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Study of Coagulation Profile in Patient with Liver Diseases

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ABSTRACT

Introduction: The liver is the primary site for the synthesis of coagulation factors and regulatory proteins. Hepatic dysfunction disrupts this haemostatic balance, predisposing patients to both bleeding and thrombotic complications. Chronic liver disease (CLD) and cirrhosis are significant global health burdens, necessitating a thorough understanding of these complex haemostatic alterations to improve clinical management.

Objectives: This study aimed to evaluate the coagulation profile in patients with liver disease, investigate the association between coagulation abnormalities and disease severity, and assess the risk of clinical bleeding.

Material and Methods: This hospital-based, prospective observational study was conducted from January 2024 to June 2025 at a medical college. A total of 100 clinically and radiologically confirmed liver disease patients (aged 20–70 years) were included. Coagulation parameters—Prothrombin Time (PT), Activated Partial Thromboplastin Time (aPTT), platelet count, and D-dimer—were measured using standard laboratory techniques and compared with existing literature.

Results: The study cohort comprised 79% males and 21% females, with a mean age of 54.4±13.1 years. Cirrhosis (49%) and alcoholic liver disease (30%) were the most common diagnoses. Haemostatic abnormalities were prevalent: 71% of patients exhibited prolonged PT, 84% had prolonged aPTT, 70% demonstrated thrombocytopenia, and 72% showed elevated D-dimer levels. A history of clinical bleeding was observed in 48% of patients, correlating with more pronounced coagulation derangements.

Conclusion: Liver disease, particularly cirrhosis, is associated with significant coagulation abnormalities, reflecting a rebalanced haemostatic state where synthetic impairment and increased fibrinolysis coexist. These profiles serve as valuable indicators of disease progression. Regular assessment of these parameters is essential for evaluating severity and guiding effective patient care.

Keywords: Coagulation Profile, Liver Disease, Prothrombin Time.

INTRODUCTION

The liver acts as a manufacturing facility responsible for the production of the majority of proteins involved in blood clotting. These include procoagulant factors such as fibrinogen, prothrombin, and factors V, VII, IX, X and XI as well as natural anticoagulants including protein C, protein S and antithrombin III. In addition, the liver synthesizes key components of the fibrinolytic system, thereby contributing to the dissolution of clots and maintenance of vascular integrity.[1] The liver

is responsible for the production of thrombopoietin (also known as the c-Mpl ligand), which is the primary physiological growth factor for megakaryocytes and platelet production. Furthermore, the liver serves as a storage site for essential elements required for haematopoiesis, including iron, vitamin B12, and folic acid. Another important function of the liver is the clearance of activated clotting factors, fibrin degradation products, and other circulating substances once they are no longer required, thereby preventing excessive coagulation and maintaining haemostatic balance. [2] Haemostasis consists of three major components: primary, secondary and fibrinolysis.[1]

Since the liver is involved in both coagulation and anticoagulation pathways, any hepatic dysfunction leads to disruption of this balance, resulting in significant haemostatic abnormalities. An impaired liver may have insufficient capacity to produce adequate quantities of clotting proteins. Even when synthesized, their functional activity may be compromised. Certain liver conditions may also lead to increased intravascular coagulation, resulting in reduced availability of clotting factors. Additionally, impaired clearance of activated clotting factors further contributes to haemostatic imbalance.[3] Bleeding in liver disease may result from decreased levels of haemostatic proteins synthesized by the liver. Multiple mechanisms contribute to coagulation abnormalities, including reduced synthesis of clotting factors and natural anticoagulants, vitamin K deficiency, production of abnormal clotting proteins, increased consumption and decreased clearance. Hyper fibrinolysis and in some cases, disseminated intravascular coagulation may also occur.[4]

Several laboratory investigations includes prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count and platelet function tests. Normally, PT ranges from 11–13 seconds, with mild prolonged and aPTT ranges from 26–40 seconds. Prolongation indicates impaired haemostasis; however, PT-INR does not always accurately predict bleeding risk and should be interpreted cautiously.[5] PT mainly reflects vitamin K–dependent clotting factors, whereas aPTT reflects intrinsic pathway factors.[6]

Coagulation followed by fibrinolysis results in decreased fibrinogen levels and increased fibrin degradation products. D-dimer is widely used in the diagnosis of thrombotic conditions.[7] In chronic liver disease, levels of natural anticoagulants such as antithrombin III, protein C and protein S are reduced further contributing to haemostatic imbalance.[8] Therefore, coagulopathy in liver disease represents a complex rebalanced haemostatic state rather than a purely hypo coagulable condition.[3] Normocytic normochromic anaemia is the most common anaemia seen in chronic liver disease and is often associated with chronic inflammation.[9]

The present study is therefore undertaken to evaluate the coagulation profile in patients with liver disease to better understand haemostatic abnormalities and improve patient care.

AIMS AND OBJECTIVES

The study aims to examine coagulation profile alterations in liver disease patients, assess the association between coagulation abnormalities and liver disease severity, evaluate bleeding risk, and compare findings with similar studies.

MATERIAL AND METHODS

This hospital-based, prospective observational study was conducted from January 2024 to June 2025 at Department of Pathology, Jawaharlal Nehru Medical College, Ajmer. A total of 100 clinically and radiologically confirmed liver disease patients (aged 20–70 years) were included. Patients without a liver disease diagnosis, pregnant women and individuals with Disseminated Intravascular Coagulation (DIC). Additionally, patients unwilling to provide complete medical history, those with prior coagulation disorders or taking medications like aspirin, NSAIDs, or anticoagulants, recent blood transfusion recipients (<7 days), and known malignancy cases were excluded. A written informed consent was taken from all patient included in the study. History taking, relevant clinical findings and routine laboratory investigations was done. Permission from the institutional ethical committee was taken for conducting the study.

Methodology: Venous blood samples were collected[10] using standard phlebotomy techniques. For complete blood count (CBC), 2ml of blood was drawn into a lavender-top tube containing K3EDTA anticoagulant (1.5mg/ml blood). CBC parameters, including hemoglobin, total leukocyte count, differential leukocyte count, platelet count, and red blood cell count, were analyzed using an automated analyzer (Sysmex XP-1000), with platelet counts confirmed manually. For coagulation profiling, blood was collected in a vacutainer with 3.2% sodium citrate without a tourniquet to avoid hemoconcentration. Prothrombin time (PT) samples were not refrigerated to prevent F-VII activation, while aPTT plasma was stored at 4°C for up to 4 hours to prevent F-V and FVIII degradation. D-dimer samples were collected in blue-top tubes with 3.2% sodium citrate[2].

Coagulation parameters—Prothrombin Time (PT), Activated Partial Thromboplastin Time (aPTT), platelet count, and D-dimer—were measured using standard laboratory techniques.

Prothrombin Time[11] : The prothrombin time measures the clotting time of recalcified plasma in the presence of an optimal concentration of tissue extract (thromboplastin) and indicates the overall efficiency of the extrinsic clotting system.

Reagents: Thromboplastin reagent: This contains Tissue factor and phospholipid.
Normal range of PT: -11 to 13.5 sec
International normalized ratio (INR): - by international sensitivity index chart was provided by the reagent manufacturer.
Normal range of INR: -1.0

aPTT (Activated partial thromboplastin time)[11] : The test measures the clotting time of plasma after the activation of contact factors and the addition of phospholipid and CaCl2 but without added tissue thromboplastin and so indicates the overall efficiency of the intrinsic pathway.

Reagents:[12] Ck priest, calcium chloride
D-Dimer[11] : Sandwich immunoluminometric assay; Use an anti-D-dimer monoclonal antibody to label ABEI, and use another monoclonal antibody to label FITC. Sample, Calibrator or Control, ABEI Label, FITC Label and magnetic micro beads coated with anti-FITC are mixed thoroughly and incubated at 37℃, forming a sandwich; after sediment in a magnetic field, decant the supernatant, then cycle washing for 1 time Subsequently, the starter reagents are added and a flash chemiluminescent reaction is initiated. The light signal is measured by a photomultiplier as RLU within 3 seconds and is proportional to the concentration of D-DIMER present in controls or samples.

Statistical analysis: The qualitative data was expressed in proportions and percentages and the quantitative data was expressed as mean and standard deviations. The appropriate test was applied using computer software (SPSS Trial version 23 and primer). Statistical significance was kept at $p<0.05$.

RESULTS

Table 1 : Age and Sex Wise Distribution of Cases with Liver Disease

		Number of Patients	Percentage
Age Range (years)	20-40	20	20
	41-60	41	41
	61-80	39	39
Mean Age (years)		**54.4±13.1**	
Sex Distribution	Male	79	79
	Female	21	21

Table 2 : Distribution of Patients According to Ultrasonographic Findings

USG	Number of Patients	Percentage
Gross Ascites with Hepatomegaly	60	60
Moderate Ascites	19	19
Mild Ascites	12	12
Ascites with Hepatosplenomegaly	6	6
Hetero-echoic lesion	3	3
Total	**100**	**100**

Table 3 : Distribution of Patients According to Clinical Diagnosis

Clinical Diagnosis	Number of Patients	Percentage
Cirrhosis	49	49
Alcoholic Liver Disease	30	30
Non-Alcoholic Liver Disease	9	9
Viral Hepatitis	9	9
Liver Abscess	3	3
Total	**100**	**100**

Table 4 : Mean Values of Liver Function and Coagulation Profile Parameters among Patients

Parameter		Mean	SD
LFT	AST (IU/L)	192.9	159.5
	ALT (IU/L)	101.8	76.5
	ALP (IU/L)	99	49.7
Coagulations Profile	PT (sec.)	25.62	15.54
	INR	2.95	3.83

	APTT (Sec.)	52.69	26.38
	Platelets Count (K)	174.21	122.62
	D-Dimer (µg/ml)	3.91	2.77

Table 5 : Distribution of Patients According to Prothrombin Time (PT) Severity; Activated Partial Thromboplastin Time Severity; Platelets Count Severity; and D-Dimer Levels

Parameter		Number	Percentage
Prothrombin Time (Sec.)	Normal (11 to 13)	29	29
	Mildly Prolonged (14-20)	54	54
	Moderately Prolonged (21-50)	18	18
	Severely Prolonged (≥51)	8	8
aPTT	Normal (25 -29)	16	16
	Mildly Prolonged (30 - 50)	59	59
	Moderately Prolonged (51-100)	15	15
	Severely Prolonged (>100)	10	10
Platelets Count (K)	Normal (150-400)	**30**	30
	Mild thrombocytopenia (75-150)	45	45
	Moderate thrombocytopenia (50-75)	13	13
	Severely thrombocytopenia (< 50)	12	12
D-Dimer (µg/ml)	Normal (≤ 0.5)	28	28
	Elevated (> 0.5)	72	72

Table 6 : Variations in Coagulation Parameters by Hepatic Pathology

Coagulation Parameters	Cirrhosis	Alcoholic Liver Disease	NALD	Viral Hepatitis	Liver Abscess
PT	Moderate	Normal	Normal	Normal/Mild	Mild
aPTT	Moderate	Mild	Normal	Normal	Mild
Platelets Count	Mild	Mild	Mild	Normal	Normal
D-dimer	Elevated	Normal	Normal	Normal	Normal

Table 7 : Correlation of Hemostatic Markers with Clinical Bleeding Status

Coagulation Parameters	Bleeders (n=48)	Non-Bleeders (n=52)
PT	Moderate	Normal
aPTT	Moderate	Mild
Platelets Count	Moderate	Mild
D-dimer	Elevated	Normal

DISCUSSION

The present study was conducted from January 2024 to June 2025 on 100 cases of liver diseased. The mean age observed was 54.42 ± 13.16 years, which was comparable to the study of Raza F et al. (2025)[14] (46.2 ± 14.5 years).

In the present study, males constituted 79% of the study population, while females accounted for 21%, demonstrating a marked male predominance among patients with liver disease. The gender distribution observed in the present study closely resembles that reported by Raut AA et al. (2025)[17] who documented 78% males and 22% females. **(Table 1)**

In the present study, cirrhosis was the most common liver disease, accounting for 49% of cases, followed by alcoholic liver disease at 30%. Non-alcoholic fatty liver disease (NAFLD/NASH) and viral hepatitis each contributed 9.00% of the cases, while liver abscess constituted 3.00% of the study population. In the present study, the prevalence of cirrhosis observed in the (49%) was highly. Similarly, Raut AA et al.[17] documented cirrhosis in 45% of patients, which closely resembles our findings. In the present study, the prevalence of alcoholic liver disease observed in (30%) was represented the second most common etiology. The prevalence observed in our study was higher than that reported by Melkamu A et al. (8.4%)[16]. In the present study, the prevalence of Non-alcoholic fatty liver disease (NAFLD/NASH) accounted for 9% of cases. The

prevalence observed in our study was higher than that reported by Mistry PA et al. (3%)[19]. In the present study, viral hepatitis accounted for 9% of cases. The prevalence was lower than that reported by Raja F et al. (24.2%)[14]. In the present study, Liver abscess accounted for 3% of cases was highly similar to the study by Shakyawal B et al (2.7%)[15]. **(Table 3)**

In the present study, the mean AST, ALT, and ALP levels were 192.9 IU/L, 101.8 IU/L, and 99 IU/L respectively. Mistry PA et al. (2024)[19] study reported mean AST and ALT levels of 123 IU/L and 124.6 IU/L, respectively. In contrast, the present study demonstrated substantially higher AST levels and a clear predominance of AST over ALT. In the present study, the mean AST value was found to be 192.9 IU/L which was markedly higher than the mean ALT value of 101.8 IU/L.

In the present study, the mean Prothrombin Time (PT) was 25.6 ± 15.5 seconds. Similarly, Agrawal KV et al. (2023)[20] reported 27±13 seconds . In the present study, the mean International Normalized Ratio (INR) was 2.9 ± 3.8 was higher than the values reported by Aryaprabha et al. (2023)[21] (1.7 ± 2.6). In the present study the mean Activated Partial Thromboplastin Time (APTT) was 52.6 ± 26.3 seconds. Similarly, study by Mistry PA et al. (2024)[19] (48.9 ± 19.3 seconds). In the present study, the mean platelet count was 174.2±122.6 × 10^3/µL which was similar to the study by Mistry PA et al. (2024)[19] (177.8 ± 121.2× 10^3/µL). In the present study, the mean D-dimer level was 3.9 ± 2.7 µg/mL, similarly reported by Agrawal KV et al. (2023)[20] (3.7 ± 3.9 µg/mL). Overall, the coagulation profile observed in the present study indicates significant impairment of hemostatic function, particularly among patients with advanced liver dysfunction and cirrhosis. **(Table 4)**

In the present study, based on severity grading, 30.0% of patients had normal PT values (11–13 seconds), while 70.0% demonstrated varying degrees of PT prolongation. Mild prolongation (14–20 seconds) was observed in 49.0% of cases, moderate prolongation (21–50 seconds) in 15.0% and severe prolongation (>51 seconds) in 6.0% of patients. When compared with previous studies, the mean PT in the present study (25.62 ± 15.53 seconds) was higher than that reported by Patil AY et al. (2020)[6] (18.50 ± 11.56 seconds). The proportion of patients with normal PT values in the present study (30.0%) was identical to that reported by the findings of Kotadiya TP et al. (2018)[23] (25.0%). In the present study, the combined prevalence of mild and moderate PT prolongation was 64.0%, which is comparable to the proportions reported by Bhatia G et al. (2017)[24] (62.0%). **(Table 5)**

In the present study, only 16.0% of patients had normal APTT values (25–29 seconds), whereas 84.0% demonstrated varying degrees of prolongation. Mild prolongation (30–50 seconds) was observed in 45.0% of cases, moderate prolongation (51–100 seconds) in 29.0% and severe prolongation (>100 seconds) in 10.0% of patients. The mean APTT observed in the present study (52.69 ± 26.37 seconds) was substantially higher than that reported by Patil AY et al. (2020)[6] (35.56 ± 19.85 seconds). The proportion of patients with normal APTT values in the present study (16.0%) was lower than that reported by Srinivas GKP et al. (2025)[22] (30.0%). Mild APTT prolongation was observed in 45.0% of patients which was comparable to the 48.33% reported by Patil AY et al. (2020)[6]. Moderate prolongation was present in 29.0% of cases, compared with 5.0% reported by Patil AY et al. (2020)[6]. 74.0% of patients demonstrated moderate degrees of APTT prolongation, a proportion slightly higher than that reported by Srinivas GKP et al. (2025)[22]. Severe APTT prolongation (>100 seconds) was observed in 10.0% of patients in the present study, compared with only 2.5% reported by Patil AY et al. (2020)[6]. Overall, the significantly prolonged APTT values observed in the present study highlight the severity of coagulation abnormalities associated with advanced liver disease and cirrhosis. **(Table 5)**

The mean platelet count observed in the present study (174.21 ± 122.62 × 10^3/µL) was lower than that reported by Garg RP et al. (2020)[13] (215.70 ± 79.60 × 10^3/µL). The proportion of patients with normal platelet counts in the present study (30.0%) was comparable to the findings of Garg RP et al. (2020)[13] (29.0%). In the present study, the combined prevalence of mild and moderate thrombocytopenia was 58.0%, which was comparable to the rates reported by Mistry PA et al. (2024)[19] (51.0%). Severe thrombocytopenia (<50 × 10^3/µL) was observed in 12.0% of patients in the present study. **(Table 5)**

In the present study, a threshold of 0.5 µg/mL was used to categorize D-dimer levels. Normal D-dimer levels (≤0.5 µg/mL) were observed in 28% of patients while 72% showed elevated levels (>0.5 µg/mL). These findings are comparable to those reported by Rai V et al. (2017)[26], who observed normal D-dimer levels in 30% and elevated levels in 70% of patients. Bohania N et al. (2021)[27] reported a higher prevalence of elevated D-dimer levels (86.67%), with only 13.33% of patients having normal values. In contrast, Garg RP et al. (2020)[13] found elevated D-dimer levels in 58% of cases and normal levels in 42%, suggesting the inclusion of patients with less severe liver dysfunction. **(Table 5)**

CONCLUSION

Advanced liver disease, particularly cirrhosis and alcoholic liver disease was associated with significant coagulation abnormalities that were commonly observed in middle-aged and elderly male patients. These abnormalities included prolonged Prothrombin Time (PT), prolonged Activated Partial Thromboplastin Time (aPTT), thrombocytopenia and elevated D-dimer levels, reflecting impaired hepatic synthetic function and disease progression. Such derangements were important indicators of liver dysfunction and may contribute to complications associated with portal hypertension.

Therefore, regular assessment of coagulation parameters may be useful for evaluating disease severity and guiding patient management. In contrast, patients with chronic hepatitis or early-stage liver disease may initially demonstrate normal laboratory findings despite underlying hemostatic alterations. This phenomenon is explained by a rebalanced hemostatic state in which both bleeding and thrombotic risks coexist. In the present study, elevated D-dimer levels were observed in 72% of patients, suggesting that D-dimer may serve as a valuable marker of advanced liver disease and ongoing coagulation activation.

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