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CASE REPORT

Acidosis and ketonuria in an 8-Year-Old Male

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Case Summary

The patient is an 8-year-old Caucasian male with no significant past medical history referred to the emergency department by his pediatrician for possible appendicitis. The day prior to presentation, his mother noted decreased energy and appetite. He went to sleep soon after arriving home from school and woke up twice overnight with non-bloody, non-bilious emesis. He began complaining of right-sided back and abdominal pain in the morning and was brought to his pediatrician, where he was immediately referred to the Emergency Department (ED).

In the ED, the patient was afebrile but tachycardic. He was described as ill appearing with sunken eyes and dry mucous membranes. Physical exam was notable for mild abdominal pain, inconsistent with appendicitis. Initial laboratory workup was pursued, including complete blood count, basic metabolic panel, C-reactive protein, venous blood gas and urinalysis. Baseline labs showed severe anion gap metabolic acidosis (pH 7.13, HCO_3 9.7, anion gap 29) with normal lactate (1.7), ketonuria and leukocytosis with left shift (white blood cell count 15.3, 89% neutrophils). The patient was also hypoglycemic, requiring a dextrose push for a glucose level of 59. A chest x-ray was obtained and showed no focal consolidation. The patient was re-hydrated with a total of 30 milliliters/kilogram (mL/kg) of normal saline (NS) and was admitted to the pediatric intensive care unit for further work-up.

Review of systems was negative for fevers, chills, headaches, respiratory symptoms, urinary symptoms and rash. He had not traveled recently. His older brother had self-resolving abdominal pain the week prior. The patient and his mother denied toxic ingestions. However, his mother noted that the patient drank from a public water fountain that tasted "sweet" on the day prior to presentation.

Upon arrival to the PICU, the patient received an additional 20 mL/kg NS bolus and was started on 1.5 times maintenance fluids. On further discussion with the patient's mother, it was discovered that he had been purposefully limiting calorie intake over the preceding two-three weeks in attempts to lose weight. His mother estimated an approximate six-pound weight loss in this period. Blood gases and electrolytes were trended closely over the next twelve hours with resulting closure of anion gap and return of normal pH without further intervention. Additional labs were obtained and resulted as normal throughout hospital course, including salicylate level, acetone, hepatic panel, thyroid function, ammonia, and pyruvate. Serum amino acid panel revealed low lysine, ornithine and tryptophan, possibly indicating low protein intake. Urine organic acids were nonspecifically elevated, suggesting a catabolic state. An abdominal ultrasound was also obtained, which showed no intra-abdominal pathology.

Given the patient's history and improvement with aggressive re-hydration alone, the leading diagnosis was a ketoacidosis state secondary to fasting. Child psychiatry was consulted to further explore psychosocial factors contributing to this presentation. Thorough psychiatric evaluation revealed an anxious character structure with some obsessional features. The patient and his mother admitted to longer standing concerns about his weight, especially in the context of being "chubbier" than his fa-

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mily members and occasionally being teased at school. The psychiatry team did not feel that the patient was at any imminent risk for self-harm but deemed it prudent to monitor anxiety and obsessive symptoms closely in the future. They also recommended outpatient nutrition follow up. He was discharged on hospital day two with pediatrician follow up.

Discussion

The broad differential diagnosis of high anion gap acidosis includes inborn errors of metabolism, ingestions of toxic chemicals (methanol, propylene glycol, ethylene glycol) or standard medications (iron, isoniazid or salicylates), chronic kidney disease, diabetic ketoacidosis, infection, and lactic acidosis. Our patient's laboratory findings and history quickly ruled out most of these etiologies. Normal BUN/creatinine, normal lactate and hypoglycemia eliminated chronic kidney disease, lactic acidosis and diabetic ketoacidosis, respectively. Infection was low on the differential given normothermia and no focal physical findings. Ingestion was denied by the patient and his mother.

An underlying metabolic disorder was initially high on the initial differential. However, the patient's age and lack of medical history would make an inborn error of metabolism less likely. Normal ammonia and pyruvate were reassuring, and abnormal serum amino acids and urine organic acids are non-specific and can be explained by a fasting ketoacidosis state. This case helps review the differential diagnosis and pathophysiology of high anion gap metabolic acidosis and also highlights the importance of reviewing the psychosocial history.