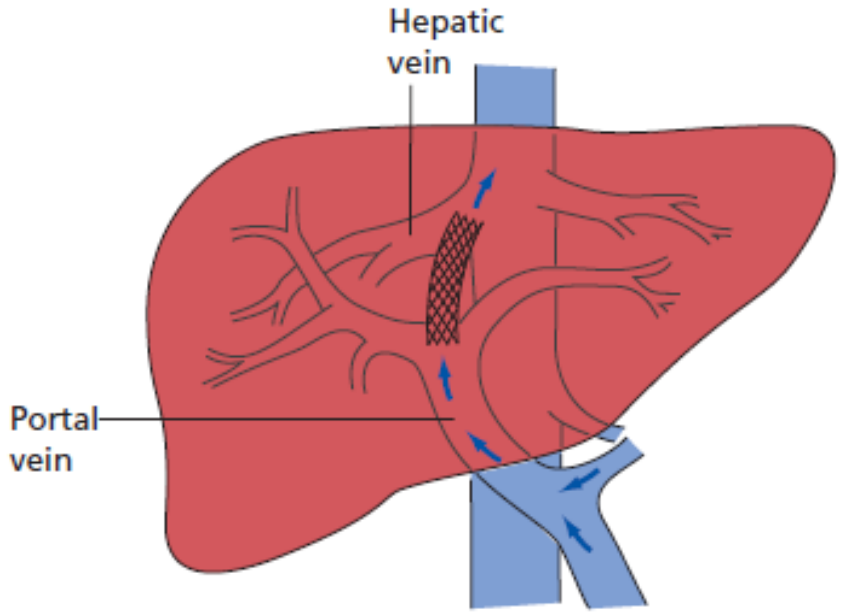
Acute Liver Failure



Learning outcomes

LO1: Definition of liver failure

LO2: Classification of liver failure

LO3: Causes of liver failure

LO4: The Clinical features of liver failure

LO5: The diagnosis of liver failure

LO6: Treatment of liver failure

LO1: Acute Liver Failure

- Acute liver failure describes the clinical syndrome of severe impairment of liver function -

✓Encephalopathy

✓Coagulopathy

✓ jaundice

- Within 6 months of the onset of symptoms.

LO1:

- The acute onset of liver disease with no known evidence of chronic liver disease.
- Biochemical and/or clinical evidence of severe liver dysfunction:
- Hepatic-based coagulopathy – prothrombin time [PT] ≥15 seconds or international normalized ratio [INR] ≥1.5 that is not corrected by parenteral vitamin K in presence of clinical hepatic encephalopathy
- PT is ≥20 seconds or INR is ≥2.0 in presence or absence of HE .

LO2: Classification

	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

LO2:

- An alternative classification

➢ **Fulminant**

- liver failure - time from jaundice to encephalopathy less or more than 2 weeks

➢ **Sub-fulminan**t

- Late onset liver failure describes encephalopathy developing more than 8 weeks (but less than 24 weeks) after the first symptoms

LO3: major causes of fulminant hepatic failure : the ABC's

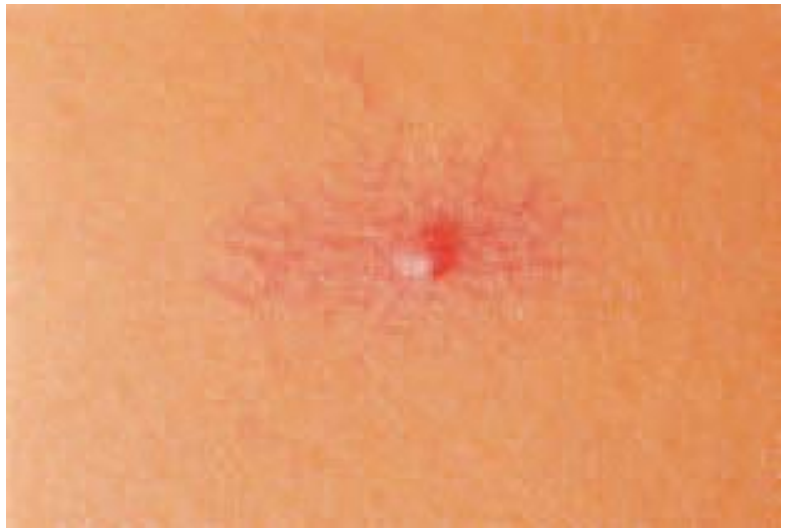
A.	Acetaminophen, hepatitis A, autoimmune hepatitis
B.	Hepatitis B
C.	Hepatitis C, cryptogenic
D.	Hepatitis D, drug
E.	Esoteric causes – Wilson's disease, Budd-Chiari syndrome
F.	Fatty infiltration – acute fatty liver of pregnancy, Reye's syndrome

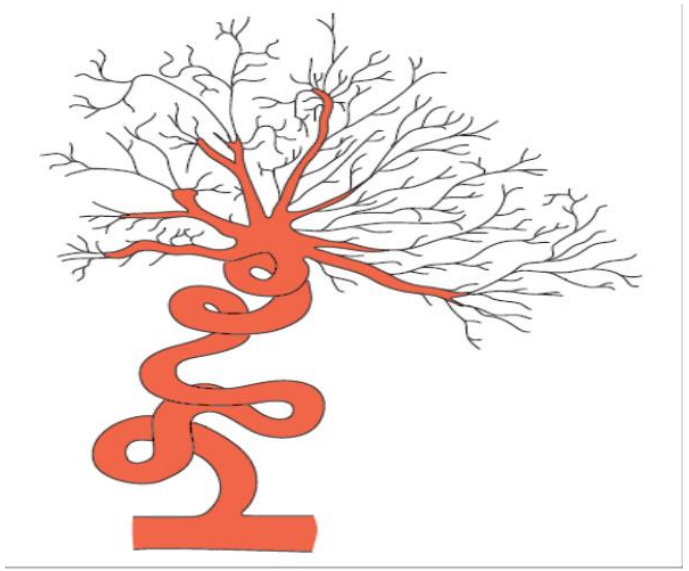
LO4: Clinical features

- The patient, previously having been well, typically develops non-specific symptoms such as nausea and malaise.
- Progressive Jaundice.
- Vomiting is common
- Abdominal pain .
- Fetor hepaticus
- Rapid decrease in liver size without clinical improvement
- Ascites
- Tachycardia, hypotension, hyperventilation and fever are later features
- Later coma and encephalopathy features

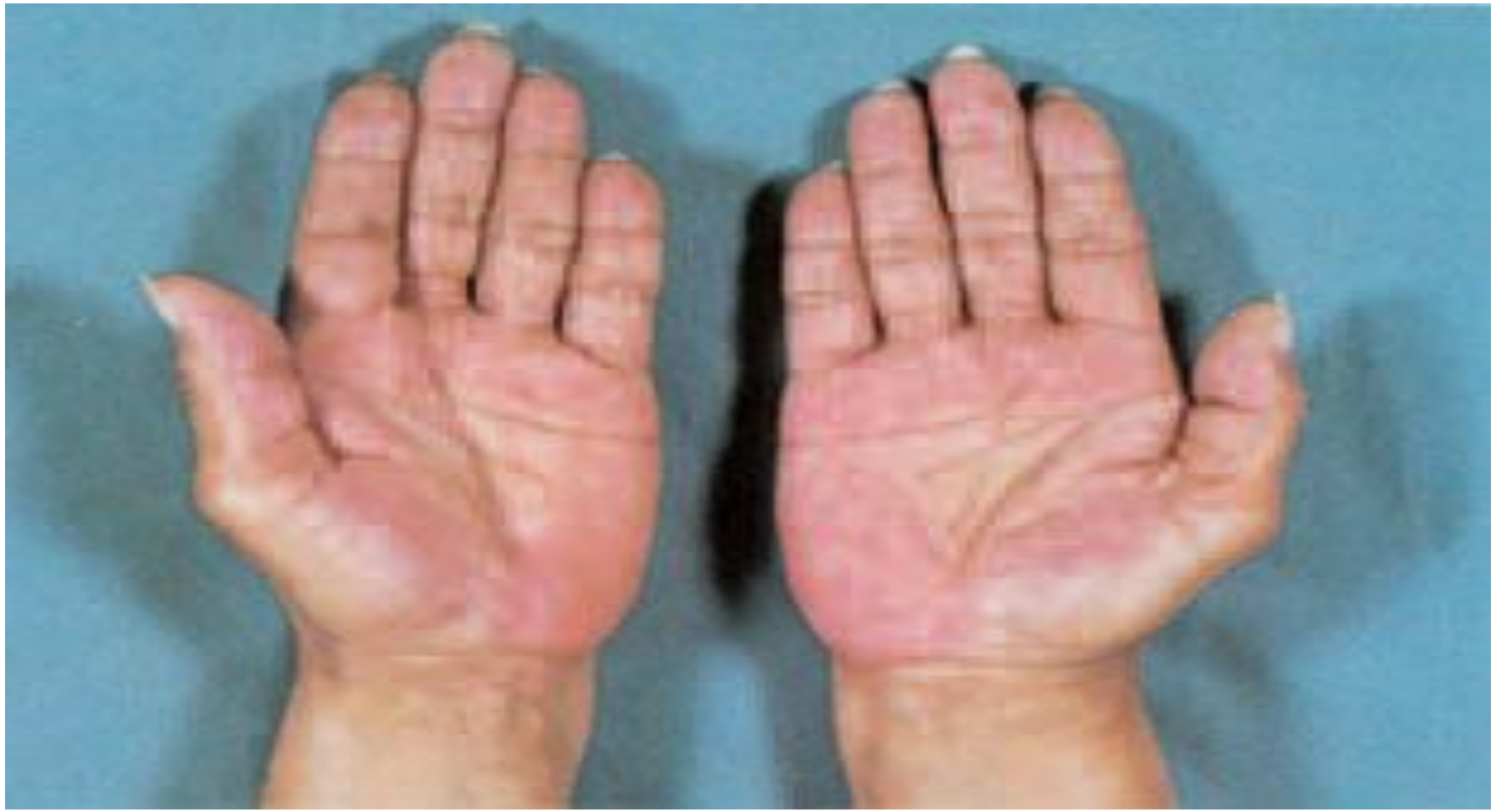
LO4:
Skin changes - Vascular spiders

Common sites are the necklace area, the face, forearms and dorsum of the hand





LO4: Palmar erythema (liver palms)



LO4:

Hepatic encephalopathy

The brain is exposed to increased levels of ammonia, neurotransmitters and their precursors because of failed hepatic clearance.

- Neurological and psychiatric components.

- Features of encephalopathy can be separated into changes in consciousness, personality, intellect and speech.

LO4:

- Disturbed consciousness with disorder of sleep is usual.

- Hypersomnia appears early and progresses to reversal of the normal sleep pattern.
- Speech is slow and slurred and the voice is monotonous

- The most characteristic neurological abnormality is the ‘flapping’ tremor (asterixis).

- Coma at first resembles normal sleep, but progresses to complete unresponsiveness.

LO5: Diagnosis

Haematology

- The prothrombin time to the assessment of the severity of the clinical situation, and its progress.
- Haemoglobin and white count are obtained.
- A falling platelet count may reflect disseminated intravascular coagulation.

LO5:

Biochemical

- Serum bilirubin
- Serum Albumin – initially normal but later low albumin carries poor prognosis
- Transaminases – of little prognostic values as levels tends to fall as condition worsens
- Blood Glucose
- Blood Urea
- Serum Creatinine
- Serum Electrolytes

LO5:

Virological markers

- Serum HBsAg
- IgM Anti HBc
- IgM anti HAV
- Anti HCV
- HCV RNA

LO5:

Encephalopathy

- Cerebrospinal fluid - usually clear and under normal pressure , cell count is normal

- EEG changes occur very early even before psychological or biochemical disturbances.

- CT scan to show cerebral oedema and cortical atrophy

LO6: Treatment

General measures

- Volume resuscitation should be carried out aggressively
- Fluids should be glucose
- Strict input output charting

LO6:

Treatment of Hepatic Encephalopathy

- Treatment broadly divides into three areas.

1) Identification and treatment of the precipitating cause.
2) Intervention to reduce the production and absorption of gut-derived ammonia and other toxins.

- Involves reduction and modification of dietary protein,
- Alteration of enteric bacteria and the colonic environment -antibiotics, oral lactulose
- Stimulation of colonic emptying - enemas, lactulose

3) Agents to modify neurotransmitter balance directly- bromocriptine, Flumazenil (benzodiazepine antagonist) limited clinical value at present.

LO6:

Treatment of cerebral oedema

- Head should be elevated to 30 degrees
- High levels of PEEP should be avoided – it may increase hepatic venous pressure and intracranial pressure
- Mannitol bolus of 0.5 g/kg as 20 % solution over 15 minutes – can be repeated if serum osmolality less than 320 mOsm/L
- Other methods 3% hypertonic saline
- **STEROIDS ARE NOT INDICATED** IN TREATMENT OF cerebral oedema in ALF – as it may complicate infection and cause gastric erosions

LO6:

Treatment coagulopathy

- Iv vitamin K to correct any reversible coagulopathy
- FFP – to be given in case of haemorrhage or if coagulopathy is severe (PT>60sec)
- Thrombocytopenia to be corrected
- Prophylaxis for GI bleed – administration of PPI , H2 blocker

• Treatment Hepatorenal syndrome

- is the most common cause of renal insufficiency in ALF
- Secondary to renal vasoconstriction
- Primarily focused on decreasing splanchnic circulation –

1. Vasoconstrictors – Terlipressin
2. Alpha agonist- nor-epinephrine , medodrine

   Very effective in reversal of functional renal insufficiency

LO6:

Liver transplantation