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Case Report

Salt Poisoning in a 6-year-old Child: A Case Report

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ABSTRACT

Background: Hypernatremia due to salt poisoning is clinically rare and standard care procedures have not been established. We report this case of pediatric salt poisoning due to chronic craving for table salt. Immediate recognition of salt toxicity and aggressive resuscitative measures are critical in the treatment of this lethal poisoning. Despite heroic measures, pediatric deaths due to salt toxicity still occur from irreversible neurological damage.

Case presentation: A 6-year-old boy presented to our pediatric emergency department referred from other hospital with fatigue, polyuria and polydipsia and remarkable hypernatremia with a serum sodium concentration of 203 mmol/L. The initial lab work showed blood glucose 5.7 mmol/l, Serum Sodium (203 mmol/l) from referring hospital) and venous blood gases showed PH 7.26 PCO2 47 mmHg HCO3 21.5 mmol/l. We reduced his serum sodium level slowly over 84 hours, as its toxicity happened over a period of 2 months.

Conclusion: After reviewing instances of resuscitation following salt intoxication, aggressive rapid correction of serum sodium concentration should only be considered in acute phases of hypernatremia within a few hours from ingestion.

Keywords: Salt poisoning; Children; Toxicity

INTRODUCTION

Salt poisoning is a serious cause of hypernatremia in children and represents a diagnostic challenge for the treating physician. The most important aspect is to actually consider this diagnosis, given its rarity and the severe medical and social consequences associated with it [1-3]. Moreover, salt poisoning is exceedingly rare. While the true incidence is unknown, as the diagnosis is likely missed in some cases, the annual incidence of recognized non-accidental salt poisoning in the UK in one study was approximately 1 in 1,00,000 children aged under 16 years [4]. Hypernatremia is a common electrolyte disturbance, most often caused by volume depletion. Hypernatremia due to sodium excess occurs less frequently and fatal hypernatremia solely from ingestion of table salt is reare [5]. Salt toxicity is a rare form of hypernatremia that typically occurs after a single massive ingestion of salt or it can occur over a period of time (days to months). It is a dangerous imbalance capable of causing significant neurological injury; quick recogition of salt toxicity is crucial to allow treatment before permanent brain injury occurs [6].

CASE REPORT

A 6-year-old boy presented to our pediatric emergency department, referred from other hospital with polyuria and polydipsia for two weeks and fatigue and inability to walk for 1 day. There was no history of diarrhea or vomiting. He was not having any underlying medical condition before, except moderate language delay. Patient was referred form peripheral hospital because his serum sodium was 203 mmol/l. He was received a normal saline bolus at rate 20 ml/kg in referring hospital. On arrival to our emergency, the patient was conscious, oriented and alert. Vital signs, blood pressure of 99/60 mmHg, heart rate of 106 beats/min, respiratory rate of 26 breath/min, and temperature of 36.7°C. A general examination revealed weight 20 kg, no dysmorphic feature. There were no signs of dehydration. Hydration status was normal. Examination of cardiovascular, respiratory and gastro intestinal system revealed unremarkable findings. Central nervous examination showed Glasgow Coma scale of 15/15, alert and active. Muscle tone and reflexes were normal. Pupils were equal and reactive in both sides. The Initial laboratory investigations yielded the following results; CBC hemoglobin was 9 g/dl, WBC 8 × 10^3/ µl and platelet count was 78 × 10^3/ µl. Serum chemistry; serum sodium,185.4 mmol/L; serum chloride 156 mmol/L; serum potassium 2.9 mmol/L, serum calcium 2.1 mmol/l, serum phosphorus 0.71 mmol/l, serum magnesium 0.96 mmol/l, BUN 8.2 mmol/l; creatinine, 53.8 µmol/l, plasma glucose, 3.7 mmol/l. The calculated serum osmolality was 390 mOsmole/kg. Initial venous blood gas measurements while the patient was breathing in room air was

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pH 7.26, PCO2 47 mmHg, PO2 74 mmHg, HCO3 21.5 mmol/l and anion gap 8 mmol/l (normal 8 to 16). Urinary electrolytes showed Urine sodium 250 mmol/l, Urine chloride 275 mmol/l, Urine potassium 9.9 mmol/l, Urine phosphorus 8.6 mmol/l, Urine magnesium 3.7 mmol/l, Urine Osmolality 840.2 mosmole/kg H_2O. Chest x-rays showed normal findings. BUN/Creatinine ratio was 41. Reviewing further history from both parents and grandmother, they admitted that he had poor growth since birth and they also noticed that he is craving for salt in his meals. They were adding one teaspoon of salt to his meals 2-3 times per day for last 2 months and he was insisting to do so while his parents were trying to prevent adding salt to his meals. With history, physical finding and laboratory investigations patient was diagnosed as a case of salt poisoning. He was admitted to pediatric intensive care unit. Patient was treated with hypotonic intravenous fluid (0.45% saline in 5% dextrose water) to provide more free water. As the patient was conscious he was allowed to take orally specially water. Serum sodium was closely monitored and aim was to reduce over 72-84 hours. His urine output was between 1-2 ml/kg/hour over the 4 consecutive days. Serum sodium declined gradually to normal level over 3 days. He was monitored specially for neurological complications (decrease level of consciousness, convulsions). No neurological complications were noted. Patent was discharged home with outpatient follow up appointment.

DISCUSSION

Salt toxicity is a rare form of hypernatremia that typically occurs after a single massive exposure to salt (sodium chloride) over a short period of time, such as minutes to hours [2,7,8]. It is difficult to determine the specific levels of salt intake capable of causing salt toxicity. Using data from 2 children, Campbell et al. [9] estimated that the lethal dose of salt ingestion in children was less than 5 teaspoons. In children, mortality in salt toxicity is high when the plasma sodium level

exceeds 190 mEq/L.2 [10]. Ingestion of as little as two tablespoons of salt has been reported to increase serum sodium levels by as much as 30 mmol/L with the potential to cause severe irreversible neurological damage [8,11]. Many of the fatalities were related to the administration of salt therapeutically as an emetic agent, where toxicity may have been, in part, related to co-ingested drugs. Our patient presented with severe hypernatremia. The way of distinguishing the causes hypernatremia is challenging as the differential diagnosis includes hypernatremic dehydration, diabetes insipidus and rarely salt intoxication. Hypernatremia is due to either salt excess e.g. salt poisoning or due to water deficit e.g. hypernatremic dehydration and diabetes insipidus. We initially suspect hypernatremic dehydration but careful history from parents, he never had gastroenteritis. We also consider diabetes insipidus but we could able to exclude by consequent analysis of both plasma and urinary electrolytes and osmolality. With the history of extra salt ingestion for a period of time, salt intoxication is likely diagnosis in our patient. Diagnosis of salt poisoning can be supported by recent weight gain and measuring fractional excretion of sodium (FENa). Parent had no record of pre admission weight but he gains weight after therapy started. We could not do FENa because consequent urinary creatinine could not be done in our patient. A massive ingestion of salt causes the plasma sodium concentration to elevate within minutes and peak within hours [2,10]. As a result, the cells shrink because fluid is drawn osmotically from the intracellular spaces; this process is particularly dangerous in brain cells. Irreversible neurological injury can result; intracranial hemorrhages are found commonly during the autopsy of salt toxicity victims and are believed to result from shearing forces in the brain due to osmotic fluid shifts [10,12,13]. In an effort to correct the fluid shift from the cells, the brain cells pump in electrolytes and endogenous osmolytes to regulate cell size [7,14]. Although brain injury in salt toxicity is attributed generally to osmotic fluid shifts, Blohm et al. point out that cerebral edema may develop in response to diffuse cellular injury, as evidenced in case reports in which cerebral edema occurred in patients before treatment [2]. The symptoms associated with salt toxicity can include nausea, vomiting, abdominal cramps, and diarrhea, presumably due to the mucosal irritation and fluid shifts associated with the hypertonic solution. The neurological symptoms may include lethargy, weakness, twitching, seizures, and coma [15]. Without a clear history of salt intake, it may be difficult to distinguish salt toxicity from hypernatremia due to excessive water loss [2]. Occasionally, patients with acute salt poisoning may develop acute kidney injury and oliguria; in such patients, dialysis is indicated [5]. In addition, seizure prophylaxis is important because seizures are commonly present in patients with salt toxicity [2]. We consider long term hypernatremia in our patient because salt was given over a period of two months and no neurological symptoms were present. Management is recommended in chronic hypernatremia (>24 hour) is to lower the plasma sodium level with hypotonic intravenous fluids and target should reduce the sodium not more than 12 mmol/L/day (0.5 mmol/L/day) [10]. The plasma sodium concentration can be safely lowered more quickly when salt toxicity has been present for less than 24 hours, because brain's adaptation to hypernatremia is incomplete, minimizing the risk of neurological injury from rapid correction of the hypernatremia [16]. Blohm et al. recommend rapid correction of the plasma sodium concentration in pediatric patients who present within 24 hours of the high salt ingestion [2].

CONCLUSION

Salt toxicity is very rare condition around 1 in 10 million. Mortality rate is high amongst the children. It can be happened in all ages and reasons can be varied from use of salt water as an emetic, child abuse and a suicide attempt. For patients whose massive exposure to salt is recent (such as minutes to hours), rapidly reducing the serum sodium concentration may prevent irreversible neurological injury, but when hypernatremia is occurred over a longer period of time, the gradual reduction of serum sodium is advised along with close monitoring in intensive care unit settings and nephrologist consultation.

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