LIVER FAILURE

Causes

Fulminant hepatic failure results from massive necrosis of liver.
Major causes:

- Viral hepatitis
- Drugs and toxins – e.g. paracetamol overdose
- Acute decompensation of chronic liver disease
- Occasionally – inadequate liver reserve post hepatectomy

Management

Resuscitation
Watch out for complication
(1) Hepatic encephalopathy

Grade 1 confused
Grade 2 drowsy but speaking
Grade 3 drowsy, incoherent or no speech but still arousable
Grade 4 comatose, unresponsive to stimulus

Management: correct aggravating factors

- GI bleed, hypoxia, hypercarbia, hypoglycaemia, infection (eg spontaneous bacterial peritonitis), electrolyte imbalance, sedation
- Septic work-up, include paracentesis if ascites present. If suspect infection, start empirical antibiotics (check with senior and the unit's antibiotic guidelines)
- May need endoscopy to R/O gastrointestinal bleeding
- dietary restriction of protein
- regular lactulose 20-30mls Q6H po to achieve 3-4 bowel motions per day
- head up 30 degrees
- others (to discuss with senior)
  Neurologic monitoring: Fulminant hepatic failure is associated with increased intracranial blood volume and cerebral hyperaemia early in the course, followed later by the development of cerebral oedema and decreased cerebral blood flow. Best is to assess mental status therefore avoid sedatives, ICP monitoring, jugular venous oxygen saturation (rarely performed in our unit)
  mannitol, thiopentone for cerebral oedema
- Liver transplantation. May be the only chance of survival in many patients. Refer to King's College criteria

(2) cardiovascular problems
patient is usually hyperdynamic and vasodilated – fluids and vasopressors

as required

(3) respiratory problems
may be unable to protect airway because of encephalopathy
lung shunts are common (hepatopulmonary syndrome)

(4) renal problems
renal failure common eg. hepatorenal syndrome (our hepatologist may suggest the use of terlipressin), often cause is multifactorial - ensure adequate hydration. In general renal function improves when liver function improves. Continuous renal replacement therapy may be required to treat volume overload or acid-base disturbances

(5) metabolic problems
hypoglycaemia – rapid correction with D50 then prevent by starting an infusion of D10 or D20. Monitor blood glucose closely

(6) haematological problems/ infection
coagulopathy, thrombocytopenia – vitamin K, FFP, platelets as indicated
Note: Preferred not to prophylactically correct the coagulopathy unless bleeding or pending an invasive procedure. However, if INR of >5, warrant attempt to correct this severe coagulopathy

For patients with underlying chronic liver failure, development of portal hypertension may result in varices, splenomegaly and ascites.
Ascites should be drained for diagnostic purposes
to relief diaphragmatic splinting
to relief abdominal compartmental syndrome
Drainage should be done with FFP and platelet cover and best done in ill patients by interventional radiologist.
Ascites fluid should be sent for biochemistry (protein, glucose, amylase)
microbiology – urgent Gram stain, C/ST, AFB and culture
hematology lab (during office hours) – WCC count

- WCC >250 x 10^6 /l and a positive culture in the absence of a primary source is diagnostic of a primary peritonitis (correction for traumatic tap, compare the peripheral RBC and WCC count, usually 1000 RBC for 1 WCC)
- Mixed growth may indicate secondary peritonitis e.g. perforated bowel

cytology