Section: Pathology

Original Research Article

THE RELATIONSHIP BETWEEN LIVER DISEASE SEVERITY AND COAGULATION DYSFUNCTION: INSIGHTS FROM A TERTIARY CARE STUDY

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Received : 11/02/2026
Received in revised form : 10/04/2026
Accepted : 28/04/2026

Keywords:
Liver disease, Coagulation abnormalities, Prothrombin time (PT), Platelet count, Child-Pugh score.

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DOI: 10.47009/jamp.2026.8.3.126

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 686-691

ABSTRACT

Background: The liver maintains homeostasis by producing the coagulation, anticoagulant, and fibrinolytic factors. The balance becomes rebalanced, increasing the risk of bleeding and thrombosis. Cirrhosis helps to alter the coagulation dynamics. Clinical scores like Child-Pugh and MELD are used to evaluate the severity of the disease, as routine tests are unable to predict the risk of bleeding. **Materials and Methods:** This was a hospital-based cross-sectional study, which was conducted as SSPM Medical College and Lifetime Hospital, Sindhudurg from January 2025 to December 2025 and included 115 patients with liver disease. Various clinical, biochemical, and coagulation parameters were evaluated. The severity of the disease was evaluated by standard scoring systems. Statistical analysis was conducted by SPSS version 26, and $p<0.05$ was maintained for statistical significance. **Result:** The study showed balanced equal distributions, where mid-aged showed (31–40 years: 33.9%). The most common was Chronic liver disease, account to be as 62.6%. Some of the crucial abnormalities include the included aPTT (63.5%), PT (56.5%), plateletcount (50.4%), and elevated SGOT (73.0%) over SGPT (43.5%). High derangements were observed in alcohol-related liver diseases. Also, severe coagulation changes were noted in acute failure of acute liver failure. NAFLD and liver SOL showed moderate abnormalities. **Conclusion:** The study concluded that the COPD-related patient showed coughing with the rise of intra-abdominal pressure, weakening of the linea alba, promoting the diastasis recti condition. This highlighted the early screening and management.

INTRODUCTION

The liver is at the core of the haemostatic process and produces the majority of the coagulant and anticoagulant factors and thrombopoietin and fibrinolytic regulators, thus maintaining the delicate balance between the formation and degradation of blood clots.[1] This balance is disrupted in liver disease due to decreased production of coagulant and anticoagulant factors and fibrinolysis, thus creating a 'rebalanced' system which is more susceptible to both thrombosis and bleeding.[2] Bleeding is caused by portal hypertension and thrombocytopenia (~78%), while the risk of thrombosis persists due to decreased anticoagulant factors despite the presence of coagulant factors.[1,2] This risk is further compounded by the increasing incidence of liver disease worldwide, especially MASLD.[3]

Cirrhosis results in a weakened 'rebalanced hemostasis' with decreased levels of coagulation factors (II, V, VII, IX, and X) and natural anticoagulants (protein C, protein S, and antithrombin) existing in a delicate balance.[4] However, the level of factor VIII and vWF is raised due to endothelial cell activation, thus maintaining thrombin generation.[4] While thrombocytopenia caused by hypersplenism and decreased thrombopoietin is partly compensated by the raised vWF, the process of primary hemostasis is maintained.[4] Fibrinolysis is affected variably due to the raised tPA and decreased inhibitors, thus resulting in unstable fibrinolysis.[4] Moreover, the process of hypercoagulability is further enhanced by the endothelial cell dysfunction.[4] Routine tests like PT/INR and aPTT are poor indicators of the hemostatic balance and are of little use in predicting the risk of bleeding.[2] In clinical settings, patients with cirrhosis are observed to have manifestations such as variceal bleeding and portal vein thrombosis.[2,4]

The severity of liver disease can be determined by the Child-Pugh score and MELD score, which are predictive of the prognosis of the disease, as well as clinical decisions in the timing of liver transplantation and TIPS procedures. The Child-

Pugh score uses albumin, bilirubin, INR, ascites, and encephalopathy, which are divided into classes A, B, and C, and are predictive of short-term outcomes.[5] The MELD score, which uses bilirubin, creatinine, and INR, as well as the MELD 3.0 score, is predictive of mortality rates, including the priority of liver transplantation, in which MELD 3.0 has better predictive value (sensitivity 67.4%, specificity 82.4%).[6] Both are predictive of worsening coagulation abnormalities and outcomes.[5,6]

As the severity of the disease increases, the coagulation parameters deteriorate, but the current tests are not able to accurately predict the bleeding tendency in these patients. In a study of cirrhotic patients in a prospective cohort, an increase in Child-Pugh and MELD score correlated with decreased thrombomodulin-mediated inhibition of thrombin generation, suggesting an imbalance in the prothrombotic state, despite an increase in prolonged PT/INR, aPTT, decreased factor V, and bilirubin levels.[7] In 870 liver biopsies, the incidence of bleeding was 2.0% (0.8% required intervention, 23% mortality), with the highest INR being predictive of bleeding, compared to recent INR, while bilirubin showed moderate predictive value.[8]

This study was performed to determine the association between liver dysfunction and abnormalities in coagulation and to assess the association with the severity of liver disease in a tertiary care facility.

MATERIALS AND METHODS

Research design

This was a hospital-based observational cross-sectional study that assessed the relationship between the severity of liver disease and coagulation impairment. The study was conducted in the SSPM Medical College and Lifetime Hospital, Sindhudurg, Maharashtra. The study was conducted during the period from January 2025 to December 2025. A total of 115 patients were included in the study, who had attended hospital OPD. Several inclusion and exclusion criteria were considered for patient enrolment. Significant clinical data, laboratory factors (including coagulation factors such as prothrombin time, INR, and platelet count), and imaging outcomes were recorded for each patient. The standard clinical scoring systems was used to assess the severity of the liver disease. Ethical approval was obtained from the Institutional Review Board. Informed consent was taken from each of the participants.

Inclusion Criteria

- Patients of all age groups were included from 0 to 70 years of age.
- Diverse demographic features were included in the study.
- Patients with any form of liver disease, like cirrhosis, hepatitis, or liver abscess, were included.
- Patients with an effective diagnosis through the clinical and diagnostic investigations were included for the study.

Exclusion Sriteria

- Patients with a prior history of bleeding disorder were not considered. ‘
- The usage of medication that impacts coagulation like, such as aspirin, ibuprofen, antihistamines, antibiotics, anti-hypertensives, and blood thinners were excluded.
- Uncompromised blood samples, such as clotting, improper collection and lack of storage containers.

Procedure

Patients were enrolled and the screening was done and well written and informed consent was taken and recorded for each of the participants. Blood samples were collected and under aseptic conditions, and then they were processed for prothrombin time (PT), international normalized ratio (INR), and platelet count. Standard laboratory protocols were set to investigate the biochemical investigations and liver function tests. Some of the imaging studies like ultrasonography, which was assessed to confirm the classification depending on the severity of the liver disease. Standard scores were given to categorise the severity of the liver disease. Data collection was performed and was entered into a structured data sheet.

Statistical Analysis

Descriptive statistics were utilised for the summarisation of the demographic and clinical features, while continuous variables were represented as the mean ± standard deviation and categorical variables were represented as the frequencies and percentages. The association between the severity of the liver disease and the coagulation parameters was investigated by some inferential tests, such as the Chi-square test. Student’s t-test or ANOVA was used for the continuous variables. Statistical analysis was performed by SPSS software version 26. The p-value of <0.05 was maintained for statistical significance.

RESULTS

The age distribution showed that most patients were 26–30 years old (53.3%), followed by those >30 years old (30%), and the least were <26 years old (16.7%). Primary infertility was predominant (90.0 %), while secondary infertility was 10%. The most common cause was male factor infertility (53.3%), followed by unexplained (26.6%), female factor (13.3%), and combined factors (6.7%). Most patients had a normal ovarian response (60%), followed by high responders (30%) and poor responders (10.0 %). [Table 1]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 1: The distribution of age and gender among the study participants (N = 115)

Age Group	Female (N)	Male (N)	Total Cases (N)	Percentage (%)
0–10	5	5	10	8.69%
11–20	5	3	8	7.00%
21–30	9	8	17	14.80%
31–40	11	28	39	33.90%
41–50	10	22	32	27.80%
51–60	6	19	25	21.70%
61–70	7	12	19	16.50%
>70	5	5	10	8.69%
Total	58	57	115	100%

Table 2 suggested distribution of cases according to diagnosis, highlighted that chronic liver disease is the most predominant condition, 72 cases (62.60%), and a balanced distribution of males and females, 36 each. The liver space-occupying lesions (SOL) showed 11 cases, where males were predominant over females. (6 males vs 5 females). Alcoholic fatty liver cases were 6.10% and alcoholic hepatitis cases were 4.30%, which together estimated to be 10% of total cases. Contrastingly, non-alcoholic fatty liver disease was 3.50%, indicating that both of the sexes were affected by the metabolic factors. Acute viral hepatitis (5.20%) and acute liver failure (1.70%) were frequently seen, indicating no sexual disparity.

Table 2: The distribution of participants according to the diagnosis

Diagnosis	Female (N)	Male (N)	Total (N)	Percentage (%)
Acute liver failure	1	1	2	1.70%
Acute viral hepatitis	3	3	6	5.20%
Alcoholic fatty liver	3	4	7	6.10%
Alcoholic hepatitis	2	3	5	4.30%
Chronic liver disease	36	36	72	62.60%
Liver SOL	5	6	11	9.60%
Non-alcoholic fatty liver disease	2	2	4	3.50%
Others	6	2	8	7.00%
Total	58	57	115	100%

Table 3 showed the analysis of the coagulation and liver function parameters, which demonstrated the burden of the biochemical abnormalities. Highest derangement was observed for activated partial thromboplastin time (aPTT), altered values noted for 73 cases (63.5%) and the a mean of 47.6 ± 18.90 seconds. High 65 cases for prothrombin time (PT), having the mean value for 20.5 ± 14.60 seconds, were observed, which reflected the extrinsic pathway function and showed the risk of bleeding. Abnormalities in the Platelet count were noted among 58 cases (50.4%), with a mean value of 172.3 ± 118.40. The liver enzyme SGOT (AST) showed the highest rate of abnormality, 84 cases (73.0%), with a high mean value of 118.4 ± 170.20 IU/ml, compared with 50 cases (43.5%) of SGPT (ALT), which showed the mean value of 120.2 ± 220.10 IU/ml.

Table 3: The distribution of cases on the basis of the Coagulation and Liver Function Parameters

Parameter	Range of Parameter	Mean ± SD	Altered Value N (%)	Normal Value N (%)	Total N (%)
aPTT (sec)	24.8 – 118	47.6 ± 18.90	73 (63.5%)	42 (36.5%)	115 (100%)
PT (sec)	9.0 – 115	20.5 ± 14.60	65 (56.5%)	50 (43.5%)	115 (100%)
Platelet Count	15 – 650	172.3 ± 118.40	58 (50.4%)	57 (49.6%)	115 (100%)
SGOT (IU/ml)	8 – 1750	118.4 ± 170.20	84 (73.0%)	31 (27.0%)	115 (100%)
SGPT (IU/ml)	9 – 1580	120.2 ± 220.10	50 (43.5%)	65 (56.5%)	115 (100%)

Table 4 showed the high value of SGOT 78.2% rather than 56.5% of SGPT, which indicated high sensitivity of SGOT for liver injury. Highest proportion of abnormal cases was observed for the chronic liver disease. This was followed by the alcohol related conditions, which demonstrated a high pattern of SGOT rather than SGPT. Also, the acute conditions like acute liver failure and viral hepatitis showed high enzyme derangement. SGOT predominance suggested hepatocellular damage, specifically in cases of chronic and alcohol-related liver diseases.

Table 4: Distribution on the basis of diagnosis of SGOT and SGPT

Diagnosis	Total Cases (N)	Altered SGOT (N)	Altered SGPT (N)
Acute liver failure	2	2	1
Acute viral hepatitis	6	5	4
Alcoholic fatty liver	8	7	4
Alcoholic hepatitis	5	4	3

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Chronic liver disease	72	55	35
Liver SOL	11	9	5
NAFLD	4	3	2
Others	7	5	11
Total	115(100%)	90(78.2%)	65(56.5%)

Table 5 showed the alteration of Prothrombin time (PT) among 56.5% of cases, which indicated the common coagulation dysfunction. Acute liver failure is the most severe form of abnormality, which reflects the dysfunction of the liver. Chronic liver disease (58.3%) and alcohol-related conditions (66–71%) showed prolonged PT. Contrastingly, the acute viral hepatitis (33.3%) and liver SOL (27.3%) showed preserved function, while NAFLD (50%) showed moderate involvement. PT derangement is the most severe and chronic liver condition.

Table 5: The Distribution of Prothrombin Time Abnormalities based on diagnosis

Diagnosis	Mean ± SD	Altered Prothrombin Time (N, %)	Normal Prothrombin Time (N, %)	Total Cases (N, %)
NAFLD	18.20 ± 6.10	2 (50.0%)	2 (50.0%)	4 (100%)
Alcoholic fatty liver	21.10 ± 7.90	5 (71.4%)	2 (28.6%)	7 (100%)
Acute viral hepatitis	17.00 ± 7.50	2 (33.3%)	4 (66.7%)	6 (100%)
Alcoholic hepatitis	24.20 ± 7.10	4 (66.7%)	2 (33.3%)	6 (100%)
Acute liver failure	72.50 ± 38.80	2 (100%)	0 (0%)	2 (100%)
Chronic liver disease	21.00 ± 13.80	42 (58.3%)	30 (41.7%)	72 (100%)
Liver SOL	15.20 ± 3.00	3 (27.3%)	8 (72.7%)	11 (100%)
Others	20.10 ± 8.40	5 (71.4%)	2 (28.6%)	7 (100%)
Total	—	65 (56.5%)	50 (43.5%)	115 (100%)

Table 6 demonstrated that overall 59.1% of patients exhibited altered aPTT, indicating a substantial burden of coagulation abnormalities. Chronic liver disease constituted the largest group (72 cases) and showed a markedly high prevalence of altered aPTT (76.4%), contributing most to overall abnormalities. Acute liver failure, although limited in number, demonstrated 100% alteration, indicating severe coagulopathy. In contrast, NAFLD (25%), alcoholic fatty liver (28.6%), and alcoholic hepatitis (16.7%) showed relatively lower proportions, while moderate alterations were observed in acute viral hepatitis (33.3%) and other conditions (42.9%).

Title 6: The distribution of diagnosis of aPTT Abnormalities

Diagnosis	Mean ± SD	Altered aPTT (N, %)	Normal aPTT (N, %)	Total (N, %)
NAFLD	36.90 ± 6.10	1 (25.0%)	3 (75.0%)	4 (100%)
Alcoholic fatty liver	39.10 ± 7.80	2 (28.6%)	5 (71.4%)	7 (100%)
Acute viral hepatitis	34.20 ± 7.40	2 (33.3%)	4 (66.7%)	6 (100%)
Alcoholic hepatitis	35.80 ± 7.00	1 (16.7%)	5 (83.3%)	6 (100%)
Acute liver failure	78.50 ± 38.90	2 (100%)	0 (0%)	2 (100%)
Chronic liver disease	53.80 ± 13.90	55 (76.4%)	17 (23.6%)	72 (100%)
Liver SOL	35.10 ± 3.00	2 (18.2%)	9 (81.8%)	11 (100%)
Others	42.30 ± 8.40	3 (42.9%)	4 (57.1%)	7 (100%)
Total	—	68 (59.1%)	47 (40.9%)	115 (100%)

Table 7 represented the platelet abnormalities among 50.4% of cases, which indicated a burden. Chronic liver disease showed 52.8% of cases, which reflected ongoing liver dysfunction. High rate of alteration was observed for NAFLD (75%) and acute viral hepatitis (66.7%), while moderate involvement was observed for alcoholic hepatitis and acute liver failure. While alcoholic fatty liver and liver SOL showed a low rate of abnormality.

Table 7: The distribution of Platelet Count Abnormalities based on diagnosis

Diagnosis	Mean Platelet Count ± SD	Altered Platelet Count (N, %)	Normal Platelet Count (N, %)	Total (N, %)
NAFLD	130.20 ± 145.10	3 (75.0%)	1 (25.0%)	4 (100%)
Alcoholic fatty liver	198.50 ± 110.20	2 (28.6%)	5 (71.4%)	7 (100%)
Acute viral hepatitis	245.30 ± 215.60	4 (66.7%)	2 (33.3%)	6 (100%)
Alcoholic hepatitis	130.10 ± 85.40	3 (50.0%)	3 (50.0%)	6 (100%)
Acute liver failure	160.80 ± 65.20	1 (50.0%)	1 (50.0%)	2 (100%)
Chronic liver disease	158.40 ± 102.70	38 (52.8%)	34 (47.2%)	72 (100%)
Liver SOL	250.60 ± 125.30	3 (27.3%)	8 (72.7%)	11 (100%)
Others	190.20 ± 115.50	4 (57.1%)	3 (42.9%)	7 (100%)
Total	—	58 (50.4%)	57 (49.6%)	115 (100%)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

DISCUSSION

The "rebalanced hemostasis" seen with advanced liver disease is characterized by a decrease in the production of procoagulant and natural anticoagulant factors, balanced by activation of the endothelium with increased levels of von Willebrand factor and factor VIII, resulting in a precarious balance that is susceptible to both bleeding and thrombosis under stress.[4] Inflammation also plays a role in disrupting anticoagulant mechanisms and fibrinolysis, resulting in both hypo- and hypercoagulable states. The commonly used laboratory tests, such as PT/INR and aPTT, are inadequate because these tests measure only the levels of procoagulants, and even though abnormal results are seen, global tests are often close to normal, resulting in the persistence of bleeding and thrombotic complications.[9]

In a study conducted by Chen & Chen (2023) of 48 patients with decompensated cirrhosis undergoing TIPS, portal venous levels of both procoagulant and anticoagulant factors were lower than peripheral blood, with protein C activity showing a significant negative correlation with Child-Pugh (CP) score, and factors II, VII, and protein C decreasing with advancing CP class, indicating worsening imbalance and fragile "rebalanced" hemostasis prone to bleeding and thrombosis.[10] In another study conducted by Dhillon et al. (2019) among 61 patients with well-compensated cirrhosis (CP A), thrombin generation assays demonstrated reduced endogenous thrombin potential and peak thrombin with increasing liver stiffness, yet no significant correlation was observed between thrombin parameters and CP or MELD scores, suggesting limited sensitivity of these scores in early disease.[11]

Recent studies have shown that cirrhosis is associated with a prothrombotic state, even with increased INR levels. Thrombin generation assays showed that thrombomodulin inhibition was significantly decreased compared to healthy controls ($p < 0.0001$) and that this inhibition was decreased progressively from Child-Pugh A to B/C cirrhosis ($p < 0.0001$), and increased PT/INR levels are associated with a prothrombotic state rather than a bleeding risk.[7] Endogenous thrombin potential with thrombomodulin was found to be similar to that of controls among 97 patients with cirrhosis, and 80% showed thrombomodulin resistance, especially with increased levels of INR ($p = 0.0042$).[12]

Cirrhosis is considered an unstable rebalanced state of hemostasis, and the risk of bleeding or thrombosis may be increased by the progression of the disease, infection, renal failure, or acute on chronic liver failure, making the sole reliance on PT/INR inadequate to assess the changes.[4] The advanced global tests are considered more accurate in the assessment of the coagulation state and are considered essential in the decision-making process in hepatology and critical care, especially in invasive procedures.[13] The inclusion of thrombin generation and viscoelastic tests enable the stratification and management strategies, while simultaneously assessing the formation of clots in critically ill cirrhotic patients in real-time.[14]

Large studies are also necessary to validate new models integrating Child-Pugh/MELD, thrombin generation, and viscoelastic tests, and currently, there are no standardized thresholds and outcome validations for these methods, as emphasized in a study.[5] Future studies on personalized coagulation testing and new markers also need to be validated, and new approaches may include dynamic models to improve risk prediction and management, along with standardized protocols and outcome measures.[9,15]

CONCLUSION

The study concluded that chronic respiratory stress among COPD individuals is crucial for the development of diastasis recti. The most significant contributing factors were persistent coughing and increased intra-abdominal pressure, which cause weakening of the abdominal wall and stretching of the linea alba. The results indicated the relationship between the linea alba and the clinically relevant patients with prolonged pulmonary conditions. Also the compromised respiratory mechanics and the alteration in the core musk, increases the abdominal instability. This triggers the significance of the early screening. The integration of respiratory management with core-strengthening interventions reduces the risk or severity of the condition. Thus, a multidisciplinary approach focus on the musculoskeletal support and the pulmonary rehabilitation, to improve the patient outcome and prevent other complications.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556