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Clinical and Biochemical Predictors of Mortality in Patients With Decompensated Liver Cirrhosis

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Abstract: Background: Decompensated liver cirrhosis carries a high short-term mortality, driven by a complex interplay of hepatic dysfunction, renal impairment, and systemic complications. Identifying reliable clinical and biochemical predictors of mortality is critical for risk stratification and timely intervention.**Objectives:** To identify clinical and biochemical variables that independently predict in-hospital mortality in patients with decompensated liver cirrhosis and to compare the prognostic performance of Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores.**Methods:** This prospective observational study enrolled 35 consecutive patients with decompensated liver cirrhosis admitted to the Department of Medicine, Government Medical College, Satna (M.P.) over a period of 18 months (January 2023 – June 2024). Clinical parameters, biochemical investigations, CTP scores, and MELD scores were recorded at admission. Patients were followed until discharge or death. Statistical analysis included univariate and multivariate logistic regression.**Results:** Of 35 patients, 10 (28.6%) died during hospitalization. Non-survivors had significantly higher MELD scores (mean 26.4 ± 4.1 vs. 14.2 ± 3.8; $p < 0.001$), serum creatinine (3.1 ± 0.9 vs. 1.3 ± 0.4 mg/dL; $p < 0.001$), total bilirubin (12.6 ± 3.2 vs. 5.1 ± 2.4 mg/dL; $p < 0.01$), and INR (2.8 ± 0.6 vs. 1.7 ± 0.3; $p < 0.001$). Hepatic encephalopathy (grade III–IV) and hepatorenal syndrome were the strongest clinical predictors of mortality. MELD score showed superior discriminatory ability (AUROC 0.89) compared to CTP score (AUROC 0.78).**Conclusion:** MELD score, serum creatinine, serum bilirubin, INR, and presence of hepatic encephalopathy grade III–IV are significant independent predictors of in-hospital mortality in decompensated liver cirrhosis.

Keywords: Decompensated liver cirrhosis, mortality predictors, MELD score, Child-Pugh score, hepatic encephalopathy, hepatorenal syndrome

INTRODUCTION

Liver cirrhosis is the end stage of progressive hepatic fibrosis. Furthermore, it is a major cause of global morbidity and mortality resulting in ~1.03 million deaths.[1] The natural history of cirrhosis is one of a prolonged compensated phase followed by decompensation leading to ascites, variceal bleeding, hepatic encephalopathy (HE) or jaundice.[2] Survival post-first decompensation is <2 years vs >12 years in the compensated phase.[3]

In India, the major etiologies of cirrhosis are alcohol-related liver disease, and chronic hepatitis B and C. The burden of decompensated disease is significant in tertiary care settings.[4] In-hospital mortality in patients with decompensated liver cirrhosis is about 20-50% depending on the type and number of complications.[5] Gastrointestinal bleeding, spontaneous bacterial peritonitis (SBP), hepatic encephalopathy, and hepatorenal syndrome (HRS)

worsen prognosis independently and coexist; the latter two together worsen mortality risk.[6]

A scoring system which has been validated for prognostication in cirrhosis is Child-Turcotte-Pugh (CTP) score which uses bilirubin, albumin, prothrombin time, ascites and encephalopathy.[7] A more recent scoring system which uses serum creatinine, bilirubin and INR is MELD score.[7] Both scores have good accuracy but there is more evidence that MELD score may be superior for short-term mortality prediction, particularly in decompensated patients with renal dysfunction.[8] A further improvement, MELD-Na has been described which incorporates serum sodium in the prognostication. This is because sodium represents the severity of circulatory dysfunction and portal hypertension.[9]

Limited transplant facilities in central India must have a scant but not incomplete data compliance and literature. In such cases, proper identification of patients at high risk will help in prioritizing the patients for intensive monitoring and intervention and referral for liver transplant. As a result, clinical and laboratory exam (biochemical) predictors of in-hospital mortality in patients of decompensated liver cirrhosis were undertaken the present study. CTP and MELD scoring systems were compared for performance.

Material and Methods

Study Design and Setting

This was a prospective observational study conducted in the Department of Medicine, Government Medical College, Satna (M.P.), India over 18 months from January 2023 to June 2024.

Participant Selection

Inclusion Criteria: Adult patients (≥18 years) with established liver cirrhosis (diagnosed clinically, biochemically, or by imaging) admitted with at least one feature of decompensation — ascites, variceal bleeding, hepatic encephalopathy, or jaundice (serum bilirubin >3 mg/dL) — were enrolled consecutively.

Exclusion Criteria: Patients with hepatocellular carcinoma, non-cirrhotic portal hypertension, pregnancy, concomitant terminal malignancy, or those who refused consent were excluded.

A total of 35 patients meeting inclusion criteria were enrolled after obtaining written informed consent. The study was approved by the Institutional Ethics Committee.

Data Collection

At admission, the following data were recorded:

- Demographic details: Age, sex, BMI
- Etiology of cirrhosis: Alcohol, viral hepatitis B or C, cryptogenic, autoimmune
- Clinical features: Ascites (grade 1–3 by ultrasonography), hepatic encephalopathy (West Haven Criteria grade I–IV), variceal bleeding, SBP, hepatorenal syndrome
- Biochemical parameters: Hemoglobin (Hb), total leucocyte count (TLC), platelet count, serum bilirubin (total and direct), serum albumin, AST, ALT, alkaline phosphatase (ALP), serum creatinine, serum sodium, serum potassium, blood urea nitrogen (BUN), prothrombin time / INR
- Scoring: CTP score (Class A/B/C) and MELD score (calculated as $3.78 \times \ln[\text{bilirubin mg/dL}] + 11.2 \times \ln[\text{INR}] + 9.57 \times \ln[\text{creatinine mg/dL}] + 6.43$)

The study was conducted at Government Medical College, Satna, Madhya Pradesh.

The affiliation of the first author reflects his institutional appointment and does not indicate patient recruitment from the Institute of Liver and Biliary Sciences, New Delhi.

Outcome

The primary outcome was in-hospital mortality (death before discharge). Patients were categorized as survivors and non-survivors for comparative analysis.

Statistical Analysis

Categorical data were expressed as frequencies and percentages; continuous data as mean ± standard deviation (SD). Comparison between survivors and non-survivors was performed using the independent samples *t*-test for continuous variables and chi-square or Fisher's exact test for categorical variables. Univariate logistic regression was applied to identify potential predictors, followed by multivariate logistic regression for independent predictors. Receiver operating characteristic (ROC) curves were plotted for MELD and CTP scores; areas under the curve (AUROC) were compared using DeLong's method. A p-value < 0.05 was considered statistically significant. Statistical analysis was performed using SPSS software version 26.0 (IBM Corp., USA).

Results

Of 35 enrolled patients, 10 (28.6%) died during hospitalization (non-survivors) and 25 (71.4%) were discharged (survivors).

Table 1: Baseline Demographic and Etiological Profile of Study Patients (n = 35)

Characteristic	Total (n = 35)	Survivors (n = 25)	Non-Survivors (n = 10)	p-value
Age (years), mean ± SD	48.3 ± 11.6	46.1 ± 10.8	53.4 ± 12.9	0.08
Sex (Male), n (%)	27 (77.1%)	19 (76%)	8 (80%)	0.78

BMI (kg/m²), mean ± SD	22.4 ± 3.1	23.1 ± 2.9	20.6 ± 3.4	0.04
Etiology of Cirrhosis				
Alcohol-related	18 (51.4%)	12 (48%)	6 (60%)	0.49
Hepatitis B	8 (22.9%)	6 (24%)	2 (20%)	0.79
Hepatitis C	4 (11.4%)	3 (12%)	1 (10%)	0.86
Cryptogenic	5 (14.3%)	4 (16%)	1 (10%)	0.63
Duration of illness (months)	28.6 ± 14.2	30.1 ± 13.8	24.4 ± 15.1	0.27

Alcohol-related liver disease was the most common etiology (51.4%), consistent with the pattern seen in central Indian tertiary care hospitals. Non-survivors were significantly younger in BMI (p = 0.04), reflecting greater nutritional depletion and sarcopenia in those who died.

Table 2: Clinical Features and Complications at Admission

Clinical Feature	Total (n = 35)	Survivors (n = 25)	Non-Survivors (n = 10)	p-value
Ascites (grade 2–3), n (%)	30 (85.7%)	20 (80%)	10 (100%)	0.14
Variceal bleeding, n (%)	14 (40%)	9 (36%)	5 (50%)	0.42
Hepatic Encephalopathy (any), n (%)	22 (62.9%)	12 (48%)	10 (100%)	0.003
HE Grade III–IV, n (%)	10 (28.6%)	2 (8%)	8 (80%)	<0.001
Spontaneous Bacterial Peritonitis, n (%)	11 (31.4%)	6 (24%)	5 (50%)	0.10
Hepatorenal Syndrome, n (%)	8 (22.9%)	1 (4%)	7 (70%)	<0.001
Jaundice (bilirubin >3 mg/dL), n (%)	28 (80%)	18 (72%)	10 (100%)	0.06
Gastrointestinal bleeding, n (%)	14 (40%)	9 (36%)	5 (50%)	0.42

Hepatic encephalopathy (HE) grade III–IV was present in 80% of non-survivors compared to only 8% of survivors (p < 0.001). Hepatorenal syndrome was noted in 70% of non-survivors versus 4% of survivors (p < 0.001), underscoring its lethal significance in decompensated cirrhosis.[6]

Table 3: Biochemical Parameters at Admission — Survivors vs. Non-Survivors

Parameter	Survivors (n = 25)	Non-Survivors (n = 10)	p-value
Hemoglobin (g/dL)	9.6 ± 1.8	8.4 ± 1.6	0.06
Total Leucocyte Count (×10³/µL)	7.2 ± 2.9	10.8 ± 4.1	0.003
Platelet Count (×10³/µL)	88.4 ± 32.6	61.3 ± 28.1	0.01
Serum Bilirubin – Total (mg/dL)	5.1 ± 2.4	12.6 ± 3.2	<0.001
Serum Albumin (g/dL)	2.8 ± 0.5	2.1 ± 0.4	<0.001
AST (IU/L)	78.4 ± 31.2	114.6 ± 48.3	0.006
ALT (IU/L)	52.3 ± 22.6	74.8 ± 30.1	0.02
Alkaline Phosphatase (IU/L)	168.2 ± 54.8	212.4 ± 68.1	0.04
Serum Creatinine (mg/dL)	1.3 ± 0.4	3.1 ± 0.9	<0.001
Blood Urea Nitrogen (mg/dL)	22.4 ± 9.6	58.6 ± 18.3	<0.001
Serum Sodium (mEq/L)	132.6 ± 5.2	124.8 ± 6.1	<0.001
INR	1.7 ± 0.3	2.8 ± 0.6	<0.001
Prothrombin Time (seconds)	18.4 ± 3.1	26.8 ± 4.6	<0.001

Key biochemical markers of poor prognosis included elevated serum creatinine, raised total bilirubin, low albumin, prolonged INR, thrombocytopenia, and hyponatremia — all significantly different between survivors and non-survivors.

Table 4: Scoring Systems — CTP and MELD Score Comparison

Score / Category	Survivors (n = 25)	Non-Survivors (n = 10)	p-value
CTP Score (mean ± SD)	8.6 ± 1.4	11.4 ± 1.2	<0.001
CTP Class A (score 5–6), n (%)	2 (8%)	0 (0%)	—
CTP Class B (score 7–9), n (%)	16 (64%)	2 (20%)	—
CTP Class C (score 10–15), n (%)	7 (28%)	8 (80%)	0.005
MELD Score (mean ± SD)	14.2 ± 3.8	26.4 ± 4.1	<0.001
MELD < 15, n (%)	18 (72%)	1 (10%)	—
MELD 15–24, n (%)	6 (24%)	3 (30%)	—

MELD ≥ 25, n (%)	1 (4%)	6 (60%)	<0.001
AUROC — MELD score	—	—	0.89
AUROC — CTP score	—	—	0.78

MELD score ≥ 25 was associated with 60% mortality in this cohort. The AUROC for MELD (0.89) was superior to that of CTP (0.78) for predicting in-hospital mortality, consistent with published evidence.

Table 5: Multivariate Logistic Regression — Independent Predictors of In-Hospital Mortality

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
MELD Score ≥ 25	14.6	2.8 – 76.2	0.001
Serum Creatinine > 2.0 mg/dL	9.4	1.9 – 46.3	0.006
HE Grade III–IV	8.2	1.6 – 42.5	0.011
Serum Sodium < 125 mEq/L	6.8	1.3 – 35.6	0.024
INR > 2.5	5.9	1.1 – 31.8	0.038
Serum Bilirubin > 10 mg/dL	4.7	0.9 – 24.6	0.067
Serum Albumin < 2.0 g/dL	3.8	0.7 – 20.4	0.118

On multivariate analysis, MELD score ≥ 25 (OR 14.6; 95% CI 2.8–76.2; p = 0.001), serum creatinine > 2.0 mg/dL (OR 9.4; p = 0.006), HE grade III–IV (OR 8.2; p = 0.011), hyponatremia (serum sodium < 125 mEq/L; OR 6.8; p = 0.024), and INR > 2.5 (OR 5.9; p = 0.038) emerged as independent predictors of in-hospital mortality.

DISCUSSION

The current study evaluated the clinical and biochemical predictors of in-hospital mortality in a study group of 35 patients with decompensated liver cirrhosis admitted to a tertiary care teaching hospital in central India. The in-hospital mortality rates were 28.6% which is comparable to previous studies. Arora et al found a 30-day mortality of around 24-30% in patients with decompensated cirrhosis in Indian tertiary care.[4] In like manner, Saad et al. reported that mortality was around 20% to 40% depending upon complications present.[6]

In the present study, 80% of non-survivors had HE grade III-IV, but only 8% of survivors had it (p < 0.001), which was among the best clinical predictors. According to Hussain et al. (2020), there is 100% mortality recorded for patients with grade IV HE. Hussain et al. (2020) also mentioned 100% mortality in their cohort of hospitalized cirrhotics, who had grade IV HE.[5] Cerebral edema, inflammation, increased ammonia load, and multiple organ dysfunction are all implicated in hepatic failure. The strong predictors of in-hospital mortality in cirrhosis were gastrointestinal bleeding and hepatic encephalopathy. This was revealed by a study published in 2024 by Saad et al. The current study found that 70% of non-survivors had HRS versus 4% in survivors (p<0.001). [6] HRS is a type of functional AKI that is very serious, caused by splanchnic vasodilatation and decrease in effective arterial blood volume and renal vasoconstriction.

The results of the current study are in agreement with a systematic review and meta-analysis by Singh et al., published in IJMPR in 2024. [2] It showed that renal impairment is one of the strongest contributors to mortality from cirrhosis with ascites, independent of severity of hepatic dysfunction. The scarcity of renal replacement therapy and liver transplantation options in our country increased the prognostic significance of hepatorenal syndrome (HRS) furthermore, the MELD score was found to be a better predictor of in-hospital mortality with an AUROC of 0.89 compared to AUROC of 0.78 for CTP score. Many studies that support this have been published. Kamath et al. validated the MELD score for the prediction of 3-month mortality after TIPS insertion with AUROC of 0.84 vs 0.70 of the Child-Pugh score.[9] As suggested by the study of Verma et al (2023), the MELD score being an AUROC value of 0.87 is a good predictor of thirty-day mortality in decompensated cirrhosis in an Indian tertiary care setting.[8] The numbers we find here closely match those from our study. In the present cohort study, in-hospital mortality was observed to be thirty per cent in patients with a MELD score of twenty-five or more. In contrast, in-hospital mortality was four per cent for patients with MELD < 15. Consequently, these two groups can be distinctly categorized into high-risk groups and low-risk groups. Although it is easier to use clinically, the CTP score applies subjective parameters (ascites, degree of encephalopathy) that allow inter-observer variability. Still, it may be useful in circumstances where serum creatinine measurement is not uniformly available. Ahuja et al. recently discovered that mortality prediction by CTP score revealed AUROC of 0.85 while MELD revealed 0.78 at 3 months time frame.[7] In other terms, CTP may keep similar prognostic value in some settings. Yet, the present results vary. The study population, length of follow-up, and higher frequency of renal impairment in the present cohort may explain this difference. It is likely that the creatinine-inclusive MELD formula is preferred due to a higher prevalence of renal dysfunction.

According to a single biochemical variable, the best predictors of clinical outcome were serum creatinine > 2.0 mg/dL, INR > 2.5, serum sodium < 125 mEq/L

and total bilirubin > 10 mg/dL. Low sodium level in the blood has been added as an independent prognostic marker over and above MELD. MELD-Na, which includes serum sodium, has better predictive accuracy for waitlist mortality in candidates for liver transplant and may also improve short-term prognosis in hospitalised patients. Progressive hepatic synthetic failure characterized by severe hypoalbuminemia (< 2.0 g/dL) increases the risk of infection [10] Thrombocytopenia is a good surrogate marker of the degree of portal hypertension and secondary hypersplenism according to Giannini et al. [11].

The mean TLC in non survivors was found to be twice as much as that in survivors (10.8 ± 4.1 7.2 ± 2.9 $\times 10^3/\mu L$; p = 0.003). There could be a concurrent infection, either SBB or pneumonia, or it may just be a systemic inflammatory response. Both of these were shown to degrade the outcome. Many cases ACLF though the incidence of bacterial infection in a patient of decompensated liver disease is high.[12] The mortality rates are found to be very high in the short-term between 45-70%. Liver disease caused by alcohol makes up 51.4% of cases. The pattern observed in central India was comparable to this. 77.1% of total cases are males owing to more alcohol consumption. In this cohort, age was not a significant predictor of mortality but non-survivors were older than survivors (53.4 years for non-survivors vs 46.1 years for survivors; p = 0.083). The lower BMI found in non-survivors (20.6 versus 23.1 kg/m2; p=0.04) is in keeping with emerging data on sarcopenia and nutritional depletion as determinants of cirrhosis prognosis.1 There are numerous limitations to this study. The 35 patient sample size limits the power of the study. This single-centre study from a tertiary care Hospital in Central India may have referral bias. Failure to obtain follow-up data post discharge for this patient. Due to resource constraints, we were unable to conduct ascitic fluid cultures for increasing suspicion of spontaneous bacterial peritonitis and HVPG measurement to quantify portal hypertension in all patients.

CONCLUSION

1. In-hospital mortality for decompensated liver cirrhosis was 28.6%. Independent mortality risk factors for in-hospital mortality are MELD score ≥ 25 serum creatinine > 2.0 mg/dL hepatic encephalopathy grade III–IV serum sodium < 125 mEq/L INR > 2.5. The MELD score outperformed CTP score which is shown by MELD score having AUROC 0.89 and AUROC 0.78 for CTP score. According to their findings, patients at risk should receive care escalation and may benefit from being referred for evaluation for liver transplantation using MELD score at admission routinely. Larger multicentric prospective studies with adequate follow up are needed to further validate these findings in the Indian context.

2. **Conflict of Interest**
3. The authors declare no conflict of interest.
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