Hepatorenal syndrome is the development of renal failure in a patient with advanced liver disease. - Hepatorenal syndrome is characterized by impaired renal function, abnormalities in the arterial circulation, and activation of the endogenous vasoactive system - Divided into two types (type 1 is rapidly progressive; type 2 is slowly progressive)

Epidemiology:
- The prevalence of hepatorenal syndrome in patients with end stage cirrhosis ranges between 7% and 15%

Risk factors:
- (i) Na and H2O retention (indicated by a urinary Na of <5 mEq/L and dilutional hyponatremia),
- (ii) low mean arterial blood pressure,
- (iii) poor nutrition,
- (iv) reduced glomerular filtration rate, and
- (v) esophageal varices.

Investigation:
- (i) urine and serum toxicology screens
- (ii) hepatitis serologies
- (iii) ceruloplasmin
- (iv) antinuclear antibodies
- (v) smooth-muscle antibodies
- (vi) serum protein electrophoresis
- (vii) CMV and EBV serology

Diagnostic criteria:
- Once a patient is diagnosed with fulminant hepatic failure, the patient should be stabilized and transferred to a liver transplant center
- Certain pathogeneses demand immediate specific treatment, including:
  - (i) zinc and trientine therapy for Wilson’s disease
  - (ii) transjugular intrahepatic portosystemic shunt, surgical decompression or liver transplantation for patients with acute Budd-Chiari; and
  - (iii) aspiration precautions and fluid maintenance.

Management:
- Liver transplantation offers the best long-term survival, with an overall posttransplantation 1-yr survival of about 60%.
- Short-term extracorporeal hepatic support for patients with fulminant hepatic failure may ultimately serve to improve overall survival and provide support as a bridge to liver transplantation, but it remains experimental (2 types are cell-based and non-cell based).

Criteria for transplant in acute liver failure
- Fulminant hepatic failure is a clinical syndrome characterized by the rapid onset of hepatic encephalopathy in conjunction with a marked decline in hepatic synthetic function.

Treatment:
- (i) dialysis
- (ii) liver transplant
- (iii) TIPS
- (iv) iv clonidine has been shown to improve GFR by 25% (oral is ineffective)
- (v) midodrine / octreatide / terlipressin
- (vi) albumin administration

Hepatic encephalopathy
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Diagnosis:
- Early neuropsychological changes include disturbance in sleep patterns such as insomnia and hyperactivity.
- Neurologic abnormalities seen in more advanced presentations include disorientation, somnolence, and asterixis.
- Focal neurologic signs may be detected in some patients during episodes of hepatic encephalopathy, with hemiplegia being the most common deficit.

Management:
- Management also includes supportive measures such as restoring electrolyte balance, fluid maintenance, aspiration precautions, and rapid sequence intubation for airway protection in grades 3-4 hepatic encephalopathy.
- Management has been proposed as a possible therapeutic agent for hepatic encephalopathy based on the theory that endogenous benzodiazepines may be present in patients with hepatic encephalopathy. Meta-analyses suggest that flumazenil was associated with a significant improvement in encephalopathy compared with placebo; however, the benefit was short term and may have been confined to patients who otherwise had a favorable prognosis.