Portal Hypertension

Definition

portal pressure gradient of 12mm Hg or more compared with a normal figure of 5- 10mm Hg. It is secondary to many conditions but the commonest is cirrhosis.

Aetiology:

Prehepatic	Intrahepatic	Posthepatic
Thromboses: portal vein, splenic vein Congenital portal vein atresia or stenosis Extrinsic compression e.g. tumour Splanchnic arteriovenous fistula	Cirrhosis Acute alcoholic hepatitis Acute and fulminant hepatitis Primary biliary cirrhosis Idiopathic portal hypertension Myeloproliferative or metastatic disease infiltration Polycystic liver disease Granulomatous disease (Sarcoid, TB) Venoocclusive disease Schistosomiasis Idiopathic portal hypertension Congenital hepatic fibrosis Vitamin A toxicity Peliosis hepatis Nodular regenerative hyperplasia	IVC obstruction Budd-Chiari syndrome Right heart failure Constrictive pericarditis Tricuspid regurgitation Arterial-portal venous fistula ↑Portal or splenic blood flow

Liver disease causes a reduction in the lumen of the portal vein and thus a marked increase in resistance. Increased blood flow also results from a hyperdynamic state due to the release of endogenous vasodilators.

History:

- Jaundice, blood transfusion, especially abroad. Sexual or drug taking lifestyle.
- Family history of liver disease such as Wilson's disease or hereditary haemochromatosis
- Alcohol consumption

The following aspects seek evidence of complications of portal hypertension.

- Haematemesis or melaena are features of upper GI and suggest bleeding varices
- Mental changes e.g. lethargy, irritability & changes in sleep suggest encephalopathy
- Increased abdominal girth and rapid weight gain suggests ascites
- Abdominal pain and fever suggest spontaneous peritonitis

Examination:

May indicate liver disease, porto-systemic anastomosis and hyperdynamic circulation.

- Jaundice, spider naevi, palmar erythema, Dupuytren's contracture, asterixis
- Abdo exam: ascites, splenomegaly, dilated epigastric veins, caput medusa, haemorrhoids
- Testicular atrophy and gynaecomastia
- Muscle wasting
- Examination of the pulse shows a bounding, hyperdynamic circulation with warm periphery
- Arterial blood pressure is low

Investigations:

Bloods:

- LFTs, FBC, platelets, PTT, Albumin,
- Screen for viral hepatitis
- Anti-nuclear antibody, anti-mitochondrial antibody, anti-smooth muscle antibody
- Ferritin, Alpha-1-antitrypsin, Ceruloplasmin

Imaging

- Doppler USS - ascites, portal blood flow and thrombosis of portal or splenic veins. Liver & spleen anomalies.
- CT or MRI may show portal vasculature and be useful if USS inconclusive.
- Selective angiography of the superior mesenteric artery may be used.
- Endoscopy to exclude or treat any oesophageal varices.
- Liver biopsy may be performed if coagulation studies are satisfactory.

Management - Aims is to reduce bleeding by reducing portal pressure

Non-Drug

- Bed rest, restrict intake of salt to <90mmol/day, water restriction.

Drugs

- Reduce portal pressure with: beta blockers (e.g. **propranolol**), vasodilators (e.g. **isosorbide mononitrate**), splanchnic vasoconstrictor (e.g. **Terlipressin**, a vasopressin analogue), **octreotide** and **lanreotide** (somatostatic analogues)
- Reduce ascites with diuretics (**spironolactone** at high dose ± **frusemide**), paracentesis

Surgical

- Portocaval shunts are no longer used but instead procedures such as transjugular intrahepatic portosystemic shunts (TIPS). Shunts may be total, partial or selective. 90% can achieve long term patency but 30 to 40% of patients develop encephalopathy.
- Devascularisation procedures include splenectomy or gastro-oesophageal transection.
- Liver transplantation is the ultimate treatment.
- Ligation seems better than sclerotherapy to treat active bleeding but as a prophylactic measure neither is beneficial and may even have an adverse outcome.

Complications:

- Variceal haemorrhage (esp. oesophageal)
- Almost 90% of patients with cirrhosis develop varices, and ~30% of varices bleed.
- First episode of variceal haemorrhage is estimated to carry a mortality rate of 30-50%.

Prognosis

The Child-Pugh classification system indicates prognosis:

Criterion	Score 1 point	Score 2 points	Score 3 points
Serum albumin (g/L)	>3.5	3.0-3.5	<3.0
Serum bilirubin (g/dL)	<2.0	2.0-3.0	>3.0
PTT (s) or INR	PTT=1-4 or INR<1.7	PTT=4-6 or INR=1.7-2.3	PTT>6 or INR>2.3
Ascites	none	moderate	severe
Encephalopathy	none	mild	severe

Score 5-6 = Class A, Score 7-9 = Class B, Score ≥10 = Class C

Patients with Class A or B have a 5-years survival rate of 70% to 80%.
Class C patients have a 1-year survival around 50%
Ascites, albumin <3.2gm/l, recent episode SBP are each assoc with a one-year survival of ≤50%.