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Case Report
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Hyponatremia-Induced Seizures Secondary to Magnesium Citrate Colon Cleansing Agent

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Abstract

Mild and clinically insignificant electrolyte disturbances are a well-recognised complication of all bowel preparation. Seizure as a complication is very rare. Magnesium citrate, a hyperosmolar substance, is often used for bowel preparation before colonoscopy and colon surgery. We report a 74-years-old female, with no prior history of seizures, who had her first seizure associated with hyponatremia following ingestion of magnesium citrate for bowel preparation. A careful correction of serum sodium must be undertaken. The patient improved her mental status to baseline after intravenous infusion of hypertonic saline. The other causes of hyponatremia and seizure were ruled out. This case highlights the association between magnesium citrate bowel preparations with significant hyponatremia and seizure. The physicians in the ED and general practice need to be aware of this serious complication of magnesium citrate bowel preparation to diagnose them early and initiate timely management.

Keywords: Hyponatremia; Bowel preparation; Seizures; Hyponatremic seizures; Magnesium citrate

Introduction

Hyponatremia (<135 mEq/L) usually represents a relative excess of water in relation to sodium in the blood. Neurologic symptoms of hyponatremia include confusion, somnolence and loss of consciousness, seizures, permanent brain damage, and brain herniation. Only a few cases of hyponatremic seizure associated with magnesium citrate bowel preparation are published [1-3].

Case Presentation

A 74-years-old female was brought to the Emergency Department (ED) of a comprehensive cancer center with a complaint of confusion and tonic-clonic seizure. The patient had two episodes of voluminous watery bowel movement few hours following magnesium citrate intake (4 hours before the presentation to the ED) to prepare her bowel for colon surgery. The patient had no previous history of seizure, head trauma, smoking, alcohol intake or illicit drug use. The patient had a history hypertension, hypothyroidism, depressive disorder, and recently diagnosed localized colon cancer. She had no history of chemotherapy. Her medications were losartan (50 mg daily), Metoprolol extended release (100 mg daily), levothyroxine (88 mg daily) and escitalopram (20 mg daily).

At the ED, the patient's Glasgow Coma Scale (GCS) was 10 (E2, M5, V3), vitals were normal except for blood pressure 175/85 mmHg. The patient was moving all four extremities randomly. No full neurological examination was carried out because of her mental condition. Other systemic examinations were unremarkable. Computed tomography (CT)-head was normal. Bedside Electro-Encephalography (EEG) showed no epileptiform discharges. Initial laboratory work-ups showed sodium 115 (135-145 mEq/L), magnesium 1.6 (1.8-2.2 mg/dL), lactate 1.2 (0.5-2.1 mg/dL), serum osmolarity 250 (275-295 mOsm/kg), urine osmolarity 404 (300-900 mOsm/kg of water), and urine sodium 142 (random normal around 20 mEq/L). Other serum electrolytes, BUN, creatinine, TSH and free T4 were within normal range. The patient received 200 ml of 3% saline in 40 minutes and responded well with improvement in her mental status. She had polyuria following the hypertonic saline infusion. Four hours later, repeat lab showed serum sodium 123 mEq/L, urine sodium 62 mEq/L, and urine osmolarity 140 mOsm/kg of water. The patient received one dose of desmopressin to prevent overcorrection of serum sodium. The serum sodium was serially monitored (Figure 1).

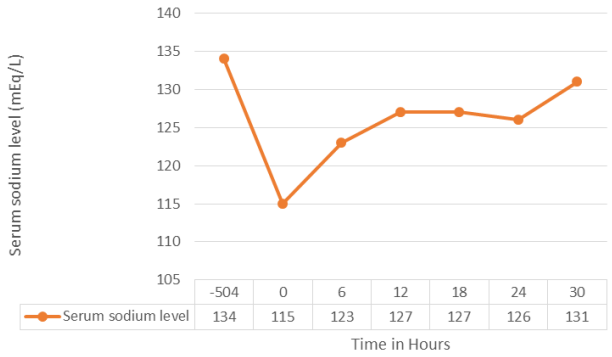

Figure 1: The chart showing serum sodium level before and after the patient presented to the Emergency Department(ED). Zero (0) hour represented when the patient presented to our emergency department. Serum sodium was closely monitored. The serum sodium was rapidly corrected, shown at 12 hours when the patient received one dose of desmopressin. The patient had serum sodium level 134 mEq/L one month before presentation to the ED.

The patient was admitted to inpatient service for close monitoring. Subsequently, the patient remained in baseline neurological status with normal serum sodium level and was successfully discharged home on day-4 without any change in home medications. Serum sodium level remained normal on outpatient follow-up.

Discussion

Magnesium citrate, being a hypertonic solution, extracts and then retains fluid by osmosis into the intestinal lumen. This stimulates the colon and increases the frequency of peristalsis to empty the colon and rectum of faecal contents resulting in osmotic diarrhoea [4]. The development of severity of neurological symptoms is related to the rapidity of the change in the serum sodium concentration rather than severity of hyponatremia [5]. In this patient, magnesium citrate-induced osmotic diarrhoea led to the loss of both water and sodium. However, concurrent intake of water and decreased urinary output (likely secondary to increased tubular reabsorption) could contribute to the rapid decline in serum sodium level than intravascular water content. Additionally, the elevated blood pressure causes the physiological suppression of aldosterone. Hence the patient lost sodium in the urine in spite of being in the hyponatremic state. The increased urinary output (with more water than sodium) after hypertonic saline infusion, likely secondary to osmotic diuresis and auto-correction by her healthy kidneys, led to the rapid increase serum sodium level. Desmopressin helped to prevent the overcorrection of serum sodium level. Current guidelines recommend correcting serum sodium concentration by no more than 8 mEq/L/day for patients at high risk of osmotic demyelination syndrome, and 10-12 mEq/L in 24 hours and 18 mEq/L in 48 hours for patients at average risk of osmotic demyelination syndrome [6]. The use of desmopressin is recommended to prevent or slow high rates of serum sodium correction [7]. We excluded the possibility of the syndrome of inappropriate antidiuretic hormone (SIADH) due to escitalopram use because the patient continued to have normal serum sodium in spite of the continuous use of the medication. The patient neither had metastatic disease nor on any chemotherapy.

Since cancer patients often are on chemotherapy, where hyponatremia is a possible risk, then physicians need to be more vigilant regarding cancer patient monitoring when prescribing magnesium citrate pre-colonoscopy or colon surgery, given the side-effects of magnesium citrate may cause a potentially dangerous, if not lethal, decrease in their electrolytes, especially sodium. Patient with advanced age, potential chronic sodium depletion, such as patients on thiazide diuretics, or with baseline electrolyte disturbances are at high risk of developing significant hyponatremia [1,3]. Concurrent ingestion of free water, as in this case, may contribute to the development of severe hyponatremia. Careful monitoring is needed in such patients when prescribing the magnesium citrate bowel preparation to prevent the potential complication and identity in time and manage appropriately. There is a need for more research in the future to understand the drug-induced complication better.

Conclusion

This case highlights the association between magnesium citrate bowel preparations with hyponatremic seizure, especially in an older cancer patient. ED physicians need to be aware of these potential complications to diagnose them early and initiate timely management.

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