Hepatic Failure

Definitions

Hepatic failure = liver loses the ability to regenerate or repair and decompensation occurs. Characterised by: encephalopathy, haemorrhagic diathesis, ascites and jaundice.
Fulminant hepatic failure = encephalopathy + Factor V <50% of norm <2w after jaundice onset.
Acute hepatic failure = decompensation occurring ≤ 8 weeks of the onset of liver disease,
Subacute fulminant hepatic failure = decompensation with encephalopathy + Factor V <50% of normal 2 weeks to 3 months after onset of jaundice.
Late onset hepatic failure = decompensation 8 to 26 weeks after onset of liver disease.
Chronic decompensated hepatic failure = ≥ 6 months from onset of liver disease.
Chronic hepatic failure = Chronic liver disease (e.g. cirrhosis) complicated by one or more characteristic features of liver failure.

Epidemiology

M=F.

Causes

- Infectious
  - Viral hepatitis (A, B[+D], C, E), Adenovirus, EBV, CMV & viral haemorrhagic fevers
  - Systemic infection
- Drugs
  - Paracetamol, co-amoxiclav, ciprofloxacin, doxycycline, erythromycin, isoniazid, nitrofurantoin, halothane, statins, cyclophosphamide, methotrexate, disulfiram, gold, propylthiouracil, allopurinol, colchicine, etc.
  - Poisoning by amanita phalloides, paraquat, P, CCl_4 & other organic solvents
  - Illicit drugs including Ecstasy and cocaine
  - Herbals: ginseng, pennyroyal oil, Teucrium polium, chaparral or germander tea
- Autoimmune liver disease
  - Idiopathic
  - IBD
  - PBC
- Deposition
  - Haemachromatosis
  - Wilson's disease
  - Glycogen storage diseases
- Other
  - Hepatocellular carcinoma or metastatic carcinoma
  - Reye's syndrome
  - Acute fatty liver of pregnancy
  - Ischaemia, veno-occlusive disease, Budd-Chiari syndrome
  - Metabolic disease (esp infants): alpha-1-antitrypsin deficiency, fructose intolerance, galactosaemia and tyrosinaemia
  - Idiopathic in 15%.

History

Encephalopathy may limit history from patient. Jaundice, EtOH use, medications, exposure risk for viral hepatitis, toxin ingestion, past & family history of liver disease.

Examination

- Jaundice, other stigmata (asterixis, spider naevi, palmar erythema etc.) absent if acute.
- Hepatic encephalopathy is graded from 0 to 4:
    - *Grade 0* - Subclinical; normal LOC, minimal changes in cognition/co-ordination
    - *Grade 1* - Mild confusion, euphoria or depression, decreased attention, slowing of ability to perform mental tasks, irritability, disorder of sleep pattern.
    - *Grade 2* - Drowsiness, lethargy, deficits in ability to do mental tasks, obvious personality changes, inappropriate behaviour, intermittent disorientation.
    - *Grade* 3 - Somnolent but rousable, unable to perform mental tasks, disorientation, marked confusion, amnesia, occasional fits of rage, speech incomprehensible.
    - *Grade 4* - Coma, with or without response to painful stimuli. ↑Plantars
- Hyperdynamic circulation with multiple organ failure may mimic septic shock
- Abdominal distension and abdominal masses, including: ascites or hepatosplenomegaly
- Cerebral oedema with ↑ICP, may produce papilloedema, hypertension, and bradycardia

Investigations

Blood: LFT(↑Transaminases,bilirubin±ALP), FBC (↑plts), UEC (?RF), BSL may ↓, Coags (↑INR,↑FDP in DIC), ↑NH_3, blood cultures, viral serology
Special tests: free copper for Wilson's disease and paracetamol levels.
Imaging: USS (?hepatic vein patent - Budd-Chiari syndrome, ?Ca, ?ascites), CT or MRI liver,CT brain (r/o other cause of confusion or cerebral oedema), EEG, Liver biopsy (avoid if INR↑)

Management

- Treat ↑ICP: mannitol/hypertonic NaCl, head elevation, low norm pCO_2,
- Poisoning with drugs such as paracetamol or mushrooms may require specific Rx. E.g. NAC
- Lactulose, often with neomycin, is given to reduce ammonia production.
- Coagulation deficits require Vit K, FFP and platelets.
- RF: may require CVVH in preference to haemodialysis which may ↓BP
- Monitor glucose and other biochemical parameters. Usually give 5%Dex as maint. fluid.
- Protein restriction may not be as important as is traditionally taught.
- Liver transplantation may be life-saving

Complications

The major complications that cause death, even after transplantation are bleeding (oesophageal varices, retroperitoneal), sepsis (spontaneous peritonitis, opportunistic or access line infection common), cerebral oedema, renal failure, and respiratory failure.

Prognosis

Prognosis is dependent upon the cause of the hepatic failure.

- Features indicating poor prognosis include:
    - Arterial pH<7.3
    - PT>100s
    - Bilirubin>300µmol/L
    - >7 days jaundice before encephalopathy
    - Age<11 or >40.
- Hepatitis A has a good prognosis with a 50 to 60% survival.
- Survival generally <25% with fulminant hepatic failure, better with HAV, paracetamol.
- Wilson's disease normally fatal without Tx if presents as fulminant hepatic failure.
- Transplantation increases 1 yr survival.