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D-Lactic acidosis 25 years after bariatric surgery due to *Salmonella enteritidis*

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ABSTRACT

D-lactic acidosis is a rare complication that occurs in patients with short bowel syndrome due to surgical intestine resection for treatment of obesity. The clinical presentation is characterized by neurologic symptoms and high anion gap metabolic acidosis. The incidence of this syndrome is unknown, probably because of misdiagnosis and sometimes symptoms may be incorrectly attributed to other causes. Therapy is based on low carbohydrate diet, sodium bicarbonate intravenous, rehydratation, antiobiotics, and probiotics that only produce L-lactate. In the case we describe, D-lactic acidosis encephalopathy occurred 25 y after bypass jejunoileal, due to *Salmonella enteriditis* infection.

Introduction

D-lactic acidosis (DLA) is an uncommon neurologic syndrome that occurs in patients with jejunoileal bypass, small bowel resections, and other forms of short bowel syndrome. The clinical manifestations of DLA are episodes of encephalopathy and metabolic acidosis with an increased anion gap. Most of the cases described in the literature have occurred within a few months to 10 y after surgical resection of the bowel, especially after ingestion of high-grade carbohydrates. We present a case of DLA that occurred 25 y after bypass jejunoileal caused by *Salmonella enteritidis*, which was able to change intestinal mucosa, raising load D-lactate-producing bacteria. The important decrease in urine and plasma D-lactate levels and clinical resolution after antibiotic therapy suggest that selective bacterial overgrowth by Salmonella was the determining factor in the development of the recurrent syndrome.

Case report

A 51-y-old Caucasian woman was admitted to the emergency department in an acute confusional state characterized by disorientation, weakness, nausea, dehydration, blurred vision, and slurred speech, appearing drunk. Her medical history was significant for a jejunoileal bypass procedure in 1985 for obesity with residual intestinal absorption of 80-90 cm; 9 y later, she was hospitalized for neurologic syndrome, probably due to bacterial overgrowth. Since then, she had begun therapy with rifaximine, 2 weeks every month, until hospitalization. The patient has been free of symptoms and any complications related to short bowel syndrome for 15 years. On admission, her temperature was 37.6°C, blood pressure 120/80 mmHg, pulse 79 bpm. Physical examination was negative except for mild and diffuse abdominal pain with no organomegaly or ascites. Arterial blood gas analysis showed normocloremic metabolic acidosis with increased anion gap; laboratory tests are summarized in Table 1. No drugs, alcohols, lactate, ketones, or salicylates were detected in her blood. Neurologic testing, including computed tomography, was unrevealing. Thus, the treatment with intravenous administration of 150 mEq sodium bicarbonate was started and acid-base imbalance was normalized with improvement of neurologic symptoms within a few hours. The patient was transferred to our department of internal medicine to evaluate the etiology of metabolic acidosis and neurologic symptoms. Because of her previous bypass jejunoileal, DLA was suspected, and we proposed a carbohydrate load that the patient refused. Three days later, after a meal, the patient showed the same neurologic disturbances and physical weakness as presented on admission. During this acute episode, arterial blood gas analysis showed high anion gap normocloremic metabolic acidosis, and sera and urine were collected to measure the level of D-lactic acid (colorimetric assay kit, EnzyChrom D-Lactate Assay Kit; BioAssay

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Table 1
Summary of daily serum and urine chemistries

Laboratory data	First episode		Second episode		Third episode		Start treatment		
	Day 1	Day 2	Day 1	Day 2	Day 1	Day2	Day 1	Day 5	Day 30
Sodium (mmol/L)	140	141	143	141	142	140	142	138	140
Potassium (mmol/L)	4.6	4.2	3.5	3.1	3.6	3.7	4.1	3.8	4.2
Chloride (mmol/L)	104	106	105	104	105	103	104	106	105
BUN (mg/dL)	29	28	21	22	26	32	30	28	31
Creatinine (mg/dL)	1.4	1.2	1.5	1.1	1.2	1	1.4	1.2	1.3
Glucose (mg/dL)	90	88	94	85	77	102	96	81	75
Ammonium	31	32	28	—	—	—	—	—	—
Urine ketones	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Bicarbonate (mmol/L)	10	16	11	23.8	16.8	23.7	22.8	21.4	23
Anion gap (mmol/L)	26	19	27	12	20	13	15	11	12
Plasma L-lactate (mmol/L)	0.6	1.3	1.1	0.8	0.7	0.9	1.1	0.7	1.1
Plasma D-lactate (mmol/L)	—	—	27.2	26.6	27	24	9.51	4.2	2.9
Urine D-lactate (mmol/L)	—	—	38.5	7.45	23	5.45	1.06	1.04	1.01
pH	7.17	7.43	7.28	7.44	7.33	7.43	7.41	7.42	7.4
Pco_2 (mmHg)	28	24	25	35	31	35	36	33	37
Po_2 (mmHg)	103	93	123	94	91	94	92	99	98
BE (B) (mmol/L)	−17	−6	−13	0	−8.5	−0.7	−1.2	−2.5	−1.5

Systems, Hayward, CA, USA) (Fig. 1, Table 1). The levels of serum and urinary D-lactic acid were high at 27.2 and 32.4 mmol/L, respectively (normal range, 0-3 mmol/L). Treatment was started with intravenous administration of sodium bicarbonate and, over the next 12 h, the patient gradually recovered from neurologic impairment and the acid-base imbalance was normalized (Table 1). After this second episode, we decided to reduce carbohydrate intake. A surgical consultant suggested bypassing the conversion, which the patient refused. However, 2 d later, the patient presented a third episode of metabolic acidosis with confusion, slurred speech, anxiety, and weakness, appearing drunk. Sera and urine collected of D-lactic acid levels were high (Fig. 1). Because of increased numbers of evacuations from three to seven daily, a rise in temperature to 38.5°C, and diffuse abdominal pain, stool cultures were performed showing positivity for *S. enteritidis* responsive to ciprofloxacin. Therefore, the patient started ciprofloxacin 500 mg twice daily for a week. She also received medical nutrition therapy planning with 1485 Kcal in divided meals with 49.5% carbohydrates, 22.6% proteins, and 27.9% fats. Over the next 7 d, no episodes of encephalopathy or metabolic acidosis occurred; stool cultures, Widal reaction, D-lactic acid in serum and urine were negative, and the patient was discharged with antibiotics and probiotics (rifaximine and *Lactobacillus* GG). No more attacks happened in 6 mo of follow-up.

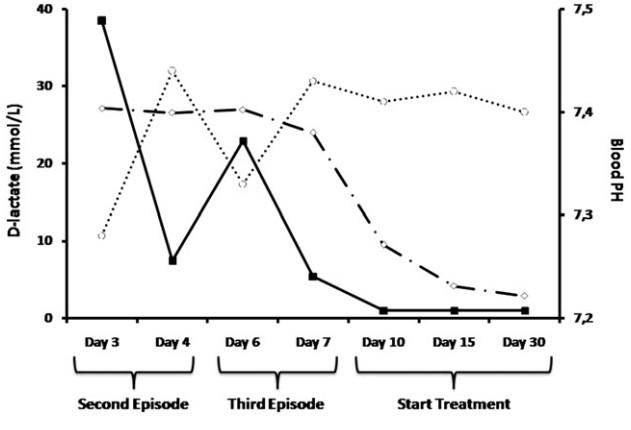

Fig. 1. Correlation between serum and urine D-lactate and blood pH.

Discussion

DLA is an uncommon and often misdiagnosed neurologic syndrome that occurs in patients with jejunoileal bypass, small bowel resections, and other forms of short bowel syndrome. The clinical manifestations of DLA are episodes of encephalopathy and metabolic acidosis with an increased anion gap. Headache, weakness, delirium, visual disturbances, slurred speech, ataxia, cranial nerve palsies, and aggressive and abusive behavior are hallmarks of DLA encephalopathy, with the patients often appearing drunk [1]. The incidence of DLA is unknown, probably due to misdiagnosis; sometimes symptoms may be incorrectly attributed to other causes. DLA is well documented in veterinary medicine; however, encephalopathy after jejunoileal bypass was first described in humans by De Wind in1976 and, to date, there are approximately 40 cases reported in literature [2]. DLA can occur within a few months to 10 y after surgical resection of bowel and most cases occur after ingestion of high-grade carbohydrates [3]. However, Narula et al. reported an unusual case that occurred 23 y after jejunoileal bypass [2]. The diagnosis was performed on the basis of neurologic manifestations and high level of D-lactate. No universal reference values for D-lactate are available and normally are undetectable, but usually, when the measured concentration results in an excess of 3 mmol/L, it contributes to the acidosis [4]. Blood gas analysis reveals a metabolic acidosis usually with an increased anion gap [1]; L- and D-lactate are found at low concentrations in the stool of healthy subjects as a result of conversion of both isomers of lactate to short-chain fatty acid by the intestinal bacteria. The development of DLA is promoted by an alteration of the normal intestinal flora [5], often secondary to surgical procedures. In these patients, malabsorption in the proximal bowel causes a large amount of carbohydrate available to colonic bacteria. Subsequently, carbohydrates are fermented, producing lactic acid and short-chain fatty acids able to reduce the luminal pH of the colon favoring overgrowth of lactate-generating bacteria (L- and D-lactate). Progressive increase of lactic-acid-producing bacteria and further decrease of pH promote growth and survival of gram-positive organisms producing D-lactate,