Anesthetic Management of Severely Hypokalemic Patients for Liver Transplantation

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Practitioners continue to defend arbitrary serum potassium values as a prerequisite for elective surgery. Recent studies, however, have failed to detect an increased frequency of intraoperative arrhythmias associated with chronic hypokalemia (K+ = 1.9 mEq/l) in either healthy populations or patients with known heart disease.1,2 We report the course of four severely hypokalemic patients (K+ = 1.9 mEq/l) with end-stage liver disease (ESLD) presenting for urgent orthotopic liver transplantation (OLT).

CASE REPORTS

Four adult patients with ESLD and severe preoperative hypokalemia (1.9 mEq/l) were admitted for OLT (tables 1–3). None of the four patients demonstrated electrocardiographic evidence of hypokalemia (e.g., QRS widening, S-T segment abnormalities, progressive diminution of the T-wave, or evidence of a U wave) during the postoperative period. Preoperative ECG monitoring in the intensive care unit (ICU) demonstrated a regular sinus rhythm without ectopy. Serum potassium determinations were made inoperatively every half hour. The low serum potassium concentrations were unchanged during the dissection and anhepatic phases, and increased an average of 0.5 mEq/l following reperfusion of the donor liver (fig. 1). Only one patient received intraoperative potassium replacement, a total of 180 mEq/l over 3 h. The absence of arrhythmias for all patients was assessed inoperatively by continuous monitoring of the ECG. The only premature ventricular contractions (PVCs) noted were during insertion of the pulmonary artery catheters; however, they abated spontaneously.

DISCUSSION

Hypokalemia is usually defined as a serum potassium of less than 3.5 mEq/l and may reflect either depletion of total body potassium or an acute redistribution of potassium ions between the intracellular and extracellular compartments. Potassium, which is approximately 200 times more permeable than sodium, is concentrated intracellularly. The primary factors contributing to total body potassium depletion include gastrointestinal losses (e.g., vomiting, diarrhea) or increased renal losses due to diuretics,§ hyperosmolar states, excess aldosterone secretion,§ excess cortisol secretion, or surgical trauma. Altered distribution of potassium can also be due to respiratory or metabolic alkalosis, intracellular uptake of glucose, and beta-2 agonist stimulation.

Potential detrimental effects of hypokalemia include diminished cardiac conduction and contractility, dysfunction of the neuromuscular junction resulting in muscular weakness, ileus, and polyuria.4,5 Electrophysiologic data suggest that hypokalemia should play a significant role in the genesis of cardiac dysrythmias. The distribution of potassium across the cellular membrane is responsible for the resting membrane potential. Hypokalemia, with intracellular depletion, has been shown to increase pacemaker discharge from an increase in the slope of phase IV diastolic depolarization with concurrent decreases in threshold potential, thus favoring emergence of potential pacemakers for the development of dysrythmias.6 Hypokalemia has also been shown to cause slowing of conduction, dispersion of refractoriness, and unidirectional block, thereby favoring re-entrant cardiac dysrythmias, especially in patients with known ischemic heart disease, digitalis toxicity, or history of cardiac arrhythmia.7 However, it is generally accepted that patients suffering from chronic hypokalemia are at less arrhythmogenic risk than patients in whom an acute lowering of serum potassium has occurred.

Metabolic changes may also acutely affect potassium distribution in patients undergoing OLT. Liver transplantation is accompanied by hyperglycemia due to multiple factors. These include decreased glucose utilization by the diseased liver, administration of glucose-containing blood products, and release of glucose from the ischemic hepatocytes.8 Although many patients show a reduced ability to shift glucose intracellularly, this function along with other cellular functions recovers and may predispose the patient to hypokalemia, especially during the postreperfusion phase where hyperglycemia is seen most commonly. Consequently, serum potassium concentration should be carefully monitored during that period. In our patients, the blood glucose concentration increased to an

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average of 328 ± 53 mg/dl after reperfusion and remained elevated during the remainder of the operation.

Usually, cirrhotic patients are alkalotic in response to hypoxia with subsequent hypokalemia. In our patients (those with available arterial blood gases), there was no alkalosis to explain the hypokalemia. Acidosis is frequently seen during the first two phases of OLT and is due to blood transfusion and the inability of the liver to metabolize the acidic metabolites.

Alkalosis occurs during the postrevascularization phase when the donor liver starts metabolizing citrate from transfused blood products. Intraoperatively, alkalosis was avoided and the pH maintained between 7.35 and 7.45 by changing respiratory parameters. In addition, metabolic acidosis observed during the initial two phases of transplantation was not fully corrected in order to avoid further hypokalemia.

Sources of exogenous potassium during OLT are primarily from the packed cells transfused during the case. In the four patients described, blood transfusion ranged from 6–8 units of packed cells, a quantity unlikely to affect serum potassium (table 3). Another source of potassium is from the donor liver preservation solution (K = 155 mEq/l). It has been reported that plasma potassium increases an average of 1 mEq/l at reperfusion of the donor liver. In our institution, the donor liver is flushed profusely before revascularization resulting in an average increase in potassium of only 0.5 mEq/l.

The interrelationship of potassium, magnesium, and calcium is important in maintaining the normal negative sites on sodium channels, thus setting the threshold for depolarization.9 Magnesium controls calcium ion movements and so regulates myocardial excitability and contractility. Hypermagnesemia depresses atrioventricular and intraventricular conduction and can cause ventricular fibrillation or asystole. Like potassium, magnesium is depleted by diuretic therapy. Unfortunately, magnesium concentrations were not measured in these patients. Hypokalemia, due to citrate toxicity, is common during liver transplantation.10 Serum-ionized calcium was maintained in the normal range as determined by serial measurement every 30 min. Appropriate doses of calcium chloride were administered when indicated.

At reperfusion of the donor liver, some patients experience a decrease in their systemic blood pressure by more than 30 mmHg. Common treatment includes the use of beta agonists to support circulation. These agents promote intracellular shift of K+ and sensitize the myocardium to arrhythmias and should be avoided in the presence of hypokalemia.11 Three of our patients had a decrease in systolic blood pressure of less than 20 mmHg, and in the fourth patient systolic blood pressure decreased

### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Disease</th>
<th>Age</th>
<th>Preoperative Arrhythmias</th>
<th>Preoperative Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Primary Biliary Cirrhosis</td>
<td>38</td>
<td>None</td>
<td>Diuretics, Potassium supplement</td>
</tr>
<tr>
<td>2</td>
<td>Primary Biliary Cirrhosis</td>
<td>40</td>
<td>None</td>
<td>Diuretics, Potassium supplement</td>
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<tr>
<td>3</td>
<td>Sclerosing Cholangitis</td>
<td>41</td>
<td>None</td>
<td>Diuretics, Potassium supplement</td>
</tr>
<tr>
<td>4</td>
<td>Biliary Atresia</td>
<td>29</td>
<td>None</td>
<td>Diuretics, Potassium supplement</td>
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</tbody>
</table>

### Table 2. Preoperative Laboratory Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Disease</th>
<th>Bilirubin (mg/dl)</th>
<th>SGOT (U/l)</th>
<th>SGPT (U/l)</th>
<th>Albumin (g/dl)</th>
<th>Alkaline Phosphatase (U/l)</th>
<th>PT (s)</th>
<th>aPTT (s)</th>
<th>Platelets</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Primary Biliary Cirrhosis</td>
<td>12.3</td>
<td>235</td>
<td>45</td>
<td>2.3</td>
<td>550</td>
<td>30</td>
<td>76.3</td>
<td>100,000</td>
</tr>
<tr>
<td>2</td>
<td>Primary Biliary Cirrhosis</td>
<td>21</td>
<td>187</td>
<td>84</td>
<td>1.9</td>
<td>359</td>
<td>29</td>
<td>69</td>
<td>89,000</td>
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<tr>
<td>3</td>
<td>Sclerosing Cholangitis</td>
<td>14.2</td>
<td>1040</td>
<td>565</td>
<td>1.8</td>
<td>224</td>
<td>40.4</td>
<td>67</td>
<td>110,000</td>
</tr>
<tr>
<td>4</td>
<td>Biliary Atresia</td>
<td>11.4</td>
<td>220</td>
<td>167</td>
<td>2.1</td>
<td>1274</td>
<td>35</td>
<td>76</td>
<td>200,000</td>
</tr>
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</table>
53 mmHg. An alpha agonist (phenylephrine 200 μg iv) was used to restore the blood pressure to normal.

The rationale for our decision to proceed with these cases was based on several factors encompassing both physiologic and clinical considerations. First, the patients’ conditions were progressively deteriorating and delay of the procedures may have put them at greater risk for increased bleeding, advancing encephalopathy, and renal failure related to hepatic failure. All had denied any history of palpitations or syncpe, and preoperative ECG monitoring had failed to document any rhythm abnormality. Although a serum potassium of 1.9 mEq/l represents a severe deficit in total body potassium (up to several thousand mEq), repletion therapy was not a viable option because of the emergent nature of the surgery and the need for several days to accomplish replenishment safely (0.5 mEq/kg/h).

Although the first patient of the series was given 180 mEq of potassium over 3 h, the serum potassium concentration was unchanged during the dissection and anhepatic phases. This finding was observed in the other three, nonsupplemented patients as their potassium concentrations were similar. In addition, there is a case report demonstrating that cirrhotic patients are less tolerant of exogenously administered potassium as a result of a decrease in hepatic uptake of potassium. In view of these factors and recent clinical studies suggesting that chronic hypokalemia does not add to perioperative morbidity or mortality, the decision was made to proceed without repletion.

### CONCLUSION

Severe preoperative hypokalemia in any surgical patient is not an ideal situation to confront in the operating room when there is no time for preoperative restoration of potassium body stores. In the severely ill or emergency surgical patient, such as those undergoing liver transplant, intraoperative and postoperative metabolic and electrolyte derangements may potentiate hypokalemia. However, on the basis of experience with these cases, the authors suggest that a preoperative potassium concentration of less than 2.0 mEq/l may not constitute a contraindication to urgent anesthesia in a chronically hypokalemic patient.

### REFERENCES

5. Hill GE, Wong KC, Shaw CL, Blatnick RA: Acute and chronic

### Table 3.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Blood Products</th>
<th>Supplementation of K⁺ (mEq)</th>
<th>pH</th>
<th>Preoperative Arterial Blood Gases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>FIO₂</td>
</tr>
<tr>
<td>Primary</td>
<td>8 units</td>
<td>180</td>
<td>7.45</td>
<td>.21</td>
</tr>
<tr>
<td>Biliary</td>
<td>21 FFP</td>
<td></td>
<td></td>
<td>.21</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>9 units</td>
<td>None</td>
<td>7.45</td>
<td>not available</td>
</tr>
<tr>
<td>Sclerosing</td>
<td>11 units</td>
<td>None</td>
<td></td>
<td>not available</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>17 FFP</td>
<td></td>
<td></td>
<td>not available</td>
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<tr>
<td>Biliary</td>
<td>7 units</td>
<td>none</td>
<td></td>
<td>not available</td>
</tr>
<tr>
<td>Atresia</td>
<td>11 FFP</td>
<td></td>
<td></td>
<td>not available</td>
</tr>
</tbody>
</table>

A New Technique for Replacing an Endobronchial Double-Lumen Tube with an Endotracheal Single-Lumen Tube

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Although endobronchial intubation with a double-lumen tube (DLT) is both useful and safe in thoracic surgical operations, the need for gradual separation from mechanical ventilation postoperatively may necessitate placement of a single-lumen endotracheal tube (SLT) before emergence from anesthesia. This maneuver may be difficult, either due to preexisting anatomic considerations or because of postoperative changes such as upper airway edema. This report describes a technique for rapidly replacing a DLT with an SLT using a flexible bronchoscope as a guide while maintaining nearly continuous ventilation.

CASE REPORT

A 70-yr-old, 45-kg woman with cancer of the mid-esophagus was scheduled for esophagectomy. Her physical findings, blood chemistries, and pulmonary function tests were within normal limits. Her ECG showed sinus tachycardia at 105 beats per min with Q waves in V1–V2 and diffuse, nonspecific S-T segment abnormalities. Intraoperative monitors included ECG, digital pulse oximetry, left radial arterial catheter, and mass spectrometry.

While breathing oxygen and after receiving d-tubocurare (1.5 mg iv), anesthesia was induced with sufentanil (30 µg iv) and thiopental (200 mg iv). Succinylcholine (80 mg iv) was administered to facilitate tracheal intubation while cricoid pressure was applied by an assistant. It was not possible to visualize the glottis directly using a 3 MacIntosh laryngoscope blade despite the patient being in the “sniffing position.” The difficulty in visualization of the glottis appeared to be caused by a combination of a long incisor-to-epiglottis distance, a relatively small mouth, and limited head extension. An attempt was made to use a long and narrow laryngoscope blade (a no. 3 Miller blade), but glottic visualization remained unsuccessful despite repositioning the head with slight extension of the neck.

While the lungs were ventilated with 100% oxygen via a mask, a no. 35 left-sided (9 mm ID), polyvinyl-chloride DLT (Mallinckrodt) was prepared by removing the stilet and passing a 4-mm diameter flexible fiberoptic bronchoscope (FFB; Olympus LF-1) through the DLT bronchial lumen. The DLT-FFB combination was then passed into the trachea, and the FFB was advanced into the left main bronchus and served as a guide for placement of the DLT. The FFB was then removed, and the Y-adapter was attached; it was possible to ventilate the lungs within 50 s from the start of the FFB-DLT intubation. Exact placement of the DLT was confirmed both by auscultation of breath sounds and by FFB examination of the bronchial lumen of the DLT via the tracheal lumen as described by Bemunof. In general anesthesia was maintained with isoflurane (0.3–0.8% in oxygen), sufentanil (50 µg iv), and vecuronium. The operation proceeded uneventfully with the right lung deflated when surgically necessary.

At the conclusion of surgery, bilateral lung inflation was reinstituted successfully. It was decided, however, that the lungs should be mechanically ventilated in the Post Anesthesia Care Unit. Because of the previous difficulty during direct laryngoscopy, uncertainty regarding the patient’s upper airway anatomy, and the possible development of postoperative laryngeal edema, it was felt that changing from the DLT to an SLT should be performed under FFB guidance.

After assuring that the patient was completely paralyzed, both the tracheal and bronchial cuffs of the DLT were deflated, and the DLT was withdrawn until the bronchial lumen was above the level of the carina. The tracheal lumen adapter was cross-clamped, and bilateral ventilation was maintained via the bronchial lumen. With the tracheal lumen now isolated, a no. 10 scalpel was used to create a 5×5-mm opening in the lateral wall of the tracheal lumen distal to the reinforced area connecting the bronchial and tracheal lumina. A 25-cm long, 7-mm OD SLT was advanced over the FFB until the 15-mm adapter

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