D-lactic acidosis in short bowel syndrome: are probiotics friend or foe? A case report

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Case presentation

A 6-year-old Chinese boy with short bowel syndrome (SBS) presented to the emergency department with excessive drowsiness. He was born full term with an unremarkable perinatal history and had good past health. At 3 years of age, he underwent extensive small bowel resection and a right hemicolectomy due to intestinal malrotation with midgut volvulus, resulting in a residual length of 66 cm of proximal small bowel and distal colon, with loss of the ileocaecal valve.

Initially dependent on total parenteral nutrition, he achieved enteral autonomy 3 years later, consuming an oral diet supplemented with vitamins, iron, and a hydrolysed formula of 1 kcal/mL, contributing approximately 20% of total energy intake. A timeline summarising key clinical events, including enteral and parenteral nutrition milestones, is presented in the Figure.

He was reviewed monthly by a multidisciplinary team with regular assessments of his nutritional status, growth parameters, and biochemical profile. He demonstrated good growth, maintaining weight and height at the 50th percentile, with regular bowel movements with daily oral loperamide. His biochemical profile, including blood counts, liver function, electrolytes, blood gas, and trace elements, remained stable throughout the follow-up period.

On this admission, the patient was drowsy and lethargic but not confused. Blood tests indicated high anion gap metabolic acidosis, with a pH of 7.31, bicarbonate 10.8 mmol/L, pCO_2 2.9 kPa, and

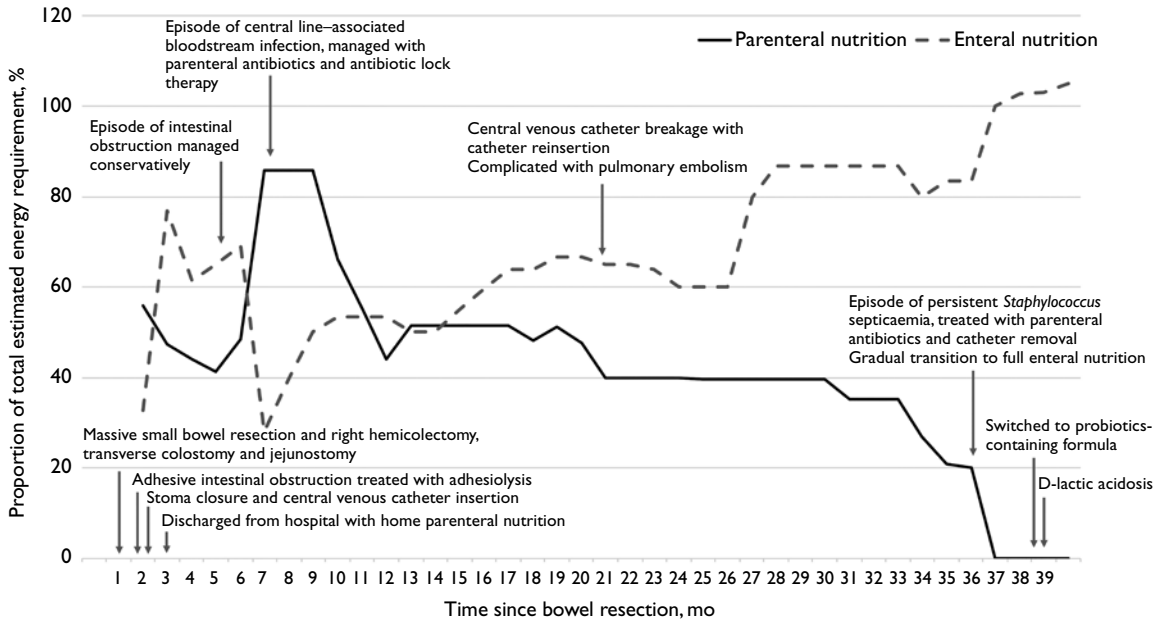

FIG. Summary of major events and respective percentages of total estimated energy requirement contributed by parenteral nutrition and enteral nutrition

L-lactate 1.6 mmol/L (reference range, 1.0-2.4 mmol/L). Complete blood counts, liver enzymes, ammonia, electrolytes, glucose levels, and computed tomography of the brain were normal. D-lactic acidosis (D-LA) was confirmed by an elevated serum D-lactate concentration of 1.7 mmol/L (normal range, <0.5 mmol/L). Further enquiry revealed that one week prior, his family had switched to an alternative commercially available enteral formula containing probiotics (*Lactobacillus paracasei* and *Bifidobacterium longum*) as the original formula was temporarily out of stock (Table). The rest of his oral diet remained unchanged. Total carbohydrate (CHO) intake accounted for 40% to 50% of his total enteral intake, with the formula contributing 20%.

His condition improved rapidly with bowel rest and oral sodium bicarbonate. He was treated with a course of oral metronidazole. The probiotic-containing formula was stopped, and he was instructed to resume the original probiotic-free hydrolysed formula along with CHO-restricted meals. His carers were re-educated on CHO counting and avoidance of simple sugars. He remained clinically stable the following months, during which he maintained good dietary compliance.

Discussion

Short bowel syndrome refers to a condition of intestinal malabsorption resulting from loss or surgical resection of the small intestine and is the leading cause of intestinal failure. It encompasses a heterogeneous group of patients with various aetiologies and bowel anatomies. Effective management requires a multidisciplinary approach to promote enteral autonomy, support growth, and prevent complications such as catheter-related bloodstream infections and intestinal failure–associated liver disease.

D-lactic acidosis, first described in SBS by Oh et al in 1979,[1] has gained increasing recognition as a rare but serious metabolic complication. It results from intestinal malabsorption and overgrowth of colonic microbiota (eg, *Lactobacillus* spp, *Bifidobacterium* spp), leading to excessive fermentation of unabsorbed CHO. The process is exacerbated by factors such as high CHO intake, elevated gut pH, impaired gut motility, antimicrobials, probiotics, and intestinal infections. The overproduction of D-lactic acid leads to a neurological syndrome and high anion gap metabolic acidosis. Clinical manifestations include acidotic breathing, altered mental state, ataxia, slurred speech, nystagmus, gait disturbance, behavioural change, and fatigue. A high index of clinical suspicion and measurement of D-lactic acid are essential for diagnosis, as serum lactate concentration (reflecting L-lactate) is often normal.[2,3]

TABLE. Comparison of contents of the original and new formula

Contents (per 100 g powder)	Original formula	New formula
Energy	465 kcal	463 kcal
Carbohydrate	62.8 g	60.7 g
Protein	13.7 g	13.9 g
Total fat	17.5 g	18.3 g
Lactobacillus paracasei	-	1×10^8 CFU
Bifidobacterium longum	-	1×10^8 CFU

The mainstays of acute management of D-LA include correction of metabolic acidosis with bicarbonate and rehydration, restriction of enteral CHO intake, administration of poorly absorbed oral antibiotics, and avoidance of antimotility agents or lactate-containing solutions. Additional treatment may include thiamine and riboflavin supplementation, insulin, and short-chain fatty acids. Metabolic acidosis and neurological symptoms often improve rapidly with early and appropriate intervention. To prevent recurrence, CHO restriction and avoidance of D-lactate–containing foods (eg, pickles and yoghurt) are essential. In selected cases, suppression of abnormal gut flora with antimicrobials or surgery to increase bowel absorptive area may be considered.[3,4]

Probiotics have gained popularity as health-promoting agents in medicines and dietary supplements, including in the management of SBS to prevent and treat small intestinal bacterial overgrowth. Certain species, such as *Lactobacillus casei*, produce only L-lactate. Among commercially available probiotics, *Lactobacillus* and *Bifidobacterium* are the most commonly used genera.[2,5] The European Society for Paediatric Gastroenterology, Hepatology and Nutrition has summarised the latest evidence on probiotic use across various paediatric gastrointestinal disorders.[6] Strain-specific benefits have been demonstrated in conditions such as acute gastroenteritis, antibiotic-associated diarrhoea, infantile colic, functional abdominal disorders, and in the prevention of necrotising enterocolitis and nosocomial diarrhoea.[7]

Animal studies and clinical case reports suggest that probiotics may confer potential benefits in patients with SBS through mechanisms such as enhancement of gut barrier function, suppression of pathogens, and modulation of immune responses.[8] Nevertheless, clinical studies evaluating their efficacy remain limited, and there is insufficient evidence to support the routine use in SBS. Conversely, case reports have raised safety concerns, such as the development of D-LA and sepsis in children with SBS following probiotic administration.[8] In our

case, the addition of probiotics via the new milk formula suggests a possible role of probiotics in the development of D-LA. This highlights the need for cautious and selective use of non–D-lactate–producing probiotic strains in patients at high risk of D-LA.

This report illustrates a case of D-LA in a paediatric patient with SBS, precipitated by the intake of a probiotic-containing enteral formula. Early recognition of D-LA, based on characteristic clinical features and confirmed by D-lactate measurement, with prompt treatment to normalise acidosis and suppress D-lactate production, is essential. Cautious dietary management, including caregiver awareness of formula contents and dietary CHO restriction, is equally important. Despite the increasing medical use of probiotics, there is a lack of clinical trials to support their routine use or provide clear guidance for their use in paediatric SBS. Careful consideration is warranted, with awareness of potential strain-specific benefits and risks, particularly in patients with altered intestinal microbiota and malabsorption.

Author contributions

Concept or design: All authors.
Acquisition of data: All authors.
Analysis or interpretation of data: All authors.
Drafting of the manuscript: BPY Leung.
Critical revision for important intellectual content: All authors.

All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

All authors have disclosed no conflicts of interest.

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Declaration

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Ethics approval

The patient was treated in accordance with the Declaration of Helsinki and provided written informed consent for all treatments, procedures and the publication of this case report.

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