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D-Lactic acidosis in a 2-year-old girl with acute gastroenteritis: A case report

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Abstract

D-Lactic acidosis is common in patients with short bowel syndrome. We report a 2-year-old girl treated for gastroenteritis at a peripheral local hospital with antibiotics and probiotics. She was drowsy, lethargic and hyperventilating. She had wide anion gap acidosis with normal lactate levels. She needed multiple doses of bicarbonate boluses and infusion for correction of acidosis. Her neurological status improved as acidosis got corrected.

Keywords

D-Lactic acidosis; wide anion gap acidosis; probiotics; gastroenteritis

Introduction

D-Lactic acidosis is a well documented state in patients with short bowel syndrome. The abnormal colonic bacterial flora produces D-Lactic acid. Symptoms of D-Lactic acidosis can thus be treated with enteric antibiotics.

Case Report

A 2-year-old girl presented with history acute gastroenteritis with profuse diarrhea and vomiting. She had associated loss of appetite and poor intake. There was no history of fever, cough, jaundice and rashes. She was initially evaluated and treated for gastroenteritis at a peripheral local hospital with antibiotics and probiotics.

On examination, she was drowsy, lethargic and hyperventilating. System examination did not show any abnormalities except for drowsiness. Her pH was 7.18, pCO_2 was 21, pO_2 was 90, bicarbonate was 4 mmoll/l, sodium was 140, chloride was 102 and albumin was 2.4. Thus, her blood gas analysis showed high anion gap metabolic acidosis. Surprisingly, her lactic acid was within normal limits. She needed multiple

doses of bicarbonate boluses and infusion for correction of acidosis. Her neurological status improved as acidosis got corrected. She did not have any family history of inborn errors of metabolism.

Discussion

Clinically our patient had wide anion gap metabolic acidosis with lethargy, weakness and dehydration. The reason for wide anion gap acidosis was difficult to explain. Blood sugar was within normal limits. There was no evidence of infection as her blood count was normal with normal C - reactive protein and negative blood culture. She had a wide anion gap acidosis with normal urine pH ruling out renal tubular acidosis. There was no previous or family history to suggest inborn error of metabolism. Serum ammonia was normal. Liver and renal functions were within normal limits. There was no history to suggest any accidental drug ingestion.

D-Lactic acidosis can be suspected in our patient with wide anion gap metabolic acidosis that cannot be explained by uremia or ketonemia. Moreover, she had normal lactic acid levels [0.7mmol/L]. D-Lactic acidosis is usually confirmed by elevated levels of D-Lactic acid in serum and urine [1,2]. Normal values of D-lactic acid in blood are undetectable, being those above 3mmol/L pathological [3]. Unfortunately, these tests are not being done in India.

D-Lactic acidosis has been previously reported by Duran et al. in 1977 in a mentally retarded child who did not have any intestinal disease or bowel surgeries [4,5]. The source was unknown. We attribute D-Lactic acidosis in our patient due to the over use of probiotics containing lactobacillus preparation. Fermentation by non-spore-forming anaerobic colonic bacteria like Bifidobacterium, Lactobacillus and Eubacterium produces D-Lactic acid and absorption of fermentation products into blood causes acidosis.

Lactic acid exists as two optical isomers in nature: D-lactic acid and L-lactic acid. L-lactic acid is the most abundant form in humans and mammals. Both are produced and metabolised by the enzyme, lactate dehydrogenase [LDH]. LDH is isomer-specific; L-lactate is produced by L-LDH and D-lactate by D-LDH. L-LDH is the only form of the enzyme present in mammals. Many carbohydrate-fermenting bacteria, including *Lactobacillus* sp. and *Bifidobacterium* sp., possess both forms of the enzyme and are able to produce both stereoisomers in varying proportions.

Concentrations of 5–20 μmol/L of D-lactate have been found in the blood of healthy subjects. It has been explained as produced by bacteria in the GI tract, absorbed into the bloodstream. However, this explanation appears to be insufficient to explain the levels present in human blood. Recently, it has become clear that D-lactate is both produced and metabolised by human cells, by an alternative pathway, the methylglyoxal pathway. The methylglyoxal pathway is an offshoot of glycolysis, which converts glucose into methylglyoxal and then into D-lactate.

Current methods for measurement of plasma and whole blood lactate only detect the L-lactic acid isomer. D-lactic acidosis (plasma D-lactate >3.0 mmol/L with blood pH<7.35) is one complication of short bowel syndrome, secondary to diffuse small bowel disease or resection. Both D-lactic acid and L-lactic acid

are produced by these bacteria and absorbed systemically. While the L-LDH in the body can metabolise L-lactic acid, humans have limited capacity to metabolise D-lactic acid, resulting in a metabolic acidosis. The levels of D-lactic acid needed to cause an academia are more than a hundred times the levels seen in healthy individuals. D-lactic acidosis presents with neurological dysfunction and encephalopathy.

This case is a reminder of the importance of suspecting this rare entity in children presenting with neurological symptoms and high anion gap metabolic acidosis. We thus stress the importance of using probiotics judiciously in children with gastroenteritis by reporting this case.

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