

0.3 - 1.2.

Total bilirubin — 0.2 - 1 mg%
direct bilirubin — 0 - 0.2 mg%
indirect bilirubin — 0.2 - 0.8 mg%
~1

JAUNDICE

- Accumulation of bilirubin in body tissues

BILIRUBIN METABOLISM

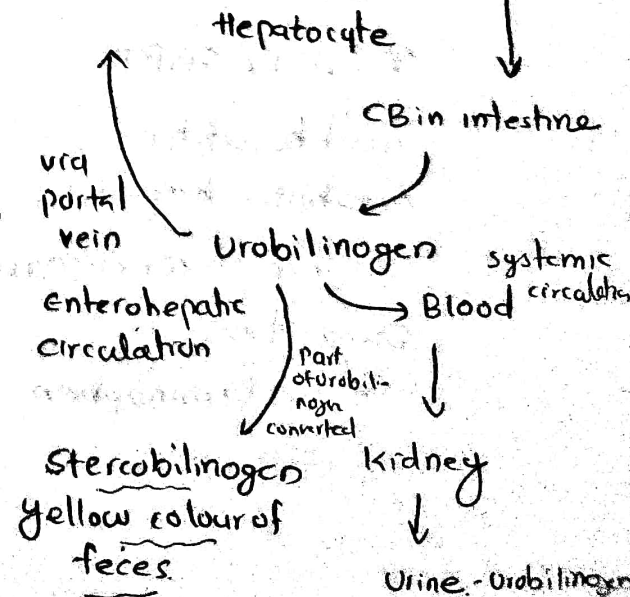
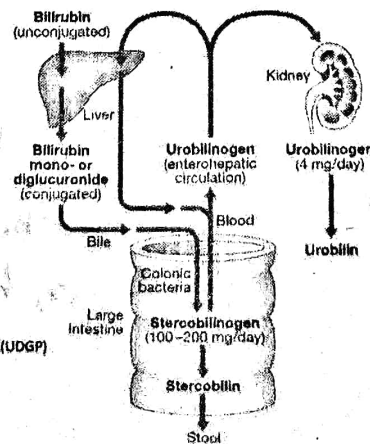
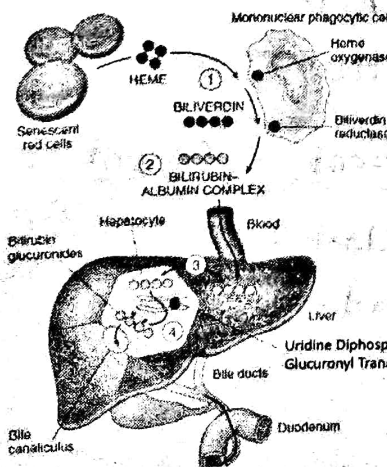
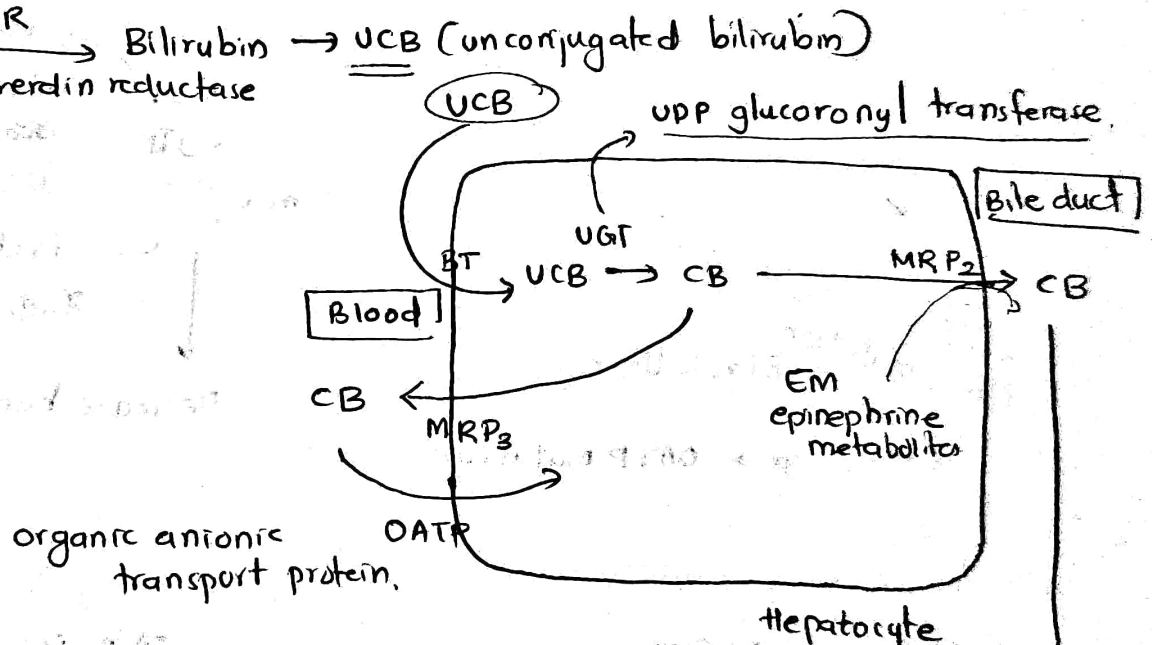
- Bilirubin is the major breakdown product of hemoglobin

Heme $\xrightarrow{HO}$ Biliverdin
Heme Oxygenase

Biliverdin $\xrightarrow{BR}$ Bilirubin $\rightarrow$ UCB (unconjugated bilirubin)
Biliverdin reductase

[UCB + Albumin]
water insoluble

MRP2 - multidrug resistant P2



CB $\geq$ 20% T. Bilirubin
conjugated hyperbilirubinemia.

Bilirubin increased - Liver enzymes normal.

ISOLATED HYPERBILIRUBINEMIA

✓ I. Indirect hyperbilirubinemia

- A. Hemolytic disorders - RBC destruction in periphery
- B. Ineffective erythropoiesis - RBC destruction in bone marrow
- C. Increased bilirubin production
 - 1. Massive blood transfusion
 - 2. Resorption of hematoma

Thalassemia
vitamin B12 deficiency

D. Drugs

- 1. Rifampin
- 2. Probenecid
- 3. Ribavirin
- 4. Protease inhibitors (Atazanavir, Indinavir)

→ ↓ hepatic uptake of UCB

E. Inherited conditions

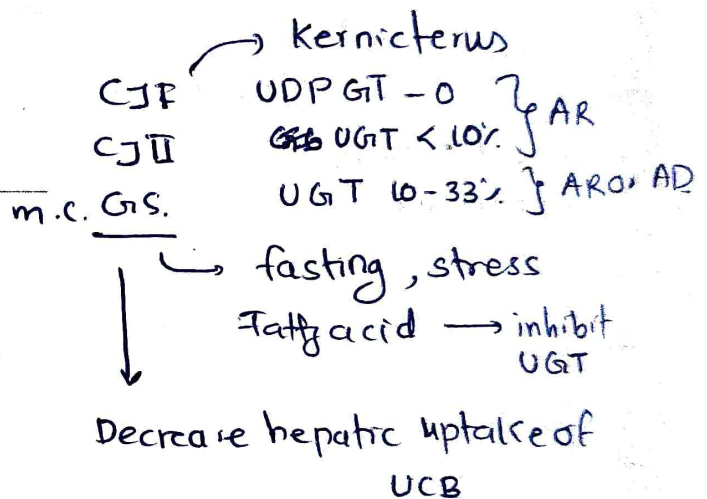
- 1. Crigler-Najjar types I and II
- 2. Gilbert's syndrome

✓ II. Direct hyperbilirubinemia (inherited conditions)

- A. Dubin-Johnson syndrome
- B. Rotor syndrome

DJ
MRP2 mutation → Black liver.

Rotor syndrome → OATP mutation.



HEPATOCELLULAR

↑ SGOT / SGPT

viral hepatitis

Alcoholic hepatitis

chronic liver disease.

Drug toxicity

- Acetaminophen

- INH

CHOLESTATIC

↑ ALP

↑ HBRD

Present

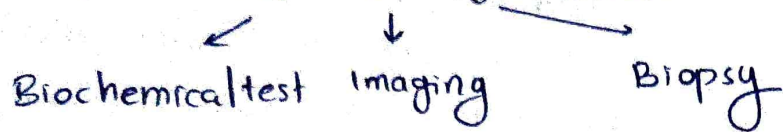
Absent

choledocholithiasis
ca head of pancreas.

PBC
PSC

LIVER FUNCTION TESTS

Test to detect liver dysfunction



Focal nodular hyperplasia → not called cirrhosis

CIRRHOSIS

- Diffuse hepatic fibrosis. Distortion of hepatic architecture → nodules

ETIOLOGIES

Alcoholism Chronic viral hepatitis Hepatitis B Hepatitis C Autoimmune hepatitis Nonalcoholic steatohepatitis NASH • Biliary cirrhosis ✓ Primary biliary cholangitis ✓ Primary sclerosing cholangitis ✓ Autoimmune cholangiopathy	Cardiac cirrhosis Inherited metabolic liver disease • Hemochromatosis • Wilson's disease • α_1 -Antitrypsin deficiency • Cystic fibrosis Cryptogenic cirrhosis
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Heart failure → congestive hepatomegaly
↓
cirrhosis

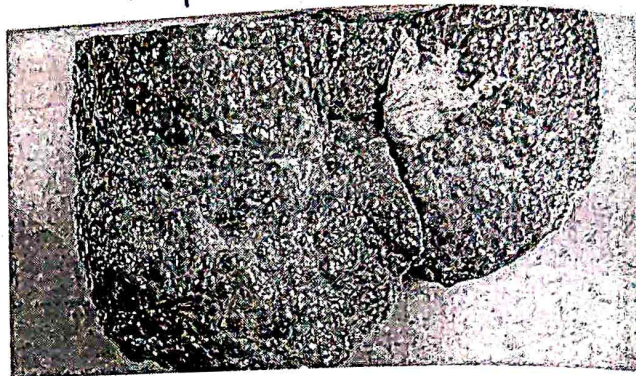
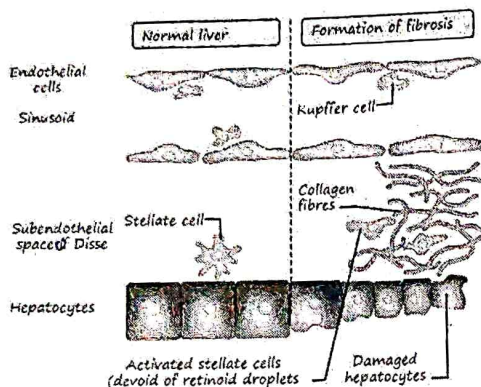
Hepatic Stellate cell (HSC) — stores vitamin A
 Fibroblastogenic stimuli
 HSC → Myofibroblast

collagen, Fibronectin -

PATHOLOGY

Nodules
 micronodular < 3mm - Alcoholism
 macronodular > 3mm - chronic viral hepatitis

Hepatocyte damage



CLINICAL FEATURES

compensated cirrhosis

- Early stage - Asymptomatic → non specific symptoms
- Decompensated stage
- Other symptoms
- Features related to etiology
- Examination

(Anorexia, weight loss, muscle cramps, Fatigue)
 worsening of jaundice, upper GI bleed, ascites, hepatic encephalopathy, PT INR > 2

Head

Parotid - enlargement of parotid (alcohol related)

Fetor hepaticus (pungent odor - ↑ dimethyl sulphide)

Hands

Palmar erythema

Clubbing (Hypoalbuminemia)

Terry's nails

Asterixis (flapping tremor)

Dupuytren's contracture

Trunk

spider naevi (aka spider angioma)
 dilated central arteriole with branching vessel in radiating manner
 - spider naevi - pathological
 ↳ (pregnancy, malnutrition)
 ✓ no. & size correlated severity.
 ✓ gynecomastia
 - estrone → estradiol
 → breast atrophy

hypogonadism, menstrual irregularities

Abdomen

Ascites.
 hepatomegaly
 splenomegaly
 caput medusae (dilated opening of collateral)
 - dilated abdominal veins with flow away from umbilicus
 - crurillier & Baumgarten murmur (venous hum)
 - umbilical hernia

Testicular atrophy, skin bruise, itch marks
loss of axillary & pubic hair

INVESTIGATIONS

↑ disease activity

- LFT - Hypoalbuminemia, Serum bilirubin, SGOT, SGPT
Alkaline phosphatase (ALP) ↑, GGT ↑ - alcohol, PT-INR ↑
decompensated state
- Other blood investigations
Anemia, thrombocytopenia (hypersplenism), viral markers
HBsAg, AntiHCV
- Imaging

Ultrasonography - Surface nodularity & regenerative
portal vein diameter, Ascites, splenomegaly

Elastography - [fibro scan] - Ultrasound waves
propagation velocity & elasticity

- Biopsy
- Wilson's disease
- Auto immune hepatitis
- Hemo chromatosis

SCORING SYSTEMS

CHILD - PUGH scoring
- MELD use:

FACTOR	POINTS TOWARD TOTAL SCORE			
	UNITS	1	2	3
Serum bilirubin	μmol/L	<34	34-51	>51
	mg/dL	<2.0	2.0-3.0	>3.0
Serum albumin	g/L	>35	30-35	<30
	g/dL	>3.5	3.0-3.5	<3.0
Prothrombin time	seconds prolonged	<4	4-6	>6
	INR ^a	<1.7	1.7-2.3	>2.3
Ascites		None	Easily controlled	Poorly controlled
Hepatic encephalopathy		None	Minimal	Advanced

- ✓ Prognosis
- ✓ need for liver transplantation

Model for Endstage liver disease

Serum bilirubin

PT-INR

Serum creatinine

CPA - 5, 6 - compensated cirrhosis

CPB - 7, 8, 9 - decompensated cirrhosis

CP C - 10-15

MELD - Na⁺

Serum Na⁺

- Histologic scoring system
- METAVIR scoring
- Ishak scoring

ASCITES

- Abnormal accumulation of fluid in peritoneal cavity

ETIOLOGIES

- Liver cirrhosis with portal hypertension
- Cardiac failure
- Tuberculosis ; TB peritonitis
- Malignancies ; ca Lung, Breast, colon, ovary, mesothelioma
Peritoneal carcinomatosis
- Pancreatic diseases
- Hypothyroidism

Hypoalbuminemia - Nephrotic syndrome.

MECHANISM IN LIVER CIRRHOSIS

→ Increased sinusoid pressure - capillary leakage in peritoneum
Portal hypertension

- Sodium & water retention

Increase in NO - splanchnic vasodilation

→ RAAS

Hypoalbuminemia

CLINICAL FEATURES

↑ abdominal girth

Buddle sign - 120ml, shifting dullness - 500ml, Fluid thrill - 1500ml

Signs of underlying etiology

• Complications

- ✓ SBP - Spontaneous bacterial peritonitis
- ✓ Hyponatremia → Solute free water reabsorption
- ✓ Refractory ascites
- ✓ Hepatorenal syndrome

INVESTIGATIONS

1. Ultrasound — Ascites ⊕/⊖, etiology

2. CT Scan

3. Diagnostic paracentesis

Appearance, Total WBC, Absolute neutrophil count

Total protein, Albumin, culture

Other test to detect underlying etiology → cytology

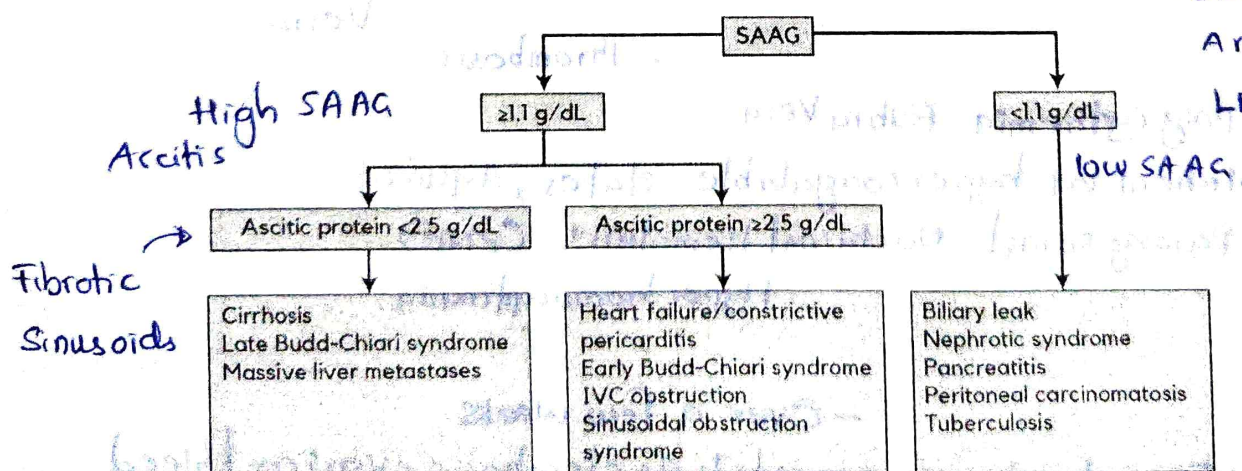
Peritoneal biopsy

CBNAAT, ADA

Amylase, lipase

LDH.

SAAG: Serum Ascites Albumin Gradient



- serum albumin — Ascites fluid Albumin
- Gradient correlates with portal pressure.

TREATMENT

Sodium intake $< 2g/day$

• Grade 1 - mild Salt restriction

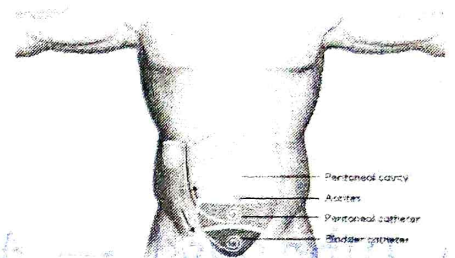
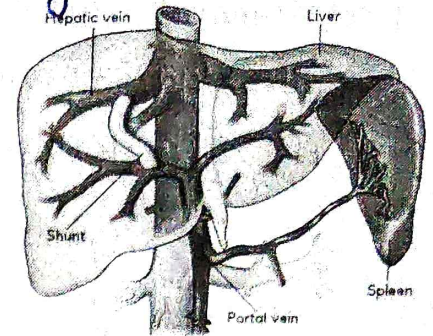
• Grade 2 - moderate salt restriction +

Drugs: Diuretics (Frusemide + spironolactone)

• Grade 3 - severe - salt restriction

+ Diuretics + therapeutic
paracentesis (LVP)
+ supplement with
Albumin

• Refractory ascites



✓ TIPS

✓ α pump system

✓ midodrine, clonidine

BUDD CHIARI SYNDROME

- Abnormal hepatic venous outflow obstruction $\rightarrow$ IVC $\rightarrow$ hepatic Vein.

PREDISPOSING FACTORS - Thrombosis.

Poly cythemia Rubra Vera.

Inherited hypercoagulable states, Acquired
Paroxysmal Nocturnal Hematuria, (PNH)

Hyper homocystinuria.

CLINICAL FEATURES

- Most commonly subacute - Over a few weeks

Acute, chronic is presented as cirrhosis - varices/bleed

Abdominal pain, abdominal distension (ascites)

icterus, hepatomegaly.

INVESTIGATIONS

- Imaging Doppler Ultrasonography
CE MRI

- Liver biopsy → Centrilobular congestion → Fibrosis

TREATMENT

- Treatment of ascites — salt restriction + diuretics
- Anticoagulation LMWH → oral anticoagulation.
- Thrombolysis — only if ^{no} response with anticoagulation.
- Adenosine receptor agonist — Defibrotide
- TIPS
- Liver transplantation

SBP — Spontaneous Bacterial peritonitis

- Bacterial infection of ascitic fluid & no other identifiable intra abdominal source.

MECHANISM

- Alteration of gut liver axis

Pathological bacterial translocation (along the gut wall first to the blood, subsequently to lymphatics then to peritoneum.)

CLINICAL FEATURES

Ascites

Abdominal pain, fever, vomiting, diarrhea
worsening of ascites

AKI, sepsis, hepatic encephalopathy

INVESTIGATIONS - Diagnostic paracentesis

- Neutrophil count in ascitic fluid

ANC $> 250 / \text{mm}^3$

or

Total WBC $> 500 / \text{mm}^3$

Culture

Spontaneous - monomicrobial

- E. coli

2° bacterial

- polymicrobial

TREATMENT

- Empirical antibiotic therapy

3rd generation cephalosporin /

Fluoroquinolone.

- Prophylaxis

- To prevent recurrence of SBP

norfloxacin, Trimethoprim - sulfamethoxazole.

HEPATIC ENCEPHALOPATHY

- Alteration in mental status in the presence of liver failure

Spectrum

PATHOPHYSIOLOGY

in case Hepatic dysfunction

- Gut derived neurotoxins (ammonia) $\rightarrow$ Brain - cerebral edema

- Abnormal synaptic neurotransmission - elevated level
inhibitory neurotransmitter $\rightarrow$ GABA

CLINICAL FEATURES

$\downarrow$ excitatory neurotransmitter $\rightarrow$ Glutamate

- Reversal of Sleep rhythm

- Forgetfulness, confusion

- Bad handwriting

- Delirium $\rightarrow$ Seizures $\rightarrow$ coma

Examination $\rightarrow$ Bilateral extensor plantar

Flapping tremor (asterixis), constructional apraxia

Grading :
- West Haven
- SONIC

- Precipitants : Alcohol
Blood in gut
Constipation
Dehydration
Electrolyte imbalance (hypokalemia, hyponatremia)
Fever.

INVESTIGATIONS

- No specific lab finding
Serum ammonia level - elevated.
EEG - Abnormal. - Triphasic waves, Delta waves.

TREATMENT

- Treat precipitating factor
- Oral lactulose - Reduces intestinal pH - $\text{NH}_3 \rightarrow \text{NH}_4^+$
↓ ammonia absorption from intestine
- Oral antibiotics - Rifaximin. modifies intestinal bacteria
- LOLA L-ornithine - L aspartate. → ↑ ammonia pool.
excretion
- Zinc - mild benefit

HEPATORENAL SYNDROME

- Functional Renal Failure due to severe hepatic injury - cirrhosis with Ascites

PATHOPHYSIOLOGY

- Altered renal hemodynamics

↑ NO → splanchnic vasodilation → ↓ renal perfusion
↓
AKI

Acute

TYPES ↓ relatively slowly progressive.

- HRS AKI, HRS NAKI - Type 2 HRS

Formerly Type 1 HRS

CRITERIA HRS AKI

- Diagnosis of cirrhosis and ascites
- Diagnosis of AKI
- No response after 2 consecutive days of diuretic withdrawal and plasma volume expansion with albumin
- Absence of shock
- No current or recent use of nephrotoxic drugs
- No macroscopic signs of structural kidney injury — Proteinuria, Hematuria.

CRITERIA HRS NAKI

- HRS AKD - eGFR < 60ml/min for < 3 months
- HRS CKD - eGFR < 60ml/min for ≥ 3 months

TREATMENT

- **Pharmacotherapy** - stop diuretics

IV Albumin ⊕

IV Terlipressin

IV Norepinephrine

Oral Midodrine + s.c or iv Octreotide

} splanchnic
v.c.

- **Definitive treatment**

Liver transplantation.

Alcohol use disorder
 $> 2 \text{ drinks/day}$ in female, $> 3 \text{ drinks/day}$ in male
 1 drink = 14g methanol.

Chronic & **ALCOHOLIC LIVER DISEASE**

- Excessive alcohol use - Fatty liver, Alcoholic hepatitis, Cirrhosis

Fatty liver

- ✓ **Fatty liver** → reversible after alcohol cessation.

Inhibition of beta oxidation Fatty acids
 mostly asymptomatic, Hepato megal +1-

- ✓ **Alcoholic hepatitis** → reversible After alcohol cessation,

Acetaldehyde, TNF-alpha liver inflammation

Rapid onset jaundice, Tender hepatomegaly

signs of hepatic decompensation - Ascites, encephalopathy,
 Coagulopathy, upper GI bleed.

- ✓ cirrhosis.

INVESTIGATIONS

Rapid decline in Hepatic cell
 - ~~FH~~ Fulminant Hepatitis

Fatty liver - $\uparrow \text{AST}$, $\uparrow \text{ALT}$, $\uparrow \text{AGT}$

Alcoholic Hepatitis - Bilirubin $\uparrow$, $\uparrow \text{AST}$, $\uparrow \text{ALT}$, PT-INR

Cirrhosis

Zieve's syndrome

↳ hemolytic anemia

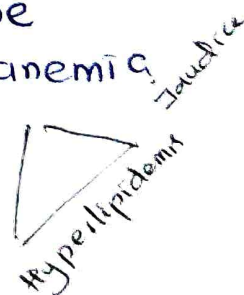
TREATMENT

- Fatty liver - stop alcohol
- Severe alcoholic hepatitis - stop alcohol

D.O.C - oral prednisolone.

- Cirrhosis

Thiamine Supplementation



MELD > 20

MDF > 32

Maddrey's discriminant function

$4.6 (\text{PT}_{\text{test}} - \text{PT}_{\text{control}}) + \text{s. bilirubin}$

NAFLD:

Metabolic dysfunction associated Steatotic liver disease.

MASLD

- Steatotic liver disease + at least 1 component of metabolic syndrome

↑ waist circumference

↑ serum Triglyceride

↓ s. HDL, BPT, ↑ Blood glucose

ETIOPATHOGENESIS

- Insulin resistance

Obesity, DM, Hypertriglyceridemia,

Cushing Syndrome, steroids, GSA, PCOS, Red meat,

soft drinks

CLINICAL FEATURES: Fatty liver, MASH, cirrhosis

Asymptomatic

hepatomegaly

Jaudice

Tender hepatomegaly

INVESTIGATIONS

Fatty liver: De Ritis ratio < 1 , GGT ↑
NAFLD.

↳ cirrhosis > 1

TREATMENT

- Lifestyle modifications — Diet, Exercise, weight reduction.
- Vitamin E, Pioglitazone, Obeticholic acid, Resmetirom

Thiazolidinediones

↳ Bile acid analogues

↳ Thyroid hormone receptor B-agonist

- Advanced cirrhosis

→ liver transplantation

AUTOIMMUNE HEPATITIS

- Immune mediated chronic hepatitis

ETIOPATHOGENESIS

- Loss of immune tolerance to self antigens
- Lymphocytic infiltration
- Interface hepatitis, periportal necrosis, emperipolesis

Portal tract & } inflammation
Hepatocytes. } happen.

cell within
cell
within hepatocytes,
lymphocytes get engulfed.

CLASSIFICATION

- Type 1 m.c

90% cases

Autoantibodies — ANA, ASMA, Anti actin antibody,
Anti sLA (soluble liver antigen)

- Type 2

10% cases

Autoantibodies Anti LKM-1, Anti LKM-3,
anti LC
↓
Liver cytosol.

CLINICAL FEATURES

- Asymptomatic
- Chronic non specific symptoms - fatigue, malaise
- Acute onset symptoms - Acute hepatitis - jaundice
- Fulminant presentation / fulminant hepatitis
- Cirrhosis
- Extra-hepatic - SLE

INVESTIGATIONS

Bilirubin increased , USG liver
SGOT, SGPT ↑↑ , cirrhosis workup
ALP ↑ - Auto
Globulin ↑.
Liver Biopsy.

TREATMENT

D.O.C → Biopsy followed by
taper steroid. Azathioprine,
steroid sparing agent mmf

PRIMARY BILIARY CHOLANGITIS

- Old name - Primary Biliary Cirrhosis

ETIOPATHOGENESIS

- Autoimmune destruction of small intrahepatic bile ducts

CLINICAL FEATURES

- Age group — > 50yr F > M
- Common symptoms — Fatigue, pruritis
- Other manifestations — osteoporosis, steatorrhea, skin pigmentation
- Examination Icterus (+), hepatomegaly, splenomegaly

INVESTIGATIONS

- LFT : cholestatic pattern.
- Antibody Anti mitochondrial antibody (AMA) 90% can.
- Biopsy — Granuloma, ductal proliferation → Fibrosis

TREATMENT

D.O.C — UDCA — Ursodeoxycholic acid
Definite R — Liver transplantation

WILSON'S DISEASE

- Inherited disorder of copper metabolism

ETIOPATHOGENESIS

- Mutations in ATP7B gene — Chromosome 13q

↓
Copper not excreted into Bile
↘ defective copper incorporation into apoceruloplasmin.

Free copper → (↑)
Bound copper → (↓)

- Inheritance — AR.
- Toxic accumulation of copper in the liver, brain, and other organs

CLINICAL FEATURES

- Age group wide range 2nd-3rd decade
- Hepatic - hepatitis, cirrhosis
- Neurologic tremor (wing beating type), parkinsonism, dystonia.
- Ocular Kring, sunflower cataract.
- Others Psychiatric manifestations



INVESTIGATIONS

- Screening → 24 hour urinary excretion[↑] copper, serum ceruloplasmin[↓] (most sensitive)
- Confirmation

Liver Biopsy → hepatic dry weight of copper - [↑]

most specific test • MRI Brain - Face of giant panda sign

TREATMENT

- Remove excess copper LFT - hepato ~~test~~/cirrosis, slit lamp - Kring, peripheral smear.
- D-penicillamine, Trientine, Zinc, Tetrathiomolybdate



Worsen Neuro ~~penicillamine~~ Wilson disease.

HEMOCHROMATOSIS

- Inherited iron storage disorder

ETIOPATHOGENESIS

- Hereditary - mutation in HFE gene on chromosome 6p.

- Secondary iron overload

sideroblastic anemia, Thalassemia
multiple blood transfusion.

} parenchymal
iron
deposition.

→ mucosal iron absorption $> 4 \text{ mg/day}$

CLINICAL FEATURES

- Age group $> 50 \text{ yrs}$
- Early symptoms non specific - fatigue, Malaise
- Hepatic Hepatitis, cirrhosis, HCC

- Others Pancreas - Diabetes
Skin - hyperpigmentation } Bronze diabetes, Pituitary - Hypo gonadism

INVESTIGATIONS

- Screening Transferrin Saturation, serum ferritin

$$\frac{\text{Serum iron}}{\text{TIBC}} \times 100 > 45\% \quad > 300 \text{ mg/L}$$

- Confirmation

Liver biopsy.

→ perls' prussian blue

, genetic study

LFT, RFT, PT, aPTT.

Liver - USG.

TREATMENT

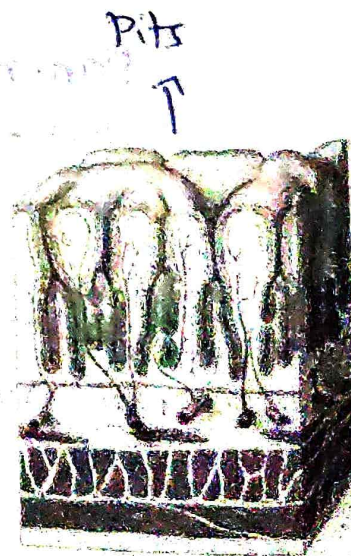
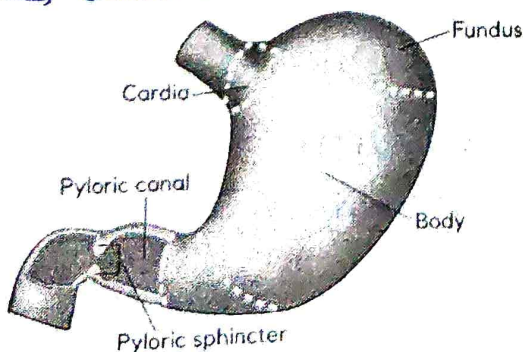
- Dietary modification $\downarrow$ take of iron rich foods
- Phlebotomy - T.O.C
initially weeks $\rightarrow$ later once in 2 weeks $\rightarrow$
Once in 2-3 months
- Iron chelators
Deferasirox
Deferoxamine
End stage: Liver transplantation

PEPTIC ULCER DISEASE

- Break in the duodenal or gastric mucosa

$\downarrow$ $\rightarrow$ Ulceration

Erosion $< 5\text{mm}$ does not penetrate muscularis mucosa,
> 5mm, penetrate muscularis mucosa,



Duodenum
— 1st part of duodenum

Stomach
along the lesser curvature.

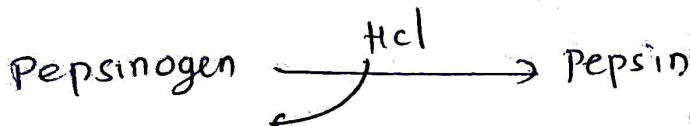
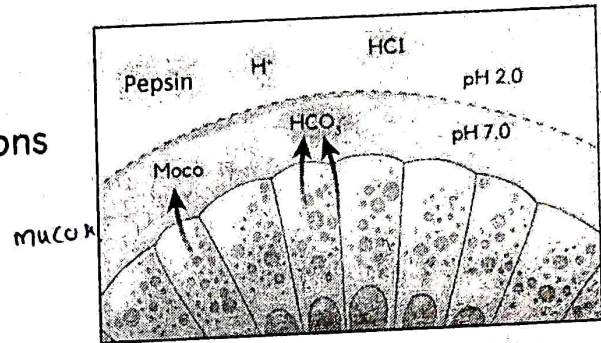
ETIOPATHOGENESIS

- Risk factors — *Hy. pylori*, NSAIDs
Smoking, Alcoholism, stress, Type A personality
Cushing syndrome, Hyperparathyroidism, ZES, o blood group
Drug → steroid, Bisphosphonates,
- Acid and pepsin overwhelm the defense mechanisms

✓ Pre-epithelial : mucus, bicarbonate

✓ Epithelial : cell membrane, tight junctions

✓ Post-epithelial : mucosal blood flow

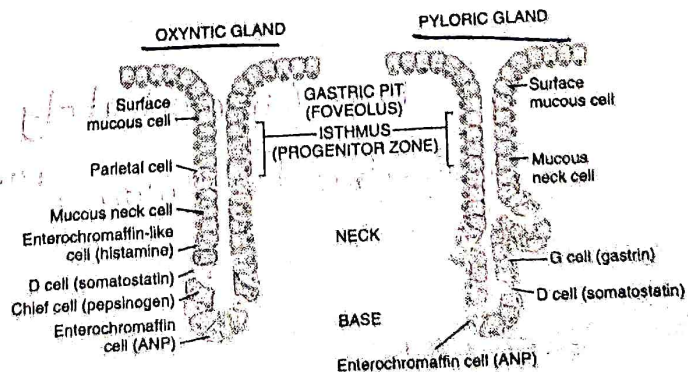
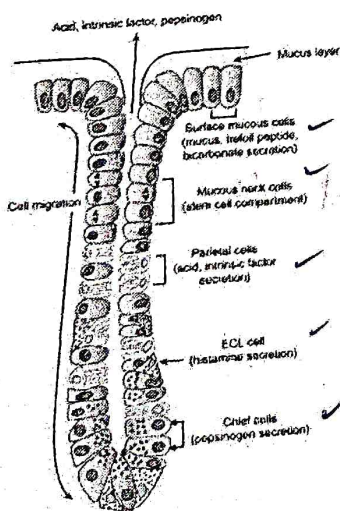


Stimulus

Gastrin — a cells

Histamine — ECL cells

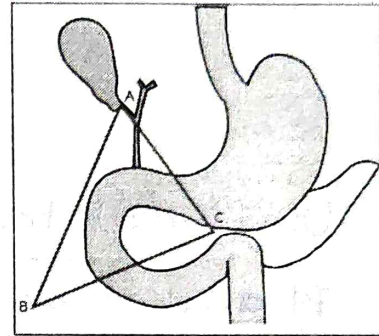
Acetyl choline



ZOLLINGER ELLISON SYNDROME

- Gastrin-secreting gut neuroendocrine tumors *Gastrinoma*
- Result in hypergastrinemia and acid hypersecretion
- Recurrent ulceration of stomach, duodenum and jejunum

60% gastrinomas occur in
- Gastrinoma triangle
a/w MEN-1



INVESTIGATIONS

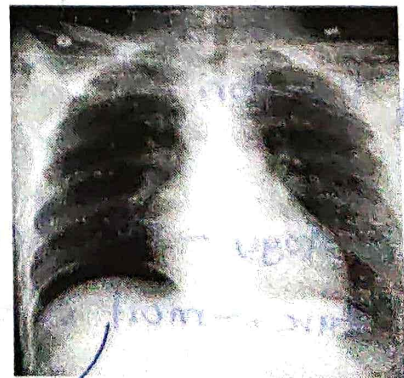
- Endoscopy (Gastroduodenoscopy)

Trial of PPI

↓ 4-8 weeks

No relief

Elderly
significant weight loss } indication.



Air under
diaphragm.

DIAGNOSTIC TESTS - H PYLORI

• Non-Endoscopic Tests

Urea breath test

Serology

Stool antigen

• Endoscopic Tests

Rapid urease test

Histology

Culture

Urea Breath test

Pt drink a soluⁿ of urea with C^{13} .



Serology

- IgG detection

mlc Stool antigen test

RUT → gastric biopsy specimen

placed into a gel containing urea as indicator.

H. pylori →

↑ pH → colour change

Histology - modified Grams, silver stain

Culture - most specific.

TREATMENT

- Antacids — Aluminum & magnesium hydroxide
- H₂ receptor antagonists Ranitidine, famotidine
- Proton pump inhibitors Pantoprazole, omeprazole
- Mucosal protective agents Sucralfate, Misoprostol
Bismuth containing preparation
colloidal Bismuth subcitrate
Bismuth subsalicylate
- Potassium competitive channel blockers
↳ Vopran.

H. pylori eradication regimen

REGIMEN	DRUGS (DOSES)	DOSING FREQUENCY	DURATION (DAYS)
Clarithromycin triple	PPI (standard or double dose)	bid	14
	Clarithromycin (500 mg)		
	Amoxicillin (1 g) or metronidazole (500 mg tid)		
Bismuth quadruple	PPI (standard dose)	bid	10-14
	Bismuth subcitrate (120-300 mg) or subsalicylate (300 mg)	qid	
	Tetracycline (500 mg)	qid	
	Metronidazole (250-500 mg)	qid (250 mg) tid to qid (500 mg)	
Concomitant	PPI (standard dose)	bid	10-14
	Clarithromycin (500 mg)		
	Amoxicillin (1 g)		
	Nitroimidazole (500 mg) ^c		

Surgical treatment

- chronic non healing gastric ulcer
 - partial gastrectomy with Billroth I or antistomum
- Duodenal ulcer
 - vagotomy & drainage
 - HSV
 - vagotomy + Antrectomy

CELIAC DISEASE

- Chronic Immune mediated enteropathy → duodenum & jejunum
- Celiac sprue. Gluten sensitive enteropathy

ETIOPATHOGENESIS

- Precipitated by gluten in genetically predisposed individuals
- HLA DQ2 and DQ8
 - ↳ wheat, Barley, rye
 - ↳ certain varieties of oats

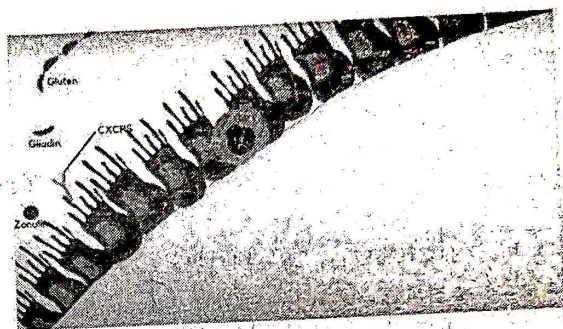
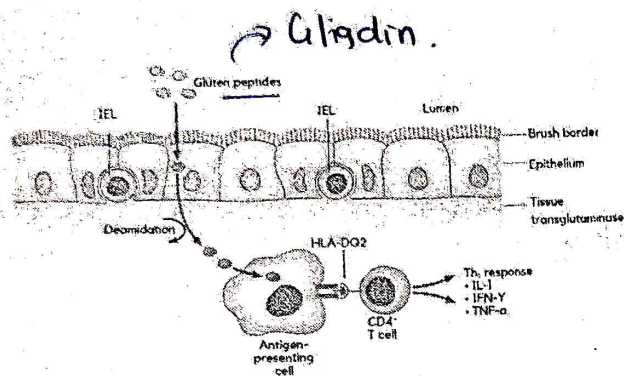
Glutens

deamidation $\rightarrow$ amide $\rightarrow$ deamidated
glutens $\rightarrow$ Ape $\rightarrow$ cytokine $\rightarrow$ damage
intestines / cells

intestinal epithelial cell produce

Zonulin + CXCR5

loosening of "tight junctions"



CLINICAL FEATURES

- Asymptomatic
- Infants — Diarrhea, malabsorption, failure to thrive.
- Children — growth retardation, delayed puberty
- Adults — malabsorption — steatorrhea
 $\rightarrow$ dyspeptic symptoms
 Folate deficiency, osteoporosis
- Associated diseases

Dermatitis herpetiformis

Type 1 DM

INVESTIGATIONS

• Serological tests

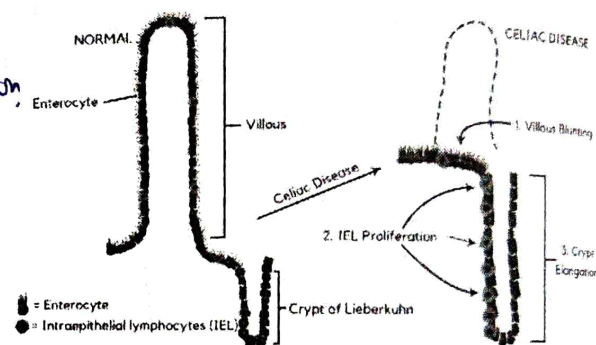
IgA Tissue transglutaminase

Anti- endomysial

Deamidated anti-gliadin

• Small intestinal biopsy - confirmation

- ① villous blunting
- ② IEL proliferation
- ③ Crypt elongation.



TREATMENT

- Gluten free diet
- Nutrient supplementation Fe, Folic acid, calcium.
- DEXA scan T score, Z score.

COMPLICATIONS

Enteropathy associated T cell lymphoma
Small bowel Carcinoma
Squamous cell carcinoma of esophagus

WHIPPLE'S DISEASE

- Chronic multiorgan infection

ETIOPATHOGENESIS

- Tropheryma whipplei Gram +ve, Teco-oral
- Invades into macrophages of bowel mucosa

Lymphatic obstruction



Fat malabsorption.

CLINICAL FEATURES

- G.I symptoms — diarrhea, Ab. pain, weight loss
mesenteric lymphadenopathy
- Neurologic — dementia, ataxia, encephalopathy.
- Joint disease, Cardiac, Psychiatric
arthritis pericarditis

INVESTIGATIONS

Biopsy — villous atrophy, crypt hyperplasia, apoptosis of surface epithelial cells

↳ PCR from Biopsy specimen

PAS +ve diastase resistant granules

TREATMENT

Foamy macrophage

I.V ceftriaxone . 2g I.V daily x 2 weeks

Followed by oral cotrimoxazole x 1 year.

IRRITABLE BOWEL SYNDROME

- Abdominal pain or discomfort and altered bowel habits
in the absence of detectable structural abnormalities

ETIOPATHOGENESIS - multifactorial

- Behavioural and psychosocial factors - stress
(serotonin)
- Abnormal 5HT metabolism
↓
not quite clear
- Mucosal inflammation - mild degree
- Luminal factors - SIBO (small intestine bacterial overload)

CLINICAL FEATURES

- IBS-C, IBS-D, IBS-M - mixed.

ROME IV DIAGNOSTIC CRITERIA

Recurrent abdominal pain, on average, at least 1 day per week in the last 3 months, associated with ≥2 of the following criteria

1. Related to defecation
2. Associated with a change in frequency of stool
3. Associated with a change in form (appearance) of stool

IBS-C

hard pellets

IBS-D

diarrhea

Diagnosis of exclusion.

TREATMENT - Reassure

- IBS-C : ↑ Fiber content
Osmotic laxatives
Drugs: Lubiprostone, Linaclotide, prucalopride,
 cl⁻-channel activator ↳ acts on guanylate cyclase receptor. ↳ 5HT₄ agonist
- IBS-D
Loperamide, codeine,
cholestyramine