J Crit Care 89 (2025) 155138

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.journals.elsevier.com/journal-of-critical-care

Letter to the Editor

Letter to the editor: "What every intensivist should know about type D hyperlactatemia"

ARTICLE INFO

Keywords:
D-lactic acidosis
Hyperlactatemia
Critical care
Therapy
Prevention

The recent article by Ballarin et al., titled "What every intensivist should know about type D hyperlactatemia", is commendable and provides a valuable overview of the condition [1].

While the treatment section is well presented, the overall therapeutic approach remains limited, and preventive measures, which are crucial to avoiding the occurrence of type D hyperlactatemia, are not addressed.

A recent case report described the use of synbiotics not only as a treatment but also as a preventive approach for type D hyperlactatemia in patients with short bowel syndrome (SBS) [2].

Synbiotics are a combination of probiotics and prebiotics that have recently been applied in various gastrointestinal conditions, including infectious enteritis, inflammatory bowel disease, and bowel obstruction. Probiotics are live microbial supplements that exert beneficial effects on the host by improving intestinal microbial balance. Prebiotics are non-digestible food components that selectively promote the growth and activity of specific bacteria in the colon, thereby improving host health. In recent years, several reports have described the use of probiotics or synbiotics in the management of SBS.

Takahashi et al. described the case of a patient with recurrent D-lactic acidosis associated with SBS, who experienced repeated neurologic symptoms. In addition to fasting, the patient was treated with antibiotics to eliminate D-lactate–producing bacteria. After the failure of antibiotic treatment, a stand-alone synbiotic treatment was initiated, consisting of *Bifidobacterium breve* Yakult and *Lactobacillus casei* Shirota as probiotics, and galacto-oligosaccharide as a prebiotic. Serum D-lactate levels decreased, and the patient has remained free of recurrence for three years without dietary restrictions. Synbiotic therapy allowed the reduction in colonic absorption of D-lactate by both preventing overgrowth of D-lactate-producing bacteria and stimulating intestinal motility, leading to remission of D-lactate acidosis [2].

Several reports have described the development of D-lactic acidosis in patients with SBS following the administration of various antibiotics, including tetracycline, metronidazole, and vancomycin. In this reported case, after the failure of antibiotic therapy, other strategies had to be considered. Although surgical intervention remained an option for the prevention of D-lactic acidosis [3], a synbiotic treatment was chosen as a promising alternative due to its safety and favorable compliance rates.

Kanamori et al. reported a remarkable improvement in intestinal absorptive function and motility in a patient with SBS treated with synbiotics [4]. Uchida et al. described the successful management of recurrent D-lactic acidosis in SBS using a combination of antibiotics and probiotics over an 8 months period [5].

Notably, Godey et al. reported that enteral treatment with Lactobacillus specifically should be avoided due to its association with increased D-lactate production [6].

Bustos et al. reported that fecal concentrations of D-lactate tended to be high in patients with SBS even when serum D-lactate levels were undetectable. The authors noted that the development of D-lactic acidosis depends not only on overproduction of D-lactate in the colon, but also on additional factors such as enhanced absorption or impaired metabolism of D-lactate [7].

Synbiotics have been shown to increase the production of short-chain fatty acids by correcting intestinal microbiota [6]. These fatty acids promote proliferation of the intestinal epithelium and stimulate intestinal motility [8]. Through enhanced bowel movements, D-lactate produced by colonic fermentation can be eliminated in feces before excessive absorption occurs in the colon. In the present case, although fecal D-lactate concentration was elevated (24.8 mmol/L), serum D-lactate concentration (2.4 mmol/L) remained below the threshold (2.5 mmol/L) for symptom onset, which allowed the patient to remain asymptomatic.

In conclusion, we believe that the original commentary omits key considerations in the management and prevention of type D hyperlactatemia, particularly the role of synbiotic therapy as a viable and underrecognized option.

CRediT authorship contribution statement

Julien Moury: Conceptualization, Writing – original draft, Writing – review & editing. **Thibault Gennart:** Writing – original draft, Writing – review & editing. **Sydney Blackman:** Writing – original draft, Writing – review & editing. **Nathan De Lissnyder:** Writing – original draft, Writing – review & editing. **Oumayma Errais:** Writing – original draft, Writing – review & editing. **Patrick M. Honore:** Conceptualization, Supervision, Writing – original draft, Writing – review & editing.

https://doi.org/10.1016/j.jcrc.2025.155138
Received 12 May 2025; Accepted 2 June 2025
Available online 7 June 2025

Consent for publication

Not applicable.

Ethics approval and consent to participate

Not applicable.

Funding

None.

Declaration of competing interest

The authors declare to have no competing interests.

Acknowledgements

None.

Data availability

Not applicable.

References

[1] Ballarin RS, Lazzarin T, Minicucci MF. What every intensivist should know about type D hyperlactatemia. J Crit Care 2025 Mar 6;88:155049.
[2] Takahashi K, Terashima H, Kohno K, Ohkohchi N. A stand-alone synbiotic treatment for the prevention of D-lactic acidosis in short bowel syndrome. Int Surg 2013 Apr-Jun;98(2):110–3.
[3] Modi BP, Langer M, Duggan C, Kim HB, Jaksic T. Serial transverse enteroplasty for management of refractory D-lactic acidosis in short-bowel syndrome. J Pediatr Gastroenterol Nutr 2006;43(3):395–7.
[4] Kanamori Y, Hashizume K, Sugiyama M, Morotomi M, Yuki N. Combination therapy with Bifidobacterium breve, Lactobacillus casei, and galactooligosaccharides dramatically improved the intestinal function in a girl with short bowel syndrome: a novel synbiotics therapy for intestinal failure. Dig Dis Sci 2001 Sep;46(9):2010–6.
[5] Uchida H, Yamamoto H, Kisaki Y, Fujino J, Ishimaru Y, Ikeda H. D-lactic acidosis in short-bowel syndrome managed with antibiotics and probiotics. J Pediatr Surg 2004;39(4):634–6.
[6] Godey F, Bouasria A, Ropert M, Diakite M, Le Treut A, Balençon M. Don't forget to test for D-lactic acid in short bowel syndrome. Am J Gastroenterol 2000;95(12):3675–7.
[7] Bustos D, Pons S, Pernas JC, Gonzalez H, Caldarini MI, Ogawa K, et al. Fecal lactate and short bowel syndrome. Dig Dis Sci 1994;39(11):2315–9.
[8] Cherbut C, Aubé AC, Blottière HM, Galmiche JP. Effects of short-chain fatty acids on gastrointestinal motility. Scand J Gastroenterol Suppl 1997;222:58–61.

Julien Moury[a], Thibault Gennart[a], Sydney Blackman[b], Nathan De Lissnyder[c], Oumayma Errais[d], Patrick M. Honore[a,*]
[a] *ICU, CHU UCL Godinne Namur, UCLouvain Medical School, Belgium*
[b] *CHIREC, ULB University, Brussels, Belgium*
[c] *UZ Brussel, VUB University, Brussels, Belgium*
[d] *Medical Student, ULB University, Brussels, Belgium*

[*] Corresponding author at: ICU Dept, CHU UCL Godinne Namur, UCL Louvain Medical School, Campus Godinne, Avenue G Thérasse 1, 5530 Yvoir, Belgium.
E-mail addresses: Julien.Moury@chuuclnamur.uclouvain.be (J. Moury), Thibault.Gennart@chuuclnamur.uclouvain.be (T. Gennart), Oumayma.errais@ulb.be (O. Errais), patrick.honore@chuuclnamur.uclouvain.be (P.M. Honore).