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Case report

Metabolic acidosis due to d-lactate in a patient with intestinal resection: Diagnostic challenges and nutritional strategies

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ABSTRACT

Introduction: Metabolic acidosis, marked by decreased plasma bicarbonate and arterial pH, is a common complication following extensive abdominal surgeries. D-lactate acidosis presents additional diagnostic challenges due to nonspecific symptoms.

Presentation of case: A 65-year-old woman with hypertension and morbid obesity was admitted to the ICU for intestinal obstruction and peritonitis due to an incarcerated hernia. Extensive bowel resection required ileostomy and prolonged antibiotic therapy. She developed refractory metabolic acidosis, suspected to be D-lactate acidosis. Management included sodium bicarbonate for acid-base correction and carbohydrate restriction via enteral nutrition. Gradual carbohydrate reintroduction resolved the acidosis. After clinical stabilization, elevated D-lactate levels were confirmed, and she transitioned to an oral diet with protein supplementation.

Discussion: Treatment focused on carbohydrate restriction to limit D-lactate production by reducing intestinal fermentation. Fructose was initially considered for its unique absorption properties that prevent fermentation, but limited formula availability led to complete carbohydrate elimination. Complex carbohydrates were gradually reintroduced to meet metabolic requirements without worsening acidosis. Intravenous bicarbonate, probiotics, and antibiotics were employed to manage severe acidosis. This case emphasizes the importance of individualized, multidisciplinary approaches in managing D-lactic acidosis and underscores the need for accessible, effective nutritional formulas.

Conclusion: Early diagnosis of D-lactate acidosis enables effective management through carbohydrate restriction, reducing bacterial fermentation and improving clinical outcomes.

1. Introduction

Metabolic acidosis is an acid-base imbalance characterized by a reduction in plasma bicarbonate (HCO_3^-) concentration and arterial pH, resulting from the accumulation of organic acids or loss of bases [1]. It is rarely observed in patients with short bowel syndrome (SBS) or other malabsorption conditions. This disorder is associated with neurological symptoms such as confusion, speech difficulties, ataxia, and irritability, often triggered by carbohydrate intake. Metabolic acidosis can occur in hospitalized patients, particularly those who have undergone extensive abdominal surgeries, and are influenced by factors such as intraoperative bicarbonate loss, systemic inflammatory response, and electrolyte imbalances [2].

The length of the remaining intestine, the distance from the duodenum and the presence or absence of the ileocecal valve are crucial factors that influence nutrient absorption and the risk of developing complications, including D-lactic acidosis. These factors increase the risk of severe complications and postoperative mortality.

Among the variants of metabolic acidosis, D-lactate acidosis poses a significant diagnostic challenge, especially in patients undergoing intestinal resection [3–6]. Its nonspecific symptoms and similarity to other metabolic disorders complicate diagnosis. Assessment of the anion gap is critical: a normal gap suggests hyperchloremic acidosis, while an elevated gap indicates acidosis due to acid gain, such as ketones, nitrogen derivatives, and lactates (both L and D) [6]. D-lactate accumulation, associated with abdominal surgery, results from alterations in

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intestinal glucose metabolism, independent of hypoperfusion [1,7].

D-lactate builds up due to an overload of undigested carbohydrates in the small intestine. These carbohydrates are fermented by colonic microbiota, promoting the growth of D-lactate-producing lactic acid bacteria. As humans lack the enzyme D-lactate dehydrogenase, they primarily metabolize L-lactate (Fig. 1) [8,9].

Intestinal resection induces an imbalance in the colonic microbiota, leading to an increased fermentation of unabsorbed carbohydrates. As a result, there is an overproduction and accumulation of D-lactate, an isomer of lactate that cannot be efficiently metabolized by the body, causing metabolic acidosis.

Diagnosis is confirmed through an elevated serum anion gap with normal lactate levels and increased D-lactate (≥3 mmol/L) [6]. Management involves dietary modifications (such as a low-carbohydrate diet), antibiotic therapy, bicarbonate therapy to correct acidosis, and adequate hydration. In severe cases, surgical intervention may be necessary. Early recognition is crucial to prevent complications.

D-lactate accumulation has significant clinical implications, increasing the risk of mortality. Early intervention and targeted management are critical. Nutritional strategies play a key role in managing this acidosis by optimizing carbohydrate absorption and reducing D-lactate levels [9]. This report presents an unusual case of D-lactate acidosis in a patient with intestinal resection, emphasizing the diagnostic challenges and nutritional therapeutic strategies.

2. Case presentation

This paper is reported in line with the SCARE 2023 criteria [10]. A 65-year-old female patient with a history of arterial hypertension and obesity class 1 (BMI 30) was admitted to the intensive care unit (ICU) for intestinal obstruction and peritonitis secondary to an incarcerated inguinal hernia. The patient underwent exploratory laparotomy due to clinical deterioration and lack of improvement after the initial surgery. She presented with shock, metabolic acidosis, and hyperlactatemia secondary to hypoperfusion. During the procedure, extensive necrosis of approximately 5 m of the small intestine was identified, involving the distal jejunum and most of the ileum, leaving a minimal ileal remnant. Necrotic intestinal segments were resected, preserving as much functional bowel as possible to minimize the risk of short bowel syndrome. An end-to-end anastomosis was performed between the remaining jejunum and the preserved distal ileum using absorbable sutures to ensure intestinal continuity and minimize leak risk.

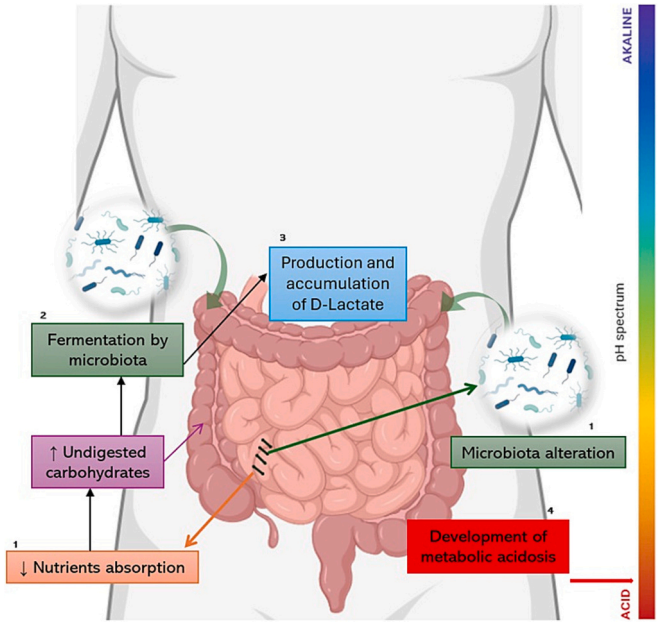

Fig. 1. Pathophysiology of D-Lactate acidosis in intestinal resection.

The abdominal cavity was thoroughly irrigated with warm saline to reduce contamination and sepsis risk. A negative pressure wound therapy (NPWT) system was applied at −125 mmHg. The surgical time was approximately 3 h, during which 3 to 4 l of Lactated Ringer's solution were administered, along with intravenous piperacillin/tazobactam. A transfusion of one unit of packed red blood cells was given due to an estimated blood loss of 800 mL, and norepinephrine was used to manage blood pressure. In the ICU, laboratory tests (Table 1) were ordered, and post-operative fasting was prescribed for 24 h, with gradual reduction of vasopressor support.

At 72 h, a second laparotomy was performed for further abdominal irrigation. The anastomosis was inspected and reinforced, ensuring adequate perfusion and closure. A terminal ileostomy was created, and the abdominal cavity was closed.

The management of intestinal evacuation was closely monitored following the creation of the ileostomy, which became functional within 48 h, with the first significant drainage of intestinal content recorded. The consistency and volume of the evacuated content were monitored, ensuring favorable progression without signs of complications.

ICU management was multidisciplinary, focused on stabilizing the patient and addressing complications from peritonitis and septic shock. The patient was intubated and managed with conscious sedation, without the need for neuromuscular blockers. Intubation was maintained until metabolic acidosis and septic shock were corrected. Prolonged antibiotic therapy with meropenem and caspofungin was administered according to the antibiogram. High-volume intravenous Lactated Ringer's solution was infused via an infusion pump to achieve adequate intravascular volume.

During her hospitalization, she experienced episodes of hemodynamic instability and hydroelectrolytic imbalance (Table 1), developing refractory metabolic acidosis (pH 7.32, pCO_2 22.2 mmHg, HCO_3 11.6 mmol/L, anion gap 33 mEq/L) without uremia, ketosis, or hyperlactatemia. Given the elevated anion gap (>12), D-lactate acidosis was suspected due to the extensive bowel resection, which predisposes the patient to fermentation of unabsorbed carbohydrates in the colon. Despite the absence of immediate laboratory results, empirical treatment was initiated.

The therapeutic approach focused on correcting the acid-base disorder and implementing nutritional management to prevent D-lactate production. Sodium bicarbonate ($NaHCO_3$) infusion was initiated to correct the acidosis, while nutritional management involved carbohydrate restriction through total enteral nutrition (TEN). During the first three days, carbohydrate intake was limited, and a protein module was provided, contributing 1.5 g/kg/day. As the acidosis was corrected and HCO_3^- levels normalized, slow-absorbing carbohydrates were progressively introduced using a high-protein, high-calorie formula, starting at 20 cc/h, with gradual increases based on tolerance. Subsequently, she is discharged to the hospital and then to her home.

3. Discussion

This study presents a case of D-lactate metabolic acidosis in a patient with intestinal resection, emphasizing diagnostic challenges and nutritional management strategies. Biochemical analysis confirmed elevated D-lactate levels (>6 mmol/L) 13 days post-admission. Clinical suspicion was present from the onset, suggesting that treatment response may aid in diagnosis prior to laboratory confirmation.

Nutritional intervention, focused on carbohydrate restriction, was essential for managing D-lactate acidosis, as carbohydrates are the primary substrate for colonic bacterial fermentation. After a 24-hour postoperative fast to resolve ileus and ensure anastomosis integrity, the patient initially received 20 kcal of standard nutrition. However, 48 h later, due to suspected lactic acidosis, the nutritional therapy was adjusted. Carbohydrate intake was completely restricted for the first

Table 1
Clinical laboratory values.

Laboratory parameters	Patient's values					
	Pre-surgical	Day 1	Day 2	Day 3	Day 4	Day 5
Hematology						
Hemoglobin (13.5–17.5 g/dL)	14,5	14	14	13,8	13,5	13
Hematocrit (40–50 %)	60 %	42 %	42 %	41 %	41 %	43 %
Platelets (150,000-450,000/μL)	255,000	250,000	249,000	247,000	230,000	238,000
Leukocytes (4500-11,000/μL)	13,200	7000	7000	7800	8900	8422
C-reactive protein (<2 mg/L)	3	2	1.5	1.3	1	0.6
Biochemistry						
Electrolytes						
Sodium (135–145 mmol/L)	142	140	136	138	144	140
Potassium (3.5–4.5 mmol/L)	3.7	4.0	3.6	4.2	4.1	4.0
Chloride (95–105 mmol/L)	101	100	102	104	104	100
Calcium (8.5–10.2 mg/dL)	9.5	9.2	9.0	8.9	9.1	9.3
Magnesium (1.7–2.2 mg/dL)	1.9	1.8	1.7	1.6	1.7	1.8
Arterial blood gases, mmHg						
pH (7.35–7.45)	7.24	7.32	7.05	7.06	7.30	7.39
pCO_2 (35–45)	26	22	26	24	27	37
PaO_2 (75–100)	86	75	79	83	80	83
HCO_3^- (22–26)	10	11	7	6	13	22
BE (−2–+2)	−13	−18	−13	−14	−7	−3
Lactate (0.5–2.2 mmol/L)	8	1.0	0.4	0.2	0.2	0.1
Ketonemia (<0.6 mmol/)		0.5		<0.5		
Renal function						
Creatinine (0.7–1.3 mg/dL)	0.8	0.9	0.9	0.9	1.0	1.1
Blood urea nitrogen (7–20 mg/dL)	27	15	18	18	17	18
Polycultures						
Blood cultures	*Negative*					
Peritoneal fluid culture	*Escherichia coli* *Enterococcus faecalis* *Bacteroides fragilis*					
Urine culture	Negative					

three days post-fast to prevent colonic fermentation and D-lactate production. The patient received TEN, based on a protein module providing 1.5 g/kg/day, with dietary fats to meet caloric needs without increasing fermentable substrates. As acid-base parameters stabilized and bicarbonate levels normalized, carbohydrates were gradually reintroduced following ileostomy placement, starting with slow-absorbing carbohydrates in a high-protein, high-calorie enteral formula. The infusion rate began at 20 cc/h to minimize the risk of exacerbating acidosis and was gradually increased based on patient tolerance and clinical stability. Although fructose-based formulas were considered due to their favorable absorption profile via facilitated diffusion, their limited availability prevented their use. Instead, complex carbohydrates were prioritized for their slower absorption and reduced risk of colonic fermentation. Nutritional intake, including caloric count, protein levels, and nitrogen balance, was closely monitored to prevent catabolism and ensure an adequate energy supply. Literature supports carbohydrate restriction to reduce bacterial fermentation, particularly in patients with bacterial overgrowth [1,2,11,12].

Intravenous bicarbonate was essential in stabilizing blood pH and improving clinical markers during episodes of severe acidosis. Although oral antibiotics may reduce D-lactate-producing bacteria, their use must be weighed against the risk of antimicrobial resistance [13,14]. There is no standardized nutritional protocol for D-lactate acidosis, but clinical evidence suggests that appropriate interventions can manage acute episodes and prevent complications. Clinicians should consider D-lactate acidosis in patients with poor nutrient absorption and an increased anion gap, and exclude other causes of high anion gap metabolic acidosis, such as L-lactate, ketonemia, and uremia [6,7,15].

The patient's management focused on carbohydrate restriction, which effectively reduced intestinal fermentation by eliminating substrates that promote D-lactate production. Limiting carbohydrate intake suppresses the growth of D-lactate-producing bacteria, leading to improved clinical outcomes [2,11]. Carbohydrate restriction can be achieved using enteral formulas that consist exclusively of fructose or

starch. A case study [16] highlighted those reintroducing carbohydrates in a pediatric patient with D-lactate acidosis resulted in increased D-lactate levels, emphasizing the need for careful management. Fructose, absorbed via facilitated diffusion, prevents other carbohydrates from reaching the colon for fermentation. In patients with short bowel syndrome, bacterial overgrowth and mucosal inflammation can impair active transport mechanisms, leading to poor absorption of glucose and sucrose, unlike fructose [1,3].

Fructose-based feeding was considered, but it was infeasible due to challenges in meeting nutritional requirements with available formulas in Colombia. Therefore, all carbohydrates were initially eliminated, and protein intake was tailored to prevent excessive fermentation and decrease hydrogen ion production, improving metabolic acidosis and its clinical manifestations [6,8].

A significant challenge was meeting the patient's daily metabolic requirements while preventing catabolism. Gradually, formulas containing complex carbohydrates were introduced, given their slower absorption compared to simple carbohydrates. This decision was based on the principle that metabolic acidosis can be reversed by decreasing the fermentable substrate [9,11]. Treatment with intravenous bicarbonate was established as an integral component of acute management, particularly during episodes of severe acidosis. Monitoring bicarbonate levels is crucial to avoid hypernatremia, highlighting the need for continuous electrolyte monitoring in critically ill patients [14,15].

Oral antibiotics may reduce bacterial load, but antibiotic resistance remains a concern. Probiotics have shown potential benefits in D-lactate acidosis cases, underscoring the importance of prompt recognition and management, particularly in conditions like short bowel syndrome (SBS) [17,18]. Documented cases of D-lactic acidosis in pediatric patients include a 14-year-old boy with SBS due to midgut volvulus, who had a D-lactate level of 11.21 mmol/L and responded positively to bicarbonate infusion and carbohydrate restriction. A 10-month-old infant with SBS secondary to gastroschisis also experienced D-lactate acidosis, with management including intravenous fluids with bicarbonate and metronidazole, resulting in normalization of neurological symptoms and acid-base balance. In another case, a child with recurrent episodes of D-lactate acidosis responded positively to probiotics, preventing further episodes [3,17].

Survival statistics for patients with D-lactic acidosis following intestinal resection, particularly those with SBS, are not well-documented. However, available data indicate a concerning prognosis for SBS patients, with a 5-year mortality rate of approximately 37.5 % [19], reflecting the significant risks associated with the condition. Although D-lactate elevation is rare, it can complicate outcomes due to diagnostic challenges, as its nonspecific symptoms are often mistaken for other metabolic or neurological disorders. Early diagnosis and intervention can improve outcomes, with some patients returning to normal activities after treatment. Nutritional strategies, such as a low-carbohydrate diet, are essential for treatment and prevention, helping reduce the risk of D-lactate accumulation and recurrence [11]. Long-term outcomes vary, but continuous monitoring and adherence to dietary restrictions are crucial for minimizing recurrence and improving functional status. Despite limited survival data for D-lactic acidosis following intestinal resection, early diagnosis, appropriate treatment, and nutritional interventions are essential for improving prognosis and quality of life [20].

Postoperative mobilization was carried out by specialized physiotherapists following an early mobilization protocol. This began in the early postoperative days, once safety criteria were met. After ICU discharge, personalized recommendations were made, focusing on short bowel syndrome management, nutrition, and complication prevention. Carbohydrate intake was restricted, especially simple carbohydrates, to reduce D-lactate production and prevent acidosis. Supplementation with fat-soluble vitamins (A, D, E, K), vitamin B12, and periodic evaluations of nutritional needs were prioritized.

A multidisciplinary rehabilitation plan aimed to prevent physical deconditioning and promote recovery of musculoskeletal function [21]. Regular follow-ups included nutritional assessments, liver and renal function monitoring, and laboratory tests (complete blood count, electrolytes, vitamins, and essential minerals). Bone health was monitored annually through bone densitometry. Dietary recommendations focused on limiting simple carbohydrates, prioritizing complex carbohydrates, and ensuring adequate protein intake while moderating fat consumption. Regular physical activity and tailored rehabilitation were encouraged to maintain muscle and bone health.

The strengths of this report include its detailed clinical and nutritional approach, which serves as a valuable guide for treating patients with D-lactate acidosis. Management should be individualized based on each patient's specific conditions and limitations in accessing therapeutic resources, such as fructose-based nutritional formulas. This experience emphasizes the importance of interprofessional collaboration in optimizing the treatment of metabolic acidosis and highlights the need for future research on accessible and effective nutritional formulas.

4. Conclusion

Early diagnosis of D-lactate metabolic acidosis is crucial for effective management, which emphasizes dietary carbohydrate restriction. This intervention reduces the substrate available for intestinal bacterial fermentation, thereby limiting D-lactate production and improving the patient's clinical condition.

Abbreviations

HCO_3^-	Bicarbonate
ICU	intensive care unit
BMI	Body mass index
$NaHCO_3$	Sodium bicarbonate
TEN	enteral nutrition
SBS	short bowel syndrome

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethical approval

We certify that this kind of manuscript does not require ethical approval (exemption) by the Ethical Committee of our institution.

Guarantor

Olid Iván Ochoa

Research registration number

Does not apply, it is a case report.

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Author contribution

Leonardo Arzayus Patiño; Conceptualization, methodology, project administration, writing—review and editing, funding acquisition

Claudia Martínez Fuentes; Conceptualization, methodology, writing—review and editing

Olid Iván Ochoa; Conceptualization, methodology, writing—review

and editing

Jose Luis Estela-Zape; Methodology, validation, formal analysis, investigation, project administration, writing—original draft preparation, writing—review and editing, funding acquisition, supervision, visualization

Conflict of interest statement

Authors declare that they have no conflict of interest.

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Corrigendum

Corrigendum to "Metabolic acidosis due to d-lactate in a patient with intestinal resection: Diagnostic challenges and nutritional strategies" [Int. J. Surg. Case Rep. 126 (2025) 110801]

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