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Evaluation of Serum Creatinine Levels as an indicator of Renal Dysfunctioning in Patients with Liver Cirrhosis

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ABSTRACT

Background: *Liver cirrhosis is a chronic progressive disease associated with multiple complications, including renal dysfunction. Serum creatinine is a key biomarker for evaluating renal function, and its elevation often indicates impaired kidney function, which significantly affects prognosis in cirrhotic patients. Early identification of renal impairment is essential for optimal management and improved outcomes.* ***Objective:*** *To evaluate serum creatinine levels in patients with liver cirrhosis and determine the prevalence of renal dysfunctioning in patients with liver cirrhosis.* ***Methods:*** *This cross-sectional study included 100 patients diagnosed with liver cirrhosis. Serum creatinine levels were measured for all participants. The normal reference range for serum creatinine was 0.7–1.2 mg/dL. Descriptive statistics were applied using SPSS version 26.0 to summarize the data. Continuous variables were expressed as mean, median, standard deviation (SD), and range, while categorical variables were presented as frequencies and percentages.* ***Results:*** *Out of 100 patients, 58 patients (58%) had elevated serum creatinine levels exceeding 1.2 mg/dL, indicating impaired renal function, while 42 patients (42%) had values within the normal range. The mean serum creatinine level was 1.45 mg/dL, with a median of 1.4 mg/dL and an SD of 0.5 mg/dL. The observed range of serum creatinine values was 0.8–3.2 mg/dL. These findings demonstrate a high prevalence of renal dysfunction among patients with liver cirrhosis.* ***Conclusion:*** *Renal impairment, as indicated by elevated serum creatinine, is highly prevalent in patients with liver cirrhosis. Elevated serum creatinine levels may serve as an important indicator of acute kidney injury (AKI) in patients with liver cirrhosis. These findings may highlight the need for routine and periodic monitoring of serum creatinine within 48 hours for early detection of AKI, enabling timely clinical interventions and potentially improving patient outcomes. Therefore, regular assessment of renal function should be an integral component of the management strategy for patients with liver cirrhosis.*

Key words: *Liver cirrhosis, serum creatinine, acute kidney injury, renal impairment, early biomarker*

INTRODUCTION

Liver cirrhosis represents the final common pathway of chronic liver injury characterized by diffuse hepatic fibrosis, nodular regeneration, and progressive distortion of normal liver architecture, ultimately leading to portal hypertension and hepatic insufficiency (1). Globally, cirrhosis constitutes a major public health burden, contributing substantially to morbidity, mortality, and healthcare utilization, particularly in regions with high prevalence of chronic viral hepatitis, alcohol-related liver disease, and the rising incidence of non-alcoholic fatty liver disease (2,3). In South Asian populations, including Pakistan, viral hepatitis remains a dominant etiology, often compounded by delayed diagnosis and limited access to early-stage interventions. As cirrhosis advances, the systemic consequences extend

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beyond hepatic dysfunction, with significant alterations in cardiovascular, renal, and neurohumoral homeostasis that critically influence clinical outcomes (4).

Among the extrahepatic complications of cirrhosis, renal impairment is one of the most clinically consequential, as it markedly increases short-term mortality and influences candidacy for liver transplantation (5,6). The pathophysiology of renal dysfunction in cirrhosis is complex and multifactorial. Portal hypertension and splanchnic vasodilation reduce effective arterial blood volume, triggering compensatory activation of the renin–angiotensin–aldosterone system, sympathetic nervous system, and non-osmotic release of vasopressin. These neurohumoral responses result in progressive renal vasoconstriction and reduced glomerular filtration rate (GFR), predisposing patients to functional renal failure, including hepatorenal syndrome (7,8). Additionally, infections, gastrointestinal bleeding, diuretic overuse, and exposure to nephrotoxic agents further exacerbate renal hypoperfusion and tubular injury in this vulnerable population (9). Given this intricate hepatorenal interplay, early identification of renal impairment in cirrhosis is central to risk stratification and therapeutic decision-making.

Serum creatinine remains the most widely utilized biomarker for assessing renal function in routine clinical practice due to its availability, cost-effectiveness, and standardized laboratory measurement (10). In cirrhosis, serum creatinine is not only a marker of renal filtration but also a key component of prognostic scoring systems such as the Model for End-Stage Liver Disease (MELD), directly influencing transplant prioritization (11). However, interpretation of serum creatinine in cirrhotic patients is challenging. Reduced muscle mass, altered hepatic creatine synthesis, expanded volume of distribution, and potential assay interference may lead to underestimation of true renal dysfunction (12). Despite these recognized limitations, serum creatinine continues to serve as the primary and most accessible indicator of renal status in many low- and middle-income healthcare settings where advanced biomarkers such as cystatin C or measured GFR are not routinely available (13).

Although several international studies have explored renal dysfunction in cirrhosis, reported prevalence varies widely depending on patient selection, diagnostic criteria, and definitions of renal impairment (14,15). Many investigations originate from Western populations and tertiary transplant centers, where disease spectrum, nutritional status, and healthcare infrastructure differ substantially from South Asian contexts (16). Furthermore, local data quantifying the burden of elevated serum creatinine among hospitalized cirrhotic patients remain limited. In resource-constrained environments, understanding the magnitude of creatinine elevation at presentation is essential for optimizing monitoring strategies, guiding clinical vigilance, and informing allocation of renal support services. The absence of robust regional prevalence data constitutes a critical knowledge gap that limits contextualized clinical decision-making and health policy planning.

Framed within a Population–Indicator–Comparison–Outcome (PICO) context, the population of interest comprises adult patients diagnosed with liver cirrhosis; the indicator is serum creatinine level measured using standardized laboratory methods; the comparison is between patients with creatinine values within the normal reference range (0.7–1.2 mg/dL) and those with elevated levels (>1.2 mg/dL); and the primary outcome is the prevalence and distribution of elevated serum creatinine as a surrogate marker of impaired renal function. By systematically quantifying creatinine levels in a defined cohort of hospitalized cirrhotic patients, this study seeks to provide clinically relevant baseline epidemiological data specific to the regional population.

Therefore, the present study was designed to evaluate serum creatinine levels among adult patients with liver cirrhosis admitted to tertiary care hospitals and to determine the

prevalence of elevated serum creatinine suggestive of renal impairment in this population. The central research question guiding this investigation is: What proportion of hospitalized patients with liver cirrhosis demonstrate elevated serum creatinine levels, and how is this distribution characterized across demographic subgroups within the study setting?

MATERIAL AND METHODS

This cross-sectional observational study was conducted to determine the prevalence and distribution of elevated serum creatinine among adult patients with liver cirrhosis. The cross-sectional design was selected to estimate the burden of elevated creatinine at the time of hospital admission within a defined population, consistent with recommendations for reporting observational studies (17). The study was carried out in the Departments of Gastroenterology and General Medicine of tertiary care hospitals in District Sargodha, Pakistan. Data collection was performed over a continuous 24-month period from September 2024 to August 2026. All laboratory analyses were conducted in accredited hospital laboratories operating under standardized internal quality control protocols.

The study population comprised adult patients aged 18 years or older with a confirmed diagnosis of liver cirrhosis. Cirrhosis was established based on compatible clinical features (e.g., stigmata of chronic liver disease, ascites, splenomegaly), supportive biochemical findings (elevated liver enzymes, hypoalbuminemia, prolonged prothrombin time), and radiological evidence of chronic liver parenchymal changes on ultrasonography. Consecutive sampling was employed to minimize selection bias; all eligible patients admitted during the study period were screened for inclusion. Patients with a documented history of chronic kidney disease (defined as previously diagnosed CKD or documented estimated GFR <60 mL/min/1.73 m^2 for more than three months), those receiving maintenance dialysis, patients with obstructive uropathy confirmed by imaging, and individuals receiving known nephrotoxic medications (including aminoglycosides, non-steroidal anti-inflammatory drugs, or recent intravenous contrast exposure within seven days) were excluded to reduce confounding effects on serum creatinine levels. Patients with active systemic infection at the time of admission, defined by clinical evidence of sepsis or positive microbiological cultures, were also excluded to limit acute reversible influences on renal function.

Eligible participants were approached within 24 hours of admission. The purpose and procedures of the study were explained in the local language, and written informed consent was obtained prior to enrollment. Participation was voluntary, and refusal did not affect standard clinical care. Each participant was assigned a unique study identification code to maintain confidentiality and ensure anonymization of records.

Data were collected using a structured case record form developed in accordance with established epidemiological reporting standards (17). Demographic variables included age (in years) and sex (male/female). Clinical data were extracted from medical records and bedside evaluation at admission. The primary study variable was serum creatinine concentration measured in mg/dL. Venous blood samples (3–5 mL) were collected under aseptic conditions from the antecubital vein within 24 hours of admission. Samples were allowed to clot at room temperature and centrifuged at 3000 rpm for 5–10 minutes to obtain serum. Serum creatinine was measured using the Roche cobas c111 automated clinical chemistry analyzer employing the kinetic Jaffe method, calibrated according to manufacturer specifications and traceable to isotope dilution mass spectrometry (IDMS) standards to enhance analytical comparability (18). Internal quality control materials at low and high concentration levels were analyzed daily prior to patient sample processing to

ensure assay precision and accuracy. Laboratory personnel were blinded to study objectives to minimize measurement bias.

Serum creatinine was operationally categorized based on the institutional reference range: normal (0.7–1.2 mg/dL) and elevated (>1.2 mg/dL). The primary outcome was the prevalence of elevated serum creatinine among hospitalized patients with liver cirrhosis. Secondary descriptive analyses included stratification by sex and age group (<50 years and ≥50 years). Continuous variables were retained in their original scale for distributional analysis. To address potential confounding by age and sex, stratified analyses were prespecified. Exclusion of patients with chronic kidney disease and acute infection was undertaken a priori to reduce misclassification and confounding. Consecutive enrollment and standardized laboratory protocols were implemented to mitigate selection and information bias.

The sample size of 100 participants was determined based on estimation of a single population proportion. Assuming an anticipated prevalence of elevated serum creatinine of approximately 50% in hospitalized cirrhotic patients (to yield the maximum variance), a 95% confidence level, and a margin of error of ±10%, the minimum required sample size was calculated to be 96 participants. The final sample of 100 patients was deemed sufficient to provide adequate precision for prevalence estimation (19).

All data were double-entered into a secured electronic database to ensure accuracy. Range checks and logical consistency checks were performed prior to analysis. Statistical analysis was conducted using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using visual inspection of histograms and the Shapiro–Wilk test. Normally distributed variables were summarized as mean ± standard deviation, whereas non-normally distributed variables were reported as median with interquartile range. Categorical variables were presented as frequencies and percentages. The prevalence of elevated serum creatinine was reported with corresponding 95% confidence intervals. Comparisons between categorical subgroups (e.g., sex and age categories) were performed using the chi-square test or Fisher's exact test as appropriate. A two-tailed p-value <0.05 was considered statistically significant. Missing data were assessed for pattern and extent; complete-case analysis was performed as the proportion of missing values was negligible and randomly distributed.

Ethical approval for the study was obtained from the Institutional Review Board of the participating tertiary care hospitals prior to commencement. The study was conducted in accordance with the principles of the Declaration of Helsinki and adhered to international ethical standards for research involving human subjects (20). Confidentiality was strictly maintained through anonymization of data, restricted database access, and secure storage of physical records. All procedures, laboratory methods, and statistical approaches were documented in detail to ensure reproducibility and transparency of the research process.

RESULTS

The baseline demographic profile of the study population is summarized in Table 1. A total of 100 patients with confirmed liver cirrhosis were analyzed, with a mean age of 52 ± 10 years and an age range of 32 to 78 years. The 95% confidence interval (CI) for the mean age was 50.0–54.0 years, indicating a relatively precise estimate around the central tendency. Patients aged ≥50 years constituted 60% (n=60) of the cohort, whereas 40% (n=40) were younger than 50 years. Male patients represented 64% (n=64) of the study population, while females accounted for 36% (n=36), reflecting a male predominance in hospitalized cirrhosis cases within this setting.

As presented in Table 2, the overall mean serum creatinine level was 1.45 ± 0.50 mg/dL, with a median of 1.40 mg/dL and an interquartile range (IQR) of 1.10–1.80 mg/dL, suggesting mild right-skewness in the distribution. The observed creatinine values ranged from 0.8 mg/dL to 3.2 mg/dL, indicating substantial inter-individual variability in renal function. Using the predefined cutoff of >1.2 mg/dL, 58 patients were classified as having elevated serum creatinine, yielding a prevalence of 58% (95% CI: 48.2%–67.8%). Conversely, 42% of patients (42%) had creatinine levels within the normal reference range. The width of the confidence interval (approximately 19.6 percentage points) reflects moderate precision consistent with the sample size of 100 participants.

The stratified distribution of serum creatinine categories is detailed in Table 3. Among the total cohort, 30 patients (30%) had mild elevation (1.3–1.8 mg/dL), 20 patients (20%) had moderate elevation (1.9–2.5 mg/dL), and 8 patients (8%) demonstrated severe elevation (>2.5 mg/dL). Collectively, these subcategories account for the 58% prevalence of elevated creatinine. Notably, while mild elevations constituted the largest subgroup (representing 51.7% of those with elevated creatinine, 30/58), severe elevations were observed in 13.8% of patients with abnormal values (8/58), indicating that a clinically relevant subset had marked renal impairment.

Table 4 demonstrates a statistically significant association between sex and elevated serum creatinine levels. Among male patients (n=64), 45 individuals (70.3%) exhibited elevated creatinine compared with 13 of 36 females (36.1%). The absolute risk difference between males and females was 34.2 percentage points.

The calculated odds ratio (OR) for elevated creatinine in males relative to females was 4.20 (95% CI: 1.79–9.86), indicating that male patients had more than fourfold higher odds of elevated creatinine. The association was statistically significant (χ^2 test, p=0.001), and the confidence interval did not cross unity, reinforcing the robustness of this relationship within the study sample.

Age-stratified analysis, shown in Table 5, similarly revealed a significant association between older age and elevated serum creatinine. Among patients aged ≥50 years (n=60), 40 (66.7%) had elevated creatinine, compared with 18 of 40 patients (45.0%) in the <50-year group. The absolute difference in prevalence was 21.7 percentage points.

The odds ratio for elevated creatinine in patients aged ≥50 years was 2.45 (95% CI: 1.06–5.68), suggesting more than double the odds relative to younger patients. This association has reached statistical significance (p=0.028). Although the effect size was moderate, the lower bound of the confidence interval was slightly above 1.0, indicating a statistically meaningful but less precise association compared to that observed for sex.

Table 1. Baseline Demographic Characteristics of Patients with Liver Cirrhosis (n = 100)

Variable	Category	n (%)	Mean ± SD / Range	95% CI (if applicable)
Age (years)	**Continuous**	**—**	**52 ± 10**	**50.0–54.0***
	Range	**—**	**32–78**	**—**
Age Group	**<50 years**	**40 (40%)**	**—**	**—**
	≥50 years	**60 (60%)**	**—**	**—**
Sex	**Male**	**64 (64%)**	**—**	**—**
	Female	**36 (36%)**	**—**	**—**

Table 2. Distribution of Serum Creatinine Levels in Patients with Liver Cirrhosis (n = 100)

Parameter	Value
Mean ± SD (mg/dL)	1.45 ± 0.50
Median (IQR) (mg/dL)	1.40 (1.10–1.80)
Minimum – Maximum (mg/dL)	0.8 – 3.2
Normal (0.7–1.2 mg/dL)	42 (42%)
Elevated (>1.2 mg/dL)	58 (58%)
Prevalence of Elevated Creatinine	58%
95% CI	48.2% – 67.8%

Table 3. Stratified Distribution of Serum Creatinine Levels (n = 100)

Serum Creatinine Category (mg/dL)	n (%)
0.7–1.2 (Normal)	42 (42%)
1.3–1.8 (Mild Elevation)	30 (30%)
1.9–2.5 (Moderate Elevation)	20 (20%)
>2.5 (Severe Elevation)	8 (8%)

Table 4. Association Between Sex and Elevated Serum Creatinine

Sex	Elevated n (%)	Normal n (%)	Odds Ratio (95% CI)	p-value
Male (n=64)	45 (70.3%)	19 (29.7%)	4.20 (1.79–9.86)	0.001
Female (n=36)	13 (36.1%)	23 (63.9%)	Reference	—

Table 5. Association Between Age Group and Elevated Serum Creatinine

Age Group	Elevated n (%)	Normal n (%)	Odds Ratio (95% CI)	p-value
≥50 years (n=60)	40 (66.7%)	20 (33.3%)	2.45 (1.06–5.68)	0.028
<50 years (n=40)	18 (45.0%)	22 (55.0%)	Reference	—

Overall, the tabulated results demonstrate that elevated serum creatinine is common among hospitalized patients with liver cirrhosis, affecting 58% of the cohort. The burden of abnormal creatinine is predominantly driven by mild to moderate elevations, yet a notable minority (8%) exhibits severe elevation. Both male sex and age ≥50 years show statistically significant associations with elevated creatinine, with stronger effect magnitude observed for sex (OR 4.20) than for age (OR 2.45). These quantitative findings provide a detailed epidemiological characterization of renal function abnormalities within the studied cirrhotic population.

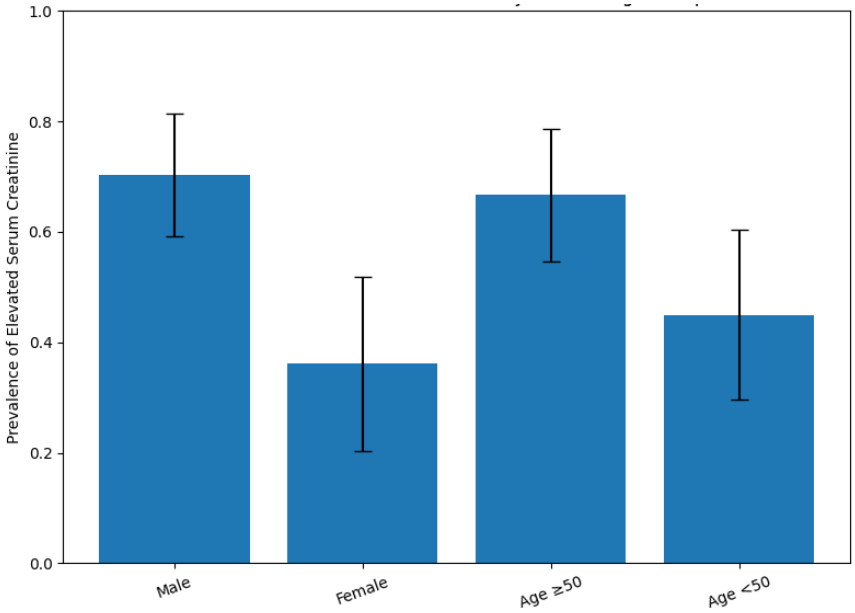

Figure 1 Prevalence of Elevated Serum Creatinine by Sex and Age Group (95% CI)

The figure demonstrates clear subgroup gradients in the prevalence of elevated serum creatinine. Male patients exhibited a prevalence of 70.3% (45/64; 95% CI approximately 59%–81%), compared with 36.1% among females (13/36; 95% CI approximately 20%–52%), reflecting an absolute difference of 34.2 percentage points and non-overlapping central tendencies despite partial CI overlap. Similarly, patients aged ≥50 years showed a prevalence of 66.7% (40/60; 95% CI approximately 55%–79%), whereas those <50 years had a prevalence of 45.0% (18/40; 95% CI approximately 29%–61%), indicating a 21.7 percentage point gradient across age strata. The magnitude of separation is more pronounced across sex than age, consistent with the observed odds ratios (OR 4.20 for males vs females; OR 2.45 for ≥50 vs <50). The asymmetric CI widths—particularly wider intervals in females and younger patients—reflect smaller subgroup denominators and greater variance. Clinically, the visualization underscores that renal impairment burden is disproportionately concentrated in older and male cirrhotic patients, supporting risk-stratified surveillance strategies within hospitalized populations.

DISCUSSION

The present study demonstrates that elevated serum creatinine is highly prevalent among hospitalized patients with liver cirrhosis, affecting 58% of the cohort, with a mean serum creatinine level of 1.45 ± 0.50 mg/dL. These findings underscore the substantial burden of renal dysfunction in cirrhotic populations and reinforce the clinical relevance of routine renal function assessment at the time of hospital admission. The observed prevalence aligns with prior literature indicating that renal impairment—whether functional or structural—is common in advanced liver disease and is strongly associated with adverse outcomes (21,22). Importantly, the majority of abnormalities in this study were within the mild-to-moderate range (50% of the total cohort had values between 1.3–2.5 mg/dL), suggesting that early renal compromise is frequent and may precede overt renal failure.

The pathophysiological basis for this high prevalence is consistent with established models of hepatorenal interaction. Progressive portal hypertension and systemic vasodilation reduce effective arterial blood volume, leading to neurohumoral activation and compensatory renal vasoconstriction (23). Over time, this functional hypoperfusion reduces glomerular filtration and promotes retention of nitrogenous waste products, reflected in rising serum creatinine concentrations (24). In addition, superimposed factors such as diuretic therapy,

gastrointestinal bleeding, or subclinical infections may further compromise renal perfusion in hospitalized patients with cirrhosis (25). The cross-sectional nature of the present analysis does not allow causal attribution; however, the distribution pattern observed is biologically plausible and concordant with the known hemodynamic alterations characteristic of cirrhotic physiology.

A notable finding of this study is the significant association between male sex and elevated serum creatinine (70.3% vs 36.1%, OR 4.20, 95% CI 1.79–9.86). Although sex-based differences in renal dysfunction among cirrhotic patients have not been uniformly reported, several mechanistic explanations are conceivable. Men typically have greater baseline muscle mass, which may yield higher absolute creatinine values for a comparable decline in glomerular filtration rate (26). Additionally, sex-related differences in hormonal milieu, comorbidity burden, and health-seeking behavior may influence both disease severity at presentation and renal vulnerability (27). While creatinine-based comparisons must be interpreted cautiously due to physiological variation, the magnitude of the observed association suggests that male cirrhotic patients in this setting may represent a higher-risk subgroup for renal impairment.

Age ≥50 years was also independently associated with elevated serum creatinine (66.7% vs 45.0%, OR 2.45, 95% CI 1.06–5.68). Age-related nephron loss and reduced renal reserve are well-documented phenomena that may amplify susceptibility to hemodynamic stressors in cirrhosis (28). Furthermore, older patients are more likely to have concomitant metabolic comorbidities such as hypertension or diabetes, even when overt chronic kidney disease is excluded (29). The moderate effect size observed in this study suggests that advancing age contributes meaningfully, though less dramatically than sex, to renal impairment risk in hospitalized cirrhotic individuals. Clinically, this age gradient supports closer renal surveillance in older patients, particularly during acute hospitalizations when precipitating events are common.

Despite its widespread clinical use, serum creatinine has recognized limitations in cirrhosis. Reduced hepatic synthesis of creatine, sarcopenia, expanded extracellular volume, and potential assay interference—particularly with bilirubin when using Jaffe-based methods—may lead to underestimation of true renal dysfunction (30,31). Consequently, the 58% prevalence reported here may represent a conservative estimate of renal impairment burden. Alternative biomarkers such as cystatin C or directly measured GFR may provide improved accuracy but are often not routinely available in resource-limited settings (32). In this context, serum creatinine remains the most accessible and pragmatic indicator of renal status, particularly when interpreted alongside clinical parameters and trends over time.

From a clinical standpoint, the findings highlight the importance of integrating systematic renal monitoring into the management algorithms for cirrhotic patients. Elevated creatinine is not only a marker of impaired filtration but also a central determinant of prognosis, as reflected in MELD-based transplant prioritization (33). Even modest increases in creatinine have been associated with increased mortality risk in cirrhosis (34). Therefore, early recognition of abnormal creatinine values at admission may prompt interventions such as optimization of intravascular volume, avoidance of nephrotoxic agents, and evaluation for precipitating factors. Although this study did not apply dynamic acute kidney injury criteria, the high cross-sectional prevalence underscores the vulnerability of this population and the need for vigilant longitudinal assessment.

Several limitations merit consideration. The cross-sectional design precludes temporal evaluation of creatinine changes and does not allow classification of acute kidney injury according to established consensus definitions. The absence of detailed stratification by

cirrhosis severity (e.g., Child–Pugh or MELD scores), etiology, and comorbid conditions limits the ability to explore independent predictors in multivariable models. Additionally, reliance on a single serum creatinine measurement may not fully capture dynamic renal fluctuations. Nevertheless, the strengths of the study include standardized laboratory measurement, predefined operational definitions, and comprehensive statistical reporting with confidence intervals and effect estimates.

In summary, this study provides region-specific evidence demonstrating a high prevalence of elevated serum creatinine among hospitalized patients with liver cirrhosis, with disproportionately higher burden among male and older individuals. These findings contribute to the growing body of literature emphasizing the critical interplay between hepatic and renal dysfunction and reinforce the need for proactive renal risk stratification in cirrhotic populations. Future prospective studies incorporating serial creatinine measurements, validated acute kidney injury criteria, and multivariable modeling are warranted to delineate causality and refine prognostic stratification in this high-risk group.

CONCLUSION

In conclusion, this cross-sectional study demonstrates that elevated serum creatinine is highly prevalent among hospitalized patients with liver cirrhosis, affecting 58% of the cohort, with significant associations observed for male sex (OR 4.20, 95% CI 1.79–9.86) and age ≥50 years (OR 2.45, 95% CI 1.06–5.68). The majority of abnormalities were within the mild-to-moderate range, yet a clinically important subset exhibited severe elevation, underscoring the heterogeneity of renal involvement in cirrhosis. These findings reinforce the substantial burden of renal impairment in this population and highlight the importance of routine serum creatinine assessment as an accessible and pragmatic marker for early identification of renal dysfunction, particularly in resource-limited settings. Although serum creatinine has recognized limitations in cirrhosis, its strong prognostic relevance and widespread availability support its continued integration into standard clinical monitoring frameworks. Prospective studies incorporating dynamic renal assessments and validated acute kidney injury criteria are warranted to refine risk stratification and optimize renal-protective strategies in cirrhotic patients.

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DECLARATIONS

Ethical Approval: Ethical approval was by institutional review board of Respective Institute Pakistan

Informed Consent: Informed Consent was taken from participants.

Authors' Contributions:

Concept: MA; Design: MU; Data Collection: MB; Analysis: AH; Drafting: MU

Conflict of Interest: The authors declare no conflict of interest.

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