ACUTE & CHRONIC LIVER FAILURE

PRESENTER: FELISTERS BOSIBORI
FACILITATOR: DR. SOME

OUTLINE

- DEFINITION
- ANATOMY AND PHYSIOLOGY
- ETIOLOGY AND EPIDEMIOLOGY
- PATHOPHYSIOLOGY
- CLINICAL FEATURES
- DIAGNOSTIC EVALUATION
- TREATMENT
- COMPLICATIONS
- PROGNOSIS

DEFINITION

- A clinical syndrome that results from massive necrosis of liver cells leading to hepatic encephalopathy and impaired synthetic function causing coagulopathy (**INR>1.5**) in a person with previous normal liver or compensated liver disease.

ALF includes;

(1)Biochemical evidence of acute liver (usually <8 wks duration)

(2) no evidence of a known chronic liver disease;

(3)hepatic-based coagulopathy that is not corrected by parenteral administration of vitamin K;

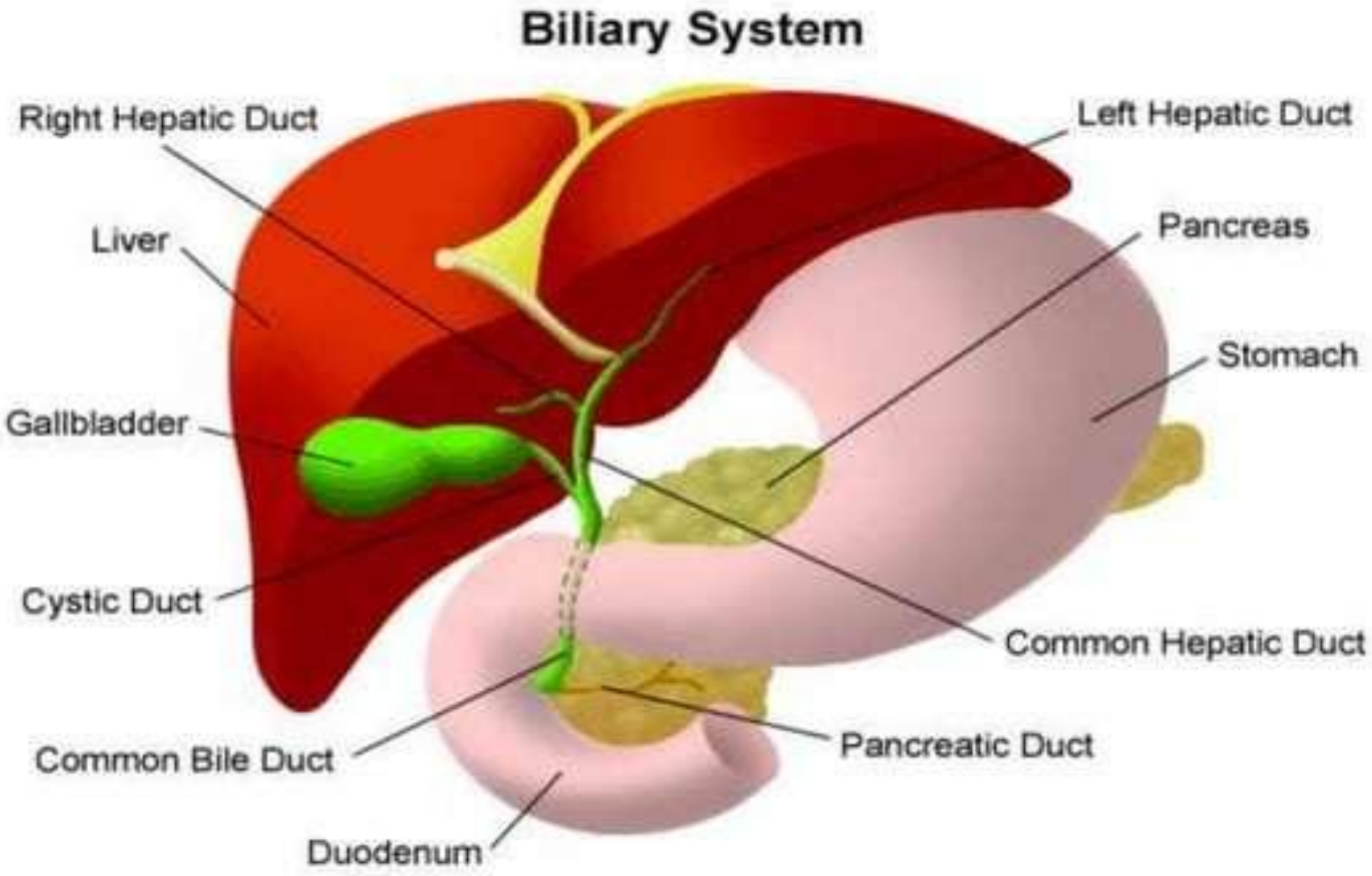
(4) hepatic encephalopathy must be present if the uncorrected prothrombin time (PT) is ≥ 15 seconds or international normalized ratio (INR) is 1.5 to 1.9, respectively; and

(5) hepatic encephalopathy is not required if the PT is ≥ 20 seconds or INR ≥2.0, respectively.

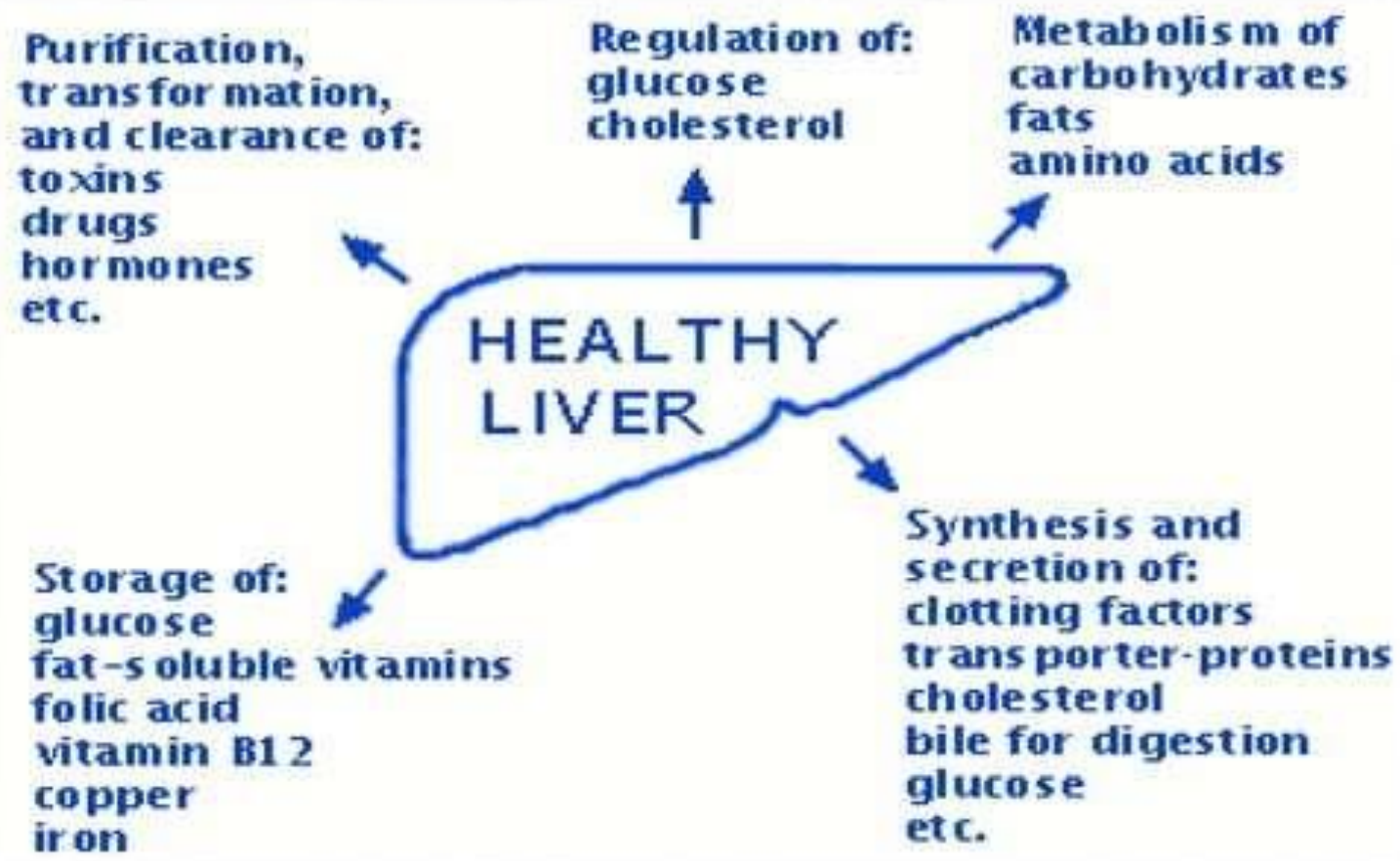
CLASSIFICATION

- **Fulminant HF** is acute hepatic decompensation **within 8wks** from onsent of illness.
- **Subfulminant HF/ late onset HF** is liver disease **up to 26wks** prior to development of encephalopathy.

	Interval: jaundice to encephalopathy	Cerebral oedema	Prognosis	Leading causes
Hyper-acute	<7 days	Common	Moderate	Virus A, B; acetaminophen
Acute	8–28 days	Common	Poor	Non-A/B/C; drugs
Sub-acute	29 days to 12 weeks	Poor	Poor	Non-A/B/C; drugs


Biliary System
Right Hepatic Duct
Left Hepatic Duct
Liver
Pancreas
Stomach
Gallbladder
Cystic Duct
Common Hepatic Duct
Common Bile Duct
Pancreatic Duct
Duodenum

Functions of liver



Blood Supply

- The liver receives a blood supply from two sources.
- The first is the **hepatic artery** which delivers ***oxygenated blood*** from the ***general circulation.***
- The second is the **hepatic portal vein** delivering ***deoxygenated blood*** from the ***small intestine*** containing *nutrients*.

- The blood flows through the liver tissue to the hepatic cells where many metabolic functions take place.
- The blood drains out of the liver via the **hepatic vein.**
- The liver tissue is ***not vascularised with*** a ***capillary network*** as with most other organs, but consists of **blood filled sinusoids** surrounding the hepatic cells.

EPIDEMIOLOGY

- approximately 2000 cases annually occurring in the United States.
- Acetaminophen or paracetamol overdoses are prominent causes of FHF in Europe and, in particular, Great Britain.
- In the developing world, acute HBV infection dominates as a cause of fulminant hepatic failure because of the high prevalence of HBV.
- more often in women (73%) than in men
- Patients younger than 10 years and older than 40 years tend to fare relatively poorly
- women with acute liver failure- older (39 y) than men (32.5 y)

ETIOLOGY

- **Idiopathic** – 15%
- **Acetaminophen overdose**: commonest in developed countries.
- **Viral Hepatitis**:
  - Hepatotrophic viruses: HBV, HDV, HEV, HAV
  - Non-hepatotrophic viruses: adenovirus, CMV, HSV, EBV, Paramyxovirus, Hemorrhagic fever virus.
- **Pregnancy**: acute fatty liver of pregnancy
- **HELLP syndrome**

- **Drugs**: idiosyncratic hypersensitivity or dose dependent.
- **Toxins**: Aflatoxicosis, Aspergillus Flavus
- **Vascular**: Portal V thrombosis, Budd Chiari Syn
- **Metabolic**: wilsons disease
- **Autoimmune** disease
- **Malignancy**: primary and metastatic
- **Miscellaneous**: heat stroke, graft failure.

NB: Broadly classified as Acetaminophen and non-acetaminophen causes for prognostication.

Etiology of ALF In different age group

	Neonates	Infants
Infection	HSV, Adeno virus HBV	HAV, HBV, HSV
Inborn errors of metabolism	Galatosemia, tyrosinaemia, Hereditary fructose	hereditary fructose Intolerance
Immune mediated	Intolerance. Neonatal Hemochromatosis,	Autoimmune hepatitis
Ischemia & abnormal Perfusion	Hemophagocytic syndrome Congestive heart failure, severe aspyxia	Congestive heart Failure,
Drugs & toxins		Acetaminophen,INH, valproate malignancy
other		

Etiology Cont....

	2-10 year old	10-18 yrs old
Infection	HAV, HBV, HSV	HAV, HBV, HSV, HCV
Drugs & toxins	Acetaminophen,INH, valproate	Acetaminophen,INH, valproate
Immune mediated	Autoimmune hepatitis, hemophagocytic syndrome	Autoimmune hepatitis, hemophagocytic syndrome
Ischemia & abnormal Perfusion	Budd-Chiari syndrome, Heart failure, cardiac Surgery, myocarditis	Budd-Chiari syndrome, Heart failure, cardiac Surgery, myocarditis
Metabolic	Wilson's disease, Reye's syndrome,	Wilson's disease, Reye's syndrome,
other	Hemophagocytic syndrome, septicemia, heat stroke	Hemophagocytic syndrome, septicemia, heat stroke

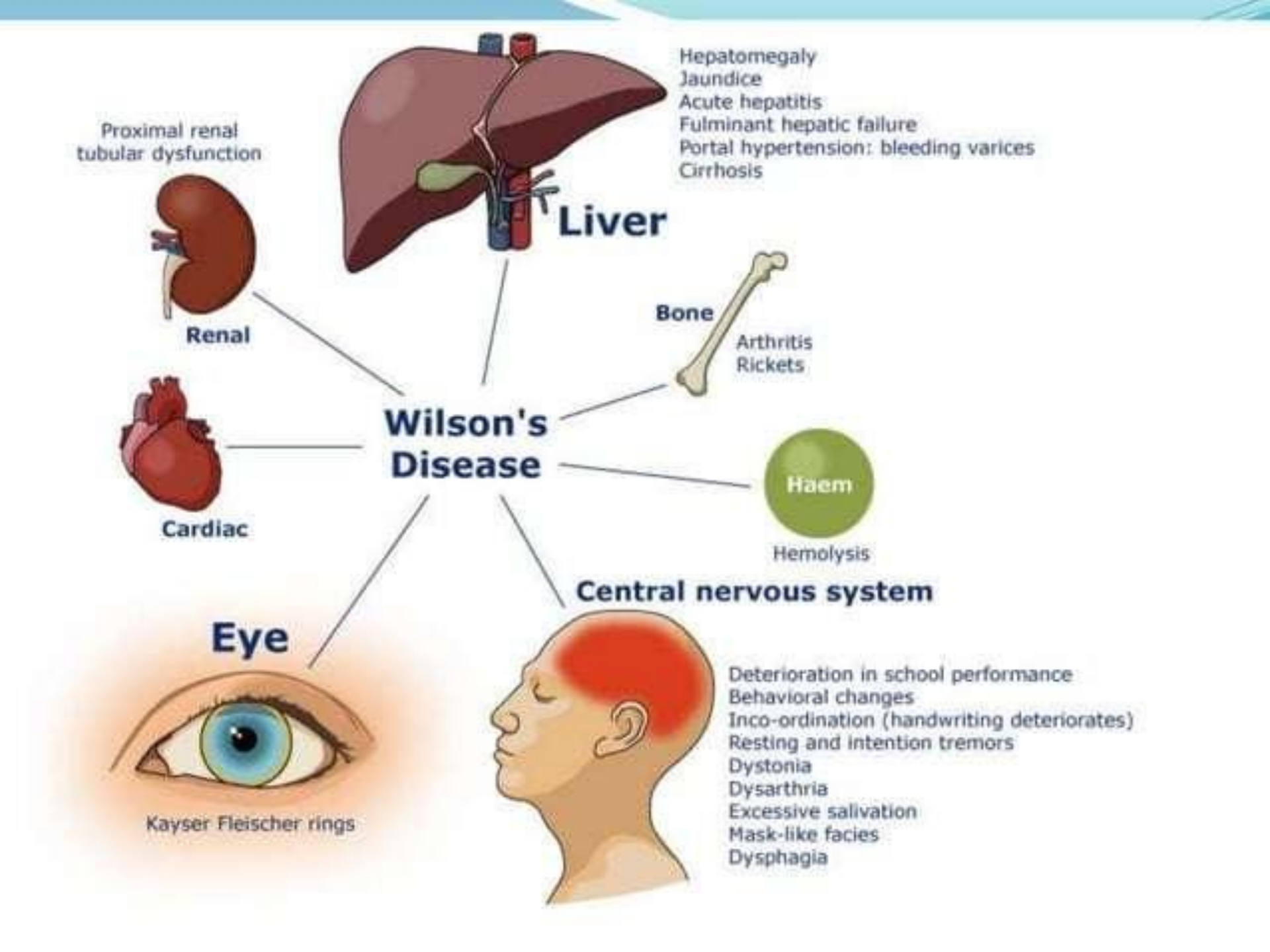
Important drugs responsible for ALF

- Dose dependant:
  - ❖ Acetamenophen
  - ❖ CCBs
  - ❖ Mushroom (aminata phalloides)
- Idiosyncratic:
  - ❖ INH
  - ❖ Valproic acid
  - ❖ Phenytoin
  - ❖ Carbamazepine
  - ❖ Propylthiouracil
  - ❖ Halothane
  - ❖ Nitrofurantoin
  - ❖ Herbal drugs

Wilson disease

- It is an autosomal recessive disorder.
- The condition is due to mutations in the Wilson disease protein (*ATP7B*) gene
- May present as ALF in young adults
- ALF due to wilson disease should be suspected in any pt with.

  -ALF + Coomb's negative hemolytic anemia.


Hepatomegaly
Jaundice
Acute hepatitis
Fulminant hepatic failure
Portal hypertension: bleeding varices
Cirrhosis
Liver
Proximal renal tubular dysfunction
Renal
Bone
Arthritis
Rickets
Wilson's Disease
Cardiac
Haem
Hemolysis
Central nervous system
Deterioration in school performance
Behavioral changes
Inco-ordination (handwriting deteriorates)
Resting and intention tremors
Dystonia
Dysarthria
Excessive salivation
Mask-like facies
Dysphagia
Eye
Kayser Fleischer rings

PATHOPHYSIOLOGY

Mechanisms of hepatic injury

- **Immune mediated hepatocellular injury**
  - Viral infections
  - Drug hepatotoxicity (dihydralazine, halothane)
- **Direct hepatocellular injury**
  - Hepatotrophic virus family-HAV, HBV, HCV
  - Toxic or reactive metabolites- acetaminophen
  - Toxic metabolites of compounds-metabolic diseases
- **Ischemic hepatocellular Injury**
  - Shock states, SIRS

Effects are:

- Impaired hepatocyte regeneration
- Altered parenchymal perfusion
- Endotoxemia
- Decreased hepatic reticulo-endothelial function

Hepatic failure

Pathophysiology ctd…

CEREBRAL EDEMA

- development of cerebral edema/intracranial hypertension is the major cause of morbidity and mortality of patients suffering from acute liver failure- ?etiology- multifactorial
- increase of intracranial blood volume and cerebral blood flow- because of disruption of cerebral autoregulation- elevated systemic concentrations of nitric oxide, bacterial endotoxin, tumor necrosis factor-alpha (TNF-a), and interleukin-1 (IL-1) and -6 (IL-6)
- Accumulation of glutamine in astrocytes leads to swelling and edema.

MULTISYSTEM ORGAN FAILURE

- Hyperdynamic circulation state leads to low systemic vascular resistance (mimics sepsis) circulatory insufficiency and poor organ perfusion may initiate FHF or lead to complications

Pathophysiology ctd…

ENCEPHALOPATHY

Wide variety of agents, including ammonia, free fatty acids, mercaptans, phenols, bile acids and aromatic amino acids combine to produce hepatic encephalopathy by several different mechanisms:

- direct cellular effect
- indirect: cause metabolic derangement
- conversion to false neurotransmitters
- alteration of permeability of BBB: allows entry of toxic metabolites into the brain and thus contributes to cerebral oedema - major cause of death: present in 32% of patients at PM.

CLINICAL PRESENTATION

HISTORY

- Duration of illness.Onset of jaundice and encephalopathy.
- Risk factors for hepatitis: eg- street food, sanitation, BT, surgery.
- Drug & immunization history
- H/O any bleeding episode.
- Features of hepatic encephalopathy-Initially reduced alertness and poor concentration, progressing through behavioural abnormalities such as restlessness and aggressive outbursts, to drowsiness and coma

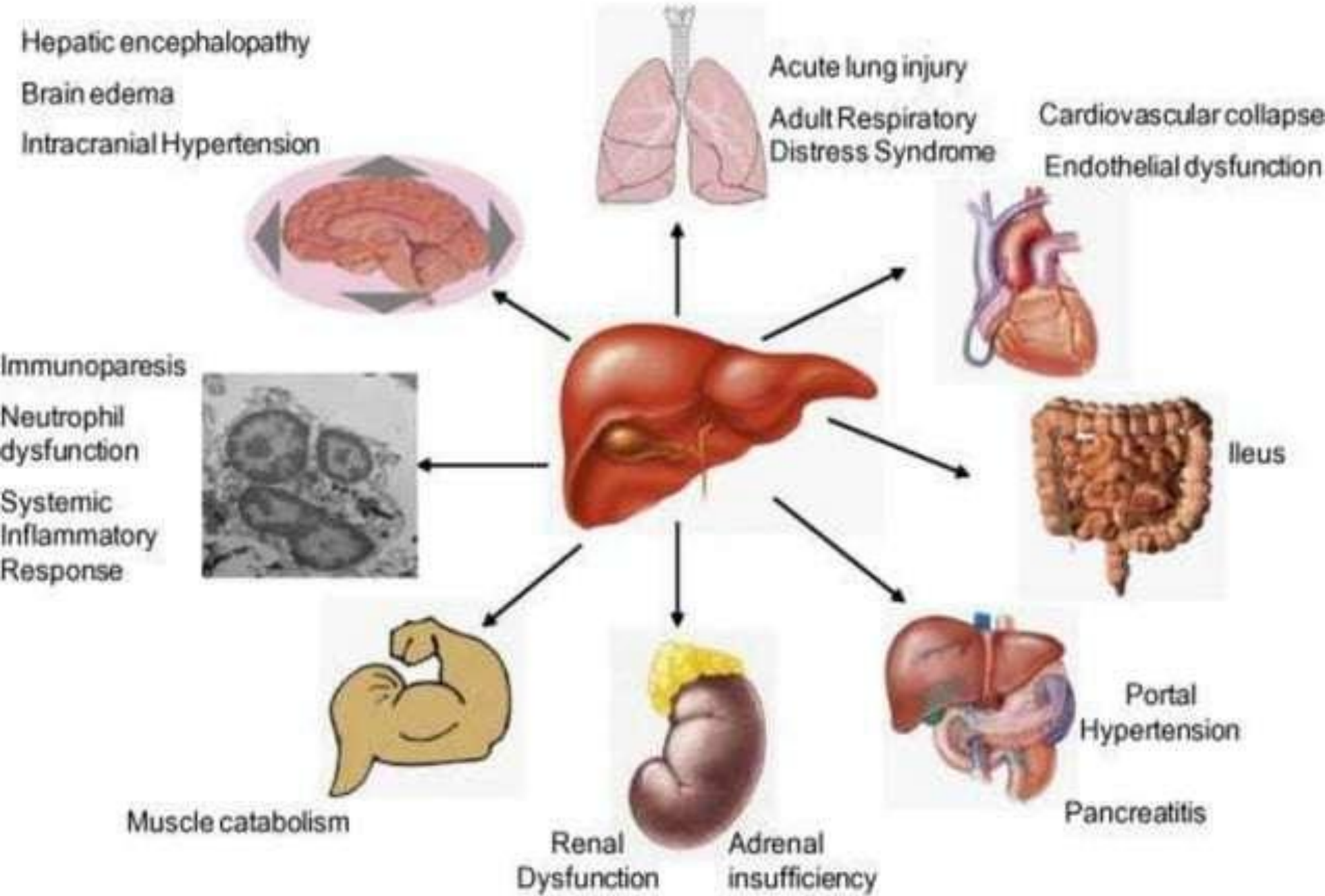
- weakness, nausea and vomiting
- Right hypochondrial discomfort
- Family history
- Systemic enquiry

Physical examination

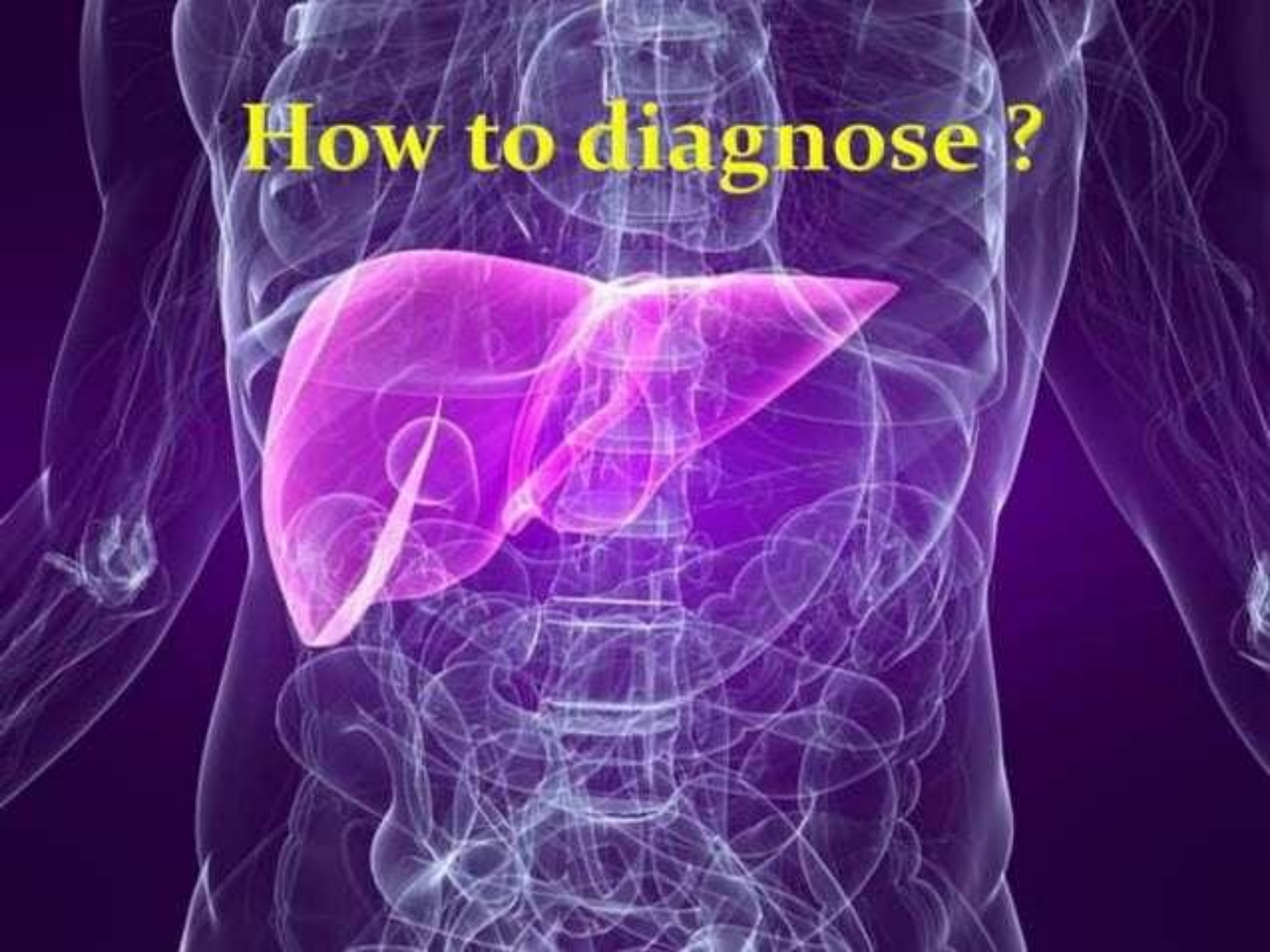
- Jaundice
- Anaemia
- Vital signs: Hypotension, tachycardia, tachypnoea.
- Mental status.
- Hepatomegaly –uncommon, liver usually N size - smaller
- Splenomegaly
- Stigmata of chronic liver disease- ALF 2' to compensated diseases(caput medusa, spider nevi, spider angiomata, ascites, contractures, palmar erythema)
- Ascites
- Features of raised ICP- unequal n fixed pupils
- flapping 'hepatic' tremor
- profuse sweating, local or general myoclonus, focal fits or decerebrate posturing
- Papilloedema occurs rarely and is a late sign
- Fetor hepaticus

- Cardiovascular- hypotension with low SVR
- Pulmonary- resp alkalosis, impaired peripheral O2 uptake, pulmonary edema, ARDS
- GIT- GI bleeding(decreased clotting factors & platelets, DIC), pancreatitis due to ischaemia
- Renal-ATN, hepatorenal syndrome, hyponatremia, hypokalemia, hypophosphatemia
- Hematology- coagulopathy, infection
- Endocrine- hypoglycemia, adrenal insufficiency

Systemic Manifestations of Acute Liver Failure



How to diagnose ?



Investigation

a. Complete blood count: Leucocytosis , thrombocytopenia

b. LFT: Serum bilirubin ↑

- ALT: raised
- AST :raised esp in paracetamol- 100-500x normal
- Alkaline phospatase: may be normal or ↑
- PT and INR:prolonged, to see the severiry of coagulopathy and to asses prognosis.
- Albumin: usually normal unless in prolonged course

c. Blood grouping and cross matching

d. blood cultures

e. Blood sugar levels

Inv.continued

E . Biochemical test:U/E/Cs

- S. Electrolytes- Hypokalaemia and others.
- S. Glucose- Hypoglycaemia.
- S. Creatinine
- S. Ammonia- ↑
- S. Calcium , Phosphate, Magnesium
- S. lactate: At 4 hours(>3.5) or at 12 hours(>3) are early predictors of outcome in acetamenophan induced ALF.

F . Blood gas analysis.

To detect cause

G . Serological marker

- Anti HAV IgM
- HBsAg, AntiHBcore IgM antibody
- Anti HEV IgM
- Screening for other viruses- HSV, CMV etc.

Immunological test

- S. immunoglobulin, ANA, anti SMA, antiLKM1

J . Serum acetamenophen level

K . USG of HBS

L . Screening for metabolic disease.

M . EEG- encephalopathy has xteristic pattern

N . Liver biopsy :in auto-immune, metabolic causes of liver failure. Transjugular, Percutaneous CI due to coagulopathy

Screening for Wilsons disease

- Slit Lamp Eye examination for KF ring & cataract
- Serum copper levels
- Serum Ceruloplasmin
- 24 hours urinary copper

Management of ALF

- **Medical:** To maintain physiological functions of liver
  - Supportive care
  - Specific treatment
- **Surgical:**
  - Extracorporeal systems
  - Liver transplantation

Supportive care.

- quiet environment.
- ICU- hemodynamic n ventilatory support
- Avoid sedation and stimulation.
- Fluid restriction- 60-80% of daily requirement.

  Choice of fluid: 0.225% Nacl in 10% dextrose.
- Prevention of hypoglycemia(maintain glucose level >4 mmol/L).

- IV H_2 blocker or PPI.
- Antibiotics: Cefuroxime, amoxicillin, fluconazole or

Cefotaxime+ flucoxacillin

- Anaemia should be corrected to ensure maximum oxygen supply to tissue.
- Lactulose-metabolized to organic acids by colonic bacteria.Reduces ammonia prodn by bacteria & traps ammonia in acidic intestinal contents. Avoid diarrhea!
- IV N-acetylcysteine benefits pts after paracetamol overdose even with encephalopathy and coagulopathy- same dose as PCM

- early intubation, as soon as gag reflex lost or patient develops grade III/IV encephalopathy
- IV midazolam or propofol can be used for intubated patients who require sedation. Sedatives should not be used in patients who are not intubated.
- Immunosuppressive RX- steroids may help in auto-immune hepatitis
- Antivirals e.g pleconaril for enteroviral hepatitis,Acyclovir for HSV.
- Consider plasma exchange in Wilson's disease
- AminataPhylloides poisoning- Gastric lavage, High dose Pen G
- Budd Chiari- Ttansjugular intrahepatic portocaval Shunt and Anticoagulation

- Management of complications!

Close Monitoring

- Continous O_2 saturation
- Clinical status: Pulse & BP hourly, liver size 12 hrly
- Strict input-output chart : Avoid fluid overload
- CVP, Foley's and arterial line : MAP >60 mmHg
- Frequent evaluation of blood glucose and neurological status.
- 12 hourly electrolyte and coagulation studies

Plasmapharesis

- Blood purification procedure

- High volume plasmaphoresis associated with rise in conscious level, haemodynamics and reduction in arterial ammonia concentrations
- Disadv. is that possible hepatic growth factors are removed along with hepatotoxic mediators

Liver transplantation

- Pts with anticipated survival of < 80% and no contraindication to transplantation should be placed on waiting list as soon as possible.
- Multiorgan failure is major C/I to transplantation.
- Cerebral perfusion pressure <40 mmHg is a relative contraindication.

Table 8.7. King's College Hospital criteria for liver transplantation in acute liver failure [58]

Acetaminophen (paracetamol)

pH < 7.30 (irrespective of grade of encephalopathy)

or

Prothrombin time >100 s (INR > 7) and serum creatinine >300 μmol/l in patients with grade III or IV encephalopathy

Non-acetaminophen patients

Prothrombin time >100 s (INR > 7) (irrespective of grade of encephalopathy)

or

Any three of the following variables (irrespective of grade of encephalopathy)

- age <10 or >40 years
- aetiology: non-A–E hepatitis, 'viral' hepatitis no agent identified, halothane hepatitis, idiosyncratic drug reaction
- duration of jaundice before onset of encephalopathy >7 days
- prothrombin time >50 s (INR > 3.5)
- serum bilirubin >300 μmol/l

Management of complications

ALF: common complications

- Encephalopathy
- cerebral edema
- Raised ICP
- Infection
- Coagulapathy
- Hypoglycemia
- Dyselectrolytemia
- Acid base disturbance.
- Multi-organ failure

Hepatic encephalopathy

- Complex neuropsychiatric manifestation in a pt with liver disease, after exclusion of brain disease.
- Ranges from altered mental status to coma.
- Failure of liver to detoxify toxins from intestines
- Pathogenesis multifactorial but ammonia major factor
- Rapid progression to coma, seizures and decerebrate rigidity. Ass with cerebral edema in late stages.
- Death due to both herniation and hypoxia

Stages of encephalopathy

Encephalopathy	Symptoms	Signs
Stage I (mild)	Alert, euphoric, occasionally depression. Poor concentration,Insomnia slow mentation and affect,reversed sleep rhythm.	Trouble drawing figures, performing mental task, EEG-normal
Stage II	Drowsiness, lethargic, inappropriate behavior, disorientation, slurred speech	Asterexis,fetor hepaticus,EEG slowing of wave.
Stage III	Stuporous but easily arousable, marked confusion/disorientation, incoherent speech	Asterexis, hyperreflexia, rigidity, EEG-Triphasic wave
Stage IV	Coma, unresponsive but may respond to painful stimulus.	Planter extensor, Rigidity ,Areflexia, Flacidity.EEG-delta wave.

Aggravating factors of HE

- GI bleeding
- Hypovolemia
- Hypokalemia
- Hypoglycemia
- Sedatives
- Uremia
- Sepsis
- High protein diet
- Constipation
- Paracentesis

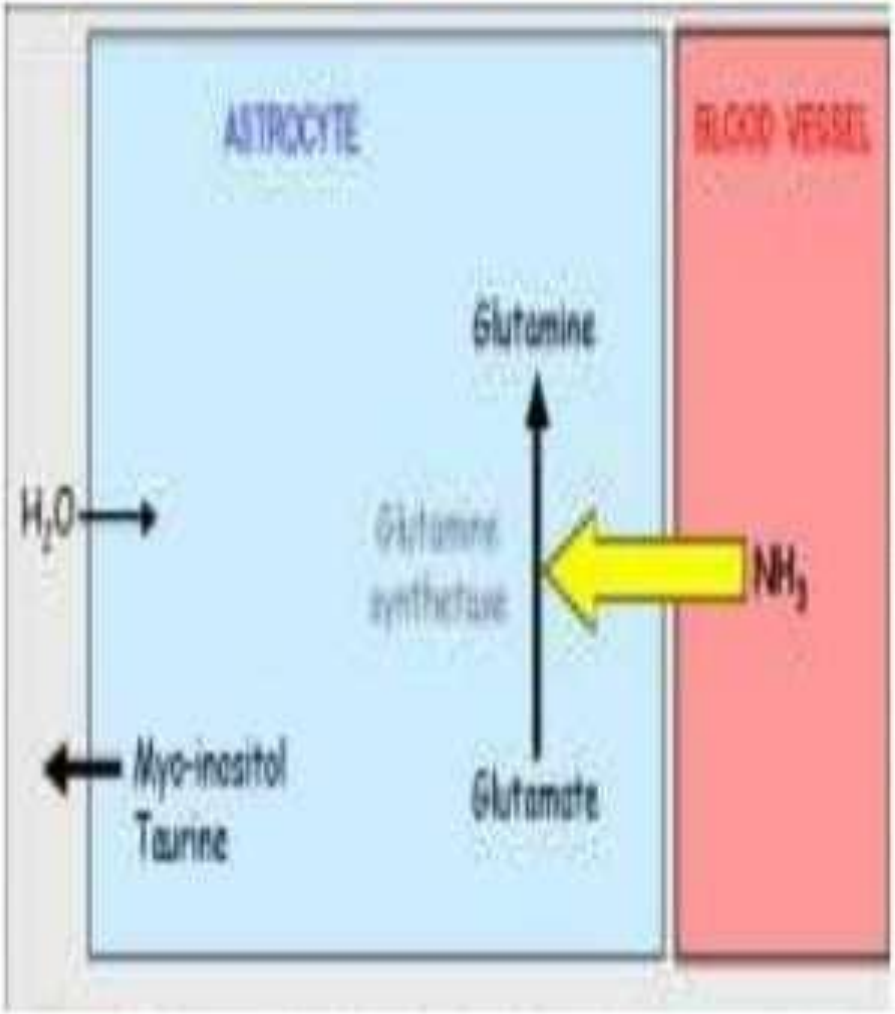
Encephalopathy

Treatment

- Low protein diet
- Lactulose 10-50ml 2-4 hourly
- Acid enema: Lactulose enema, vinegar-can be given 6 hourly
- Treat sepsis, bleeds and electrolyte imbalance.
- Avoid sedation esp benzodiazepines
- Intubate in Grade 3 and 4- risk aspiration
- Oral Neomycin, Metronidazoleor Vancomycin to decrease colonic conc. Of ammoniagenic bacteria

- Flumazenil : Short lived results.

Cerebral edema



- Occurs in 80% of pts who eventually develop stage 4 encephalopathy.
- Hyperammonia induces accumn of glutamine within the cells
- Diagnosis usually difficult,usually at postmotrtem
- Antemoterm dx suggested by deterioration of conciousness,neurological signs eg increased muscle tone,myoclonus,focal seizures.
- Signs of increased intracranial pressure

Cerebral Edema

- Treatment
- Head elevated 20-30° ↓ ICP
- IV mannitol
- Hypertonic saline
- Hyperventilation may help ↓ ICP
- Hypothermia ↓ ICP
- Avoid hyperthermia.

BLEEDING

- Due to inability of liver to synthesize the clotting factors, abnormality in platelet structure and function, mild DIC
- Pts have a tendency to bleed at venupuncture sites

Treatment

- Inj. Vitamin K
- Fresh frozen plasma (FFP)- necessary if spontaneous bleeding occurs (5% pts). Prophylactic use not assocd with reduction in morbidity or mortality. Avoidance of FFP has advantage that PT remains valid prognostic marker.
- Platelet transfusion –Indicated in severe thrombocytopenia;If platelet<10000/mm^3 or platelet count<50000 with bleeding. Fresh whole blood transfusion.
- Recombinant factor VII.

ELECTROLYTE DISTURBANCES

- Hyponatremia

- Hypokalemia – decreased dietary intake , chronic illness , secondary hyperaldosteronism , frequent GI losses .
- Hypophosphatemia – due to amount of regenerative liver mass as phosphate be a substrate for various kinase enzymes that phosphorylate proteins for liver regeneration

- Hypoglycemia – due to impaired hepatic glucose release due to glycogen depletion (failure of hepatic gluconeogenesis) , high plasma insulin levels due to decreased uptake

ACID BASE IMBALANCES

- Respiratory alkalosis – due to hyperventilation – direct stimulation of respiratory centre by toxic substances
- Metabolic acidosis-due to lactate accumulation

Treatment

- Volume expansion.
- Dopamine infusion.
- $NaHCO_3$.
- Mechanical ventilation for M. acidosis.
- Plan for liver transplantation.

SEPTICAEMIA

- Gram-positive (in 2/3rd cases):
  - Staphylococcus
  - Streptococci
- Gram-negative (in 1/3rd cases):
- Fungal infection

Septicemia ctd...

- Septic workup : culture blood, sputum & urine
- Cefuroxime, Amoxycillin, Fluconazole.
- Fungal infection : IV amphoterecin .
- We use fluconazole orally.
- Iv cefuroxime and flucloxacillin
- late rise in INR is usually a pointer to uncontrolled sepsis and anti-fungals should be introduced if a comprehensive regime of antibacterials is already being used.

RENAL COMPLICATIONS

- Renal failure may occur in 50% pt. with ALF especially in paracetamol poisoning.

Due to

- Hepatorenal syndrome.
- ATN by due to sepsis, endotoxemia, bleeding hypotension.
- Directly by nephrotoxic drug.

- Treatment
- Dopamine infusion.
- Correct fluid overload, acidosis & hyperkalemia.
- Avoid nephrotoxic drugs.
- Dialysis

HEPATORENAL SYNDROME

Development of renal failure in pts with advanced liver disease.

-common, associated with Spontaneous Bacterial Peritonitis

-may rapidly progress

-often associated with cerebral edema

-functional, due to vasoconstriction of renal vessels

-plasma urea/creat unreliable

RX: Ideal Rx is liver transplant

HEPATOPULMNARY SYNDROME

Syndrome of shortness of breath and hypoxemia coased by vasodilation in lungs in patients with liver disease.

-decreased vascular resistance

-pulmonary edema.

-Hypoxaemia

-hyperventilation

-aspiration with progression

Prognosis

Without transplantation

- up to 80% die
- in specialist units approx 30% survive

Following transplantation

- up to 80% 1 year survival.
- long term prognosis is good

- High mortality
  -severe encephalopathy
  -EEG abnormalies
  -severe coagulopathy ptt>100sec
  -age <5yrs and >40yrs
  -halothane,paracetamol
  -major complications eg ARF,GI h'age

Non acetaminophen predictors

Poor prognosis in;

Age younger than 10yrs or >40yrs

Halothane hepatitis or idiosyncratic drug reactions

Jaundice present for > 1wk before onset of encephalopathy

Prothrombin time > 50s

Serum bilirubin > 300mmol/L

Chronic liver disease

Cirrhosis

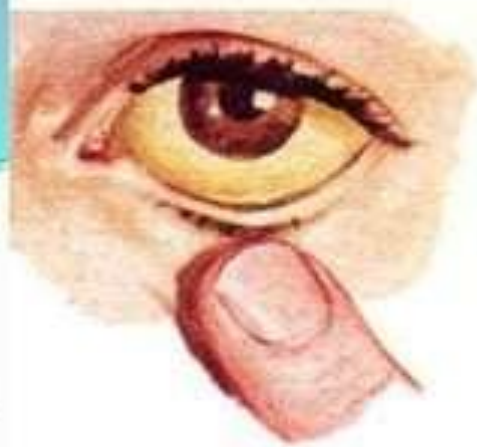
- A consequence of CLD
- Characterized by replacement of liver tissue by fibrosis & regenerative nodules
- Leads to irreversible loss of liver function & its complications
- Micronodular- alcohol or Macronodular- chronic viral hepatitis
- Decompensated= jaundice, variceal bleed, encephalopathy and ascites

Causes

- Alcoholic liver disease
- Chronic viral hepatitis- C or B
- Non-alcoholic steatohepatitis
- Autoimmune hepatitis
- PBC & PSC
- Hemochromatosis, Wilson's disease
- α1-antitrypsin deficiency
- Cystic fibrosis

Stigmata of CLD

- Muscle wasting
- Scratch marks
- Pallor, jaundice
- Parotid enlargement
- Xanthelasma
- Clubbing
- Palmar erythema
- Dupuytren's contracture
- Spider nevi/angiomata
- Petechiae, purpura
- Decreased body hair
- Gynecomastia
- Testicular atrophy
- Caput medusae
- Edema, ascites
- Splenomegaly
- Asterixis
- Fetor hepaticus



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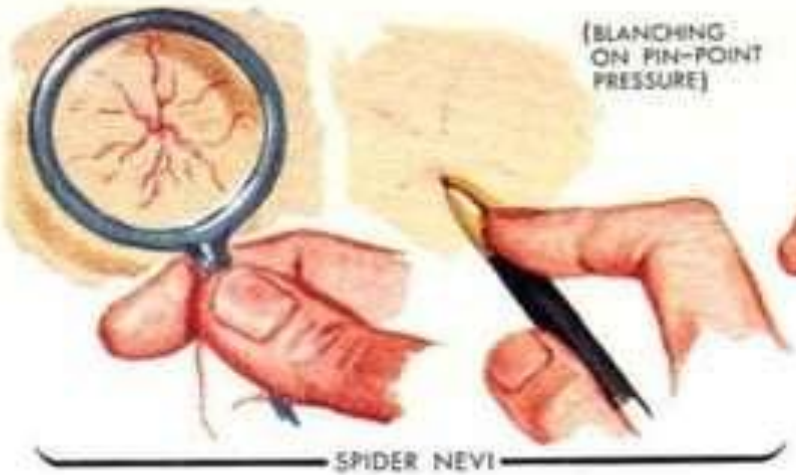
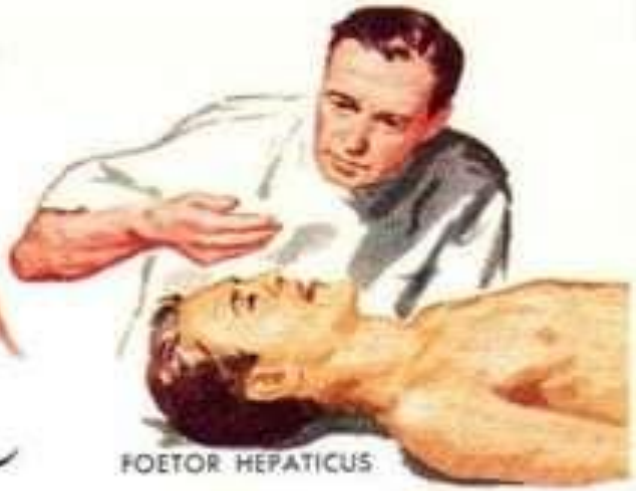
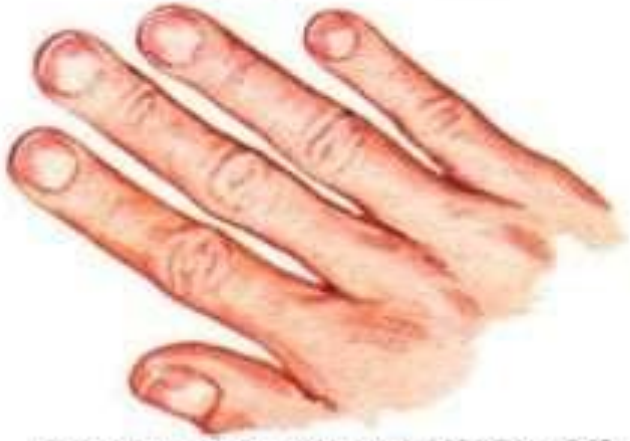


SPIDER NEVI

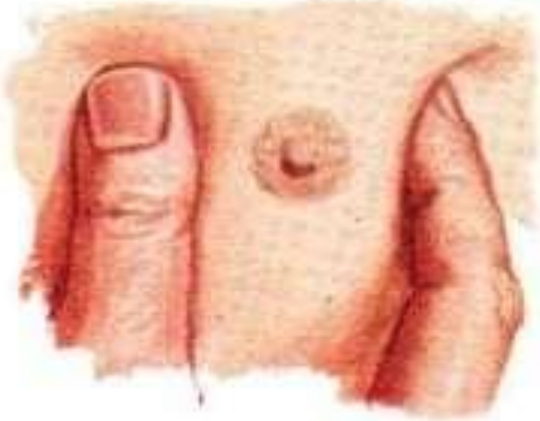


FOETOR HEPATICUS

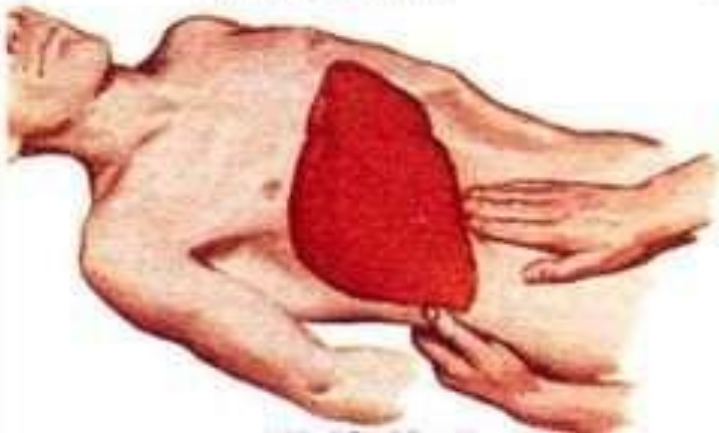
PALMAR ERYTHEMA

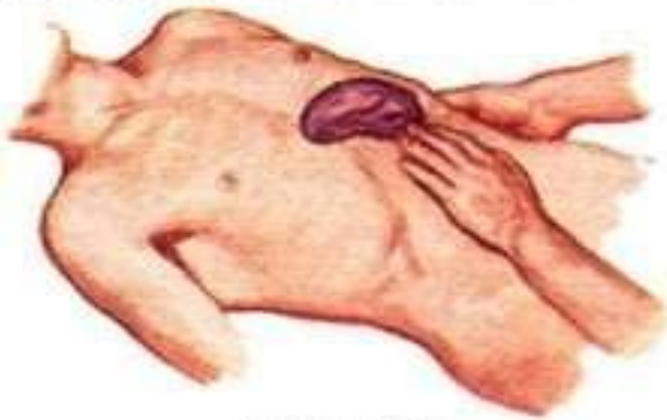


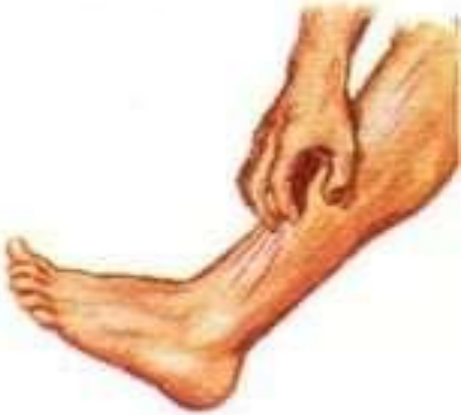
FINGER CLUBBING AND NAIL BED CHANGES



GYNECOMASTIA







Investigation

- Liver biopsy- gold standard, but not always necessary
- Deranged LFT-
  - ± elevated AST, alkaline phosphatase, ALT
  - Increased bilirubin
  - Low albumin, increased globulins
  - Increased PT/INR
  - Thrombocytopenia
  - Low sodium
- Ultrasound- shrunken liver, ± portal HT/HCC

Staging of CLD

- Based on Child-Turcotte-Pugh scoring system
- Includes- each given score of 1-3
  - Ascitis
  - Encephalopathy
  - Bilirubin
  - Albumon
  - PT/INR
- Class- total score
  - A- 5-6
  - B- 7-9
  - C- 10-15

Treatment

- To retard progression & reduce complications
- Abstinence from alcohol
- Vaccination- Hep A & B
- Treat underlying cause

Complications

- Ascites
- Spontaneous bacterial peritonitis- SBP
- Variceal bleed
- Hepatic encephalopathy
- Hepatorenal syndrome
- Hepatocellular carcinoma- HCC

Ascitis

- Diagnostic paracentesis- SAAG >1.1
- Causes
- Portal HT
- Hypoalbuminemia- nephrotic, malnutrition, protein loosing enteropathy.
- Raised renin-angiotensin-aldosterone levels causing Na retention by kidneys

Ascitis- treatment

- Salt ± fluid restriction
- Diuretics- Spironolactone ± Furosemide
- Large volume paracentesis-
  - With massive or refractory ascitis
  - >5 lit. fluid removed in one go
  - Albumin- ~8 gm/lit. fluid removed
  - Avoid hepatorenal syndrome
- TIPS- transjugular intrahepatic portosystemic shunt
  - For refractory ascitis or refractory variceal bleed
  - Preferred for short duration, pending liver transplant
  - Increases risk of hepatic encephalopathy, shunt occlusion/infection

Spontaneous Bacterial Peritonitis

- s/s-
  - Abdominal pain, fever,worsened ascitis & encephalopathy
- Dx- paracentesis
  - PMN >250/microlitre
  - Ascitic fluid culture- bedside,commonly Gram –ve bacteria
- Rx- Cefotaxime/Ciprofloxacin
- Prophylaxis- Ciprofloxacin/Co-trimoxazole
- Prognosis- 30% mortality during hospital stay & 70% within 1 year

Variceal bleed

- Varices- dilated submucosal veins, in esophagus or stomach
- Cause- portal HT
- Causes ~80% of UGI bleed in Chronic Liver Dse
- Risk factors for bleed-
  - Size of varices
  - Severity of liver disease
  - Continued alcohol intake
  - UGIE- wale markings, hematocystic/red spots on varix
- Dx- UGI Endoscopy

Management

- Acute-
  - Resuscitation
  - FFP, platelets, vit. K
  - Terlipressin/octreotide
  - Lactulose

  - Banding/sclerotherapy
  - Balloon tamponade
  - TIPS
  - Surgery

- Prevent rebleed-
  - Band ligation- over repeated sessions
  - Non-selective β- blockers- Propanolol/Nadolol
  - TIPS- for recurrent bleed or bleed from gastric varices
  - Surgery- portosystemic shunts
  - Liver transplantation

Hepatic encephalopathy

- Confusion→drowsiness→stupor→coma
- Ammonia is an identified/measurable toxin
- Precipitants-
  - GI bleed
  - Constipation
  - Alkalosis, hypokalemia
  - Sedatives
  - Paracentesis→hypovolemia
  - Infection
  - TIPS
- Dx- clinical- s/s of CLD
  with asterixis & altered sensorium

Management

- Correct underlying precipitating factor
- Avoid sedatives
- Restrict dietary protein intake
- Lactulose- 2-3 loose stools a day
- Oral antibiotic- Metronidazole, Rifaximin, Neomycin

Hepatorenal syndrome

- Occurs in patients with advanced CLD & ascitis
- Marked by renal impairment in the absence of any renal parenchymal disease or shock
- Oliguria, hyponatremia & low urinary Na accompany raised creatinine
- Albumin infusion, with vasoconstrictors (norepinephrine, terlipressin/ornipressin, octreotide) may help
- Liver transplantation is Rx of choice

Hepatocellular carcinoma

- Associated with cirrhosis in ~80%
- Suspect if- worsening of CLD, enlarged liver, hemorrhagic ascitis, weight loss
- Dx-
  - CT/MRI with contrast-
  - Raised AFP- α-fetoprotein
  - Liver biopsy
- Rx-
  - Early-resection
  - Advanced- liver transplantation or local palliative treatment
- Screening- US & AFP q 6 months

Liver transplantation

- Donor, cost, technical expertise
- Option in ESLD
- GVHD, recurrence

- THANK YOU