Clinical Observations

Sodium intoxication caused by use of baking soda as a home remedy

MARK S. PUCZYNSKI,* MD
DANIEL G. CUNNINGHAM,† MD
JOANNE C. MORTIMER,* MD

Excessive sodium ingestion is an unusual cause of hypernatremia in childhood. Incorrectly mixed formula, excessive administration of sodium bicarbonate for cardiac arrest and heparinized saline flushes have led to excessive total body sodium content.[1-3] We describe an unusual case of sodium intoxication.

Case report

A 3-year-old girl presented to the emergency department of another hospital with a 5-day history of fever, vomiting, lethargy and gait imbalance. She appeared acutely ill. Initial serum electrolyte determinations revealed very high levels of sodium (210 mmol/l) and chloride (185 mmol/l).

When the patient was transferred to Loyola University Hospital she demonstrated minimal response to verbal and painful stimuli. Her weight was 14.1 kg, temperature 39.6°C, heart rate 128 beats/min and blood pressure 96/50 mm Hg. She had a large contusion over the right orbit and several minor facial lacerations. Neurologic examination revealed a slight resistance to neck flexion, hyperactive deep-tendon reflexes and bilateral extensor plantar responses. She appeared moderately dehydrated and had doughy skin turgor and dry mucous membranes. Initial laboratory investigation gave the following values in blood or serum: glucose 257 mg/dl (14.3 mmol/l), sodium 210 mmol/l, potassium 2.9 mmol/l, chloride 175 mmol/l, carbon dioxide content 10 mmol/l, urea nitrogen 72 mg/dl (urea 25.7 mmol/l), creatinine 1.2 mg/dl (106 μmol/l), osmolality 456 mOsmol/kg and pH 7.33. Arterial blood gas studies showed a partial pressure of oxygen of 110 mm Hg, a partial pressure of carbon dioxide of 19.3 mm Hg, a pH of 7.34 and a standard bicarbonate deficit of 14 mmol/l. The results of liver function studies and the coagulation profile were normal, as were skull roentgenograms and a computed tomography scan. The child's foster parents later admitted to giving the child 8 to 10 tablespoons of baking soda a day for 10 days because she was having mild abdominal pain.

The hypernatremia was slowly corrected with fluid therapy. Initially we used 5% dextrose and 0.9% saline, the rate of administration being calculated to correct the dehydration over a 48-hour period. After 18 hours the serum sodium level was 190 mmol/l and treatment was changed to 5% dextrose and 0.45% saline. Thirty-six hours after her admission to hospital her serum sodium level was 175 mmol/l and her serum osmolality 352 mOsmol/kg. Her blood glucose level had reached 412 mg/dl (22.9 mmol/l), but insulin therapy was not begun. Treatment was changed to 5% dextrose in water, the rate of administration being calculated to meet maintenance requirements. After 3 days in hospital the patient had a serum sodium level of 155 mmol/l and was normoglycemic. At this time she had a brief generalized seizure, which responded to treatment with diazepam. By the next day her serum sodium level and serum osmolality had returned to normal. Over the next 3 weeks her neurologic status slowly improved. She was discharged to a new foster home. Two years after discharge she was experiencing mild language and cognitive delays.

Discussion

Our patient's serum sodium level was high because of excessive sodium ingestion. The slightly acidic pH of 7.33 was an unexpected finding. We cannot explain the increased serum chloride level, since the foster parents denied having given the patient any substance other than baking soda. The absence of alkalemia may be explained by metabolic acidosis secondary to acute renal failure. After the patient was rehydrated the pH and renal function returned to normal.

Specific dosage recommendations for the use of baking soda as an antacid are printed on the container, the maximum recommended dose for an adult being 8 half-teaspoons over a 24-hour period. A formal investigation of the child's foster parents failed to reveal intentional abuse; evidently they thought sodium bicarbonate was a panacea for abdominal pain.

Previous reviews have noted a high incidence of acute neurologic symptoms associated with hypernatremia, including altered consciousness, hypertonicity, seizures and coma, which result from intracranial vascular damage and cerebral edema during rapid rehydration.[4,5] Permanent neurologic sequelae are

From the department of pediatrics, Stritch School of Medicine, Loyola University, Maywood, Illinois

*Assistant professor
†Associate professor

Reprint requests to: Dr. Mark S. Puczynski, Department of pediatrics, Stritch School of Medicine, Loyola University, 2160 S First Ave., Maywood, IL 60153, USA

found in 16% to 37% of such patients.[4,5] A common finding is generalized cerebral dysfunction manifested by impaired intellect, attention and behaviour. The mild language and cognitive delays experienced by our patient may have been unrelated to the hypernatremia since she had previously exhibited delayed language development.

On the second day in hospital our patient was noted to have hyperglycemia. No insulin was administered for fear that a rapid reduction in the serum glucose level would enhance the risk of cerebral edema. Hyperglycemia has been described in about half the patients with hypernatremic dehydration.[6] Injection of a hypertonic solution has been shown to affect carbohydrate metabolism in experiments with animals.[7] Hyperosmolality may adversely affect the cellular metabolism of glucose by increasing the level of circulating catecholamines or injuring the central nervous system at the hypothalamic level.

Our patient's hypernatremia was slowly corrected with fluid therapy, as a rapid reduction in the serum sodium level carries the risk of cerebral edema and seizures. Another form of management would have been peritoneal dialysis,[8] which has previously been reported to correct serum sodium levels rapidly in cases of severe salt intoxication; however, its use is not without complications.[2]

References

1. FINBERG L, KILEY J, LUTTRELL CN: Mass accidental salt poisoning in infancy. A study of a hospital disaster. *JAMA* 1963; 184: 187–190
2. SAUNDERS N, BALFE JW, LASKI B: Severe salt poisoning in an infant. *J Pediatr* 1976; 88: 258–261
3. WHANG R: "Bicarbonate overshot": an indication for acetazolamide therapy. *South Med J* 1975; 68: 733–734
4. MACAULEY D, WATSON M: Hypernatremia in infants as a cause of brain damage. *Arch Dis Child* 1967; 42: 485–491
5. MORRIS JONES PH, HOUSTON IB, EVANS RC: Prognosis of the neurological complications of acute hypernatremia. *Lancet* 1967; 2: 1385–1389
6. FINBERG L: Hypernatremic (hypertonic) dehydration in infants. *N Engl J Med* 1973; 289: 196–198
7. NITZAN M, ZELMANOVSKY S, TIKVA P: Glucose intolerance in hypernatremic rats. *Diabetes* 1968; 17: 579–581
8. MILLER NL, FINBERG L: Peritoneal dialysis for salt poisoning. Report of a case. *N Engl J Med* 1960; 263: 1347–1350

Eikenella corrodens endocarditis: case report and review of the literature

STEPHAN J. LANDIS, MD, FRCP[C]
JOHN KORVER, ART

Eikenella corrodens, a bacterium usually found in the oral cavity and gastrointestinal tract, has been reported to cause cutaneous, wound and abdominal abscesses as well as meningitis, osteomyelitis and endocarditis.[1] Although it is most often associated with mixed bacterial infections, it may also be the sole infecting agent.

We report the first case, to our knowledge, of *E. corrodens* endocarditis involving a porcine heterograft valve.

Case report

A 58-year-old man was admitted to hospital with a 4-week history of malaise, night sweats, palpitations and low back pain. Five years earlier he had undergone an aortic porcine heterograft valve replacement for calcific aortic valve stenosis. He denied use of illicit drugs.

Four months before his admission to hospital the patient had had his teeth cleaned by his dentist. Although he was not allergic to penicillin he was asked to take erythromycin, 250 mg four times daily, 1 day before, the day of and the day after the cleaning procedure as prophylaxis.

At the time of admission the patient was obese and dehydrated and looked ill. His blood pressure was 90/60 mm Hg, pulse rate 90 beats/min and irregular, and oral temperature 38°C. The appearance of the oral cavity indicated poor oral hygiene, but there was no tenderness. His sternotomy scar was well healed. Examination of the heart revealed no enlargement but a variable first heart sound, a normal second sound, a grade 2/6 systolic ejection murmur in the aortic area, a grade 2/6 systolic murmur in the mitral area and a grade 1/6 diastolic murmur along the left sternal border. The tip of the spleen was felt. A conjunctival hemorrhage was noted in the left eye. The rest of the physical examination, including funduscopy, revealed no other peripheral evidence of endocarditis or concurrent skin infection.

The hemoglobin level was 110 g/l, the leukocyte count 29.0 × 10^9/l (50% mature polymorphonuclear leukocytes, 34% band cells, 10% monocytes and 6% lymphocytes), the platelet count 0.23 × 10^9/l and the erythrocyte sedimentation rate (ESR) 88 mm/h. The following levels were found in blood or serum: urea nitrogen 151 mg/dl (urea 54 mmol/l), creatinine 3 mg/dl (265 μmol/l), sodium 126 mmol/l, potassium 4.7 mmol/l, chloride 96 mmol/l and carbon dioxide content 20 mmol/l. Urinalysis revealed occasional erythrocytes but no casts. The prothrombin time, partial thromboplastin time and thrombin time were normal. A chest roentgenogram revealed left ventricular enlargement, and an electrocardiogram atrial fibrillation and left ventricular hypertrophy. Two-dimensional echocardiography showed moderate left ventricular enlargement, a thickened mitral valve with diastolic fluttering of its anterior leaflet, and a prominent echo in the angle of the left ventricular outflow tract near the base of the aortic valve prosthesis and the mitral valve. It was thought that the echo might represent a

From the division of infectious diseases, department of medicine, and the department of laboratory medicine, Hamilton General Hospital, McMaster University, Hamilton, Ont.

Reprint requests to: Dr. Stephan J. Landis, Department of medicine, Hamilton General Hospital, 237 Barton St. E, Hamilton, Ont. L8L 2X2