Aesculapius Journal (Health Sciences & Medicine)

Volume 5 Article 2

February 2024

A Case of SIADH After Bowel Preparation for Routine Colonoscopy

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Introduction:
Colonoscopy is the gold standard modality for screening of colorectal cancer.[1] Bowel preparation is necessary for adequate visualization during the procedure. Several bowel preparation agents are available, and they are generally safe and well tolerated for the majority of patients.[1,2] Severe electrolyte abnormalities, though uncommon, can occur after use of oral bowel-cleansing preparations.[3]

Case Report:
We present the case of a 78-year old female with a history of hypertension on losartan and diltiazem, hyperlipidemia on atorvastatin, hypothyroidism on levothyroxine, severe aortic stenosis, and Alzheimer's dementia on donepezil who presented to the emergency department (ED) for evaluation of altered mental status (AMS) and suspected witnessed seizure. The patient's family reported the patient was scheduled to have a colonoscopy that morning and had completed the bowel preparation with NuLYTELY [polyethylene glycol (PEG) plus electrolytes] the evening prior. When the patient's family arrived at her residence to transport her to the scheduled colonoscopy in the morning, the patient was found to be disoriented. En route to the hospital, the patient reportedly had convulsive seizure-like activity which lasted 10 minutes. The patient had no prior history of seizures. Upon arrival to the ED, the patient had findings consistent with post-ictal state including confusion, inability to follow commands, and evidence of a bite mark on anterior tongue. Vitals were significant for heart rate of 48 beats per minute, blood pressure of 160/105, and oxygen saturation in the upper 90 percent range on 2 Liters supplemental oxygen via nasal cannula.

In the ED, i-STAT labs were collected and revealed a sodium of 117 mEq/mL (133-145 mEq/L). Aside from the bitten tongue, no other apparent trauma or injury was noted. The patient's chest x-ray was unremarkable and head computerized tomography (CT) showed no evidence of acute hemorrhage or intracranial pathology. Metabolic panel confirmed significant hyponatremia with a sodium level of 115 mEq/L. Chart review of sodium levels over the three years prior to this presentation ranged from 132-135 mEq/L.

While in the ED, the patient was given an initial bolus of intravenous (IV) 3% hypertonic saline which resulted in improved neurologic status including mentation, ability to follow simple commands, and movement of all extremities equally. However, repeat sodium came back low at 115 mEq/mL, thus the patient was given an additional bolus of hypertonic saline with the next sodium resulting at 118 mEq/mL. Following this, the patient was started on IV normal saline at 100 mL/hour due to concerns for volume depletion secondary to bowel preparation. The patient's potassium was mildly low at 3.2 mEq/mL, which was replaced with IV potassium chloride. The patient was admitted to the hospital and the nephrology service was consulted.

Nephrology evaluated the patient and had concern for syndrome of inappropriate antidiuretic hormone secretion (SIADH) secondary to bowel preparation. With concern for SIADH, they stopped normal saline, which can worsen the electrolyte dysfunction. Laboratory values resulted and were consistent with SIADH: serum osmolality 247 mOsm/kg (275-295 mOsm/kg), urine osmolality 469 mOsm/L, and elevated urine sodium 129 mmol/L. Additional laboratory workup at this time revealed a TSH of 1.44 (0.450-5.330 mIU/L), BUN of 8 mg/dL (6-24 mg/dL) 0.7, Cr of 0.7 (0.5-1.1 mg/dL), albumin of 4.4 g/dL (3.5-5.5 g/dL), and total

protein of 7.5 g/dL (6-8.3 g/dL). The patient was placed on a fluid restriction with repeat electrolyte checks scheduled every two hours. The goal of sodium correction in acute hyponatremia is no more than 6 mmol/L over 24 hours to decrease the risk of osmotic demyelination syndrome. Over the next several hours, the patient began to answer questions more appropriately and follow commands. The patient was given one additional dose of hypertonic saline which resulted in additional improvement of mentation. In 24 hours, the patient's sodium had increased from 115 to 122 mEq/mL, though it later decreased back to 120 mEq/mL. Sodium chloride tablets were added at this time. Over the next several days, the patient's neurologic status returned to baseline and serum sodium continued to normalize with fluid restriction and sodium chloride tablet supplementation.

After 6 days in the hospital, the patient was discharged home with home health for physical and occupational therapy due to overall deconditioning. Additionally, the patient continued with sodium supplementation with 1 mg sodium chloride tablets three times daily. The patient was to follow up with their primary care physician to recheck electrolytes and assess the need for continued use of sodium chloride tablets. The patient was also instructed not to drive for 6 months following the seizure.

Discussion:

Oral bowel preparations are used to clean the colon prior to colonoscopy for optimal examination. They are safe for the majority of patients. Symptomatic electrolyte abnormalities, like hyponatremia, can occur after use.[2,3] PEG is one of the most routinely utilized agents for bowel preparation, because it is an isosmotic agent there is minimal absorption in the GI tract and less risk for electrolyte shifts.[2,3]

Reported risk factors for developing hyponatremia after bowel preparation include age >65 years, female gender, renal insufficiency, prior electrolyte abnormalities, heart failure, excessive fluid intake, prior gastrectomy, and use of angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers (ARB), thiazide diuretics, or antidepressants.[3,4,5,6,7] The incidence of bowel preparation induced hyponatremia is estimated to be 1-7%.[4,5,8] Hyponatremia may be asymptomatic, or range from mild to severe symptoms including nausea, vomiting, headache, confusion, fatigue, myalgias, seizures, coma, and death.[4] The incidence of symptomatic acute hyponatremia after bowel preparation is not well established.

The development of hyponatremia following bowel preparation is thought to occur from multiple physiologic mechanisms. Volume depletion that occurs with bowel preparations can lead to an increase in antidiuretic hormone (ADH, also known as arginine vasopressin (AVP)) secretion from the posterior pituitary gland.[4,7,9] The basic function of ADH is to regulate the amount of water in the body. A condition known as SIADH occurs when excess ADH is released regardless of serum osmolality[4,9,12] Factors that lead to non-osmotic release of ADH include anxiety, nausea, vomiting, pain and stress, several of which may occur in anticipation of the procedure.[8,10,11] The lack of protein and sodium intake while fasting for bowel preparation may also contribute to the development of hyponatremia.[10] Additionally, patients may be at increased risk of developing hyponatremia as they consume large volumes of water to counteract the loss of fluid through the colon.[3,4,7,10]

Diagnostic criteria indicating SIADH was initially described by Bartter and Schwartz in 1967 can be seen in *Table 1*, along with the patient's laboratory values.[13] Slight variations to this diagnostic criteria have been used, however, the criteria by Bartter and Schwartz is still commonly utilized today.[14]

	SIADH Criteria	Patient's Laboratory Values
Serum osmolality	<275 mOsm/kg	247 mOsm/kg
Urine osmolality	>100 mOsm/L	469 mOsm/L
Urine sodium	>20 mmol/L	129 mmol/L
	Euvolemic and no other cause for hyponatremia	

Table 1. Criteria for SIADH as described by barter and Schwartz and the patient's lab values consistent with SIADH.[13]

The patient presented in this case report experienced severe hyponatremia following bowel preparation with PEG and had laboratory values consistent with SIADH. The patient had risk factors that may have increased their chances of experiencing hyponatremia after bowel preparation including age, gender, and use of an ARB. In addition to fluid loss causing ADH release, the patient likely experienced symptoms leading to non-osmotic release of ADH, such as nausea, anxiety, pain, or stress. Even with the patient's risk factors, the development of SIADH is an unlikely event to occur in those preparing for colonoscopy. Another factor which was not confirmed, but may have contributed to the severe hyponatremia, included lack of sodium and protein intake prior to preparation and/or excessive fluid intake. With fluid restriction, sodium supplementation, and hypertonic saline the patient's sodium level and neurologic status returned to baseline.

This case emphasizes that physicians should be aware of the potential for severe hyponatremia or even SIADH to occur after utilizing oral bowel preparations for colonoscopy, especially in patients with multiple risk factors.

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