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Study of Serum Sodium Level in Decompensated Alcoholic Liver Disease and it's Clinical Significance in Tertiary Care Centre

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HIGHLIGHTS

- DALD causes major global morbidity.
- Study included 180 male patients.
- Hyponatremia common in liver cirrhosis.
- Low sodium linked with severity.
- MELD-Na improves prognosis prediction.

Key Words:
Decompensated alcoholic liver disease
Serum sodium
Hyponatremia
MELD-Na score
Child-Pugh classification

ABSTRACT

Introduction: Decompensated alcoholic liver disease (DALD) is a major cause of morbidity and mortality worldwide, with complications including ascites, hepatic encephalopathy, hepatorenal syndrome, and spontaneous bacterial peritonitis. Hyponatremia, defined as serum sodium <135 mEq/L, is common in cirrhosis and strongly influences prognosis. This study was conducted to evaluate the clinical significance of serum sodium levels and their correlation with disease severity in patients with DALD. **Material & Methods:** A prospective observational study was conducted at a tertiary care center from July 2023 to June 2025. A total of 180 male patients aged >18 years, diagnosed with DALD, were enrolled based on clinical, biochemical, and imaging findings. Patients with cardiac failure, chronic kidney disease, or on interfering medications were excluded. Clinical history, laboratory investigations, and imaging were recorded. Disease severity was assessed using Model for End-Stage Liver Disease (MELD), MELD-Na, and Child-Pugh scoring systems. Data were analyzed using SPSS, with p <0.05 considered significant. **Results:** Among 180 patients, the majority were aged 36–50 years (62.78%), with farming as the most common occupation (27.78%). Hyponatremia (<135 mEq/L) was present in 34.44%, normonatremia in 51.11%, and hypernatremia in 14.44%. Patients with sodium <135 mEq/L demonstrated higher MELD scores (27–31 in 13.89% cases) and were more frequently classified as Child-Pugh class C (22.78%). Hyponatremia was strongly associated with severe complications, including ascites, hepatic encephalopathy, and hepatorenal syndrome. **Conclusion:** Serum sodium is a simple, inexpensive, and reliable biomarker in DALD. Hyponatremia correlates with advanced disease severity and poor prognosis, emphasizing its utility in risk stratification. Integration of serum sodium into prognostic scoring systems like MELD-Na enhances predictive accuracy, supporting early intervention and improved patient management in tertiary care settings.

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INTRODUCTION

Chronic liver disease poses a significant global health challenge due to its high morbidity and mortality. Its most advanced stage, decompensated liver disease (DCLD), is characterized by complications such as ascites, hepatic encephalopathy, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome (HRS), and variceal bleeding. Among the various etiologies, alcoholic liver disease (ALD) is a leading contributor to liver decompensation. Monitoring serum sodium, a key clinical parameter, is essential for the accurate diagnosis and effective management of decompensated alcoholic liver disease (DALD) [1].

Patients with decompensated cirrhosis frequently present with hyponatremia, defined as serum sodium levels below 135 mEq/L. The pathophysiology of hyponatremia in cirrhosis primarily involves increased vasopressin secretion and reduced renal water excretion, leading to a dilutional decrease in serum sodium. This electrolyte imbalance is closely associated with disease severity, complications, and mortality. Recent studies have highlighted the inclusion of serum sodium in prognostic scoring systems such as the Model for End-Stage Liver Disease (MELD-Na) to improve predictive accuracy [2,3].

Despite the recognized association between low serum sodium and disease severity, its clinical significance in DALD within tertiary-care settings remains inadequately explored. With the rising prevalence of alcoholic liver disease, evaluating serum sodium as a prognostic biomarker could enhance early intervention and optimize patient management. The primary objective of this study is to assess serum sodium concentrations in patients with decompensated alcoholic liver disease and determine their clinical relevance in a tertiary-care environment. In decompensated cirrhosis, splanchnic vasodilation triggers compensatory neurohormonal responses, including activation of the sympathetic nervous system, antidiuretic hormone release, and stimulation of the renin–angiotensin–aldosterone system. These mechanisms promote sodium and fluid retention, ultimately resulting in hyponatremia. Two primary types of hyponatremia are observed in DALD: dilutional hyponatremia, caused by water retention exceeding sodium retention, and hypovolemic hyponatremia, resulting from sodium loss due to diuretic therapy or gastrointestinal fluid losses [4].

Multiple studies have demonstrated an inverse correlation between serum sodium levels and disease severity as measured by MELD and Child-Pugh scores. Hyponatremia is consistently associated with higher mortality rates, increased risk of complications, and poorer clinical outcomes. Serum sodium has emerged as a robust predictor of both mortality and transplant outcomes in liver disease. Patients with severe hyponatremia, defined as serum sodium below 125 mEq/L, exhibit markedly reduced survival compared to those with normal sodium levels. The incorporation of serum sodium into the MELD-Na score has improved its predictive value, particularly for liver transplant prioritization [5].

Hyponatremia also contributes to several cirrhosis-related complications. In hepatic encephalopathy (HE), low sodium levels are linked to brain edema and cognitive dysfunction, exacerbating disease severity [6]. In patients with ascites and spontaneous bacterial peritonitis (SBP), hyponatremia is frequently associated with refractory ascites, necessitating repeated paracentesis and albumin therapy [7]. Moreover, in hepatorenal syndrome (HRS), hyponatremia serves as an indicator of poor renal perfusion and progressive kidney dysfunction, often culminating in type 1 HRS, which carries a high mortality rate [8].

Given its pathophysiological significance and prognostic value, serum sodium is a crucial biomarker in the diagnosis and management of decompensated alcoholic liver disease. Understanding its implications allows clinicians in tertiary-care settings to perform early risk assessments, implement timely interventions, and optimize patient outcomes, ultimately improving survival and reducing complications in this vulnerable population.

The aimed of the study is to evaluated serum sodium levels in patients with decompensated alcoholic liver disease. The primary objective is to assess the association of serum sodium levels with decompensated alcoholic liver disease, while the secondary objective focuses on examining the correlation between serum sodium levels and the severity of the disease.

MATERIAL & METHODS

This prospective observational study was conducted at the Department of General Medicine, at a tertiary care hospital from July 2023 to June 2025. Ethical approval has been obtained from the Ethical Approval Committee of a tertiary care hospital.

Study Population

The study population will include patients diagnosed with decompensated alcoholic liver disease who are over 18 years of age and willing to participate by providing written informed consent. Patients with cardiac failure, chronic kidney disease, or those receiving medications such as diuretics, selective serotonin reuptake inhibitors, tricyclic antidepressants, monoamine oxidase inhibitors, or cytotoxic drugs will be excluded to ensure accurate assessment of serum sodium levels in relation to the severity of alcoholic liver disease.

Data Analysis

Patient data were collected using a standardized proforma, including demographic details, medical history, clinical findings, laboratory and imaging results, and then securely entered into an electronic database for analysis. Data were processed using Microsoft Excel and SPSS (IBM, USA), with quantitative variables expressed as mean ± standard deviation and qualitative variables as proportions. Group means were

compared using the unpaired t-test, categorical associations with Chi-square or Fisher's exact test, and correlations between continuous variables with Pearson's coefficient, considering $p < 0.05$ as significant.

RESULTS

In this study of 180 male patients with decompensated alcoholic liver disease, the majority (62.78%) were aged 36–50 years, followed by 21.11% aged 51–65 years, and 16.11% aged 18–35 years. Regarding occupation, most patients were farmers (27.78%), followed by those in service (23.33%), laborers/servants/sweepers (21.67%), self-employed (13.89%), and businessmen (13.33%). Only 11.67% of patients practiced exercise, while 88.33% did not. Body weight was predominantly between 51–60 kg (57.22%), with 24.44% between 61–70 kg, 11.11% ≤50 kg, and 7.22% >70 kg. In patients with decompensated alcoholic liver disease, 6.11% had hypertension, while 8.33% had diabetes mellitus. Among patients with decompensated alcoholic liver disease, 3.33% tested positive for Hepatitis B, while 96.67% tested negative. In patients with decompensated alcoholic liver disease, 51.11% had serum sodium levels between 135–145 mEq/L, 34.44% had levels below 135 mEq/L, and 14.44% had levels above 145 mEq/L. In patients with decompensated alcoholic liver disease, 48.89% had serum potassium below 3.5 mEq/L, 44.44% between 3.5–5.0 mEq/L, and 6.67% above 5.0 mEq/L, while all patients had total bilirubin above 1.2 mg/dL. In patients with decompensated alcoholic liver disease, 97.78% had aspartate transaminase (SGOT) levels above 40 U/L, while 2.22% had levels between 10–40 U/L.

In patients with decompensated alcoholic liver disease, 94.44% had alanine transaminase (SGPT) levels above 45 U/L, while 5.56% had levels between 15–45 U/L.

In this study of 180 patients with decompensated alcoholic liver disease, 60% had KFT urea levels above 40 mg/dL, while 40% were between 7–40 mg/dL. Creatinine levels were 0.6–1.2 mg/dL in 53.33% of patients, above 1.2 mg/dL in 40%, and below 0.6 mg/dL in 6.67%. INR values exceeded 1.4 in 91.11% of patients. Serum albumin was below 3.5 g/dL in 59.44% and between 3.5–5.5 g/dL in 40.56% of patients. Ultrasound of patients with decompensated alcoholic liver disease showed that 42.78% had liver cirrhosis with portal hypertension, moderate ascites, and spleenomegaly, 10% had cirrhosis with portal hypertension and mild ascites, and 7.22% had altered liver echotexture with mild ascites. Among patients with decompensated alcoholic liver disease, 30% had a MELD score of 17–20, 22.22% scored 23–26, and 20% scored 21–22. Among patients with decompensated alcoholic liver disease, 44.44% had a Child-Pugh score of B, 32.78% scored A, and 22.78% scored C. Among patients with decompensated alcoholic liver disease, those with serum sodium below 135 mEq/L mostly had MELD scores of 27–31 (13.89%) and 23–26 (9.44%), sodium 135–145 mEq/L mostly had MELD 17–20 (23.89%) and 23–26 (9.44%), and sodium above 145 mEq/L had MELD 17–20 and 21–22 equally (5% each).

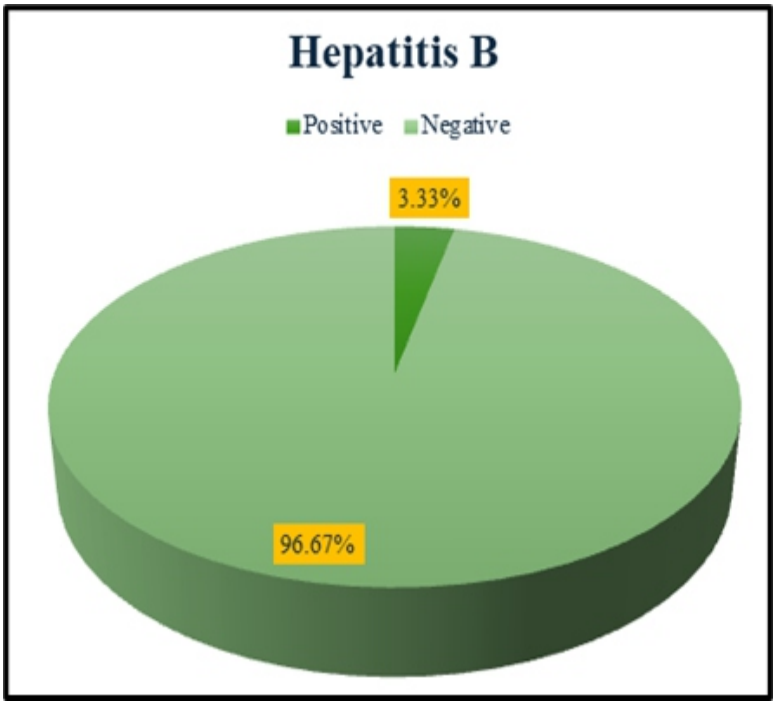

Figure 1: Hepatitis B of Patients with Decompensated Alcoholic Liver Disease

Table 1: Laboratory findings of Patients with Decompensated Alcoholic Liver Disease

Laboratory Parameters	Present	Absent	Total
Hypertension (HTN)	11 (6.11%)	169 (93.89%)	180 (100.00%)
DM (Diabetes Mellitus)	15 (8.33%)	165 (91.67%)	180 (100.00%)

Table 2: Serum Sodium of Patients with Decompensated Alcoholic Liver Disease

Serum Sodium	Count (%)
<135 mEq/L	62 (34.44%)
135-145 mEq/L	92 (92.11%)
>145 mEq/L	26 (14.44%)
Total	180 (100.00%

Table 3: Serum potassium of Patients with Decompensated Alcoholic Liver Disease

Serum potassium	Count (%)
<3.5 mEq/L	88 (48.89%)
3.5-5.0 mEq/L	80 (44.44%)
>5.0 mEq/L	12 (6.67%)
Total	180 (100.00%)

Table 4: Asparatate Transaminase (SGOT) of Patients with Decompensated Alcoholic Liver

Asparatate Transaminase (SGOT)	Count (%)
10–40 U/L	4 (2.22%)
>40 U/L	176 (97.78%)
Total	180 (100.00%)

Table 5: Alanine Transaminase (SGPT) of Patients with Decompensated Alcoholic Liver Disease

Alanine Transaminase (SGPT)	Count (%)
15–45 U/L	10 (5.56%)
>45 U/L	170 (94.44%)
Total	180 (100.00%)

Table 6: USG (Ultrasound Sonography) of Patients with Decompensated Alcoholic Liver Disease

USG (Ultrasound Sonography)	Count (%)
Altered Ecotexure, Mild Ascites	13 (7.22%)
Altered Liver Ecotexure	6 (3.33%)
Altered Liver Ecotexure, Mild Free Interbowel Fluids	7 (3.89%)
Liver Cirrhosis With Mild Ascites	6 (3.33%)
Liver Cirrhosis, Mod Ascites, Spleenomegaly	7 (3.89%)
Liver Cirrhosis, Portal Htn, Gross Ascites, Spleenomegaly	13 (7.22%)
Liver Cirrhosis, Portal Htn, Gross Ascites	7 (3.89%)
Liver Cirrhosis, Portal Htn, Mild/ Mod Ascites	12 (6.67%)
Liver Cirrhosis, Portal Htn, Mild Ascites	18 (10.00%)
Liver Cirrhosis, Portal Htn, Mild Ascites, Spleenomegaly	7 (3.89%)
Liver Cirrhosis, Portal Htn, Mod Ascites	7 (3.89%)
Liver Cirrhosis, Portal Htn, Mod Ascites, Spleenomegaly	77 (42.78%)
Total	180 (100.00%)

Table 7: MELD Score of Patients with Decompensated Alcoholic Liver Disease group

MELD Score	Count (%)
<17	2 (1.11%)
17 – 20	54 (30.00%)
21 – 22	36 (20.00%)
23 – 26	40 (22.22%)
27 – 31	35 (19.44%)
>=32	13 (7.22%)
Total	180 (100.00%)

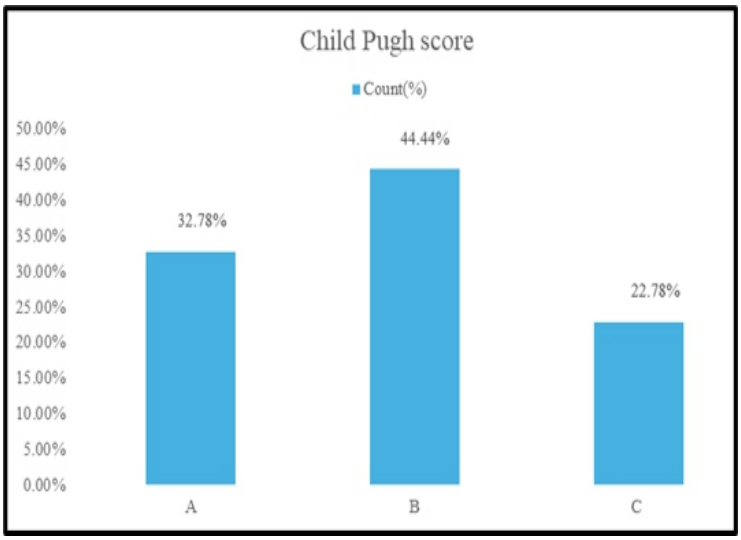

Figure 2: Child Pugh score of Patients with Decompensated Alcoholic Liver

Table 8: Cross-table for Serum sodium level Vs MELD Score of Patients with

	Serum Sodium (Count (%))			
MELD Score	**<135 mEq/L**	**135 - 145 mEq/L**	**>145 mEq/L**	**Total**
<17	0 (0.00%)	1 (0.56%)	1 (0.56%)	2 (1.11%)
17 - 20	2 (1.11%)	43 (23.89%)	9 (5.00%)	54 (30.00%)
21 - 22	11 (6.11%)	16 (8.89%)	9 (5.00%)	36 (20.00%)
23 - 26	17 (9.44%)	17 (9.44%)	6 (3.33%)	40 (22.22%)
27 - 31	25 (13.89%)	10 (5.56%)	0 (0.00%)	35 (19.44%)
>=32	7 (3.89%)	5 (2.78%)	1 (0.56%)	13 (7.22%)
Total	62 (34.44%)	92 (92.11%)	26 (14.44%)	180 (100.00%)

DISCUSSION

The findings of this study strongly support the prognostic and diagnostic significance of serum sodium levels in patients with decompensated alcoholic liver disease (DALD), providing valuable clinical insights consistent with existing literature. In our observational study of 180 male patients from a tertiary care hospital, we evaluated the relationships between serum sodium and disease severity using established scoring systems, including MELD and Child-Pugh classifications. The results highlight serum sodium as a reliable, cost-effective biomarker for risk stratification and prognosis in DALD, supporting its clinical utility for guiding patient management and intervention strategies [9].

Hyponatremia, defined as serum sodium <135 mEq/L, was observed in 34.44% of our study population, a prevalence comparable to prior reports by Channakeshava SP. 2019, who noted 56.7% hyponatremia in decompensated cirrhosis, and Raja MK, et. al; 2017, who reported 52% in cirrhotic patients. This finding underscores hyponatremia as a common electrolyte disturbance in DALD, particularly in alcohol-related cirrhosis. The slightly lower prevalence in our study may be attributed to the exclusion of patients with coexisting renal or cardiac conditions and the homogeneous male sample, in contrast to more heterogeneous populations in prior studies [10,11].

A significant correlation was observed between serum sodium levels and MELD scores. Specifically, 13.89% of patients with sodium <135 mEq/L had MELD scores between 27–31, and 9.44% were between 23–26. This trend confirms that lower sodium levels reflect worsening liver function and higher mortality risk. Our results align with Kumar VS. 2020, and Mobin AQ, et. al; 2022, who reported strong inverse relationships between serum sodium and both MELD and Child-Pugh scores, establishing sodium as an independent prognostic marker [12,13].

Similarly, Child-Pugh classification demonstrated a significant association with serum sodium levels. Patients in Class C (22.78%) exhibited the most severe sodium derangements, followed by Class B (44.44%). These results are supported by Singh N, et. al; 2022, who observed that 63.3% of CLD patients with serum sodium ≤135 mEq/L had worse Child-Pugh scores, reinforcing sodium's role as a surrogate marker for hepatic decompensation severity [14].

Complications associated with low serum sodium were also noted. Levels <135 mEq/L were significantly linked to hepatic encephalopathy, ascites, and hepatorenal syndrome, consistent with Shetty VN & Nayak UB. 2023, who observed higher complication rates in 23.7% of patients with sodium <130 mEq/L. Nareddy SR, et. al; 2020, reported associations with SBP and HRS, though not ascites or variceal bleeding; our study noted correlations with ascites, likely due to high prevalence of ultrasound-confirmed ascitic presentations [15,16].

Interestingly, 14.44% of patients had hypernatremia (>145 mEq/L), a relatively underreported finding in DCLD research. Most prior studies either excluded hypernatremic patients or did not document such cases, likely due to their rarity. Given the hemodynamic instability and increased mortality associated with hypernatremia in ICU patients, as reported by Kiaei BA, et. al; 2018, this observation warrants further investigation in the context of DALD [17].

The age distribution of hyponatremic patients revealed a predominance in the 36–50-year range, reflecting a working-age population critical to socioeconomic productivity. Mobin AQ, et. al; 2022, highlighted a significant burden of cirrhotic complications in younger adults and emphasizing the public health importance of early screening and intervention.

Biochemical parameters further supported the association between liver decompensation and sodium disturbances, with all patients exhibiting elevated bilirubin, SGOT, SGPT, and INR, 91.11% with INR >1.4, and 59.44% presenting with hypoalbuminemia, consistent with prior literature [13].

Statistical analyses confirmed the predictive value of hyponatremia, with 13.89% of patients having sodium <135 mEq/L and MELD ≥27, indicating high short-term mortality risk. These findings support earlier reports by Elkady MS, et. al; 2016 which emphasized that MELD-Na scoring improves mortality prediction and transplant prioritization. Lifestyle factors were notable, with 88.33% of patients not engaging in exercise, potentially contributing to sarcopenia, metabolic dysfunction, and poor resilience to complications. Additionally, a substantial portion of the cohort were farmers (27.78%) and laborers (21.67%), suggesting occupational stress and limited healthcare access as contributors to delayed diagnosis and worse outcomes [3].

These results highlight the importance of early detection and serum sodium monitoring in DALD management, aligning with global recommendations for MELD-Na utilization. Therapeutic strategies, including vaptans for dilutional hyponatremia, cautious sodium supplementation, and fluid restriction, have shown promise but remain limited by cost and availability in resource-constrained settings. Incorporating such interventions where feasible could improve patient outcomes, emphasizing the clinical utility of serum sodium as a prognostic and therapeutic guide [18,19].

CONCLUSION

Serum sodium is not just a biochemical measurement but a crucial prognostic and therapeutic marker in decompensated alcoholic liver disease, reflecting systemic physiological disturbances associated with hepatic decompensation and serving as an early indicator of potential complications. Regular monitoring of serum sodium in clinical practice can enhance patient outcomes by enabling timely interventions, minimizing complications, and optimizing resource use in tertiary care settings. Ongoing research and policy efforts are essential to standardize sodium assessment protocols and incorporate them into universal liver disease management guidelines for improved care.

ABBREVIATIONS

DALD: Decompensated Alcoholic Liver Disease
MELD: Model for End-Stage Liver Disease
MELD-Na: MELD with Serum Sodium
SPSS: Statistical Package for the Social Sciences
mEq/L: Milliequivalents per Liter

LIMITATIONS & FUTURE PERSPECTIVES

The study was limited by its single-centre design, relatively small sample size, and short duration, which may restrict generalizability. Future research could focus on multicenter studies with larger cohorts to validate findings, evaluate long-term outcomes, and explore innovative diagnostic and management strategies for appendicular perforation, improving patient prognosis and reducing complications.

CLINICAL SIGNIFICANCE

Timely detection and management of acute appendicitis are crucial to prevent perforation, reducing morbidity and mortality. The study identifies high-risk groups, such as males and individuals at age extremes, highlighting the need for targeted preventive strategies and clinical vigilance. Delayed presentation significantly increases perforation risk, underscoring the importance of early healthcare access and dawareness campaigns. Postoperative complications, including surgical site infections and prolonged ileus, emphasize the need for thorough preoperative risk assessment and tailored postoperative care. Recognizing the distal third of the appendix as the most common perforation site aids surgeons in effective intraoperative planning and management.

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION
All necessary consent for publication was obtained by authors.

DATA AVAILABILITY
All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)
The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

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