to provide nutrients for the repair of liver

9. Frequent oral hygiene and a pleasant environment should be provided.

10. Change patient's position frequently

11. Use padded side rails, if patient becomes agitated or restless.

12. Administer diuretics, potassium and vitamin supplements as ordered.

13. Carefully evaluate any injury because of the possibility of internal bleeding.

14. Patient is instructed to notify physician immediately if any evidence of gastrointestinal bleeding is noted, so that re-evaluation can be initiated quickly.

COMPLICATIONS OF LIVER CIRRHOSIS

1. Portal hypertension
2. Peripheral edema and Ascites
3. Hepatic encephalopathy
4. Hepatorenal syndrome
5. Esophageal and gastric varices
6. Hepatic failure
7. Splenomegaly
8. Malnutrition
9. Hemorrhage

PORTAL HYPERTENSION

Portal hypertension is also a vascular disease. Hypertension or high blood pressure is a disease in which pressure of increases in blood. When blood pressure increases in portal vein and its branches then the condition known as Portal hypertension. Portal vein is the large blood vessel that carries blood from the abdominal organs (digestive system) to the liver.

Portal hypertension is defined as portal pressure gradient of 12 mm Hg or greater and is often associated with varices and ascites. Portal pressure gradient can be defined as the difference in pressure between the portal vein and the hepatic veins.

High blood pressure in the portal vein or portal venous system is known as portal hypertension. Obstructed blood flow through the damaged liver results in increased blood pressure throughout the portal venous system. It is the leading side effect of cirrhosis. Major consequences of portal hypertension are as follows:

1. **Ascites:** Fluid accumulation in the abdominal cavity.

2. **Varices:** Varicosities which develop from elevated pressure transmitted to all veins that drain into portal system.

Etiology

Causes of portal hypertension according to site of vascular obstruction

A. Pre-hepatic pre-sinusoidal

B. Intrahepatic pre-sinusoidal

C. Sinusoidal

D. Intrahepatic post-sinusoidal

E. Post-hepatic post-sinusoidal

Pre-hepatic pre-sinusoidal

- Portal vein thrombosis due to sepsis (umbilical, portal pyemia) or procoagulopathy or secondary to cirrhosis
- Abdominal trauma including surgery

Intrahepatic pre-sinusoidal

- Schistosomiasis
- Congential hepatic fibrosis
- Drugs
- Vinyl chloride
- Sarcoidosis

Sinusoidal

- Cirrhosis
- Polycystic liver disease
- Nodular regenerative hyperplasia
- Metastatic malignant disease

Intrahepatic post-sinusoidal

- Veno-occlusive disease

Post-heaptic post-sinusoidal

- Budd-Chiari syndrome

Risk Factors

1. Cirrhosis

2. Congestive heart failure

3. Arteriovenous malformations.

Pathophysiology

The pressure in any vascular system is directly proportional to the flow and resistance in that system. Portal hypertension develops due to:

- *Increased vascular resistance in the portal venous system* - from various mechanical causes, and also as an active process, in which liver damage activates stellate cells and myofibroblasts, contributing to the abnormal blood flow patterns.
- Increased blood flow in the portal veins - from splanchnic arteriolar vasodilatation, caused by an excessive release of endogenous vasodilators.
  The raised portal pressure opens up venous collaterals, connecting the portal and systemic venous systems. These occur in various sites:
- *Gastro-oesophageal junction* - producing varices which are superficial and easily bleed
- *Anterior abdominal wall:* Via the umbilical vein visible as caput medusae radiating from the umbilicus. May also occur where adhesions exist between abdominal viscera and the parietal peritoneum, or at sites of stomas or previous surgery
- *Anorectal junction* - rarely cause bleeding.
- *Veins from retroperitoneal viscera* - communicate with systemic veins on the posterior abdominal wall.

Portal hypertension and cirrhosis produce a hyperdynamic circulation, with splanchnic vasodilatation, increased cardiac output, arterial hypotension, and hypervolaemia. There is salt and water retention, ascites and hyponatraemia.

Clinical Manifestations.

1 Slightly tortuous epigastric vessels that branch the area of the umbilicus and lead towards the sternum and ribs.

2. Ascites, which typically appears when there is concurrent liver disease.

3. Enlarged veins of the esophagus and stomach.

4. Increased abdominal girth and abdominal pain.

5. Fluid and electrolyte imbalances are common.

6. Jaundice

7 Shortness of breath.

8. Reduced platelet count.

9. Umbilical hernia.

10. Gynecomastia

Diagnostic Evaluation

1. **Blood tests:** Low platelet count is the most common blood test as a sign of portal hypertension.

2. On percussion of the abdomen, a fluid wave is likely to be found only when a large amount of fluid is present.

3. Daily measurement and recording of abdominal girth and body weight assess the progression of ascites and respond to treatment.

4. **Abdominal ultrasound:** Performed to detect the size of liver and spleen, ascites, portal blood flow and thrombosis of the portal and splenic veins.

5. **Doppler ultrasound:** It can show the direction of flow in blood vessels.

6**. CT scan:** Rule out portal vasculatures.

7. **Liver biopsy:** It helps to diagnose the underlying cause of portal hypertension.

8. **Hepatic venography:** It is used when hepatic vein block or idiopathic portal hypertension is suspected

Management

Medical Management

1. Beta blockers, such as propranolol:

a. Prescibed to reduce portal pressure.

b. Helps to reduce bleeding or rebleeding in patients with esophageal varices.

2. Vasopressin (Pitressin):

a. Its administration achieves temporary lowering of portal pressure. These agents reduce portal blood flow by constricting afferent arterioles.

b. Direct infusion of vasopressin into the superior mesenteric artery is most effective.

c. Sometimes, it is administered intravenously

3. Vitamin K: Prescribed for gastrointestinal bleeding.

4. Diuretics:

a. Use of diuretics along with sodium restriction is successful in 90% of patients with ascites.

b. Spironolactone (Aldactone): It is an aldosterone blocking agent. It is most often the first line therapy in patients with ascites from cirrhosis.

c. Furosemide (Lasix): It may be used cautiously because with long term use, it may induce severe sodium depletion.

d. Ammonium chloride and acetazolamide (Diamox): They are contraindicated because of the possibility of precipitating hepatic coma.

4. Endoscopic therapy: This is usually the first line of treatment for variceal bleeding and consists of either banding or sclerotherapy. Banding is a procedure in which a gastroenterologist uses rubber bands to block off the blood vessel. Sclerotherapy is occasionally used when banding cannot be used and is a procedure in which a sclerosant solution (ethanolamine oleate or sodium tetradecyl sulphate) is injected into the bleeding varices to cause them to scar.

Other Treatment Options

For those with portal hypertension, if endoscopic therapy, drug therapy, and/or dietary changes do not successfully control variceal bleeding, may require one of the following procedures to reduce the pressure in these veins. Decompression procedures include:

1. ***Transjugular Intrahepatic Portosystemic Shunt (TIPS***): This procedure involves placing a stent (a tubular device) in the middle of the liver. The stent connects the hepatic vein with the portal vein. During the TIPS procedure, a radiologist makes a tunnel through the liver with a needle, connecting the portal vein to one of the hepatic veins. A metal stent often covered with a thin plastic material, is placed in this tunnel to keep the tunnel open. The procedure reroutes blood flow in the liver and reduces pressure in all abnormal veins, not only in the stomach and esophagus, but also in the bowel and the liver.

2. Distal Splenorenal Shunt (DSRS): The DSRS is a surgical procedure during which the vein from the spleen (splenic vein) is detached from the portal vein and attached to the left kidney (renal) vein. This surgery selectively reduces the pressure in the varices and controls the bleeding associated with portal hypertension. It is usually performed only in patients with good liver function.

3. Devascularization: A surgical procedure that removes the bleeding varices. This procedure is done when a TIPS or a surgical shunt is not possible or is unsuccessful in controlling the bleeding.

4. Liver transplant: This is done in cases of end-stage liver disease.

5. Paracentesis: This is a procedure in which the accumulation of fluid in the abdomen (ascites) is directly removed.

Procedure of paracentesis

Pre-procedure

- Explain the procedure to patient
- Reassure the patient
- Instruct the patient to void, if he/she desires.
- Collect all the articles
- Place the patient in upright position on the edge of bed with feet supported on stool (sit on chair)

Or

- Fowler's position for patient confined to bed
- Place BP cuff on patient's arm to monitor BP during procedure

Procedure:

- Following the aseptic technique, doctor will insert the trocar through a puncture wound below the umbilicus. The fluid drains from the abdomen through a drainage tube into a container
- Help the patient for maintaining position through the procedure
- Monitor and record the BP at frequent interval beginning from the procedure
- Monitor the patient closely for vascular collapse pallor, tachycardia, decreased BP

Post procedure

- Make the patient comfortable by returning back to bed
- Record the amount, color, quality of the collected fluid.
- Label the sample and send to laboratory.
- Monitor vital signs: Every 15 minutes for 1 hour. Every 30 minutes over 2 hours. Every hour over 2 hours. Then, every four hours
- Observe the puncture site for bleeding and leakage.

- Assess the patient for fluid and electrolyte loss, change in mental status, etc.

Teaching at the time of discharge:

- Monitor the puncture site for bleeding, excess drain.
- Avoid heavy lifting.
- Change position very slowly.

1 Paracentesis is the removal of fluid (ascites) from the peritoneal cavity through a small surgical incision or puncture made through the abdominal wall under sterile conditions.

2. Ultrasound guidance may be indicated in some patients at high risk for bleeding.

3. For analysis, a sample of fluid may be sent to laboratory

4. Cell count, albumin and total protein levels, culture and occasionally other tests are performed.

6. Peritoneovenous Shunt

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Nursing Management

1. Assess the respiratory rate of patient.

2. Monitor vital signs.

3. Observe patient's abdomen for striae, umbilical hernia and distended veins.

4. Enquire patient about alcohol intake.

5. Provide fowler's position to patient.

6. Encourage patient to take deep breaths.

7. Change patient's position 2 hourly

8. Assess the pain and discomfort level of patient.

9. Promote bed rest.

10. Provide diet high in carbohydrates and low in sodium.

11. Provide small and frequent meals.

12. Maintain skin hygiene.

13. Always involve family members in doing patient care.

Complications

1. Serious kidney problems.

2. Internal bleeding.

3. Fluid buildup in the stomach.

Peripheral Edema and Ascites. Peripheral edema occurs in the lower extremities and presacral area. Peripheral edema can occur before, concurrently with, or after ascites development. Edema results from decreased colloidal oncotic pressure from impaired liver synthesis of albumin and increased portacaval pressure from portal hypertension.

PERIPHERAL EDEMA AND ASCITES

Peripheral edema occurs in the lower extremities and presacral area. Peripheral edema can occur before, concurrently with, or after ascites development. Edema results from decreased colloidal oncotic pressure from impaired liver synthesis of albumin and increased portacaval pressure from portal hypertension.

Ascites is the accumulation of serous fluid in the peritoneal or abdominal cavity. It is a common manifestation of cirrhosis.

Several mechanisms lead to ascites.

One mechanism of ascites occurs with portal hypertension, which causes proteins to shift from the blood vessels into the lymph space

- When the lymphatic system is unable to carry off the excess proteins and water, they leak into the peritoneal cavity.
- The osmotic pressure of the proteins pulls additional fluid into the peritoneal cavity

A second mechanism of ascites formation is,

- hypoalbuminemia resulting from the liver's decreased ability to synthesize albumin. The hypoalbuminemia results in decreased colloidal oncotic pressure.

A third mechanism of ascites is hyperaldosteronism,

- which occurs when the hormone aldosterone is metabolized by damaged hepatocytes. The increased level of aldosterone causes increased sodium reabsorption by the renal tubules.
- This retention of sodium, combined with an increase in antidiuretic hormone in blood, leads to additional water retention.
- Because of edema formation, there is decreased intravascular volume and, subsequently, decreased renal blood flow and glomerular filtration.

Ascites is manifested by abdominal distention with weight gain (Fig. 40-8). If the ascites is severe, the increase in abdominal pressure from the fluid accumulation may cause eversion of the umbilicus.

Abdominal striae with distended abdominal wall veins may be present.

Patients may show signs of dehydration (e.g., dry tongue and skin, sunken eyeballs, muscle weakness) and a decrease in urine output.

Hypokalemia is common and is due to an excessive loss of potassium caused by hyperaldosteronism. Low potassium levels can also result from diuretic therapy used to treat the ascites.

Management of ascites

- Management of ascites focuses on sodium restriction, diuretics, and fluid removal.
- Patients may be encouraged to limit sodium intake to 2 g/day. Patients with severe ascites may need to restrict their sodium intake to 250-500 mg/day. Very low sodium intake can result in reduced nutritional intake and subsequent problems associated with malnutrition.
- The patient is usually not on restricted fluids unless severe ascites develops. When caring for patients with ascites, accurately assess and monitor fluid and electrolyte balance.
- Albumin infusion may be used to help maintain intravascular volume and adequate urine output by increasing plasma colloid oncotic pressure

- Diuretic therapy is an important part of management. Often a combination of drugs that work at multiple sites of the nephron is more effective than a single agent. Spironolactone is an effective diuretic, even in patients with severe ascites. Spironolactone is also an antagonist of aldosterone and is potassium sparing. Other potassium-sparing diuretics include amiloride and triamterene. A high-potency loop diuretic, such as furosemide, is frequently used in combination with a potassium-sparing drug.
- Paracentesis
- TIPS

HEPATIC ENCEPHALOPATHY.

- Hepatic encephalopathy is a neuropsychiatric manifestation of liver disease.
- The pathogenesis is multifactorial and includes the neurotoxic effects of ammonia, abnormal neurotransmission, astrocyte swelling, and inflammatory cytokines.
- A major source of ammonia is the bacterial and enzymatic deamination of amino acids in the intestines.
- The ammonia that results from this deamination process normally goes to the liver via the portal circulation and is converted to urea, which is then excreted by the kidneys.
- When blood is shunted past the liver via the collateral vessels or the liver is so damaged that it is unable to convert ammonia to urea, the levels of ammonia in the systemic circulation increase.
- The ammonia crosses the blood- brain barrier and produces neurologic toxic manifestations.
- Hepatic encephalopathy can occur after placement of transjugular intrahepatic portosystemic shunt (TIPS), which is used to reduce portal hypertension by diverting blood flow around the liver.

Pathophysiology

Due to etiological factors

⬇

Damaged liver cells fail to detoxify and convert ammonia into urea

⬇

Ammonia accumulates in the body

⬇

Ammonia enter the blood stream and ammonia blood level rises

⬇

Increased permeability of blood - brain barrier to ammonia

⬇

Ammonia is delivered to brain and cause brain damage and dysfunction

⬇

Hepatic Encephalopathy

Staging Of Hepatic Encephalopathy

In patients with cirrhosis and overt encephalopathy, two staging classifications have been used for patients with hepatic encephalopathy.

Stage 0

- Lack of detectable changes in personality or behavior
- Asterixis absent

Stage 1

- Trivial lack of awareness
- Shortened attention span
- Impaired addition or subtraction
- Hypersomnia, insomnia, or inversion of sleep pattern
- Euphoria or depression
- Asterixis can be detected.

Stage 2

- Lethargy or apathy
- Disorientation
- Inappropriate behavior
- Slurred speech
- Obvious asterixis

Stage 3

- Gross disorientation
- Bizarre behavior
- Semistupor to stupor
- Asterixis generally absent

Stage 4

Coma with or without response to painful stimuli

Clinical manifestations of encephalopathy are changes in neurologic and mental responsiveness; impaired consciousness; and inappropriate behavior, ranging from sleep disturbances to trouble concentrating to deep coma.

- Changes may occur (1) suddenly because of an increase in ammonia in response to bleeding varices or infection or (2) gradually as blood ammonia levels slowly increase.
- A characteristic manifestation of hepatic encephalopathy is asterixis (flapping tremors). This may take several forms, with the most common involving the arms and hands. When asked to hold the arms and hands stretched out, the patient is unable to hold this position and performs a series of rapid flexion and extension movements of the hands.
- Impairments in writing involve difficulty in moving the pen or pencil from left to right and apraxia (inability to construct simple figures).
- Other signs include hyperventilation, hypothermia, tongue fasciculations, and grimacing and grasping reflexes. Fetor hepaticus (musty, sweet odor of the patient's breath) occurs in some patients with encephalopathy. This odor is from the accumulation of digestive by-products that the liver is unable to degrade.

Management of hepatic encephalopathy

- The goal of management of hepatic encephalopathy is the reduction of ammonia formation. Ammonia formation in the intestines is reduced with *lactulose, a drug that traps ammonia in the gut. It can be given orally, as an enema, or through a nasogastric (NG) tube*. The laxative effect of the drug expels the ammonia from the colon.
- *Antibiotics* such as rifaximin may also be given, particularly in patients who do not respond to lactulose.
- *Constipation should be prevented*, and regular and frequent bowel movements are necessary to minimize the ammonia buildup.

- Control of hepatic encephalopathy also involves treatment of precipitating causes. This includes *lowering one's dietary protein intake, preventing and controlling GI bleeds, and in the event of a bleed, removing the blood promptly from the GI tract to decrease the protein accumulation in the gut*

HEPATORENAL SYNDROME.

- Hepatorenal syndrome is a type of renal failure with azotemia, oliguria, and intractable ascites. In this syndrome, the kidneys have no structural abnormality.
- The etiology is complex, but the final common pathway is likely to be portal hypertension along with liver decompensation, resulting in splanchnic and systemic vasodilation and decreased arterial blood volume. As a result, renal vasoconstriction occurs, and renal failure follows.
- This renal failure can be reversed by liver transplantation. In the patient with cirrhosis, hepatorenal syndrome frequently follows diuretic therapy, GI hemorrhage, or paracentesis.

ESOPHAGEAL VARICES

Esophageal varices are abnormal, enlarged veins in the lower part of the esophagus. Esophageal varices occur most often in people with serious liver diseases. Esophageal varices develop when normal blood flow to liver is slowed. The blood then backs up into nearby smaller blood vessels, such as those in esophagus, causing the vessels to swell. Sometimes, esophageal varices can rupture, causing life-threatening bleeding.

Causes of Esophageal Varices

Pre-hepatic

- Portal vein thrombosis
- Portal vein obstruction - congenital atresia/stenosis
- Increased portal blood flow - fistula
- Increased splenic flow

Intra-hepatic

- Cirrhosis due to various causes, including alcoholic, chronic hepatitis (e.g. viral or autoimmune)
- Idiopathic portal hypertension (hepatoportal sclerosis)
- Acute hepatitis (esp. alcoholic)
- Schistosomiasis
- Congenital hepatic fibrosis
- Myelosclerosis

Post-hepatic

- Compression (e.g. from tumour)
- Budd-Chiari syndrome
- Constrictive pericarditis (and rarely right-sided heart failure)

Factors that increase the risk of Variceal Bleeding

- Decompensation of liver disease
- Malnourishment
- Alcohol intake
- Physical exercise
- Circadian rhythms
- Increased intra-abdominal pressure
- Aspirin
- NSAIDS
- Bacterial infection (cause of initial, and recurrence of, bleeding)

Pathophysiology

Portal hypertension (caused by resistance to portal flow and increased portal venous inflow)

⬇

Development of pressure gradient of 12 mmHg or greater between portal vein and inferior vena cava (portal pressure gradient)

⬇

Venous collaterals develop from high portal system pressure to systemic veins in esophageal plexus, hemorrhoidal plexus, and retroperitoneal veins

⬇

Abnormal varicoid vessels form in any of above locations

⬇

Vessels may rupture causing life-threatening hemorrhage

Clinical Manifestations

Esophageal varices usually don't cause signs and symptoms unless they bleed.

Symptoms

- Haematemesis most commonly
- Abdominal pain
- Dysphagia / odynophagia (pain on swallowing; uncommon) Confusion secondary to encephalopathy (may even be obtunded)
- Symptoms of anemia and shock in severe cases
- Vomiting blood, blood in the stool, black tarry stool, and red or maroon stool.

Signs

- Pale
- Peripherally shut down
- Pallor
- Hypotension and tachycardia (i.e. shock)
- Reduced urine output
- Melaena
- Signs of chronic liver disease
- Reduced Glasgow Coma Scale
- Signs of sepsis may also commonly be present

Diagnostic Evaluations

- CBC - haemoglobin may be low, MCV may be below, WCC may be raised high, normal or low, platelets may also
- Clotting including INR
- Renal function
- Liver function tests
- Group and cross-match
- CXR - patients may have aspirated or have chest sepsis
- Ascitic tap may be needed if bacterial peritonitis suspected
- Computerized tomography (CT) scans and magnetic resonance imaging (MRI)

Management

Treatment for esophageal varices involves reducing the risk of bleeding from the varices. Treatment for esophageal varices includes avoidance of alcohol, medications to reduce the risk of bleeding, and surgical procedures to stop variceal bleeding.

Several treatments can help lower the risk of vessel rupture or to stop bleeding if it starts. Treatment options include the following:

1 Endoscopic Band Ligation: During endoscopic band ligation, a rubber band is tied around the bulging veins to prevent ruptures or stop bleeding. This is considered the first line treatment

Endoscopic Variceal Ligation (Esophageal Banding Therapy)

- In variceal banding also referred to as EVL, a modified endoscope loaded with an elastic rubber band is passed through an overtube directly onto the varix (or varices) to be banded.
- After the bleeding varix is suctioned into the tip of the endoscope, the rubber band is slipped over the tissue, causing necrosis, ulceration, and eventual sloughing of the varix.
- Variceal banding is comparable to endoscopic sclerotherapy in its effectiveness in controlling acute bleeding.
- Esophageal band ligation has replaced sclerotherapy as the treatment of choice in the management of esophageal varices.
- Complications include superficial ulceration and dysphagia, transient chest discomfort, and, rarely, esophageal strictures.
- Band ligation in combination with pharmacologic therapy may be more effective than monotherapy (i.e., a single mode of therapy) in the treatment of acute hemorrhage.
- EVL is recommended for patients who have experienced variceal bleeding while receiving beta-blocker therapy and for those who cannot tolerate beta-blocking agents

2. Balloon Tamponade: This procedure involves the passage of a balloon through the nose to help compress the bleeding varices.

3. Sclerotherapy: During sclerotherapy, drugs intended to slow bleeding are injected into the bleeding vein and sometimes into the surrounding area. The drugs cause clots to form and harden the vein to stop bleeding.

Endoscopic Sclerotherapy

- In endoscopic sclerotherapy also referred to as injection sclerotherapy, a sclerosing agent (i.e., sodium morrhuate, ethanolamine oleate, sodium tetradecyl sulfate, or ethanol) is injected through a fiberoptic endoscope into or adjacent to the bleeding esophageal varices to promote thrombosis and eventual sclerosis
- The process of sclerotherapy causes inflammation of the involved vein with eventual thrombosis and loss of the lumen of the vessel.
- The procedure has been used successfully to treat acute GI hemorrhage but is not recommended for prevention of first and subsequent variceal bleeding episodes where endoscopic variceal ligation (EVL), also known as esophageal banding therapy (discussed later), is the first-line treatment
- After treatment for acute hemorrhage, the patient must be observed for bleeding, perforation of the esophagus, aspiration pneumonia, and esophageal stricture.
- Antacids, histamine-2 (H_2) antagonists such as cimetidine (Tagamet), or proton pump inhibitors such as pantoprazole (Protonix) may be given after the procedure to counteract the chemical effects of the sclerosing agent on the esophagus and the acid reflux associated with the therapy.

4 Drug Therapy: Drugs used to relieve blood pressure include terlipressin, vasopressin, nitroglycerin, octreotide, and somatostatin.

5. Transjugular Intrahepatic Portosystemic Shunting (TIPS): TIPS involves threading a catheter from a neck vein to the liver. A stent, a small tube designed to keep veins open, is bound to the catheter and inserted into the liver to increase blood flow through the portal vein and relieve blood pressure in esophageal varices. This procedure can control bleeding in over 90% of cases.

Additional Therapies

- The use of endoscopically placed tissue adhesives and fibrin glue has been successful in the treatment of gastric and esophageal varices.
- Coated expandable stents (placed via endoscope) have also been used effectively for the same purpose.

Surgical Management

Surgical Bypass Procedures

(A) Portocaval shunt. The portal vein is anastomosed to the inferior vena cava, diverting blood from the portal vein to the systemic circulation.

(*B) Distal splenorenal shunt.* The splenic vein is anastomosed to the renal vein. The portal venous flow remains intact while esophageal varices are selectively decompressed. (The short gastric veins are decompressed.) The spleen conducts blood from the high pressure of the esophageal and gastric varices to the low-pressure renal vein

Devascularization and Transection

- Devascularization and staple-gun transection procedures to separate the bleeding site from the high-pressure portal system have been used in the emergency management of variceal bleeding.
- The lower end of the esophagus is reached through a small gastrostomy incision; a staple gun permits anastomosis of the transected ends of the esophagus.
- Rebleeding is a risk, and the outcomes of these procedures vary among patient populations.

Liver Transplant: Liver transplantation is the only way to completely cure esophageal varices.

Prevention

1***. Don't drink alcohol.*** People with liver disease are often advised to stop drinking alcohol, since alcohol is processed by the liver. Drinking alcohol may stress an already vulnerable liver.

2. Eat a healthy diet. Choose a plant-based diet that's full of fruits and vegetables. Select whole grains and lean sources of protein. Reduce the amount of fatty and fried foods you eat.

3. Maintain a healthy weight. An excess amount of body fat can damage your liver Lose weight if you are obese or overweight.

4. Use chemicals sparingly and carefully. Follow the directions on household chemicals, such as cleaning supplies and insect sprays. If you work around chemicals, follow all safety precautions. Your liver removes toxins from your body, so give it a break by limiting the amount of toxins it must process.

5. Reduce risk of hepatitis. Sharing needles and having unprotected sex can increase your risk of hepatitis B and C. Protect yourself by abstaining from sex or using a condom if you choose to have sex. Ask your doctor whether you should be vaccinated for hepatitis B

LIVER OR HEPATIC FAILURE

Liver failure is the inability of the liver to perform its normal synthetic and metabolic function as a part of normal physiology. It is a life threatening condition that demands urgent medical care.

Types of Liver Failure

1. Acute liver failure (Fulminant liver failure).

2. Chronic liver failure (End stage liver disease).

ACUTE LIVER FAILURE

Acute liver Failure (ALF) is defined as, "The rapid development of hepatocellular dysfunction, specifically coagulopathy and mental status changes (encephalopathy) in a patient without known prior (encephalopathy) in a patient without known prior liver disease" It is also known as fulminant liver failure

It is a medical emergency that requires immediate medical attention.

Classification

Acute liver failure is divided into three categories which is based on the interval between development of jaundice and onset of encephalopathy (see table 5.15).

1. Hyperacute liver failure: Onset of encephalopathy is less than 7 days after the development of jaundice.

2. Acute liver failure: Onset of encephalopathy is 8 to 28 days after the development of jaundice.

3. Sub-acute liver failure: Onset of encephalopathy is more than 5 weeks but less than 12 weeks after development of jaundice.

Etiology

1. Hepatitis and other viruses: Hepatitis A, hepatitis B and hepatitis E can cause acute liver failure. Other viruses include Epstein Barr virus, cytomegalovirus and herpes simplex virus.

2. Taking 2 much acetaminophen causes most cases of acute liver failure.

3. Vascular diseases, such as Budd-Chiari Syndrome.- Budd-Chiari syndrome (BCS) is an uncommon disorder characterized by obstruction of hepatic venous outflow. The obstruction may be thrombotic or non-thrombotic

4. Metabolic disease, such as wilson's disease and acute fatty liver of pregnancy

5. Less common causes of acute liver failure: Poisoning with amanita muscaria and liver failure associated with eclampsia or preeclampsia of pregnancy

Risk Factors

1. Alcohol

2. Having poor blood flow to the liver

3. Taking two much acetaminophen.

Clinical Manifestations

1. Loss of appetite

2. Nausea

3. Diarrhea

4. Disorientation or confusion

5. Jaundice

6. Bruising or bleeding easily

7. Sleepiness

8. Tremors

9. Malaise

10. Abdominal swelling (ascites)

Diagnostic Evaluation

1. **Blood tests:** Performed to determine how well patient's liver works. It may reveal thrombocytopenia.

2. **Ultrasound:** It may recommend to determine the exact cause of liver problem.

3. **CT scans or MRI:** It will identify certain causes of acute liver failure, such as Budd-chiari syndrome or tumors.

Management

Medical Management

1. **Medications to reverse poisoning:** Acute liver failure cause by acetaminophen overdose or mushroom poisoning is treated with drugs that can reverse the effects of the toxin.

2. Vitamin supplements may also be prescribed to patient.

3**. Relieving excess fluid in the brain:** Cerebral edema caused by acute liver failure can increase pressure on brain. Medication can help to reduce the fluid buildup in the brain.

Nursing Management

1. Nursing management of patient with acute liver failure involves the intensive care monitoring of patient in need of life support.

2. Assess respiratory or hydration status of patient.

3. Monitor signs and symptoms of bleeding.

4. Observe patient's vital signs.

5. If patient is hospitalized weight and fluid intake and output are measured and recorded daily

6. Measure and record abdominal girth daily.

7 Restrict sodium and fluids; replace electrolytes as directed.

8. Keep the head of bed elevated 30 degrees with the patient's head in the neutral position.

9. Provide small, frequent meals or dietary supple- ments to conserve patient's energy

10. Provide mouth care, if patient has bleeding gums.

11. Observe for signs and symptoms of infection and possible sepsis, administer antibiotics as ordered.

12. Instruct patient and family members to contact health care provider, if any of these occur

a. New or worse swelling in legs, ankles or feet.

b. Any questions or concerns about condition or care

Complications

1. Excessive fluid in the brain (cerebral edema)

2. Bleeding disorders

3. Kidney failure

4. Encephalopathy

5. Respiratory failure

6. Pancreatitis

7. Metabolic:

a. Hypoglycemia

b. Hypokalemia

c. Hypocalcemia

d. Hypomagnesemia

e. Acid-base disturbance

CHRONIC LIVER FAILURE

Chronic liver failure in the clinical context is a disease process of the liver that involves a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis.

Etiology

Viral causes

- Hepatitis B
- Hepatitis C
- Cytomegalovirus (CMV)
- Epstein Barr Virus (EBV)

Toxic and drugs

- Alcoholic liver disease
- Amiodarone
- Methotrexate
- Nitrofurantoin

Metabolic

- Non-alcoholic fatty liver disease
- Hemochromatosis
- Wilson's disease (a rare inherited disorder that causes copper to accumulate in the liver, brain and other vital organs)

Autoimmune

- Autoimmune chronic hepatitis
- Primary biliary cirrhosis
- Primary sclerosing cholangitis

Others

Right heart failure

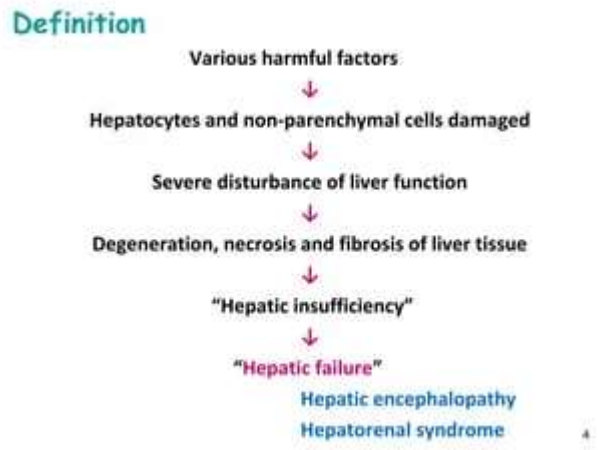
Risk Factors

1. Alcohol

2. Autoimmune hepatitis

3. Bile duct disorders

Pathophysiology



Clinical manifestation

Portal hypertension

- Esophageal varices
- Ascites
- Capute medusae
- Rectal hemorrhoids
- Enlarged spleen

Gastrointestinal

- RUQ pain
- Enlarged firm liver
- Epigastric discomfort
- Nausea
- Vomiting
- Diarrhea
- Constipation
- Clay-colored stools
- Weight loss
- Anorexia

Hematologic

- Abnormal bleeding
- Anemia
- Bruising
- Petechiae
- Bleeding of gums

Hepatic encephalopathy

- Personality/ behavior changes
- Asterixis
- Fatigue
- Drowsiness
- Confusion
- Muscle twitching

- Fetor hepaticus
- Comatose

Skin

- Jaundice
- Icterus
- Dryness
- Itching

Hepatorenal syndrome

- Decreased urine output
- Sodium retention

Reproductive

- Erectile dysfunction
- Testicular atrophy
- Amenorrhea

Most Commonly Occurs

1. Yellow skin and eyeballs (jaundice).

2. Pain in the upper right area of abdomen.

3. A general sense of not feeling well.

4. Difficulty in concentrating.

5. Disorientation or confusion.

6. Sleepiness

Signs Associated with Decompensation

1. Drowsiness

2. Hyperventilation

3. Metabolic flap or asterixis

4. Jaundice

5. Ascites

6. Leukonychia

7. Bruising

8. Peripheral edema

Signs Associated with the Etiology

1 Peripheral neuropathy

2. Kayser-Fleisher rings

3. Signs of right heart failure

4. Tattoos (hepatitis C)

Other Manifestations

1. Clubbing

2. Scratch marks

3. Testicular atrophy

4. Small irregular shrunken liver

5. Anemia

6. Gynecomastia

7 Spider nevi (angiomata)

Diagnostic Evaluation

1. **Abdominal radiographs:** It helps to identify ascites and enlargement of liver.

2. **An upper gastrointestinal (UGD) series**: It may reveal esophageal varices and evidence of gastric inflammation or ulcers.

3. **Esophagogastroduodenoscopy (EGD):** Helps to rule out any bleeding and to directly observe the esophagus, stomach and duodenum.

4. **Liver biopsy:** A physician may perform this to determine the extent and nature of the liver damage.

5. Liver serum enzymes, serum bilirubin, urobilinogen, serum ammonia and prothrombin times are elevated.

Management

Medical Management

1. Multivitamins for nutritional deficiencies are pre- scribed to patient.

2. Medical goals for managing bleeding from esoph- ageal varices are as follows:

a. Stop the bleeding.

b. Treat the fluid volume deficit caused by bleeding.

c. Prevent further fluid loss.

d. Maintain fluid and electrolyte balance

Surgical Management

Sclerotherapy:

1. Sclerotherapy is performed without general anesthesia.

2. It is usually done as part of an EGD while the patient is sedated with diazepam or midazolam hydrochloride.

3. Topical anesthesia is used.

4. Varices are injected with sclerosing agent that causes thickening and closing of the dilated vessels.

5. After this procedure, patient may complains of chest pain for upto 72 hours.

Liver transplant:

Liver transplant is required when the liver fails and there is no other alternative.

Nursing Management

1. Monitor respiratory rate, depth, use of accessory muscles, nasal flaring and breath sounds.

2. Evaluate ABG values, hemoglobin level and hematocrit.

3. Monitor the vital signs of patient.

4. Patient with chronic liver failure requires rest and other supportive measures to re-establish its functional ability.

5. Adjust patient's position on bed for maximal respiratory efficiency.

6. Measure and record abdominal girth daily

7. Assess for signs and symptoms of hemorrhage or bleeding.

8. Encourage patient to eat in a sitting position which helps to decrease abdominal tenderness and feeling of fullness.

9. Provide small, frequent meals or dietary supple- ments to conserve patient's energy

10. Provide mouth care, if patient has bleeding gums.

11. Administer medications as prescribed.

12. Maintain close follow-up for lab testing and evaluating by doctor.

Complications

1. Portal hypertension.

2. Hepatopulmonary syndrome (most common cause of respiratory insufficiency in patients with chronic liver disease).

3. Encephalopathy (damage or disease that affects the brain).

4. Hepatocellular carcinoma also known as hepatoma.

Medications used in liver failure

Antiemetics

• Odansetron

• Promethazine

Antihistamines

• Diphenhydramine

Histamine

• Ranitidine (zantac)

Vitamin K

Diuretics

• Spironolactone

Thiamine