THE AMERICAN JOURNAL of MEDICINE®

A Rare Case of Non-Acidotic High Anion Gap Pancreatic Ketonemia

To the Editor:

A 36-year-old man with a medical history of alcohol use disorder and alcoholic gastritis presented to the hospital with 6 hours of severe epigastric pain radiating to the back, nausea, emesis, and anorexia. Prior to presentation, he had consumed 750 mL of liquor daily for 5 days, followed by 2 days of abstinence. Physical examination exhibited tenderness to palpation in the mid-epigastrium. Admission laboratory values revealed lipase 1254 U/L, aspartate transaminase 332 U/L, alanine transaminase 128 U/L, sodium 132 mmol/L, chloride 86 mmol/L, carbon dioxide 21 mmol/L, anion gap 25 mmol/L, serum glucose 155 mg/dL, lactate 1.5 mmol/L, triglycerides 74 mg/dL, and undetectable alcohol level. Venous blood gas pH was 7.44, PO_2 29 mm Hg, and PCO_2 47 mm Hg. Urinalysis was notable for 3+ ketones. Given his history, presentation, and laboratory values, the patient was admitted for management of acute alcoholic pancreatitis. His symptoms improved with fluid resuscitation and pain control, but his anion gap persisted despite clinical improvement. On hospital day 3, beta hydroxybutyrate was >8 mmol/L, the upper limit of our hospital's assay; his anion gap was 21 mmol/L, lipase was 121, and serum glucose was 281 mg/dL. The patient was treated with insulin and dextrose infusions. Further testing revealed a hemoglobin A1c of 5.0% and undetectable C-peptide level; although notably, C-peptide was measured during treatment with an insulin infusion. He was discharged with a 4-times daily insulin regimen and outpatient Endocrinology follow-up.

While the patient's anion gap and ketonemia on admission were initially attributed to alcohol or starvation, his presentation was atypical in that he had a high anion gap and beta hydroxybutyrate without associated acidosis or excessive hyperglycemia. Although his symptoms resolved with supportive care, his anion gap and beta hydroxybutyrate remained persistently elevated, disproportional to what may have been expected given his mild hyperglycemia. A prior case series described pancreatic ketoacidosis as a high anion gap acidosis during an acute pancreatitis episode, thought to be due to lipase-induced adipose breakdown as well as injury to pancreatic beta cells.[1] Another case similarly discussed a patient with euglycemic diabetic ketoacidosis (though without a history of diabetes) during acute pancreatitis.[2] Our patient's course suggests a diagnosis of pancreatic ketosis similar to, and presumably on the spectrum of, pancreatic ketoacidosis, yet unique from previous cases given a lack of acidosis and a continued high anion gap despite decreasing lipase levels.[3] To our knowledge, this is the first described case of a patient with non-diabetic hyperglycemia and pancreatitis-associated high anion gap ketonemia without acidosis. This case demonstrates the importance of closely monitoring patients with acute pancreatitis for signs of insulin deficiency such as ketonemia, high anion gap, or hyperglycemia.

ACKNOWLEDGMENTS

The authors would like to acknowledge Tracy Setji, MD, Erin Hercules, MD, MBA, and Aubrey Jolly Graham, MD for their care of this patient.

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https://doi.org/10.1016/j.amjmed.2022.02.014

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Funding: None.
Conflict of Interest: None.
Authorship: All authors participated in the care of this patient and had a role in writing this manuscript.

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