Transplant Perioperative Metabolic Acidosis in a Patient with Ileal Bladder Augmentation

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A 6-year-old, 13-kg girl required an anterior spinal fusion for treatment of progressive scoliosis. Medical history was remarkable for a myelomeningocele repaired shortly after birth, a ventriculoperitoneal shunt insertion at 2 weeks of age, and a bladder augmentation with an ileal segment at 4 yr of age. Preoperative laboratory studies were as depicted in table 1. Notably, the chloride level was 108 mEq/L (normal 101–111) and the carbon dioxide level was 17.5 mEq/L (normal 18–25). The hemoglobin was 10.5 mg/dl.

Preoperatively, the patient received Dextrose 5%/0.4% normal saline at 40 ml/hr overnight. In the operating room, monitoring with an electrocardiogram, precordial stethoscope, automated blood pressure, pulse oximeter, and mass spectrometer was established. Anesthesia was induced with halothane in nitrous oxide and oxygen.

Then maintained with 0.25–0.5% isoflurane in a 2:1 mixture of nitrous oxide and oxygen. Paralysis and analgesia were accomplished by intermittent doses of vecuronium and morphine sulfate, respectively. An indwelling urinary catheter drained cloudy urine, with large amounts of mucus, as is typical in a patient with intestinal bladder augmentation.

One hour after the start of the operation, the urine output ceased and was not reestablished by fluid challenge of 200 ml of normal saline. Catheter obstruction secondary to mucus was suspected and corrected by irrigation of the urinary catheter that successfully reestablished urinal flow.

The operation proceeded uneventfully with blood pressure maintained at 110–120/70 mmHg and heart rate at 110–120 beats/min. Four hours after the start of the procedure, the blood loss was estimated at 100 ml and the patient had received 800 ml of normal saline. Blood pressure was 120/70 mmHg, pulse 115 beats/min, and temperature 35°C. Urine output was acceptable at 1 ml/kg/h. An arterial blood gas at that time demonstrated a metabolic acidosis, with pH 7.25, PaCO₂ 58 mmHg, PaO₂ 170 mmHg, and base excess (BE) –2 (table 2). The hemoglobin had decreased to 9.9 mg/dl. Further 100-ml boluses of normal saline, 50 ml 5% albumin, and 13 mEq of bicarbonate were administered, without significant changes in vital signs, and a repeat arterial blood gas showed pH 7.32, PaCO₂ 59 mmHg, PaO₂ 255 mmHg, and BE 5.0.

At the conclusion of surgery, mechanical ventilation was maintained in the intensive care unit because of concerns over the persistent metabolic acidosis. Total intraoperative fluid therapy had amounted to 1,200 ml of normal saline and 50 ml 5% albumin for the 6-h procedure. Total blood loss was estimated at 200 ml. Arterial blood gas postoperatively showed pH 7.41, PaCO₂ 45, PaO₂ 220 mmHg, and base excess (BE) 12.5 (table 2). The diagnosis of metabolic acidosis was established when serum electrolytes were measured showing sodium 147 mEq/L, potassium 3.7 mEq/L, chloride 121 mEq/L, and carbon dioxide 15.8 mEq/L. Cursatting of the sodium bicarbonate and administration of sodium bicarbonate was successful in returning the chloride and the carbon dioxide to their baseline levels on the 2nd postoperative day. The patient had an uneventful postoperative course. On follow-up visit 8 months after the procedure, the patient had normal electrolyte levels (table 1).

Discussion

Metabolic acidosis may result from bicarbonate ion loss or dilution, from increase of endogenous organic acids, or from addition of exogenous acid. Metabolic acidosis in the course of general anesthesia is most often related to hypoperfusion of essential organs, resulting in anaerobic metabolism and accumulation of lactate.

Case Report

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CASE REPORTS

Table 1. Laboratory Data

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>1 h Postoperative</th>
<th>8 h Postoperative</th>
<th>18 h Postoperative</th>
<th>28 h Postoperative</th>
<th>8 mo Postoperative</th>
</tr>
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<tbody>
<tr>
<td>Sodium (mEq/L)</td>
<td>141</td>
<td>147</td>
<td>144</td>
<td>145</td>
<td>138</td>
<td>144</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>4.5</td>
<td>3.7</td>
<td>3.8</td>
<td>3.3</td>
<td>3.3</td>
<td>4.6</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>108</td>
<td>121</td>
<td>117</td>
<td>116</td>
<td>108</td>
<td>107</td>
</tr>
<tr>
<td>Total CO₂ (mEq/L)</td>
<td>17.6</td>
<td>15.8</td>
<td>20.6</td>
<td>22.9</td>
<td>26.2</td>
<td>20.6</td>
</tr>
<tr>
<td>BUN (g/dl)</td>
<td>18</td>
<td>10</td>
<td>6</td>
<td>6</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.4</td>
<td>0.4</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Acid. Ketoacidosis may be seen in uncontrolled diabetics and in starvation situations. Dilution acidosis has been associated with rapid infusion of large amounts of normal saline, as well as mannitol and Dextrose/water in dogs. However, these experiments were terminated as soon as the acidosis was demonstrated, and steady-state situation was not examined. Further studies showed that acid titration by intracellular and bone nonbicarbonate buffers will mitigate the dilution of bicarbonate and defend against changes in extracellular hydrogen ion concentration. It was concluded that "an acute volume expansion of a degree capable of causing marked hemodynamic alterations would still be insufficient to result in a clinically appreciable degree of metabolic acidosis." In the only case of dilution acidosis reported in the literature, the total carbon dioxide decreased to 9 mEq/l after the infusion of 81 of normal saline over 15 h to an 84-yr-old, 27-kg woman. Our patient received a comparatively much smaller amount of normal saline over 10 h, and the bicarbonate level did not change significantly. Although the administration of large amounts of normal saline is purported to result in mild hyperchloremia and hypernatremia, the severity of the acidosis (BE = -12.5) and hyperchloremia (Cl = 121) experienced by our patient indicates that other factors were involved.

Metabolic hyperchloremic acidosis is a well known result of bladder augmentation. Boyd first described the disorder in 1931 in a child who developed chronic acidosis and rickets after bilateral ureterosigmoidostomy. D’Agostino et al. demonstrated bicarbonate secretion by the colon after rectal instillation of normal saline solution and suggested that it contributes to the acidosis. Koch and McDougall demonstrated that chloride, ammonia, ammonium, and potassium were the principal ions reabsorbed by the ileal segment. Chloride is the principal anion reabsorbed, and H⁺, K⁺, and ammonium are the cations reabsorbed, in equiequivalent amounts to chloride, therefore producing a normal anion gap. Ammonium absorption constitutes the major mechanism for excess hydrogen ions in the serum; a bicarbonate ion is secreted in the lumen in exchange for a chloride ion, and a sodium ion is secreted in exchange for a hydrogen ion, therefore contributing to the acidosis.

Although approximately 20% of patients with an ileostomy and normal electrolyte values on routine follow-up will have periodic episodes of hyperchloremic metabolic acidosis, the sudden development of metabolic acidosis under anesthesia has not been reported previously. We believe that the administration of a large intraoperative load of chloride ions to our patient resulted in an acute increase in reabsorption of chloride.

Table 2. Arterial Blood Gases

<table>
<thead>
<tr>
<th></th>
<th>5 h Intraoperative</th>
<th>6 h Intraoperative</th>
<th>1 h Postoperative</th>
<th>3 h Postoperative</th>
<th>7 h Postoperative</th>
<th>11 h Postoperative</th>
<th>20 h Postoperative</th>
</tr>
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<tbody>
<tr>
<td>pH</td>
<td>7.25</td>
<td>7.32</td>
<td>7.21</td>
<td>7.25</td>
<td>7.27</td>
<td>7.30</td>
<td>7.33</td>
</tr>
<tr>
<td>Pao₂ (mmHg)</td>
<td>39</td>
<td>39</td>
<td>35</td>
<td>35</td>
<td>41</td>
<td>44</td>
<td>42</td>
</tr>
<tr>
<td>Paco₂ (mmHg)</td>
<td>130</td>
<td>126</td>
<td>254</td>
<td>132</td>
<td>163</td>
<td>144</td>
<td>80</td>
</tr>
<tr>
<td>Base excess</td>
<td>-9.2</td>
<td>-5.0</td>
<td>-12.5</td>
<td>-10.6</td>
<td>-7.0</td>
<td>-4.2</td>
<td>-3.5</td>
</tr>
<tr>
<td>Hemoglobin (mg/dl) (est)</td>
<td>10.0</td>
<td>9.9</td>
<td>8.9</td>
<td>7.7</td>
<td>9.7</td>
<td>10.9</td>
<td>9.7</td>
</tr>
</tbody>
</table>

hydrogen ion, and ammonium, as witnessed by the acidosis and the hyperchloremia. The decrease in bicarbonate level from 17.6 to 15.8 mEq/l is explained by the associated bicarbonate secretion by the ileal segment of the bladder augmentation.

This clinical situation is analogous to an experiment by Piser et al.13 that explored the appropriateness of the use of a gastric segment for bladder augmentation to avoid the problem of metabolic acidosis seen, especially in patients with renal failure.13 Dogs with colostomyplasty or gastrocystoplasty were subjected to acute acid and/or intravenous normal saline loading. The authors found a net reabsorption of chloride, ammonia, and sodium ions in the colostomyplasty animals and an opposite trend in gastrocystoplasty.

The formation of mucus and intermittent plugging of the urinary catheter may have played a facilitating role in this mechanism. Bladder augmentation with an ileal segment frequently is associated with production of mucus. This may lead to intermittent obstruction of the urinary catheter and incomplete bladder emptying. The resulting stasis of urine may have allowed increased time for reabsorption of solute.

Preanesthetic evaluation of a patient with bladder augmentation should include a determination of the metabolic status and the intestinal segment used. Cystoplasties with a colon or ileum segment are associated with hyperchloremic metabolic acidosis in 70% of the cases.3 Depletion of body buffers and hypocalcemia may be present even in the absence of significant changes in serum chloride or total bicarbonate level.11 The use of jejunum may result in hypochloremic acidosis. Gastrocystoplasties may be associated with hyperchloremic metabolic alkalosis secondary to bicarbonate reabsorption, especially in renal failure patients who are unable to excrete the excess bicarbonate load. Intraoperative monitoring of electrolytes levels and arterial blood gases is advisable during long procedures. Solute administration must be tailored to the metabolic derangement caused by the cystoplasty. Frequent perioperative irrigation of the urinary catheter may be necessary to alleviate or prevent its obstruction by mucus.

In conclusion, we report on a case of obstruction of urinary catheter by mucus, and acute metabolic decompensation in a patient with ileal bladder augmentation, manifested by the occurrence of hyperchloremia and perioperative acidosis. Treatment consisted of irrigation of the urinary catheter, reduction of sodium chloride load, and administration of sodium bicarbonate.

References


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