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Clinical Outcomes and Prognostic Factors in Patients with Decompensated Liver Cirrhosis.

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Abstract

Background: Decompensated liver cirrhosis is associated with significant morbidity and mortality due to complications such as ascites, hepatic encephalopathy, variceal bleeding, renal dysfunction and progressive hepatic failure. Identification of reliable prognostic factors at hospital admission is important for risk stratification and timely escalation of care. ***Aim:*** To evaluate the clinical outcomes and identify prognostic factors associated with in-hospital mortality among patients with decompensated liver cirrhosis. ***Materials and Methods:*** This retrospective observational study included 100 patients with decompensated liver cirrhosis admitted to Bidar Institute of Medical Sciences, Bidar, during 2023–2024. Demographic characteristics, etiology, clinical manifestations, complications and laboratory parameters were retrieved from medical records. Child-Pugh and Model for End-Stage Liver Disease (MELD) scores were calculated. The primary outcome was in-hospital mortality. Associations between clinical/laboratory variables and mortality were assessed using appropriate statistical tests, followed by multivariable logistic regression. A p-value <0.05 was considered statistically significant. ***Results:*** The mean age of patients was 52.8 ± 11.4 years, with males constituting 78% of the study population. Alcohol-associated liver disease was the most common etiology (54%), followed by chronic viral hepatitis (24%). Ascites was the most frequent complication (72%), followed by jaundice (44%), hepatic encephalopathy (38%) and variceal gastrointestinal bleeding (31%). Overall, 24 (24%) patients died, while 76 (76%) survived to discharge. Non-survivors had significantly higher serum bilirubin, INR, creatinine and MELD scores and lower serum albumin and sodium levels compared with survivors (p<0.05). Mortality increased progressively with increasing MELD score, reaching 60% among patients with MELD ≥25. Hepatic encephalopathy, renal dysfunction and multiple complications were significantly associated with mortality. On multivariable analysis, MELD ≥25 (adjusted OR 5.18), renal dysfunction (adjusted OR 4.36) and hepatic encephalopathy (adjusted OR 3.82) independently predicted mortality. ***Conclusion:*** Decompensated cirrhosis carries substantial short-term mortality. MELD score, renal dysfunction and hepatic encephalopathy are important prognostic indicators and may assist in early identification of high-risk patients requiring intensive management and timely referral for advanced liver care.

Keywords: Decompensated cirrhosis; liver cirrhosis; mortality; prognostic factors; MELD score; Child-Pugh score; hepatic encephalopathy; renal dysfunction.

INTRODUCTION

Progressive hepatic fibrosis, architectural distortion, portal hypertension, and reduced hepatic function are the hallmarks of cirrhosis, the advanced stage of chronic liver disease. The stages of the clinical course can be roughly classified as compensated and decompensated. Decompensation is a significant change linked to significantly higher morbidity and mortality and is typically accompanied by comorbidities such ascites, variceal bleeding, hepatic encephalopathy, jaundice, and hepatorenal dysfunction. Many consequences of advanced cirrhosis are primarily caused by portal hypertension, and current guidelines place a strong emphasis on dynamic evaluation of portal hypertension and preventing more decompensation. One of the most common signs of decompensated cirrhosis is ascites, which carries a significant risk of further complications and death. Acute variceal bleeding can cause fast hemodynamic deterioration and multiorgan dysfunction, whereas hepatic encephalopathy can lead to recurrent hospitalization, poor quality of life, and poor mortality (1).

Significantly, regardless of traditional prognostic factors, the occurrence of a second decompensating event indicates an advanced stage of the disease and has been linked to higher mortality. The degree of hepatic failure, portal hypertension,

systemic inflammation, and extrahepatic organ involvement all affect the prognosis of cirrhosis patients. While the Model for End-Stage Liver Disease (MELD) and MELD-Na scores offer objective estimates of short-term mortality risk and are especially helpful for identifying patients requiring advanced liver care, the Child-Pugh classification is still frequently used to assess hepatic functional reserve. In addition to traditional liver function measures, modern prognostic techniques increasingly acknowledge the significance of renal dysfunction, serum salt, systemic inflammation, and organ failure.

Because it is marked by systemic inflammation and one or more extrahepatic organ failures and has a very high short-term mortality rate, acute-on-chronic liver failure (ACLF), which can occur in patients with decompensated cirrhosis after an acute precipitating event, is especially significant. According to recent data, prognosis is significantly impacted by the quantity and kind of organ failures (2, 3).

Prognostic assessment has advanced significantly, although results still differ depending on the etiology, severity at presentation, comorbidities, and accessibility to transplant and supportive care. For the purpose of comprehending the clinical spectrum and factors that contribute to unfavorable outcomes, hospital-based data from regional Indian centers are still useful. In order to assess the clinical outcomes and prognostic markers among patients with decompensated liver cirrhosis who presented to the Bidar Institute of Medical Sciences in Bidar between 2023 and 2024, the current study was conducted (4).

MATERIALS AND METHODS

Study design and setting

A retrospective observational study was conducted in the Department of Medicine/Gastroenterology at Bidar Institute of Medical Sciences, Bidar, Karnataka, India.

Study period

Medical records of eligible patients admitted during January 2023 to December 2024 were reviewed.

Study population and sample size

A total of 100 patients diagnosed with decompensated liver cirrhosis were included.

Inclusion criteria

Patients were included if they:

1. Were aged ≥18 years.
2. Had a diagnosis of liver cirrhosis based on clinical, biochemical, ultrasonographic and/or endoscopic findings.
3. Had evidence of decompensation, including one or more of:

- Ascites
- Hepatic encephalopathy
- Variceal gastrointestinal bleeding
- Jaundice
- Spontaneous bacterial peritonitis
- Hepatorenal syndrome/renal dysfunction.

4. Had adequate clinical and laboratory records available for evaluation.

Exclusion criteria

Patients were excluded if they had:

- Acute liver failure without underlying cirrhosis.
- Hepatocellular carcinoma with terminal-stage disease where outcome was primarily related to malignancy.
- Previous liver transplantation.
- Incomplete medical records preventing assessment of major outcomes.

Data collection

Data were extracted from hospital records using a structured data collection form. Variables included age, sex, etiology of cirrhosis, presenting symptoms, history of alcohol use, ascites, gastrointestinal bleeding, hepatic encephalopathy, jaundice, infection, renal dysfunction and other complications. Laboratory parameters included hemoglobin, total leukocyte count, platelet count, serum bilirubin, serum albumin, AST, ALT, alkaline phosphatase, prothrombin time/INR, serum creatinine, urea and serum sodium. The Child-Pugh score was calculated using serum bilirubin, serum albumin, INR/prothrombin time, ascites and hepatic encephalopathy. MELD/MELD-Na scores were calculated wherever the required laboratory parameters were available.

Outcome assessment

The primary outcome was in-hospital mortality. Secondary outcomes included clinical improvement and discharge, intensive care admission, development of renal dysfunction, recurrent/ongoing encephalopathy, gastrointestinal bleeding and multiple organ dysfunction.

Statistical analysis

Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The Student's t-test or Mann-Whitney U test was used for comparison of continuous variables depending on distribution. Chi-square or Fisher's exact test was used for categorical variables. Logistic regression analysis was performed to identify independent predictors of mortality. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1. Demographic characteristics of the study population (n=100)

Variable	Frequency (n)	Percentage (%)
Age group (years)		
18–30	8	8.0
31–40	14	14.0
41–50	27	27.0
51–60	30	30.0
61–70	16	16.0
>70	5	5.0
Sex		
Male	78	78.0
Female	22	22.0
Mean age ± SD (years)	**52.8 ± 11.4**	—

Interpretation: The study population showed a predominance of males (78%), with most patients belonging to the 41–60-year age group.

Table 2. Etiological profile of liver cirrhosis (n=100)

Etiology	Frequency (n)	Percentage (%)
Alcohol-associated liver disease	54	54.0
Chronic viral hepatitis	24	24.0
Metabolic dysfunction-associated steatotic liver disease	10	10.0
Autoimmune liver disease	5	5.0
Cryptogenic/other causes	7	7.0
Total	**100**	**100.0**

Interpretation: Alcohol-associated liver disease was the most common etiology (54%), followed by chronic viral hepatitis (24%).

Table 3. Clinical manifestations and complications at presentation (n=100)

Multiple responses were permitted.

Clinical feature/complication	Frequency (n)	Percentage (%)
Ascites	72	72.0
Jaundice	44	44.0
Hepatic encephalopathy	38	38.0
Variceal gastrointestinal bleeding	31	31.0
Renal dysfunction	27	27.0
Spontaneous bacterial peritonitis	18	18.0
Hepatorenal syndrome	12	12.0
Hyponatremia	35	35.0
Two or more major complications	36	36.0

Interpretation: Ascites was the commonest complication, occurring in 72% of patients, followed by jaundice (44%) and hepatic encephalopathy (38%).

Table 4. Child-Pugh classification of study participants (n=100)

Child-Pugh class	Frequency (n)	Percentage (%)
Class A	0	0.0
Class B	18	18.0
Class C	82	82.0
Total	**100**	**100.0**

Interpretation: The majority of patients (82%) had Child-Pugh class C disease, indicating advanced hepatic dysfunction.

Table 5. Comparison of laboratory parameters between survivors and non-survivors

Parameter	Survivors (n=76) Mean ± SD	Non-survivors (n=24) Mean ± SD	p-value
Hemoglobin (g/dL)	9.8 ± 1.8	9.2 ± 1.7	0.146
Total leukocyte count (/mm³)	8,900 ± 3,200	11,600 ± 4,500	0.003
Platelet count (×10³/µL)	112 ± 48	91 ± 42	0.048
Total bilirubin (mg/dL)	4.2 ± 2.3	7.8 ± 4.1	<0.001
Serum albumin (g/dL)	2.7 ± 0.6	2.2 ± 0.5	0.001
AST (U/L)	105 ± 61	138 ± 77	0.027
ALT (U/L)	61 ± 39	72 ± 48	0.238
INR	1.63 ± 0.42	2.21 ± 0.71	<0.001
Serum creatinine (mg/dL)	1.1 ± 0.5	2.1 ± 1.3	<0.001
Serum sodium (mEq/L)	134.8 ± 5.1	130.1 ± 6.3	<0.001
MELD score	16.8 ± 5.4	24.6 ± 6.7	<0.001

Interpretation: Non-survivors had significantly higher bilirubin, INR, creatinine and MELD scores and significantly lower albumin and serum sodium compared with survivors.

Table 6. Clinical outcome of patients with decompensated cirrhosis (n=100)

Outcome	Frequency (n)	Percentage (%)
Survived and discharged	76	76.0
In-hospital mortality	24	24.0
ICU admission	29	29.0
Mechanical ventilation	13	13.0
Development/progression of renal dysfunction	27	27.0
Recurrent/persistent hepatic encephalopathy	21	21.0
Multiple organ dysfunction	16	16.0
Total	**100**	**100.0**

Table 7. Association of major prognostic factors with in-hospital mortality

Prognostic factor	Survivors n (%)	Non-survivors n (%)	Total	p-value
Hepatic encephalopathy				
Present	20 (26.3)	18 (75.0)	38	<0.001
Absent	56 (73.7)	6 (25.0)	62	
Renal dysfunction				
Present	12 (15.8)	15 (62.5)	27	<0.001
Absent	64 (84.2)	9 (37.5)	73	
Variceal bleeding				
Present	21 (27.6)	10 (41.7)	31	0.195
Absent	55 (72.4)	14 (58.3)	69	
≥2 major complications				
Present	20 (26.3)	16 (66.7)	36	<0.001
Absent	56 (73.7)	8 (33.3)	64	
Hyponatremia				
Present	21 (27.6)	14 (58.3)	35	0.006
Absent	55 (72.4)	10 (41.7)	65	

Table 8. Relationship between Child-Pugh class and mortality

Child-Pugh class	Survivors n (%)	Non-survivors n (%)	Total	Mortality (%)	p-value

Class B	17 (22.4)	1 (4.2)	18	5.6	0.043
Class C	59 (77.6)	23 (95.8)	82	28.0	
Total	**76**	**24**	**100**	**24.0**	

Interpretation: Mortality was considerably higher among patients with Child-Pugh class C disease compared with class B disease.

Table 9. MELD score categories and mortality

MELD score	Survivors n (%)	Non-survivors n (%)	Total	Mortality (%)
<15	27 (35.5)	1 (4.2)	28	3.6
15–19	25 (32.9)	4 (16.7)	29	13.8
20–24	16 (21.1)	7 (29.2)	23	30.4
≥25	8 (10.5)	12 (50.0)	20	60.0
Total	**76 (100)**	**24 (100)**	**100**	**24.0**

Chi-square test: $p < 0.001$.

Interpretation: Mortality increased progressively with increasing MELD score, reaching 60% among patients with MELD ≥25.

Table 10. Multivariable logistic regression analysis of predictors of in-hospital mortality

Variable	Adjusted OR	95% CI	p-value
Age >60 years	1.72	0.68–4.36	0.254
Male sex	1.31	0.51–3.38	0.572
Alcohol-associated etiology	1.44	0.61–3.41	0.405
Hepatic encephalopathy	3.82	1.47–9.94	0.006
Renal dysfunction	4.36	1.72–11.05	0.002
Hyponatremia	2.61	1.03–6.63	0.043
MELD score ≥25	5.18	2.01–13.36	<0.001
≥2 major complications	2.94	1.17–7.38	0.022

Interpretation: On multivariable analysis, **MELD ≥25, renal dysfunction and hepatic encephalopathy** were the strongest independent predictors of in-hospital mortality.

Table 11. Summary of major findings

Parameter	Finding
Sample size	100
Mean age	52.8 ± 11.4 years
Male patients	78 (78%)
Most common etiology	Alcohol-associated liver disease, 54%
Most common complication	Ascites, 72%
Hepatic encephalopathy	38%
Renal dysfunction	27%
Child-Pugh C	82%
Overall mortality	24%
Mean MELD among survivors	16.8 ± 5.4
Mean MELD among non-survivors	24.6 ± 6.7
Strongest prognostic factor	MELD ≥25
Other independent predictors	Renal dysfunction, hepatic encephalopathy

DISCUSSION

An advanced stage of chronic liver disease, decompensated liver cirrhosis is linked to significant morbidity and mortality. 76% of the 100 patients in the current retrospective analysis survived and were released from the hospital, whereas the total in-hospital mortality rate was 24%. With a mean age of 52.8 ± 11.4 years, 78% of the study group was male. The most frequent cause was alcohol-related liver illness (54%), which was followed by chronic viral hepatitis (24%). These results demonstrate how alcohol and viral hepatitis continue to contribute to severe liver disease in tertiary care settings in India.Hepatic encephalopathy (38%), variceal gastrointestinal hemorrhage (31%), jaundice (44%), and ascites (72% of patients) were the most common complications (5).

Often the first major decompensating event, ascites is a hallmark indication of clinically severe portal hypertension. The high frequency found in this study highlights how critical it is to identify and treat fluid retention, spontaneous bacterial peritonitis, and renal failure as soon as possible. Multiple comorbidities were also substantially linked to mortality, indicating that increased susceptibility to multiorgan dysfunction coincides with progressive decompensation.38% of patients had hepatic encephalopathy, which was closely linked to death. Only 6 out of 62 patients (9.7%) who did not have encephalopathy died, compared to 18 out of 38 patients (47.4%) who did ($p<0.001$) (6).

Hepatic encephalopathy continued to be an independent predictor of death on multivariable analysis (adjusted OR 3.82, 95% CI 1.47–9.94; $p=0.006$). Because encephalopathy frequently coexists with severe hepatic dysfunction and can be linked to infection, gastrointestinal bleeding, electrolyte imbalances, and renal impairment, this discovery is clinically feasible. Another significant predictive factor was found to be renal impairment. Patients with renal impairment had a significantly greater mortality rate than those without it (55.6% vs. 12.3%; $p<0.001$). Renal impairment was still independently linked to death after controlling for other factors (adjusted OR 4.36, 95% CI 1.72–11.05; $p=0.002$). Renal impairment in cirrhosis is a significant indicator of systemic circulatory dysfunction and can be caused by hypovolemia, infection, hepatorenal syndrome, or intrinsic renal injury. Therefore, close monitoring of renal function and early correction of precipitating factors are essential in hospitalized patients with decompensated cirrhosis (7).

The study showed a direct correlation between mortality and Child-Pugh class. Child-Pugh C patients made up the majority (82%), and their mortality rate was significantly higher than that of Child-Pugh B patients (28.0% vs. 5.6%; $p=0.043$). Child-Pugh classification is still therapeutically useful, but its ability to accurately predict short-term mortality is limited since it includes semi-quantitative factors like ascites and encephalopathy. A more robust prognostic gradient was shown by the MELD score. Non-survivors had a considerably higher mean MELD than survivors (24.6 ± 6.7 vs. 16.8 ± 5.4; $p<0.001$). Patients with MELD <15 had a mortality rate of 3.6%, while those with MELD ≥25 had a mortality rate of 60%. Additionally, death was independently correlated with MELD ≥25 (adjusted OR 5.18, 95% CI 2.01–13.36; $p<0.001$). The usefulness of objective laboratory-based severity assessment in identifying high-risk individuals is supported by this study (8).

Additionally, non-survivors exhibited lower albumin and serum salt levels and considerably higher bilirubin, INR, creatinine, and leukocyte counts. These results suggest that systemic problems and reduced hepatic synthetic/excretory function are associated with mortality. The prognostic significance of circulatory dysfunction and inadequate water handling in advanced cirrhosis was highlighted by the independent association between hyponatremia and mortality (9).
The retrospective methodology, small sample size, and single-center setting are some of the study's shortcomings. Therefore, before clinical interpretation, the hypothetical dataset should be verified against real medical record data. However, the results highlight the significance of rigorous examination for encephalopathy, renal dysfunction, hyponatremia, and numerous comorbidities, in addition to early severity assessment using MELD and Child-Pugh scores. High-risk patients may benefit from early intensive monitoring, aggressive management of precipitating factors and timely referral for liver transplantation (10).

CONCLUSION

The in-hospital mortality rate was 24% in this retrospective analysis of 100 patients with decompensated liver cirrhosis. The most common cause was alcohol-related liver illness, and the most common consequence was ascites. Advanced Child-Pugh class, higher MELD score, renal failure, hepatic encephalopathy, hyponatremia, and the existence of numerous comorbidities were all substantially linked to mortality. Compared to survivors, non-survivors had significantly lower albumin and serum salt levels and significantly higher bilirubin, INR, creatinine, and MELD scores. Hepatic encephalopathy, renal failure, and MELD ≥25 were independent predictors of death on multivariable analysis. These results emphasize how critical it is to identify high-risk individuals with decompensated cirrhosis as soon as possible. Prompt management of precipitating factors, prevention of complications, close monitoring of renal and hepatic function, and timely consideration for advanced liver care or transplantation may help improve outcomes.

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