Liver Failure Physiology

Anesthetic Pearls: Anesthetic Implications and Physiologic Changes of Liver Failure

Respiratory

Chronic hypoxemia occurs for several reasons. 1) Intrapulmonary and porto-pulmonary AV shunts. 2) Pleural effusions and ascites lead to atelectasis with a restrictive pulmonary disease pattern. 3) V/Q mismatch from the impaired ability of the lungs for hypoxic pulmonary vasoconstriction (liver fails to degrade vasodilating mediators). 4) The lungs accumulate water that slows 0_2 diffusion across membranes leading to increased venous admixture (high Sv02). These changes ultimately lead to tachypnea and respiratory alkalosis. The 0_2 dissociation curve shifts to the right which is not intuitive.

Cardiovascular

A hyperdynamic CV state is the hallmark of End-Stage Liver Disease (ESLD). Cardiac output and ejection fraction increase because of numerous AV anastamoses, increased circulating blood volume, and abnormally high glucagon levels. A low SVR results from high concentrations of vasodilator substances like nitric oxide, substance P, and gama-aminobutyric acid (GABA). There is increased incidence of cardiomyopathy, pericardial effusions, dysrhythmias, HTN, and pulmonary HTN.

Neurologic

Encephalopathy occurs because of accumulation of toxic byproducts (ammonia & urea) and malnutrition (thiamine deficiency). Elevation of intracranial pressures, which cause a significant factor in mortality, contributes to symptoms of altered consciousness and damaged upper motor neuron reflexes.

Hepatic

Hypoalbuminemia leads to decreased plasma oncotic pressure translating into symptoms of ascites and edema. Laboratory parameters of liver function like annnonia, PT, transaminases are abnormal. Drugs in general have increased elimination half-life, increased free fraction, and increased efficacy.

Gastrointestinal

Portal HTN leads to gastro-esophageal varices. Gastric emptying slows which elevates the risk of aspiration. Fat and fat-soluble nutrients are malabsorbed leading to significant malnutrition because of decreased bile salt synthesis. As a result, there are bouts of diarrhea and vomiting.

Renal

The kidneys react to low effective renal perfusion by retaining sodium and increasing blood volume. High circulating aldosterone contributes to hypokalemia. For patients with obstructive jaundice, acute tubular necrosis (ATN) results from the inability to excrete bile products and toxic substances. Pre-renal azotemia result from frequent GI bleeds, paracentesis, aggressive diuresis, and ororectal fluid losses. There becomes an altered balance between vasoconstricting and vasodilating regulators within the kidneys that predispose patients to NSAID-induced renal failure and hepato-renal syndrome.

Endocrine

Glucose intolerance results from increased hyperglycemic hormones and neurotransmitters like glucagon and norepinephrine that the liver fails to catabolize. Both ADH and aldosterone become elevated. Other affected hormones include TSH (increased), growth hormone (increased), estrogens (increased), and active Vitamin D (decreased).

Hematologic

Coagulopathy is manifested from both quantitative and qualitative deficiencies in both primary and secondary homeostasis. All coagulation factors except for von Willebrand factor (vWF) and Factor-VIII are decreased. vWF, Factor-VIII, and fibrinogen become defective. The spleen sequesters platelets in patients with portal HTN while renal insufficiency contributes to the impairment of platelet-functions. Fibrinolysis is decreased due to low plasminogen and protein C & S. An ongoing low-grade DIC is associated with ESLD. The anemia becomes progressively worse because of frequent GI hemorrhages, low liver iron stores, and decreased erythropoiesis.

MELD Score

The **Model for End-Stage Liver Disease** (**MELD**) is a scoring system for assessing the severity of chronic liver disease. It was initially developed to predict death within three months of surgery in patients who had undergone a transjugular intrahepatic portosystemic shunt (TIPS) procedure and was subsequently found to be useful in determining prognosis and prioritizing for receipt of a liver transplant. MELD uses the patient's values for serum bilirubin, serum creatinine, and the international normalized ratio for prothrombin time (INR) to predict 3 month survival. It is calculated via the following formula: **MELD Score = 3.78 [serum bilirubin (mg/dL)] + 11.2 [INR] + 9.57 [serum creatinine (mg/dL)] + 6.43**. In interpreting the MELD Score in hospitalized patients, the 3 month mortality is: > 40 — 100% mortality; 30-39 — 83% mortality; 20-29 — 76% mortality; 10-19 — 27% mortality; <10 — 4% mortality.