RESEARCH PAPER

Glycemic–Electrolyte Interplay in Alcohol-Associated Liver Disease: A Severity-Stratified Retrospective Analysis Using Child–Pugh and Meld Scores in an Indian Inpatient Cohort

Marka Sheshi *, Konga Jyoshna , Ponnala Pallavi , Chikkula Pavan Kumar , Macharla Karthik Teja , Sunayana Velede

Department Of Pharmacy Practice, St. Peter's Institute of Pharmaceutical Sciences, Affiliated to Kakatiya University, 506001, Hanamkonda, Telangana, India.

ABSTRACT

Background:
Alcoholic liver disease (ALD) is a leading cause of chronic liver–related morbidity and mortality and is commonly associated with metabolic disturbances that adversely affect patient outcomes. Alterations in glucose metabolism and electrolyte balance are frequently encountered in patients with ALD and contribute to disease progression and the development of serious complications. This study was undertaken to evaluate the relationship between blood glucose levels and serum electrolyte abnormalities in patients with alcoholic liver disease.

Methods:
A retrospective observational study was conducted in a tertiary care hospital in Telangana, India, over a six-month period. Medical records of 400 patients diagnosed with alcoholic liver disease were reviewed. Demographic details, disease severity, random blood glucose levels and serum electrolyte parameters including sodium, potassium and chloride were analyzed before and after inpatient management. Statistical analysis was performed using SPSS version 26.0 and a p-value <0.05 was considered statistically significant.

Results:
The study revealed significantly elevated baseline glucose levels along with deranged electrolyte values in the majority of patients. Inpatient management resulted in marked improvement in glycemic control and correction of electrolyte abnormalities. Baseline GRBS showed a significant inverse correlation with serum sodium ($r = -0.38$, $p = 0.006$), potassium ($r = -0.34$, $p = 0.024$), and chloride ($r = -0.31$, $p = 0.032$), indicating a close metabolic interaction between these parameters. More pronounced metabolic derangements were noted in patients with moderate to severe liver disease based on Child-Pugh and MELD scores.

Conclusion:
These findings highlight a clinically meaningful metabolic interaction between hyperglycemia and electrolyte imbalance in alcohol-associated liver disease. The observed inverse correlations, particularly in moderate-to-severe disease categories, suggest that metabolic monitoring should extend beyond isolated parameter correction toward integrated biochemical assessment. Early recognition and targeted correction of these disturbances may contribute to improved inpatient stabilization and potentially better short-term outcomes.

Keywords: Blood glucose, Electrolytes, Metabolic abnormalities, Child Pugh, MELD score

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INTRODUCTION

Alcohol-related liver injury remains a significant contributor to chronic liver disease burden globally, particularly in regions with high alcohol consumption patterns. The clinical spectrum ranges from reversible steatosis to progressive fibrosis, cirrhosis, and hepatic failure. Beyond structural hepatic damage, metabolic instability is increasingly recognized as a central component influencing disease progression and short-term prognosis [1-2]. Globally, alcohol use contributes to

Corresponding Author: Marka Sheshi, markasheshi@gmail.com

approximately three million deaths annually, and alcoholic liver disease accounts for a substantial proportion of these deaths [3]. The estimated global prevalence of ALD ranges between 4–6% in the adult population, while in India, ALD constitutes nearly one-third to half of chronic liver disease admissions in tertiary care hospitals, predominantly affecting middle-aged males [1].

In addition to structural hepatic injury, alcoholic liver disease is associated with profound metabolic derangements that significantly influence disease severity, complications and prognosis [4]. Among these, disturbances in glucose metabolism and electrolyte balance are particularly important [5]. The liver serves as a metabolic regulator of glucose turnover through glycogen storage, gluconeogenesis, and insulin clearance mechanisms [6]. Chronic alcohol exposure disrupts these tightly regulated pathways by inducing hepatic insulin resistance and altering pancreatic β-cell responsiveness [7]. Consequently, stress-induced hyperglycemia and impaired glucose tolerance are frequently observed in patients with advanced liver dysfunction [8]. Persistent hyperglycemia further aggravates oxidative stress and inflammatory responses, thereby accelerating hepatocellular injury and disease progression [8].

Electrolyte abnormalities are also common in alcoholic liver disease. Portal hypertension, impaired renal perfusion, secondary hyperaldosteronism, diuretic therapy, vomiting, diarrhea and malnutrition predispose these patients to dilutional hyponatremia, hypokalemia or hyperkalemia and hypochloremia [9]. These disturbances are clinically significant because they contribute to the development of hepatic encephalopathy, cardiac arrhythmias, neuromuscular weakness, renal dysfunction and increased mortality [10].

Glucose metabolism and electrolyte balance are closely interrelated. Hyperglycemia induces osmotic diuresis, resulting in increased urinary loss of sodium, potassium and chloride [11]. Conversely, electrolyte imbalances impair insulin sensitivity and cellular glucose uptake, creating a bidirectional metabolic interaction [11]. In patients with alcoholic liver disease, this vicious cycle is intensified by compromised hepatic function, chronic inflammation, malnutrition and altered hormonal regulation, leading to worsening metabolic instability [5, 9].

Laboratory investigations in ALD commonly reveal hyperglycemia, hyponatremia, hypokalemia or hyperkalemia and hypochloremia along with elevated liver enzymes, hyperbilirubinemia, prolonged prothrombin time and hypoalbuminemia [12]. These biochemical parameters are important not only for diagnosis but also for assessing disease severity and prognosis [12]. Diagnosis of alcoholic liver disease is based on clinical history of alcohol consumption, biochemical evaluation, ultrasonographic liver grading and severity scoring systems such as Child-Pugh and Model for End-Stage Liver Disease (MELD) scores [13]. The Child-Pugh scoring system is a widely used clinical tool for assessing liver disease severity and prognosis based on five parameters: serum bilirubin, serum albumin, prothrombin time (or INR), ascites and hepatic encephalopathy [14]. Patients are classified into Class A, B or C, corresponding to mild, moderate and severe liver dysfunction, respectively [14, 15]. The MELD score is an objective prognostic model calculated using serum bilirubin, creatinine and INR values and is primarily used to estimate short-term mortality risk and prioritize patients for liver transplantation [14, 16]. Together, these scoring systems provide standardized and reliable assessment of hepatic functional reserve and disease severity in patients with alcoholic liver disease [13, 14, 16].

Despite the high prevalence and clinical relevance of these metabolic abnormalities, limited data are available regarding the combined evaluation of glucose levels and electrolyte disturbances in patients with alcoholic liver disease, particularly in the inpatient setting [17]. Understanding their interrelationship and response to treatment is essential for early identification of high-risk patients, optimization of inpatient management and prevention of disease-related complications [18,26].

Although both hyperglycemia and electrolyte disturbances are individually well documented in chronic liver disease, few studies have explored their simultaneous interaction within a severity-stratified framework using validated prognostic scoring systems. In the Indian inpatient setting, where late presentations are common, such integrated evaluation remains limited. This study was therefore designed to examine the relationship between glycemic status and electrolyte imbalance in alcohol-associated liver disease, with correlation across Child–Pugh and MELD classifications.

MATERIALS AND METHODS

This single-center retrospective analytical study reviewed medical records of patients admitted with alcohol-associated liver disease over a six-month period (02 June 2025 to 08 November 2025) at a tertiary care hospital in Telangana, India. A total of 400 patients diagnosed with alcoholic liver disease (ALD) and admitted to inpatient departments during the study period were included. Inclusion criteria included adult patients (>18 years) diagnosed with alcohol-associated liver disease and having complete biochemical records. Patients with pre-existing type 1 diabetes mellitus, chronic kidney disease, or incomplete laboratory data were excluded. Patient data were obtained from medical record files available in the hospital records department.

Demographic variables, associated comorbidities, hospitalization duration, ultrasonographic grading, and laboratory parameters were extracted from institutional medical records. Disease severity was categorized using Child–Pugh and MELD scoring systems as documented in patient files.

Laboratory parameters recorded at admission and discharge included random blood glucose (GRBS), serum sodium, potassium, and chloride levels. These variables were analyzed to evaluate the relationship between glucose levels and electrolyte disturbances in patients with alcoholic liver disease.

Descriptive statistical methods were used for baseline analysis, including measures of central tendency and dispersion. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage. The paired Student's t-test was applied to compare pre-treatment and post-treatment biochemical parameters. Pearson's correlation coefficient was used to determine the relationship between blood glucose levels and serum electrolyte concentrations. All statistical analyses were performed using SPSS software version 26.0, and a two-tailed p value of < 0.05 was considered statistically significant.

RESULTS

A total of 400 patients diagnosed with alcoholic liver disease (ALD) were included in the study. Demographic characteristics, ultrasonographic liver grading, duration of hospitalization, glycemic status and serum electrolyte parameters were analyzed. Minor variability in electrolyte correction was observed depending on associated comorbidities and duration of hospitalization.

Table 1. Age group–wise distribution of GRBS before and after treatment

Age group (years)	n	GRBS Before (Mean ± SD)	GRBS After (Mean ± SD)
18–30	53	199.6 ± 33.4	132.8 ± 26.3
31–40	95	206.9 ± 34.7	136.1 ± 27.8
41–50	102	213.2 ± 35.9	139.4 ± 28.6
51–60	87	218.1 ± 37.5	142.3 ± 29.1
≥60	63	221.7 ± 39.8	145.6 ± 30.4

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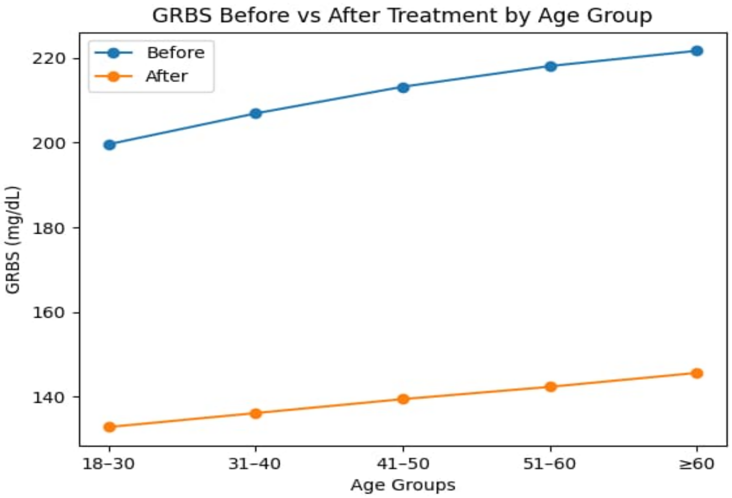


Figure 1 — GRBS Before vs After by Age

Table 1 and Fig 1 shows the age-wise distribution of GRBS before and after treatment. Baseline GRBS values increased progressively with age. A statistically significant reduction in GRBS was observed after treatment across all age groups ($p < 0.05$).

Table 2. Baseline demographic profile, Liver grading and hospital stay (N = 400)

Category	Parameter	n (%)
Gender	Male	332 (83.0)
	Female	68 (17.0)
Diagnosis	ALD only	164 (41.0)
	ALD with sepsis	74 (18.5)
	ALD with pancreatitis	62 (15.5)
	ALD with UTI	48 (12.0)
	ALD with cellulitis	32 (8.0)
	ALD with ACLF	20 (5.0)
USG - Liver Grading	Grade I (mild)	136 (34.0)
	Grade II (moderate)	158 (39.5)
	Grade III (severe/cirrhotic)	106 (26.5)
Hospital Duration (Days)	1–5	118 (29.5)
	6–10	164 (41.0)
	>10	118 (29.5)

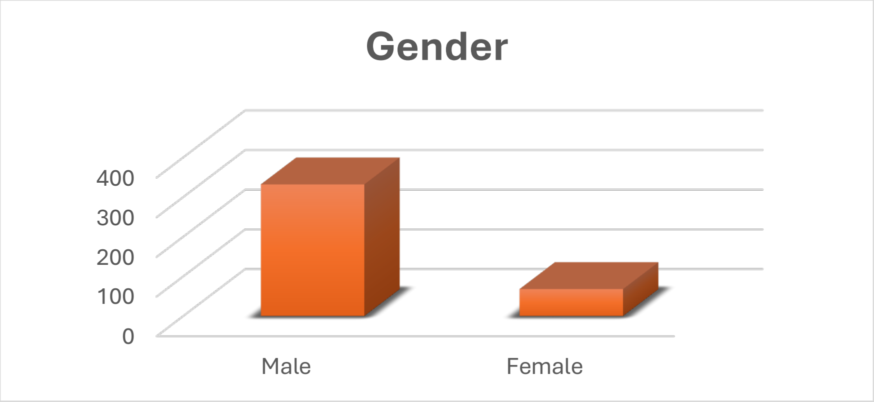


Figure 2 Gender (N = 400)

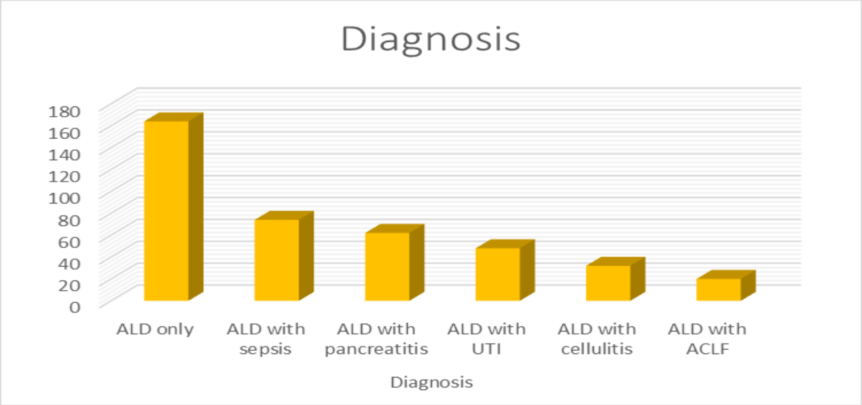


Figure 3 Baseline demographic profile (N = 400)

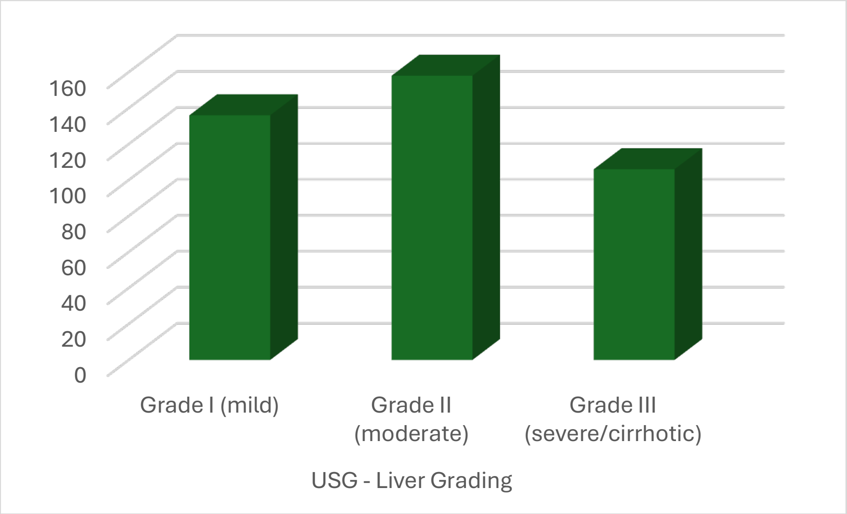


Figure 4 Baseline Liver grading (N = 400)

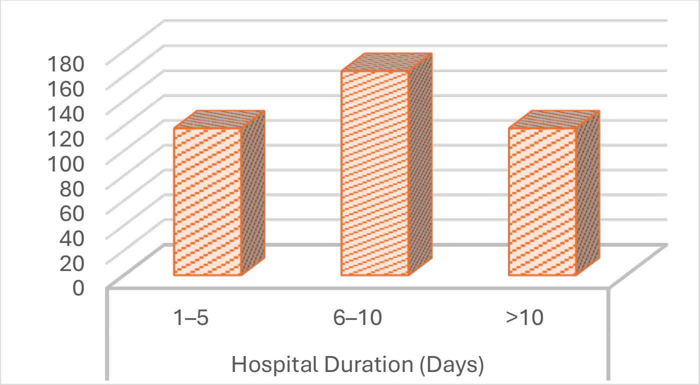


Figure 5 Baseline hospital stay (N = 400)

Table 2 and Fig 2,3,4,5 presents demographic and clinical characteristics of the study population. The majority were male (83%). Most patients had moderate ultrasonographic grading (39.5%), and 41% required hospitalization for 6–10 days.

Table 3. Comparison of biochemical parameters before and after treatment

Parameter	Before (Mean ± SD)	After (Mean ± SD)	Mean Change (p-value)
GRBS (mg/dL)	209.8 ± 33.8	136.9 ± 27.6	−72.9 ($p = 0.012$)
Sodium (mEq/L)	128.3 ± 5.7	139.1 ± 4.3	+10.8 ($p = 0.021$)
Potassium (mEq/L)	3.19 ± 0.58	4.32 ± 0.44	+1.13 ($p = 0.034$)
Chloride (mEq/L)	94.9 ± 7.1	102.5 ± 6.6	+7.6 ($p = 0.019$)

The magnitude of sodium correction was more prominent among patients with moderate and severe ultrasonographic grading, suggesting that electrolyte normalization paralleled overall clinical stabilization.

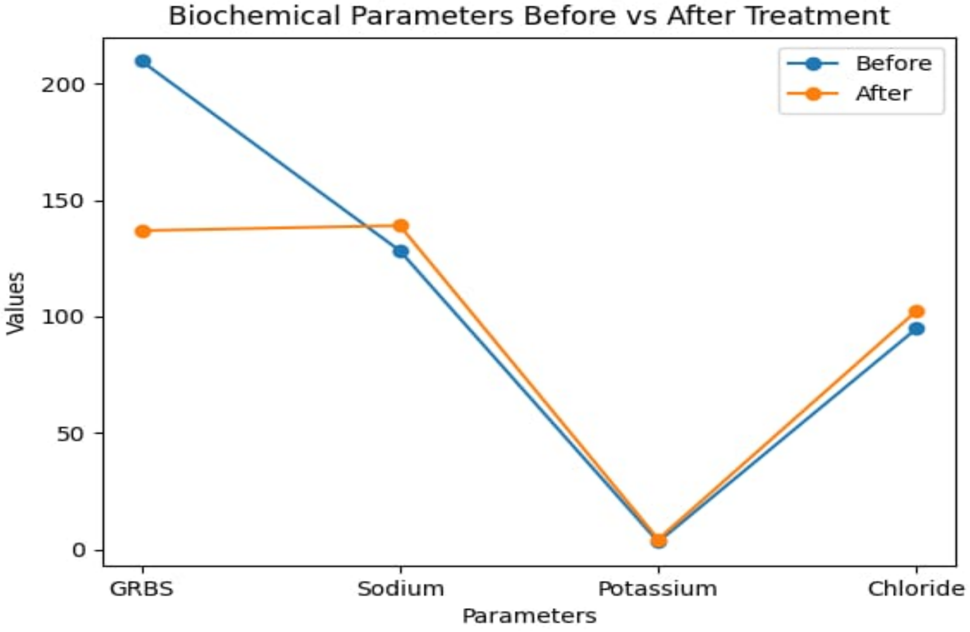


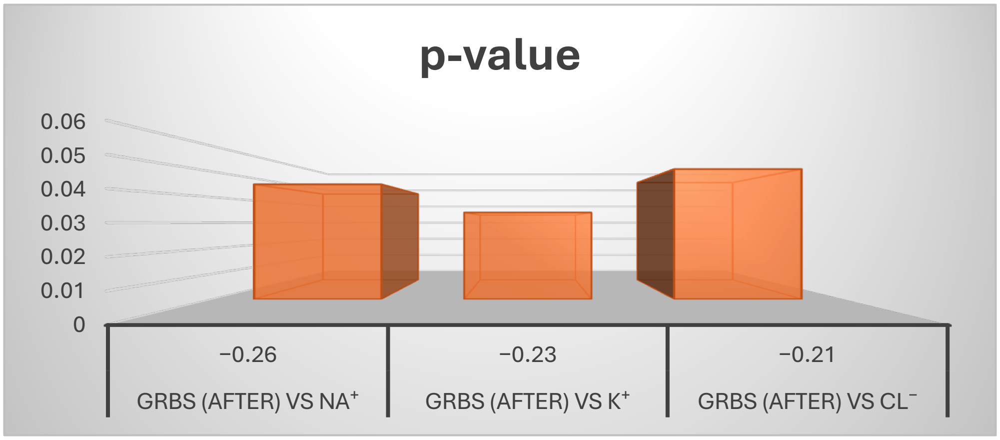
Figure 6 — Biochemical Parameters Before vs After

Table 3 and Fig 3 compares biochemical parameters before and after treatment. Significant reductions were observed in GRBS, while sodium, potassium, and chloride levels showed significant improvement following inpatient management ($p < 0.05$).

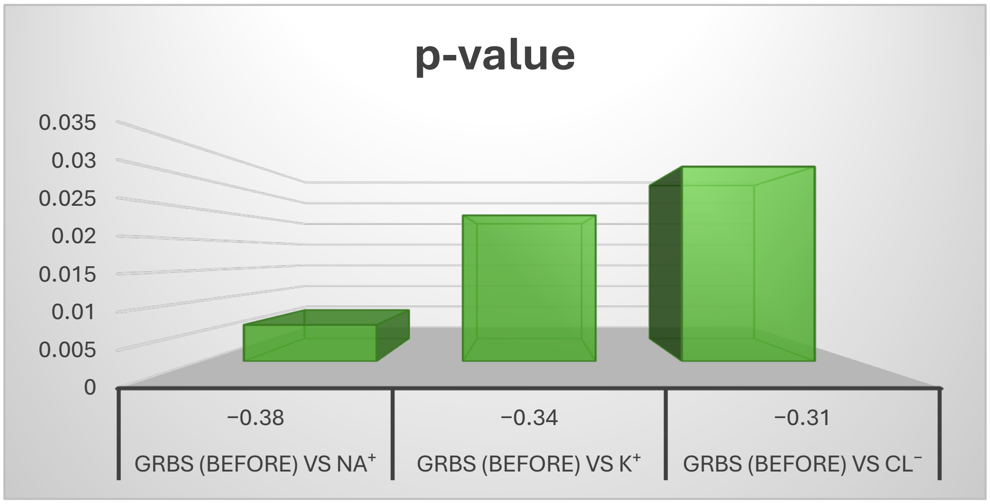
Table 4. Correlation between glucose levels and serum electrolytes

Comparison	r	p-value
GRBS (Before) vs Na^+	−0.38	0.006
GRBS (Before) vs K^+	−0.34	0.024
GRBS (Before) vs Cl^-	−0.31	0.032
GRBS (After) vs Na^+	−0.26	0.045
GRBS (After) vs K^+	−0.23	0.034
GRBS (After) vs Cl^-	−0.21	0.051

Table 4 shows Pearson's correlation between GRBS and serum electrolytes. A statistically significant inverse correlation was observed between baseline GRBS and sodium (r = −0.38, p = 0.006), potassium (r = −0.34, p = 0.024), and chloride (r = −0.31, p = 0.032).



Before



After

Figures 7 — Correlation Between Glucose and Electrolytes.

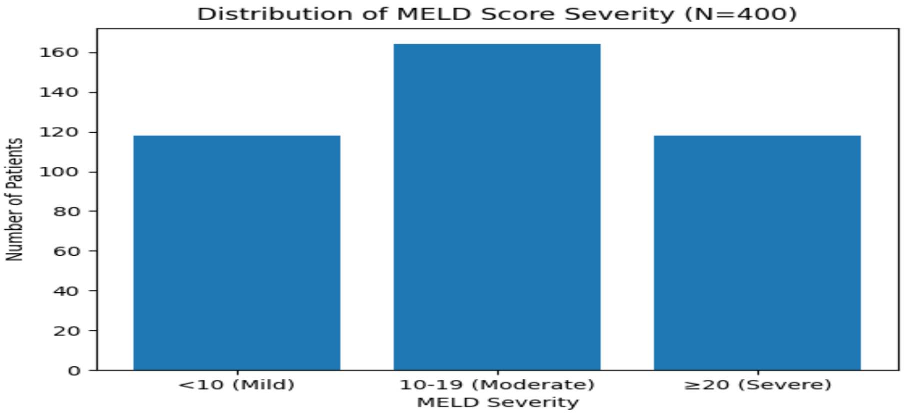


Figure 8. Child-Pugh Score Distribution

Figure 5 shows the distribution of patients according to Child–Pugh classification. The majority of patients were categorized under Class B, followed by Class A and Class C.

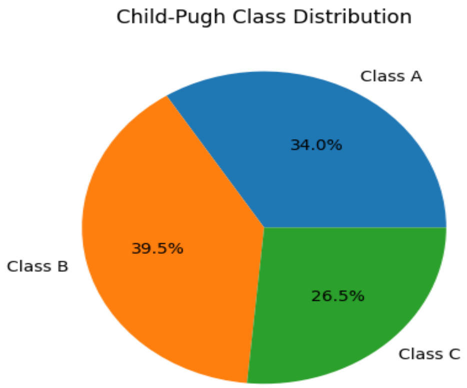


Figure 9. MELD Score Severity Distribution

Figure 6 illustrates the distribution of patients based on MELD score severity categories, including mild, moderate, and severe groups.

DISCUSSION

The present analysis underscores the metabolic vulnerability observed in patients with alcohol-associated liver disease, particularly those presenting with moderate to advanced severity [19]. Our findings demonstrate that hyperglycemia and electrolyte disturbances are not isolated abnormalities but appear to coexist within a shared pathophysiological framework [20, 6, 11]. Elevated random blood glucose levels were observed in patients across all age groups, with a tendency toward higher values in older individuals [21,25]. This pattern can be explained by the metabolic effects of chronic alcohol exposure, which impairs insulin sensitivity and alters normal glucose regulation. Persistent hyperglycemia may worsen liver injury by increasing oxidative stress and inflammatory responses, thereby accelerating disease progression. The reduction in glucose levels following inpatient care suggests that timely medical management can significantly improve glycemic control in patients with alcoholic liver disease [8, 21].Electrolyte disturbances were also common among the study population. Lower levels of sodium and chloride, hypokalemia and hyperkalemia were frequently observed, indicating the presence of dilutional hyponatremia, hypo & hyperkalemia and hypochloremia [11]. Hyponatremia observed in this cohort likely reflects neurohormonal activation secondary to portal hypertension, including increased antidiuretic hormone secretion and renin–angiotensin–aldosterone system activation. Potassium imbalance may be influenced by diuretic exposure, altered renal perfusion, and intracellular shifts driven by hyperglycemia. Such disturbances are clinically significant, given their association with encephalopathy, arrhythmias, and renal dysfunction. [22].The improvement in electrolyte levels after inpatient management emphasizes the importance of regular monitoring and early correction of these abnormalities. Maintaining electrolyte balance plays a key role in stabilizing patients and preventing life-threatening complications [9].The inverse correlations identified between baseline glucose levels and serum sodium, potassium, and chloride suggest a bidirectional metabolic interaction. Hyperglycemia-induced osmotic diuresis may accelerate electrolyte loss, while concurrent electrolyte imbalance may further impair insulin-mediated glucose utilization [23,24]. This interaction potentially amplifies metabolic instability in advanced liver disease. This bidirectional interaction may contribute to worsening metabolic instability in alcoholic liver disease [24]. Furthermore, a considerable proportion of patients in this study were categorized under moderate to severe disease based on Child-Pugh and MELD scores, indicating that many patients present at advanced stages of liver disease [13, 14, 16]. More severe disease was associated with greater metabolic derangements, highlighting the need for early diagnosis and continuous metabolic monitoring [11, 12].Overall, the findings of this study underline the clinical importance of routinely evaluating glucose and electrolyte levels in patients with alcoholic liver disease. Early detection and prompt management of metabolic abnormalities may reduce complications, improve clinical stability and enhance patient outcomes [18]. Several limitations warrant consideration. The retrospective design limits causal inference, and single-center data may restrict generalizability. Additionally, longitudinal follow-up beyond discharge was not available, preventing assessment of long-term outcome implications. Prospective multicenter validation would strengthen these findings.

CONCLUSION

In this cohort of patients with alcohol-associated liver disease, significant hyperglycemia and electrolyte derangements were frequently observed and demonstrated meaningful inverse correlations. The integration of glycemic and electrolyte assessment within severity stratification frameworks such as Child–Pugh and MELD may offer improved insight into metabolic instability during hospitalization. Focused correction of these biochemical abnormalities may enhance short-term stabilization, particularly in moderate-to-severe disease categories.

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