Abstract

D-lactic acidosis is a rare form of lactic acidosis that can occur in individuals following jejuno-ileal bypass surgery or short bowel syndrome. A 48-year-old female with Crohn's disease s/p subtotal small bowel resection 3 years ago, with resultant short bowel syndrome presented with persistent weight loss, abdominal pain, postprandial nausea, headache, confusion, blurred vision, dizziness with ataxia, intermittently ongoing for several months. She complained of feeling as if she was intoxicated, although she denied any alcohol ingestion. She had a history of intestinal intussusception and CT enterography ordered 9 days prior showed dilated loops of distal small bowel consistent with partial small bowel obstruction. Lab work revealed normal CBC, LFTs, but also metabolic acidosis with serum bicarbonate at 16mmol/L, with anion gap of 12; VG showed pH 7.15, pCO2 44mm Hg, pO2 17mmHg, bicarb 15mmol/L. However, serum lactic acid level was normal at 1mmol/L. Serum D-lactate was drawn showing elevation to 6.15mmol/L -- thus confirming the diagnosis of D-lactic acidosis. She was given fluids, bicarbonate and placed on Flagyl and Levofloxin for bacterial overgrowth. Her acidosis along with her neurological symptoms shortly thereafter resolved. PEG tube was placed and she was started on enteral feeding for malnutrition. Her partial SBO also resolved with supportive care.

Discussion

*Patients often appear drunk in the absence of ethanol consumption.

*D-lactic acidosis should be considered in short bowel patients with an anion gap metabolic acidosis, a normal serum lactate; characteristic neurological findings and acidosis following food intake.

*Diagnosis is confirmed with special enzymatic assay measuring serum D-lactate.

*Treatment involves sodium bicarbonate, low carbohydrate diet and antibiotics (usually Vancomycin, Neomycin or Metronidazole) to decrease bacteria overgrowth.

Background

*D-lactic acidosis is occurs in patients with short bowel syndrome where glucose and starch are metabolized in the colon into D-lactic acid.

*Symptoms typically occur after ingestion of a high-carbohydrate meal being metabolized by the colon rather than the small bowel-with subsequent absorption into circulation and systemic acidemia.

*D-lactate accumulates as L-lactate dehydrogenase is unable to convert this type of lactate into pyruvate.

Discussion

*In individuals with intact small intestine, few carbohydrates and starch reach the colon. Delivery of glucose is markedly magnified when the small bowel is removed, bypassed or diseased.

*In patients with short bowel syndrome, glucose reaches the colon, and when in combination with bacterial overgrowth (usually gram-positive anaerobes, eg. Lactobacilli)-- D-lactic acid is formed.

*Episodic metabolic acidosis can occur after meals with neurological symptoms including altered mental status, confusion, dysarthria, cerebellar ataxia, and concentration/memory difficulties.

Conclusion

*D-lactic acidosis should be considered in all patients with short bowel and/or other malabsorption syndrome whose acidosis corresponds with food intake and resolves after cessation of ingestion.

*Classic symptoms are described as being intoxicated with ethanol-slurred speech, memory loss, cerebellar ataxia, confusion.

*Treatment involves a low carbohydrate diet, sodium bicarbonate infusion to correct acute acidemia and antibiotics to minimize D-lactate producing bacteria.