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Electrolyte Replacement in Bartter Syndrome With Abnormal Small Bowel: A Case Report

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Abstract

Bartter syndrome is a rare disorder that is characterized by weakness and fatigue with laboratory findings of hypokalemia and metabolic alkalosis with increased aldosterone and angiotensin. It specifically acts on the ascending loop of Henle, characterized by miscoded proteins affecting NaCl transports and channels. Patients will require replacement of potassium and sometimes magnesium due to the kidneys' inability to reabsorb these ions. So what happens when the body's other primary mechanism of absorption of these elements are taken out? In this article, we present the case of a 47-year-old woman with Bartter syndrome on oral potassium 40 mg BID (twice a day) and magnesium oxide 800 TID (thrice a day), who recently had a small bowel resection that required intravenous potassium and magnesium throughout her hospital admission. Significant questions arose as to how her electrolytes should be managed, given her unusual presentation with rare underlying disorder. We discuss the implications of her bowel resection in the context of Bartter syndrome and our views on her future course based on available literature.

Keywords

Bartter syndrome, short bowel syndrome, hypokalemia, hypomagnesemia

Introduction

In 1962, Frederic Bartter described a new syndrome of weakness and fatigue with laboratory findings of hypokalemia and metabolic alkalosis with increased aldosterone and angiotensin.[1] Bartter syndrome is a genetically inherited disorder of the renal tubules, specifically the ascending loop of Henle, characterized by miscoded proteins affecting NaCl transports and channels.[2] The result is salt wasting with hypokalemia and a metabolic alkalosis with anhydrochloria, and in some cases hypomagnesemia.[3] There is debate as to the inciting event causing the precipitation of symptoms seen in this syndrome. In one case report, potassium wasting was evident even after aldosterone levels were down to a normal level, although conversely magnesium infusion decreased potassium wasting. The authors of that case report suggest that the primary defect is with reabsorption of sodium chloride rather than potassium wasting. The current accepted pathophysiological mechanism is the reduction of potassium absorption back into the tubules causing an imbalanced electrochemical gradient.[4-6] In patients with kidney disease, electrolyte homeostasis can be maintained in part by absorption in the gastrointestinal tract.[7]

Potassium and magnesium absorption in the small intestine do vary somewhat based on location. One study showed that approximately 85% of net potassium intake in an individual is absorbed in the terminal ileum.[8] Magnesium absorption takes place in the distal jejunum and ileum.[9] When these electrolytes are low, replenishment is most commonly achieved through oral supplementation. However, when confronted with an abnormal gastrointestinal (GI) tract, supplementation can become a tedious task. Maximization of absorption must be considered, including intravenous (IV) supplementation to overcome a shorter bowel.[10] The clinical picture is unclear when both the kidney and bowel are unable to maintain homeostasis.

Case Presentation

We present the case of a 47-year-old woman with a past medical history of restless leg syndrome, peptic ulcer disease,

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Table 1. Daily Laboratory Values and Supplementation of Potassium and Magnesium.

Dates	Potassium (mEQ) (3.5-5.1)	Supplementation (mEQ)	Magnesium (mg/dL) (1.6-2.3)	Supplementation (mg/dL)
First admission				
Day 1	3.4	N/A	N/A	N/A
Day 2	3.3-3.5	40 mEQ (IV)	1.0-1.4	4000 mg (IV)
Day 3	3.8	80 mEQ (IV)	1.6	2000 mg (IV)
Day 4	3.0	60 mEQ (IV)	1.3	2000 mg (IV)
Day 5	3.9-3.9	N/A	1.5-2.5	2000 mg (IV)
Second admission				
Day 1	3.5	N/A	1.9	N/A
Day 2	3.0	80 mEQ (IV)	1.3	4000 mg (IV)
Day 3	3.2	60 mEQ (IV)	1.4	4000 mg (IV)
Day 4	3.4	40 mEQ (IV)	1.4	2000 mg (IV)
Day 5	3.8	N/A	1.6	2000 mg (IV)

Abbreviations: N/A, not available; IV, intravenous.

achalasia, and Bartter syndrome who presented to our facility with intractable abdominal pain, nausea, loss of appetite, and hematemesis that started 24 hours prior to admission. Medications on admission included Pepcid, Carafate, Sertraline, Ropinirole, Keppra, ferrous sulfate, B_{12}, potassium chloride 40 mg BID (twice a day), and magnesium oxide 800 mg TID (thrice a day). Her electrolyte levels had been previously stable on those home doses. The abdominal pain had been intermittent before admission, but had worsened in the past few days. She endorsed that she had similar symptoms in the past because of a bleeding peptic ulcer. Of note, she had also recently suffered a small bowel obstruction and underwent a small bowel resection in Mexico about 8 weeks before this admission, a scenario that prevented us from obtaining any records of that procedure itself. With the records, our hope would have been to know how much small bowel was left intact post-resection. The surgical site had become swollen and began to drain after an episode of hematemesis and continuous retching the day before.

On admission, she was noted to have hypokalemia at 3.4 mEq/L and hypomagnesemia of 1.0 mg/dL despite her home regimens of potassium chloride and magnesium oxide. Due to her recent abdominal surgery and significant discomfort, a CT A/P (computed tomography of abdomen/pelvis) was ordered, which showed constipation, no bowel obstruction, a right-sided pelvic fibroid, and multiple hypodense areas in kidneys. Also noted were surgical changes in the small bowel with part of the stomach removed. She continued to report of abdominal pain and persistent hematemesis, and was placed on Protonix. Our surgery team was consulted, who recommended an upper GI endoscopy for further evaluation of hematemesis. Endoscopy showed a tortuous esophagus with a small amount of clotted blood and mild gastritis. In order to further workup the bleeding, surgery recommended to perform a CTA A/P (computed tomography angiography of abdomen/pelvis), which did not give a defined etiology for her bleeding. It is suspected that her bleeding was due to gastritis secondary to an ulcer, consistent with her history of ulcers.

Throughout her stay, the patient did not require any transfusion of blood products. She did however require significant daily IV supplementation of magnesium and potassium, which can be seen in Table 1. Despite aggressive replacement of both, she would be hypokalemic and hypomagnesimic daily. She was discharged home with the same home potassium chloride and magnesium oxide regimens as there was no previous history of hypokalemia and hypomagnesemia. She had returned nearly a month later again with complaints of abdominal pain and hematemesis. On this admission, her initial potassium and magnesium were 3.5 and 1.9, respectively, both within normal ranges. However, she once again became hypokalemic and hypomagnesimic as her hospitalization extended and again required daily IV supplementation as seen in Table 1. She was again discharged on her home medication regimen with no changes made to her magnesium oxide and potassium chloride. She has not had further electrolyte abnormalities after discharge. During her hospital stay, her lowest potassium was recorded at 3.0 mEq/L and lowest magnesium at 1.0 mg/dL.

Discussion

To the authors' knowledge, there are no case reports reporting a patient with Bartter's syndrome with concurrent significant small bowel resection. This presents an interesting dilemma in terms of electrolyte management and replacement during hospital management. In the case of our patient, large doses of IV potassium and magnesium were required daily in order to maintain homeostasis. When potassium is below the normal threshold of 3.5 mEq/L, the body has already lost much of its potassium due to most potassium being stored intracellularly.[11]

Patients with short bowel syndrome can have major electrolyte abnormalities resulting in significant morbidity.

Although CT was obtained on admission, it was not able to be determined the exact amount of small bowel that had been removed during the patient's previous surgery, and we are unable to retrieve prior records. While it is unclear if our patient meets the criteria for short bowel syndrome, which is defined as possessing <150 cm of small bowel, the principle remains the same. Intravenous replacement of electrolytes is a mainstay of therapy in the initial phases, but the remaining bowel can undergo dilation, increase villus length, and augmentation of crypts to allow more surface area for increased absorption and possible oral supplementation.[12] It is also not unreasonable to assume her partial gastrectomy, as noted on CT, also played a role in malabsorption.[13] It is not unreasonable to postulate that our patient could possibly be in the "IV supplementation" stage of recovery following small bowel resection, and that she may be able to fully replace her decreased stores with oral supplements.

We surmise that in patients with underlying kidney deficiencies in absorption of necessary electrolytes that the GI tract may be able to overcome this and result in electrolyte homeostasis. When this mechanism of absorption is knocked out, through removal in the case of our patient, the resulting outcome relies on aggressive replenishment of these ions in order to avoid deleterious effects of low electrolyte counts. This may be exacerbated in times of other underlying illness and GI illness. Seeing how oral supplementation was inadequate to maintain electrolyte balance in our patient during her hospitalization, we argue that during acute illness, the patient may require IV supplementation in order to avoid adverse outcomes. Careful attention should be paid to electrolyte homeostasis in those with underlying gastrointestinal and renal abnormalities that affect absorption of electrolytes, especially in episodes of acute illness as such in our case.

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or cases series.

Informed Consent

Verbal informed consent was obtained from the patient for their anonymized information to be published in this article.

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