بسم الله الرحمن الرحيم
SURGERY OF PORTAL HYPERTENSION
CIRRHOSIS OF THE LIVER

- Hepatic cirrhosis is a *necrosis* of the liver followed by *fibrosis* and *regeneration* of liver cells, involving the whole organ.

- **Aetiology**

  Cirrhosis can be the late result of any type of liver cell damage, and is the result of a combination of fibrosis and irregular degeneration leading to nodules. Viral hepatitis, alcohol and Bilhar. are the most common causes.

  **1- Primary biliary cirrhosis.**

  Common in female the onset is insidious and the diagnosis is suggested by the presence of *antimitochondrial antibodies* and confirmed by *liver biopsy*. **TT by Transplantation is the only cure.**
2- **Secondary biliary cirrhosis.**

- This is caused by prolonged *partial blockage of the bile duct* accompanied by cholangitis. *Total* blockage either leads to liver failure or is relieved *surgically* before cirrhosis develops. (choledecholithotomoy)

3- **Autoimmune chronic active hepatitis.**

- It is due to *autoimmune reaction* and can be diagnosed by detecting *antinuclear antibodies* (ANAs) and *smooth muscle antibodies* (SMAs) in the blood. Common in female TT by *steroids and azathioprine*.

4- **Primary sclerosing cholangitis.**

- This condition related to *inflammatory bowel disease*, is characterised by inflammation of the wall of the *bile ducts* and lead to chronic liver damage and *cirrhosis*. 
It can be complicated or confused with malignant change in the bile ducts. Transplantation is treatment of choice.

5- **Metabolic liver disease.**

1- **Haemochromatosis.**

- **In this disease, abnormal amount of iron are absorbed from the gut and deposited in various organs, particularly the liver and pancreas.** The condition is almost always confined to men, probably because regular menstruation prevents iron accumulation in women. Progression can be prevented by iron chelation and regular blood letting. Malignant change can be occurs multifocal.

2- **Hepatolenticular degeneration (Wilson‘s disease)**

- **Is an uncommon condition that is confined to children and young adults due to a hereditary error in copper metabolism.**
Muscular rigidity is common. Kayser-Fleischer rings in the eye due to peripheral corneal opacity are present. Liver biopsy show cirrhosis. Serum ceruloplasmin and serum copper are decreased, and urinary copper increased. TT by Penicillamine, which may have to be given for several years.

3- α1- Antitrypsin deficiency. This is an inherited disorder where there is a faulty production of the enzyme α1-antitrypsin. TT hepatic transplantation.

4- Cystic fibrosis. This inherited disease primary affects the lung and pancreas in children but can also cause strictures of the bile duct, gallstones and cirrhosis. TT by ursodeoxycholic acid.

5- Galactosaemia. Error in metabolism of lactose they will develop cirrhosis.
Morphological types.

1- **Micronodular.** Showing small regenerating nodules less than 3 mm in diameter. (alcoholism)

2- **Macronodular.** Nodules are greater than 3 mm diameter. (postnecrotic).

3- **Mixed.** Liver show both micro-and macronodular feater.

**Clinical features:**

- Often the presentation is by a complication, such as bleeding from oesophageal varices or the development of ascites. Other clinical signs are palmare erythema, spider naevi on the face, upper trunk and occasionally on the hands.
Gynecomastia and testicular atrophy are the result of high oestrogen levels and women may develop amenorrhoea.

- **The liver** may be markedly enlarged in the early stages but become small and shrunken in the late stages when the spleen will usually be large from the portal hypertension.

**Investigations:**

- Liver function test – C.B.C
- Abdominal ultrasound
- Liver biopsy

**Complications.**

- 1- Ascites (fluid retention)  2- Hepatic failure.
- 3- Malignant change.    4- Portal hypertension.
# Child’s Classification

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
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<tr>
<td>Bilirubin</td>
<td>&lt; 2</td>
<td>2 – 3</td>
<td>&gt; 3</td>
</tr>
<tr>
<td>Albumin</td>
<td>&gt; 3.5</td>
<td>2.8 – 3.5</td>
<td>&lt; 2.8</td>
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<tr>
<td>Ascites</td>
<td>None</td>
<td>Controlled</td>
<td>Uncontrolled</td>
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<tr>
<td>Enceph</td>
<td>None</td>
<td>Minimal</td>
<td>Advanced</td>
</tr>
<tr>
<td>Nutrition</td>
<td>Excellent</td>
<td>Good</td>
<td>Poor</td>
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## Child-Pugh Classification

<table>
<thead>
<tr>
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<th>Points</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td><strong>Bilirubin (mg/dL)</strong></td>
<td>&lt; 2</td>
</tr>
<tr>
<td><strong>Albumin (g/dL)</strong></td>
<td>&gt; 3.5</td>
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<tr>
<td><strong>Prothrombin time (seconds ↑)</strong></td>
<td>1 – 3</td>
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<tr>
<td><strong>Ascites</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>Encephalopathy</strong></td>
<td>None</td>
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Grade A, 5-6 points; Grade B, 7-9 points; Grade C, 10-15 points
PORTAL HYPERTENSION

- **Aetiology**: The portal venous pressure, normally 100-150 mm. of water or 7-11 mm.hg, may rise to abnormally high levels as a result of obstruction to the portal flow at any of 3 levels.

  1- **Prehepatic**
  2- **Intrahepatic**, 3- **Posthepatic**.

- **1-Prehepatic.** About 20% of patients belong to this group. The patient with prehepatic portal obstruction is nearly young, and often a child. On examination the liver is impalpable, but the spleen is enlarged. The *anaemia* is usually due to oozing from *oesophageal varices*, but sometimes *hypersplenism* is a factor. The obstruction arises in one of the following ways:
1- There is congenital absence or abnormality of the portal vein.

2- *Thrombosis of the portal vein* due to extension of the normal obliterative process of the *umbilical vein* and *ductus venosus* sometimes associated with *omphalitis* of newborn.

3- *Obstruction of the portal vein* in adults may be due to chronic *pancreatitis* and *carcinoma of the pancreas*, or thrombosis of the vein may follow acute pancreatitis, although these may just thrombose the splenic vein leaving the portal vein patent.
2- *Intrahepatic* account for nearly 80% of all cases.

- The causes are cirrhosis and *schistosomiasis* in which the blood flow through the liver is obstructed.
- Enlarged portosystemic venous communications are present long before serious oesophageal haemorrhage occurs.
- Enlargement of veins of the abdominal wall radiating from the umbilicus may be present.

3- *Posthepatic* is rare. It may be caused by a constrictive pericarditis and tricuspid valvular incompetence, and it is also a component of the Budd-Chiari syndrome.
Portal Vein Anatomy

- Liver
- Pyloric vein
- Superior pancreaticoduodenal vein
- Portal vein
- Left gastric vein (coronary)
- Pancreas
- Splenic vein
- Inferior mesenteric vein
### Site of anastomosis

<table>
<thead>
<tr>
<th>Portal vessels</th>
<th>Systemic vessels</th>
<th>Signs and symptoms</th>
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<tbody>
<tr>
<td><strong>Plexus around lower end of oesophagus</strong></td>
<td>Oesophageal branches of left gastric vein and short gastric veins</td>
<td>Lower systemic oesophageal veins</td>
</tr>
<tr>
<td><strong>Around umbilicus</strong></td>
<td>Paraumbilical veins (accompany the round ligament of the liver)</td>
<td>Superficial veins of the anterior abdominal wall</td>
</tr>
<tr>
<td><strong>Plexuses around lower third of rectum and anal canal</strong></td>
<td>Superior haemorrhoidal vein</td>
<td>Middle and inferior haemorrhoidal veins</td>
</tr>
<tr>
<td><strong>Extraperitoneal surfaces of abdominal organs</strong></td>
<td>Tributaries of superior and inferior mesenteric veins</td>
<td>Subdiaphragmatic and retroperitoneal veins</td>
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</table>
Portal Vein Collaterals
When the obstruction is prehepatic:

- Collateral between the portal vein above and below the obstruction enlarge. Thus insignificant venae comitantes of the hepatic artery and of the portal vein widely dialated.
- Depending on the site of the obstruction, some of the collaterals between the portal and systemic venous systems, notably the oesophageal plexus, also become dialated.

When the obstruction is intrahepatic:

- Anastomosis channels outside the liver between the portal and systemic systems become engorged, dilated and so an increasing proportion of the obstructed portal venous blood bypasses the liver. The only dilated veins that are dangerous to life are those in the submucosa of the oesophagus and
upper end of the stomach.

- In patients with portal hypertension, haemorrhoids, if present, are usually of the idiopathic type but rectal varices are common and can lead to severe haemorrhage.

**Oesophageal varices:**

- **Oesophageal varices are dilatation of the normal submucosal oesophageal veins and are the most important collaterals of the portal circulation.**
- **They may extend from below the gastro-oesophageal junction for 10-15 cm up the oesophagus.**
- **Perforating veins through the oesophageal wall connect them to the perioesophageal plexus.**
• Bleeding, which is nearly always from the lower 5 cm of the oesophagus, may be a slow ooze or sudden and severe.

• **Fibreoptic oesophagoscopy:** used to demonstrate presence of oesophageal varices.

• **Radiology Barium swallow:**

• **Angiography:** The venous phase of a coeliac axis angiogram is now preferred to direct puncture of the spleen
Fibreoptic oesophagoscopy
Gastric varices

1- they may be simple extensions of oesophageal varices across the gastro-oesophageal junction and down the lesser curve.

2- They may be extension through the fundus and upper body of the stomach in the presence of oesophageal varices.

3- They may occur in the absence of oesophageal varices, because they are collateral between the spleen and portal vein when there is splenic vein thrombosis.
Management of bleeding oesophageal varices.

I- General TT (first aid measure)

1- Put the patient in flat position with raising of the bed feet.

2- Ensuring a clear air way and rapid replacement of blood volume by fresh blood are the priorities (avoid fluids containing sodium)

3- Prevention of hepatic coma (ammonicial encephalopathy)

* Nasogastric suction of blood and instillation of solution of Mg sulphate (purgative and neomycin solution)
*Repeated enema with Mg sulphat solution.

*Giving amonia binding agent as ion exchange – prevent absorption of amonia from the intestine, glutamic a and arginine.

5- Supportive measures of the liver  Ca, Vit C, hypertonic glucose.

6- Correction of hypokalaemia and alkalosis by
• Infusion of KCL or Kadalex to correct hypokalaemia.
• Infusion of arginine sorbitol solution to correct alkalosis.

7- Continuous observation of temp, pulse, Bp, central venous pressure, haematocrite value, blood gases, blood electrolytes.
II-Specific TT

1- Arrest of haemorrhage.
   • Tamponade. By Sengstaken tube or linton tube

2- Drugs:
   A-I.V. injection of vasopressin.
   One unit in 200 ml glucose i.v. over 20 minutes to be repeated after 2 hours

Action of vasopressin
   • Systemic v.c. → Correction of low B.P.
Oesophageal aspiration channel

Oesophageal balloon
20-30mmHg greater than predetermined pressure

Gastric balloon
at least 300ml of air

Gastric aspiration channel

Fig. 45.23 Balloon tamponade. The four-channel approach is used to arrest gastrointestinal haemorrhage for control.
Splanchnic V.C. → Portal pressure due to ↓ portal blood volume → ↓ bleeding from oesophageal varices.

Selective V.C. of left gastric vein → ↓ bleeding.

↑ intestinal peristalsis to help evacuation of blood of the intestine rapidly → ↓ amonia concentration in the blood.

**Contraindications**

- Old patient with a history of anginal attack.
- Recently long acting vasopressin called Glypressin is used as a bolus injection initially 2 ampules, then 1 ampule every 1 hour up to 5 ampules.
**B-Octreotide (Sandostatin)**

- Is effective in lowering variceal blood pressure I. V. 50 µg bolus followed by an infusion of 50 µg/hour.

**C- Metoclopramide** 20 mg i.v. arrest oesophageal variceal bleeding temporarily, by constricting the gastro-oesophageal sphincter.

**D- Beta-blocking drugs,**

**E- Selective arterial mesenteric injection of vasopressin** to avoid systemic effect of vasopressin.
- **Endoscopic sclerotherapy**: 
- By using a fibreoptic flexible oesophagoscope

- **Intravariceal** method 5-6 ml of ethanolamine oleate into each varix at or just above the gastro-oesophageal junction to produce thrombosis.

- **Paravariceal**, very small quantities (0.5 ml) of sclerosant are injected alongside each varix to produce perivascular fibrosis.
Operations:

I- Devascularisation and transection
II- Portosystemic shunting

I- Devascularisation and transection

1- Borema-Crilea: Transthoracic porto-azygos disconnection.
2- Tanner's porto-azygos disconnection: Trans abdominal
3- Siguira (Japan) and Hassab (Egypt) If Vagal trunks are damaged pyloroplasty must be added.
4- Stapling gun makes the transection and reanastomosis relatively easy.
II-PORTOSYSTEMIC SHUNT OPERATIONS

1- Portocaval anastomosis.

2- Splenorenal anastomosis (proximal or central splenorenal shunt) splenectomy done.

3- Superior mesentericocaval anastomosis

4- Distal splenorenal shunt (Warren- Salam)

5- Inokuchi shunt: Joining Lt gastric vein to IVC by short saphenous graft.

6- Transjugular intrahepatic portosystemic shunt (TIPS)
BUDD-CHIARI SYNDROME

- This results from obstruction to the hepatic veins.

Clinical features

1-In acute cases nausea, vomiting and severe pain, due to rapid enlargement of the liver as a result of congestion, are often followed by death from hypotensive shock.

2-A less sudden onset Ascites and signs of portal hypertension, oedema of lower limb.

3-Chronic cases resemble liver cirrhosis

TT: *In obstruction of IVC with a membranous web in suprahepatic Portion. a transatrial meatotomy is the best treatment

*In whom PV and IVC are patent portocaval or mesocaval shunt

*In patient with a blocked inferior vena cava A mesoatrial shunt can be done
THANK YOU