ELSEVIER

Pitfalls in Anesthesia for Liver Transplantation

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PREOPERATIVE ASSESSMENT

With advances in surgical and anaesthetic techniques, patients who are previously deemed unsuitable are now presenting themselves for orthoptic liver transplantation (OLT). Age is no longer a strict exclusion; older patients (above 70 years) do just as well as the young (below 70).[1] However, age-related illness take on greater significance.

Patients with chronic liver failure can present with hypoxemia. This may be due to ascites, pleural effusion, ventilation-perfusion imbalances, or pulmonary hypertension. Hepatic pulmonary syndrome (HPS) is a triad of liver dysfunction, intrapulmonary vasodilation, and hypoxemia (Pao_2 <70 mm Hg on room air). Orthodeoxia (decrease in Pao_2 >3 mm Hg on standing from supine) is also present. Hypoxemia due to HPS can be treated by OLT and is not a contraindication to surgery.

Liver transplantation is associated with intense cardiovascular changes. Preoperative cardiovascular assessment can be difficult because these patients may not be ambulatory. This could include stress ECG, echocardiography, angiography, or MUGA scans. Patients with significant cardiovascular history were more prone to perioperative complications like arrhythmias, hypotension, myocardial infarct, and pulmonary edema. However, long-term survival after transplant was not affected.[2] Pulmonary hypertension is often associated with the development of portal hypertension. Mild and moderate pulmonary hypertension (mean < 35 mm Hg) are not contraindications to surgery. Patients with increased pulmonary resistance and mean pulmonary artery pressures > 25 mm Hg did not have worse outcome after OLT.[3]

PERIOPERATIVE EVENTS

Postreperfusion syndrome (30% decrease in mean blood pressure for more than 1 minute during the first 5 minutes of reperfusion) occurs in 8% to 30% of OLT. This may be due to acidosis, metabolic disturbances, or release of vasoactive substances from the transplanted liver. The hypotension can be accentuated by insufficient increase in preload on removal of vascular clamp. These patients show a more marked decrease in cardiac output and less increase in systemic vascular resistance in response to caval clamping.[4] Venovenous bypass (VVB) did not prevent reperfusion syndrome. Treatment consists of fluid loading, correction of acidosis, use of calcium, and inotropes. VVB has been used for venous decompression of the splanchnic circulation and the reduction of intraoperative blood loss. Renal function did not improve with bypass. All patients with or without bypass suffered a deterioration in renal function.[5] Complications of VVB include vascular problems, thromboembolic events, heat loss, and fluid sequestration.

There are no reliable predictors for perioperative bleeding. Factors influencing amount of bleeding include experience of transplant team, patient's preoperative status, previous portosystemic shunts, and use of reduced liver grafts. Patients who require more perioperative transfusions are associated with lower graft and patient survival rates.[6] This may be due to the increased release of cytokines like interleukin-6 in response to excessive intraoperative bleeding. Endotoxin concentration rises and graft function worsens.[7] The prevention of excessive intraoperative bleeding and the subsequent response of proinflammatory cytokines may affect the success of liver transplant surgery.

Coagulopathies associated with liver disease may be due to reduced production of clotting factors, platelet sequestration, or dysfunction. This can worsen during surgery due to massive blood loss, fibrinolysis, and release of heparinoids during reperfusion. Treatment must be multifaceted. The underlying defects must be corrected including giving protamine, administration of blood products, maintaining normothermia, and correcting hypocalcemia. Prophylactic use of aprotinin does not reduce bleeding.[8] The thromboelastograph (TEG) is a useful tool to rationalize therapy. Because many patients undergoing OLT can be hypothermic intraoperatively, TEG studies, which are performed at 37°C, may overestimate the quality of clotting.[9]

Patients with hepatic encephalopathy can present with raised intracranial pressure owing to cerebral edema. Treatment during OLT would be aimed at maintaining cerebral perfusion pressure (>40 mm Hg). Intracranial pressure rises soon after induction and worsens during reperfusion due to release of vasoactive mediators. This rise can continue into the early postoperative period even with a

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functioning graft.[10] Therapeutic measures including moderate hyperventilation, diuretics, and barbiturate coma should continue into the postoperative period. Neurologic complications can also arise from electrolyte abnormalities especially sodium. Rapid correction of hyponatremia (>12 mmol/L over 24 hours) can be associated with central pontine myelinolysis.

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