Acute Liver Failure (ALF)

What:
ALF is characterized by extensive liver injury in the absence of prior liver disease. Acute is defined by < 26 weeks, & signs of liver failure include coagulopathy with INR ≥ 1.5 as well as hepatic encephalopathy (HE).

It is important to note that ALF **is not** a patient with decompensated cirrhosis or acute on chronic liver failure (ACLF). Patients will typically be younger without many prior medical problems. They will typically have a hepatocellular pattern of injury where there ALT is greatly elevated compared to the ALP. An R factor can be calculated to determine the pattern of injury. It is important to get an accurate history with a list of old & new prescriptive & OTC medications, supplement use, teas, mushrooms or illicit drug use. Knowing if they have prior liver disease is important as management differs. Keep in mind if this their first time presenting with elevated liver enzymes, they may have ALF vs. chronic liver injury.

Etiology & Workup:
In the US, acetaminophen toxicity is the most common cause of ALF, followed by drug related, viral hepatitis & indeterminate causes. Uncommon causes of ALF includes autoimmune diseases, thrombosis or ischemic hepatitis, Wilson disease or pregnancy related ALF.

Basic workup includes CBC, BMP, liver enzymes, hepatitis & iron panel, alcohol, acetaminophen, salicylate levels and a drug screen. An abdominal US with doppler can check portal flow & potentially budd-chiari syndrome. If HE is present, non-contrasted CTH should be done.

Extrahepatic Manifestations:
HE is the most important complication to watch for as a result of increased ammonia & cerebral edema. The West Haven criteria can be used to determine severity & subsequent management.

Hypotension in ALF results from a vasodilatory state from systemic & splanchnic vasodilation, & third spacing from capillary leak. Capillary leak can lead to edema in various parts of the body including the brain.

Maintaining an adequate MAP maybe challenging & involves a combination of IV fluids & pressors.

Acute kidney injury from acute tubular necrosis can be seen from hypotension, direct nephrotoxicity or a combination of both. CRRT may be needed to correct electrolyte abnormalities & hypervolemia.

Coagulopathy is a complex process & even moreso in ALF. The INR does not correlate with bleeding risk & the SCCM recommends using viscoelastic testing such as ROTEM or TEG to identify increased bleeding risk.

Patients with ALF have a high risk of infection & deteriorating into septic shock, and appropriate treatment should be immediately started if suspected.

Prognosis & Treatment:
ALF is a potentially fatal disease with a complicated pathophysiology made worse with multiple deadly complications. Quick diagnosis is necessary to triage & treat the patient & if necessary, refer to a liver transplant center. The AASLD & SCCM have published very informative guidelines & should be referenced if needed. Treatment is unfortunately too complex for this quick overview.

References:
AASLD Guidelines: DOI: 10.14309/ajg.0000000000002340
SCCM Guidelines: DOI: 10.1097/CCM.0000000000004192