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Opinion

Intra Operative Anesthesia Considerations in Liver Transplant

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Hepatic disease causes endothelial dysfunction that impairs all organs of the body. Thus each organ must be carefully managed throughout the operative and post-operative period.

Anesthesia typically begins with a rapid sequence induction which is necessitated by the emergent nature of the surgery, recent upper gastrointestinal bleeding and the presence of ascites. Semi upright position during induction prevents rapid oxygen desaturation until abdomen is opened. Establish large bore intravenous catheters. Rapid infusion pump must be available. Routine hemodynamic monitoring includes intraarterial access and central venous catheter. The patient's temperature must be maintained actively with use of fluid warmers and forced air surface warming devices. Goal directed hemodynamic and fluid management using arterial and pulse wave analysis, esophageal doppler or TEE is routinely used in many centers.

Balanced anesthetic technique is used. Anesthesia is usually maintained with a volatile agent where concentration is limited to less than 1 minimum alveolar concentration. Patients with fulminant liver failure are at risk of raised intra cranial pressure, inhalation agents must be avoided and TCI propofol used. Opioid usually fentanyl is chosen to blunt the sympathetic response to stimulation. Cisatracurium may be the preferred neuromuscular agent because of its organ independent elimination.

Transfusions are targeted to maintain hemoglobin more than 7g/dl. FFP is transfused approximately 2 units per unit of blood given. Clotting is monitored and managed with aid of viscoelastic coagulation assay or conventional tests of coagulation. Potassium and calcium should be monitored regularly and supplemented when required.

Liver transplantation is divided into three stages: dissection, an hepatic and enterohepatic periods.

Stage 1 of the operation is dissection and hemostasis. This is highlighted by the management of hemodynamic changes due to blood loss and surgical compression of major vessels. With abdominal incision and drainage of ascites, hypovolemia typically occurs which should be treated in an anticipatory fashion with colloid-containing fluid.

Stage 2 is the an hepatic phase where the hepatic artery, portal vein, hepatic veins and bile ducts are divided. This stage ends with reperfusion. When the liver is removed, citrate load from blood products is no longer metabolized and causes hypocalcemia and secondary myocardial depression. Periodic calcium chloride administration is necessary guided by ionized calcium concentration measurements.

Stage 3 is the neohepatic phase beginning with re-establishment of blood flow through the liver.

This may be accompanied by a reperfusion syndrome or reperfusion injury. Reperfusion is associated with abrupt increases in potassium and hydrogen ion concentrations, an increase in preload, and a decrease in systemic vascular resistance and blood pressure. Signs of graft function that may be observed in the operating room include decreased calcium requirements, improvement in acidosis, increased urine output, a rise in core temperature, and bile output from the graft.

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