Case Report

A Non-Fatal Case of Sodium Toxicity

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Abstract

A non-fatal case of sodium toxicity in a six-year-old boy is presented. Hypernatremia is the clinical term for an excessive concentration of sodium relative to water in the body. The diagnosis of hypernatremia was made at serum sodium (Na⁺) concentrations exceeding 150 mEq/L, and few people have been reported to survive concentrations greater than 160 mEq/L. This case involves a six-year-old boy who was taken to the hospital following a seizure attack, and lab analyses revealed serum sodium (Na⁺) levels of 234 mEq/L and serum chloride (Cl⁻) levels of 205 mEq/L. Clinical tests ruled out diabetes insipidus, dehydration, renal pathology, and other primary causes of hypernatremia. The child’s purported history of pica, and the lab results indicating corresponding increases in levels of serum sodium (Na⁺) and serum chloride (Cl⁻), led to a diagnosis of acute sodium toxicity by ingestion of sodium chloride. A search of the boy’s house led to the discovery of rock salt in the cabinet and a container of table salt. Extrapolating from the serum sodium (Na⁺) level, it was estimated that the child had ingested approximately four tablespoons of rock salt, leading to the acute toxicity. A literature search revealed that the serum sodium (Na⁺) concentration in the present report was the highest documented level of sodium in a living person.

Introduction

Sodium (Na) is a frequently encountered element, and it is also a key component of the body’s electrolyte system. The daily intake of sodium is chiefly in the form of common salt, or sodium chloride (NaCl). Hypernatremia, or very high sodium concentration, may be due to a variety of causes like dehydration, diabetes insipidus (DI), sodium ingestion, and kidney disease. The cutoff for a diagnosis of hypernatremia is a serum sodium (Na⁺) level of 150 mEq/L. However, reports describe a few non-fatal cases with serum sodium (Na⁺) levels ranging from 193 to 209 mEq/L (1–3). Prior to this report, Dominguez et al. (4) have documented a case of hypernatremia with sodium ion levels of 216 mEq/L, which was the highest known level of serum sodium (Na⁺), in a non-fatal case. The following is a case of sodium toxicity secondary to the suspected ingestion of rock salt in a six-year-old boy.

Case History

A six-year-old boy was taken to the hospital following respiratory failure after a seizure attack. The patient was thrashing in his sleep, thus awakening the foster parents, who attempted to give him CPR after he turned unresponsive. The foster parents called emergency services, and an ambulance transported him to the hospital. At the hospital, the lab findings indicated serum sodium levels in excess of 200 mEq/L; the result was beyond the linearity of the instrument. The child was then transferred to a children’s hospital, where the presenting symptoms included respiratory failure and altered mental status.

A CT scan revealed the presence of bilateral chronic subdural hematomas. Lab results at the children’s hospital indicated severe hypernatremia with a serum sodium (Na⁺) value of 234 mEq/L. Additionally, hyperchloremia was observed, with a serum chloride (Cl⁻) value of 205 mEq/L. Other metabolic abnormalities included hypokalemia and metabolic acidosis. In the ER, the patient was administered fluids, lorazepam (Ativan®), and vecuronium. Subsequently, the patient was placed on saline, sodium bicarbonate, potassium chloride, potassium phosphorus, and Ringer’s lactate.

The patient had a general history of pica, a disorder involving the ingestion of non-food items, and a specific history of glass ingestion. He displayed signs of a burn wound on his left hand, and the wound looked approximately two-weeks old. Records also revealed that he had a past history of physical and sexual abuse and was diagnosed with Post Traumatic Stress Disorder (PTSD) and Attention Deficit Hyperactivity Disorder (ADHD). The foster parents of the child reported that he complained of thirst, urinated frequently, and had vomited twice in two days.
However, they denied any knowledge of salt ingestion.

The sodium levels started to progressively decrease without any additional treatment, and this was followed by a corresponding improvement in neurological status. The patient had a past history of seizure activity and was taking the prescription anti-convulsant drug divalproex sodium (Depakote®). Other medications administered to the patient during the hospital stay included albuterol, epinephrine, norepinephrine, dopamine, fosphenytoin, lorazepam, rocuronium, ranitidine (Zantac®), clindamycin, desmopressin acetate (DDAVP), vaso-pressin, and hydrochlorothiazide.

### Discussion

Sodium is a critical electrolyte and changes in sodium concentration could manifest as hyponatremia or hypernatremia. Increase in the sodium concentration may imply a relative decrease in the water concentration, or an absolute increase in sodium concentration (1). Hypernatremia results in cellular dehydration, because of the shift of water from the intracellular to the extracellular compartment (5). Physiological compensatory mechanisms to combat the increase in Na concentrations include polydipsia (thirst) and the release of the antidiuretic hormone (ADH). As the sodium concentration in the body increases, it leads to the release of ADH/vasopressin, which decreases diuresis and the ensuing water loss, thereby maintaining homeostasis. Coulthard et al. (6) refer to the rare disorder Essential Hypernatremia, in which escalating levels of sodium do not produce a thirst response or release of antidiuretic hormone (ADH). The etiology of hypernatremia includes dehydration due to diarrhea and vomiting, use of diuretics, renal disease, DI, and excessive intake of sodium. The clinical features include thirst, lethargy, weakness, nausea and vomiting, hypertension, and irritability (5). Seizures, coma and pulmonary edema have also been reported in certain cases of hypernatremia (5,6). Serum Na levels above 160 mEq/L have been associated with a high mortality rate (60%) and neurologic damage (7). DI, which is a disorder of decreased secretion of ADH, is a common cause of hypernatremia. There have also been reports of lithium-induced DI leading to hypernatremia (8).

This patient, however, did not have a history of medication with lithium, which is often used to treat bipolar disorders. Additionally, the lab findings were not consistent with a diagnosis of DI, with the urine appearing normal (specific gravity 1.01 and osmolality 457 mOsm/L) instead of dilute, as expected in cases of DI and renal pathology (300 mmol/kg to 50 mmol/kg) (6). The administration of DDAVP and vasopressin, drugs of choice for the treatment of DI, did not produce any results, thus ruling out DI as the cause of hypernatremia in this case. No signs of dehydration were evident; the urine output was elevated, heart rate was normal, and there was no increase in hemoglobin/hematocrit values. None of the medications taken by the patient were known to cause this electrolyte imbalance. The patient was also hyperchloremic (205 mEq/L), and this finding was not consistent with any etiology other than the ingestion of sodium chloride (NaCl).

A search of the patient’s house revealed a container of table salt on a countertop and rock salt in a closet. The similarity between the serum concentrations of Na⁺ (234 mEq/L) and Cl⁻ (205 mEq/L) and the patient’s history of pica suggested that the hypernatremia might have been a consequence of acute NaCl ingestion. Salt poisoning is known to increase the serum sodium (Na⁺) concentrations and also leads to an increased osmolality, often above 400 mOsm/kg, due to impaired renal excretion (9).

Pica refers to the ingestion of non-food substances, which may include dirt, clay, chalk, cigarette ashes, sand, soil, and paint. Mental retardation (10), stress resulting from maternal deprivation (10), parental neglect, and a variety of behavior disorders (11) have been suggested as possible etiologies of pica. This patient was a victim of sexual abuse, neglect, and maternal deprivation and had a purported history of pica, particularly the ingestion of glass.

One of the statements by the child alleged that his foster mother used to make him drink salt water. Adrogue and Madias (12) list sodium-chloride emetics as a possible cause of hypertonic sodium gain. Salt-water emetics have been known to cause hypernatremia; however, cases of salt water emetic poisoning are not as frequently encountered as before (5,13,14). Although there was no evidence to determine the manner of ingestion, we hypothesized that the child had ingested NaCl, as a result of his pica condition, and thereby developed acute sodium toxicity. Shapiro et al. (10) describe the case of a young woman with salt pica, secondary to iron deficiency. In the present case, however, there was no evidence of a deficiency of iron in the diet.

Being a monobasic salt, NaCl contains similar proportions of Na⁺ and Cl⁻, and both serum sodium (Na⁺) and serum chloride (Cl⁻) levels were elevated in this patient. In the current case, information about the methodology used by the ER to evaluate electrolyte concentration was not available. However, these electrolytes are typically assessed by ion selective electrodes. We performed a series of back calculations to estimate the approximate quantity of ingested sodium that would produce a serum concentration of 234 mEq/L. Cases of salt poisoning in the literature describe lethal doses ranging from 0.75 g/kg body weight to 3 g/kg body weight (15). Extrapolating from the aforementioned data, the lethal dose for a child who weighs 25 kg would range between approximately 19 g and 75 g. Assuming the normal sodium concentration to be 140 mEq/L, and using the following equation, the ingested quantity of NaCl was calculated to be approximately 83 g.

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\text{Dose} = \frac{C_p \times V_d}{C_p - \text{plasma concentration}}
\]

where \(V_d\) = volume of distribution and \(C_p\) = plasma concentration.

The average tablespoon of rock salt contained about 21.6 g of NaCl; therefore, the patient was suspected to have consumed about four tablespoons of rock salt.

The CT scan of the patient exhibited the presence of bilateral subdural hematomas. One of the prevailing theories in the 1950s was based on the dehydration induced by hypernatremia.
leading to shrinkage of the brain (1), thereby decreasing its size and stretching the bridging veins, resulting in a subdural hematoma (9), subarachnoid hemorrhage, and cerebral bleeding (12). Handy et al. (9), however, failed to identify any causal link between hypernatremia and the subsequent occurrence of subdural hematomas.

References