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FREQUENCY OF HYPOGLYCEMIA IN PATIENTS WITH LIVER CIRRHOSIS

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Abstract

Background: *Impaired glucose metabolism due to alterations in glycogen storage, gluconeogenesis, and hormone clearance leads to hypoglycaemia in patients with liver cirrhosis. Although it is clinically significant, local estimates for the prevalence and predictors of hypoglycaemia in patients with liver cirrhosis are underreported in Low- and Middle-Income Countries (LMICs).*

Objective: *To assess the prevalence and severity, as well as investigate clinical predictors of hypoglycaemia in adult non-diabetic patients with liver cirrhosis.*

Method: *This cross-sectional study was conducted at Sharif Medical City Hospital, Lahore, Pakistan. A total of 131 patients aged 18-60 with a diagnosis of cirrhosis were selected. Clinical demographic data was recorded, and blood glucose concentrations were determined using standardized point-of-care testing equipment. Prevalence of hypoglycaemia was classified into mild, moderate, or severe. Chi-square statistics or t-tests, and regression analyses were utilized to identify clinical predictors.*

Results: *A total of 131 patients were surveyed, where 72(55%) were male, 59(45%) were female, and the mean age was 38.34±12.42 years. Hypoglycaemia was detected in 93 patients (71%), with mild being the most prevalent (45%). Significant differences were seen in the duration of cirrhosis ($p = .015$) and in the blood glucose concentrations for hypoglycaemia ($p < .001$), whereas gender, comorbidities, and Child–Pugh class were not significantly associated with hypoglycaemia.*

Conclusion: *Hypoglycemia is highly reported in patients with cirrhosis, especially in the situations of significant amounts of cirrhosis. Regular glucose levels should be taken into consideration in treatment plans when providing care to patients with cirrhosis in the inpatient setting to assist in earlier recognition and improved outcomes.*

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INTRODUCTION

The liver has an integral role in the homeostasis of systemic glucose levels: it stores glycogen, carries out the process of gluconeogenesis, and clears the circulating insulin and additional hormones that are related to glucose levels (Shen et al., 2025). Once chronic liver injury progresses to cirrhosis, there is a breakdown of the metabolic function of the liver, leading to depletion of glycogen stores, abnormalities of gluconeogenesis, impaired insulin clearance, and often malnutrition, leading to complications of hypoglycemia in patients with cirrhosis in ways that hypoglycemia occurs in the absence of diabetes or the use of a hypoglycemic agent. Furthermore, episodes of hypoglycemia are associated with subsequent worsening and potentially, worse outcomes in the short-term in patients with cirrhosis (Chung, Promrat, and Wands, 2020; Hung et al., 2021).

The reported prevalence estimates of hypoglycemia have varied across settings and study design that reflect the difference in inclusion/exclusion criteria for patient selection, study designs, measures and definitions of hypoglycemia (Yen et al., 2023). Several single-center series from South Asia and across the globe also documented particularly high prevalence rates: one Pakistan study reported a prevalence of nearly 58% among hospitalized patients with cirrhosis, while similarly, reports from elsewhere reported prevalence variations of generally about 48% to 51%. All studies typically did not include patients who had a diagnosis of diabetes so as not to impede the measurement of liver-related dysregulation of glucose levels (Khan et al., 2024; Majeed et al., 2017; Singh et al., 2015).

Different investigations have also underscored the clinical importance of hypoglycemia in liver disease. Analyses of national inpatient datasets and matched cohort studies reported that hypoglycemia occurs relatively frequently among hospitalized cirrhotic patients and is associated with increased healthcare utilization, intensive care admission, and mortality compared with cirrhotic patients without hypoglycemia (Badshah et al., 2018). In addition, studies of acute-on-chronic liver failure and cirrhosis with infectious complications highlighted that hypoglycemia often marks severe illness and poorer prognosis in these contexts (Govindarajan et al., 2025).

Mechanistically, hypoglycemia in cirrhosis arises from a combination of factors: depleted hepatic glycogen due to chronic malnutrition or fasting, failure of gluconeogenesis because of reduced hepatocyte mass or impaired substrate availability, impaired hormonal counter-regulatory responses, and, in some cases, sepsis or adrenal dysfunction (Hung et al., 2021; Lee et al., 2022). Iatrogenic contributors (glucose-lowering agents) were deliberately excluded in many prior studies to isolate liver-specific mechanisms; this methodological choice also guided the present work's exclusion criteria. Understanding the relative contribution of these mechanisms in local patient populations is important for risk stratification and for developing monitoring and prevention strategies (Yen et al., 2023).

Despite recognition of the problem, high-quality prevalence data remain limited in many low- and middle-income settings where advanced liver disease is common and routine glucose monitoring may be inconsistent. Given the potential for hypoglycemia to precipitate encephalopathy, hemodynamic instability, and increased mortality, accurate estimates of its frequency and of clinical predictors among non-diabetic cirrhotic patients are needed to inform monitoring protocols and early interventions. Accordingly, this study aimed to determine the prevalence of hypoglycemia and its severity categories among adults with liver cirrhosis admitted to a tertiary hospital in Lahore, and to evaluate clinical factors associated with hypoglycemia in this population.

Methodology

This cross-sectional study aimed to investigate the prevalence of hypoglycemia among patients with liver cirrhosis. This study was conducted between November 2024 and April 2025, in the Medical Ward and the Emergency Department of Sharif Medical City Hospital, Lahore, after approval from the institutional ethical review board. The methods and results are reported in accordance with the STROBE guidelines for reporting observational studies to allow efficiency and reproducibility. Sample size was determined based on WHO formula for estimating a single population proportion, assuming a confidence level of 95%, a proportion of hypoglycemia at 58%, and margin of error as 5%. Based on the above

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parameters, a calculated sample size of 131 patients was determined. Non probability consecutive sampling was used to transform the sample, inviting all patients meeting eligibility criteria who were noted during the study period until the necessary sample was obtained. Patients aged 18 to 60 years and with a clinical, radiological or histological diagnosis of liver cirrhosis were included in the study. This population also included patients diagnosed with complications related to cirrhosis, such as ascites, hepatic encephalopathy, or hepatocellular carcinoma. Patients were excluded from the study if they had a diagnosis of diabetes mellitus or impaired fasting glucose prior to liver disease, were treated with a medication that affects glucose metabolism (e.g. systemic corticosteroids, insulin, etc.), were pregnant, alcohol-dependent, or had advanced chronic kidney disease.

Upon obtaining written informed consent from each subject's legak guardian (or the subject directly if not encephalopathy) to participate, demographic details, clinical history, comorbidities, the etiology and severity of cirrhosis and a list of medications taken were captured on a pre-structured form. Cirrhosis was classified as mild, moderate or severe based on the Child Pugh classification and severity was also determined using the MELD score. Capillary blood glucose was determined with a point of care glucometer with ISO 15197:2013 accuracy standards. If there were symptoms of hypoglycemia, a repeat vein blood glucose was obtained (if able to do so). Hypoglycemia was classified as mild (≥55 & <70) mg/dL) moderate (≥35 & <55 mg/dL) or severe (less than <35 or required assistance due to neuroglycopenic symptoms). Hypoglycemia clinical symptoms and any need of clinical intervention (i.e. intravenous dextrose or glucagon) were also documented.

To ensure reliability of the data, all data collectors were trained on how to obtain consent, effectively administer glucometer readings, and accurately fill out case report forms. All statistical analysis was carried out using SPSS v.25.0. Continuous variables (age, disease duration, blood glucose) were summarized as means ±SD, while dichotomous variables (gender, Child-Pugh class, and presence of hypoglycemia) were summarized as frequencies (%). The overall prevalence of hypoglycemia and severities were reported with 95% CI. The association of hypoglycemia with categorical predictors was tested using Chi-square tests and continuous predictors were analyzed using independent-sample t-tests. Regression analysis was conducted to ascertain the independent predictors of hypoglycemia by reporting adjusted odds ratios and a 95% CI. Significance was established at $p \leq 0.05$ using two-tailed tests. The study adhered to ethical principles: informed consent was obtained, patient data was anonymized to maintain confidentiality, and patients with hypoglycemia were treated per hospital protocols as quickly as possible. Indicating that the participation in the study would not affect any patient's clinical care in anyway.

Results

The participants consisted of 131 patients with liver cirrhosis; 72 (55%) patients were male and 59 (45%) were female (Table 1). The mean age was 38.34±12.42, and the mean duration of liver cirrhosis was 35.98±32.44 months. The mean blood sugar was 80.61±48.11 mg/dL, and the mean Child-Pugh score was 4.24±2.34. In terms of Child-Pugh score, 39 (29.8%) patients were in class A, 45 (34.4%) were in class B, and 47 were in class C (35.9%). Hypoglycemia was identified in 93 patients (71%); most were classified as mild hypoglycemia, while a small percentage (9.2%) were in the severe category (Table 1). The most prevalent complication was ascites (23.5%), while hepatic encephalopathy (8.5%) and variceal bleed (5.9%) were found mildly less often. Interestingly, 35.2% of patients had no complications. The most common co-morbidities found were hypertension (22.4%) and thyroid disorders (14.9%).

Table 1: Baseline Characteristics of Patients with Liver Cirrhosis (N = 131)

Variable	Category	n (%)	Mean
Gender	Female	59 (45.0)	

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	Male	72 (55.0)	
Age (years)			38.34±12.42
Duration of cirrhosis (months)			35.98±32.44
Blood sugar (mg/dL)			**80.61±48.11**
Child–Pugh score			4.24±2.34
Child–Pugh class	A	39 (29.8)	
	B	45 (34.4)	
	C	47 (35.9)	
Hypoglycemia	Absent	38 (29.0)	
	Present	93 (71.0)	
Severity of hypoglycemia	None	38 (29.0)	
	Mild	59 (45.0)	
	Moderate	22 (16.8)	
	Severe	12 (9.2)	

Figure 1: Age distribution of Cirrhotic patients

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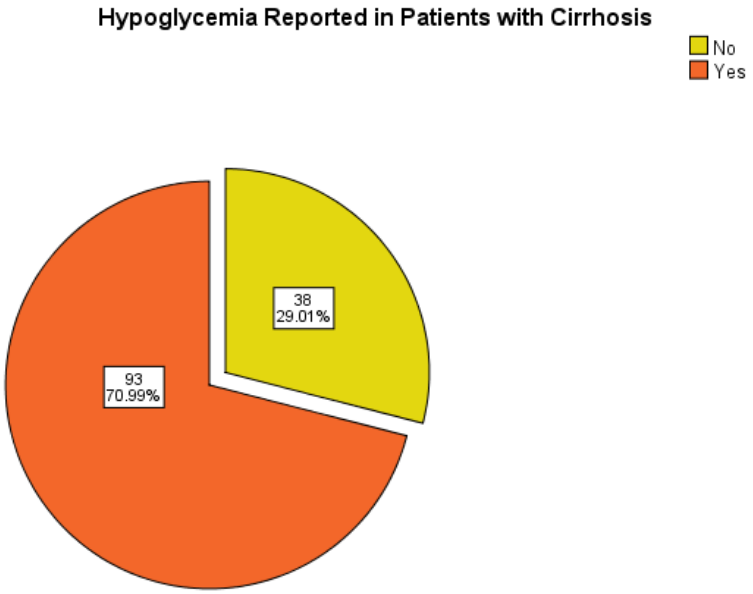

Figure 2: Prevalence of Hypoglycemia in Patients with Cirrhosis

Hypoglycemia was more frequent among males (72.2%) compared with females (69.5%) with p = .723. Across Child–Pugh classes, hypoglycemia occurred in 71.8% of Class A patients, 62.2% in Class B, and 78.7% in Class C, with no significant association identified (p = .217). When analyzed by the type of blood sugar measurement, hypoglycemia was observed in 65.9% of patients tested during fasting and 73.3% of those tested with random glucose levels, again showing no meaningful difference (p = .382). Similarly, the prevalence of hypoglycemia did not differ significantly across comorbid conditions, including hypertension (60.0%), ischemic heart disease (73.7%), no comorbidity (71.2%), and thyroid disorder (81.0%) (p = .467) (Table 2).

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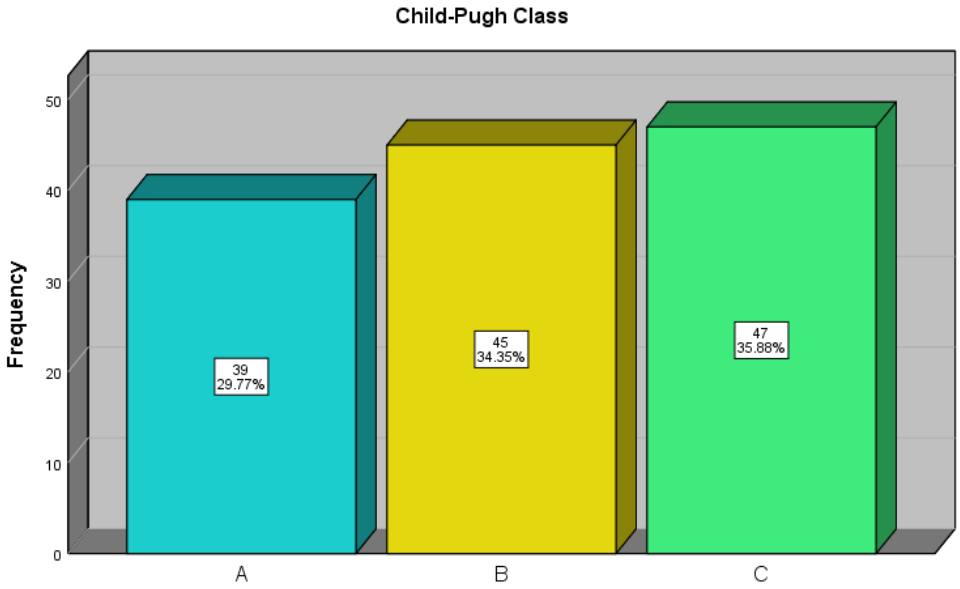

Figure 3: Frequency of Child-Pugh class in Cirrhotic Patients

Table 2: Associations Between Clinical and Demographic Variables and Hypoglycemia in Patients with Liver Cirrhosis (N = 131)

Variable	Category	Hypoglycemia Absent (n, %)	Hypoglycemia Present (n, %)	Total	*p*-value
Gender	Female	18 (30.5%)	41 (69.5%)	59	
	Male	20 (27.8%)	52 (72.2%)	72	.723
Child–Pugh Class	A	11 (28.2%)	28 (71.8%)	39	
	B	17 (37.8%)	28 (62.2%)	45	
	C	10 (21.3%)	37 (78.7%)	47	.217
Blood Sugar Type	Fasting	14 (34.1%)	27 (65.9%)	41	
	Random	24 (26.7%)	66 (73.3%)	90	.382
Comorbid Condition	Hypertension	10 (40.0%)	15 (60.0%)	25	
	Ischemic heart disease	5 (26.3%)	14 (73.7%)	19	
	None	19 (28.8%)	47 (71.2%)	66	
	Thyroid disorder	4 (19.0%)	17 (81.0%)	21	.467
Total		38 (29.0%)	93 (71.0%)	131	

Independent sample t-tests showed significant differences in the duration of cirrhosis and blood sugar levels between patients with and without hypoglycemia. Patients without hypoglycemia had a longer mean duration of cirrhosis (46.71 ± 36.75

months) compared to those with hypoglycemia (31.59 ± 29.61 months; p = .015). Hypoglycemic patients

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demonstrated significantly lower values (54.14 ± 15.27mg/dL) compared to non-hypoglycemic patients (145.39 ± 38.48mg/dL; p < .001). However, no significant difference was found in Child–Pugh scores between hypoglycemic and non-hypoglycemic patients (p = .386) (Table 3).

Table 3: Comparison of Duration of Cirrhosis, Blood Sugar Levels, and Child–Pugh Score by Hypoglycemia Status (N = 131)

Variable	Hypoglycemia Absent (M ± SD)	Hypoglycemia Present (M ± SD)	*t*	*p*-value
Duration of cirrhosis (months)	46.71 ± 36.75	31.59 ± 29.61	2.467	.015
Blood sugar (mg/dL)	145.39 ± 38.48	54.14 ± 15.27	19.494	< .001
Child–Pugh score	3.95 ± 2.20	4.35 ± 2.39	–0.904	.386

Multiple linear regression analysis was conducted to identify predictors of hypoglycemia (Table 4). The model explained 7.15% of the variance in hypoglycemia status (R^2 = .0715, p = .000).

Table 4: Multiple Linear Regression Predicting Hypoglycemia Status in Patients with Liver Cirrhosis (N = 131)

Variable	B	SE	β	t	p	R^2	p-value
Constant	2.357	.102		23.016	.000	.0715	.000
Age	-.001	.002	-.038	-.841	.402		
Gender	.041	.041	.045	1.011	.314		
Cirrhosis duration	.000	.001	-.024	-.511	.610		
CPS	.002	.009	.010	.222	.824		
BSR level	-.008	.000	-.858	-18.601	.000		

Discussion

Liver cirrhosis produces profound metabolic disturbances that impair the organ's capacity to maintain glucose homeostasis, principally because of reduced glycogen stores, impaired gluconeogenesis, and altered hormone clearance; these pathophysiologic changes make hypoglycemia a recognized complication in cirrhosis with important prognostic implications (Pfortmueller & Lindner, 2014).

In this study, the overall frequency of hypoglycemia was 71% among patients with liver cirrhosis. This observed prevalence is higher than many prior single-center reports but is within the broad range reported in the literature; for example, Tanveer and colleagues reported a prevalence near 58% in a Pakistani cohort, Singh et al. reported 67% in another regional series, and Nouel et al. reported approximately 50% in their hospitalized cirrhotic patients, indicating substantial heterogeneity across cohorts (Majeed et al., 2017; Singh et al., 2015; Hung et al., 2021).

More contemporary and larger observational analyses likewise documented frequent hypoglycemic events among hospitalized cirrhotic patients, albeit with variable point estimates. Khan et al. (2024) found hypoglycemia in nearly half of their cirrhotic cohort undergoing endoscopy, while Hung et al. (2021) reported frequent hypoglycemia among hospitalized patients and highlighted its association with worse

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short-term outcomes (Khan et al., 2024; Hung et al., 2021). These studies corroborate that hypoglycemia is common in inpatient cirrhosis and confirm that prevalence estimates are sensitive to patient selection (eg, emergency vs. elective admissions), measurement timing (fasting vs. random glucose), and hypoglycemia thresholds used.

Our prevalence was higher than several reports from higher-resource settings that used strict laboratory confirmation or excluded patients with acute infections, and this discrepancy may reflect differences in case-mix: a sizable proportion of our sample belonged to Child–Pugh classes B and C (70.2%), indicating more advanced disease where gluconeogenic reserve is reduced and hypoglycemia risk is amplified (Yen et al., 2023; Yang et al., 2023). In addition, settings where prolonged fasting occurs (for procedures or due to decreased oral intake) or where nutritional surveillance is limited tend to show higher point prevalences (Yen et al., 2023; Yang et al., 2023).

Consistent with several prior reports, we found that hypoglycemia in cirrhosis often clusters with markers of severity and complications. Although Child–Pugh class itself did not reach statistical significance in our bivariate tests, the numerically greater proportion of hypoglycemia in Class B/C and the literature linking low albumin, sepsis, and hepatic decompensation to hypoglycemia suggest that impaired hepatic reserve remains a principal driver (Saiman & Mahmud, 2021; Govindarajan et al., 2025). Moreover, other studies have shown that hypoglycemia at admission predicts higher short-term mortality and ICU utilization; Hung and colleagues reported substantially increased 30-day mortality among hypoglycemic cirrhotic inpatients, and more recent analyses have similarly associated hypoglycemia with worse clinical trajectories (Hung et al., 2021). These outcome studies reinforce the clinical importance of the phenomenon we observed. Recent evidence further highlights the clinical importance of hypoglycemia in cirrhotic patients. A study reported that 48.6% of patients experienced hypoglycemia, and those with hypoglycemia had significantly lower event-free survival compared to those without ($p = 0.007$) (Sasaki et al., 2023).

Our study showed that younger age and shorter duration of diagnosed cirrhosis were independently associated with hypoglycemia, whereas Child–Pugh score and BSR type were not. Comparable nuances were observed in other series where both disease acuity and intercurrent illnesses (eg, sepsis) rather than age per se modulated hypoglycemia risk (Yang et al., 2023; Khan et al., 2024).

The extremely large effect size for measured glucose simply confirms that measured capillary/venous glucose strongly discriminates between cases and non-cases by design; nonetheless, it also highlights the clinical relevance of routine glucose monitoring because low glucose values were highly predictive of the hypoglycemia outcome and of the need for intervention (Hung et al., 2021).

Mechanistically, hypoglycemia in cirrhosis arises from impaired hepatic glucose production (both glycogenolysis and gluconeogenesis), malnutrition with depleted substrates, impaired hormonal counter-regulation, and occasionally adrenal insufficiency or sepsis—factors repeatedly described in mechanistic reviews and clinical series (Pfortmueller & Lindner, 2014; Yen et al., 2023). In an analysis, hypoglycemia ($p = 0.034$) and Child–Pugh class B ($p = 0.039$) were predictors of adverse liver-related outcomes, including gastrointestinal bleeding, infection, ascites, encephalopathy, and liver-related death (Sasaki et al., 2023). These findings suggest that, beyond simple glucose monitoring, FGM provides prognostic value in predicting liver-related events among cirrhotic patients These combined pathways explain why hypoglycemia can occur in both advanced disease and in acutely unwell patients irrespective of absolute Child–Pugh score. Our findings align with this multifactorial model.

Clinically, the high point prevalence observed in our cohort supports routine point-of-care glucose checks on admission and during periods of clinical deterioration among cirrhotic patients, and a low threshold for confirmatory venous testing when capillary values are low (Coman et al., 2021). Several authors have advocated for targeted monitoring protocols in high-risk groups (advanced Child–Pugh class, malnutrition, sepsis, and HCC), and our data reinforce that such protocols may be warranted in similar tertiary-care settings (Hung et al., 2021; Iqbal et al., 2024). Prompt recognition and treatment of hypoglycemia could plausibly reduce short-term morbidity and mortality, although prospective interventional data are needed.

The study was single-center and observational, limiting external generalizability; although our sample was large,

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residual confounding (eg, variable nutritional assessments, unmeasured infections, or undocumented medication exposures) could explain part of the observed associations. Also, although point-of-care glucometers were used and confirmatory venous samples were obtained when feasible, capillary measurements have inherent limitations which we acknowledged. Finally, differences in hypoglycemia definitions among studies complicate direct prevalence comparisons. Future multicenter prospective studies should incorporate standardized laboratory confirmation, nutritional status indices, and outcome follow-up to better define causality and optimal management strategies.

Conclusion

This study concluded that hypoglycemia is a highly prevalent complication among patients with liver cirrhosis, affecting nearly three-quarters of the study population. While most events were mild, a significant fraction of cases involved moderate to severe hypoglycemia, illustrating the clinical importance of this metabolic disturbance. Younger age and shorter duration of disease were independent predictors. These findings suggest that hypoglycemia in cirrhosis is influenced by multifactorial mechanisms beyond conventional markers of disease severity.

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