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ILEAL NEOBLADDER: AN IMPORTANT CAUSE OF NON-ANION GAP METABOLIC ACIDOSIS

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☐ **Abstract—Background: The differential diagnosis for a non-anion gap metabolic acidosis is probably less well known than the differential diagnosis for an anion gap metabolic acidosis. One etiology of a non-anion gap acidosis is the consequence of ileal neobladder urinary diversion for the treatment of bladder cancer. Case Report: We present a case of a patient with an ileal neobladder with a severe non-anion gap metabolic acidosis caused by a urinary tract infection and ureteroenterostomy. Why Should an Emergency Physician Be Aware of This?: Part of the ileal neobladder surgery includes ureteroenterostomy and predisposes patients to several clinically significant metabolic derangements, including a non-anion gap metabolic acidosis. These patients have an increased chronic acid load, bicarbonate deficit, and hypokalemia, which should be appreciated when resuscitating these patients.** © 2017 Elsevier Inc. All rights reserved.

☐ **Keywords—non-anion gap metabolic acidosis; neobladder; ileal conduit; urology**

INTRODUCTION

The differential diagnosis for a severe non-anion gap metabolic acidosis is less commonly known than the differential diagnosis for a severe anion-gap metabolic acidosis. The most common mechanisms involve loss of bicarbonate through the gastrointestinal or renal system, excessive chloride administration, Addison's disease, or medications such as acetazolamide. Commonly used mnemonics to remember the differential for the non-anion gap metabolic acidosis include "ABCD" for "Addison's, Bicarbonate loss, Chloride (administration), and Drugs," as well as "HARDUP" for "Hyperchloremia, Acetazolamide and Addison's, Renal Tubular Acidosis, Diarrhea, Ureteroenterostomies, and Pancreaticoenterostomies" (Table 1) (1).

Ureteroenterostomies are when the ureter has an abnormal connection with a segment of intestine. These connections may be the complication of fistulating Crohn's disease, or may be the intended outcome from a urological surgery. Many patients with bladder cancers are treated with a cystectomy and urinary diversion. To avoid permanent external urinary diversion, these patients may undergo a procedure to create a new "neobladder" from a segment of intestine. This neobladder serves as a conduit and reservoir between the kidneys and ureters and the urethra, and sits in the same orthotopic position as a urinary bladder. The neobladder passively fills with urine, and when the patient needs to void, often in a timed manner, they use a Valsalva or Credé maneuver to assist in emptying their reservoir via the urethra. Although these procedures may increase the quality of life for patients, there are specific medical complications to this procedure that emergency physicians should appreciate. This is a

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Table 1. The "ABCD" and the "HARDUP" Mnemonics for the Differential Diagnosis of a Non-Anion Gap Metabolic Acidosis

Mnemonic	Etiologies
"ABCD"	Addison's disease, Bicarbonate loss, Chloride (administration), Drugs
"HARDUP"	Hyperchloremia, Acetazolamide, Addison's disease, Renal Tubular Acidosis, Diarrhea, Ureteroenterostomies, Pancreaticoenterostomies

case report of a patient with a previous neobladder reconstruction from an isolated piece of her ileum, and it serves to highlight that these patients are predisposed to a non-anion gap metabolic acidosis.

CASE REPORT

A 57-year-old woman presented to the Emergency Department (ED) for confusion and fevers. For the previous few days she had malaise, fevers, and chills at home without any specific localizing infectious symptoms. The patient had no active medical issues, though she had been successfully treated for bladder cancer remotely. She took no medications chronically and had no known allergies. She had a history of bladder cancer, which was successfully treated 2 years prior with a cystectomy and neobladder using a portion of her ileum. In the surgery a portion of her ileum was isolated by ligating it proximally and distally from the rest of her alimentary canal while leaving the mesentery intact, and bowel continuity was re-established with a standard small bowel anastomosis. The harvested piece of ileum was detubularized and reconfigured into a spherical pouch, the patient's ureters were reimplanted into the pouch, and then an opening in the pouch was anastomosed to the urethra. This allowed her to store and empty urine in a continent fashion.

The physical examination found no fever at present (though she took acetaminophen prior to arrival), tachycardia to 103 beats/min, a blood pressure of 94/52 mm Hg, tachypnea of 24 breaths/min, and an oxygen saturation of 98%. Her point-of-care glucose was normal. Her electrocardiogram showed sinus tachycardia. She was ill appearing with labored breathing, there was no evidence of trauma, and her neurological examination was notable for severe confusion and delirium without focal deficits. The head, eyes, ears, nose, and throat were normal on inspection. There was no meningismus. The heart and lungs and abdomen and extremities revealed no significant findings. The patient was given a 30-mL/kg bolus of normal saline, and empirically administered 1 g ceftriaxone and 1 g vancomycin after blood cultures were drawn. A portable chest x-ray study demonstrated no acute pathology, and a noncontrast head computed tomography scan showed no acute pathology.

Her complete blood count demonstrated a leukocytosis of 17.9 K/μL with 15% bandemia, hemoglobin of 9.3 g/dL, and platelets of 234 K/μL. Her lactate returned normal at 1.4 mmol/L. Her chemistries demonstrated a sodium of 129 mEq/L, potassium of 5.4 mEq/L, chloride of 107 mEq/L, bicarbonate < 5 mEq/L, blood urea nitrogen of 73 mg/dL, a creatinine of 3.1 mg/dL, and a glucose of 90 mg/dL. Her anion gap was calculated to be < 17. She had normal electrolytes and a normal creatinine documented a few months prior at our institution.

A Foley catheter was placed for urine output monitoring, and 200 cc were collected over 4 h. Urinalysis demonstrated 44 white blood cells, 6 red blood cells, few bacteria, and large amounts of leukocyte esterase, but no nitrite.

The patient's mental status continued to decline and she was intubated for work of breathing and airway protection. On the ventilator she was initially hyperventilated while maintaining low tidal volumes, and her postintubation arterial blood gas analysis showed a pH of 7.09, pCO_2 14 mm Hg, and pO_2 511 mm Hg, with a base excess of -23. A right femoral vein triple lumen catheter was placed for norepinephrine infusion. She was given an additional liter bolus of lactated Ringer's solution, a dose of 100 mg hydrocortisone, and she was admitted to the medical intensive care unit.

Supportive care was continued in the intensive care unit and the patient was administered more intravenous fluids, a bolus of sodium bicarbonate, and she was continued on norepinephrine and hydrocortisone. In the intensive care unit the patient developed a fever, and had a renal ultrasound on the first hospital day, which suggested pyelonephritis, though her urine culture did not grow anything.

Over time her condition improved and her electrolytes and creatinine normalized completely, and she was liberated from the ventilator on the second hospital day. After a period of stability, the patient was transferred to the floor and then ultimately discharged from the hospital on a course of cefpodoxime for presumed pyelonephritis. Her blood cultures had no growth, and no other source of her infection was found.

DISCUSSION

Neobladder surgeries are relatively common, and are often performed after a cystectomy for bladder cancer to facilitate continent urinary storage. From 1998 to 2005, over 31,300 such surgeries were performed in Medicare beneficiaries, with ileal conduit surgery representing the vast majority (80% of total), but parts of the