SURVIVAL OF ACUTE HYPERNATREMIA DUE TO MASSIVE SOY SAUCE INGESTION

David J. Carlberg, MD, Heather A. Borek, MD, Scott A. Syverud, MD, and Christopher P. Holstege, MD

Department of Emergency Medicine, University of Virginia Medical Center, Charlottesville, Virginia

Reprint Address: Heather A. Borek, MD, Department of Emergency Medicine, Division of Medical Toxicology, Einstein Medical Center, 5501 Old York Road, Korman B-6, Philadelphia, PA 19141

Abstract—Background: Intentional massive sodium chloride ingestions are rare occurrences and are often fatal. Objectives: There are a variety of treatment recommendations for hypernatremia, ranging from dialysis to varying rates of correction. We report a case of acute severe hypernatremia corrected with rapid free-water infusions that, to our knowledge, has not been previously reported.

Case Report: A 19-year-old man presented to the Emergency Department in a comatose state with seizure-like activity 2 hours after ingesting a quart of soy sauce. He was administered 6 L of free water over 30 min and survived neurologically intact without clinical sequelae. Corrected for hyperglycemia, the patient’s peak serum sodium was 196 mmol/L, which, to our knowledge, is the highest documented level in an adult patient to survive an acute sodium ingestion without neurologic deficits. Conclusion: Emergency physicians should consider rapidly lowering serum sodium with hypotonic intravenous fluids as a potential management strategy for acute severe hypernatremia secondary to massive salt ingestion. © 2013 Elsevier Inc.

Keywords—acute hypernatremia; hypotonic fluids; resuscitation; sodium chloride

INTRODUCTION

Acute sodium chloride (salt) ingestion has long been known to have dire consequences. In ancient China, salt ingestion was a traditional method used for suicide (1). In the 1960s and 1970s, hypertonic saline ingestion was recommended for inducing emesis after poisoning. These recommendations were rescinded after recognition of the associated iatrogenic morbidity and mortality (1,2). Other iatrogenic causes of acute hypernatremia include incorrect dilution of oral rehydration solution, as well as hypertonic intravenous fluids, enemas, gastric lavage, and peritoneal lavage (1,3–5). Acute sodium chloride poisoning in adults generally occurs as a suicide attempt in the setting of mental or emotional disorders (2).

CASE REPORT

A 19-year-old man with no significant past medical history was brought to a local community hospital by his friends with altered mental status. He drank a quart-sized bottle of soy sauce on a challenge from friends approximately 2 h prior. On arrival to the Emergency Department (ED), he was unresponsive to verbal or painful stimuli. He exhibited teeth grinding, with arms stiffly fixed at his side, clinically suspicious for seizure activity. He was started on lactated ringers at 200 mL/h for resuscitation purposes before the return of any laboratory results. A nasogastric (NG) tube was placed to suction, with return of brown material with scant streaks of blood. He was given 150 mL/h of free water through the NG tube. A head computed tomography (CT) scan was normal. He received a total of lorazepam 12 mg and fosphenytoin 1 gram phenytoin equivalent during his 1.5-h ED course, which resulted in a cessation of seizure-like activity.
Upon arrival at the tertiary care hospital 4 h after ingestion, he remained comatose, with a Glasgow Coma Scale score of 3 and no apparent seizure activity. His vital signs were: blood pressure 160/94 mm Hg; pulse 147 beats/min; respirations 48 breaths/min; temperature 39.4°C (102.9°F) rectally; weight 72 kg. The physical examination was significant for multiple beats of inducible ankle clonus bilaterally, pupils 4 mm bilaterally with a disconjugate gaze, and tachycardia with good pulses. An electrocardiogram revealed sinus tachycardia with a QRS of 148 ms in a right bundle-branch block pattern and no evidence of central pontine myelinolysis. One month after ingestion he had returned to school and was doing well with normal mental status. A follow-up MRI showed persistent high signal in the right hippocampus, favored to represent postictal change. He was maintained on phenytoin for seizure prophylaxis and was discharged home on hospital day 4 without any neurologic deficits.

On follow-up 9 days after ingestion, the patient self-extubated. He remained confused, but was oriented to person and place on hospital day 2. His mental status returned to normal on hospital day 3, at which point a magnetic resonance imaging (MRI) scan of the brain showed swelling, abnormal signal, and restricted diffusion of the right hippocampus, favored to represent postictal change. He was maintained on phenytoin for seizure prophylaxis and was discharged home on hospital day 4 without any neurologic deficits.

Soy sauce contains 17–18% sodium chloride and has the potential to cause severe hypernatremia. The estimated lethal dose of sodium chloride is 0.75–3 g/kg (1,6,7). For reference, a tablespoon of table salt weighs approximately 15 g (2,4,6). Our patient drank approximately a quart of soy sauce, which contained between 160 and 170 g of salt, and was in the reported lethal range for his weight. The two cases of soy sauce ingestion published in English describe middle-aged women with extensive psychiatric histories who drank large volumes of soy sauce in suicide attempts (6,7). Blood sodium concentrations were 177 mmol/L and 187 mmol/L, respectively, and both of these cases resulted in death. Our case differs from previous case reports in that the patient was male, had no past medical or psychiatric history, and was not suicidal. His sodium level of 196 mmol/L is, to our knowledge, the highest reported in an adult patient to survive an acute sodium ingestion neurologically intact.

Patients who become acutely hypernatremic are frequently tachycardic and tachypneic, and often develop seizures (1,2,4,6). Fever, mild leukocytosis, altered

### Table 1. Blood Laboratory Values

<table>
<thead>
<tr>
<th></th>
<th>2</th>
<th>4</th>
<th>4.5*</th>
<th>5†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mmol/L)</td>
<td>177</td>
<td>182</td>
<td>191.3</td>
<td>154</td>
</tr>
<tr>
<td>Corrected sodium (mmol/L)</td>
<td>182</td>
<td>187</td>
<td>196</td>
<td>170</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>5.6</td>
<td>4.8</td>
<td>5.1</td>
<td>2.5</td>
</tr>
<tr>
<td>Chloride (mmol/L)</td>
<td>150</td>
<td>&gt;150</td>
<td>157</td>
<td>130</td>
</tr>
<tr>
<td>Bicarbonate (mmol/L)</td>
<td>19</td>
<td>15</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>16</td>
<td>18</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>1.6</td>
<td>1.8</td>
<td>2.1</td>
<td>2</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>384</td>
<td>414</td>
<td>365</td>
<td>1116</td>
</tr>
<tr>
<td>Salicylate (mg/dL)</td>
<td>&lt;3</td>
<td>&lt;3</td>
<td>&lt;3</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Acetaminophen (ug/mL)</td>
<td>&lt;1.3</td>
<td>&lt;1.3</td>
<td>&lt;1.3</td>
<td>&lt;1.3</td>
</tr>
<tr>
<td>Ethanol (mg/dL)</td>
<td>&lt;10</td>
<td>&lt;10</td>
<td>&lt;10</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

BUN = blood urea nitrogen; INR = international normalized ratio; PTT = partial thromboplastin time.

* Arterial blood gas.
† After 6 L 5% dextrose in water.
mental status, confusion, combativeness, and coma have been described (1,4,6,8). Other neurologic findings include increased muscle tone, involuntary muscle twitches, and hyperactive reflexes with clonus (7). Autopsy examinations frequently demonstrate intracranial hemorrhages, which are believed to occur via shearing forces in the brain secondary to osmotic fluid shifts (6,7). Patients may develop a high-output heart failure, pulmonary edema, venous congestion, and acute renal insufficiency (1,2,6,7). Blood gas analysis on these patients frequently shows a metabolic acidosis (4).

The treatment of hypernatremia in the setting of acute salt ingestion is controversial. Both dialysis and hypotonic fluid administration have been recommended (1,8–13). Although the importance of early recognition and treatment is well accepted, the rate of correction is still debated. Adrogue and Madias stated that rapid correction of acute excess sodium ingestion improves prognosis and does not increase the risk of cerebral edema as the brain has not had time to adapt to the increased osmolarity (14). Some authors recommend correction at 1–2 mmol/L/h (1,2). Treatment with these correction rates has produced mixed results. A positive neurologic outcome was reported in an elderly woman with a sodium level of 193 mmol/L whose level was corrected using 150 mL/h of D5W; however, the acuity of this sodium ingestion was not reported (8). MacDonald described the treatment of a patient with an acute sodium elevation to 186 mmol/L using intravenous D5W at a rate of 125 mL per hour, but this patient had a poor neurologic outcome (13). McGouran presented the case of a middle-aged woman who presented 21 h after a salt ingestion with a sodium level of 185 mmol/L. Her serum sodium was lowered over 18 h, but she had a poor neurologic outcome. The author postulated that this was because the patient had presented so late after ingestion, and not because treatment was too aggressive (12).

Survival with an acute elevation of sodium beyond 175 mmol/L is rare, and the prognosis is particularly poor when hypernatremia develops rapidly (1,6). Ofran et al. suggested that ingestions over the course of minutes to hours are uniformly associated with a poor prognosis; with more prolonged salt intake, survival from acute hypernatremia improves (2). At the time of our patient’s presentation to the tertiary care center, the treating physicians were aware of the poor outcomes reported in previous salt poisonings with this degree of serum sodium elevation. In view of these outcomes, the physicians felt that aggressive intervention was warranted despite the potential complications that may occur with rapid lowering of serum sodium. After 6 L of D5W over 30 min, the patient’s serum sodium decreased significantly and his neurologic status improved. The patient left the hospital 4 days later without any neurologic deficits. Such a rapid sodium correction has never, to our knowledge, been reported in a patient with a positive neurologic outcome.

In cases of slowly developing hypernatremia, it is well documented that rapid correction can result in cerebral edema and death. The underlying cause of hypernatremia must be recognized (e.g., pure water loss, hypotonic sodium loss), and treatment tailored appropriately (14). The rapid correction of chronic hypernatremia has the potential to cause cerebral edema, and can also result in central pontine myelinolysis, also known as osmotic demyelination syndrome (ODS) (15). ODS is often encountered in the case of inappropriately rapid correction of chronic hyponatremia. The mechanism is not fully understood, but is thought to be related to the loss of brain volume and osmotic shrinkage of cells that occurs (16). In this patient, ODS potentially could have developed due to the excess electrolytes and subsequent osmotic shrinkage in the brain parenchyma due to the ingestion of a high solute load.

This patient presented with apparent status epilepticus, and he remained at continued risk for seizures, subsequent anoxic brain injury, and intracranial hemorrhage while his sodium remained profoundly elevated. Due to the exceedingly rapid rise, the decision was made to aggressively lower the sodium level using free water, followed by a more conservative, slower correction rate after initial stabilization. Although his MRI revealed edema due to postictal changes, he had no radiographic abnormalities related to the rapid correction of his serum sodium.

Limitations

The patient in this case report presented with a clear history and timeline of acute sodium chloride ingestion. His renal function was appropriate and adequate for correction using intravenous free water replacement. However, such an aggressive approach to rapid correction may be deleterious if the history is unclear, the rate of rise of sodium is unknown, or the patient does not have adequate renal function.

CONCLUSION

Emergency physicians should consider rapidly lowering serum sodium with hypotonic intravenous fluids as a potential management strategy for acute hypernatremia secondary to massive salt ingestion. The rapidity of correction should be based on the rapidity of the ingestion, the time from ingestion, the patient’s clinical presentation, and comorbid medical conditions that could limit large-volume fluid infusions. Clinician judgment plays a significant role in balancing the risks of slow
correction, including brain parenchymal contraction and intracranial hemorrhage, with the risks of rapid correction, including cerebral edema.

REFERENCES