PORTAL HYPERTENSION
INTRODUCTION

• Elevation of portal pressure >10-12 mm Hg (Normal 5-7 mm Hg).
• Common cause of Upper GI bleeding.
• Major cause of morbidity & mortality from liver disease.
• Remarkable difference in aetiology between geographic areas.
• Prognosis depends on presence of complications.
ETIOLOGY

Results from obstruction to portal blood flow
Classified as-
• Pre-Hepatic
• Intrahepatic
• Post-Hepatic
• Idiopathic
ETIOLOGY (contd.)

PREHEPATIC (EXTRAHEPATIC)

• Portal vein thrombosis* / atresia / stenosis / cavernous malformation
• Splenic vein thrombosis / AV fistula

INTRAHEPATIC

(A) HEPATOCELLULAR CAUSES

• Acute and chr. hepatitis
• Cirrhosis of liver*
• Congenital hepatic fibrosis

*common cause
ETIOLOGY (contd.)

- Wilson’s disease
- $\alpha_1$-antitrypsin deficiency
- Glycogen storage disease type IV
- Sarcoidosis
- Schistosomiasis
- Veno-occlusive disease

(B) BILIARY TRACT DISEASE

- Biliary atresia (intra/extrahepatic)
- Sclerosing cholangitis
- Choledochal cyst
ETIOLOGY(CONTD.)

POSTHEPATIC
• Budd-Chiari syndrome
• CCF & constrictive pericarditis.

IDIOPATHIC PORTAL HTN (50%)
- Non-cirrhotic portal fibrosis
- 3rd order or 4th order small branches of portal vein are thrombosed
ETIOLOGY (contd.)

EHPVO – most common cause
• Occurs due to thrombosis secondary to-
  • Infection: umbilical sepsis with or without catheterisation, Intra-abdominal infections.
  • Hypercoagulable states
  • Trauma
  • Infiltration and compression by malignancy
  • Idiopathic
CLINICAL FEATURES

Presentation depends on type of Portal Hypertension.

**Extrahepatic**: common in India

- Mean age of presentation- 5-6 yrs
- Hematemesis & melena- very common
  - occurs spontaneously in a healthy child
- Splenomegaly - almost universal, occurs early
  - size may be 6-10 cm
  - f/o hypersplenism may be present

- Ascites- persistent or massive -uncommon
CLINICAL FEATURES

Intrahepatic:
- Stigmata of Chronic Liver disease (CLD) - may be present
  CLD → cirrhosis → most of the manifestations
- Hematemesis & Melena - profuse and recurrent.
- F/O Hepatic failure - may be present
  e.g. deepening jaundice, progressive ascites, hepatic encephalopathy, bleeding manifestations

Posthepatic:
- Prominent veins may be seen in Rt. flank and back.
- Liver markedly enlarged specifically the caudate lobe
  (Budd-Chiari syndrome)
CLINICAL FEATURES OF INTRAHEPATIC PORTAL HYPERTENSION

JAUNDICE

PALMAR ERYTHEMA

CLUBBING

RECTAL VARICES

SPIDER ANGIOMA

ASCITES WITH DILATED ANT. ABDOMINAL VEINS
COMPLICATIONS

- Variceal bleeding
- Haemorrhoids
- Hepatic Encephalopathy
- Hepato-Pulmonary syndrome
INVESTIGATIONS

- Complete hemogram, liver function test
- USG, Doppler flow USG
- Upper GI endoscopy
- Barium swallow, contrast enhanced CT scan
- MR angiography
- Splenoportovenography
- Percutaneous transhepatic portovenography
UGI ENDOSCOPY (conn ‘s staging)

• GRADE I = Straight vessels in inspiration only
• GRADE II = Straight vessels in both inspiration & expiration
• GRADE III = Straight projecting into lumen <50%, can be effaced.
• GARDE IV = Tortuous vessels projecting into lumen >50%, Cannot be effaced
The steps of Management (Mgt.) are:

- Emergency Mgt. of acute Upper GI bleeding
- Mgt. of variceal bleeding
- Prevention of variceal bleeding
MANAGEMENT OF A/C UPPER GI BLEEDING

• Vital signs monitoring avoid fluid overload
• Hemodynamic stabilisation – crystalloids, vasopressors, blood transfusion
• Correction of coagulopathy (Inj. Vit. K, FFP)
• Nasogastric Aspiration of blood
• H2 receptor blocker
• Pharmacotherapy
• Therapeutic Endoscopic sclerotherapy
• Balloon Tamponade
PHARMACOTHERAPY

- Vasopressin - causes splanchnic vasoconstriction & ↓ portal blood flow.
- Vasopressin + nitroglycerine. (↓ side-effects on heart, bowel & kidney)
- Terlipressin (less side-effects)
- Somatostatin analogue
  Octreotide (selective splanchnic vasoconstrictor)
  Dose 1-2 μg/kg bolus followed by continuous Infusion of 1-5 μg/kg/hr till 48 hrs no bleeding.
ENDOSCOPIC SCLEROTHERAPY

• Intra and paravariceal injection of sclerosant – causes thrombosis & obliteration of vessels.
• Common Complications - bleeding, oesophageal ulcerations & stricture, perforation, bacteraemia.
• **Endoscopic variceal ligation**
  Better option than sclerotherapy.
ENDOSCOPIC VARICEAL LIGATION

• Intra- and paravariceal injection of sclerosant - causes thrombosis & obliteration of vessels.

• Common Complications - bleeding, oesophageal ulcerations & stricture, perforation, bacteraemia.

• Endoscopic variceal ligation - better option than sclerotherapy.
**Sengstaken Blakemore Tube** A tube made of 3 smaller tubes with 2 balloon located distally for the oesophagus and the stomach, 3 proximal and 1 distal opening.

**COMPLICATIONS**

- Asphyxia by pressure of the oesophageal balloon on the trachea
- A ruptured balloon can set of an air embolism
MANAGEMENT OF VARICEAL BLEEDING

• After emergency mgt. by pharmacotherapy and endoscopic sclerotherapy (EST), further sittings of EST required.
• To start - wkly for 3 sittings then 3 wkly till varices obliterated.
• Endoscopic variceal ligation (EVL) + low dose EST may have better outcome.
• Surgical interventions
SURGICAL INTERVENTIONS

• DECOMPRESSIVE SHUNTS
  Aim is to divert portal blood flow to decrease portal pressure
  Eg: Mesocaval & Distal Splenorenal shunt
  REX Shunt (Mesenterico – left portal vein bypass)
  TIPS(Transjugular intrahepatic porto systemic shunt)

• LIVER TRANSPLANTATION
  Better option in intrahepatic disease
PREVENTION OF VARICEAL BLEED

• Long term T/t by nonselective β- blocker
  (↓ cardiac output, lowers portal perfusion
   Splanchnic vasoconstriction)

• Propranolol (0.5-2 mg/kg 3-4 div. doses titrated upwards till 25% ↓ in HR from baseline and ↓ BP by 15mm Hg)
THANK YOU