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ORIGINAL ARTICLE

Clinical and Laboratory Indicators Associated with Cirrhosis Severity

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SUMMARY

Background: **This study aimed to evaluate clinical and laboratory indicators associated with varying cirrhosis severity, with the objective of supporting more accurate clinical assessment and stratification.**
Methods: **A retrospective analysis was conducted on 180 patients diagnosed with cirrhosis and treated at the People's Hospital of Zhengzhou between January 1, 2020, and February 1, 2023. Patients were stratified into three groups based on the Child-Pugh classification: grade A (n = 73), grade B (n = 68), and grade C (n = 39). Statistical analyses were performed to identify associations between clinical and laboratory variables and cirrhosis severity.**
Results: **No statistically significant differences were observed among the three groups with respect to gender, age, diarrhea, abdominal distension, or abdominal pain ($p > 0.05$). Fever and spontaneous bacterial peritonitis were more frequently observed in patients with advanced disease ($p < 0.05$). The presence of hepatitis B, hepatitis C, cholestasis, malnutrition, circulatory disorders, and other etiologies showed no significant association with cirrhosis severity ($p > 0.05$). Similarly, portal vein diameter, upper gastrointestinal hemorrhage, alcohol use history, smoking status, and esophagogastric varices did not differ significantly among groups ($p > 0.05$). Laboratory parameters significantly associated with cirrhosis severity included serum calcium (Ca^{2+}), white blood cell count (WBC), platelet count (PLT), C-reactive protein (CRP), hemoglobin (Hb), fibrinogen (FIB), total bile acids (TBA), apolipoprotein A1 (APOA1), direct bilirubin (DBIL), cholinesterase (CHE), cystatin C (CYSC), and bicarbonate (HCO^{3-}) ($p < 0.05$). TBA and DBIL levels were positively correlated with disease severity, while FIB, APOA1, CHE, and HCO^{3-} levels were negatively correlated.**
Conclusions: **Fever, spontaneous bacterial peritonitis, and several laboratory markers, namely CRP, PLT, FIB, TBA, Ca^{2+}, APOA1, CHE, CYSC, and HCO^{3-}, may serve as relevant indicators for assessing cirrhosis severity. Recognition of these indicators may support improved clinical stratification and guide management decisions.**
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KEYWORDS

cirrhosis, clinical features, diagnostic indicators, etiology, related factors

LIST OF ABBREVIATIONS

Ca - serum calcium
WBC - leukocyte
PLT - thrombocyte
CRP - C-reactive protein
HB - hemoglobin
FIB - fibrinogen

TBA - total bile acid
APOA1 - apolipoprotein A1
DBIL - direct bilirubin
CHE - cholinesterase
CYSC - cystatin C
HCO_3^- - bicarbonate

INTRODUCTION

Cirrhosis is a chronic liver disease marked by persistent inflammation and progressive fibrosis, histologically characterized by diffuse hepatic scarring, pseudolobule formation, and vascular remodeling involving both intrahepatic and extrahepatic vessels [1,2]. The clinical course typically advances from a compensated phase, often asymptomatic, to a decompensated phase, which is defined by the onset of complications such as ascites, upper gastrointestinal bleeding, and hepatic encephalopathy, all of which signify clinical decompensation [3].

Despite substantial advances in the understanding and management of cirrhosis, it continues to represent a major global health burden, accounting for an estimated 1 to 2 million deaths annually. Patients in the decompensated phase frequently experience significant reductions in quality of life and have a generally unfavorable prognosis [4].

Although research efforts have increasingly focused on therapeutic strategies and prognostic evaluation in cirrhosis, there remains a relative paucity of studies examining the relationship between cirrhosis severity and its clinical manifestations and associated factors. The present study aims to characterize clinical and laboratory indicators associated with varying degrees of cirrhosis severity, using the Child-Pugh classification system as a stratification tool. Particular emphasis is placed on the potential utility of biomarkers such as total bile acids (TBA), direct bilirubin (DBIL), fibrinogen (FIB), and C-reactive protein (CRP) in the clinical evaluation of disease severity.

MATERIALS AND METHODS

Study population

A total of 180 patients diagnosed with cirrhosis were enrolled at the People's Hospital of Zhengzhou between January 1, 2020, and February 1, 2023. Based on the Child-Pugh classification criteria, patients were stratified into three groups: Child-Pugh A (n = 73), Child-Pugh B (n = 68), and Child-Pugh C (n = 39) [5]. Clinical and laboratory data were compared across groups to examine associations between cirrhosis severity and related clinical features. The study included both outpatient evaluations and the management of patients with advanced disease requiring timely intervention.

Inclusion and exclusion criteria

Inclusion criteria

1) Patients meeting the diagnostic criteria for cirrhosis as outlined in the Guidelines for the Diagnosis and Treatment of Cirrhosis by the Chinese Medical Association, with confirmation based on abdominal ultrasound, gastroscopy, liver function tests, complete blood count, coagulation profile, and additional laboratory evaluations;
2) Availability of complete clinical data.

Exclusion criteria

1) Pregnant or breastfeeding women;
2) Patients with hematologic disorders such as idiopathic thrombocytopenic purpura;
3) Patients lacking complete clinical data.

Data collection

All patients underwent fasting blood tests. Collected data included demographic variables (age, gender), etiology, clinical symptoms (e.g., diarrhea, abdominal pain, abdominal distension), and the presence of fever or spontaneous bacterial peritonitis. Laboratory parameters included platelet count (PLT), hemoglobin (Hb), CRP, white blood cell count (WBC), FIB, serum calcium (Ca^{2+}), TBA, apolipoprotein A1 (APOA1), DBIL, cholinesterase (CHE), cystatin C (CYSC), and bicarbonate (HCO^{3-}).

Screening for hepatitis B and hepatitis C virus infections was performed. Etiology was determined through a review of medical history and relevant clinical investigations. The occurrence of upper gastrointestinal bleeding was assessed using patient history, clinical presentation, and gastroscopy findings. All patients underwent gastroscopy to evaluate for esophagogastric varices. The diameter of the main portal vein trunk was measured using abdominal ultrasonography.

Statistical analysis

Statistical analyses were conducted using SPSS version 26.0. Quantitative data are expressed as mean ± standard deviation ($\bar{x} \pm s$). Normality testing was performed to determine appropriate statistical methods. Intergroup comparisons were conducted using one-way analysis of variance (ANOVA) or the Kruskal-Wallis test, as applicable. Post hoc pairwise comparisons were performed using the Z-test for variables with significant intergroup differences.

Categorical data are presented as frequencies or percentages and analyzed using the chi-squared test. The Q-test was used for pairwise comparisons of categorical variables with significant differences. A two-tailed p-value of < 0.05 was considered statistically significant.

Table 1. Comparison of relevant clinical factors among patients with cirrhosis with varying disease severity.

Factors	Child-Pugh A grade	Child-Pugh B grade	Child-Pugh C grade	F/χ^2 value	p-value
DBIL	9.67 ± 5.32	18.98 ± 17.06	92.65 ± 113.09	33.36	< 0.001
HB	118.67 ± 4.96	103.84 ± 32.18	104.79 ± 23.19	4.61	0.011
APOA1	1.29 ± 0.37	0.98 ± 0.28	0.61 ± 0.29	58.64	< 0.001
Portal vein diameter	11.90 ± 1.93	12.60 ± 2.29	12.50 ± 1.70	2.36	0.098
PLT	101.00 ± 51.06	118.04 ± 90.17	82.36 ± 54.87	3.37	0.032
WBC	4.18 ± 2.27	5.03 ± 2.94	5.90 ± 3.19	5.09	0.006
Ca	2.22 ± 0.16	2.10 ± 0.15	2.10 ± 0.18	12.03	< 0.001
FIB	2.31 ± 0.59	2.02 ± 0.60	1.38 ± 0.66	29.61	< 0.001
CRP	2.25 ± 5.88	15.43 ± 31.57	19.39 ± 27.60	8.70	< 0.001
TBA	23.46 ± 27.38	62.13 ± 60.35	130.13 ± 125.45	28.5	< 0.001
CHE	7,216.16 ± 1,423.19	4,744.23 ± 2,152.31	2,415.84 ± 877.13	111.72	< 0.001
CYSC	0.95 ± 0.27	0.99 ± 0.32	1.36 ± 0.77	12.02	< 0.001
HCO^-_3	24.54 ± 2.46	23.22 ± 2.37	20.90 ± 3.22	24.82	< 0.001
History of alcohol consumption					
Yes	20 (27.40)	23 (33.82)	12 (30.77)	0.69	0.710
No	53 (72.60)	45 (66.18)	27 (69.23)		
History of smoking					
Yes	15 (20.55)	13 (19.12)	8 (20.51)	0.53	0.974
No	58 (79.45)	55 (80.88)	31 (79.49)		
Esophagogastric varices					
Yes	42 (57.53)	44 (64.71)	18 (46.15)	3.50	0.174
No	31 (42.47)	24 (35.29)	21 (53.85)		
Upper gastrointestinal bleeding					
Yes	13 (17.81)	21 (30.88)	12 (30.77)	3.88	0.114
No	60 (82.19)	47 (69.12)	27 (69.23)		

Table 2. Statistical analysis of CRP, ALB, HB, PLT, FIB, and total bilirubin levels.

Factors	Comparison between Child-Pugh A and Child-Pugh B grades (p-value)	Comparison between Child-Pugh A and Child-Pugh C grades (p-value)	Comparison between Child-Pugh B and Child-Pugh C grades (p-value)
DBIL	0.001	0.000	0.000
HB	0.019	0.058	1.000
APOA1	0.000	0.000	0.000
PLT	1.000	0.286	0.840
WBC	0.425	0.011	0.718
Ca	0.010	< 0.001	1.000
FIB	0.047	< 0.001	< 0.001
CRP	< 0.001	< 0.001	0.313
TBA	< 0.001	< 0.001	0.034
CHE	0.000	0.000	0.000
CYSC	1.000	0.000	0.000
HCO^{3-}	0.047	0.000	0.001

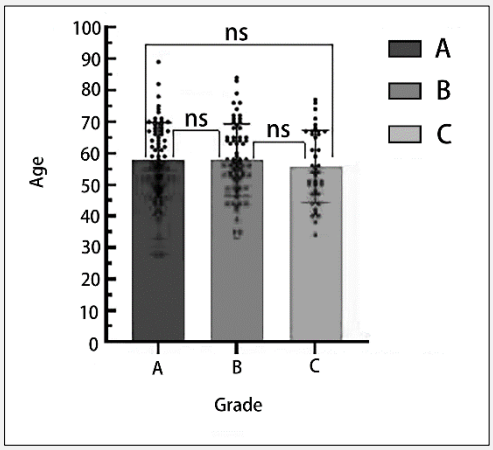

Figure 1. Distribution of clinical features across three Child-Pugh groups in patients with cirrhosis.

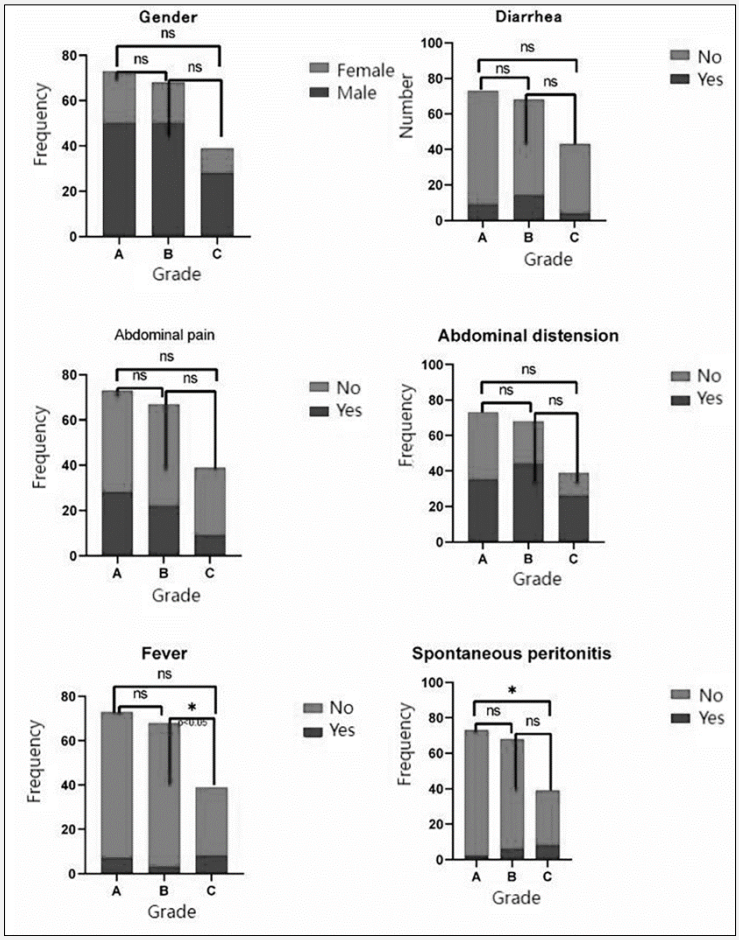

Figure 2. Comparison of clinical manifestations among patients with varying cirrhosis severity based on Child-Pugh classification.

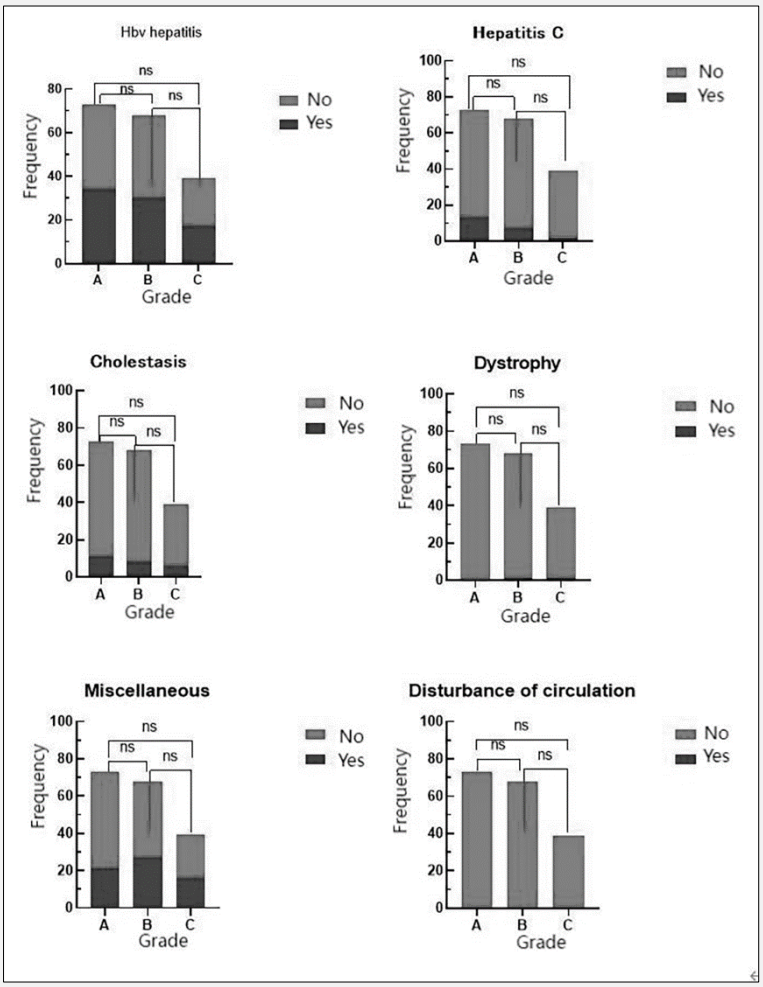

Figure 3. Correlation analysis of cirrhosis etiology among Child-Pugh groups.

No significant differences were observed for hepatitis B ($\chi^2 = 0.126$, $p = 0.939$), hepatitis C ($\chi^2 = 4.128$, $p = 0.123$), cholestasis ($\chi^2 = 0.414$, $p = 0.813$), malnutrition ($\chi^2 = 0.165$, $p = 0.438$), circulatory disorders ($\chi^2 = 0.000$), and other etiologies ($\chi^2 = 2.489$, $p = 0.488$).

RESULTS

Clinical manifestations across Child-Pugh groups

Among the 180 patients with cirrhosis, 73 (40,56) were classified as Child-Pugh A, 68 (37.78%) as Child-Pugh B, and 39 (21.63) as Child-Pugh C. No statistically significant differences were observed among the three groups in terms of gender, age, diarrhea, abdominal pain, or abdominal distension ($p > 0.05$). However, significant differences were identified in the incidence of fever ($p = 0.028$) and spontaneous bacterial peritonitis ($p = 0.007$) (Figures 1 and 2).

Etiological comparisons among severity groups

No statistically significant association was found between cirrhosis severity and etiological factors such as hepatitis B, hepatitis C, cholestasis, malnutrition, circulatory disorders, or other causes ($p > 0.05$) (Figure 3).

Comparison of related variables across disease stages

Related factors were categorized as qualitative or quantitative variables. Unordered qualitative variables were analyzed using the χ^2 test, with cirrhosis severity used as the dependent variable (Child-Pugh A = 1, B = 2, C = 3). Variables including upper gastrointestinal bleeding, alcohol use history, smoking history, and esophagogastric varices were coded as present (4) or absent (5).

Quantitative variables were assessed for normality. Variables with normal or approximately normal distribu-

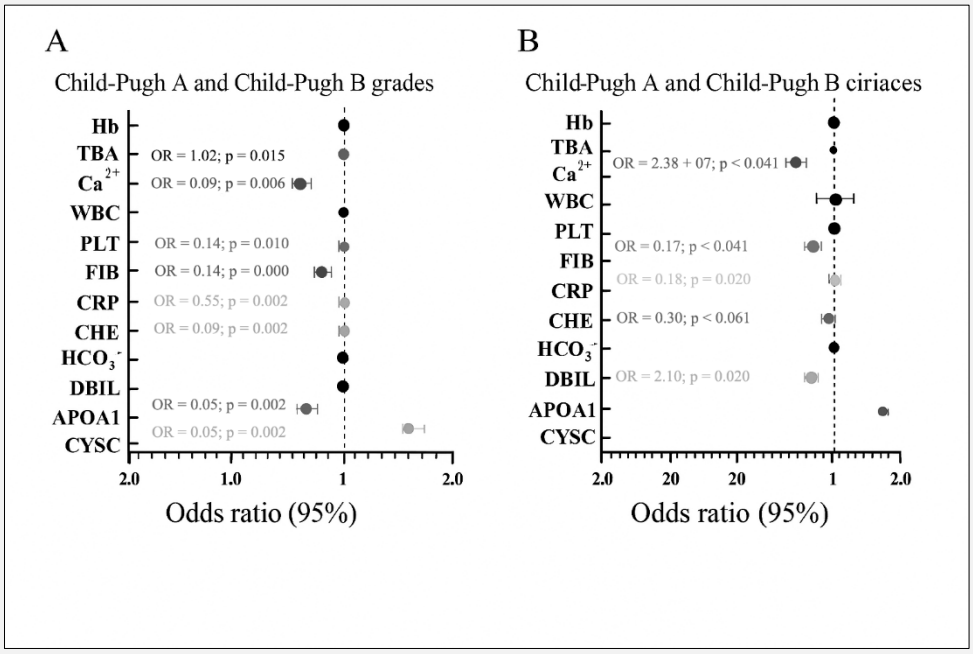

Figure 4. Logistic regression analysis showing the association between cirrhosis severity and relevant clinical and laboratory variables.

tions were analyzed using ANOVA, while those with non-normal or skewed distributions were evaluated using the Kruskal-Wallis test. No statistically significant differences were observed among the three groups for portal vein diameter, upper gastrointestinal bleeding, alcohol use history, smoking history, or esophagogastric varices ($p > 0.05$). However, statistically significant differences were found in CRP, PLT, WBC, Hb, FIB, TBA, Ca^{2+}, APOA1, DBIL, CHE, CYSC, and HCO^{3-} ($p < 0.05$) (Table 1).

Pairwise comparisons of significant variables

Pairwise comparisons were conducted for variables that showed significant intergroup differences (as presented in Table 1). Between the Child-Pugh A and B groups, statistically significant differences were observed in DBIL, Hb, APOA1, Ca^{2+}, FIB, CRP, TBA, CHE, and HCO^{3-} ($p < 0.05$). Comparisons between the Child-Pugh B and C groups revealed significant differences in DBIL, APOA1, WBC, Ca^{2+}, FIB, CRP, TBA, CHE, CYSC, and HCO^{3-} ($p < 0.05$). Additionally, the Child-Pugh A and C groups differed significantly in DBIL, APOA1, FIB, TBA, CHE, CYSC, and HCO^{3-} ($p < 0.05$) (Table 2).

Logistic regression analysis of related variables

Multinomial logistic regression was performed to evaluate the association between selected variables and cirrhosis severity, with the latter serving as the dependent variable (Child-Pugh A = 1, B = 2, C = 3). Independent variables included CRP, PLT, WBC, Hb, FIB, TBA, Ca^{2+}, APOA1, DBIL, CHE, CYSC, and HCO^{3-}. Child-Pugh A was used as the reference category.

The model was constructed to identify which factors were significantly associated with higher severity classifications. Data visualization was conducted using GraphPad software, and statistically significant variables were annotated accordingly. The analysis indicated that abnormal levels of CRP, PLT, FIB, TBA, Ca^{2+}, APOA1, CHE, CYSC, and HCO^{3-} were significantly associated with increased cirrhosis severity (odds ratio < 1, $p < 0.05$) (Figure 4).

DISCUSSION

Cirrhosis is a chronic liver condition with diverse etiologies and a prolonged clinical course. During the compensated phase, the disease may be mild or asymptomatic. However, progression to the decompensated phase is typically marked by severe complications that significantly accelerate clinical deterioration and increase mortality risk [3,6]. Both Chinese and international studies emphasize that early detection and intervention are critical to slowing disease progression and, in some cases, achieving partial reversal of hepatic dysfunction

[1,7]. Therefore, timely evaluation and accurate assessment of cirrhosis severity are essential for guiding clinical management.

In the present study, significant differences in clinical manifestations, specifically fever and spontaneous bacterial peritonitis (SBP), were observed among patients stratified by cirrhosis severity ($p < 0.05$). Patients in the decompensated stage (Child-Pugh C) were more likely to present with fever, suggesting an association between systemic inflammatory response and advanced liver dysfunction. These findings are consistent with those of Khan et al., who reported a higher incidence of SBP among patients in the decompensated phase of cirrhosis [3].

Further analysis indicated that although the incidence of SBP was higher in the Child-Pugh C group, the Child-Pugh classification itself was not identified as an independent risk factor for SBP. This observation supports the findings of Hu et al., who similarly noted the need for further investigation into the complex relationship between cirrhosis severity and SBP occurrence.

Interpretation of laboratory data must consider the retrospective, single-center nature of the study. Clinical and biochemical indicators at admission may have been influenced by prior medical management, including medication and nutritional support. Specifically, the use of antiviral agents (e.g., entecavir), hepatoprotective drugs (e.g., glutathione), albumin infusions, and nutritional supplementation may have altered levels of serum transaminases, total protein, albumin, TBA, and PLT. These effects may reduce or obscure the true differences among Child-Pugh groups, limiting the precision of disease severity assessment.

Additionally, pre-admission nutritional interventions, such as amino acid or albumin infusions, may confound indicators like APOA1, calcium, and PLT. Consequently, laboratory findings at the time of admission may reflect not only hepatic dysfunction but also the effects of recent treatments, complicating the interpretation of their correlation with cirrhosis severity.

Our analysis of cirrhosis etiology revealed no significant association between disease severity and factors such as hepatitis B, hepatitis C, cholestasis, malnutrition, or circulatory disorders ($p > 0.05$). However, due to incomplete data on certain etiologies within subgroups, these results should be interpreted cautiously. Further studies are needed to clarify the impact of specific etiologies on the clinical progression of cirrhosis.

Among laboratory variables, serum calcium (Ca^{2+}) was significantly higher in the Child-Pugh B and C groups than in the A group, although no significant difference was noted between the B and C groups ($p < 0.05$). This finding contrasts with prior work by Manrai et al., who reported a negative correlation between serum calcium and cirrhosis severity [8]. Such discrepancies may be attributable to improved clinical practices, early intervention, enhanced access to diuretics, and better nutritional status in modern patient populations.

CRP, a liver-synthesized acute-phase protein, increases rapidly in response to infection, tissue injury, and systemic inflammation, and is widely used in clinical practice as a biomarker of inflammatory activity [9]. Notably, CRP levels are generally unaffected by hormonal therapies or cytotoxic treatments, enhancing its reliability in various clinical settings. However, in this study, no significant difference in CRP levels was observed between the Child-Pugh B and Child-Pugh C groups, which contrasts with findings reported by Papp et al. [10]. These discrepancies may reflect variability in disease stage, presence of complications, or pre-hospital interventions, and underscore the need for further investigation into the inflammatory profiles of patients with advanced cirrhosis.

Variations in CRP levels among patients with cirrhosis may be influenced by multiple clinical and methodological factors. The inflammatory status can differ significantly between early and late stages of diagnosis, and between the acute decompensation phase and the more stable phase of disease. Additionally, the presence of complications such as spontaneous bacterial peritonitis or upper gastrointestinal bleeding may contribute to elevated CRP levels. Chronic comorbid conditions, including diabetes mellitus and cardiovascular disease, also add to the systemic inflammatory burden and may confound CRP interpretation. Moreover, prior or ongoing treatments, such as prophylactic antibiotics, immunosuppressive therapy, or nonsteroidal anti-inflammatory drugs, can alter inflammatory responses and suppress or exaggerate CRP expression, thereby impacting its diagnostic value.

Beyond clinical variables, non-biological factors may introduce further variability in CRP measurements. These include demographic differences (e.g., age, gender, race, and genetic background), as well as socioeconomic status and healthcare access. Technical variability in laboratory procedures-such as timing and conditions of sample collection, reagent specificity and sensitivity, and degree of standardization - can also affect measured CRP values. Regional differences in reference ranges and laboratory infrastructure may further complicate inter-study comparisons and generalizability.

Despite these limitations, CRP remains an important indicator of systemic inflammation in patients with cirrhosis. Persistent hepatocellular injury and fibrosis promote chronic inflammatory activation, and elevated CRP levels may reflect increased disease activity and portend poorer outcomes.

TBIL, another important marker included in the Child-Pugh scoring system, reflects hepatic capacity for bilirubin metabolism and excretion. In advanced cirrhosis, impaired hepatocellular function leads to elevated serum bilirubin levels, which are directly associated with disease severity.

Cholinesterase (CHE), predominantly synthesized by hepatocytes, serves as a sensitive marker of hepatic synthetic function. As liver cell mass and functional capacity decline with disease progression, CHE levels decrease accordingly. This makes CHE a valuable param-

eter for assessing liver reserve and prognosticating clinical outcomes in cirrhosis.

FIB, a large glycoprotein synthesized by hepatocytes, plays a vital role in the coagulation cascade. Its production declines in the setting of hepatic dysfunction, making it a useful indicator of liver synthetic capacity. TBAs, which include glycodeoxycholic acid, deoxycholic acid, and ursodeoxycholic acid, are synthesized by hepatocytes and normally reabsorbed in the ileum and colon, then recirculated via the portal vein to the liver for metabolism [11]. In the context of liver dysfunction, impaired bile acid clearance leads to elevated serum TBA levels, which reflect the degree of hepatic impairment.

APOA1, synthesized primarily in the liver and intestines, is a major structural component of high-density lipoprotein (HDL). Studies have suggested that serum APOA1 concentrations correlate with prognosis in cirrhosis [12-14]. Infections, which are common in patients with advanced cirrhosis (Child-Pugh C), may contribute to reduced HDL levels and thereby lower APOA1 concentrations.

DBIL is a byproduct of hemoglobin metabolism processed in the liver. Its elevation in cirrhosis reflects impaired hepatocellular excretion function. Bicarbonate (HCO^{3-}), which reflects systemic alkaline reserve, plays a key role in acid-base homeostasis.

CYSC, a low molecular weight protein expressed in multiple organs including the liver, lungs, stomach, pancreas, and placenta, has been proposed as a potential marker of renal and hepatic function [14]. In our study, no significant difference in CYSC levels was observed between the Child-Pugh A and B groups. However, pairwise comparisons revealed significant differences between both the Child-Pugh B and C groups, and the Child-Pugh A and C groups, suggesting that CYSC may be associated with more advanced stages of liver dysfunction.

PLT is another clinically relevant parameter, often reduced in cirrhosis due to hypersplenism, bone marrow suppression, or decreased thrombopoietin production. Additionally, impaired absorption of vitamin K, required for the synthesis of coagulation factors, may contribute to coagulopathy in liver disease [15]. In this study, significant differences in PLT levels were found across the three Child-Pugh groups ($p < 0.05$). Pairwise analysis indicated a significant difference between the Child-Pugh B and C groups ($p < 0.05$), while no significant differences were observed between the A and B groups or between the A and C groups. These results differ from some previously published findings [16]. Several factors may explain the observed variability in serum calcium levels among patients with cirrhosis. Nutritional status plays a key role; for instance, protein-energy malnutrition can impair vitamin D metabolism and calcium absorption. Patients with alcoholic cirrhosis are frequently magnesium-deficient, which disrupts calcium homeostasis. Gastrointestinal dysfunction may further compromise the absorption of calcium and vitamin D. In addition, regional and cultural differences in dietary calcium intake may contribute to interpatient variability.

Treatment-related factors also act as confounders. Diuretic use may increase renal calcium excretion, while long-term proton pump inhibitor therapy is known to impair calcium absorption. Corticosteroid use can negatively affect bone calcium metabolism. Moreover, prior use of calcium or vitamin D supplementation may influence baseline calcium levels and complicate their interpretation in the context of liver function.

Upon reviewing clinical data, several outliers were identified. Detailed examination revealed that a subset of patients had received hepatoprotective therapy, albumin supplementation, or nutritional support prior to admission. Analysis of historical laboratory records showed that total bilirubin and albumin levels decreased rapidly following administration of antiviral and hepatoprotective medications. In contrast, PLT either changed minimally or decreased, a pattern not entirely consistent with the expected progression of liver dysfunction. Given that the Child-Pugh score incorporates both total bilirubin and albumin, pre-hospital interventions may have influenced cirrhosis severity classification, potentially skewing group assignments.

The results of multivariate regression analysis indicated that abnormal levels of CRP, PLT, FIB, TBA, Ca^{2+}, APOA1, CHE, CYSC, and HCO^{3-} were significantly associated with cirrhosis severity. These indicators may serve as important adjuncts for clinical assessment and risk stratification in patients with chronic liver disease.

This study has several limitations. First, it utilized a retrospective, single-center design, which may limit the generalizability of the findings. Second, laboratory and clinical data collected at the time of admission may have been significantly influenced by pre-hospital interventions, including nutritional support and pharmacologic treatment. Such interventions can confound the interpretation of parameters related to liver function. For example, nutritional therapy may elevate serum albumin and APOA1 levels; lipid-lowering agents may reduce TBA and transaminase levels; blood transfusions may transiently stabilize PLT; and hepatoprotective medications may attenuate biochemical signs of liver injury. These effects may obscure true intergroup differences attributable solely to hepatic dysfunction.

Third, some data trends in this study, such as inconsistent decreases in platelet and calcium levels with worsening Child-Pugh classification, and the lack of a clear rise in CRP with advancing liver dysfunction, diverged from previous literature. These inconsistencies may be due to the aforementioned confounding factors, patient comorbidities, variable timing of blood sample collection, and relatively small sample size.

This variability underscores the importance of accounting for pre-existing treatments, nutritional status, and sampling conditions when interpreting clinical and biochemical data in cirrhosis research. Future studies should aim to reduce these confounding influences by

employing large-sample, multicenter, prospective designs. Detailed documentation of patients' medication history and nutritional interventions prior to admission will facilitate appropriate stratification and adjustment. Such rigor will help elucidate the underlying mechanisms driving changes in laboratory and clinical markers of liver dysfunction and will support the development of more accurate and individualized diagnostic and therapeutic strategies.

CONCLUSION

In summary, this study demonstrates that the presence of fever during the decompensated phase may serve as a clinical indicator of advanced cirrhosis. Laboratory findings revealed that cirrhosis severity is positively correlated with TBA and DBIL levels, and negatively correlated with FIB, APOA1, CHE, and HCO^{3-}. Additionally, abnormal levels of CRP, PLT, Ca^{2+}, CYSC, and the aforementioned markers may serve as valuable indicators for evaluating the cirrhosis severity. These findings may contribute to improving risk stratification and guiding clinical decision-making in patients with chronic liver disease.

Ethics Approval and Consent to Participate:
This study was conducted with approval from the Ethics Committee of Zhengzhou People's Hospital (No. 20230 11141). This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

Source of Fund:
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Data Availability:
The datasets used or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of Interest:
The authors declare that they have no conflict of interest regarding this work.

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