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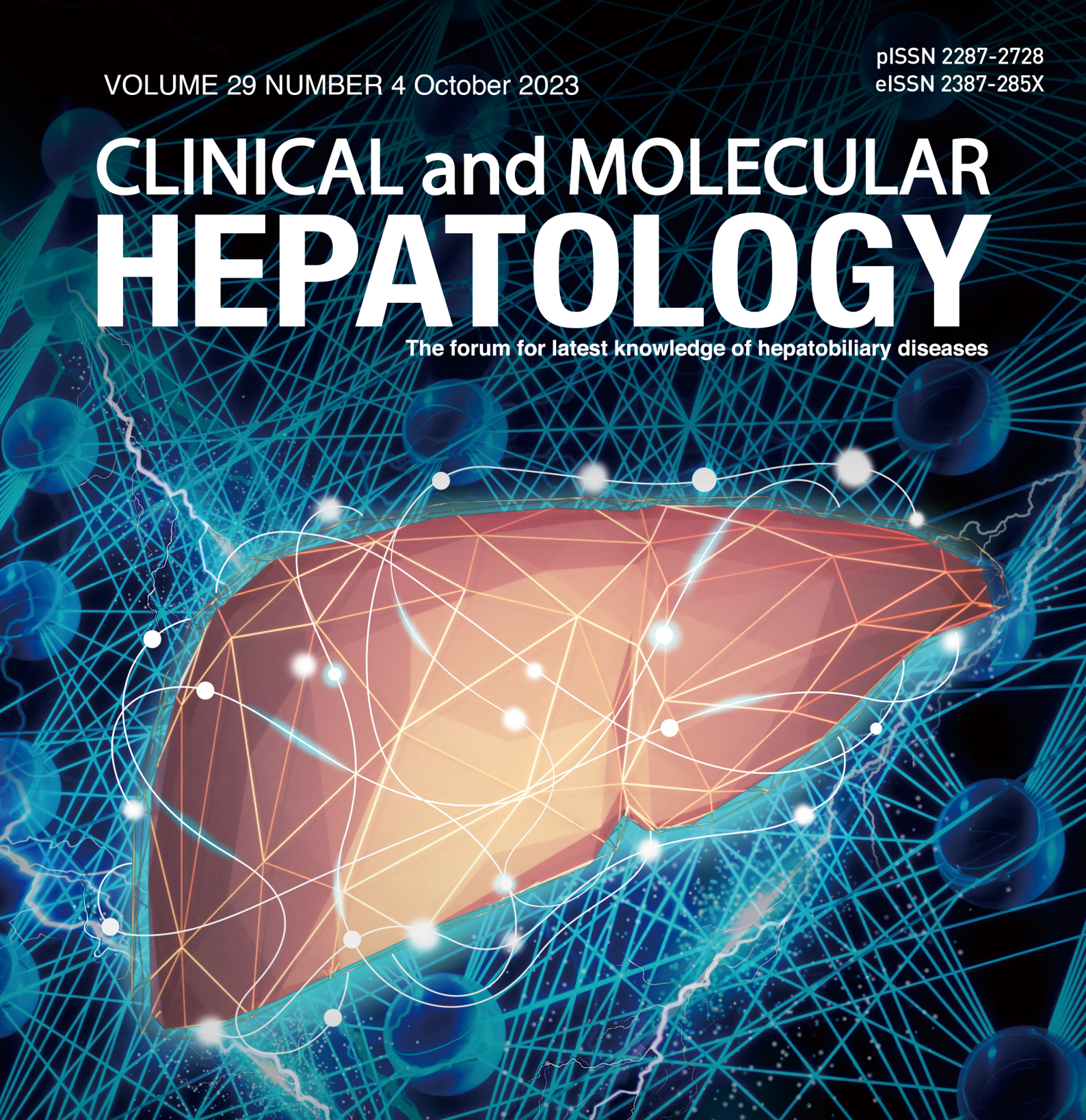

Deep learning-based CT analysis in CHB patients

Lean NAFLD & mortality
Ranking of locoregional treatments for small HCC
Visceral fat obesity, sarcopenia, myosteatosis in NAFLD without obesity

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Correspondence

Correspondence on Letter regarding "Evidence-based hyponatremia management in liver disease"

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Keywords: Hyponatremia; Liver cirrhosis; Liver disease; Water-electrolyte imbalance

Dear Editor,

We appreciate Theodorou and coauthors[1] for introducing clinical manifestation of osmotic demyelination syndrome (ODS) including reversible and irreversible sequelae and emphasizing that magnetic resonance (MR) imaging is needed for early detection and proper diagnosis of ODS in hyponatremic patients with liver disease. Furthermore, MR imaging might have prognostic value due to the regenerative potential of neuroglial cells in patients with liver disease and resolving ODS.[1,2] Generally, ODS is diagnosed clinically and by MR image.[3,4]

Our review focused on pathophysiology, diagnosis, and treatment of hyponatremia in patients with liver diseases.[5] We were unable to discuss ODS itself thoroughly. ODS is symmetric, non-inflammatory demyelination of neurons, which can be classified into two types based on location: central pontine myelinolysis and extrapontine myelinolysis.[6-8] It occurs as a result of apoptosis of oligodendrocytes and infiltration of myelin degrading macrophages.[8,9] Hyponatremia and overly rapid correction of hyponatremia have been well-known as potent causative factors of ODS.[4,10-12] The only recommendation of ODS till date is conservative treatment. The best approach is focused on prevention strategies with two aspects: identifying patients at risk and implementing proper correction, especially with a strict maximum of 8 mmol/L per day for individuals at risk of ODS.[10-12] However, it should be noted that ODS can occur even in the absence of hyponatremia or overcorrection of hyponatremia in patients with high risk of ODS. Patients with chronic alcohol consumption (the most common) or liver cirrhosis/liver transplantation (third largest group) are more susceptible to ODS because of reduced ability of astrocytes to synthesize new intracellular osmolytes in response to osmotic changes.[4,7,10-15] In a recent study involving 547,544 adult inpatients with cirrhosis, ODS was found to be developed in only 0.02% of patients. It was associated with alcohol-related cirrhosis, young age, and female gender. ODS was not associated with liver disease severity (decompensated cirrhosis) or specific complications including ascites or hepatic encephalopathy.[13] Patients undergoing liver transplant are also at risk for rapid correction of serum sodium due to intraoperative administration of

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intravenous crystalloids, blood products, and sodium bicarbonate during operation, in addition to preexisting conditions.[4] The incidence of ODS is 0.8% to 1,4%.[3,4] Symptom onset is known to be within 1 to 2 weeks after liver transplantation.[3] Additionally, although relatively less prevalent, ODS can occur in patients with burns, malnutrition, chemotherapy, diabetes mellitus, adrenal insufficiency, acquired immune deficiency syndrome, severe illness/sepsis, hypoglycemia/hypokalemia/hypophosphatemia, and renal disease with or without liver disease.[4]

In summary, individuals with advanced liver disease are more susceptible to ODS. For patients with liver diseases accompanied by aforementioned predisposing disease or circumstances or those undergoing liver transplantation, greater attention should be paid to ODS.

Authors' contribution

JYR: drafting of the manuscript. SHB, SK: critical review and final approval of the manuscript.

Conflicts of Interest

The authors have no conflicts to disclose.

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Abbreviations:
ODS, osmotic demyelination syndrome; MR, magnetic resonance