

D-Lactic Acidosis in Crohn's Disease and Short Bowel Syndrome

Hasan Ali, DO; Sung Choi, MD; Sushil Ahlawat, MD.

Introduction

Lactic acidosis (LA) in a patient presenting with abdominal pain raises concern for bowel ischemia. The physician is prompted to administer intravenous fluids, locate the ischemic area, and consider invasive procedures. However, different isomers of lactic acid exist. Elevation of D-lactic acid reveals an alternate diagnosis. Understanding D-lactic acidosis (D-LA) can guide physicians towards effective treatment and prevent harm from unnecessary imaging and invasive intervention.

Case Description

43 year old male with Crohn's Disease (CD) and bowel resection presents with 2 weeks of diffuse abdominal pain. On examination he was hypertensive and somnolent with slurred speech. His abdomen was soft but tender, with scarring from abdominal surgeries. Blood work revealed leukocytosis and anion-gap metabolic acidosis (AGMA) with LA. Testing for ketoacidosis and toxic ingestion was negative. Gastrointestinal pathogen panel was negative. Computed tomography (CT) was negative for bowel thickening or enhancement, and CT angiography was negative for mesenteric thrombosis. Endoscopy did not reveal active CD. Lactic acid remained elevated despite intravenous fluids. The patient was noted consuming juice and several packets of sugar with tea, raising concern for D-LA. After restricting carbohydrates, lactic acid and AGMA rapidly normalized. A blood sample sent out from time of admission showed elevated D-lactic acid.

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Discussion

This patient's neurologic symptoms, AGMA and LA, simple carbohydrate intake and prior bowel resection raised concern for D-LA, a rare condition observed in patients with short bowel syndrome (SBS). Decreased intestinal absorption in SBS results in delivery of carbohydrates to the colon, where fermentation by colonic flora form L- and D-Lactic acid and other organic acids. Systemic absorption can cause AGMA, LA and neurologic symptoms, such as encephalopathy, dysarthria and ataxia. Diagnosis involves clinical suspicion, AGMA that resolves with carbohydrate restriction, and neurologic symptoms. D-lactate dehydrogenase assay confirms diagnosis. Treatment involves correction of acidosis and oral carbohydrate restriction. Enteric antibiotic use has been proposed to reduce acid-forming bacteria. Physicians should suspect D-LA in patients with CD post-bowel resection and otherwise unexplained AGMA, as this could prevent harm through inappropriate treatment including intravenous antibiotics and corticosteroids, radiation exposure, and invasive endoscopic evaluation.