International Journal of Nephrology and Renovascular Disease

Dovepress

open access to scientific and medical research

Open Access Full Text Article

CASE REPORT

Hypernatremia in a patient treated with sodium polystyrene sulfonate

This article was published in the following Dove Press journal:
International Journal of Nephrology and Renovascular Disease
14 November 2010
Number of times this article has been viewed

Manish Nepal
Ion Dan Bucaloiu
Evan R Norfolk

Geisinger Medical Center,
Department of Nephrology,
Danville, PA, USA

Abstract: Severe hyperkalemia requires urgent medical attention and correction in order to prevent arrhythmic complications. Sodium polystyrene sulfonate (SPS) is a cation exchange resin commonly used in the management of hyperkalemia. A recent review raised concerns regarding its effectiveness and potential adverse effects. Hypernatremia in adults in the setting of sodium polystyrene sulfonate therapy has not been described in the literature. We report the case of a woman who developed hypernatremia in the setting of excessive SPS administration and hope to increase awareness among clinicians regarding this potential side effect of SPS therapy.

Keywords: SPS, hyperkalemia

Background

Hyperkalemia is a medical emergency primarily due to its cardiotoxic effects. In addition to cardiac membrane stabilization with intravenous calcium and redistribution of cellular potassium with insulin, beta antagonists, or bicarbonate, therapy with a cation exchange resin is often used in order to promote net potassium loss in the intestinal tract.[1,2] A recent review raised concerns regarding its effectiveness and potential adverse effects of this agent.[3] Two cases of hypernatremia in the setting of sodium polystyrene sulfonate (SPS) treatment have been reported in neonates.[4] We describe the case of an adult female patient who developed hypernatremia during treatment with SPS.

Case report

A 44-year-old female nursing home resident, with normal baseline renal function was admitted to the hospital for confusion and lethargy. She had poor oral intake for the week prior to admission and recently her narcotic medications have been increased for worsening back pain. Her outpatient medications included furosemide, spironolactone, and lisinopril. Her medical history was notable for cirrhosis due to hepatitis C, hypothyroidism, hypertension, and chronic back pain. Initial evaluation revealed acute renal failure with creatinine of 5.1 mg/dL and hyperkalemia (7.1 mmol/L). Her initial serum sodium was normal (140 mEq/L); however, in the following hours she developed hyponatremia (128 mEq/L). On physical examination she appeared dehydrated and lethargic. Laboratory values on admission and subsequently on follow-up are presented in Table 1. Her electrocardiogram revealed sinus tachycardia without T-wave changes.

She was treated with intravenous insulin and dextrose, intravenous saline infusion, and SPS (60 g every 6 hours). Her urine output improved, and the patient

Correspondence: Manish Nepal
Nephrology, Geisinger Medical Center, Danville, PA 17822, USA
Tel +1 570 271 6393
Fax +1 570 271 5623
Email mnepal1@geisinger.edu

Table 1 Results of laboratory tests[a]

Variables	Normal values	Day 0 (Hours after admission)				Day 1	Day 2	Day 3
		1 hour (SPS given)	6 hours (SPS given)	12 hours (SPS given twice)	16.5 hours			
Sodium (mmol/L)	135–145	140	128	141	149	151	143	140
Potassium (mmol/L)	3.8–4.8	7.1	7.5	8.7	6.1	5.2	4.2	3.7
Chloride (mmol/L)	100–108	95	98	104	110	108	109	105
Carbon dioxide (mmol/L)	23.0–31.9	17	18	18	22	26	24	27
Urea Nitrogen (mg/dL)	8–25	69	72	71	70	65	36	31
Creatinine (mg/dL)	0.6–1.5	5.1	4.7	4.1	3.5	2.0	0.9	0.9
Glucose (mg/dL)	70–110	74	115	96	102	116	102	131
Calcium (mg/dL)	8.5–10.5	7.4	7.6	7.9	7.5	7.9	8.6	8.9
Magnessium (mg/dL)	1.4–2.8	–	–	2.2	–	2.0	–	–
Osmolality (mOsm/kg)	278–305	–	–	–	303	–	–	–

Notes: [a]To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for glucose to millimoles per liter, multiply by 0.5551. To convert the values for calcium to millimoles per liter, multiply by 0.250. To convert the values for magnesium to millimoles per liter, multiply by 0.25. Trend of rising sodium after giving sodium polystyrene sulfonate (SPS) treatment for hyperkalemia. Patient received a total of four doses of 60 g SPS in less than 24 hours, thereby raising the sodium level up to 151 mmol/L, which was normalized by free water hydration.

developed diarrhea over the next hours. A total of 240 g of SPS was administered during a 24-hour period, before the discontinuation of this order. Her serum sodium started at 140 Meq/L and then decreased to 128 mEq/L. Twenty-four hours after admission, her sodium level was 141 mEq/L, then increased over the second hospital day to 151 mEq/L. During the first 2 days in the hospital she passed large amounts of liquid stool. Given the concerning rate of serum sodium rise, an infusion of dextrose water solution was administered and sodium levels were closely monitored. Her thyroid and adrenal function were adequate. Her serum sodium normalized over the next 3 days with replacement of the free water deficit. Her renal function and potassium levels returned to normal. Her low fractional excretion of sodium and physical signs of volume depletion led us to believe that the net free water loss due to SPS-induced diarrhea resulted in this patient's hypernatremia rather than a sodium–potassium exchange mechanism. The patient had no major long-term neurologic complications.

Discussion

SPS is a cation exchange resin used to induce a net potassium loss in patients with moderate to severe hyperkalemia. With the growing chronic kidney disease and end stage renal disease patient population, hyperkalemia is a relatively common electrolyte abnormality encountered in clinical practice. SPS is commonly used when serum potassium levels are high as an adjunct medical treatment measure. Although its safety and efficacy are being questioned,[4] the mechanism of action of SPS is thought to be the net exchange of potassium in the intestinal lumen with sodium in order to maintain electrical neutrality. Each gram of resin may bind as much as 1 mEq of potassium and exchange it for 1–2 mEq of sodium. To facilitate the passage of the resin through the gastrointestinal tract, sorbitol was added to the resin, resulting in a cathartic effect.[2] Complications of SPS therapy, although rare, include ischemic colitis and colonic necrosis,[2,4] hypocalcemia,[5] volume overload, and iatrogenic hypokalemia.

The hypernatremia in our patient is likely due to net intestinal water loss, in the setting of profuse osmotic diarrhea induced by SPS therapy. It is unclear whether salt loading from the cation exchange mechanism played a role in the pathogenesis. Only two cases of combined water loss combined with a sodium loading have been reported in the literature, both in low birth neonates.[3] In our patient, the relatively rapid correction of hyponatremia may also have been contributed to by antidiuretic hormone suppression in the setting of the restoration of intravascular volume. Although the relative change

of serum sodium was significant, the patient's serum sodium returned to the initial normal value within the first hospital day and then increased by 9 mEq/L in the next 24 hours. This change was slightly higher than the currently recommended rates of change,[6] but there were no adverse clinical consequences observed in long-term follow-up.

This case illustrates a potential side effect of excessive administration of SPS in hospitalized adults. As a quality improvement measure, our institutional pharmacy no longer accepts standing orders for SPS. We hope to raise awareness of this potential side effect of SPS among the medical community.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Flinn RB, Merrill JP, Welzant WR. Treatment of the oliguric patient with a new sodium-exchange resin and sorbitol. *N Engl J Med*. 1961;264:111–115.
2. Rashid A, Hamilton SR. Necrosis of the gastrointestinal tract in uremic patients as a result of sodium polystyrene sulfonate (Kayexalate) in sorbitol: an underrecognized condition. *Am J Surg Pathol*. 1997;21:60–69.
3. Sterns RH, Rojas M, Bernstein P, et al. Ion-exchange resins for treatment of hyperkalemia: are they safe and effective? *J Am Soc Nephrol*. 2010;21(5):733–735.
4. Filippi L, Cecchi A, Dani C, et al. Hypernatraemia induced by sodium polystyrene sulphonate (Kayexalate) in two extremely low birth weight newborns. *Paediatr Anaesth*. 2004;14(3):271–275.
5. Ng YY, Wu SC, Cheng CT, et al. Reduction of serum calcium by sodium sulfonated polystyrene resin. *J Formos Med Assoc*. 1990;89:399–402.
6. Sterns RH, Hix JK, Silver S. Treatment of hyponatremia. *Curr Opin Nephrol Hypert*. 2010;19(5):493–498.