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# Clinical and Biochemical Predictors of Complications Among Patients With Chronic Liver Disease: A Prospective Observational Study.

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## Abstract

***Background:*** Chronic liver disease (CLD) progresses through variable clinical stages, and the development of decompensating complications substantially worsens prognosis. Simple clinical and biochemical markers that identify patients at increased short-term risk can support closer monitoring and timely intervention. ***Objectives:*** To evaluate clinical and biochemical predictors of major complications among patients with CLD and to determine independent factors associated with adverse outcomes. ***Methods:*** This prospective observational study included 80 adults with CLD managed in the Department of General Medicine, MGM Hospital/Kakatiya Medical College, Warangal, Telangana, India. Baseline demographic, clinical and biochemical variables were recorded, and Child-Pugh and Model for End-Stage Liver Disease (MELD) scores were calculated. Participants were prospectively monitored for major complications. Variables associated with complications were assessed using univariate and multivariable logistic regression. ***Results:*** The mean age was 52.8 ± 11.7 years; 67.5% were male. Alcohol-related liver disease was the leading aetiology (41.3%). During follow-up, 38 patients (47.5%) developed at least one major complication. Hepatic encephalopathy occurred in 21.3%, spontaneous bacterial peritonitis in 15.0%, acute kidney injury in 13.8%, and variceal bleeding in 11.3%. Patients with complications had higher bilirubin, INR, creatinine, Child-Pugh and MELD scores and lower albumin, platelet count and sodium levels. Albumin <3.0 g/dL, creatinine ≥1.5 mg/dL, sodium <135 mmol/L and MELD score ≥15 remained independent predictors; MELD ≥15 showed the strongest association. ***Conclusion:*** Complications were frequent in CLD and were independently associated with hypoalbuminaemia, renal dysfunction, hyponatraemia and higher MELD scores. Routine biochemical parameters combined with severity scoring can provide practical early risk stratification.

**Keywords:** chronic liver disease; cirrhosis; complications; MELD score; hypoalbuminaemia; hyponatraemia; renal dysfunction.



## INTRODUCTION

Chronic liver disease (CLD) encompasses a broad spectrum of progressive hepatic disorders that can culminate in cirrhosis, portal hypertension, hepatic insufficiency and hepatocellular carcinoma. Liver disease contributes substantially to global morbidity and mortality, with cirrhosis and its complications accounting for a major share of liver-related deaths.1 The contemporary burden is driven principally by alcohol-related liver disease, metabolic dysfunction-associated steatotic liver disease and chronic viral hepatitis, although the relative contribution of these causes varies across regions and populations. In India, alcohol has emerged as a leading cause of cirrhosis in adults, while metabolic and viral causes remain important contributors to the overall CLD burden.2,3. The clinical course of cirrhosis is heterogeneous. Patients can remain compensated for prolonged periods, but the occurrence of ascites, variceal haemorrhage, hepatic encephalopathy, jaundice, spontaneous bacterial peritonitis or renal dysfunction marks a transition toward decompensated disease and a markedly poorer prognosis. Natural-history studies have demonstrated that decompensation represents a clinically meaningful turning point, with subsequent complications increasing the probability of recurrent hospitalisation, organ failure and death.4,5 Progressive portal hypertension, systemic vasodilatation, circulatory dysfunction, bacterial translocation and systemic inflammation contribute to this transition and help explain why apparently modest biochemical abnormalities can precede major clinical deterioration.

Risk stratification is therefore central to the management of CLD. The Child-Pugh classification has traditionally been used to describe hepatic functional reserve, whereas the Model for End-Stage Liver Disease (MELD) score provides an objective estimate based on serum bilirubin, creatinine and the international normalised ratio. The MELD score has established prognostic value across advanced liver disease.6 Serum sodium adds important information because dilutional

hyponatraemia reflects severe circulatory dysfunction and is associated with ascites, hepatorenal physiology and increased mortality.7,8 Likewise, hypoalbuminaemia indicates impaired hepatic synthetic function and altered circulatory homeostasis, while rising serum creatinine can signal clinically important renal dysfunction. These routinely available variables are attractive for bedside prediction because they are inexpensive, reproducible and obtainable in most secondary and tertiary care hospitals. Despite the availability of prognostic scores, patients with similar global severity can follow different short-term clinical trajectories. Local prospective data assessing the combined contribution of bedside findings and biochemical abnormalities to subsequent complications remain valuable, particularly in Indian tertiary-care settings where the aetiological spectrum and stage at presentation differ from many transplant-based cohorts. Identification of high-risk profiles can support intensified surveillance, earlier management of precipitating factors and appropriate referral. The objective of the present study was to evaluate the clinical and biochemical predictors of complications among patients with CLD treated at a tertiary-care teaching hospital in Warangal, Telangana. The study further aimed to compare baseline clinical and laboratory characteristics between patients who developed major complications and those who did not, and to identify independent predictors using multivariable analysis, with particular attention to serum albumin, creatinine, sodium, Child-Pugh class and MELD score.

## MATERIALS AND METHODS

**Study design and setting:** This prospective observational study was conducted in the Department of General Medicine, Mahatma Gandhi Memorial (MGM) Hospital/Kakatiya Medical College, Warangal, Telangana, India, a tertiary-care teaching centre serving patients from Warangal and surrounding districts. The overall study period was from May 2025 to April 2026. Recruitment was performed from May 2025 through March 2026, and the final scheduled short-term assessment was completed by April 2026.

**Study population and eligibility:** Consecutive adults aged 18 years or older with established CLD/cirrhosis were screened. Patients were eligible when sufficient baseline clinical and laboratory information was available and they provided written informed consent. Patients with acute liver failure without underlying CLD, known hepatocellular carcinoma at enrolment, severe extrahepatic illness expected to dominate short-term outcome, pregnancy, incomplete essential investigations, or inability to complete the planned assessment were excluded.

**Sample size and sampling:** Assuming an anticipated complication proportion of 50%, a 95% confidence level and an absolute precision of approximately 11%, the minimum sample size calculated using $n = Z^2p(1-p)/d^2$ was approximately 80. Therefore, 80 eligible patients were enrolled using consecutive sampling.

**Clinical and laboratory assessment:** At enrolment, demographic variables, aetiology of CLD, previous decompensating events, ascites, pedal oedema, splenomegaly, gastrointestinal bleeding and hepatic encephalopathy were documented through history, examination and record review. Baseline investigations included complete blood count, serum bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, serum albumin, prothrombin time/INR, serum creatinine, sodium and potassium. Child-Pugh class was assigned from standard clinical and biochemical components, and MELD score was calculated from bilirubin, INR and creatinine using the established prognostic model.6

**Outcome assessment:** Participants were prospectively monitored during the index clinical episode and subsequent follow-up for development of major CLD-related complications. Prespecified outcomes included hepatic encephalopathy, spontaneous bacterial peritonitis, acute kidney injury, variceal upper gastrointestinal bleeding, hepatorenal syndrome, severe or refractory ascites, hospitalisation for decompensation and short-term death. Ascites, spontaneous bacterial peritonitis and hepatorenal complications were identified using accepted cirrhosis guidance,9 while hepatic encephalopathy was diagnosed clinically after exclusion of alternative causes.11 Renal dysfunction was assessed using serum creatinine and accepted acute kidney injury concepts in cirrhosis.10

**Bias control and ethics:** Consecutive recruitment reduced selection bias. Baseline measurements were obtained before outcome classification, predefined definitions were used, and the same case-record form was applied to all participants. Laboratory testing was performed through the hospital laboratory using standardised methods. The study protocol was approved by the Institutional Ethics Committee of Kakatiya Medical College/MGM Hospital (approval number to be inserted), and written informed consent was obtained from each participant.

**Statistical analysis:** Data were analysed using appropriate statistical software. Continuous variables were summarised as mean ± standard deviation and categorical variables as frequency and percentage. Between-group comparisons used the independent-samples t test for continuous variables and chi-square test or Fisher's exact test, as appropriate, for categorical variables. Univariate logistic regression identified candidate predictors of complications. Clinically relevant variables showing significant univariate associations were entered into multivariable logistic regression. Odds ratios with 95% confidence intervals were reported, and a two-sided p value $<0.05$ was considered statistically significant.

## RESULTS

A total of 86 patients with chronic liver disease were assessed for eligibility during the study period. Six were excluded because of incomplete essential biochemical investigations, acute liver failure without established chronic disease, or inability to complete the planned assessment. The remaining 80 patients constituted the final study population. The mean age was 52.8 ± 11.7 years (range 24-76 years), and 54 (67.5%) were male. Alcohol-related liver disease was the most frequent aetiology, followed by metabolic dysfunction-associated steatotic liver disease and chronic viral hepatitis. Ascites was present in more than half of the cohort, and 45.0% of patients were classified as Child-Pugh class B (Table 1).

**Table 1. Baseline demographic and clinical characteristics of study participants (N=80)**

| Characteristic | Value |
|---|---|
| Age, years, mean ± SD | 52.8 ± 11.7 |
| Age ≤50 years | 32 (40.0%) |
| Age >50 years | 48 (60.0%) |
| Male | 54 (67.5%) |
| Female | 26 (32.5%) |
| Alcohol-related liver disease | 33 (41.3%) |
| Metabolic dysfunction-associated steatotic liver disease | 19 (23.8%) |
| Chronic viral hepatitis | 15 (18.8%) |
| Other causes | 13 (16.3%) |
| Ascites | 42 (52.5%) |
| Pedal oedema | 26 (32.5%) |
| Splenomegaly | 31 (38.8%) |
| Previous hepatic encephalopathy | 18 (22.5%) |
| Gastrointestinal bleeding at/before enrolment | 16 (20.0%) |
| Child-Pugh score, mean ± SD | 8.4 ± 2.1 |
| MELD score, mean ± SD | 14.7 ± 5.8 |
| Child-Pugh class A | 21 (26.3%) |
| Child-Pugh class B | 36 (45.0%) |
| Child-Pugh class C | 23 (28.8%) |

Values are n (%) unless otherwise indicated. SD: standard deviation; MELD: Model for End-Stage Liver Disease.

The biochemical profile demonstrated reduced hepatic synthetic function and frequent haematological abnormalities. Mean haemoglobin was 10.8 ± 2.1 g/dL and mean platelet count was 118.6 ± 54.3 ×10³/µL. Mean serum albumin was 2.9 ± 0.6 g/dL, total bilirubin 3.4 ± 2.7 mg/dL and INR 1.55 ± 0.43. Hyponatraemia, defined as serum sodium <135 mmol/L, was observed in 27 (33.8%) patients, while serum creatinine ≥1.5 mg/dL was present in 17 (21.3%) (Table 2).

**Table 2. Baseline biochemical profile of patients with chronic liver disease**

| Biochemical parameter | Value |
|---|---|
| Haemoglobin, g/dL | 10.8 ± 2.1 |
| Total leucocyte count, cells/mm³ | 7,860 ± 3,140 |
| Platelet count, ×10³/µL | 118.6 ± 54.3 |
| Total bilirubin, mg/dL | 3.4 ± 2.7 |
| Direct bilirubin, mg/dL | 1.9 ± 1.6 |
| AST, U/L | 72.8 ± 46.2 |
| ALT, U/L | 51.6 ± 35.4 |
| ALP, U/L | 168.4 ± 76.9 |
| Serum albumin, g/dL | 2.9 ± 0.6 |
| INR | 1.55 ± 0.43 |
| Serum creatinine, mg/dL | 1.18 ± 0.67 |
| Serum sodium, mmol/L | 135.1 ± 5.2 |
| Serum potassium, mmol/L | 4.1 ± 0.6 |
| Serum albumin <3.0 g/dL | 39 (48.8%) |
| Total bilirubin ≥3 mg/dL | 31 (38.8%) |
| INR ≥1.5 | 34 (42.5%) |
| Serum creatinine ≥1.5 mg/dL | 17 (21.3%) |
| Serum sodium <135 mmol/L | 27 (33.8%) |

Continuous variables are expressed as mean ± SD. AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; INR: international normalised ratio.

During prospective observation, 38 of 80 patients (47.5%) developed at least one major complication, whereas 42 (52.5%) remained free of a major complication. Hepatic encephalopathy was the most frequent event, occurring in 17 (21.3%) patients, followed by severe or refractory ascites in 14 (17.5%), spontaneous bacterial peritonitis in 12 (15.0%), acute kidney injury in 11 (13.8%) and variceal upper gastrointestinal bleeding in 9 (11.3%). Seven patients (8.8%) died during short-term observation. Individual complications were not mutually exclusive (Table 3).

**Table 3. Complications observed during the study period**

| Complication | n (%) |
|---|---|
| Any major complication | 38 (47.5) |
| Hepatic encephalopathy | 17 (21.3) |
| Spontaneous bacterial peritonitis | 12 (15.0) |
| Acute kidney injury | 11 (13.8) |
| Variceal upper gastrointestinal bleeding | 9 (11.3) |
| Hepatorenal syndrome | 6 (7.5) |
| Severe/refractory ascites | 14 (17.5) |
| Hospitalisation for decompensation | 24 (30.0) |
| Short-term mortality | 7 (8.8) |

Individual complications were not mutually exclusive.

Patients who developed complications had significantly greater liver disease severity and more pronounced biochemical derangement. Mean Child-Pugh and MELD scores were higher in the complication group than in the group without complications. Serum bilirubin, INR and creatinine were also higher, whereas serum albumin, platelet count and sodium were lower. Ascites and a previous history of hepatic encephalopathy were significantly more frequent among patients who subsequently experienced complications. Age and sex were not significantly different between the two groups (Table 4).

**Table 4. Comparison of clinical and biochemical parameters according to occurrence of complications**

| Parameter | Complications (n=38) | No complications (n=42) | p value |
|---|---|---|---|
| Age, years | 55.1 ± 11.0 | 50.7 ± 12.1 | 0.093 |
| Male sex | 27 (71.1%) | 27 (64.3%) | 0.518 |
| Ascites | 27 (71.1%) | 15 (35.7%) | 0.002 |
| Previous hepatic encephalopathy | 13 (34.2%) | 5 (11.9%) | 0.017 |
| Haemoglobin, g/dL | 10.3 ± 2.0 | 11.3 ± 2.1 | 0.033 |
| Platelet count, ×10³/µL | 101.4 ± 45.8 | 134.2 ± 57.1 | 0.006 |
| Total bilirubin, mg/dL | 4.8 ± 3.0 | 2.1 ± 1.6 | <0.001 |
| Serum albumin, g/dL | 2.5 ± 0.5 | 3.2 ± 0.5 | <0.001 |
| INR | 1.79 ± 0.43 | 1.33 ± 0.30 | <0.001 |
| Serum creatinine, mg/dL | 1.48 ± 0.80 | 0.91 ± 0.39 | <0.001 |
| Serum sodium, mmol/L | 132.9 ± 5.1 | 137.1 ± 4.5 | <0.001 |
| Child-Pugh score | 9.6 ± 1.8 | 7.3 ± 1.7 | <0.001 |
| MELD score | 18.1 ± 5.3 | 11.6 ± 4.3 | <0.001 |

Continuous variables are mean ± SD. MELD: Model for End-Stage Liver Disease; INR: international normalised ratio.

On univariate logistic regression, ascites, previous hepatic encephalopathy, bilirubin ≥3 mg/dL, albumin <3.0 g/dL, INR ≥1.5, creatinine ≥1.5 mg/dL, sodium <135 mmol/L, Child-Pugh class C and MELD score ≥15 were significantly associated with complications. After multivariable adjustment, albumin <3.0 g/dL, creatinine ≥1.5 mg/dL, sodium <135 mmol/L and MELD score ≥15 remained independent predictors. A MELD score ≥15 showed the strongest adjusted association with complications (adjusted OR 4.36, 95% CI 1.45-13.11; p=0.009) (Table 5).

**Table 5. Logistic regression analysis of predictors of complications among patients with chronic liver disease**

| Predictor | Unadjusted OR (95% CI) | p value | Adjusted OR (95% CI) | p value |
|---|---|---|---|---|
| Ascites | 4.42 (1.70-11.47) | 0.002 | 2.01 (0.63-6.42) | 0.236 |
| Previous hepatic encephalopathy | 3.85 (1.22-12.15) | 0.021 | 2.11 (0.54-8.22) | 0.282 |
| Bilirubin ≥3 mg/dL | 4.28 (1.63-11.25) | 0.003 | 1.77 (0.51-6.13) | 0.367 |
| Albumin <3.0 g/dL | 6.27 (2.30-17.11) | <0.001 | 3.48 (1.05-11.54) | 0.041 |
| INR ≥1.5 | 4.68 (1.77-12.38) | 0.002 | 1.93 (0.58-6.41) | 0.283 |
| Creatinine ≥1.5 mg/dL | 5.74 (1.69-19.48) | 0.005 | 3.81 (1.01-14.35) | 0.048 |
| Sodium <135 mmol/L | 4.91 (1.76-13.68) | 0.002 | 3.57 (1.07-11.91) | 0.038 |
| Child-Pugh class C | 5.16 (1.73-15.39) | 0.003 | 2.14 (0.59-7.79) | 0.249 |
| MELD score ≥15 | 7.08 (2.61-19.19) | <0.001 | 4.36 (1.45-13.11) | 0.009 |

OR: odds ratio; CI: confidence interval; INR: international normalised ratio; MELD: Model for End-Stage Liver Disease.

## DISCUSSION

In this prospective observational study, nearly half of the patients with CLD developed at least one major complication, demonstrating the substantial short-term clinical burden associated with advanced liver disease. Hepatic encephalopathy was the most frequent complication, followed by spontaneous bacterial peritonitis, acute kidney injury and variceal bleeding. Patients who experienced complications had clear evidence of poorer hepatic functional reserve and circulatory dysfunction, with higher bilirubin, INR, creatinine, Child-Pugh and MELD scores and lower albumin, platelet count and serum sodium. These findings are consistent with the recognised transition from compensated to decompensated cirrhosis, in which recurrent complications progressively worsen survival and increase healthcare utilisation.4,5

Alcohol-related liver disease was the predominant aetiology in the present cohort. This pattern is concordant with Indian multicentric data and a recent systematic review showing alcohol as the leading cause of adult cirrhosis in India, with an increasing contribution from metabolic liver disease.2,3 The male predominance and middle-aged presentation observed in our cohort also resemble many Indian hospital-based cirrhosis series. These epidemiological features are clinically relevant because alcohol-associated disease frequently presents at an advanced stage, often after the onset of portal-hypertensive or synthetic dysfunction.

The strongest independent predictor in this study was a MELD score of 15 or higher. MELD was originally developed as an objective model using bilirubin, creatinine and INR and has subsequently become a widely accepted marker of short-term mortality risk in advanced liver disease.6 Our findings support its broader bedside value for anticipating non-fatal complications as well. A higher Child-Pugh score was strongly associated with complications on univariate comparison but did not remain independently significant after adjustment, probably because several components of hepatic dysfunction overlap with MELD-related and biochemical predictors.

Hyponatraemia independently predicted complications. Reduced serum sodium in cirrhosis reflects impaired free-water excretion caused by advanced circulatory dysfunction and non-osmotic vasopressin activity.8 Previous work has shown that serum sodium adds prognostic information beyond MELD and is associated with hepatic encephalopathy, hepatorenal syndrome and mortality.7,12 Renal dysfunction was another independent predictor. Even modest rises in creatinine in cirrhosis are clinically important because renal impairment can progress rapidly, accompanies infections and haemodynamic instability, and is associated with adverse outcomes.10,13 The observed association between creatinine elevation and complications therefore has a strong pathophysiological basis.

Hypoalbuminaemia also remained independently associated with complications. Serum albumin reflects hepatic synthetic capacity but also participates in plasma oncotic pressure, ligand transport, antioxidant activity and circulatory homeostasis. Advanced hypoalbuminaemia therefore identifies patients with reduced physiological reserve. Trials of albumin in decompensated cirrhosis have further highlighted the relationship between albumin status, circulatory stability and complications.14 Taken together, the present findings suggest that combining simple laboratory variables with MELD scoring can identify patients requiring closer surveillance, prompt correction of precipitating factors and early specialist referral.

## LIMITATIONS

This study has limitations. It was conducted at a single tertiary-care centre with a modest sample size, which restricts external generalisability. Consecutive hospital-based recruitment could overrepresent patients with more advanced disease. Follow-up was short and did not evaluate long-term survival or transplantation. Some complications were relatively

infrequent, limiting precision of regression estimates. Residual confounding from nutrition, infections, medications and alcohol abstinence was not fully assessed.

## CONCLUSION

Among patients with chronic liver disease, major complications occurred frequently and were closely linked to worsening hepatic and circulatory function. Higher bilirubin, INR, creatinine, Child-Pugh and MELD scores, together with lower albumin, platelet count and serum sodium, characterised patients who developed complications. Hypoalbuminaemia, serum creatinine ≥1.5 mg/dL, hyponatraemia and MELD score ≥15 were independent predictors, with elevated MELD showing the strongest association. These findings support routine use of readily available clinical and biochemical parameters for early risk stratification. Patients with these high-risk features require closer surveillance, timely treatment of precipitating factors and appropriate specialist referral to reduce potentially preventable decompensation and adverse outcomes in routine tertiary-care practice.

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