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Hemofiltration in Parallel to the Venovenous Bypass Circuit for Oliguric Hypervolemia during Liver Transplantation

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PATIENTS undergoing liver transplantation often require blood components perioperatively for the treatment of coagulation factor deficiencies, thrombocytopenia, and anemia. Preexisting hypervolemia may limit this therapy, particularly when oliguric renal failure complicates endstage liver disease. Intravascular volume reduction is then often accomplished with continuous arteriovenous hemodialysis (CAVHD), continuous arteriovenous hemofiltration (CAVH) or continuous venovenous hemofiltration (CVVH). This report concerns intraoperative CVVH, in parallel to the venovenous bypass (VVB) system, without anticoagulants during liver transplantation. With this CVVH technique, transmembranal pressure gradients and ultrafiltration rates resemble that of CAVH.

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Key words: Hypervolemia; liver failure; pressure gradient; renal failure.

Case Report

A 38-yr-old man was awaiting liver transplantation for the treatment of cirrhosis secondary to chronic hepatitis C virus infection. Spontaneous retroperitoneal and gastrointestinal hemorrhages had previously developed in the patient and he had received more than 50 units of packed red blood cells and fresh frozen plasma (FFP), in the medical intensive care unit over 5 days. Encephalopathy, shortness of breath and hypoxemia ensued, requiring tracheal intubation and mechanical ventilation. Serial

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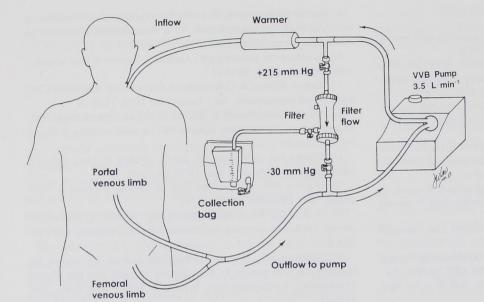


Fig. 1. The hemofilter in parallel to the liver transplant recipient on venovenous bypass (VVB). The pressure gradient across the filter in part governs the rate of hemofiltration. The rate of hemofiltration removal can also be altered by applying positive or negative pressure to the collection tubing. Pressures were electromechanically transduced from either side of the filter. The negative outflow pressure, was produced by the Bernoulli effect of the VVB flow. A pressure-flow nomogram for the filter indicates that the blood flow through the filter was approximately 400 ml/min.

chest radiography showed congestive heart failure with pulmonary edema. Oliguria developed and the patient's serum creatinine level increased from 1.3–3.1 over 2 days. He underwent dialysis *via* a double-lumen venous catheter placed in his right femoral vein. A donor liver was identified for him after 7 days.

Immediately preoperatively, the patient's arterial oxygen tension (Pa_O,) was 63 mmHg (intermittent mandatory ventilation (IMV) = 8; fractional inspired oxygen tension $[Fl_{O_2}] = 0.7$; tidal volume (TV) = 800 ml; and positive end expiratory pressure (PEEP) = $5 \text{ cm H}_2\text{O}$). The patient exhibited gross ascites, anasarca, and a peak inspiratory pressure of 44 cm H₂O. He had produced only 80 ml of urine during the preceding 24 h, despite administration of 200 mg furosemide. The venovenous dialysis catheter was still in place in the right groin, but the hemofilter had been discarded. At arrival to the operating room, the patient was mechanically ventilated with an FIO2 of 1.0 and the PEEP was increased to 10 mmHg. Oxygen saturation measure by pulse oximeter (Sp_{O₂}) increased from 93-95%, Pa_{O₂} increased to 78 mmHg. Bumetanide, 5 mg, was administered intravenously. Immediate preoperative hemoglobin level was 7.8 gm/dl, platelet count was 35,000 µl, prothrombin time/partial thromboplastin time (PT/ PTT) was 18.0/36.8, and the international normalized ratio (INR) was 2.3. Fibrinogen was 151 mg/dl and fibrin degradation products were 27 μg/ ml. A pulmonary artery occlusion catheter was placed via his right internal jugular (IJ) vein. Central venous pressure (CVP) was 29 mmHg, pulmonary artery (PA) diastolic pressure was 27 mmHg, and the pulmonary artery occlusion pressure was 27 mmHg. A 16-French right internal jugular (IJ) venous catheter was then also placed percutaneously to provide for the VVB inflow. The right groin dialysis double-lumen venous catheter was dedicated to transfusion.

The patient required intravascular volume reduction to facilitate administration of blood component products. CAVH was considered, but placement of a large arterial catheter was deferred because of the history of coagulopathic bleeding. It was decided to institute hemoconcentration by a technique similar to that performed on heparinized cardiothoracic patients requiring cardiopulmonary bypass pumps. The risk of thrombosis within the hemoconcentration filter was judged to be low in this hypocoagulable patient. The filter (Renal Systems, Minneapolis, MN) was placed across the inflow and outflow limbs of the VVB pump, as shown in figure 1. At the start of surgery, partial VVB (femoral-jugular) was

instituted, and the hemofilter was placed to gravity drainage. Hemofiltrate (1400 ml) was removed in the first 20 min. The patient's central venous pressure decreased to 19 mmHg, despite the transfusion of 12 units platelets, 1 unit packed red blood cells and 2 units fresh frozen plasma. The hemofiltration drainage tubing was then partially clamped to decrease the rate of production to 40 ml/min. During this period, the abdominal incision was also made and 6,500 ml of ascitic fluid was acutely drained. These combined factors caused a decrease in venous pressures that permitted the administration of intravenous mannitol, 0.5 gm/kg. During the 2 h of VVB, a total of 41 hemofiltrate and 1,200 ml urine were obtained from the patient. CVVH ended at discontinuation of VVB. The patient continued to produce approximately 400 ml/h urine. A total of 4 units PRBCs were transfused. The hemoglobin level at the end of the case was 9.2 gm/dl.

Discussion

Although this is technically CVVH, the rate of hemofiltration achievable across the VVB pump is closer to that associated with CAVH.

This hemofiltration entails some considerations and risk for the patient. When VVB is discontinued after liver transplantation, this technique must also end. In this case, there was no need for further hemofiltration after transplantation. Opioids and protein-unbound "middle molecules" are filtered in a fashion that may necessitate readministration. The filter's polysulfone fibers may be thrombogenic. In this case, no blood clotting was observed within the filter, despite the lack of exogenous anticoagulation. Some fibrinous material was visible on the filter fibers after flushing the system with saline. Bellomo *et al.* suggested periodic visual inspection of the filter and maintaining the patient's activated coagulation time (ACT) more than 150 s.

A massive fibrinous clot would be visible through the filter's plastic surface, but smaller thrombi would probably pass unrecognized. Clotting within the filter represents one reason for diminished hemofiltrate output. Clotting can cause pulmonary embolism, or the acute loss of VVB.

Renal failure occurs in up to 75% of patients dying of cirrhosis.⁵ CAVH and CVVH have been shown to improve pulmonary and hemodynamic function during and after liver transplantation.^{4,6}

Intraoperative hemofiltration was used to help treat hypervolemia. Placement of the hemoconcentration filter across the VVB circuit in parallel to the patient provided transmembranal pressure gradients and hemofiltrate flow rates associated with CAVH without cannulation of the arterial circulation. There is also no need for venous cannulation beyond that required for VVB. The technique can effect a rapid decrease in intravascular volume and may be performed in selected patients without systemic anticoagulants.

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Respiratory Compromise and Dramatic Chest X-ray Changes during General Anesthesia in a Patient with a Bronchogenic Cyst

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BRONCHOGENIC cysts are rare congenital anomalies that may cause obstructive emphysema. We present the

case of an infant in whom acute respiratory distress was caused by a bronchogenic cyst. Dramatic changes in the chest radiograms, from hyperinflation of the left lung and atelectasis of the right lung to hyperinflation of the right and atelectasis of the left, were observed within a short period of time during general anesthesia. Surgical excision provided complete relief.

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Key words: Atelectasis; blood gases; deterioration; hyperinflation of the lung; positive pressure ventilation; ventilation.

Case Report

An 8-month-old, 8-kg boy was admitted to a nearby hospital with a 4-week history of a croupy cough, wheezing, and fever. He was the full-term product of an uncomplicated pregnancy and delivery. He had similar symptoms when he was 5 months old and was treated for asthma and pneumonia for 1 month. At admission, he was tachypneic (60–70 breaths/min) with marked inspiratory stridor unaffected by posture. There were faint breath sounds on the left side of the chest and slight wheezing was heard on the right side. Oxygen saturation measured by pulse oximetry (Sp $_{\rm O_2}$) was 74% and did not respond to

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