Gastrointestinal System

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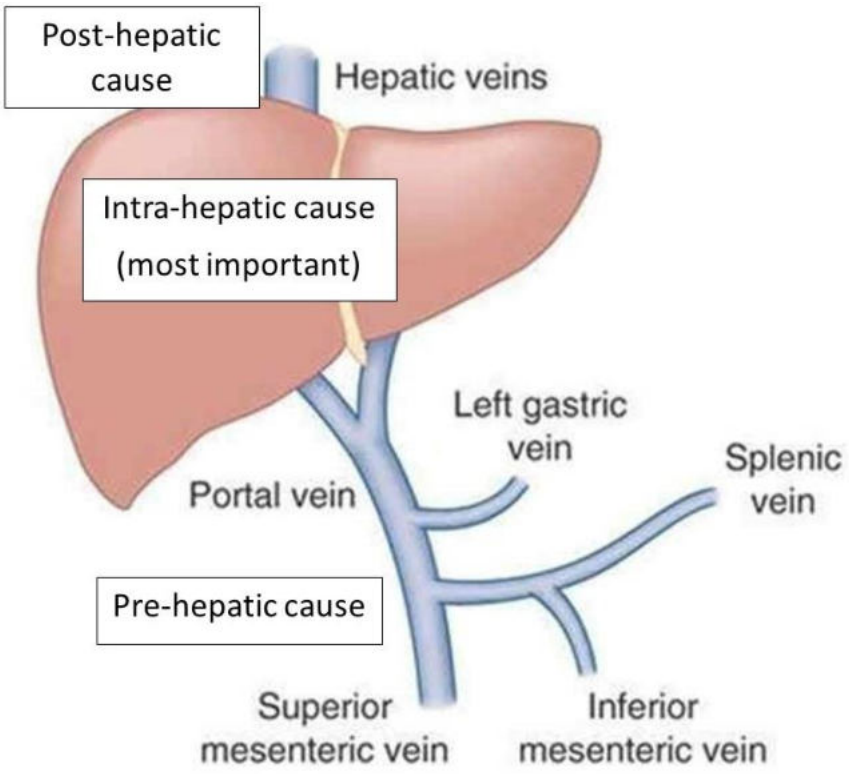
Hepatobiliary system

Portal hypertension

Characterized by an elevated portal venous pressure (normal: 9-10 mm Hg).

Causes



Pre-hepatic causes:

1. Portal vein thrombosis
2. Septic thrombus of portal vein
3. Splenic vein thrombosis
4. CA head of pancreas.

Intra-hepatic cause:

Cirrhosis

Post-hepatic cause:

1. Hepatic vein thrombosis (Budd Chiari syndrome)
2. IVC obstruction:

a. Cancer (Ex: RCC)
b. Clot (rare)

3. Long standing systemic venous congestion
   Ex:
   a. RHF
   b. Constrictive pericarditis.

Pathophysiology and Clinical features of Portal Hypertension

Pathophysiological changes	***Relevant clinical features***
Elevated portal venous hydrostatic pressure	Ascites
Opening up of portocaval collaterals	1. GI bleeding/ bleeding varices (at gastro-esophageal junction) 2. Venous prominence at superficial abdominal wall 3. Caput medusae (visible collaterals surrounding umbilicus).
Portal venous congestion--→ Splenic venous congestion	Congestive splenomegaly
Toxic products excreted by the liver bypass the liver and reaches systemic circulation through collateral circulation	1. Brain: Portocaval/ hepatic encephalopathy 2. Lung: Fetor hepaticus.
Splanchnic (GI) congestion	Congestive gastropathy
Pathophysiological changes and features of the underlying disease	

Clinical features

A	**A**scites **A**bdominal swelling
B	**B**leeding varices: ▪ Hematemesis ▪ Melena (Black semisolid stool) • It may progress into hemodynamic instability.
C	**C**aput medusae (collateral at periumbilical region) and superficial abdominal venous prominence: Direction of filling is *away from umbilicus*. **C**ongestive splenomegaly
D	Features of the underlying **d**isease
E	Portosystemic **e**ncephalopathy

F	**F**etor hepaticus (sweetish/ ammoniacal smell in the breath of the patient due to presence of mercaptan)
G	**G**astropathy (causing non-specific abdominal symptoms)
H	**H**ypersplenism (peripheral destruction of blood cells due to hyperactive reticuloendothelial [RE] cells of spleen)

Investigation

1. Blood:
   - ✓ Hb: ↓ (due to GI bleed)
   - ✓ TC, DC (Pancytopenia due to hypersplenism)
   - ✓ ESR
2. Renal function: Na+ K+ Urea Creatinine
3. Coagulation profile:
   - ✓ Platelet count
   - ✓ BT, CT
   - ✓ PT, aPTT, INR.
4. LFT: Bilirubin, Albumin, Serum protein transaminase (AST, ALT), γ-globulin
5. USG upper abdomen:
   a. Shows portal venous diameter which can roughly predict hypertension in presence of *dilated vein*
   b. Detect ascites
   c. May show splenomegaly
   d. Show any cirrhotic changes.
6. Upper GI endoscopy: To look for any varices
7. Other relevant investigations to assess the underlying cause.

Treatment

Supportive:

Treatment of:

1. Ascites
2. GI bleed
3. Underlying disease
4. Encephalopathy
5. Gastropathy.

Definitive:

Liver transplantation (if required).

Complications

1. Ascites
2. GI bleed
3. Congestive splenomegaly
4. Encephalopathy
5. Gastropathy
6. Hypersplenism.

Hepatocellular failure

Types and causes

1. Acute hepatocellular failure:

   Fulminant (Hepatic encephalopathy occurs within 8 weeks of onset of symptoms)/ Sub-fulminant (Hepatic encephalopathy occurs within 8-26 weeks of onset of symptoms):

   Causes are:
   - **A.** Acetaminophen (Paracetamol) overdose
     Acute viral hepatitis (HAV, HBV; HEV in pregnancy)
     Autoimmune hepatitis
   - **B.** Budd Chiari syndrome
   - **C.** Cardiogenic shock (Shock liver)
   - **D.** Drug induced liver injury
   - **E.** Exogenous toxin (Amanita phalloides, Aflatoxin).
2. Gradual hepatocellular failure:
   - **A.** Alcohol
     Non-alcoholic steatohepatitis
   - **B.** Budd Chiari Syndrome
     Biliary cirrhosis (Primary/ Secondary)
   - **C.** Chronic hepatitis (HCV, HBV)
   - **D.** Deposition:
     - Cu: Wilson
     - Fe: Hemochromatosis

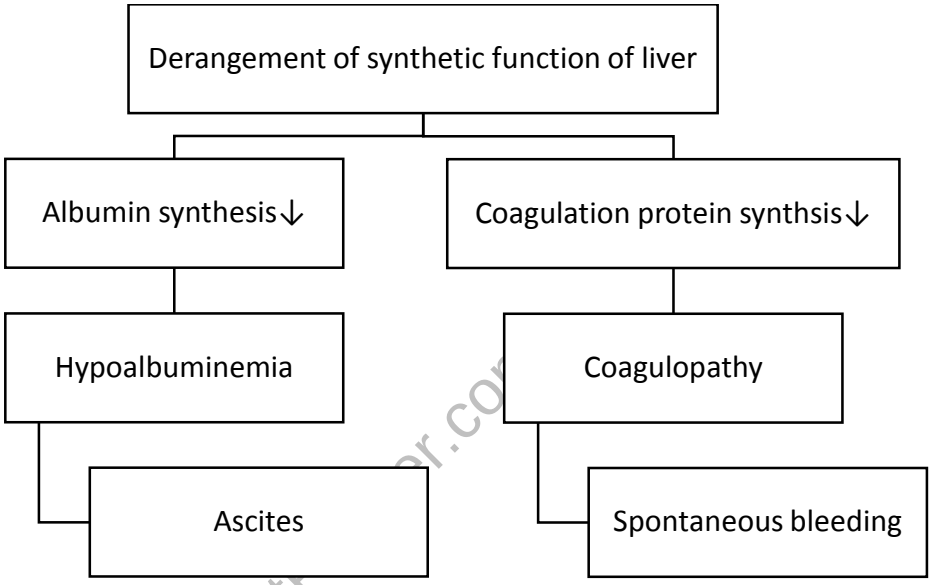
- Amyloidosis
- Glycogen storage diseases

Other uncommon causes of gradual hepatocellular failure:

- Cardiac cirrhosis
- Indian childhood cirrhosis
- Cryptogenic cirrhosis.

Pathophysiology of hepatocellular failure

1.



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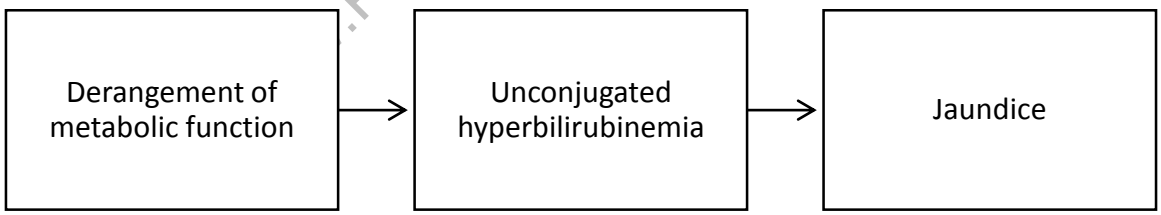

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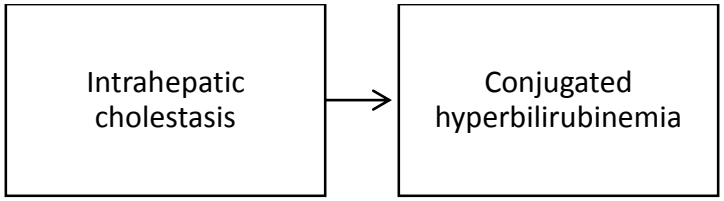

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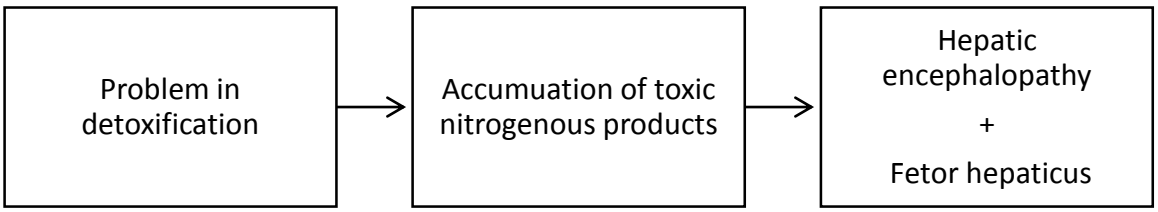

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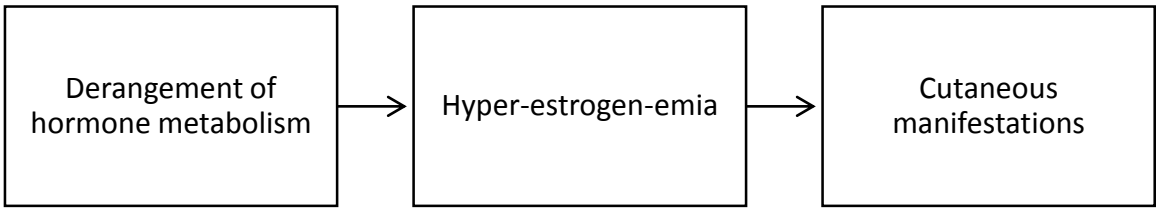

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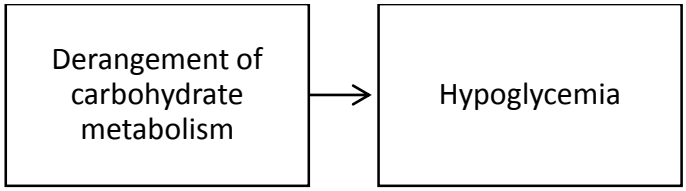

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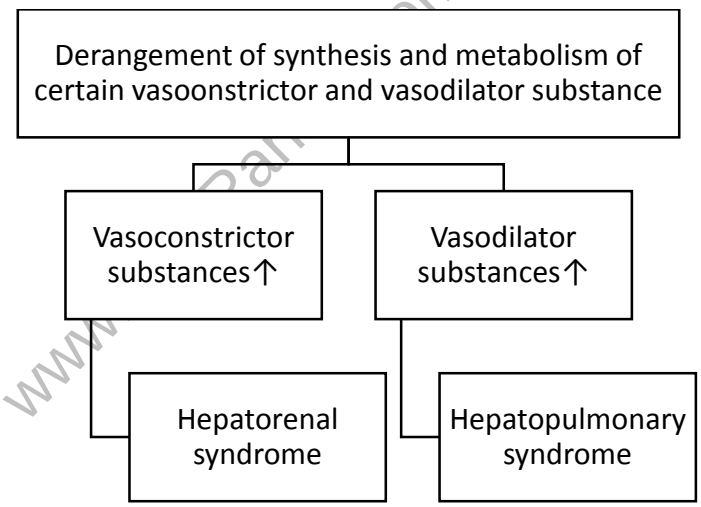


Clinical features

Symptoms (from above downwards)

- Hepatic encephalopathy (impaired consciousness)
- Yellowish discoloration of sclera (jaundice)
- Decreased frequency of shaving

- Shortness of breath (Hepatopulmonary syndrome)
- Abdominal swelling
- Pedal swelling.

Signs (from above downwards)

- Signs of hepatic encephalopathy (low GCS)
- Icterus
- Fetor hepaticus
- Loss of facial and pubic hair
- Spider nevi (at neckless area)
- Gynecomastia
- High volume collapsing pulse
- Low SpO2 (Hepatopulmonary syndrome)
- Ascites
- Testicular atrophy
- Edema
- Leukonychia (white shiny nails)
- Oliguria (Hepatorenal syndrome).

Investigations

1. Blood: Hb, TC, DC, CRP/ESR
2. Renal function: Na+ K+ Urea Creatinine
3. LFT:
   a. Bilirubin: Direct, indirect
   b. Liver enzymes: AST, ALT, GGT (signifies hepatocyte inflammation); ALP (signifies intrahepatic cholestasis)
   c. Serum albumin, globulin, total protein
   d. Coagulation profile: PT, INR (Extrinsic), aPTT (intrinsic).
4. Other relevant blood tests that diagnose the underlying cause of hepatocellular failure
5. USG abdomen:
   It can predict the underlying cause of hepatocellular failure, ascites etc.

Treatment of Hepatocellular failure

1. Supportive:
   Treatment of:
   a. Ascites

b. Bleeding diathesis
c. Underlying disease
d. Encephalopathy
e. Hepato-pulmonary/ hepato-renal syndrome
f. Hypoglycemic episodes.

2. Definitive:
   Liver transplantation.

Short note: Hyperestrogenemia in hepatocellular failure

It occurs in chronic hepatocellular failure due to derangement of testosterone metabolism, leading to:

1. Testicular atrophy
2. Loss of secondary sexual characters (sparse pubic and axillary hair)
3. Gynecomastia
4. Spider navus/ angioma.

Portocaval collaterals

Anastomotic vessels which open up between portal and systemic circulation in a patient of portal hypertension.

Effects:

Beneficial

Reduction of portal hypertension

Harmful

1. Rupture of collateral at gastro-esophageal region, leading to GI bleed
2. Toxic nitrogenous products bypass the liver and enter systemic circulation, leading to:
   a. Portosystemic encephalopathy
   b. Fetor hepaticus.

Sites:

1. Gastro-esophageal region (Varices)
2. Superficial abdominal wall
3. Falciform ligament

4. Anal canal
5. Retro-peritoneum.

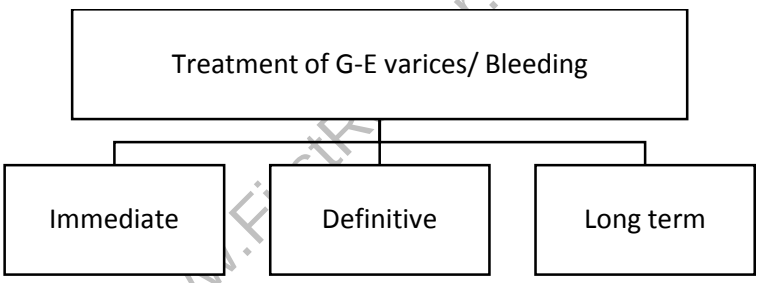
Clinical features:

1. GI bleed:
   - ✓ Hematemesis
   - ✓ Melena
   - ✓ Hemodynamic instability
   - ✓ Black tarry stool.
2. Visible collaterals:
   Superficial abdominal venous prominence ± Caput
   Direction of filling: Away from the umbilicus.
3. Portosystemic shunting through collaterals:
   - ✓ Portosystemic encephalopathy
   - ✓ Fetor hepaticus.

Investigations:

Same as portal hypertension

Treatment:



Immediate treatment

A. Airway:
- Must be protected, particularly if there is risk of aspiration
- If required: Oropharyngeal suction.

B. Breathing:
- Oxygen
- Ventilation.

C. Circulation:
- 1 wide bore cannula in each hand

- IV fluid resuscitation (Preferred fluid of choice: Normal saline)
- In case of severe bleeding: Blood transfusion (maintain a Hb level of 7-9 gm %)
- Treat any co-existing coagulopathy (platelet, vitamin K, fresh frozen plasma).

D. Drugs:

- Reduction of bleeding by splanchnic (and also systemic) vasoconstriction:
  - Vasopressin
  - Terlipressin
  - Glypressin.
- Safer and selective splanchnic vasoconstrictors (with fewer side effects):
  - Somatostatin
  - Octreotide.
- IV antibiotic therapy in all patients (to reduce the risk of potentially life-threatening infections).

Definitive treatment

E. Endoscopy:

Endoscopy confirms the presence of varices and subsequently they can be treated endoscopically by the 2 following techniques, the mechanism of both of which is stoppage of bleeding by variceal thrombosis:

- Variceal ligation (banding)
- Injection sclerotherapy:
  Intra and para-variceal administration of sclerosing agents:
  - Ethanolamine oleate
  - Sodium tetradecyl sulfate.

F. Failure of endoscopy/ not feasible:

Balloon tamponade on varices by *Sengstaken-Blackmore tube* (SSBT): It is a last resort and highly effective in controlling variceal bleeding; but it is associated with significant complications (high incidence of re-bleeding and aspiration pneumonia after removal of the tube).

Long term treatment

Prophylaxis of first episode of bleeding (primary prophylaxis)

Endoscopy

- Eradication of varices
- Surveillance.

Medical prophylaxis

- **Nonselective β blocker:**
  Propranolol reduces portal pressure by causing splanchnic vasoconstriction and reducing cardiac output.
- **Oral nitrates:**
  Nitrates reduces portal pressure by reducing hepatic vascular resistance.

Prophylaxis of subsequent bleeding episodes (secondary prophylaxis)

It is done in recurrent and severe bleeding:

Various options are:

- Transjugular intrahepatic portosystemic shunting (TIPS)
- Liver transplantation.

Ascites

Accumulation of free fluid in the peritoneal cavity.

Causes of ascites:

1. Ascites with anasarca:
   a. Right heart failure
   b. Chronic kidney disease
   c. Constrictive pericarditis
   d. Cirrhosis
   e. Nephrotic syndrome.
2. Ascites without anasarca:
   a. Peritonitis (TB/ Malignancy)
   b. Acute pancreatitis.

Clinical features:

Symptoms

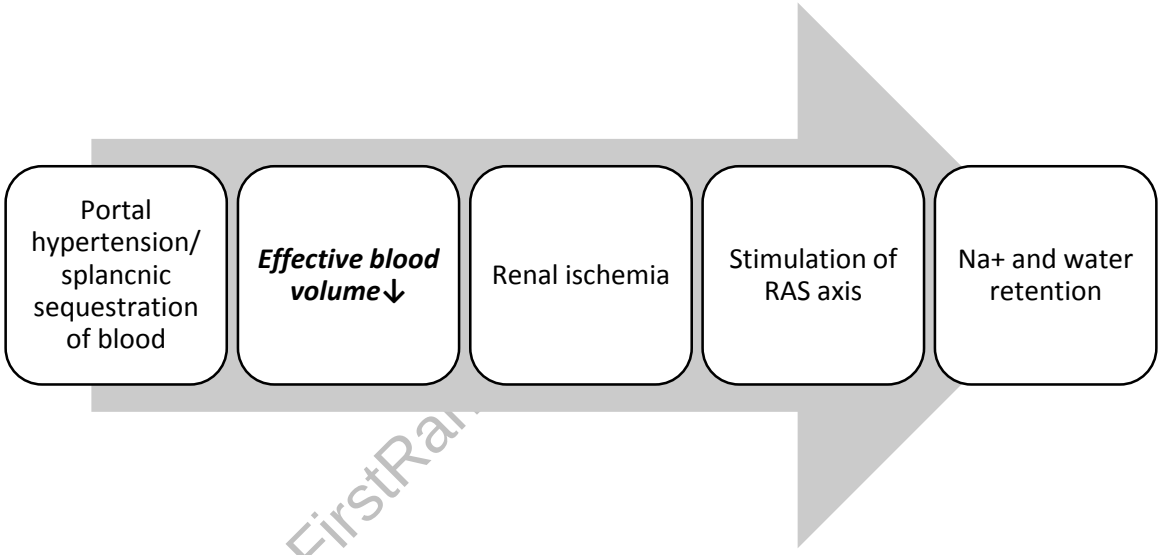
1. Progressive abdominal swelling
2. Weight gain
3. Massive ascites--→ Abdominal discomfort and shortness of breath may occur due to mechanical effect (uplifting of diaphragm).

Signs

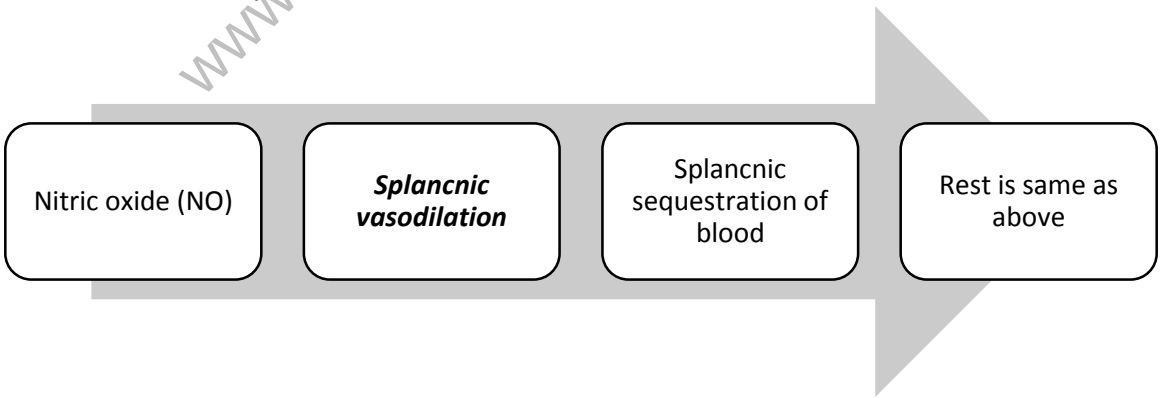
1. Shifting dullness
2. Fluid thrill may be present
3. Puddle sign (insignificant)
4. Often signs of underlying disease will help to determine causes of ascites.

Causes of ascites in cirrhosis/ CLD

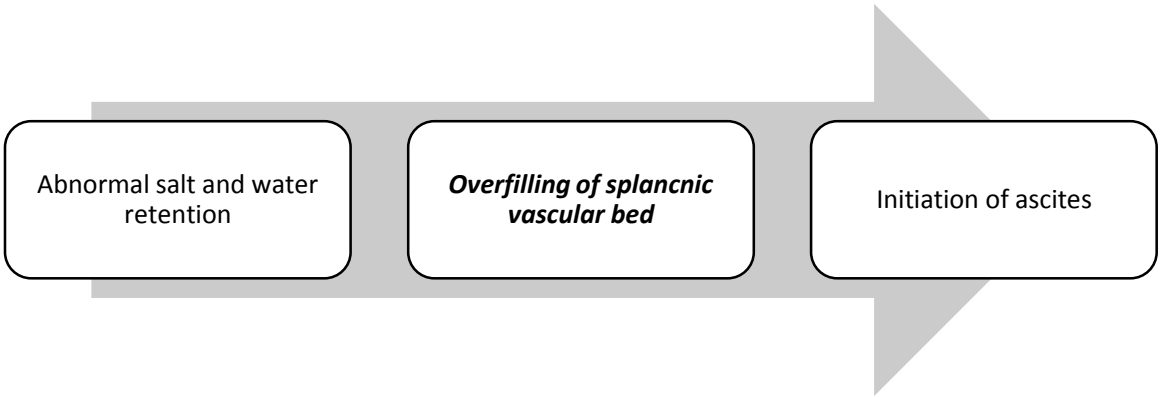
1. *Initiating factor:*
   Abnormal renal retention of Na+ and water.
   What starts this abnormality is not clear, however, there are 3 possible hypothesis:
   A. Underfilling theory:



   B. Vasodilator theory:



C. Overfilling theory:



2. *Aggravating factor:*
   A. Non-compliance to drugs/ diet prescribed
   B. Spontaneous bacterial peritonitis (SBP)
   C. Development of hepatocellular CA
   D. Tubercular peritonitis.

Investigation

1. Blood: Hb, TC, DC, CRP
2. Renal function: Na+ K+ Urea Creatinine
3. Liver function test
4. Urine analysis
5. CXR
6. ECG
7. USG
8. <u>*Diagnostic peritoneal paracentesis*</u>:
   I. Physical appearance:
      - Turbid: SBP
      - Hemorrhagic: TB/ Malignancy.
   II. Biochemical properties:
      - Serum ascitic albumin gradient (SAAG): >1.1 gm/dL: Suggestive of portal hypertension.
   III. Cytological properties:
      - WBC count:
        - ✓ Neutrophilic leukocytosis: Suggestive of SBP
        - ✓ Lymphocytic leukocytosis: Suggestive of TB/ Malignancy
      - Atypical cells: Malignancy.

IV. Microbiological properties:
- Gram stain
- AFB staining + Mycobacterial culture.

V. Special tests:
- X-PERT TB/ RIF assay:
  Detects *M.tuberculosis* genome + Rifampicin resistance (which is a very reliable indicator for MDR-TB).
- Adenosine deaminase (ADA):
  ADA levels may be high in tubercular ascites. However, it is a nonspecific marker and results should be interpreted very cautiously.

9. Endoscopy:
   If ascites is suspected to be due to cirrhosis/ portal hypertension, then look for GI varices (endoscopy).
10. Series of other tests may be required to diagnose the underlying cause.

Treatment of ascites

1. **D**iet (fluid and salt restriction)
2. **D**iuretic:
   a. Spironolactone: It is a K+ sparing diuretic; having side effects of dehydration, hyponatremia, hyperkalemia and painful gynecomastia.
   b. Furosemide: It is a loop diuretic; having side effects of dehydration, hyponatremia and hypokalemia.
3. **D**aily (regular) monitoring of the following parameters:
   a. Body weight
   b. Abdominal guts
   c. Intake and output
   d. Renal function.
4. **D**rain (Therapeutic abdominal paracentesis):
   Indication:
   a. Significant ascites (symptomatic)
   b. Ascites refractory to diuretics
   c. If required, even 5-6 L fluid may be drained in a single sitting.
   Note:
   To prevent *post-paracentesis circulatory disequilibrium*, concomitant transfusion of IV albumin preparation was practiced earlier. However, its role is doubtful.
5. Treat the underlying **d**isease.

Hepatic encephalopathy

It is a complex neuropsychiatric syndrome due to temporary cerebral dysfunction in a patient of hepatocellular failure and/or portal hypertension.

Etiopathogenesis

Underlying defect:

1. *Hepatocellular failure:*
   Toxic nitrogenous products and other toxins can't get detoxified in the liver, so they reach systemic circulation and then go to brain causing encephalopathy.
2. *Portal hypertension:*
   Due to portosystemic collaterals, toxins bypass liver and reach systemic circulation and go to brain.
   Ultimately these toxins; after reaching the brain cause cerebral dysfunction, leading to encephalopathy.

Toxins responsible for hepatic encephalopathy:

A. **A**mmonia
M. **M**ercaptan
M. **M**anganese
O. **O**ctopamine
N. Be**n**zodiazepine
I. **I**nhibitory neurotransmitters (GABA)
A. Fatty **a**cids.

Aggravating/ precipitating factors:

1. *Conditions which cause ↑ protein load:*
   - High dietary protein intake (especially animal protein)
   - GI bleeding
   - Uremia/ azotemia (pre-renal renal failure).
2. *Conditions which cause ↑ ammonia production in intestine:*
   - Constipation
   - Metabolic alkalosis (which may occur in hypokalemia caused by loop diuretics): Here conversion of NH_3 to NH_4^+ is hampered.
3. *Hepatotoxins:*
   - Alcohol
   - Sedatives.
4. *Infection:* Commonly SBP is responsible for it.

Clinical features

Symptoms

A. Altered sensorium
B. Behavioral change (Indifference to others, childish behavior)
C. Confusion, coma
D. Delirium, disturbed sleep rhythm (reversal of sleep-wake cycle), disturbed mood
E. Features of cerebral edema (due to ↑ICT)
F. Flapping tremor
G. ↓GCS.

Signs

1. Apraxia:
   Inability to perform a voluntary skillful activity in spite of normal motor, sensory cerebellar and extrapyramidal structures.
   Constructional apraxia:
   Inability to draw a 5 pointed star
   When asked to join 20 numbered points by a line, the patient either takes very much time or unable to do it.
2. Jerks:
   Usually normal, however in deep coma: hypo/a-reflexia may be seen.
3. Plantar:
   Normal, however in deep coma: plantar may be bilateral extensor/ absent.

Investigation

1. Blood: Hb, TC, DC, CRP/ESR
2. Renal function: Na+ K+ Urea Creatinine
3. Liver function test
4. Arterial NH_3 level: ↑ (But it is a nonspecific marker)
5. Arterial blood gas: To assess acid base equilibrium
6. USG abdomen
7. Upper GI endoscopy (to look for any varices)
8. Diagnostic ascitic fluid tap and analysis
9. CT head: Particularly if raised ICT is suspected/ the cause of encephalopathy is not clear.

Treatment

A. Airway:
To be protected, particularly if in deep coma.

B. Breathing:
Oxygen and if required, ventilation.

C. Circulation:
- Regular capillary blood glucose monitoring
- IV fluid (containing dextrose)

- To be avoided in presence of significant cerebral edema.

Constipation:

Prevention of constipation by:

- Lactulose (Per oral/ Per rectal)
- Lactitol
- High bowel wash enema
- Rifaximine.

D. Diet:
Dietary protein restriction.

E. Cerebral edema:
Mannitol.

Electrolyte imbalance:
Any hypokalemia is promptly treated.

F. Fever (infection):
Any infection should be promptly treated by antibiotics.

G. GI bleed:
It should be promptly treated.

H. Hypovolemia: Promptly treated.
Hepatotoxic drugs: To be avoided.

Role of lactulose in hepatic encephalopathy

1. Being a non-absorbable disaccharide, it gets metabolized in the gut, to form H+ which converts toxic NH_3 to nontoxic NH_4+.
2. It directly acts on ammonia forming colonic flora and reduces their load.
3. It also acts as a laxative.

Spontaneous bacterial peritonitis (SBP)

It is primarily a bacterial infection of ascitic fluid, in absence of any intra-abdominal focus of infection.

Risk factors:

Any patient with ascites are at risk of SBP, however, chronic liver disease/ cirrhotic patients, particularly those with ***ascitic fluid albumin level <1gm/dL*** are at greater risk as ***the fluid is often deficient in opsonins***.

Pathogens:

- E.coli
- Streptococcus

- Organisms enter ascitic fluid either by translocation across the gut wall/ through intestinal lymphatics.

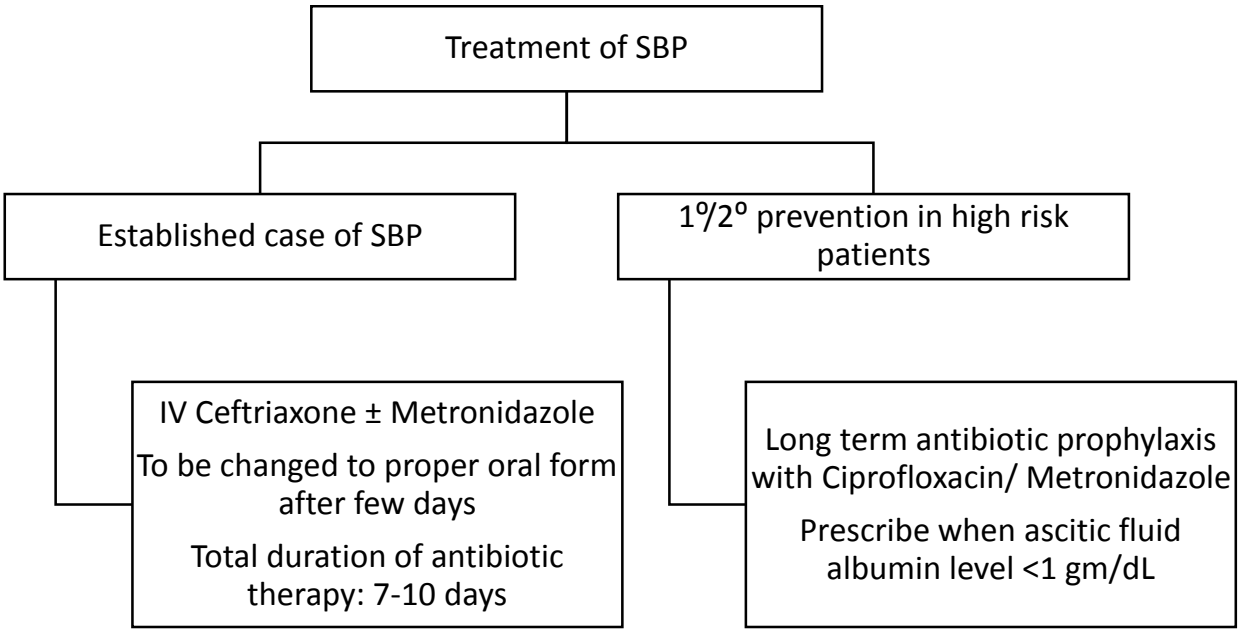
Clinical features:

1. Fever
2. Pain abdomen
3. Aggravation of ascites
4. Hepatic encephalopathy may get precipitated
5. Tenderness ± ***signs of peritonitis are usually absent***.

Investigation:

1. Blood: CBC, ESR/ CRP, Blood culture sensitivity
2. Ascitic fluid tap
3. Ascitic fluid:
   a. Cell count (WBC count > 500/cu.mm.)
   b. Gram stain + culture sensitivity.

Treatment:

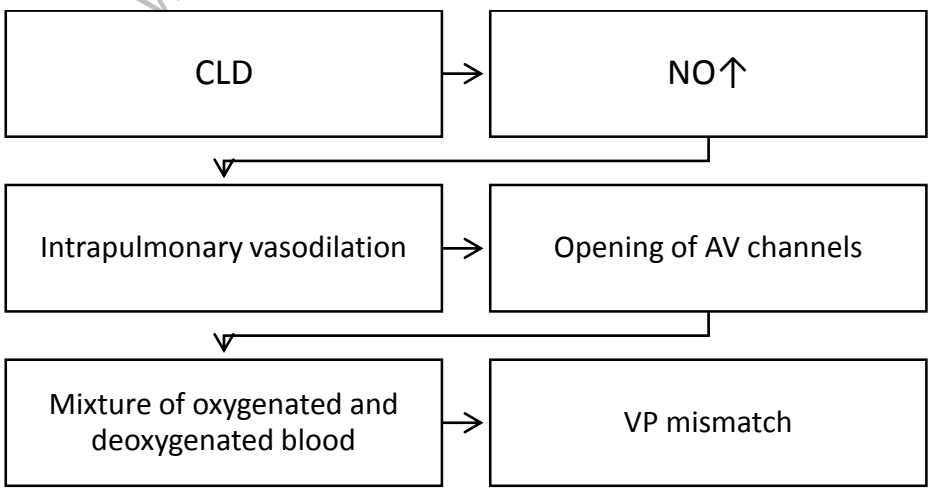


Hepatopulmonary syndrome (HPS)

It is a pulmonary complication of chronic liver disease characterized by widespread *intrapulmonary vasodilation* and *arterio-venous communication.*

Pathogenesis:

In CLD, there is abnormally high pulmonary nitric oxide (NO) level, leading to intra-pulmonary vasodilation and opening up of intrapulmonary arterio-venous communicating channels, which ultimately leads to admixture of oxygenated and deoxygenated blood and ventilation-perfusion mismatch.



Clinical features:

Symptom

Breathlessness/ shortness of breath

Signs

- Shortness of breath becomes more prominent when the patient is in standing position: ***Platypnea***.
- Arterial oxygen saturation (SaO_2) falls when the patient is in standing position: ***Orthodeoxia***.

Investigation

1. Arterial blood gas (ABG)
2. Chest X Ray (CXR): To rule out focal lung lesion
3. ECG and Echocardiogram: To rule out any cardiac abnormality
4. Highly specialized test (expensive): *Contrast echocardiogram with saline agitated microbubble*: Injected bubbles appear very quickly in the left atrium due to intrapulmonary shunting.

Treatment

1. Oxygen
2. Liver transplantation.

Portopulmonary hypertension

Pulmonary hypertension occurring as a consequence of CLD.

It occurs due to abnormal circulating level of ***Endothelin***: a potent pulmonary vasoconstrictor.

Treatment:

1. Bosentan: Endothelin antagonist
2. Sildenafil: NO donor.

Hepatorenal syndrome

A state of renal dysfunction occurring as a consequence of CLD.

Diagnostic criteria:

1. Presence of azotemia: Creatinine↑
2. No pre-renal/ intrinsic renal causes of kidney dysfunction (Ex.: dehydration)
3. No improvement of renal function even after withholding diuretic therapy for 48 hours.

Pathogenesis:

- Due to CLD, there is impaired synthesis/ malabsorption of renal vasodilators, leading to ***intense renal vasoconstriction***; leading to reduced renal blood flow.
- There is altered hemodynamics but ***no structural damage*** to kidneys.

Clinical features:

1. Oliguria/ anuria
2. Symptoms and signs of volume overload
3. Features of ***uremic encephalopathy*** (due to accumulation of toxins in blood).

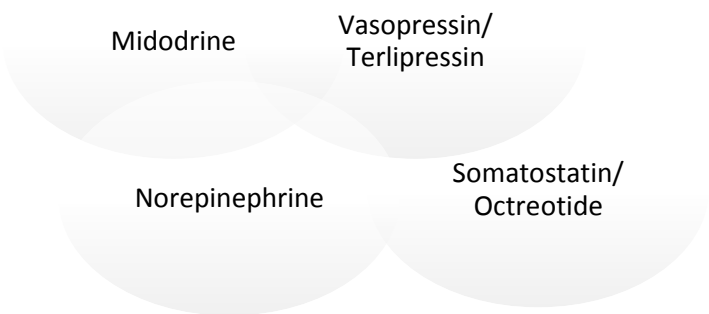
Investigations:

Renal function test: Na+ K+ Urea Creatinine

- Urea creatinine: ↑
- Serum Na+: ↓ (Dilutional hyponatremia); Urinary Na+: ↓
- Serum K+: ↑.

Treatment:

1. Any other cause of renal dysfunction needs to be treated
2. Combination of medical agents:



3. Liver transplantation
4. A special dialysis method which selectively albumin bound compound (experimental).

Coagulopathy

Cause:

Impaired synthesis of coagulation factors due to deranged hepatic function.

Clinical features:

1. Asymptomatic
2. Bleeding manifestation:
   External: Gum bleeding, epistaxis, ecchymosis
   Internal: Intracerebral hemorrhage, GI bleed, GU bleed.
- If blood loss is significant, patient may become hemodynamically unstable.

Investigation:

Coagulation profile

1. BT/ CT/ Platelet count
2. PT, INR (Extrinsic pathway) ↑
3. aPTT (Intrinsic pathway) ↑
   - Often only PT/INR is elevated in CLD as *factor VII is the first factor to get depleted*.

Treatment:

1. Fresh frozen plasma transfusion, only when there is ***active bleeding***/ before invasive procedure
2. Although it is very commonly used, ***vitamin K has no role***, unless coexistent vitamin K deficiency is suspected
3. Recombinant factor VIII concentrate.

Congestive splenomegaly

It is a common complication of portal hypertension where portal and splanchnic venous congestion leads to passive congestion in the spleen and finally, splenomegaly.

Hypersplenism

It can occur in a patient with long standing portal hypertension and defined as the combined presence of the following:

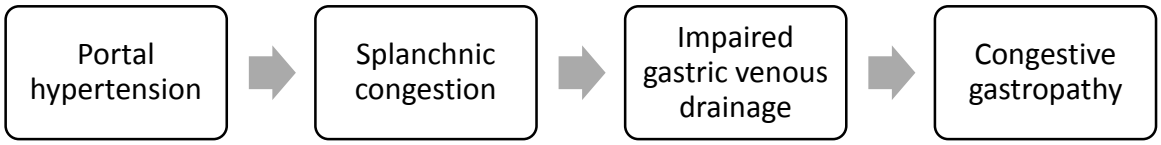
1. Splenomegaly
2. Pancytopenia (which gets corrected after splenectomy)
3. Bone marrow hyperplasia.

Cause of pancytopenia:

Excessive splenic sequestration of peripheral blood cells due to overactivity of RE cells.

Congestive gastropathy

- Pathogenesis:



- Clinical features:
  Patients are often asymptomatic; sometimes may present with nonspecific symptoms like epigastric discomfort ± pain.
- Diagnosis:
  Endoscopy shows gastric mucosal congestion and hyperemia.

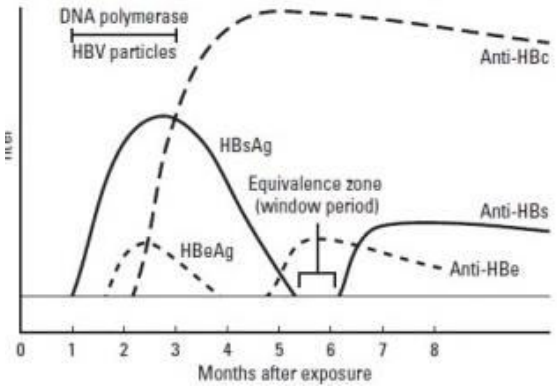
Acute viral hepatitis

Virus	Incubation period (days)	Mode of transmission	Acute hepatitis	Fulminant hepatic failure	Chronic hepatitis
HAV	15-45	Faeco-oral	+	+	-
HBV	30-180	Sexual Parenteral Vertical	+	+	++
HCV	50-160	Sexual Parenteral	-	-	++
HDV	30-180	Sexual Parenteral	+	-	-
HEV	15-60	Faeco-oral	+	+ (in pregnancy)	-

Viral markers

1. HAV: Anti-HAV antibody: IgM (acute) and IgG (remote)

2. HBV:
   - ***a. HBsAg:***
     - I. In most cases, the first viral marker to appear
     - II. Appears very early during acute hepatitis and usually disappear within few months
     - III. If persists 6 months after an episode of acute hepatitis, then the patient may be: a carrier/ entered into chronic hepatitis phase.
   - ***b. Anti-HBs-antibody:***
     - I. Appears after disappearance of HbsAg
     - II. Presence signifies immunity by vaccine/ a previous episode of acute hepatitis
   - ***c. Anti-HBc-antibody***:
     It is of 2 types:
     - I. IgM: Rises during acute infection
     - II. IgG: Develops during acute period and persists indefinitely.
   - ***d. HBeAg:***
     - I. It is a secretory form of core antigen
     - II. Appears during acute infection
     - III. It is a marker of active viral replication
     - IV. Therefore, its presence suggests *high infectivity*
     - V. Usually disappears within few months of acute infection, if persists beyond 3 months, it suggests a high possibility of chronic hepatitis.
   - ***e. Anti-HBe-antibody:***
     Appears after disappearance of HbeAg
   - ***f. HBV-DNA:***
     - I. Highly sensitive marker of viral replication
     - II. Quantification of HBV-DNA denotes *viral load* and therefore is monitored to assess *antiviral treatment response* in chronic hepatitis.



A way to remember the sequence of appearance of viral markers in hepatitis B:

Antigens:
S: HBsAg
E: HBeAg
D: HBV-DNA

Antibodies:
C: Anti-HBc (IgM)
E: Anti-HBe
S: Anti-HBs
C: Anti-HBc (IgG)

HBsAg	Anti-HBs	***Anti-HBc***	***HbeAg***	Anti-HBe	Interpretation of test results
+	-	IgM	+	-	Active hepatitis high infectivity
+	-	IgG	+	-	Chronic hepatitis high infectivity
+	-	IgG	-	+	Chronic hepatitis low infectivity
-	-	IgM	+	+	Acute infection
-	+	-	-	-	Immune (vaccinated person)
-	+	IgG	-	+	Previous infection

3. HCV:
   a. Anti-HCV:
      Usually appears during acute period of infection
      Although in substantial proportion of Hepatitis C patients will have detectable Anti-HCV, a subpopulation of the patients will be Anti-HCV –Ve.
   b. HCV-RNA:
      I. Detectable during acute period of infection
      II. Signifies *active viral replication*
      III. Quantification denotes viral load and is monitored during antiviral treatment.
4. HDV:
   It causes co/super-infection with HBV. The marker is HDV-RNA.
5. HEV:
   Anti-HEV-antibody:
   It is of 2 types: IgM and IgG.

Clinical features of viral hepatitis

Acute viral hepatitis

It goes through 3 stages:

a. Pre-icteric stage (few days-1 week):
   - ✓ Nausea and vomiting
   - ✓ Apathy/ distress for food
   - ✓ Right upper quadrant pain
   - ✓ Fever
   - ✓ Arthralgia
   - ✓ Myalgia.
   - In some patients with acute HepB, serum sickness like illness (fever + rash + arthralgia) may occur.

b. Icteric stage (1-3 week):
   - ✓ Symptoms start to subside
   - ✓ Icterus
   - ✓ Soft/ form tender hepatomegaly
   - ✓ Mild splenomegaly.

c. Recovery/ convalescent stage:
   Symptoms and signs gradually disappears and patient normalizes.

Examination may reveal RUQ pain and soft tender hepatomegaly.

Investigation

1. Blood: Hb, TC, DC, CRP/ESR: Leukopenia may be seen
   In smear, atypical lymphocytes may be seen.
2. Renal function test: Na+ K+ Urea Creatinine
3. Liver function test:
   a. Bilirubin (Unconjugated and conjugated): ↑
   b. Hepatic transaminases:
      I. ALT, AST: ↑↑↑ (often level rises to >1000 U/L)
         The rise of ALT is typically > rise of AST.
      II. GGT/ ALP: ↑ (mild to moderate rise in level)
   c. Albumin: ↓
   d. PT, INR, aPTT: ↑
      *Low albumin level and coagulation abnormalities are warning signals of an impending hepatic failure.
4. Viral markers:
   a. Anti-HAV

b. HbsAg
c. Anti-HCV

5. USG abdomen.

Treatment

A. Absolute bed rest:
Particularly when the patient is symptomatic, bed rest should be continued till transaminase levels come down/ patient becomes asymptomatic.

B. Bowel clearance: Lactulose

C. Circulatory support by IV fluid of oral intake is inadequate. Usually dextrose containing fluid is given.
Monitor capillary blood glucose regularly.

D. Diet: Normal palatable diet, restrict protein in case of encephalopathy.
Drugs: Avoid drugs which are hepatotoxic/ having hepatic metabolism.

E. Look for early signs of encephalopathy.

Complications

1. Early: Fulminant hepatic failure
2. Late:
    I. Cirrhosis
    II. Chronic hepatitis
    III. Hepatocellular carcinoma.

Alcoholic liver disease

Risk factors:

1. Alcohol related:
    - ✓ Amount (>50 gm/day for 10 years)
    - ✓ Type
    - ✓ Binge drinking.
2. Non-alcohol related:
    - ✓ Malnutrition
    - ✓ Obesity
    - ✓ Coexisting liver disease.

Stages and clinical features:

1. ***Alcoholic fatty liver (steatosis)***:
   - ✓ Often asymptomatic
   - ✓ Mild RUQ pain
   - ✓ RUQ tenderness ± Hepatomegaly.
2. ***Alcoholic hepatitis***:
   - ✓ RUQ pain
   - ✓ Loss of appetite
   - ✓ Nausea
   - ✓ Fever ±
   - ✓ RUQ tenderness ± Hepatomegaly.
3. ***Alcoholic chronic liver disease/ cirrhosis***:
   - ✓ Symptoms and signs of chronic hepatocellular failure
   - ✓ Symptoms and signs of portal hypertension.

Stigma of chronic alcohol excess:

1. ***Haptic facies***: Wasted face with facial hollowing + muddy discoloration
2. ***Bilateral parotid*** **swelling**
3. ***Dupuytren's contracture***: Fibrotic thickening of palmar fascia leading to fixed flexion deformity and limited extension of ring and little fingers
4. ***Hyperestrogenemic manifestations***: More prominent than other causes of CLD.

Investigation

1. Blood: Hb, TC, DC, ESR/ CRP
   Hb: ↓ in case of GI bleed
   TC, DC: Leukocytosis in case of alcoholic hepatitis
   Cytopenia: Due to marrow toxic effect of alcohol
   ↑MCV: Due to coexisting folic acid deficiency.
2. Renal function test: Na+ K+ Urea Creatinine:
   Urea-creatinine level may be ↓ due to low catabolic state.
3. Liver function test:
   a. Bilirubin: May be ↑
   b. ALT, AST: Mild to moderate ↑, usually don't exceed 300 U/L, $\frac{AST}{ALT} > 1$ (due to alcohol induced inhibition of pyridoxal phosphate, a coenzyme of all transaminases)
   c. GGT, ALP: Mild to moderate ↑ (markers of cholestasis)
   d. Albumin: Normal/ ↑

e. PT, aPTT, INR: Normal/ ↑.

4. USG abdomen:
   May show:
   a. Fatty liver
   b. Hepatomegaly
   c. Evidence of portal hypertension
   d. Ascites.
5. Upper GI endoscopy:
   To look for varices.

Treatment of alcoholic liver disease

A. Abstinence:
If required, drugs can be given to control acute withdrawal symptoms:
- ✓ Chlordiazepoxide
- ✓ Diazepam
- ✓ Acamprosate.

B. Bowel clearance:
Lactulose + Vitamin B supplementation

C. Circulatory support by dextrose containing IV fluid
Capillary blood glucose monitoring

D. Drugs:
Hepatotoxic drugs/ drugs with significant hepatic metabolism should be avoided in chronic liver disease.

I. Corticosteroid:
Methyl-prednisolone/ its dose equivalent: for 4 weeks
If modified Maddrey's discriminant score >32

$$Modified\ Maddrey's\ discriminant\ score = [4.6\ x\ (PTtest\ -\ PTcontrol)] +\ S.Bilirubin\ in\ mg/dl$$

II. Pentoxifylline (TNF inhibitor): For a duration of 4 weeks

E. Treatment of associated complication(s).

Non-alcoholic fatty liver disease (NAFLD)/ Non-alcoholic steatohepatitis (NASH)

Definition:

Liver disease due to fatty infiltration with/ without accompanying inflammation in absence of alcohol abuse.

Risk factors:

- High BMI
- Diabetes
- Dyslipidemia
- Hypertension
- Metabolic syndrome (Syndrome X)
- Drugs (Corticosteroid/ Amiodarone)
- Endocrine (Cushing's syndrome/ Polycystic ovarian syndrome).

Pathogenesis:

The following factor play important role in NAFLD:

Insulin resistance:
It ultimately leads to lipid peroxidation and oxidative damage to the liver

(In Alcoholic liver disease, 2 important factors are:
a. Toxic effect of acetaldehyde
b. Toxic effect of TNF).

Clinical features:

1. Often asymptomatic: Detected incidentally
2. RUQ discomfort ± Pain
3. RUQ tenderness ± Hepatomegaly
4. In advanced cases, chronic liver diseases or cirrhosis may occur.

Investigation

1. Blood: Hb, TC, DC, CRP
2. Renal function: Na+ K+ Urea Creatinine
3. Liver function:
   a. Variable deranged
   b. Usually ALT> AST
   c. In advanced cases progressing to fibrosis: AST > ALT.

4. Fasting lipid profile
5. Blood glucose: Fasting and post-prandial
6. USG abdomen:
   Evidence of steatosis: fatty infiltration is seen (steatosis and steatohepatitis are not amenable to differentiation).
7. Upper GI endoscopy: If CLD is suspected
8. Liver biopsy.

Treatment

1. Risk factor modification:
   a. Lifestyle modification
   b. Drug treatment

Lifestyle modification

I. Weight loss
II. Avoid alcohol
III. Dietary modification
IV. Drugs:
   - ✓ Anti-hypertensive
   - ✓ Anti-diabetic
   - ✓ Anti-lipidemic
   - ✓ Anti-obesity.

Drug treatment

I. Reduction of insulin resistance:
   - ✓ Metformin
   - ✓ Pioglitazone.
II. Antioxidants:
   - ✓ Vitamin E
III. Treatment of chronic liver disease (if present).

Hemochromatosis

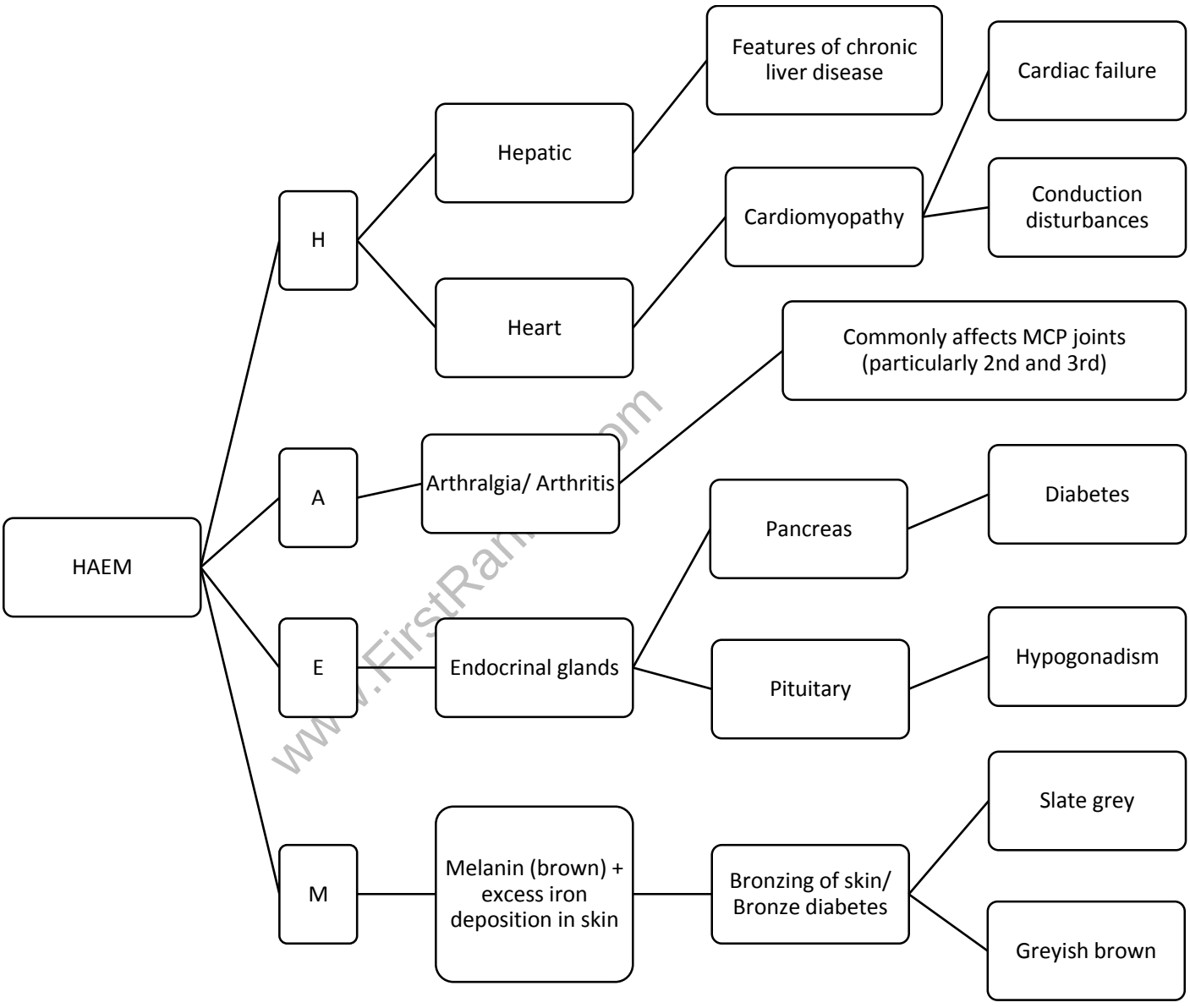
Definition:

It is a disorder of iron metabolism characterized by excessive hepatic and extrahepatic iron deposition.

Pathogenesis:

1. ***HFE gene***: This gene plays a vital role in sensing body iron stores. In mutations of this gene, there is ultimately excess intestinal iron absorption.
2. ***Hepcidin***: It is a key iron regulatory protein which is not synthetized properly. So, there is excessive hepatic iron deposition which spills over into extrahepatic sites.

Clinical features:



Investigations

To diagnose organ defect:

1. Blood & liver function: Nonspecific abnormality
2. Blood glucose: Fasting and post-prandial
3. ECG
4. Echocardiogram
5. To confirm hemochromatosis:
   a. Serum iron: ↑↑
   b. Serum ferritin: ↑↑
6. Liver biopsy: With estimation of hepatic iron index
7. Molecular testing: Detection of C282Y mutation.

Treatment

1. Supportive treatment:
   For different complications
2. Treatment of iron deposition:
   a. Regular phlebotomy
   b. Iron chelators: Desferrioxamine: SC/IV infusion.

Wilson's disease

Definition:

It is a disorder of copper metabolism characterized by excessive hepatic and extrahepatic copper deposition.

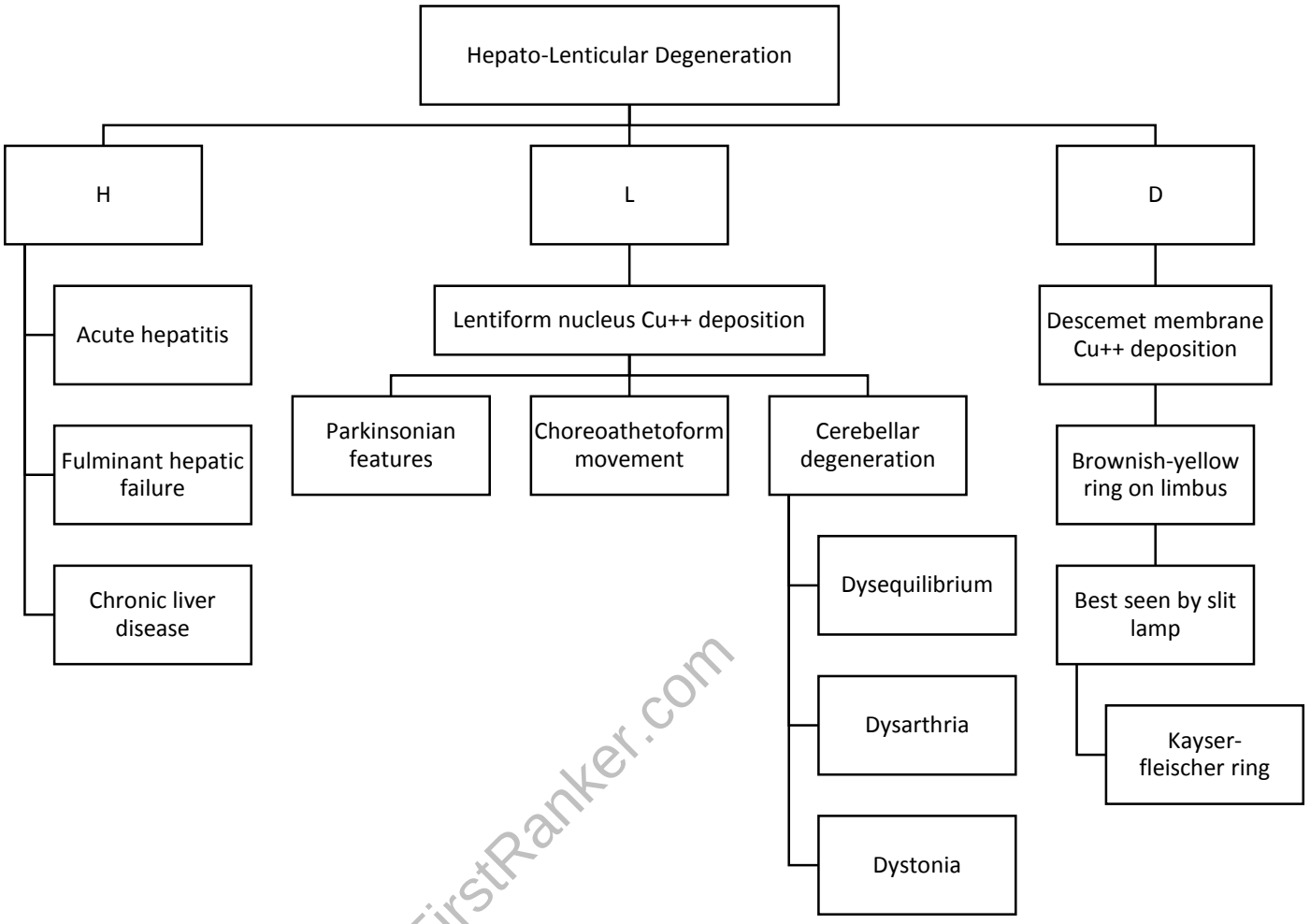
Risk factors:

2 important factors which lead to excessive hepatic copper deposition which spills over different extra-hepatic sites are:

1. Excessive intestinal Cu++ absorption
2. Impaired hepatic Cu++ excretion.

There is underlying defect in the key transporter protein ATP7B which is responsible for all of these.

Clinical features:



Investigation

To detect organ damage:

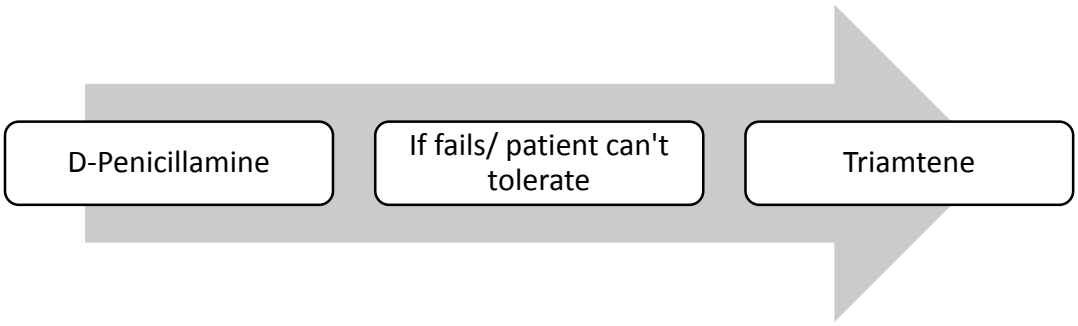
1. Liver function test
2. MRO brain.

To confirm the disease:

1. Serum Cu++: ↓
2. Ceruloplasmin: ↓
3. Urinary Cu++: ↑
4. Liver biopsy: It estimates hepatic Cu++ store.

Treatment

1. Cu++ chelating agent:



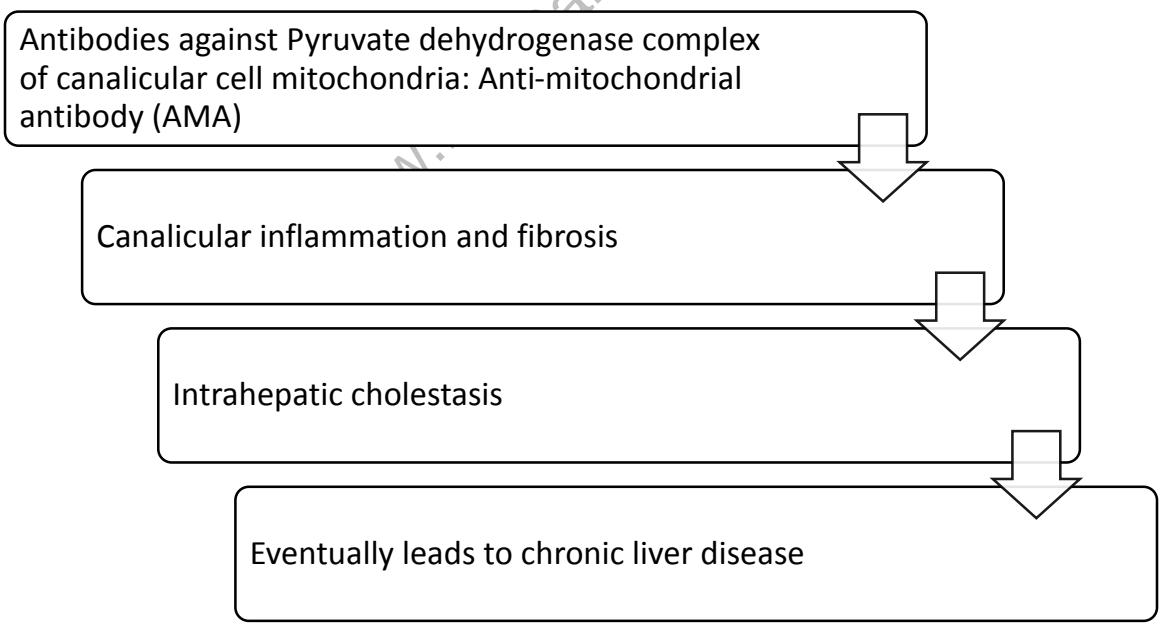
2. Reduction of Cu++ absorption from intestine: By zinc
3. Treatment of chronic liver disease.

Primary biliary cirrhosis (PBC)

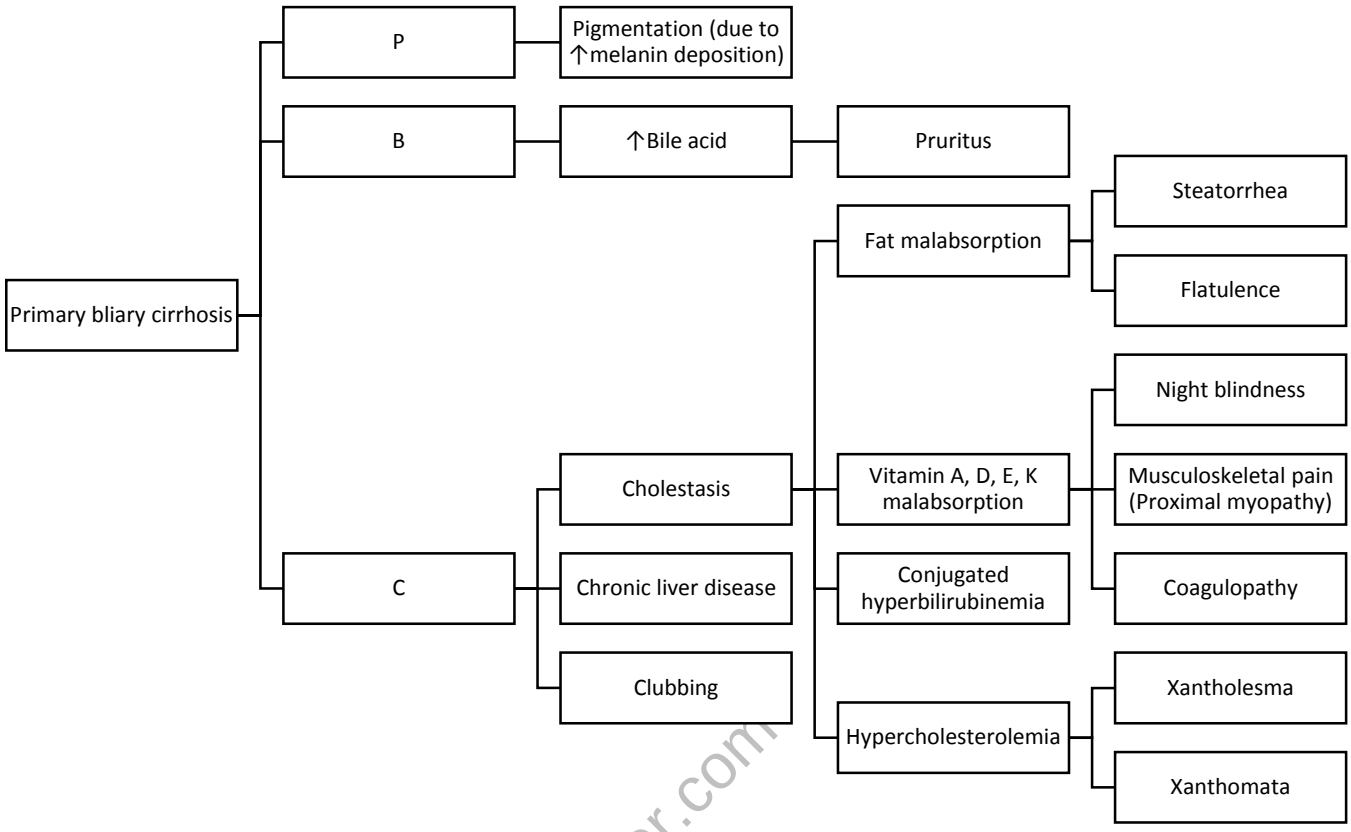
Definition:

It is an antibody mediated disease characterized by inflammation and subsequent fibrotic obliteration of intrahepatic biliary canaliculi.

Pathogenesis:



Clinical features:



Investigations:

1. Blood: Hb, TC, DC, CRP
2. Renal function: Na+ K+ Urea Creatinine
3. Liver function test:
   a. Bilirubin: Conjugated hyperbilirubinemia
   b. AST, ALT: Mild to moderate ↑
   c. Albumin: May be ↓
   d. PT, aPTT, INR: May be ↑ in vitamin K deficiency and chronic liver disease.
4. Detection of serum anti-mitochondrial antibody
5. USG
6. Upper GI endoscopy: If portal hypertension/CLD is suspected; to look for varices
7. Liver biopsy.

Treatment:

1. Cholestasis:
   Urso-deoxy-cholic acid (UDCO):

- ✓ Improves cholestasis
- ✓ Changes toxic bile salts to non-toxic ursodeoxycholate.

2. Pruritus:
   Cholestyramine (Bile acid binding agent)
3. Vitamin supplementation (parenteral, if required)
4. Treatment of chronic liver disease.

Autoimmune hepatitis

Definition:

Autoantibody mediated inflammation of liver which may lead to acute hepatitis as well as chronic liver disease.

Clinical features:

- Common in young females, sometimes associated with other autoimmune conditions like Sjogren's syndrome, autoimmune thyroiditis etc.
- Many patients are asymptomatic till they develop CLD.
- May present with *acute hepatitis ± fulminant hepatic failure*:
  For some unknown reasons, acute hepatitis like illness is triggered off by:
  a. Recent viral illness
  b. Drugs/ toxins
  c. Post-partum period.
- Many of the female patients develop amenorrhea.

Investigations

1. Blood: Hb, TC, DC, CRP
2. Renal function: Na+ K+ Urea Creatinine
3. Liver function test:
   Variable abnormal:
   a. ALT, AST: May be very ↑
   b. Hyperglobulinemia.
4. Autoantibodies:
   a. Type 1 autoimmune hepatitis:
      - ✓ Anti-nuclear antibody (ANA)
      - ✓ Anti-neutrophil cytoplasmic antibody (ANCA)
      - ✓ Anti-soluble liver antibody (ASLA).

b. Type 2 autoimmune hepatitis:
   - ✓ Anti-liver kidney microsomal antibody type 1 (ALKMA1).

5. USG abdomen.

Treatment

1. Acute hepatitis:

   Long term corticosteroid + Azathioprine

   OR

   Mycophenolate mofetil

2. Treatment of chronic liver disease.

Cirrhosis: A comprehensive review for long case discussion

It is a condition characterized:

- Clinically by: Hepatocellular failure + Portal hypertension
- Histopathologically by: Hepatic fibrosis + Necrosis + Regenerative nodules (pseudo-lobules) + Architectural destruction of liver.

Causes:

A. Alcoholic cirrhosis/ Laennec's cirrhosis (macronodular)
Non-alcoholic fatty liver disease (NAFLD)
Alpha1 antitrypsin deficiency
Autoimmune hepatitis
B. Biliary cirrhosis (primary and secondary)
C. Chronic hepatitis (B and C): Micronodular
Cryptogenic cirrhosis
D. Deposition of copper: Wilson's disease
G. Glycogen storage disease
H. Hemochromatosis
I. Indian childhood cirrhosis.

Clinical features:

Features of portal hypertension	***Features of hepatocellular failure***
• Ascites • Variceal bleed	• Hepatic encephalopathy • Icterus

• Splenomegaly • Portosystemic encephalopathy • Superficial venous prominence (direction of filling is away from umbilicus)	• Fetor hepaticus • Ascites • Edema • Miscellaneous*

*Miscellaneous points to be looked for (Hyperestrogenemic features):

- Spider nevus
- Gynecomastia
- Palmar erythema
- Sparse facial/ axillary/ pubic hair
- Testicular atrophy
- Flapping tremor.

Points to be looked for specific underlying etiology:

- Bilateral parotid swelling
- Hepatic facies: Muddy discoloration with hollowing of face due to wasting of temporalis and masseter muscles
- Dupuytren's contracture.

Points suspicious for primary biliary cirrhosis (PBC):

- Clubbing (although it may be present in any cirrhotic patient)
- Pigmentation and scratch marks on skin.

Points suspicious for Wilson's disease:

- Kayser–Fleischer ring
- Involuntary movements.

Palpation of liver:

- May be palpable (left lobe): Due to its firm consistency
- Hepatic span often reduced.

Investigations

Preliminary investigations:

1. Blood: Hb, TC, DC, CRP
   - ✓ Hb: ↓ in GI bleed

- ✓ Pancytopenia: In alcohol induced marrow toxicity/ hypersplenism
- ✓ TC, DC, CRP: ↑ in SBP.

2. Renal function: Na+ K+ Urea Creatinine
   - ✓ Urea creatinine: ↑ in Hepatorenal syndrome/ long term diuretic use
   - ✓ ***Dyselectrolytemia***: Diuretics----→ Dilutional hyponatremia
     Ex.:
     K+↑: Aldosterone antagonist
     K+↓: Loop diuretics.
3. Liver function test:
   - ✓ Bilirubin: ↑, but significant hyperbilirubinemia occurs only in end stage liver disease
   - ✓ AST, ALT: ↑, but often <300 U/L
   - ✓ AST/ALT >1 is suggestive of alcoholic liver disease
   - ✓ GGT, ALP: ↑ is suggestive of intrahepatic cholestasis
   - ✓ PT, aPTT, INR: ↑ is suggestive of synthetic function failure.
4. Arterial NH_3 level:
   ↑ in encephalopathy.
5. Chest X Ray:
   To look for any effusion.
6. USG abdomen:
   Detects liver size, texture; ascites, portal vein diameter, splenomegaly.
7. Upper GI endoscopy:
   To look for any varices.
8. Diagnostic ascitic fluid aspiration:
   - ✓ Serum ascitic fluid gradient >1.1. gm/dL
   - ✓ WBC count >500/µL with PMN fraction >50% is suggestive of SBP.

Treatment of chronic liver disease

1. Treatment of complications:
   A. Ascites:
   - ✓ Dietary Na+ and water restriction
   - ✓ Diuretics (Loop diuretics/ K+ sparing diuretics)
   - ✓ Daily body weight monitoring
   - ✓ Daily intake-output chart
   - ✓ Drainage: therapeutic ascitic fluid aspiration.

B. Bleeding varices:
   - ✓ Circulation maintenance (IV fluid + blood transfusion)
   - ✓ Drugs (Vasopressin)
   - ✓ Endoscopy (Endoscopic rubber band ligation/ sclerotherapy)
   - ✓ Prevention: β-blocker/ nitrate.

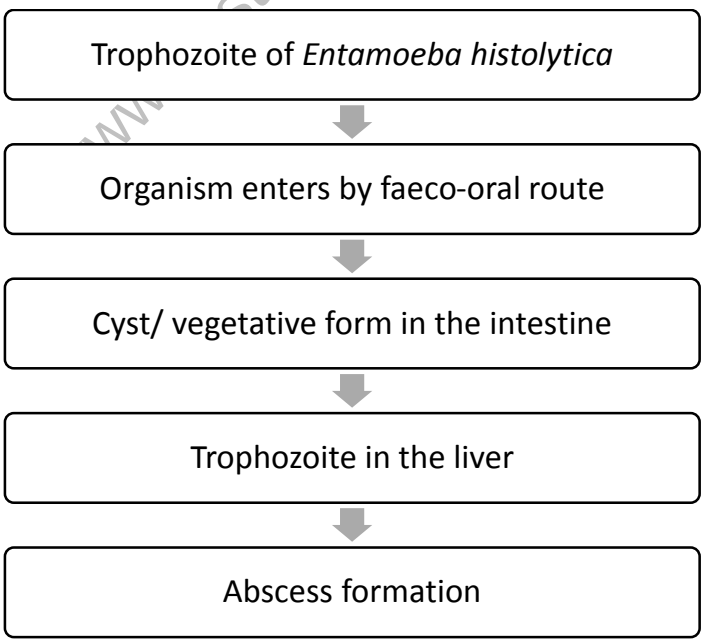
C. Coagulopathy:
   Treat by fresh frozen plasma in case of active bleeding.

E. Encephalopathy:
   - **a.** Avoid **a**lcohol/ hepatotoxic drugs
   - **b.** Urgently treat any associated GI **b**leed
   - **c.** Prevent/ treat **c**onstipation
   - **d.** **D**ietary protein restriction
   - **e.** Treat any **e**lectrolyte imbalance
   - **f.** **F**luid: Maintain proper hydration
   - **g.** **G**ut cleansing agent (Rifaximine)
   - **h.** Prevent **h**ypovolemia
   - **i.** Treat any associated **i**nfection.

Miscellaneous Topics

Amoebic liver abscess

Pathogenesis:



Clinical features:

1. Constitutional symptoms:
   a. Fever
   b. Loss of appetite
   c. Weight loss
   d. Anorexia
   e. Nausea
2. RUQ discomfort.

On examination:

1. RUQ tenderness: On percussion, tenderness over right lower intercostal spaces noted
2. Hepatomegaly may be present.

Investigation

1. Blood: Hb, TC, DC, ESR
2. Liver function test: Nonspecifically mildly deranged
3. USG abdomen: Usually shows ***a single abscess***.

Complications:

3P

P1: Pleural empyema

P2: Pericardial empyema

P3: Peritonitis.

Pyogenic liver abscess

Portal of entry:

1. Through portal vein: Pylephlebitis (infection of portal vein)
2. Through CBD: Ascending cholangitis
3. Through hepatic artery: Bacteremia.

Clinical features:

1. Constitutional symptoms:
   a. High temperature

   b. Weakness
   c. Malaise
2. Anorexia + Nausea
3. RUQ discomfort
4. RUQ tenderness ± Hepatomegaly.

Investigation

1. Blood: Hb, TC, DC, CRP
2. Renal function: Na+ K+ Urea Creatinine
3. Blood culture and sensitivity
4. Liver function test: Variably abnormal
5. USG: It shows ***multiple small abscesses***.

Treatment

- Empirically IV Ceftriaxone + Metronidazole
- To be changed to appropriate oral forms once patient starts to improve
- Total duration: Approx. 3 weeks.

Hepatocellular carcinoma (HCC)

It is a primary malignancy of liver.

Risk factors:

1. Chronic hepatitis (HBV and HCV)
2. Cirrhosis of any etiology.

Clinical features:

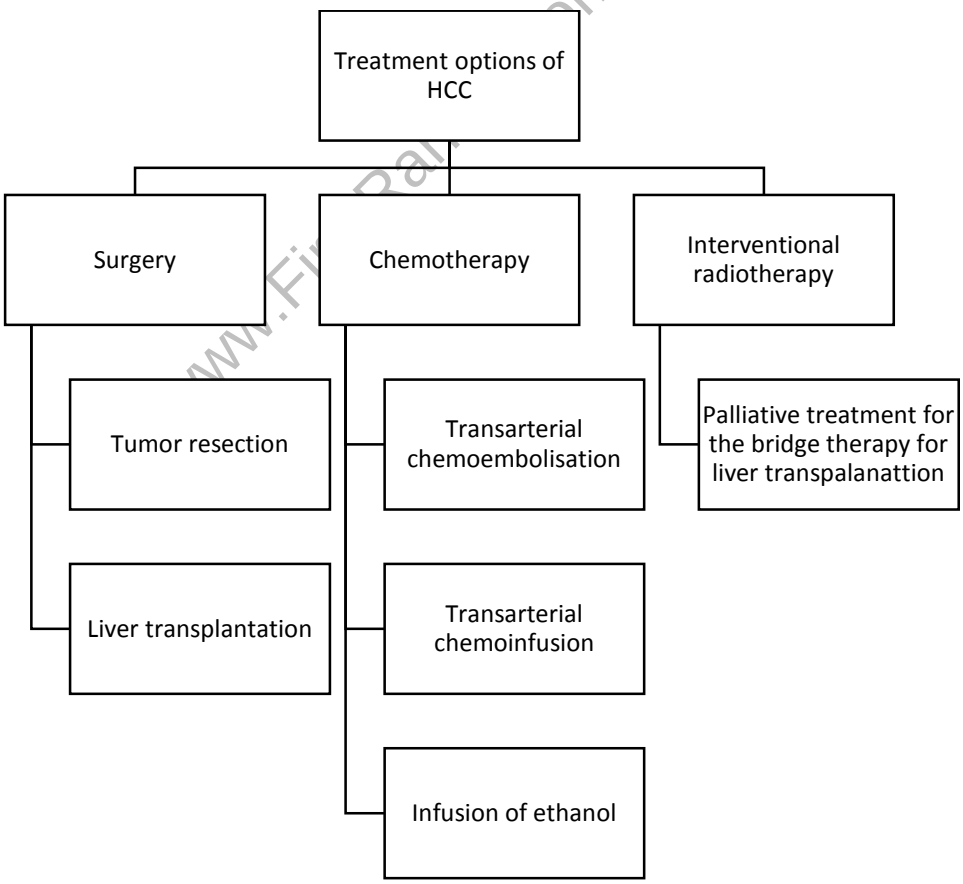
1. Constitutional symptoms:
   Weight loss
   Loss of appetite
   Fever
2. Sudden deterioration of a previously stable cirrhotic patient
   Ex.: Worsening of ascites
3. Patient may/ may not be aware of chronic hepatitis
4. Icterus, Hepatomegaly: +Ve, Evidences of CLD may be present.

Investigation

1. Blood: Hb, TC, DC, ESR
   Hb% may rise in a tumor producing erythropoietin (EPO)
   Leukocytosis is common due to underlying inflammatory condition.

2. Renal function: Na+ K+ Urea Creatinine
3. Liver function test:
   Non-specifically abnormal
   But ALP and bilirubin may rise significantly.
4. ***Serum AFP*** (α-fetoprotein): Significantly high
   Value of >200 U/L is quite specific for HCC. But it is not sensitive.
   Mild to moderate increase in value is not specific.
5. USG abdomen: Visualizes the lesion
6. CECT/ MRI
7. Biopsy of the lesion:
   Carries risk of massive bleeding as it is a highly vascular tumor.

Treatment



Budd Chiari syndrome

Thrombosis of the hepatic vein.

Causes:

1. Hypercoagulable state/ disorders:
   - ✓ Anti-phospholipid antibody syndrome
   - ✓ Nephrotic syndrome
   - ✓ Protein C/ protein S/ antithrombin 3 deficiency
   - ✓ Factor 5 Leyden.
2. Idiopathic
3. Tumor occlusion/ invasion:
   Common tumors responsible: RCC, HCC.

Clinical features:

1. Sudden RUQ pain
2. Tender soft hepatomegaly
3. Patient may develop fulminant hepatic failure
4. Ascites.

Investigation

1. USG abdomen with Doppler study
2. CECT abdomen
3. Coagulation studies.

Treatment

Anticoagulation.

Wernicke Korsakoff Syndrome

It is a neurodegenerative complication of vitamin B1 deficiency, most commonly seen in chronic alcohol abusers.

Pathogenesis:

Degeneration of different areas of brain: mammillary body/ thalamus/ median temporal lobe/ cerebellum. ***Chronic alcohol abuse interferes with absorption and metabolism of thiamine, often leading to severe thiamine deficiency.***

Clinical features:

Wernicke Korsakoff syndrome has two components:

1. Wernicke's encephalopathy
2. Korsakoff psychosis.

Clinical features of Wernicke's encephalopathy:

1. Ataxia
2. External ophthalmoplegia: Commonly lateral rectus palsy + Nystagmus
3. Psychiatric disturbances:
   Behavior/ personality disturbances/ dementia.
   - Usually these symptoms are reversible with thiamine replacement therapy.

Clinical features of Korsakoff's psychosis:

1. ***Amnesia***:
   It may be of 2 types:
   a. Anterograde amnesia: Inability to create new memories + impaired recent memory + no remote memory loss.
   b. Retrograde amnesia: Remote memory loss + new memory intact.
2. ***Confabulation***:
   Incorrect memory to which the patient holds onto/ on the basis of which patient may act.
3. Patient may have features of Wernicke's encephalopathy.

Investigation

1. CT/ MRI of brain
2. Vitamin B1 estimation.

Treatment

1. Prevention:
   I. In patients with H/O significant alcohol abuse, if requires high dose dextrose containing fluid, ***must receive high dose thiamine before/ at least along with the fluid*** (glucose oxidation is a thiamine consuming process and therefore, may unmask any underlying deficiency and may precipitate Wernicke Korsakoff syndrome).
   II. Any alcohol excess patient should be on a long term thiamine replacement therapy.

2. Established cases:
   High dose thiamine replacement.

Renson's criteria/ score

It is a predictive score which can reasonably predict prognosis, development of complication(s) in a patient of acute pancreatitis.

Renson's criteria	
On admission	***Within first 48 hours***
1. Age >55 years 2. WBC count > 60000/cu.mm 3. Blood glucose >200 mg/dl 4. AST >250 U/L 5. Serum LDH >350 U/L	1. Arterial PO_2 < 60 mm Hg. 2. ↑ in BUN value > 5 mg/dL (Normal:80) Note that, (BUN value/2) = Urea value. 3. Base deficit >4 mEq/L 4. Serum Ca++ <8 mg/dL 5. ↓ in hematocrit: >10% 6. Estimated fluid deficit >6L
Presence of ≥3 of the above predicts a complicated course.	Presence of ≥1 of the above predicts worst prognosis.

Child-Pugh Score for chronic liver disease/ cirrhosis

Points Criteria	***1***	***2***	***3***
Ascites	None	Medically controlled	Poorly controlled
Encephalopathy	None	Medically controlled	Poorly controlled
Albumin (gm/dL)	>3.5	2.8-3.5	<2.8
Bilirubin (mg/dL)	<2	2-3	>3
↑PT (sec.)	1-3	4-6	>6
Total score	5-6	7-9	10-15
Prognosis	Good	Bad	Worse

Stomach

Gastro-esophageal reflux disease (GERD)

It is a condition characterized by reflux of gastric acid content into the esophagus.

Pathophysiology:

The following factors play important roles:

1. Lower esophageal sphincter (LES) dysfunction
2. Irritant effect of refluxed acid
3. Hiatus hernia
4. Delayed esophageal emptying.

Clinical features:

1. Heartburn:
   Typically aggravates on:
   a. Lying flat after meals
   b. After alcohol ingestion.
2. A bitter/ sour test in the mouth due to regurgitation of gastric contents.
3. Atypical/ extra-esophageal manifestation:
   a. Chronic cough
   b. Chronic laryngitis
   c. Hoarseness of voce
   d. Non-cardiac chest pain.

Investigation

1. Upper GI endoscopy:
   Typically shows: reflux esophagitis.
2. Esophageal manometry with pH estimation.

Treatment

1. Lifestyle modification:
   I. Stay upright for approx. half an hour after each meal
   II. To raise the head end of the bed during night
   III. Avoid alcohol after recovery.
2. Medications:
   H_2 blockers/ PPI.

Complication: Barrett's esophagus.

Barrett's esophagus

It is condition where esophageal squamous epithelium is replaced by metaplastic columnar epithelium.

Risk factor:

Long standing GERD

Clinical features:

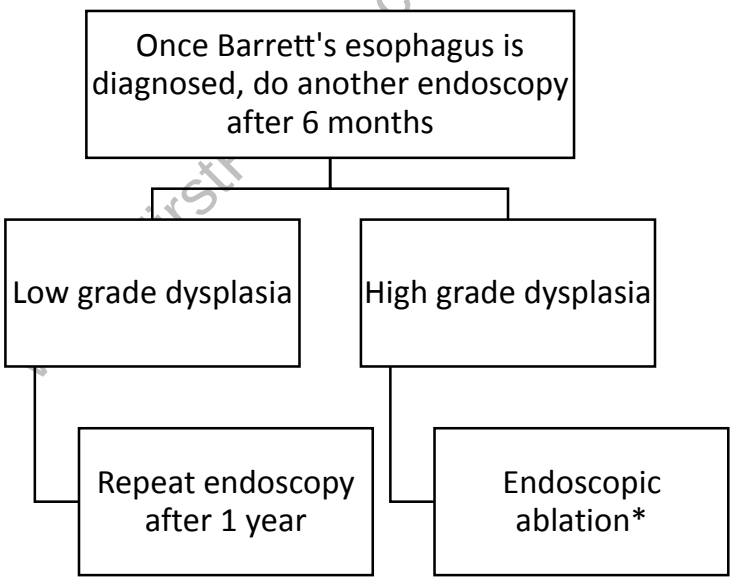
1. Asymptomatic
2. Symptoms of GERD: Present.

Investigation

1. Upper GI endoscopy:
   Shows orange, velvety gastric type epithelium in the esophagus
2. Confirmation of diagnosis by mucosal biopsy.

Treatment

1. Medications to treat GERD
2. Endoscopic surveillance:



*Resection of metaplastic mucosal nodule is done by snare dissection to prevent submucosal invasion.

Complication:

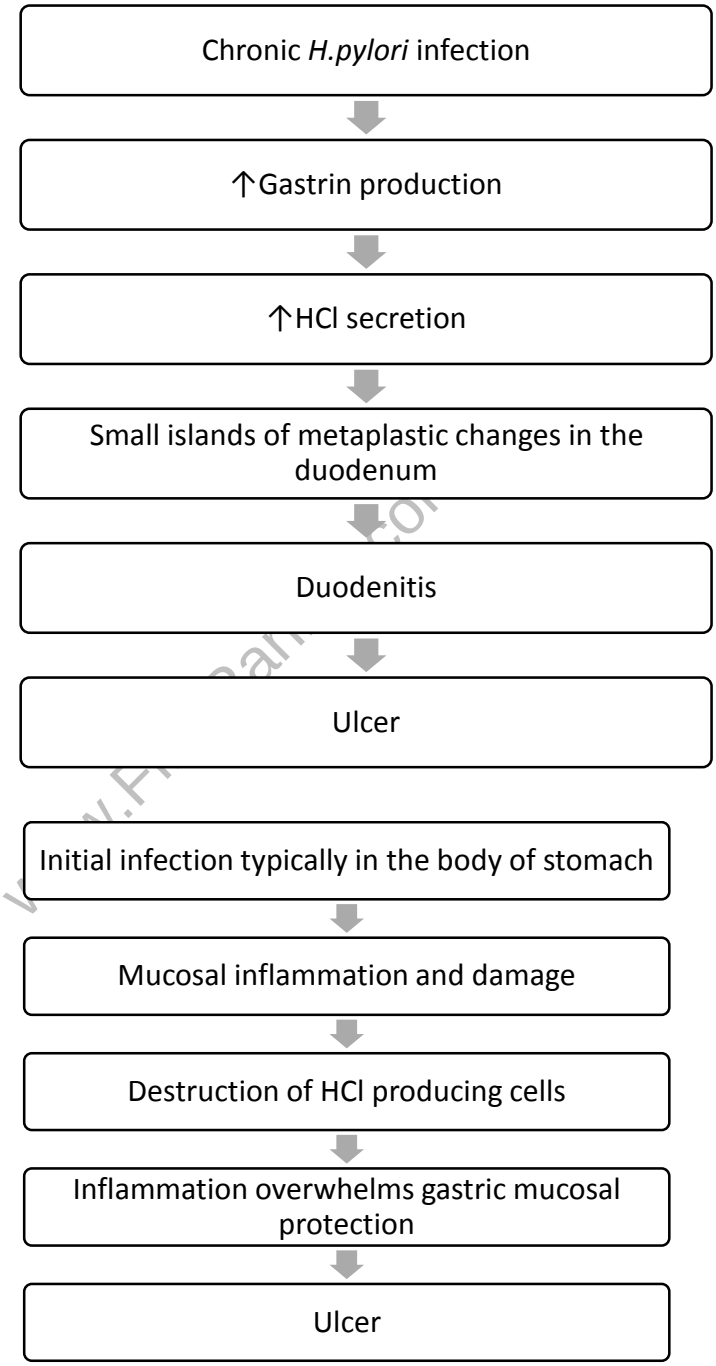
Esophageal adenocarcinoma.

Peptic ulcer disease

Breech in the continuity of gastric +/ duodenal mucosa.

Risk factors:

1. Chronic *H.pylori* infection:
   In duodenal ulcer, initial *H.pylori* infection typically occurs at the junction of body and antrum.



2. Drugs:
   NSAIDs
   Steroids
3. Stress ulcer: Can occur in any critically ill patients.
4. Malignant ulcer.

Clinical features

Pain abdomen:

- Typically occurs in epigastrium
- Variable duration
- Often occurs periodically
- Nature: Dull aching pain
- Aggravating and relieving factors:

Type	***Aggravating factor***	***Relieving factor***
Gastric ulcer	After food intake	Empty stomach
Duodenal ulcer	Empty stomach	Food/ meal

- Radiation to the back should raise the suspicion of:
  a. Perforation
  b. Pancreatitis.
- Associated symptoms:
  a. Heartburn/ indigestion
  b. Loss of appetite: Often patient is afraid of eating
  c. Hunger pain
  d. Nocturnal pain (in duodenal ulcer)
  e. *Water brush*: Regurgitation of bitter/ sour contents with sudden salivation
  f. Features usually absent in benign ulcer:
     I. Significant vomiting
     II. Unintentional weight loss.

GI bleed:

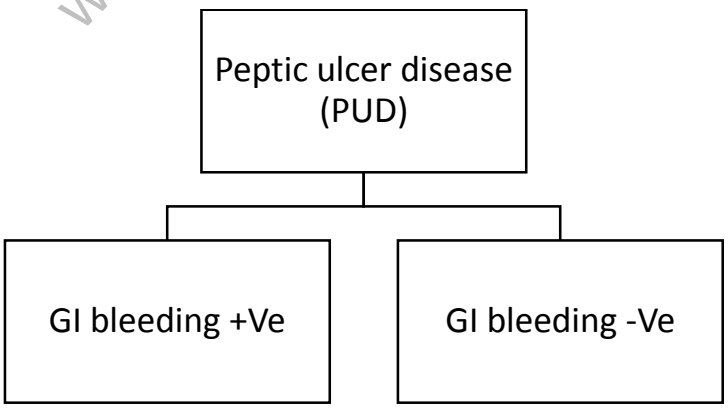
- Hematemesis ± Melena
- Bleeding may be occult
- There may be no obvious blood loss or blood loss may be massive enough to make the patient's hemodynamics unstable
- A systolic blood pressure (SBP) of <100 mm Hg indicates significant bleeding, whereas a SBP of ≥100 mm Hg indicates that the bleeding is not such severe.

- In hematemesis, the color of blood may be fresh/ altered depending upon the time elapsed between bleeding and vomiting.
- PR examination reveals melena in stool.

Investigations

1. Blood: Hb, TC, DC, CRP: If there is obvious H/O bleeding, clinically suspect for iron deficiency anemia and do the following tests:
   a. MCV
   b. Serum iron
   c. Serum ferritin.
2. Renal function: Na+ K+ Urea Creatinine
3. Lipase ± Amylase: To rule out acute pancreatitis
4. Liver function test
5. Clotting profile: Platelet count, BT, CT, PT, aPTT, INR
6. Faecal occult blood test (FOBT)
7. Upper GI endoscopy:
   a. Confirms presence of ulcer
   b. Biopsy confirms histopathological diagnosis.
8. Diagnosis of *H.pylori* infection:
   a. Endoscopically:
      I. Rapid urease test
   b. Non-endoscopically:
      I. Urea breath test
      II. H.pylori fecal antigen
      III. Blood serology.

Treatment



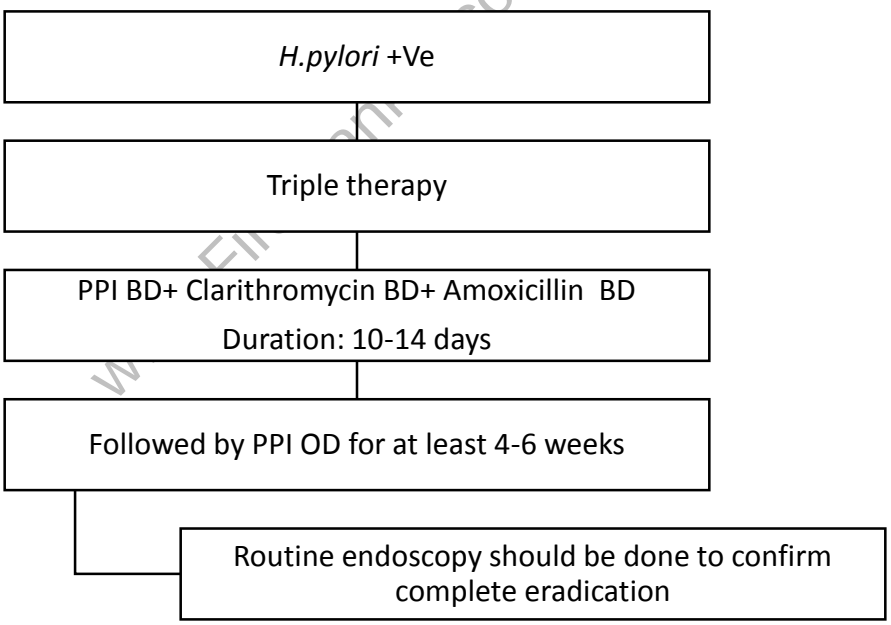
Peptic ulcer disease with significant GI bleed

Short term treatment

A. Airway: To be protected particularly if there is risk of aspiration
B. Breathing: Oxygen
   Bowel rest: Nil by mouth till bleeding is under control, nasogastric suction
C. Circulation:
   I. One wide bore cannula in each hand
   II. IV fluid resuscitation: Ringer lactate/ Normal saline
   III. Blood transfusion.
D. Drugs:
   Infusion of PPI: Pantoprazole for at least 48 hours
E. Endoscopy:
   I. Urgent endoscopy with rapid urease test to confirm the diagnosis as well as endoscopic interventions
   II. Administration of adrenaline.

Long term treatment

A.



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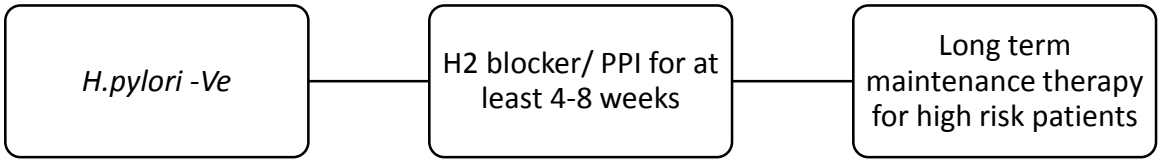
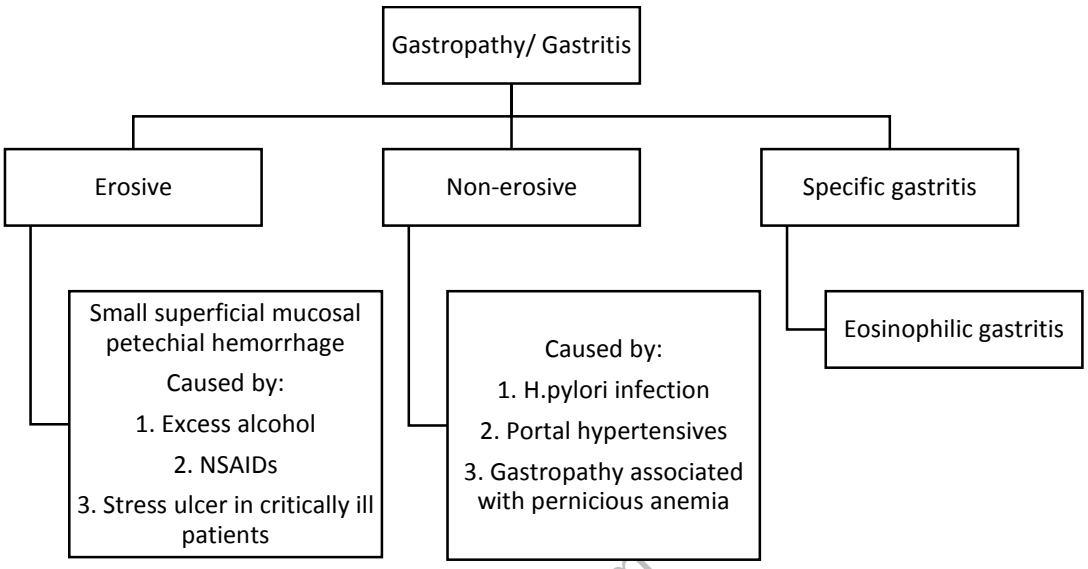


Gastropathy/ Gastritis

Damage to the mucosal epithelium of the stomach which may/ may not be accompanied by inflammation if mucosa (gastritis).

Types:



Clinical features:

- H/O intake of offending drugs/ alcohol may be present
- Epigastric pain
- Heartburn
- GI bleeding: Hematemesis ± Melena (if present at all, usually insignificant in amount).

Investigation:

Upper GI endoscopy with rapid urease test + Biopsy

Treatment:

1. Prevention:
    I. H_2 blocker/ PPI
    II. Sucralfate.
2. Established cases:
    I. Acute period: IV PPI infusion
    II. Long term treatment: H_2 blocker/ PPI
    III. *H.pylori* eradication.

Malabsorption syndrome

A group of disorders characterized by impaired absorption of different food particles and nutrients.

Examples:

I. Celiac disease
II. Tropical sprue
III. Short bowel syndrome (post-resection):
   - ✓ Terminal ileum
   - ✓ Extensive small intestinal resection
IV. Bacterial overgrowth syndrome
V. Protein losing enteropathy.

Mechanism of clinical manifestations and clinical features:

	Substances malabsorbed		***Clinical features***
A	Albumin		Swelling (anasarca)
B	Bile acids	Fat	Steatorrhea/ flatulence
		Vitamin A	Night blindness
		Vitamin D	Osteomalacia
		Vitamin E	CNS manifestations
		Vitamin K	Coagulopathy
	Vitamin B12		Anemia ± Neurological complications
C	Ca++		Muscle spasm, Tetany, Paresthesia (perioral)
D	Diet (Protein + Carbohydrate + Fat)		Weight loss and weakness
	Vitamin D		Proximal myopathy Musculoskeletal pain Bony deformity
E	Vitamin E		Neuropathy Ataxia
F	Fe++		Iron deficiency anemia
	Fluid		Watery diarrhea

Investigations:

1. ***Blood***:
   Hb, TC, DC, CRP, MCV

Hb: ↓ (in Iron deficiency/ vitamin B12 deficiency)
MCV: ↓/↑
Dimorphic film.

2. ***Iron studies***:
   Serum iron
   Serum ferritin
   Serum transferrin saturation.
   * Note that: In any acute inflammation, serum ferritin may be high as it is an acute phase reactant protein.
3. ***Ion studies***:
   Na+
   K+
   Ca++
   Mg++
4. Urea creatinine
5. Clotting profile
6. Serum vitamin B12 level + Folate level
7. Serum albumin.

Special tests:

1. Fecal fat content: ↑
2. Blood serology:
   In celiac disease, 2 antibodies are often found +Ve:
   I. IgA: Anti-endomysial antibody
   II. IgA: Tissue transglutaminase antibody.
3. Intestinal mucosal biopsy: Often confirm the diagnosis in some of the cases.

Treatment:

General treatment

1. Nutritional support
2. Supplementation of vitamins and minerals

Specific treatment

1. Celiac disease:
   As this disease is caused by immunologically mediated hypersensitivity to dietary gluten which ultimately causes malabsorption, the specific treatment consists of ***gluten free diet*** (foods containing gluten: wheat, rye, berly)

2. Bacterial overgrowth: Antibiotics.

Inflammatory bowel disease (IBD)

It is a condition characterized by widespread inflammatory damage to different parts of small and large intestine.

The pattern of inflammation and subsequent clinical features lead to 2 distinct clinical entities:

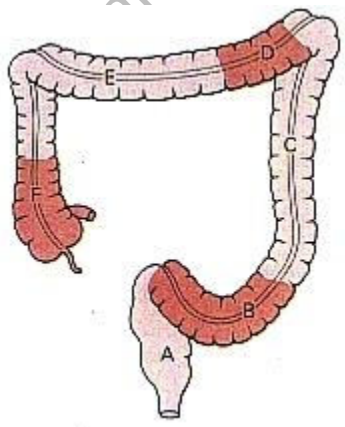
1. Crohn's disease
2. Ulcerative colitis.

Crohn's disease

Characterized by relapsing and remitting segmental/ patchy inflammation of intestine.

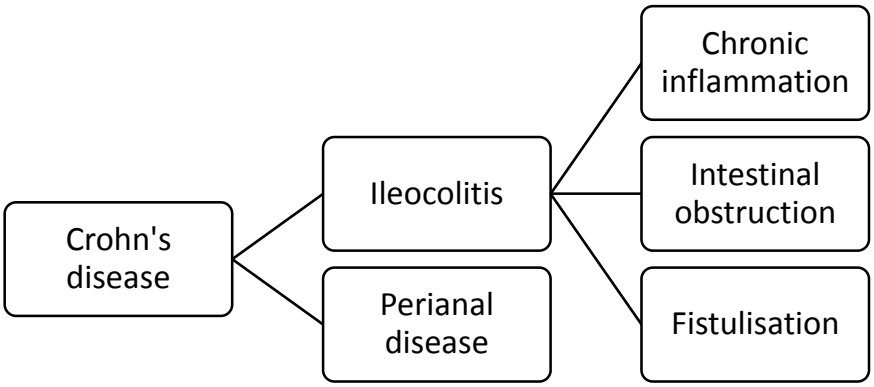
Sites (according to descending order of frequency):

1. Terminal ileum and proximal ascending colon: Ileocolitis
2. Terminal ileum: Ileitis
3. Large gut: Colitis
   However, involvement of sigmoid colon and rectum is extremely rare and this variety is usually associated with perianal manifestations.



Clinical features:

Mechanism of clinical manifestations:



Clinical features of ileocolitis:

1. ***Due to chronic inflammation***:
   I. Pain abdomen: Often in right iliac fossa
   II. Diarrhea: Usually watery, rarely bloody
   III. Fever, loss of appetite and weight loss
   IV. On palpation, tender mass may be present (matted intestine + lymph nodes + mesentery)
   V. Malabsorption syndrome.
2. ***Due to intestinal obstruction*** (fibrostenotic occlusion of intestine):
   I. Constipation
   II. Distension
   III. Vomiting.
3. ***Due to fistulisation***:
   I. Enterocolic fistula: Malabsorption
   II. Enterovesicle fistula: Feculent urine
   III. Enterovaginal fistula: Feculent per-vaginal discharge
   IV. Enteromesenteric fistula: Intra-abdominal abscess.

Clinical features of perianal disease:

1. Perianal fistula
2. Perianal abscess
3. Visible anal skin tag
4. Anal fissure.

- The disease classically relapses and remits.

Investigation

1. Blood: Hb, TC, DC, CRP/ESR

Hb: ↓ due to:
a. Chronic inflammation
b. Iron deficiency
c. B_{12} deficiency.

2. Renal function: Na+ K+ Urea Creatinine
3. If malabsorption is suspected:
   Serum Ca++, Vitamin B_{12}, Vitamin D, Iron studies.
   Stool:
   Look for ova/ parasites/ cysts.
4. Colonoscopy:
   It will show typical *mucosal inflammation/ ulceration with skip areas*
   Colonoscopic biopsy will confirm the diagnosis.
5. Barium follow through:
   Will delineate any filling defect/ any anatomical deformity.
   It is done particularly when small bowel involvement is suspected and colonoscopy is inconclusive.
6. Capsule endoscopy to visualize small gut
7. CECT abdomen: To visualize the internal structure for any obvious anatomical deviation.

Treatment

General treatment:

1. IV fluid in severe diarrhea
2. Ensure adequate fluid intake
3. Analgesic-antispasmodic:
   - ✓ Drotaverine
   - ✓ Dicyclomine.
4. Antidiarrheal:
   Loperamide (May cause paralytic ileus).

Specific treatment:

1. ***5-Amino-salicylic acid*** (5-ASA):
   Mesalamine:
   Topically acting agent typically acting on ileum.
   It is no longer the first drug of choice in Crohn's disease and can be used along with corticosteroid.

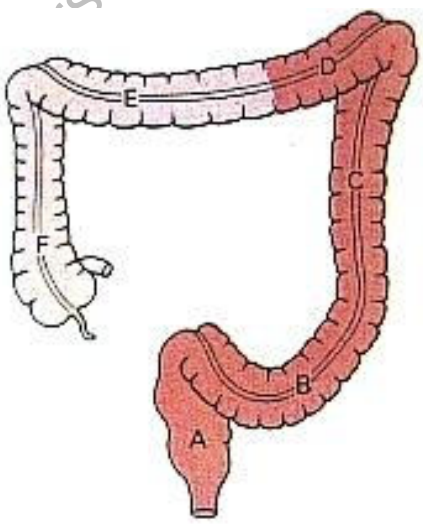
2. ***Corticosteroid***:
   - Systemic corticosteroids: Prednisolone/ Methyl-prednisolone
   - DOC in most CD patients to achieve remission
   - Often patients require long term therapy to maintain remission
   - Ileal release formulation of Budesonide can be used: it is less toxic and less effective.
3. ***Immunomodulators***:
   Used in moderate to severe disease:
   - ✓ Azathioprine
   - ✓ Methotrexate
   - ✓ Cyclophosphamide.
4. ***Biological agents***:
   - ✓ Infliximab
   - ✓ Natalizumab.

Ulcerative colitis

Characterized by relapsing and remitting inflammation of the colon which is continuous.

Common sites:

1. Sigmoid colon + rectum: Proctosigmoiditis
2. Left side of colon: Left sided colitis
3. Pancolitis.

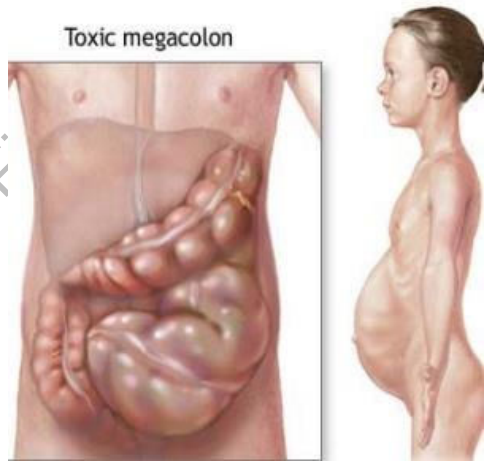


Clinical features:

Mechanism of clinical manifestations:

1. Due to chronic inflammation
2. Due to toxic megacolon
3. Extra-intestinal manifestations.

1. ***Clinical features due to chronic inflammation***:
   I. Bloody diarrhea
   II. Hemodynamic instability ± Pallor
   III. Abdominal pain: Left upper/ lower quadrant: Continuous/ spasmodic
   IV. Constitutional symptoms: Fever, weight loss, loss of appetite etc.
2. ***Clinical features due to toxic megacolon***:
   Toxic megacolon is defined as acute colonic dilatation leading to adynamic/ paralytic ileus with signs of systemic toxicity:
   I. Abdominal pain ± Distension
   II. Abdominal tenderness
   III. Sluggish bowel sound
   IV. Toxicity: Fever, tachycardia, flushing.



3. ***Clinical features due to extra-intestinal manifestations***:
   I. Ocular: Uveitis + Episcleritis
   II. Lungs: Interstitial lung disease
   III. Heart: Non-infective endocarditis
   IV. Abdomen: Gall stone (more common in CD)
   V. Liver: Primary sclerosing cholangitis

VI. Kidney: Nephrolithiasis (more common in CD)
VII. Skin:
- ✓ Erythema nodosum
- ✓ Pyoderma gangrinosum.

VIII. Joints:
- ✓ Polyarthritis
- ✓ Ankylosing spondylitis.

Investigation

1. Blood: Hb, TC, DC, CRP/ESR
   Hb: ↓ due to blood loss
   TC, DC, CRP, ESR: ↑ due to inflammation/ intra-abdominal infection.
2. Renal function: Na+ K+ Urea Creatinine
   To look for dehydration.
3. Serum albumin level
4. Stool:
   - Look for ovum/ parasite/ cyst.
   - Gram stain, culture and sensitivity.
5. ***Straight X Ray abdomen***:
   To look for toxic megacolon (diagnosed when colonic diameter is >6 cm.)
6. Sigmoidoscopy:
   Will visualize inflamed, ulcerated mucosa and biopsy confirms the diagnosis
7. Colonoscopy:
   Required particularly if sigmoidoscopy is inconclusive. However, best avoided during acute flare up as it may cause perforation.
8. CECT abdomen:
   To look for any structural abnormality.

Treatment

Treatment of Ulcerative colitis can be divided in 3 groups for simplification:

1. Supportive treatment
2. Specific treatment
3. Surgical treatment.

Supportive treatment

A. Absolute bed rest (if needed)
B. Bowel rest (nil by mouth till acute phase is over)
C. Circulation (IV fluid, blood transfusion if required)
D. Drugs:
   I. Analgesic-antispasmodic:
      Drotaverine
      Hyoscine butyl bromide.
   II. Antibiotic (Particularly if toxic megacolon is suspected):
      Ceftriaxone/ Ciprofloxacin + Metronidazole
E. Eliminate toxic megacolon:
   I. Nil per mouth
   II. IV antibiotic
   III. Colonic decompression by:
      - ✓ Flatus tube
      - ✓ Colonoscopic decompression.

Specific treatment

I. 5ASA compounds:
   - Usually 1st line therapy in mild disease
   - Act topically
   - Agents commonly used:
      - ✓ Mesalamine: More effective in CD
      - ✓ Osalazine: PO/PR enema
      - ✓ Balsalazine: PO/PR enema
      - ✓ Sulfasalazine: PO.

II. Corticosteroids:
   - Indications: Moderate to severe ulcerative colitis
   - Agents commonly used:
      - ✓ Prednisolone: PO/IV
      - ✓ Methyl-prednisolone: PO/IV
      - ✓ Hydrocortisone: Enema/ foam.
   - Many patients require long term steroid. These patients should also be prescribed the following drugs:
      - ✓ H_2 blockers/ PPI: For gastric protection
      - ✓ Bisphosphonate: For bone protection.

III. Immunomodulators:
Cyclosporine.

IV. Biological agents:
- Infliximab
- Adalimumab.

Surgery

- Surgery is done in medically refractory cases
- Surgery of choice is hemicolectomy.

Extras of ulcerative colitis

Severity of ulcerative colitis

Criteria	*Mild*	*Moderate*	*Severe*
Clinical criteria:			
Number of stools/day	<4	4-6	>6
Weight loss (% of body weight)	None	1-10%	>10%
Fever	None	99-100 ^{o}F	>100 ^{o}F
Laboratory criteria:			
Hematocrit (PCV)	Normal	30-40%	<30%
ESR	Normal	20-30 mm/hr	>30 mm/hr
Albumin (gm/dL)	Normal	3-3.5	<3

Difference between Crohn's disease and Ulcerative colitis

Points	*Crohn's disease*	*Ulcerative colitis*
Pattern of inflammation	Patchy	Continuous
Site	Ileocolitis, ileitis, colitis	Proctosigmoiditis, left sided colitis, Pancolitis
Relation to smoking	Common in smokers	Rare in smokers
Diarrhea	Not bloody	Bloody
Malabsorption	+	-
Fistulisation	+	-
Perianal disease	+	-
Intestinal obstruction	+	-
Cholelithiasis	+	-
Nephrolithiasis	+	-

Toxic megacolon	-	+
Primary sclerosing cholangitis	-	+

Irritable bowel syndrome (IBS)

Characterized by widespread GI manifestations in absence of any biochemical/ structural abnormality.

Pathogenesis:

Usually the following 4 factors play important role:

1. Intestinal dysmotility
2. Intestinal hypersensitivity: Resulting in reduced pain threshold
3. Enteric infection/ altered gut flora
4. Psychological factors.

Clinical features:

1. Lower abdominal pain:
   Often continuous/ may be spasmodic in nature.
   Typically associated with >1 of the following:
   I. Relieved after defecation
   II. Change in the form/ appearance/ consistency of stool (hard/ semisolid/ liquid)
   III. Change in stool frequency
   IV. Constipation/ diarrhea.
2. Other abdominal symptoms:
   I. Mucoid stool
   II. Urgency/ frequency
   III. Tenesmus: Sense of incomplete evacuation.
3. Extra-intestinal manifestations:
   I. Chest pain, palpitation
   II. Pain at different sites of the body.
4. ***Symptoms usually absent***:
   I. Nocturnal diarrhea
   II. Weight loss
   III. Per-rectal bleeding
   IV. Alternate constipation and diarrhea.

Investigation

Although IBS can be diagnosed with confidence from history only, if there is some confusion, organic diseases must be ruled out using the following investigations:

1. Blood: Hb, TC, DC, CRP
2. Serum albumin, serum Ca++, iron studies
3. Stool:
   a. Examine for ovum parasite cyst (OPC)
   b. Gram stain, culture and sensitivity
   c. Faecal calprotectin: If ↑: indicates inflammatory diarrhea.
4. Colonoscopy: To rule out any structural lesion.

Treatment

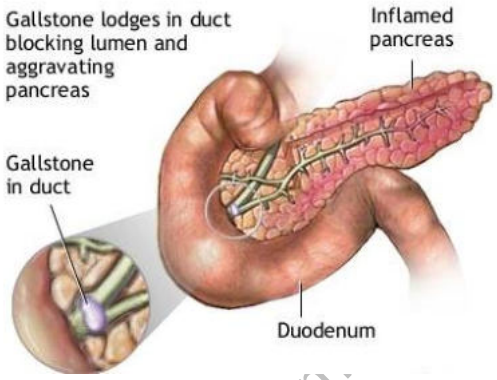
1. *Appropriate dietary modification*
2. *Antispasmodic*:
   a. Drotaverine
   b. Hyoscine butyl bromide.
3. *Anti-diarrheal*:
   Loperamide
4. *Anti-constipation agent* (Laxatives):
   a. Magnesium salts
   b. Poly-ethylene-glycol.
5. ***Anti-depressants***:
   Mechanism of action:
   a. Centrally acting pain inhibitors
   b. Anti-cholinergic effect.
   Commonly used agents are:
   a. Amitriptyline
   b. Imipramine
   c. Fluoxetine
   d. Paroxetine.
6. *Probiotics*:
   Lactobacillus spore
7. Psychological counselling.

Acute pancreatitis

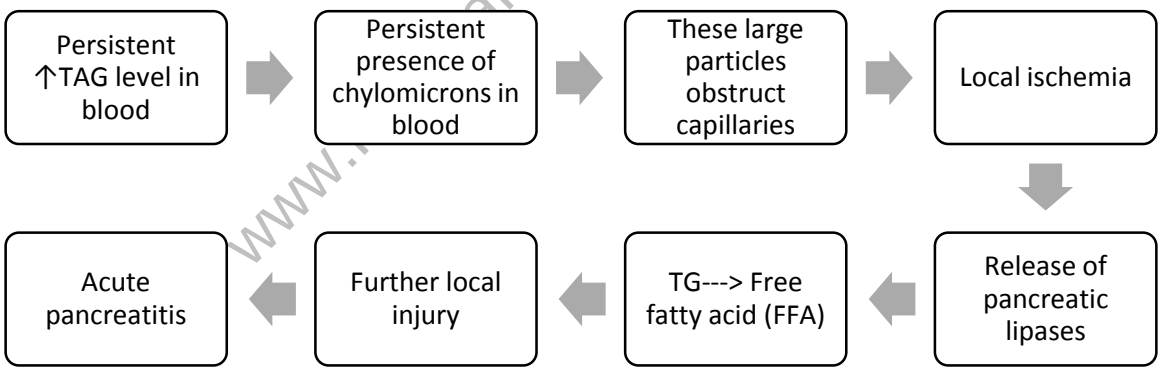
Acute inflammation of pancreas.

Causes:

1. Alcoholic pancreatitis
2. Biliary/ gallstone pancreatitis:
   Usually due to a slipped/ passed stone causing acute inflammation of pancreas.
   Many cases of so called idiopathic acute pancreatitis are thought to be caused by ***microlithiasis***.

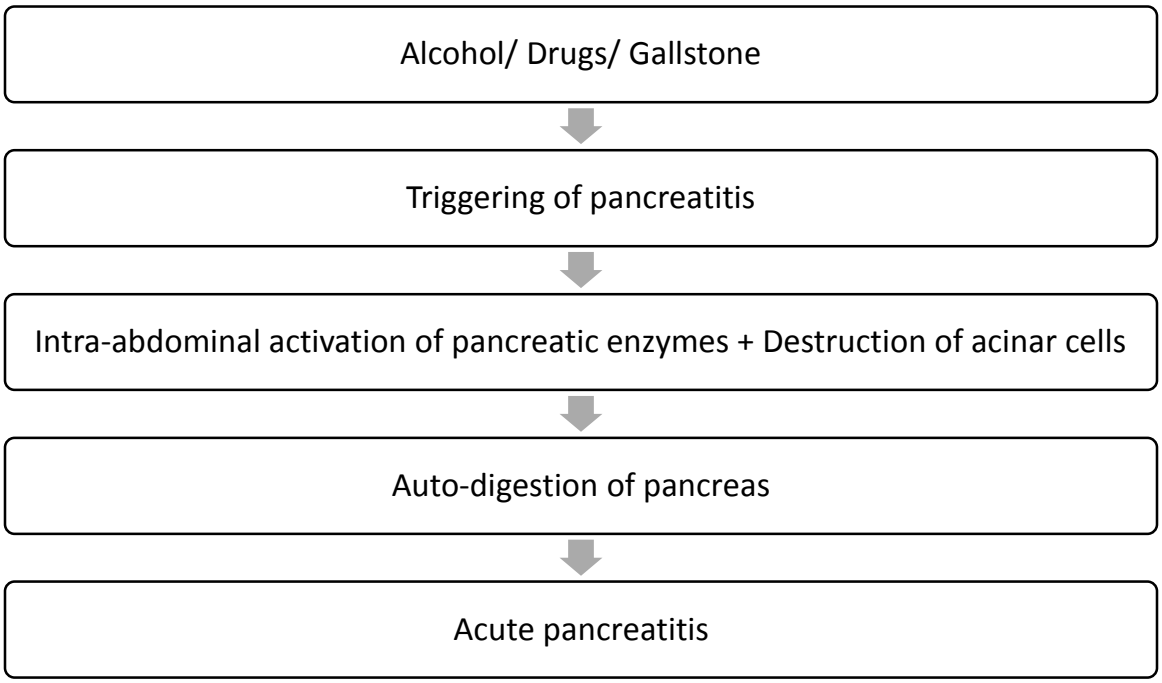


3. Cystic fibrosis, connective tissue disorders and rarely, CA head of pancreas
4. Dyslipidemia:
   Elevated levels of triglyceride is an important risk factor of acute pancreatitis.



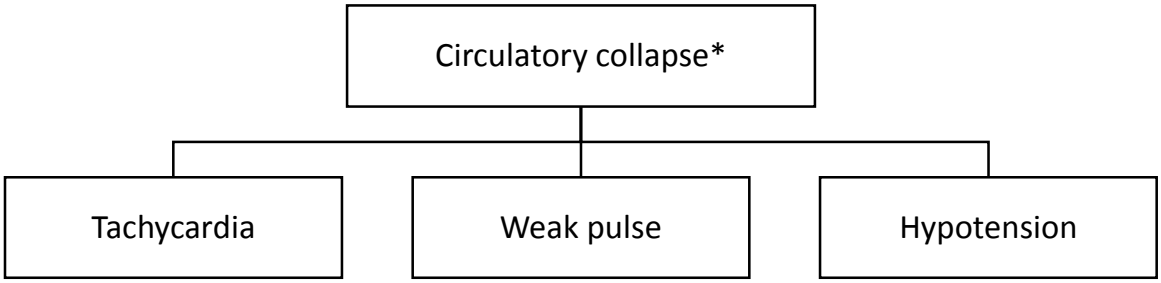
   Drugs inducing dyslipidemia:
   - ✓ Corticosteroid
   - ✓ Azathioprine
   - ✓ Valproate.
5. Elevated serum Ca++ level
6. Iatrogenic causes:
   ERCP induced pancreatitis is an important iatrogenic cause.

Pathogenesis:



Signs and symptoms:

1. Pain abdomen:
   - ✓ Onset: Sudden
   - ✓ Site: Epigastrium
   - ✓ Character: Continuous burning pain, which may be excruciating
   - ✓ Radiation: Towards the back (irritation of splanchnic nerves)
   - ✓ Aggravates on: Supine position
   - ✓ Partly relieved by: Stooping forwards with trunk flexed and knee drawn up
2. Severe nausea ± retching* ± vomiting
   * Reverse peristaltic movement of stomach and esophagus without vomiting.
3. Circulatory disturbances:



*It is caused by intra/retro-peritoneal sequestration of large amount of fluid, also termed as "3rd space fluid loss".

4. Respiratory signs and symptoms:
    a. Acute respiratory distress syndrome (ARDS):
        - ✓ Shortness of breath
        - ✓ Tachypnea
        - ✓ ↓SpO_2
        - ✓ Bilateral crepitation (due to accumulation of edematous fluid).
    b. Left sided pleural effusion:
       It results from leakage of pancreatic fluid through small pores in the diaphragm into the pleural cavity.
    c. Basal atelectasis:
       Due to prolonged bed rest.
5. Eye:
   Icterus may be present.
   Mechanism: Inflammation of the head of pancreas may lead to CBD obstruction.
6. Abdominal examination:
    a. Abdominal distension
    b. Reduced bowel sound
    c. Signs of peritonitis
    d. Ascites may be present
    e. ***Gullen's test***: Bluish discoloration around umbilicus due to extravasation of blood.
    f. ***Grey-Turner's sign***: Greenish yellow discoloration over the flanks due to tissue metabolism of bilirubin.
7. Skin:
   Erythematous skin lesion due to subcutaneous fat necrosis.

Investigations

Investigations to confirm the diagnosis of acute pancreatitis

1. Blood: Hb, TC, DC CRP
   Hb: ↓ in hemorrhagic pancreatitis
   TC, DC, CRP: ↑ due to inflammation/ intra-abdominal infection.
   Ex.:
   Necrotizing pancreatitis,
   Peripancreatic abscess,
   Peritonitis.
2. Renal function: Na+ K+ Urea Creatinine
   To look for any dehydration.

3. Liver function:
   Bilirubin: ↑
   AST, ALT, GGT, ALP: Mild ↑.
4. Pancreatic enzymes:
   - ✓ Serum lipase: Elevation of at least 3 times more than normal is suggestive of acute pancreatitis.
   - ✓ Serum amylase: Elevation of at least 3 times more than normal is suggestive of acute pancreatitis. However, there are other intra-abdominal causes of elevation of serum amylase.
   - ✓ Normalization of serum lipase usually lags behind that of serum amylase.

Investigations to detect risk factors and complications of acute pancreatitis

1. Arterial blood gas (ABG):
   To look for:
   - ✓ Metabolic acidosis
   - ✓ ↓pO_2.
2. Serum Ca++:
   - Hypercalcemia is a risk factor of acute pancreatitis
   - Acute pancreatitis leads to hypocalcemia (by saponification of fat).
3. Fasting lipid profile
4. Chest X Ray:
   To look for:
   - ✓ ARDS
   - ✓ Pleural effusion.
5. USG abdomen:
   To look for:
   - ✓ Inflammation of pancreas
   - ✓ Gallbladder stone
   - ✓ CBD dilatation
   - ✓ Ascites.
6. CECT abdomen:
   To look particularly for:
   - ✓ Necrotizing pancreatitis
   - ✓ Peripancreatic abscess
   - ✓ Pancreatic pseudocyst.
7. Aspiration of ascitic and pleural fluid:
   Show elevated levels of amylase and lipase.

Treatment

Treatment of acute pancreatitis consists of 3 parts:

1. Supportive treatment
2. Interventional treatment
3. Surgical treatment.

Supportive treatment

A. **A**bsolute bed rest

B. **B**owel rest:

- Nil by mouth
- Suction by Ryle's tube
- Gradual introduction of enteral feeding
- If required: total parenteral nutrition.

C. Maintain **c**irculation:

- IV fluid
- Proper hydration must be maintained.

D. **D**rugs (Supportive):

- Analgesic-antispasmodic: Drotaverine
- Opioids: Tramadol/ Pethidine.
  Avoid morphine as it constricts sphincter of OD.
- Antibiotics (particularly if intra-abdominal infection is suspected): Imipenem-Silastin.
- Antifungal (as intra-abdominal fungal infection is not uncommon)
- Anti-ulcer drugs: To avoid stress ulcer: H_2 blocker/ PPI.

D. Diet:
Gradual introduction of normal diet.

D. DVT prophylaxis.

Interventional treatment

A. Percutaneous drainage (paracentesis) of peripancreatic abscess/ infected pseudocyst/ ascites/ pleural effusion.
B. ERCP with stenting + sphincterotomy in patients with CBD obstruction.

Surgical treatment

A. Necrosectomy/ debridement in necrotizing pancreatitis
B. Early cholecystectomy in case of gallstone pancreatitis.

Complications of acute pancreatitis

Organ involved	***Complications***
Pancreas	1. Necrosis 2. Hemorrhage 3. Peripancreatic abscess 4. Pancreatic pseudocyst (sterile/ infected) 5. Chronic pancreatitis
Abdomen	1. Ascites 2. Paralytic ileus 3. Peritonitis
CNS	Encephalopathy
CVS	Circulatory shock/ collapse
Respiratory	1. ARDS 2. Pleural effusion 3. Atelectasis
Eye	Purtscher's retinopathy (Sudden blindness due to occlusion of posterior retinal artery by aggregated WBCs)
Kidney	Acute tubular necrosis (ATN)
Skin	Subcutaneous fat necrosis

Differential diagnosis of acute pancreatitis/ causes of hyper-amylase-emia

1. Abdominal causes:
   - ✓ Acute/ chronic pancreatitis
   - ✓ Perforation: Bowel/ peptic
   - ✓ Bowel infarction
   - ✓ Acute cholecystitis
   - ✓ Ectopic pregnancy (ruptured).
2. Non-abdominal causes:
   - ✓ Salivary gland diseases: Mumps/ Sialolithiasis (stone)/ tumor
   - ✓ Bronchogenic carcinoma
   - ✓ Ovarian carcinoma.