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Original Research Article

Coagulation Profile and Its Correlation with the Severity of Liver Dysfunction in Chronic Liver Disease: A Cross-Sectional Study from Central India

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Conflict of interest: Nil

Abstract

Background: The liver synthesises most coagulation factors and natural anticoagulants, so chronic liver disease (CLD) disturbs haemostasis. This study assessed the coagulation profile in CLD and its correlation with severity of hepatic dysfunction.

Methods: A hospital-based cross-sectional observational study was conducted at a tertiary care teaching hospital in Bhopal, central India, over 18 months. One hundred and eighty consecutive adults with CLD were enrolled. Prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), platelet count and liver function tests were measured. Severity was graded by Child–Pugh class and Model for End-stage Liver Disease (MELD) score. One-way analysis of variance with Tukey post hoc testing and Spearman rank correlation were applied.

Results: Most participants were aged 31–45 years (37.8%) and male (71.1%); alcohol-related liver disease was the commonest aetiology (54.4%). Elevated INR was the most frequent abnormality (71.1%), followed by thrombocytopenia (60.0%), prolonged PT (57.8%) and prolonged aPTT (55.0%). All parameters worsened progressively across Child–Pugh classes, INR rising from 1.33 ± 0.29 in Class A to 2.56 ± 0.63 in Class C, and across MELD categories ($p < 0.001$ for all). INR correlated most strongly with MELD score ($r = 0.62$), followed by albumin ($r = −0.59$), PT ($r = 0.57$) and platelet count ($r = −0.54$); all $p < 0.001$.

Conclusion: Coagulation abnormalities are highly prevalent in CLD and deteriorate predictably with worsening hepatic dysfunction. Routine PT, INR, aPTT and platelet count offer an inexpensive, widely available means of severity assessment and risk stratification.

Keywords: Chronic Liver Disease; Coagulation Profile; Child–Pugh Score; MELD Score; International normalized ratio; Thrombocytopenia.

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Introduction

Chronic liver disease (CLD) accounts for more than two million deaths worldwide each year, of which cirrhosis contributes approximately one million (Asrani et al., 2019; Devarbhavi et al., 2023). The burden continues to rise with the growing prevalence of viral hepatitis, alcohol use, obesity and the metabolic syndrome (GBD 2017 Cirrhosis Collaborators, 2020), and in India alcohol-related liver disease and non-alcoholic fatty liver disease (NAFLD) now rival chronic viral hepatitis as leading causes (Mackowiak et al., 2024; Younossi, 2019). Because hepatocytes synthesise almost all coagulation factors together with the natural anticoagulants protein C, protein S and antithrombin, loss of hepatic synthetic capacity disturbs haemostasis in both directions (Tripodi & Mannucci, 2011). Primary haemostasis is compromised by thrombocytopenia and qualitative platelet dysfunction, secondary haemostasis by reduced factor synthesis and impaired clearance of activated factors (Northup & Caldwell, 2013). Patients were long regarded as predominantly bleeding-prone, but pro-coagulant and anticoagulant pathways in fact decline in parallel, producing a rebalanced equilibrium that is less stable than in health and may tip towards either

bleeding or thrombosis (Lisman & Porte, 2010). Coagulation variables nonetheless remain central to severity assessment and are embedded in both instruments in routine use: prothrombin time (PT) in the Child–Pugh classification (Pugh et al., 1973) and the international normalized ratio (INR) in the Model for End-stage Liver Disease (MELD) score (Kamath et al., 2001). PT and INR are sensitive to hepatic reserve because factor VII, synthesised exclusively in the liver, has the shortest half-life of the clotting factors, while the activated partial thromboplastin time (aPTT) lengthens in more advanced disease.

How closely these inexpensive and universally available tests track hepatic dysfunction varies with the population studied, the aetiological mix and the stage at presentation, and published data from central India are sparse. We therefore measured PT, INR, aPTT and platelet count in consecutive patients with CLD attending a tertiary care hospital in Bhopal and examined their relationship with severity as graded by Child–Pugh class and MELD score.

Materials and Methods

This single-centre, hospital-based, cross-sectional observational study was carried out in the Department of General Medicine, L.N. Medical College and Research Centre & J.K. Hospital, Bhopal, Madhya Pradesh, India, Adults aged 18 years or older with an established diagnosis of chronic liver disease were eligible for inclusion.

Patients with a known inherited or acquired bleeding or thrombotic disorder, current or recent pregnancy, recent transfusion of blood products, or ongoing anticoagulant therapy were excluded. Eligible patients were enrolled consecutively until the target of 180 was reached. This figure was derived using Slovin's formula, $n = N / (1 + Ne^2)$, taking a 5% margin of error at a 95% confidence level, where N was the estimated number of patients with CLD attending the centre during the study period.

Sociodemographic details — age, sex, education, occupation, area and type of residence, and socioeconomic status by the modified Prasad classification — were recorded on a structured case record proforma, together with presenting complaints and clinical findings including ascites, jaundice, hepatic encephalopathy, gynaecomastia, loss of body hair and any history of bleeding. Aetiology was classified as alcohol-related liver disease, viral hepatitis or NAFLD/other on the basis of [state the criteria applied, including the alcohol intake threshold and the serological tests used]. All patients underwent abdominal ultrasonography as part of routine evaluation. Severity was graded in two ways: by the Child–Pugh score, derived from grade of encephalopathy, severity of ascites, serum bilirubin, serum albumin and prothrombin time prolongation or INR, with patients classified as Class A (5–6 points), Class B (7–9 points) or Class C (10–15 points); and by the MELD score, calculated as $9.57 \times \ln(\text{creatinine}) + 3.78 \times \ln(\text{total bilirubin}) + 11.2 \times \ln(\text{INR}) + 6.43$ and categorised as low (< 10), moderate (10–19) or high (≥ 20).

Venous blood for coagulation testing was collected into blue-top vacutainers containing 3.2% trisodium citrate at a ratio of nine volumes of blood to one volume of anticoagulant. Platelet-poor plasma was separated by centrifugation at 2000 g for 10 minutes and assayed immediately, or stored in cuvettes at −20 °C to −40 °C until testing. PT, INR and aPTT were determined by conventional methods on [analyser and reagent], and D-dimer was also assayed. A further 2 mL of blood collected into EDTA was used for a complete blood count including platelet estimation. Serum total and direct bilirubin, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase were measured by standard laboratory methods. Results were classified as normal or abnormal against the reference ranges of the institutional central laboratory [state the ranges applied for PT, INR and aPTT].

Data were entered into Microsoft Excel, checked for completeness and analysed using IBM SPSS Statistics version 27.0. Continuous variables are summarised as mean ± standard deviation and categorical variables as counts and percentages. Coagulation parameters and liver function tests were compared across Child–Pugh classes and MELD categories by one-way analysis of variance with Tukey honestly significant difference post hoc testing, and their association with MELD score was assessed by Spearman rank correlation. A two-sided p value below 0.05 was considered statistically significant.

Results

Of the 180 patients enrolled, most were aged 31–45 years (37.8%) and male (128 of 180, 71.1%), a male-to-female ratio of 2.46:1. Just over half lived in urban areas (57.8%) and two-thirds belonged to socioeconomic Class II–III (65.0%). Ascites was the commonest clinical finding (67.2%), followed by jaundice (58.9%) and loss of body hair (42.2%); hepatic encephalopathy was present in 31.1% and a history of bleeding in 17.8%. Alcohol-related liver disease accounted for 54.4% of cases, viral hepatitis for 27.2% and NAFLD or other causes for 18.3% (Table 1).

Most patients presented with moderate or advanced hepatic dysfunction: 41.7% were Child–Pugh Class B and 31.1% Class C, leaving 27.2% in Class A. The mean MELD score was 16.3 ± 6.1, with half

the cohort (50.6%) in the moderate range of 10–19 and 26.1% scoring 20 or above (Table 2).

Coagulation abnormalities were common. INR was raised in 71.1% of patients (mean 2.01 ± 0.67), platelet count reduced in 60.0% (147 ± 58 × 10^3/μL), PT prolonged in 57.8% (17.9 ± 4.3 seconds) and aPTT prolonged in 55.0% (43.8 ± 7.9 seconds). Hypoalbuminaemia was the commonest biochemical abnormality (77.8%; mean 2.8 ± 0.6 g/dL), followed by raised AST (70.0%), hyperbilirubinaemia (65.6%) and raised alkaline phosphatase (62.8%) (Table 3).

Every coagulation parameter deteriorated significantly and stepwise across Child–Pugh classes (Table 4, Panel A). INR rose from 1.33 ± 0.29 in Class A to 2.56 ± 0.63 in Class C and PT from 14.6 ± 2.4 to 21.8 ± 5.2 seconds (both $p < 0.001$), aPTT lengthened from 37.8 ± 5.0 to 48.6 ± 8.3 seconds ($p = 0.002$), and platelet count fell from 199 ± 53 to 91 ± 42 × 10^3/μL ($p < 0.001$). The gradient across MELD categories was of similar direction and magnitude for all four parameters ($p < 0.001$ for each; Panel B). On Spearman rank correlation, INR tracked the MELD score most closely ($r = 0.62$), followed by albumin ($r = −0.59$), PT ($r = 0.57$), platelet count ($r = −0.54$), total bilirubin ($r = 0.53$), AST ($r = 0.44$) and aPTT ($r = 0.42$); all correlations were significant at $p < 0.001$ (Panel C).

Table 1: Sociodemographic, clinical and aetiological profile of the study participants (n = 180)

Domain	Category	Frequency (n)	Percentage (%)
Sociodemographic characteristics			
Age (years)	18–30	40	22.2
	31–45	68	37.8
	46–60	52	28.9
	> 60	20	11.1
Sex	Male	128	71.1
	Female	52	28.9
Residence	Urban	104	57.8
	Rural	76	42.2
Socioeconomic status	Class II–III	117	65.0
(modified Prasad)	Class IV–V	63	35.0
Education	Illiterate	27	15.0
	Primary	54	30.0
	Secondary and above	99	55.0
Occupation	Unskilled / labourer	72	40.0
	Skilled / business	63	35.0
	Professional / salaried	45	25.0
Clinical features			
Ascites	Present	121	67.2
Jaundice	Present	106	58.9
Loss of body hair	Present	76	42.2
Hepatic encephalopathy	Present	56	31.1
Gynaecomastia	Present (males only)	38	29.7[a]
History of bleeding episodes	Present	32	17.8
Aetiology of chronic liver disease			
Alcohol-related liver disease	—	98	54.4
Viral hepatitis (HBV/HCV)	—	49	27.2
NAFLD / other	—	33	18.3

[a]Percentage calculated among male participants only (38 of 128 males); all other percentages are of the total cohort (n = 180). HBV, hepatitis B virus; HCV, hepatitis C virus; NAFLD, non-alcoholic fatty liver disease.

Table 2: Severity of liver dysfunction by Child–Pugh class and MELD score (n = 180)

Severity index	Category (definition)	Frequency (n)	Percentage (%)
Child–Pugh class	A (5–6 points)	49	27.2
	B (7–9 points)	75	41.7
	C (10–15 points)	56	31.1
MELD category	Low (< 10)	42	23.3
	Moderate (10–19)	91	50.6
	High (≥ 20)	47	26.1

Overall mean MELD score for the cohort: 16.3 ± 6.1. MELD, Model for End-stage Liver Disease.

Table 3: Coagulation profile and liver function tests of the study participants (n = 180)

Parameter	Mean ± SD	Abnormality defined as	n abnormal	% abnormal
Coagulation parameters				
Prothrombin time (seconds)	17.9 ± 4.3	[laboratory reference]	104	57.8
INR	2.01 ± 0.67	[laboratory reference]	128	71.1
aPTT (seconds)	43.8 ± 7.9	[laboratory reference]	99	55.0
Platelet count (× 10^3/µL)	147 ± 58	[laboratory reference]	108	60.0
Liver function tests				
Total bilirubin (mg/dL)	3.5 ± 2.3	> 1.2	118	65.6
Direct bilirubin (mg/dL)	2.2 ± 1.7	> 0.3	110	61.1
Albumin (g/dL)	2.8 ± 0.6	< 3.5	140	77.8
AST / SGOT (U/L)	87 ± 43	> 40	126	70.0
ALT / SGPT (U/L)	63 ± 36	> 40	105	58.3
ALP (U/L)	180 ± 76	> 120	113	62.8

INR, international normalized ratio; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; SD, standard deviation. Authors to insert the institutional laboratory cut-offs applied for PT, INR, aPTT and platelet count.

Table 4: Coagulation parameters stratified by Child–Pugh class and MELD category, and correlation of coagulation and biochemical parameters with MELD score

Panel A. Coagulation parameters by Child–Pugh class				
Parameter	**Class A (n = 49)**	**Class B (n = 75)**	**Class C (n = 56)**	**p-value**
INR	1.33 ± 0.29	1.94 ± 0.45	2.56 ± 0.63	< 0.001*
PT (seconds)	14.6 ± 2.4	18.3 ± 3.6	21.8 ± 5.2	< 0.001*
aPTT (seconds)	37.8 ± 5.0	43.1 ± 6.6	48.6 ± 8.3	0.002*
Platelet count (× 10^3/µL)	199 ± 53	143 ± 39	91 ± 42	< 0.001*
Panel B. Coagulation parameters by MELD category				
Parameter	**Low, < 10 (n = 42)**	**Moderate, 10–19 (n = 91)**	**High, ≥ 20 (n = 47)**	**p-value**
INR	1.10 ± 0.09	1.94 ± 0.32	2.89 ± 0.52	< 0.001*
PT (seconds)	12.90 ± 2.05	18.82 ± 4.52	20.59 ± 5.56	< 0.001*
aPTT (seconds)	37.47 ± 5.37	43.80 ± 7.83	49.26 ± 8.69	< 0.001*
Platelet count (× 10^3/µL)	206.52 ± 63.72	142.86 ± 49.70	101.74 ± 41.52	< 0.001*
Panel C. Spearman rank correlation with MELD score				
Variable	**Spearman r**	**p-value**	**Interpretation**	**—**
INR	0.62	< 0.001	Strong positive	
PT (seconds)	0.57	< 0.001	Moderate positive	
aPTT (seconds)	0.42	< 0.001	Moderate positive	
Platelet count (× 10^3/µL)	−0.54	< 0.001	Moderate negative	
Total bilirubin (mg/dL)	0.53	< 0.001	Moderate positive	
Albumin (g/dL)	−0.59	< 0.001	Moderate negative	
AST / SGOT (U/L)	0.44	< 0.001	Moderate positive	

Values in Panels A and B expressed as mean ± SD. *Statistically significant at $p < 0.05$; one-way ANOVA followed by Tukey HSD post hoc test. Panel C: Spearman rank correlation coefficient. INR, international normalized ratio; PT, prothrombin time; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; MELD, Model for End-stage Liver Disease.

Discussion

In 180 consecutive patients with chronic liver disease at a tertiary centre in central India, conventional coagulation tests were abnormal in the majority and deteriorated in a graded, statistically significant fashion as hepatic dysfunction advanced, whether severity was measured by Child–Pugh class or by MELD score. INR was both the most frequently abnormal parameter and the one that tracked disease severity most closely.

The cohort was young, predominantly male and largely alcohol-related, which is characteristic of CLD in this region. The concentration of cases in the 31–45 year group reflects the interval between the onset of sustained alcohol use or viral infection and clinically evident liver disease, a pattern also reported by Devrajani et al. (2012) in Pakistan and by Kotadiya et al. (2019) and Bhatia et al. (2017) in India. The male-to-female ratio of 2.46:1 is close to the 72% male proportion documented by Devrajani et al. (2012) and to the preponderance reported by

Deshmukh and Raut (2025) and Kumar (2025); Bohania et al. (2021), studying alcoholic liver disease specifically, described an entirely male cohort. Alcohol accounted for 54.4% of cases here, consistent with the systematic review of Indian cirrhosis aetiology by Swaroop et al. (2024) and the multicentric survey of Mukherjee et al. (2017), while the urban predominance accords with the high community prevalence of NAFLD reported from Bhopal by Singhai et al. (2023).

The prevalence of individual abnormalities sits within the published range. Franchini and Mannucci (2025) reported prolonged PT and elevated INR in 60–80% of patients with advanced liver disease, bracketing the 57.8% and 71.1% observed here, and Devrajani et al. (2012), Kotadiya et al. (2019), Bhatia et al. (2017) and Deshmukh and Raut (2025) all describe PT prolongation in roughly two-thirds to three-quarters of cases. Thrombocytopenia in 60.0% is comparable to the 60–70% of Mackavey and Hanks (2013) and the 68% of Majerović et al. (2016), and is attributable to hypersplenism from portal hypertension, reduced thrombopoietin production and increased platelet destruction (Gallo et al., 2022; Mitchell et al., 2016). Hypoalbuminaemia, present in 77.8%, exceeded every coagulation abnormality in frequency, which is unsurprising given that albumin is synthesised exclusively in the liver and has a half-life of roughly three weeks. A history of bleeding, by contrast, was elicited in only 17.8% — close to the 15–20% reported by McMurry et al. (2021) and Rautou et al. (2023), and a useful reminder that laboratory derangement and clinical haemorrhage are far from equivalent.

The severity gradient is the principal finding and is consistent across both scoring systems and all four parameters. Adam et al. (2020) observed the same pattern of reduced platelet counts, prolonged PT and elevated INR in Child–Pugh Class B and C relative to Class A, as did Majerović et al. (2016) and Zermatten et al. (2020). Bohania et al. (2021) found that increasing severity was accompanied not only by prolonged PT and aPTT and falling platelet counts but also by depletion of protein C, protein S and antithrombin III, and Lv et al. (2023), in 1,522 patients with cirrhosis and splenomegaly, showed that graded coagulation dysfunction predicted surgical outcome and mortality.

An important qualification applies to the strength of these associations. PT or INR contributes to the Child–Pugh score and INR to the MELD formula, so a correlation between INR and either index is in part arithmetic rather than biological. The independent signal therefore lies in aPTT and platelet count, neither of which enters either score, or both of which nonetheless separated the severity strata cleanly (aPTT $p = 0.002$ across Child–Pugh classes and $p < 0.001$ across MELD categories; platelet count $p < 0.001$ for both). That these parameters move in step with hepatic dysfunction is the more informative observation. A second qualification concerns what the tests actually measure. PT and aPTT interrogate pro-coagulant pathways alone and are blind to the parallel loss of protein C, protein S and antithrombin that characterises liver disease (Lisman & Porte, 2010). A proportion of patients accordingly remain prothrombotic despite prolonged clotting times, with portal vein thrombosis reported in 10–25% of those with cirrhosis (Franchini & Mannucci, 2025; Rautou et al., 2023).

The practical implication is twofold. PT, INR, aPTT and platelet count are inexpensive, rapidly reported and available in essentially every hospital laboratory, and their consistent relationship with both Child–Pugh class and MELD category makes them serviceable adjuncts for severity assessment and risk stratification where viscoelastic testing is unavailable. At the same time, a prolonged PT or raised INR in a patient with CLD should be read as a marker of hepatic synthetic reserve rather than as a prediction of bleeding, and should not on its own justify prophylactic transfusion of plasma or platelets before an invasive procedure.

Several limitations qualify these findings. The study was conducted at a single tertiary centre with a case mix weighted towards decompensated disease, so the prevalence figures are unlikely to generalise to primary or secondary care. The cross-sectional design establishes association rather than causation and captures a single time point in a dynamic process. No healthy control group was recruited. D-dimer was assayed but complete data were unavailable for the full cohort, so it is not reported here. Viscoelastic tests such as thromboelastography and rotational thromboelastometry, which characterise global haemostatic function, were not performed, nor were the natural anticoagulants measured. Nutritional status, vitamin K deficiency and intercurrent infection were not systematically assessed and may have influenced coagulation results.

Analysis of variance assumes approximately normal distributions, which coagulation variables often violate, and the choice of Spearman rather than Pearson correlation implies that the data were treated as non-parametric elsewhere; a non-parametric group comparison would have been more internally consistent. No adjustment was made for multiple comparisons, although the magnitude of the p values makes it unlikely that the principal findings are affected. Finally, aetiological subgroups were not analysed separately and no follow-up for bleeding, thrombosis or mortality was undertaken.

Conclusion

Coagulation abnormalities are highly prevalent in chronic liver disease and worsen in a graded, statistically significant manner with advancing hepatic dysfunction on both the Child–Pugh and MELD scales.

Because PT and INR contribute to those scores, the most informative associations are those of aPTT and platelet count, which are not score components yet separated the severity strata just as clearly. Routine coagulation testing is therefore a practical and inexpensive adjunct to severity assessment in resource-limited settings, provided the results are interpreted as indices of hepatic synthetic reserve rather than as predictors of bleeding risk.

Declarations

Ethics Approval and Consent to Participate: The study was approved by the Institutional Ethics Committee of L.N. Medical College and Research Centre, Bhopal (LNMC & RC/Dean/2024/Ethics/290). Written informed consent was obtained from all participants.

Consent for Publication: Not applicable. No individually identifiable participant data are reported.

Availability of Data: The de-identified dataset analysed during the current study is available from the corresponding author on reasonable request.

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