

**Carcinoma Stomach
Liver tumours
Portal hypertension
Chronic liver disease**

Carcinoma Stomach

Introduction

- Gastric cancer is endemic in Japan
- Late stage at diagnosis because of
 - Low incidence
 - Non specific symptoms
 - Risk factors not definable
- Biologically more aggressive
- Increasing incidence of adenocarcinomas of proximal stomach and distal esophagus

Etiology

- Low acid production
- H pylori infection



Body of stomach

- Low acid production
- Reflux



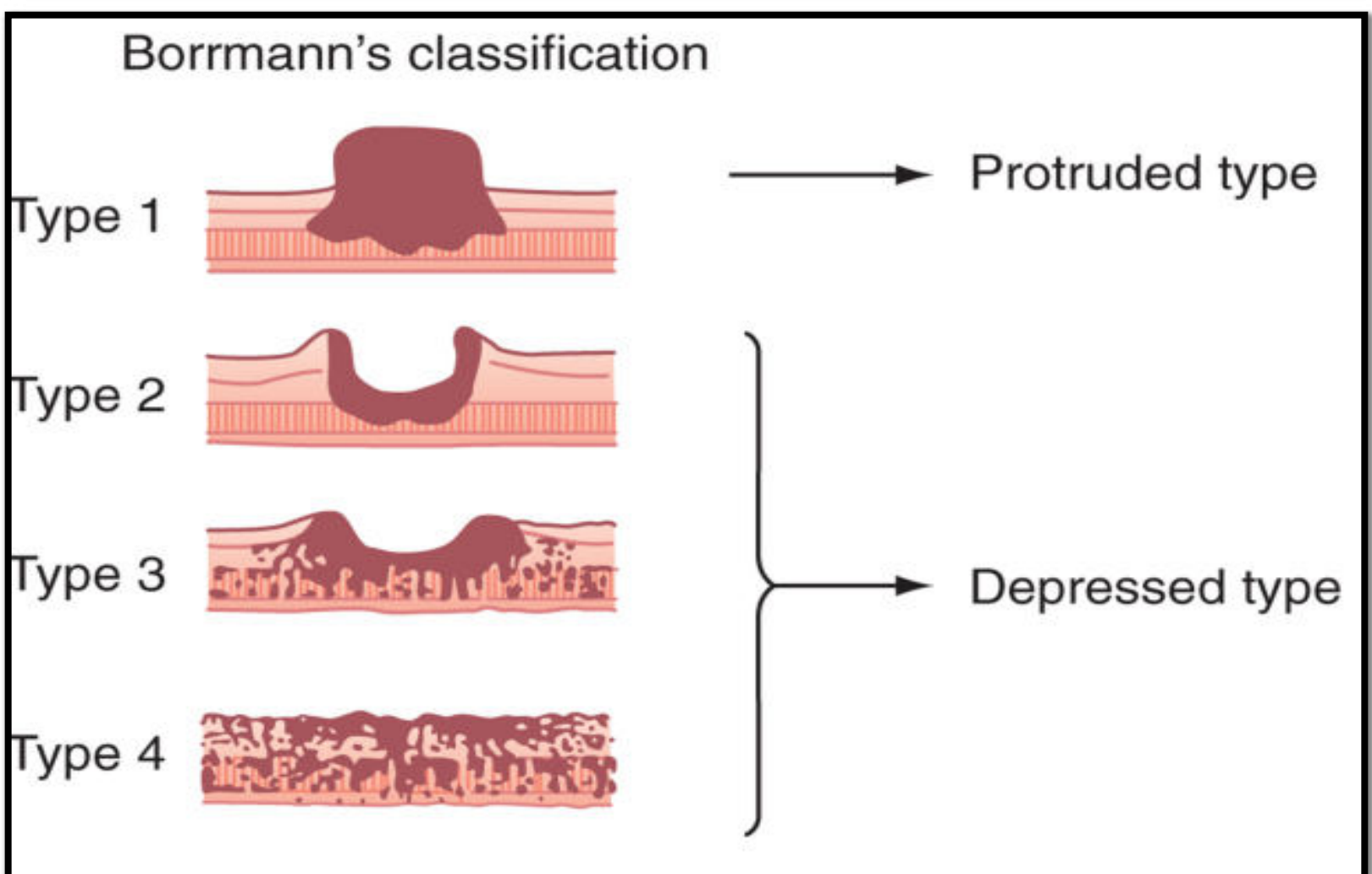
Esophagus & Cardia

- Obesity
- High BMI
- High glycaemic load diet
- GERD
- Smoking
- Alcohol
- Tobacco

Although no normal lymphoid tissue is found in the gastric mucosa, the stomach is the most common site for lymphomas of the gastrointestinal tract

Borrmann classification(depending on macroscopic appearance)

- Type I: polypoid or fungating cancers
- Type II: ulcerating lesions surrounded by elevated borders
- Type III: ulcerated lesions infiltrating the gastric wall
- Type IV: diffusely infiltrating tumors
- Type V: unclassifiable cancers



Patterns of Spread

- Local extension
- Lymphatic metastases
 - left supraclavicular fossa (Virchow's node)
 - left axilla (Irish's node)
 - S/c periumbilical tumour deposits (Sister Mary Joseph's nodes)
- Peritoneal metastases
- Distant metastases

Clinical Presentation

- Weight Loss
- Anorexia
- Early satiety (Diffusely infiltrative type)
- Recurrent Vomiting (pyloric involvement)
- Dysphagia
- Bleeding
- Anemia
- Fatigue
- Epigastric pain

Resection Classification

- R0: no gross or microscopic residual disease
- R1: microscopic residual disease (+ margins)
- R2: gross residual disease

Diagnostic work up

- Tumour Markers: CA 19.9, CA 72.4, CEA, CA 50
- Endoscopy & Biopsy:
 - Chromo endoscopy: identification of mucosal abnormalities through topical stains.
 - Magnification Endoscopy: magnify standard endoscopic fields by 1.5- to 150-fold.
 - Narrow band imaging: increased visualization of the microvasculature
 - Confocal laser Endomicroscopy : 3D microscopy including subsurface structures

- Overexpression or amplification of *HER2 (EGFR2)*:
 - occurs in approximately 20% of patients with gastric cancer
 - Recommended in metastatic, recurrent gastric cancer
 - Trastuzumab used in Her 2 neu + cancers

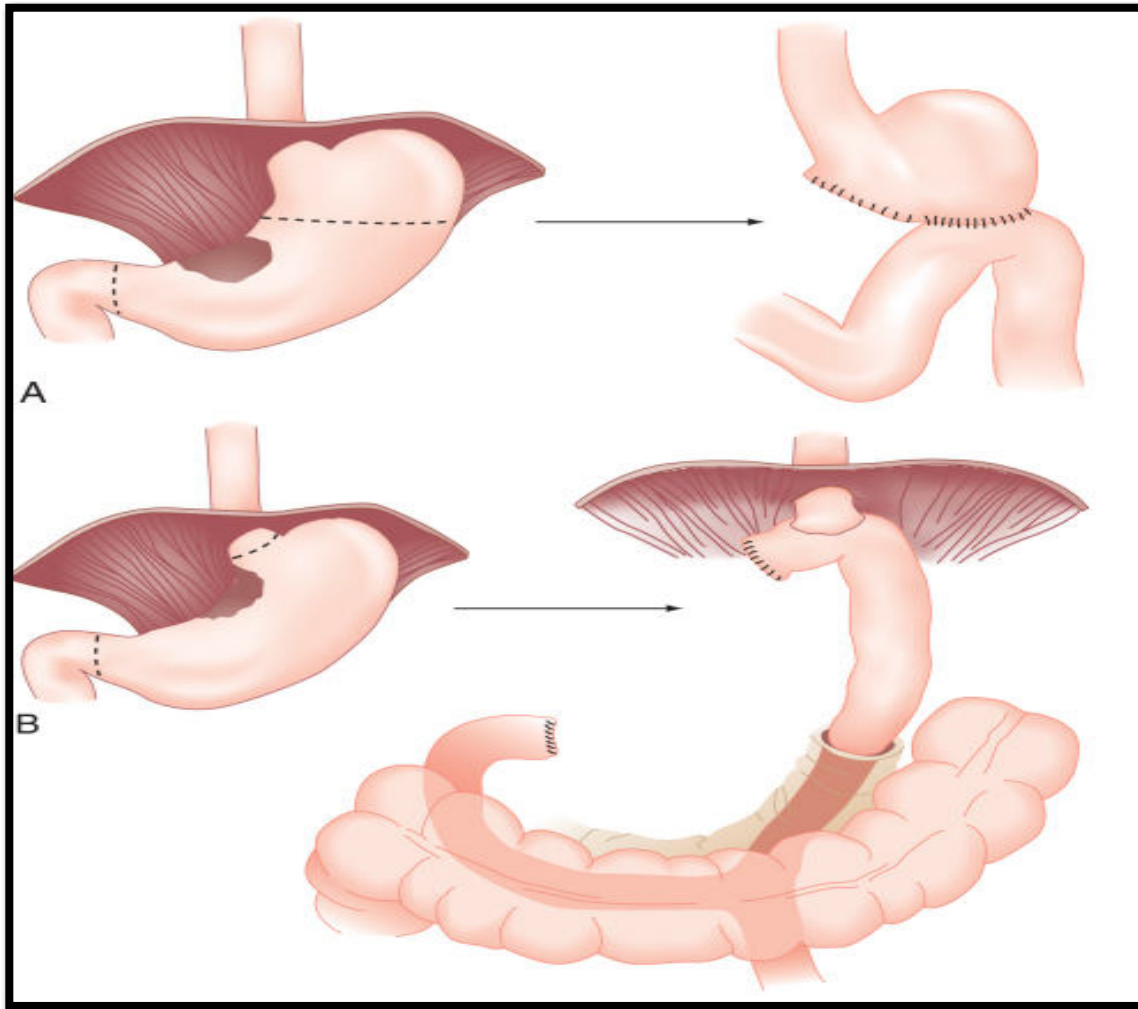
- Endoscopic ultrasound (EUS)
- Computed Tomography
- Magnetic Resonance Imaging:
 - to characterize liver lesions on CT scan
- Positron Emission Tomography:
 - 50% of primary tumors are FDG-negative
 - diffuse (signet cell) subtype most likely to be non-FDG avid

- Staging Laparoscopy and Peritoneal Cytology:
 - directly inspects the peritoneal and visceral surfaces
 - done to spare nontherapeutic operations

Treatment

- Surgery (Gastrectomy with lymph node dissection)
 - Radical Gastrectomy (Proximal gastric cancer)
 - Subtotal Gastrectomy (mid & distal gastric cancer)

When the oncologic goal of an R0 resection can be achieved by a gastric-preserving approach, partial gastrectomy is preferred over total gastrectomy



A: Subtotal gastrectomy with a Billroth II anastomosis

B: Total gastrectomy with a Roux-en-Y anastomosis

- Lymphadenectomy
 - 15 nodes for adequate pathologic staging
- Partial Pancreatectomy and Splenectomy—Resect or Preserve?
 - Splenectomy:
 - Intraoperative evidence of direct tumor extension into the spleen
 - Primary tumor is located in the proximal stomach along the greater curvature
 - Partial Pancreatectomy:
 - direct tumor extension into the pancreas

Stage specific Survival rates for gastric adenocarcinoma are higher in Japan than in Western countries because:

- Better-prognosis intestinal-type tumors are more common
- Poorer-prognosis proximal gastric cancers are less frequent
- Widespread use of extensive D2 or D3 lymphadenectomy

Chemotherapy

- Adjuvant
- Neoadjuvant:
 - Higher rate of R0 resections
 - Early treatment of micro metastatic disease
- Perioperative

Adjuvant Intraperitoneal Chemotherapy

- Hyperthermic intraoperative peritoneal chemotherapy (HIPEC)
- Normothermic intraoperative chemotherapy (NIIC)
- Early postoperative intraperitoneal chemotherapy (EPIC)
- Delayed postoperative intraperitoneal chemotherapy (DPIC)

Chemotherapy Drugs

- Cisplatin
- 5-Fluorouracil
- Taxanes
- Epirubicin
- Irinotecan
- Trastuzumab (targeted therapy in Her 2 neu +)

Radiotherapy

- Preoperative
- Intraoperative
- Postoperative

Liver Tumours

Benign tumours

- Liver Cell Adenoma
- Focal Nodular hyperplasia
- Hemangioma
- Mesenchymal hamartomas

Liver Cell Adenoma

- benign proliferation of hepatocytes
- young women (aged 20-40 years)
- associated with use of oral contraceptive pills (OCPs)
- usually singular
- Upper abdominal pain
- tumor markers are normal
- MRI scans of LCA have specific imaging characteristics
- two major risks are rupture & malignant transformation
- Hepatic artery embolization
- Resection of the mass

Focal nodular hyperplasia

- Second most common benign tumor
- young women
- small (<5 cm) nodular mass
- involves the right and left liver equally
- Related to developmental vascular malformation
- Nonspecific symptoms
- Contrast-enhanced CT and MRI scan
- Symptomatic pts: resection

Hemangioma

- Most common benign tumor of the liver
- Women more common than men (3:1 ratio)
- Mean age of about 45 years
- Occur equally in the right and left liver
- Usually single
- Lesions > 5 cm are arbitrarily called *giant hemangiomas*
- Tumor markers are usually normal
- CT and MRI scan
- Resection: enucleation with inflow control

Hepatocellular carcinoma

Introduction

- Fifth most common malignancy worldwide
- Male-to-female ratio 2.4:1
- Poor prognosis

Etiology

- Infections with hepatitis B and C viruses
- Ethanol abuse
- Obesity
- Type 2 diabetes
- Non-alcoholic fatty liver disease
- Aflatoxin B1

Pathology

- Malignant epithelial neoplasms: 85% to 95%
- Benign: 6 %to 12%
- Malignant mesenchymal tumors: 1% to 3%
- Metastatic tumors

- Tumors metastatic to the liver are:
 - Peripheral
 - Multiple
 - Cause umbilication of surface of the liver
- Primary liver tumors are:
 - Central
 - Solitary
 - Exophytic
- HCC spreads most commonly to
 - Lymph nodes around the liver
 - Peritoneal cavity
 - Lung

Characteristic feature of HCC is invasion of the portal vein

Clinical Features

Symptoms

- Abdominal pain
- Weight loss
- Weakness
- Fullness
- Anorexia
- Abdominal swelling
- Jaundice
- Vomiting

Signs

- Hepatomegaly
- Hepatic bruit
- Ascites
- Splenomegaly
- Jaundice
- Wasting
- Fever
- Virchow-Troisier nodes
- Cutaneous metastases (red-blue nodules)

Paraneoplastic Syndromes

- Hypoglycemia
- Erythrocytosis
- Hypercalcemia
- Hypercholesterolemia
- Dysfibrinogenemia
- Carcinoid syndrome
- Increased thyroxin-binding globulin
- Sexual changes (gynecomastia, testicular atrophy, and precocious puberty)
- Porphyria cutanea tarda

Diagnostic work up

- Liver function tests
- S. Alpha feto protein (70%)
- S.Des- γ -carboxy prothrombin protein (80%)
- Four-phase CT scan: unenhanced, arterial, venous, and delayed phases
- MR imaging

Characteristic feature of HCC: rapid enhancement during the arterial phase of contrast administration and “washout” during the later portal venous and delayed phases



Portal venous phase of triphasic computed tomography scan

- PET CT Scan: not recommended
- Core biopsy: hallmark feature of HCC is stromal invasion

CHILD-PUGH SCORE			
Chemical and Biochemical Parameters	Scores (Points) for Increasing Abnormality		
	1	2	3
Encephalopathy (grade) ¹	None	1-2	3-4
Ascites	None	Slight	Moderate
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time prolonged (sec) ²	1-4	4-6	>6
Bilirubin (mg/dL)	<2	2-3	>3
• For primary biliary cirrhosis	<4	4-10	>10

Class A = 5-6 points; Class B = 7-9 points; Class C = 10-15 points.

Class A: Good operative risk

Class B: Moderate operative risk

Class C: Poor operative risk

CHILD PUGH score for assessment of hepatic reserve

Quantitative assessment of hepatic reserve

Indocyanine green clearance test

- Retention rate <10%: all resections are possible
- 10% to 20%: bisegmentectomy is well tolerated
- 20% to 29%: single segment can be excised
- ≥30% or more: risk of liver failure is high

Management

Stage I and II HCC

- Surgical resection
- Local ablation (radiofrequency ablation)
- Local injection therapies (ethanol injection):
 - maximum size of tumor reliably treated is 3 cm
- Transplantation

Child-Pugh A: Resection

Child-Pugh B and C patients with stage I HCC tumors : transplant, if appropriate

Adjuvant Therapy

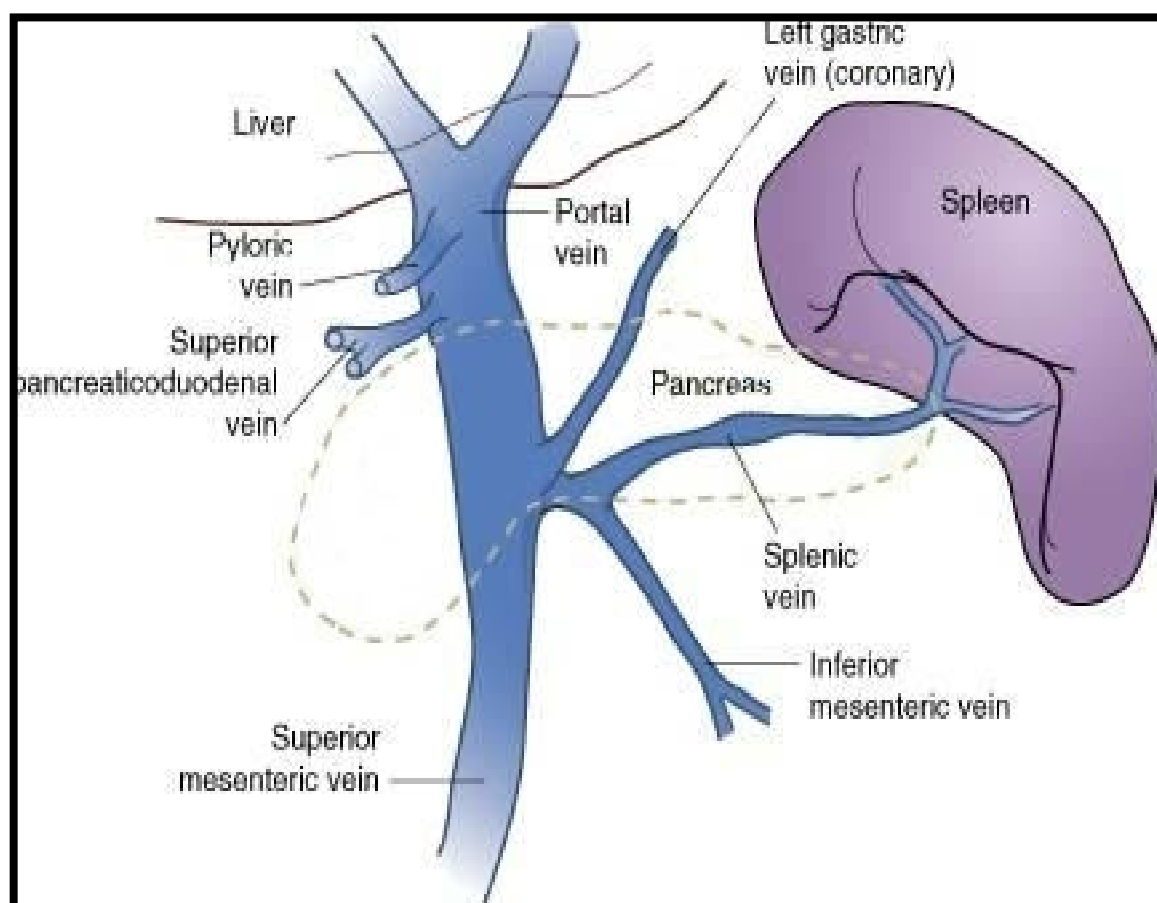
- Trans arterial chemotherapy (TACE)
- Systemic chemotherapy
 - Lipiodol
 - Doxorubicin
 - Mitomycin
 - Cisplatin

Stage III and IV Tumors

- TACE
- Regional Chemotherapy
- Systemic Therapy
- External Radiotherapy (< 8 cm)
- Hepatic Arterial Radioisotopes
 - yttrium-90 (90Y)
 - ¹³¹I antiferritin

Portal Hypertension

Anatomy



- 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

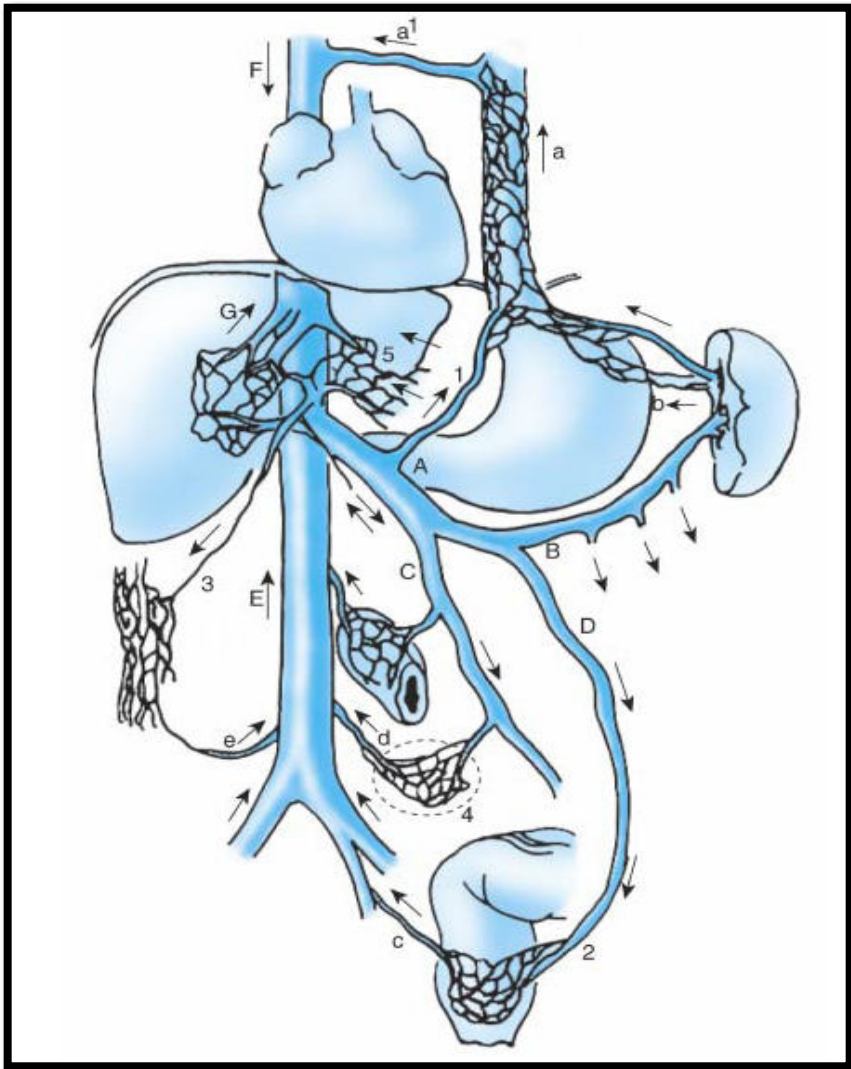
Causes

- Presinusoidal
- Sinusoidal
- Postsinusoidal

Presinusoidal
<i>Sinistral/extrahepatic</i>
Splenic vein thrombosis
Splenomegaly
Splenic arteriovenous fistula
<i>Intrahepatic</i>
Schistosomiasis
Nodular regenerative hyperplasia
Congenital hepatic fibrosis
Idiopathic portal fibrosis
Chronic active hepatitis
Myeloproliferative disorders
Sarcoid
Graft-versus-host disease
Sinusoidal
<i>Intrahepatic</i>
Cirrhosis
Alcoholic hepatitis
Postsinusoidal
<i>Intrahepatic</i>
Alcoholic terminal hyaline sclerosis
Vascular occlusive disease
<i>Posthepatic</i>
Budd-Chiari Syndrome
Inferior vena caval web
Chronic passive venous congestion

Sequelae & Clinical picture

- Porto-systemic collaterals
- Splenomegaly
- Congestion of the whole GIT
- Bleeding varices
- Ascites



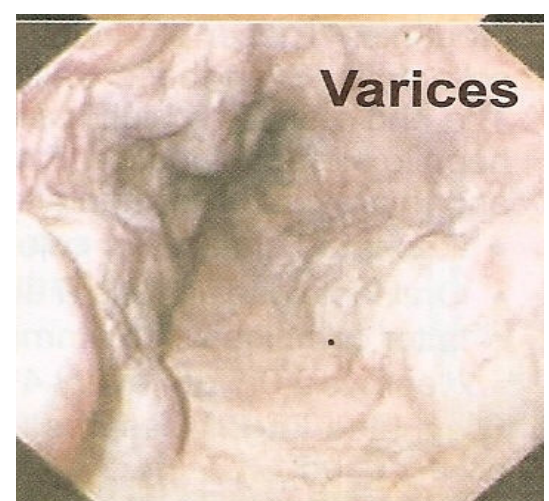
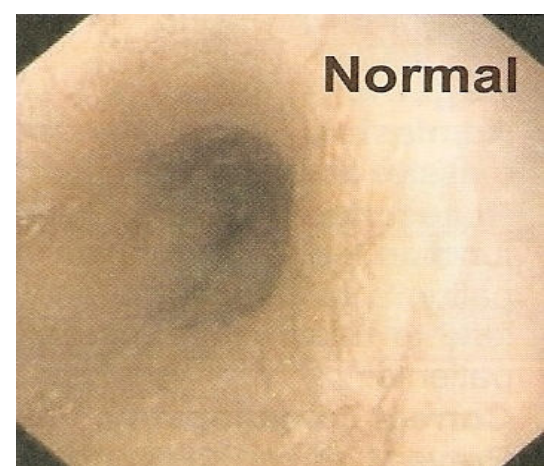
Intra-abdominal venous flow pathways leading to engorged veins (varices) from portal hypertension

Screening Tests for Portal Hypertension

Liver tests, albumin, platelet count
International Normalized Ratio
Serologies for viral hepatitis
Iron indices
Antinuclear antibody, antimitochondrial antibody, anti-smooth muscle antibody
Alpha ₁ -antitrypsin deficiency
Ceruloplasmin

- Ultrasound with doppler blood flow assessment.
- MRI venogram
- Contrast-enhanced helical CT scan

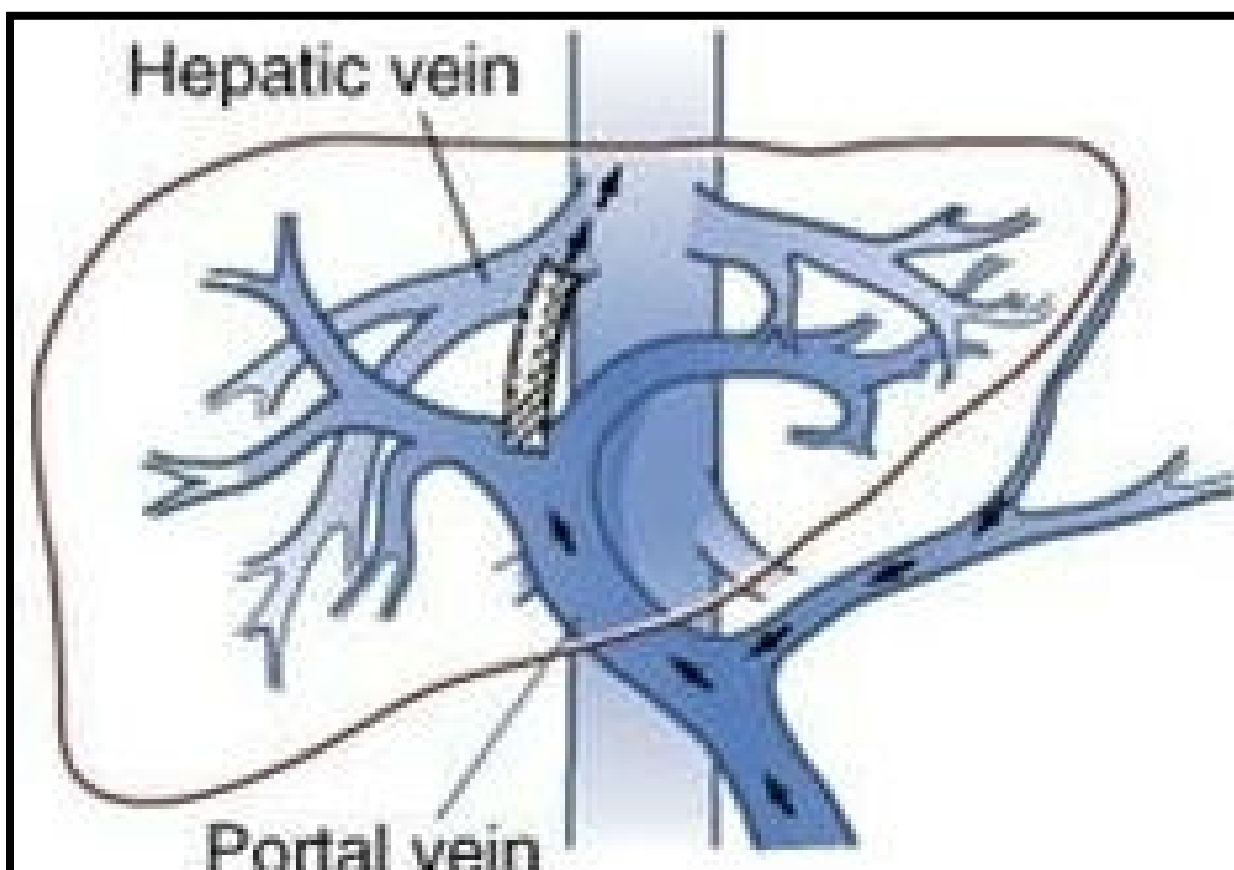
Detection of oesophageal varices



Management

- Management of patients with actively bleeding oesophageal varices
 - Resuscitation
 - Correct coagulopathy
 - Prevent encephalopathy
 - Sclerotherapy
 - Endoscopic Banding
 - Drugs: Vasopressin, Somatostatin
 - Balloon tamponade

- Trans-jugular Intra-hepatic Porto-Systemic Shunt (TIPSS)



- **Shunt operations**
 - Portocaval shunt
 - Proximal spleno-renal shunt
 - Mesocaval (Drapanas) shunt
 - Selective shunt (Warren shunt)
- **Liver Transplant**

**Chronic Liver
Disease**

Catabolism of hormones
and other serum proteins

Glucose homeostasis;
glycogenolysis & gluconeogenesis

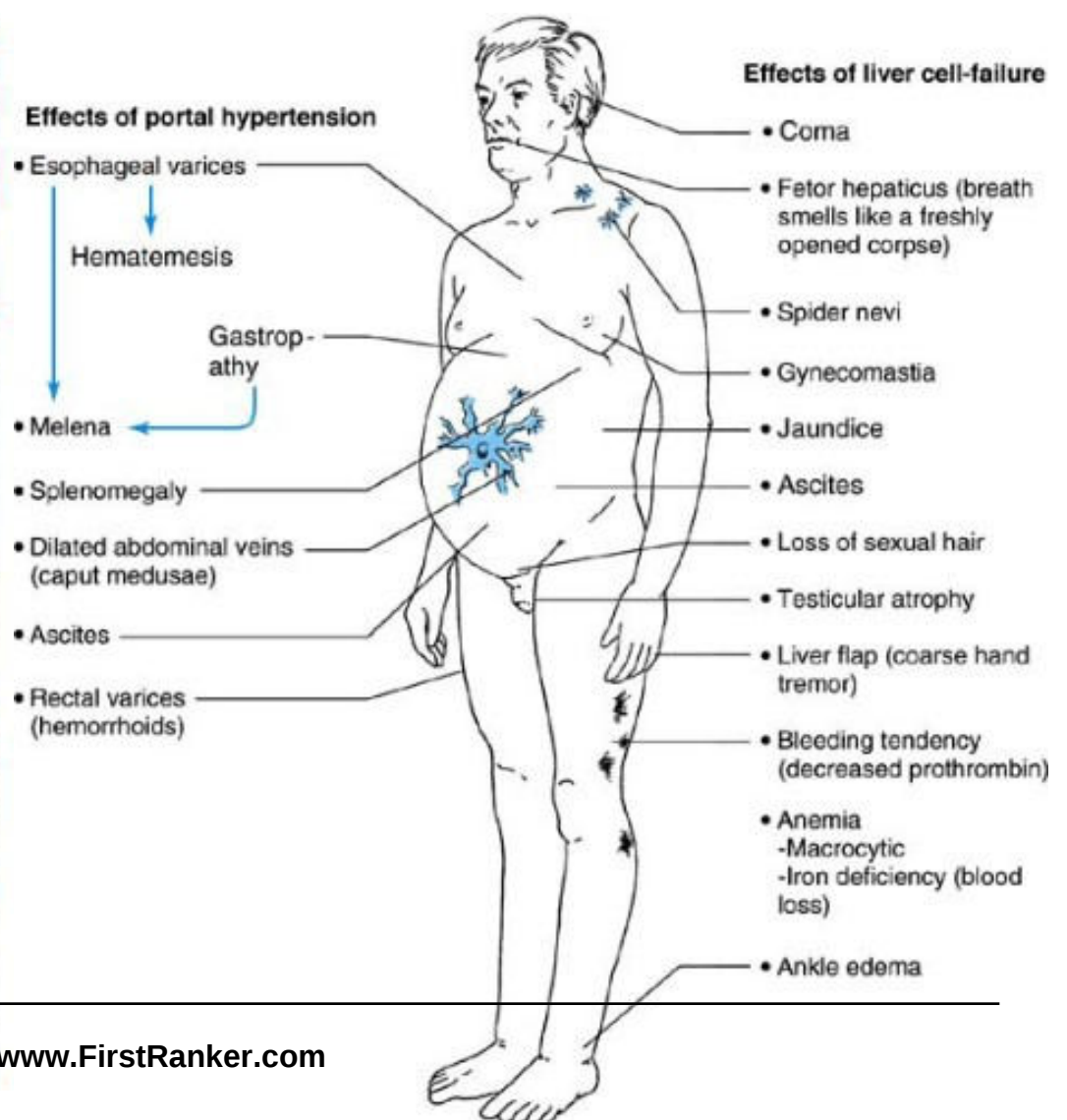
Chronic Liver Disease

Bile excretion

Synthesis:
- Albumin
- Coagulation factors

Storage:
- Glycogen
- Iron
- Cu, Iron, vitamins

Signs of CLD



Investigations

Bedside

- Observations, BM, fluid balance, weight

Blood tests

- LFTs (pre/post) (including albumin), INR
- FBC, U&Es, CRP
- Liver screen: autoantibodies, alpha-1 antitrypsin, caeruloplasmin, serum copper, ferritin, viral hepatitis serology

Imaging

- US abdomen + portal vein doppler
- CXR, CT, MRI, MRCP

Special tests

- Ascitic tap, OGD (oesophageal varices), liver biopsy

What is your management plan?

Conservative

- Alcohol abstinence, optimise nutrition, low salt diet, fluid restriction

Medical

- Vitamin B supplementation (IV/PO), chlordiazepoxide
- Diuretics
- Paracentesis (give albumin)
- NG feeding
- Antibiotics (? SBP)
- Steroids + albumin (N.B. avoid NaCl)
- Lactulose (in hepatic encephalopathy)

Surgical

- TIPSS
- Liver transplantation

Complications of CLD

- Portal hypertension: oesophageal varices, ascites
- SBP
- Hepatic encephalopathy (constipation, GI bleed, infection, renal failure)
- Hepatocellular carcinoma
- Coagulopathy
- Hepato-renal syndrome
- Liver failure

- 5 year survival rate in cirrhotic CLD 50%
- Child Pugh Score: bilirubin, INR, albumin, ascites, hepatic encephalopathy

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