



To Compare between CTP, MELD, MELD-Na, MELD + HDLc, RDW, and RDW to Platelet Ratio as a Predictor of Short-term Mortality in Cirrhosis of Liver

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ABSTRACT

Background: Liver cirrhosis indicates inflammation, necrosis, as well as fibrosis, resulting in progressively decreasing liver function. As the disease advances from a compensated to a decompensated stage, patients experience severe clinical complications, that result in elevated mortality, as well as morbidity, rates. Accurate predicting short-term mortality is essential for making clinical decisions, particularly when it comes to liver transplantation (LT). Several scores, encompassing model for end-stage liver disease (MELD), Child–Turcotte–Pugh (CTP), as well as their variants, along with specific biomarkers such as red cell distribution width (RDW) alongside RDW to platelet ratio (RPR), have been proposed for assessing these patients' prognosis. However, comparative effectiveness of these scoring systems in predicting outcomes remains underexplored.

Methods: This study involved a cohort of participants diagnosed with cirrhosis, who were evaluated to identify the most reliable predictors of 30-day mortality. The study compared the efficacy of multiple scoring systems, including CTP, MELD, model for end-stage liver disease-sodium (MELD-Na), model for end-stage liver disease-high-density lipoprotein cholesterol (MELD-HDLc), RDW, and RPR, by analyzing their correlation with patient outcomes. Data were collected on demographic profiles, clinical findings, and laboratory markers to calculate these scores and assess their predictive accuracy.

Results: The study found that among the various scores, the MELD as well as MELD-Na scores demonstrated the highest accuracy predicting 30-day mortality in liver cirrhosis patients. Alcohol emerged as the predominant etiology of cirrhosis, and there was a significant male predominance in the cohort. The results were consistent with existing literature, confirming the reliability of MELD alongside MELD-Na as stronger prognostic tools compared to the CTP score and other markers.

Conclusion: MELD along with MELD-Na scores constitute reliable indicators of mortality over the short term in individuals with cirrhosis and should be preferred in practice for assessing the need for LT and other critical interventions. These findings underscore the importance of using evidence-based scoring systems to improve patient management and outcomes in liver cirrhosis, a condition with a high global mortality burden.

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INTRODUCTION

Cirrhosis encompasses inflammation, necrosis, as well as fibrosis resulting from multiple conditions. Liver cirrhosis is initiated by hepatocyte necrosis and subsequent regeneration, leading to hepatic sinusoid capillarization along with fibrosis. Reduced hepatic parenchyma, blood flow alterations, alongside development of portosystemic shunts contribute to various complications of cirrhosis, including hepatocellular carcinoma. The progression of cirrhosis unfolds in two distinct phases: asymptomatic compensated phase, succeeded by decompensated phase characterized by various clinical manifestations, including ascites, coagulation abnormalities, encephalopathy, bleeding, and jaundice.¹ Decompensation marks a significant turning point, leading to a faster progression toward mortality or

necessity for liver transplantation (LT). Furthermore, additional complications encompassing acute kidney injury (AKI), rebleeding, hepatorenal syndrome (HRS), portopulmonary hypertension (PoPH), hepatopulmonary syndrome (HPS), cirrhotic cardiomyopathy (CCM), and bacterial infections can expedite disease progression, particularly in the decompensated stage. Cirrhosis's shift from compensated to decompensated manifests at an annual rate of approximately 5–7%. After decompensation sets in, cirrhosis evolves into a systemic condition and life expectancy drastically diminishes. Consequently, average lifespan diminishes from nearly 12 year in cirrhosis with compensation to approximately 2 year in decompensated cirrhosis. Clinical picture of decompensated cirrhosis is attributed to the hemodynamic disturbances resulting from peripheral arterial vasodilation, particularly

in the splanchnic circulatory region.² Liver disease results in 2 million fatalities each year, around 4% of worldwide deaths, with most liver-related deaths occurring in men. Cirrhosis, as well as hepatocellular cancer, were main causes, primarily due to alcohol, viral hepatitis, as well as nonalcoholic fatty liver disease (NAFLD). Affecting 25% of adults in Europe as well as America, NAFLD is the second most common contributor to end-stage liver disease as well as LT. Deaths from hepatic viruses have declined due to hepatitis B virus (HBV) vaccination and effective hepatitis C virus (HCV) treatments. In high-income countries, conditions like primary sclerosing cholangitis (linked to higher cancer risk), primary biliary cholangitis, and autoimmune hepatitis are more common.³ Early intervention is essential to halt the progression of cirrhosis and delay the onset of liver function decompensation.⁴ A straightforward, alongside trustworthy, approach must be taken to evaluate these patients' mortality risk. Traditionally, cirrhosis prognosis has been evaluated using Child–Turcotte–Pugh (CTP) or model for end-stage liver disease (MELD) scoring systems.⁵ Since 2002, MELD score—which relies on creatinine, bilirubin, as well as international normalized ratio (INR)—has been shown to be a valid indicator of early death.⁶ An expansion of classic MELD, model for end-stage liver disease-sodium (MELD-Na) score adds serum sodium (S Na) levels to the equation.

Recent research has demonstrated that MELD-Na enhances precision of short-term

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death prediction in patients with cirrhosis.⁷ Other prognostic markers gaining attention as minimally invasive techniques for evaluating liver disease include model for end-stage liver disease-high-density lipoprotein cholesterol (MELD-HDLc) scores, albumin-bilirubin (ALBI) score, red cell distribution width (RDW), RDW to platelet ratio (RPR), as well as fibrosis-4 (FIB-4) score.⁸

Given the evolving landscape of prognostic indicators in cirrhosis, comparing the predictive accuracy of these various scoring systems and biomarkers for short-term (30-day) mortality is critical. This comparative study aims to identify the most reliable predictors, facilitating improved patient management and potentially guiding clinical decision-making regarding the urgency of LT or other life-saving treatments.

MATERIALS AND METHODS

This hospital-based prospective observational investigation has been carried out in Northern India at the Department of Medicine in cooperation with the Department of Medical Gastroenterology. Following approval and clearance from the Institutional Ethics Committee and the acquisition of written consent, patients who satisfied the inclusion criteria were subsequently enlisted. The study spanned a duration of 1 year. Inclusion criteria—adults of 14–65 years old discovered with liver cirrhosis, as well as providing written informed consent. Exclusion criteria—individuals who declined to give informed consent voluntarily. The patient's medical history has been employed to determine the cirrhosis diagnosis, clinical features (e.g., ascites, jaundice, gastrointestinal bleeding, hepatomegaly, hepatic encephalopathy, and splenomegaly), elevated AST/ALT levels, hyperbilirubinemia, and supportive ultrasound findings. Patients suffering from tuberculosis (TB)-related ascites, malignancy, or nonhepatocellular carcinoma were excluded. Scores such as CTP, MELD, MELD-Na, MELD + HDLc, RDW, and RPR have been determined utilizing laboratory results obtained 24 hours after being admitted to the hospital. Outcomes were assessed at 30 days and classified as either "survived" or "deceased." CTP score comprised three categorical indicators (encephalopathy, ascites, as well as INR) alongside two continuous ones (bilirubin as well as albumin). Class A (5–6 points), class B (7–9 points), and class C (10–15 points) are three categories into which it falls. The following formulas can be used for related calculations:

$\text{MELD score} = 3.78 \times \ln [\text{serum bilirubin (mg/dL)}] + 11.2 \times \ln (\text{INR}) + 9.57 \times \ln [\text{serum creatinine (mg/dL)}] + 6.43$

$\text{MELD-Na score} = \text{MELD score} + 1.59 \times (135 - \text{Na})$, with Na values capped at a maximum of 135 mmol/L and a minimum of 120 mmol/L. For MELD + HDLc, HDL was added to the MELD scores. The AUC for CTP, MELD, MELD + HDLc, MELD-Na, RDW, and RPR was calculated. Predictive values of MELD, CTP, RDW, MELD + HDLc, MELD-Na, and RPR regarding 30-day mortality were assessed and compared.

Outcome measures, including prevalence of cirrhosis in terms of age, sex, urban or rural dwellers, number of patients in each CTP class, mean scores in terms of MELD, MELD-Na, MELD + HDLc, RDW, RPR, mean laboratory values at time of admission, and incidence in relation to outcomes at the end of 30 days (survival or death), were calculated.

Statistical Analysis

Data collected during the study were organized and entered into Microsoft Excel 2021 (Office 2021 package). Statistical analysis was carried out employing IBM SPSS software, version 24.0 (Chicago, Illinois, IBM Corp). Descriptive statistics were employed to summarize data. Categorical variables were represented as percentages alongside proportions, whereas mean and standard deviation (SD) were utilized for expressing continuous variables. Associations between variables were analyzed employing the Chi-squared test. In order to compare means of two distinct groups, an unpaired *t*-test was applied. ANOVA, on the contrary, was employed for assessing means of continuous variables among several groups. Receiver operating characteristics (ROC) analysis of prognostic scores was done, and their area under the ROC curve (AUROC), sensitivity, specificity, odds ratio, and *p*-value were calculated and compared.

RESULTS

A total of 150 patients with cirrhosis were incorporated into the investigation according to the criteria for inclusion and exclusion, and they were followed up for 30 days from their date of admission to compare various prognostic models as a mortality predictive score. Participants in the investigation were between 18 and 65 years old. The mean age was 46.41 ± 11.48 . The median age was 47.00. Out of 150 patients enrolled, 31 (20.7%) were female participants, and 119 (79.3%) were male participants. The male-to-female participant ratio was 3.8:1. Out of 150 patients enrolled, 43 (31.6%) were urban residents, and 93 (68.4%) were rural residents. About 68 (45.6%)

participants had a history of significant alcohol intake, 22 participants (14.8%) were hepatitis B reactive, 20 participants (13.4%) were hepatitis C reactive, 17 participants (11.4%) had NASH/NAFLD, 11 participants (8.1%) had an etiology under evaluation, 8 participants (5.4%) had autoimmune hepatitis, 2 participants (1.3%) had Wilson's disease, and 1 participant had cryptogenic cirrhosis.

Of the 68 patients who were alcoholic, 19 patients (28.8%) had a period of alcohol consumption of <20 years, and 49 patients (71.2%) had a history of alcohol consumption exceeding 20 years. Twenty-three patients (34.2%) had an amount of liquor (gm/day) <80, and 45 patients (66.2%) had an amount of liquor (gm/day) >80.

Out of 150 patients enrolled, 7 patients (4.7%) had no ascites, 39 patients (26.0%) had slight ascites, and 104 patients (69.3%) had moderate/severe ascites. Out of 150 patients enrolled, 11 patients (7.3%) had no encephalopathy, 24 patients (16.0%) had grade I encephalopathy, 59 patients (39.3%) had grade II encephalopathy, 43 patients (28.7%) had grade III encephalopathy, and 13 patients (8.7%) had grade IV encephalopathy.

An entire group of 150 participants has been selected for this research. The mean Hb level was 8.08 ± 2.30 gm/dL, with a range of 3.6–15.2 gm/dL. The mean total leukocyte count (TLC) was $10,042.71 \pm 7,377.58/\text{mm}^3$, ranging from 1180 to 46400/mm³. The mean platelet count was 1.05 ± 0.75 lakhs, with values ranging from 0.12 to 6.7 lakhs. The mean total bilirubin level was 4.98 ± 6.01 mg/dL, with a range of 0.14–37 mg/dL. The mean serum glutamic-oxaloacetic transaminase (SGOT) level was 100.56 U/L (range: 1.05–888 U/L), and the mean serum glutamic-pyruvic transaminase (SGPT) level was 66.76 U/L (range: 11–696 U/L). Serum albumin levels averaged 2.71 gm/dL, with a range of 1.5–3.8 gm/dL, while serum creatinine levels had a mean of 2.04 mg/dL, ranging from 0.48 to 11.6 mg/dL. The mean INR was 2.10 ± 0.90 , with a range from 0.89 to 4.9. Sodium levels had a mean of 130.16 ± 5.78 mEq/L, with a range from 111 to 145 mEq/L. The mean HDL level was 22.89 ± 10.74 mg/dL, ranging from 5 to 47 mg/dL.

All participants were followed for 30 days from admission to assess outcomes, categorized as "alive" or "expired." Of the 150 patients, 121 (80.7%) survived, while 29 (19.3%) died within 30 days. Among those who died, the mean time to mortality was 6.28 ± 3.76 days, ranging from 1 to 16 days.

Regarding disease severity, 0.7% of patients were designated as CTP class A, 23.3% as class B, and 76% as class C. The mean MELD + HDLc score was 45.86 ± 9.22 , with a median of 46.50 (range: 18–67). The mean MELD

score was 23.11 ± 10.16 , with a median of 21.00 (range: 6–40). The mean MELD-Na score was 24.69 ± 9.66 , with a median of 24.00 (range: 6–40). RDW had a mean of $16.98 \pm 2.35\%$ and a median of 16.50% (range: 12–27.32%). The mean RPR was 23.69 ± 18.87 , with a median of 17.80 (range: 2.5–136).

The Chi-squared test indicated a substantial correlation between gender and the etiology of cirrhosis ($\chi^2 = 62.560, p < 0.001$). Males predominantly had alcohol-related cirrhosis (68 cases), hepatitis B (20 cases), and Wilson's disease (2 cases), while females more frequently had cirrhosis due to hepatitis C (9 cases), NASH/NAFLD (5 cases), autoimmune conditions (7 cases), and cryptogenic causes (1 case).

Mean sodium levels were markedly elevated in the survivor cohort (131.32 mEq/L) compared to those who expired (125.31 mEq/L, $p < 0.001$). Likewise, HDL levels were

markedly elevated in survivors (mean: 25.48 mg/dL) compared to deceased patients (mean: 12.07 mg/dL, $p < 0.001$). RDW rose substantially in the expired group (mean: 18.33%) compared to survivors (mean: 16.65%, $p < 0.001$).

Variables substantially correlated with 30-day mortality ($p < 0.05$) included encephalopathy, hemoglobin, TLC, platelet count, total bilirubin, direct bilirubin, SGOT, SGPT, serum creatinine, blood urea, prothrombin time (PT), INR, sodium, HDL, CTP class, MELD + HDLc, MELD, MELD-Na, RDW, and RPR (Table 1).

Receiver Operating Characteristics Analysis of Prognostic Models

For the CTP score, although the sensitivity was found to be 100%, the specificity was low at only 0.8%. The diagnostic accuracy was 20%, and the p -value was 0.623, making it a poor

prognostic marker for mortality within 30 days for cirrhosis patients.

Receiver Operating Characteristics Analysis of MELD

Area under the ROC curve of MELD predicting outcomes: expired vs alive came out to be 0.978 (95% CI: 0.96–0.996), hence exhibiting superior diagnostic efficacy. Results were statistically significant ($p < 0.001$). At the MELD cutoff of ≥ 30 , mortality predicted with sensitivity of 100% alongside specificity of 88% (Table 2).

Receiver Operating Characteristics Analysis of MELD-Na

Area under the ROC curve of MELD-Na predicting outcomes of expired vs alive came out to be 0.977 (95% CI: 0.958–0.996), indicating exceptional diagnostic efficacy. Results were statistically significant ($p < 0.001$).

Table 1: Showing parameters significantly associated with outcome

Parameters	Outcome		p-value
	Alive (n = 121)	Expired (n = 29)	
Age (years)	46.48 \pm 11.34	46.10 \pm 12.25	0.881
Gender			0.126
Residence			0.830
Alcohol intake (yes)	43 (43.4%)	13 (59.1%)	0.183
CLD etiology			0.384
Type of liquor			1.000
Duration of alcohol intake (years)			0.753
Amount of liquor (gm/day)			0.782
Ascites			0.275
Encephalopathy			<0.001
None	11 (9.1%)	0 (0.0%)	
Grade I	24 (19.8%)	0 (0.0%)	
Grade II	55 (45.5%)	4 (13.8%)	
Grade III	28 (23.1%)	15 (51.7%)	
Grade IV	3 (2.5%)	10 (34.5%)	
Hemoglobin (gm/dL)	8.34 \pm 2.36	7.00 \pm 1.68	0.001
TLC (/mm ³)	9046.44 \pm 6873.13	14199.55 \pm 8062.00	0.001
Platelet count (lakhs)	1.17 \pm 0.78	0.54 \pm 0.22	<0.001
Total bilirubin (mg/dL)	3.59 \pm 5.02	10.79 \pm 6.37	<0.001
Direct bilirubin (mg/dL)	2.25 \pm 3.19	7.73 \pm 4.32	<0.001
SGOT (U/L)	95.02 \pm 109.98	123.68 \pm 34.50	<0.001
SGPT (U/L)	61.11 \pm 97.88	90.32 \pm 36.10	<0.001
Serum albumin (gm/dL)	2.74 \pm 0.45	2.56 \pm 0.34	0.050
Serum protein (gm/dL)	6.42 \pm 1.03	6.35 \pm 0.86	0.743
Serum creatinine (mg/dL)	1.76 \pm 1.64	3.21 \pm 1.33	<0.001
B. urea (mg/dL)	61.65 \pm 44.02	145.97 \pm 62.24	<0.001
PT (s)	23.57 \pm 8.62	36.91 \pm 11.24	<0.001
INR	1.81 \pm 0.65	3.30 \pm 0.78	<0.001
Sodium (mEq/L)	131.32 \pm 5.28	125.31 \pm 5.29	<0.001
HDL (mg/dL)	25.48 \pm 10.15	12.07 \pm 4.72	<0.001

At the MELD-Na cutoff of ≥ 32 , mortality was predicted with sensitivity of 97% as well as specificity of 89% (Table 3).

Receiver Operating Characteristics Analysis of MELD-HDLc

Area under the ROC curve of MELD + HDLc predicting outcomes of expired vs alive came out to be 0.627 (95% CI: 0.53–0.724), indicating moderate diagnostic efficacy. Results were statistically significant ($p = 0.034$). At the MELD + HDLc cutoff of ≥ 41 , mortality was predicted with sensitivity of 97%, as well as specificity of 32% (Table 4).

Table 2: ROC curve analysis showing diagnostic performance of MELD in predicting mortality ($n = 150$)

Parameter	Value (95% CI)
Cutoff (p -value)	≥ 30 (<0.001)
AUROC	0.978 (0.96–0.996)
Sensitivity	100.0% (88–100)
Specificity	88.4% (81–94)
Positive predictive value	67.4% (51–81)
Negative predictive value	100.0% (97–100)
Diagnostic accuracy	90.7% (85–95)
Positive likelihood ratio	8.64 (5.28–14.14)
Negative likelihood ratio	0 (0–NaN)
Diagnostic odds ratio	Inf (NaN–Inf)

Table 3: ROC curve analysis showing diagnostic performance of MELD-Na in predicting mortality ($n = 150$)

Parameter	Value (95% CI)
Cutoff (p -value)	≥ 32 (<0.001)
AUROC	0.977 (0.958–0.996)
Sensitivity	96.6% (82–100)
Specificity	89.3% (82–94)
Positive predictive value	68.3% (52–82)
Negative predictive value	99.1% (95–100)
Diagnostic accuracy	90.7% (85–95)
Positive likelihood ratio	8.99 (5.35–15.09)
Negative likelihood ratio	0.04 (0.01–0.27)
Diagnostic odds ratio	232.62 (29.18–1854.5)

Receiver Operating Characteristics Analysis of RDW

Area under the ROC curve of RDW (%) predicting outcomes of expired vs alive came out to be 0.804 (95% CI: 0.733–0.874), indicating exceptional diagnostic efficacy. Results were statistically significant ($p < 0.001$). At the RDW (%) cutoff of ≥ 17.5 , mortality was predicted with sensitivity of 90%, as well as specificity of 72% (Table 5).

Receiver Operating Characteristics Analysis of RPR

Area under the ROC curve of RPR predicting outcomes of expired vs alive came out to be

Table 4: ROC curve analysis showing diagnostic performance of MELD + HDLc in predicting mortality ($n = 150$)

Parameter	Value (95% CI)
Cutoff (p -value)	≥ 41 (0.034)
AUROC	0.627 (0.53–0.724)
Sensitivity	96.6% (82–100)
Specificity	31.7% (23–41)
Positive predictive value	25.5% (18–35)
Negative predictive value	97.4% (87–100)
Diagnostic accuracy	44.3% (36–53)
Positive likelihood ratio	1.41 (1.23–1.63)
Negative likelihood ratio	0.11 (0.02–0.76)
Diagnostic odds ratio	12.98 (1.7–98.94)

Table 5: ROC curve analysis showing diagnostic performance of RDW (%) in predicting mortality ($n = 150$)

Parameter	Value (95% CI)
Cutoff (p -value)	≥ 17.5 (<0.001)
AUROC	0.804 (0.733–0.874)
Sensitivity	89.7% (73–98)
Specificity	71.9% (63–80)
Positive predictive value	43.3% (31–57)
Negative predictive value	96.7% (91–99)
Diagnostic accuracy	75.3% (68–82)
Positive likelihood ratio	3.19 (2.34–4.35)
Negative likelihood ratio	0.14 (0.05–0.42)
Diagnostic odds ratio	22.18 (6.3–78.11)

0.824–0.935 (95% CI), indicating exceptional diagnostic efficacy. Results were statistically significant ($p < 0.001$). At the RPR cutoff of ≥ 23.7 , mortality was predicted with sensitivity of 90%, alongside specificity of 78% (Table 6).

MELD, MELD-Na, RPR, RDW (%), and MELD + HDLc Significantly Predicted Mortality in the Enrolled Patients

In the study, MELD, MELD-Na, RPR, RDW (%), and MELD + HDLc have been acknowledged as substantial mortality predictors. No significant difference was observed between the diagnostic performance of MELD and MELD-Na. However, MELD demonstrated significantly better diagnostic accuracy than RPR (DeLong's Test, $p < 0.001$), RDW (%) (AUC = 0.804, $p < 0.001$), and MELD + HDLc (AUC = 0.627, $p < 0.001$), with an AUC of 0.978. Similarly, MELD-Na (AUC = 0.977) outperformed RPR (AUC = 0.880, $p < 0.001$), RDW (%) (AUC = 0.804, $p < 0.001$), and MELD + HDLc (AUC = 0.627, $p < 0.001$) in terms of diagnostic performance. When comparing RPR and RDW (%), RPR (AUC = 0.880) showed significantly better diagnostic accuracy (DeLong's Test, $p = 0.034$). Additionally, RPR also had superior diagnostic performance compared to MELD + HDLc ($p < 0.001$). RDW (%) (AUC = 0.804) was found to be significantly better than MELD + HDLc (AUC = 0.627, $p = 0.004$) (Table 7 and Fig. 1).

Table 6: ROC curve analysis showing diagnostic performance of RPR in predicting mortality ($n = 150$)

Parameter	Value (95% CI)
Cutoff (p -value)	≥ 23.7 (<0.001)
AUROC	0.88 (0.824–0.935)
Sensitivity	89.7% (73–98)
Specificity	78.5% (70–85)
Positive predictive value	50.0% (36–64)
Negative predictive value	96.9% (91–99)
Diagnostic accuracy	80.7% (73–87)
Positive likelihood ratio	4.17 (2.9–5.99)
Negative likelihood ratio	0.13 (0.04–0.39)
Diagnostic odds ratio	31.67 (8.88–112.92)

Table 7: Comparison of the diagnostic performance of various predictors in predicting mortality

Predictor	AUROC	95% CI	P	Sn	Sp	PPV	NPV	DA
MELD + HDLc	0.627	0.53–0.724	0.034	97%	32%	26%	97%	44%
MELD	0.978	0.96–0.996	<0.001	100%	88%	67%	100%	91%
MELD-Na	0.977	0.958–0.996	<0.001	97%	89%	68%	99%	91%
RDW (%)	0.804	0.733–0.874	<0.001	90%	72%	43%	97%	75%
RPR	0.880	0.824–0.935	<0.001	90%	78%	50%	97%	81%

AUROC, area under ROC curve; CI, confidence interval; DA, diagnostic accuracy; NPV, negative predictive value; P, p -value; PPV, positive predictive value; Sn, sensitivity; Sp, specificity

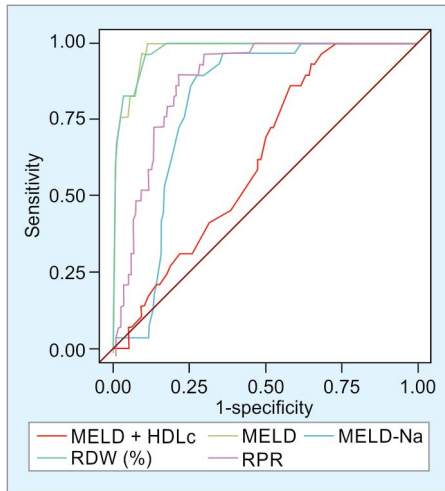


Fig. 1: Comparison of the diagnostic performance of various predictors in predicting mortality

DISCUSSION

In this study, patients included were 18–65 years old, with a mean age of 46.41 ± 11.48 years. This corresponds with observations from a systematic review by D'Amico, comprising 23,797 patients from 118 studies, which reported a mean age of 54 years.⁹ Likewise, in an investigation conducted by Cholongitas et al., which enrolled 312 patients, the mean age was 49.3 ± 11 years.¹⁰ Among 150 patients enrolled in our study, 79.3% (119) were male and 20.7% (31) were female, yielding a male-to-female ratio of 3.8:1. This male predominance aligns with an investigation conducted by Li et al., where 60.8% of participants were male.¹¹

Regarding residency, 31.6% (43) of participants were urban dwellers, while 68.4% (93) were from rural areas. This lower proportion of urban participants contrasts with a multicenter study by Mukherjee et al., which examined chronic liver disease across eleven Indian hospitals and reported a higher proportion of urban participants.¹² The difference is likely due to varying demographic profiles.

Alcohol-related liver disease (45.6%) constituted the predominant etiology in this investigation, followed by hepatitis B (14.8%). These outcomes align with findings from research conducted by Mukherjee et al. and Swaroop et al.^{12,13} Male predominance was observed across all etiologies, while in females, hepatitis C was the leading cause, followed by NASH-related cirrhosis. These findings are consistent with prior research.^{12,13}

The mean Hb level in the study was 8.08 ± 2.30 gm/dL (range: 3.6–15.2 gm/dL), reflecting anemia caused by factors such as nutritional deficiencies and variceal bleeding.¹⁴ The mean platelet count was 1.05 ± 0.75 lakhs, ranging

from 0.12 to 6.7 lakhs. Thrombocytopenia in cirrhotic patients is commonly attributed to portal hypertension, which leads to platelet sequestration in an enlarged spleen and reduced hepatic thrombopoietin production.¹⁵

The mean total bilirubin level was 4.98 ± 6.01 mg/dL (range: 0.14–37 mg/dL). This discovery corresponds with an examination conducted by Ahmad et al., reporting a progressive increase in bilirubin with advancing liver disease and significant correlations with disease severity.¹⁶ The mean SGOT and SGPT levels were 100 and 66.76 U/L, respectively. SGOT levels exceeding SGPT can be explained by reduced hepatic blood flow and a predominance of alcoholic liver disease, which is associated with decreased SGPT levels due to pyridoxal phosphate deficiency.¹⁷

Mean serum albumin level was 2.71 gm/dL, consistent with Carvalho and Machado, who reported reduced plasma albumin levels in advanced cirrhosis due to impaired hepatic synthesis, which can decline by up to 60–80% in severe cases.¹⁸ Serum creatinine had a mean of 2.04 mg/dL, likely attributable to splanchnic vasodilation, reduced effective blood volume, renal hypoperfusion, and subsequent AKI, as described by Slack et al.¹⁹ Mean sodium level was 130.16 ± 5.78 mEq/L, consistent with findings of Young et al., which showed a mean sodium level of 135.36 ± 1.41 mEq/L.²⁰ The mean HDL level was 22.89 ± 10.74 mg/dL, comparable to the findings of Trieb et al., who observed mean HDL levels of 22 mg/dL (range: 11–30 mg/dL) in patients with decompensated cirrhosis.²¹

Among the participants, 76% (114) were classified as CTP class C, which can be attributed to the enrollment of hospitalized patients with advanced disease. All patients were followed for 30 days, with 80.7% (121) surviving and 19.3% (29) expiring during this period. The mean time to mortality was 6.28 ± 3.76 days (range: 1–16 days).

Hyponatremia seemed markedly correlated with mortality. Mean sodium level within survivors was 131.32 mEq/L, compared to 125.31 mEq/L in those who expired. This discovery aligns with Biggins et al., who identified low S Na as a potential indicator of death in LT candidates.²² Additionally, mean HDL level was much lower in the expired group (12.07 mg/dL) compared to survivors (25.48 mg/dL). This aligns with Habib et al., who found that low HDL levels indicate poor prognosis in noncholestatic cirrhosis.²³

Receiver Operating Characteristics Analysis

CTP score: While sensitivity was 100%, specificity was only 0.8%, and diagnostic accuracy was 20% ($p = 0.623$), indicating poor performance as a 30-day mortality predictor.

MELD score: The AUROC was 0.978, indicating excellent discriminatory ability, consistent with Kim et al., who found MELD to be an effective predictor of 1-, 2-, and 3-year mortality, especially 1-year mortality.²⁴

MELD-Na: The AUROC of 0.977 also indicated excellent performance, aligning with findings from Peng et al., which highlighted the superior sensitivity and specificity of MELD-Na over CTP in critical care settings.²⁵

MELD + HDLc: With an AUROC of 0.627 ($p = 0.034$), this model had limited predictive ability. While Wang et al. demonstrated improved predictive performance with MELD + HDLc,²⁶ our findings suggest a need for additional parameters to enhance its utility.

RDW (%): AUROC came to 0.804 ($p < 0.001$), with 72% specificity and 90% sensitivity, demonstrating moderate predictive accuracy. This aligns with Zhou et al., who associated RDW with advanced fibrosis in NAFLD.²⁷

RPR: The AUROC was 0.88 ($p < 0.001$), with a sensitivity of 90% and specificity of 78% at a cutoff of ≥ 23.7 . These findings are consistent with Chen et al., who described RPR as a reliable and cost-efficient predictor of significant fibrosis and cirrhosis.²⁸

CONCLUSION

Most cirrhotic patients in the study were middle-aged males from rural areas. Alcohol and hepatitis B were the leading etiologies. The cohort exhibited anemia, thrombocytopenia, elevated bilirubin, low albumin, and hyponatremia. At 30 days, 19.3% mortality was observed, primarily associated with low sodium and HDL levels. MELD and MELD-Na were the most reliable mortality predictors, followed by RDW and RPR, while MELD + HDLc showed poor performance.

Limitations

The study's limitations include the sample size of 150, which, while reasonable, may not be large enough for broad generalization. The study's focus on ages 18–65 excludes older populations with cirrhosis. The majority of participants were from rural areas, limiting applicability to urban populations with different lifestyles and healthcare access. The 30-day follow-up may be too short to assess long-term outcomes. Acknowledging these limitations helps contextualize the findings and suggests areas for future research.

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REFERENCES

1. D'Amico G, Morabito A, D'Amico M, et al. Clinical states of cirrhosis and competing risks. *J Hepatol* 2018;68(3):563–576.
2. Angeli P, Bernardi M, Villanueva C, et al. EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. *J Hepatol* 2018;69(2):406–460.
3. Devarbhavi H, Asrani SK, Arab JP, et al. Global burden of liver disease: 2023 update. *J Hepatol* 2023;79(2):516–537.
4. D'Amico G, Pasta L, Morabito A, et al. Competing risks and prognostic stages of cirrhosis: a 25-year inception cohort study of 494 patients. *Aliment Pharmacol Ther* 2014;39(10):1180–1193.
5. Pugh RN, Murray-Lyon IM, Dawson JL, et al. Transection of the oesophagus for bleeding oesophageal varices. *Br J Surg* 1973;60(8):646–649.
6. Huo TI, Lee SD, Lin HC. Selecting an optimal prognostic system for liver cirrhosis: the model for end-stage liver disease and beyond. *Liver Int* 2008;28(5):606–613.
7. Biggins SW, Kim WR, Terrault NA, et al. Evidence-based incorporation of serum sodium concentration into MELD. *Gastroenterology* 2006;130(6):1652–1660.
8. Jung YK, Yim HJ. Reversal of liver cirrhosis: current evidence and expectations. *Korean J Intern Med* 2017;32(2):213–228.
9. D'Amico G. Natural history and stages of cirrhosis. In: de Franchis R, Dell'Era A. (editors). *Variceal Hemorrhage*. New York, NY: Springer; 2014.
10. Cholongitas E, Senzolo M, Patch D, et al. Risk factors, sequential organ failure assessment and model for end-stage liver disease scores for predicting short term mortality in cirrhotic patients admitted to intensive care unit. *Aliment Pharmacol Ther* 2006;23:883–893.
11. Li Q, Huang C, Xu W, et al. Accuracy of FibroScan in analysis of liver fibrosis in patients with concomitant chronic Hepatitis B and nonalcoholic fatty liver disease. *Medicine (Baltimore)* 2020;99(23):e20616.
12. Mukherjee PS, Vishnubhatla S, Amarapurkar DN, et al. Etiology and mode of presentation of chronic liver diseases in India: a multi centric study. *PLoS One* 2017;12(10):e0187033.
13. Swaroop S, Vaishnav M, Arora U, et al. Etiological spectrum of cirrhosis in India: a systematic review and meta-analysis. *J Clin Exp Hepatol* 2024;14(2):101291.
14. Manrai M, Dawra S, Kapoor R, et al. Anemia in cirrhosis: an underestimated entity. *World J Clin Cases* 2022;10(3):777–789.
15. Peck-Radosavljevic M. Hypersplenism. *Eur J Gastroenterol Hepatol* 2001;13(4):317–323.
16. Ahmad W, Ijaz B, Javed FT, et al. A comparison of four fibrosis indexes in chronic HCV: development of new fibrosis-cirrhosis index (FCI). *BMC Gastroenterol* 2011;11:44.
17. Cohen JA, Kaplan MM. The SGOT/SGPT ratio—an indicator of alcoholic liver disease. *Dig Dis Sci* 1979;24:835–838.
18. Carvalho JR, Machado MV. New insights about albumin and liver disease. *Ann Hepatol* 2018;17(4):547–560.
19. Slack A, Yeoman A, Wendon J. Renal dysfunction in chronic liver disease. *Crit Care* 2010;14(2):214.
20. Young S, Rostambeigi N, Golzarian J, et al. MELD or sodium MELD: a comparison of the ability of two scoring systems to predict outcomes after transjugular intrahepatic portosystemic shunt placement. *AJR Am J Roentgenol* 2020;215(1):215–222.
21. Trieb M, Rainer F, Stadlbauer V, et al. HDL-related biomarkers are robust predictors of survival in patients with chronic liver failure. *J Hepatol* 2020;73(1):113–120.
22. Biggins SW, Rodriguez HJ, Bacchetti P, et al. Serum sodium predicts mortality in patients listed for liver transplantation. *Hepatology* 2005;41(1):32–39.
23. Habib A, Mihas AA, Abou-Assi SG, et al. High-density lipoprotein cholesterol as an indicator of liver function and prognosis in noncholestatic cirrhotics. *Clin Gastroenterol Hepatol* 2005;3(3):286–291.
24. Kim KM, Shim SG, Sinn DH, et al. Child–Pugh, MELD, MELD-Na, and ALBI scores: which liver function models best predicts prognosis for HCC patient with ascites? *Scand J Gastroenterol* 2020;55(8):951–957.
25. Peng Y, Qi X, Guo X. Child–Pugh versus MELD score for the assessment of prognosis in liver cirrhosis: a systematic review and meta-analysis of observational studies. *Medicine (Baltimore)* 2016;95(8):e2877.
26. Wang Y, Shen W, Huang F, et al. HDL-C levels added to the MELD score improves 30-day mortality prediction in Asian patients with cirrhosis. *J Int Med Res* 2022;50(7):3000605221109385.
27. Zhou WJ, Yang J, Zhang G, et al. Association between red cell distribution width-to-platelet ratio and hepatic fibrosis in nonalcoholic fatty liver disease: a cross-sectional study. *Medicine (Baltimore)* 2019;98(30):e16565.
28. Chen B, Ye B, Zhang J, et al. RDW to platelet ratio: a novel noninvasive index for predicting hepatic fibrosis and cirrhosis in chronic hepatitis B. *PLoS One* 2013;8(7):e68780.