Portal Hypertension

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Portal vein anatomy

- The portal vein is formed in front of IVC and behind the neck of the pancreas (at the level of 2nd lumbar vertebra) by union of the splenic & SMV.

- It is 7-8 cm in length & contains no valves.

- It courses in the lesser omentum posterior to both the common hepatic artery & common bile duct.
Portal vein anatomy

- It bifurcates into the Lt & Rt trunks in, or just below the hilum of the liver.

*Its main tributaries are:*

- The coronary (Lt gastric) vein.
- Pyloric vein.
- Cystic vein.
- Pancreaticodudenal vein.
- Ligamentum teres (umbilical vein).
- Ligamentum venosum.
Portal vein anatomy
Portal vein anatomy

- The portal venous branches ramify in an arterial like pattern and end in dilated channels called sinusoids, which are equivalent to systemic capillaries.
- From here blood drains into the hepatic venous system.

- The normal portal pressure is 5-7 mmHg (8-12 cm of water).
- Portal hypertension is present when the portal vein pressure exceeds 12 mmHg.
Causes of portal hypertension

*Increased resistance to flow*

A) Pre-hepatic (portal vein obstruction):

1. congenital atresia or stenosis.
2. thrombosis of portal vein.
3. thrombosis of splenic vein.
4. Extrinsic compression (e.g., tumor).
Causes of portal hypertension

B) Intra-hepatic:

1- liver cirrhosis → obstruction is sinusoidal & post-sinusoidal.

2- bilharzial periportal fibrosis → obstruction is pre-sinusoidal.
Causes of portal hypertension

*The causes of portal hypertension in cirrhotic patients are:*

- Diminution of the total vascular bed by obliteration, distortion, & compression of sinusoids.
- Compression of the tiny radicals of portal & hepatic veins by excessive fibrosis.
- Development of multiple arteriovenous shunts between the branches of the hepatic artery & portal vein.
Causes of portal hypertension

C) Post-hepatic:

1- Budd-Chiari syndrome (hepatic vein thrombosis) \(\rightarrow\) prominent ascites, hepatomegaly & abdominal pain.

2- Veno-occlusive disease.

3- Cardiac disease:
   a- constrictive pericarditis.
   b- valvular heart disease.
   c- right heart failure.
Causes of portal hypertension

- **Increased portal blood flow**
  
  A) Arterial-portal venous fistula.

  B) Increased splenic flow:

  1- Banti’s syndrome

  (liver disease secondary to splenic disease, result from cirrhosis & other hepatic disorders)

  2- Splenomegaly (e.g. tropical splenomegaly, hematologic diseases).
Sequelae & Clinical picture

1- Porto-systemic collaterals:

- In normal conditions → collapsed.
- In portal hypertension → engorged → divert blood away from the portal circulation.
Sequelae & Clinical picture

The important sites of these collaterals are:

a) **At the lower end of oesophagus**
   - Oesophageal tributaries of Lt gastric vein (portal)
   - Oesophageal tributaries of hemiazygous vein (systemic).

b) **Around the umbilicus**
   - Para umbilical vein (portal)
   - Superior & inferior epigastric veins (systemic)
Sequelae & Clinical picture

c) Lower rectum & anal canal
Superior rectal vein (portal)
Middle & Inferior rectal veins (systemic)

d) At the back of the colon
Rt & Lt colic veins (portal)
Rt & Lt renal veins (systemic)

e) Retroperitoneum
Tributaries of superior & inferior mesentric veins { Retzius } (portal)
Posterior abdominal & subdiaphragmatic veins (systemic)
Portal vein collaterals

- Azygos vein
- Esophageal and gastric varices
- Short gastric veins
- Gastroepiploic vein
- Splenic vein
- Inferior mesenteric vein
- Superior mesenteric vein
- Superior hemorrhoidal vein
- Retroperitoneal collaterals
- Hemorrhoidal veins
- Hemorrhoids
- Umbilical vein
- Caput medusae
2- Splenomegaly

- The most constant physical finding.
- In 80% of patients regardless the cause.

* In Bilharzial cases:
  - at 1\textsuperscript{st} due to reticuloendothelial hyperplasia
  - due to absorption of bilharsial toxins.
  - With progress of portal hypertension \rightarrow due to congestion.
Sequelae & Clinical picture

3- Congestion of the whole GIT
- Leads to anorexia, dyspepsia, indigestion, and malabsorption.

4- Bleeding varices.

5- Ascites (multifactorial)
- Portal hypertension alone cannot cause ascites.
- Hypoalbuminaemia → below 3 gm/100 ml.
- Salt & water retention → high level of aldosterone, oestrogens & anti-duretic hormone.
- Increased lymphatic transudation from liver surface.
Investigations

1. Assessment of liver function tests

(a) Hypoalbuminaemia.

*The liver is the only site of albumin synthesis.*

(b) ALT & AST are moderately raised.

(c) Prothrombin time and concentration are disturbed.

*This test is the most sensitive liver function.*
Investigations

2. Detection of oesophageal varices by:
   
   (a) Fibreoptic upper endoscopy
   
   (b) Barium swallow can visualize varices in 90% of cases. 

   They appear as multiple, smooth, rounded filling defects (honey-comb appearance)

   (c) Duplex scan can show dilated portal vein and collaterals.
Variceal Bleeding

Caput Medusae

Haemorrhoids
Detection of oesophageal varices
Ascites
Shrunken Liver
Portal vein thrombosis
Splenomegaly
Investigations

3. Detection of splenic sequestration and hypersplenism.

- (a) Blood picture → anaemia, leucopenia, thrombocytopenia or pancytopenia.
- (b) Bone marrow examination → hypercellularity.

- (c) Radioactive isotope studies:
  using the patients own RBCs tagged with $^{51}$Cr → diminished half life of RBCs & increased radioactivity over the spleen.
4. Diagnosis of the aetiology of liver disease is performed by:

- (a) Immunological tests for hepatitis markers.
- (b) Liver biopsy after assessment of prothrombin time and concentration.
## Child Pugh classification

<table>
<thead>
<tr>
<th>Points</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
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<tbody>
<tr>
<td><strong>Bilirubin (mg/dL)</strong></td>
<td>&lt; 2</td>
<td>2 – 3</td>
<td>&gt; 3</td>
</tr>
<tr>
<td><strong>Albumin (g/dL)</strong></td>
<td>&gt; 3.5</td>
<td>2.8 – 3.5</td>
<td>&lt; 2.8</td>
</tr>
<tr>
<td><strong>Prothrombin time (seconds)</strong></td>
<td>1 – 3</td>
<td>4 – 6</td>
<td>&gt; 6</td>
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<tr>
<td><strong>Ascites</strong></td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
</tr>
<tr>
<td><strong>Encephalopathy</strong></td>
<td>None</td>
<td>Minimal</td>
<td>Advanced</td>
</tr>
</tbody>
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- Grade A, 5-6 points; Grade B, 7-9 points; Grade C, 10-15 points
Treatment

Management of patients with actively bleeding oesophageal varices

1. Admission.
   The patient should be admitted to hospital.

2. Resuscitation.
   - A wide bore cannula is inserted.
   - A blood sample is taken.
   - Restoration of blood volume should be rapid.
   - Overexpansion of the circulation should be avoided.
   - Morphine and Pethidine are contraindicated.
Treatment

3. Correct coagulopathy.
- Vitamin K is administered intravenously.

4. Prevent encephalopathy.
- Blood in the intestine will be fermented to ammonia and other nitrogenous products.
- Repeated enemas.
- Oral lactulose.
  - This is a disaccharide sugar, fermented by the intestinal flora → lactic acid → combines with ammonia.
- Neomycin 0.5 gm every 4 hours can reduce the bacterial flora.
Treatment

**Sclerotherapy**

- Intra- or Para- Variceal.
- 1-3 ml sclerosant (ethanolamine oleate).
- Occludes venous channels.
- Multiple sessions (2 weekly).
- Control bleeding in 80-95%.
- About 50% rebleed.
- 30% complication rate.
Endoscopic Sclerotherapy

Intra-variceal

Para-variceal
Complications of Sclerotherapy

**LOCAL**
- Ulceration.
- Stricture.
- Perforation.
- Retrosternal discomfort for few days.

**SYSTEMIC**
- Fever
- Pneumonitis
- CNS
Treatment

Endoscopic Banding

- Occludes venous channels
- Sessions < sclerotherapy
- Same results as sclerotherapy
- ↓ complications vs sclerotherapy
- Endoscopic treatment of choice
Endoscopic Banding
Treatment

Drugs

**Vasopressin** → vasoconstriction of the splanchnic circulation.

- **Dose** → 0.2 unit/kg wt, dissolved in 200 ml of 5% dextrose, over 20 minutes.

**Disadvantages**

- colicky abdominal pains, & diarrhoea.
- anginal pains, so it is contraindicated in the elderly.

- Produce temporary control of bleeding in about 80% of cases.

- To prolong its action it is combined with glycine (Glypressin).
Treatment

Somatostatin

- lower the intravariceal pressure without significant side effects.
- Initial bolus 100 microgram $\rightarrow$ continuous infusion of 25 microgram / h for 24 hs.

Beta blockare

- $\downarrow$ bleeding by $\downarrow$ cardiac output.
- Does 20-60 mg bid $\rightarrow$ 25% $\downarrow$ in HR.
- Reduces 40% of bleeding episodes
- Does *not* reduce mortality
Treatment

Balloon tamponade by Sengestaken or Linton tube.

- The gastric balloon is inflated first by 200 ml of air, and pulled upwards to press the gastric fundus.
- If bleeding continues, the oesophageal balloon is inflated.
- The pressure in the oesophageal balloon should not exceed 40 mm Hg.
- This therapy is effective in controlling bleeding in 80-90% of cases.
Treatment

Disadvantages:

- Discomfort to the patient.
- The patient cannot swallow his saliva.
- Liability to cause oesophageal ulceration or stricture.
- Once the tube is deflated, there is liability to rebleeding in 60-80% of patients.

*Balloon tamponade is only used as a temporary measure before sclerotherapy or surgery.*
Balloon tamponade

Accessory tube to suction
To esophageal balloon
Gastric tube to suction
To gastric balloon
Treatment

Emergency surgery.

If all the previous measures fail to stop bleeding, surgery is recommended.

- If the general condition of the patient is satisfactory → splenectomy, portoazygos disconnection and stapling of the oesophagus.
- If the patient is not very fit → stapling alone can be performed.
Treatment

Trans-jugular Intra-hepatic Porto-systemic Shunt (TIPSS)
TIPS
## SclTx vs TIPS

Five Randomized Trials - 360 patients

Mean Follow-up 15 mos (1-36)

<table>
<thead>
<tr>
<th></th>
<th>REBLEED*</th>
<th>ENCEPH**</th>
<th>SURVIV***</th>
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<tbody>
<tr>
<td>SCLTX</td>
<td>37%</td>
<td>8%</td>
<td>88%</td>
</tr>
<tr>
<td>TIPS</td>
<td>17%</td>
<td>32%</td>
<td>81%</td>
</tr>
</tbody>
</table>

* p < 0.05 in all but one study
** p < 0.05 in all studies
*** n.s. in all but one study where survival ↑ w/ SclTx
Treatment

**Indications for TIPSS:**

- Refractory bleeding
- Prior to transplant
- Child C
- Refractory ascites

**Main early complication:**

- Perforation of liver capsule → massive haemorrhage.
Treatment

Treatment of patients with history of bleeding oesophageal varices:

1. Repeated sclerotherapy until the varices are obliterated is the first choice.

2. Elective surgery is mainly indicated if sclerotherapy failed to stop recurrent attacks of bleeding provided that they are fit.
Treatment

Operations for portal hypertension

*Shunt operations.*

- The idea of these operations is to lower the portal pressure by shunting the portal blood away from the liver
Total shunt operations

1- Porta-caval operation

End to side  Side to side
Porta-caval operation

very efficient in lowering the portal pressure → no bleeding occurs from the varices.

**disadvantages:**

deprives the liver of portal blood flow → accelerates the onset of liver failure.

Recurrent hepatic encephalopathy in 30-50% of patients.
Proximal spleno-renal shunt

- Indicated if the portal vein is thrombosed or if splenectomy is indicated due to hypersplenism.
- The incidence of encephalopathy is less than after porta caval shunt.
- It is less effective in preventing further bleeding.
- If the splenic vein is less than 1 cm, the anastomosis is liable to thrombosis.
Mesocaval (Drapanas) shunt

- Insertion of a synthetic graft as dacron, or autogenic vein between the superior mesenteric vein and inferior vena cava.
- The incidence of thrombosis is high.
Total Shunt Results

- Prevent rebleed > 90%
- Thrombosis with graft
- Encephalopathy rate 40%
Selective shunt (Warren shunt)

- The Rt and Lt gastric vessels are ligated.
- The proximal end of splenic vein is ligated while the distal end is anastomosed to the left renal vein.
- The short gastric veins are preserved and will selectively decompress the lower end of the oesophagus.
- The incidence of encephalopathy is low, and the liver functions remain normal.
Porta azygos disconnection operations

There are many techniques for performing devascularization.

Hassab Khairy operation

- splenectomy & ligation of the Rt and Lt gastric vessels, the short gastric vessels and the vascular arcade along the greater curvature of the stomach leaving only the right gastroepiploic vessels.
All vessels surrounding the lower 5-10 cm of the oesophagus are ligated.

There is no encephalopathy following this operation and the portal blood flow is intact.

There is a low incidence of rebleeding following the operation, but it can usually be controlled by sclerotherapy.
Liver Transplant

- Indicated for liver failure
  
  *Not* for variceal bleeding.

- 24% in USA die on waiting list
Control of Ascites

- Sodium / Water Restriction.
- Spironolactone.
- Loop Diuretic.
- Large Volume → Paracentesis.
- Peritoneal-Venous Shunt.
- TIPSS
Ascites
Encephalopathy

**Etiology:** ? Nitrogen compounds

**Induced by:**
- Infection
- Dehydration
- Constipation
- Blood in gut

**No test is diagnostic**

**Therapy:**
- Hydrate
- Cleanse gut
- ↓ protein
- Find and treat cause
THANK YOU