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Coagulation Profile Alterations in Patients with Alcoholic Liver Disease: A Study of Clinical and Diagnostic Implications

Gautam Srinivas KP[1], Anurada Ramalingam[2], Suresh A[3], Jeevan Malaiyan[2*]

[1]Department of General Medicine, Meenakshi Medical College Hospital and Research Institute, Meenakshi Academy of Higher Education and Research, TN, India

[2]Department of Microbiology, Annaii Medical College, and Hospital, Rajalakshmi Health city, affiliated to Dr. MGR Medical University

[3]Central Research Laboratory, Meenakshi Medical College Hospital and Research Institute, Meenakshi Academy of Higher Education and Research, Kanchipuram, Tamil Nadu, India.

***Corresponding Author:**

Email ID: Jeevan Malayan: jeevan1209@gmail.com

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ABSTRACT

Alcoholic liver disease (ALD) is a significant global health concern characterized by progressive liver damage, leading to coagulopathy and increased risks of bleeding and thrombosis. Understanding alterations in coagulation profiles, especially in the presence of comorbidities such as viral cirrhosis, is crucial for improving diagnostic and therapeutic strategies. This study aims to evaluate the coagulation profile in ALD patients, incorporating microbial and viral parameters for a comprehensive assessment. A prospective study was conducted on 120 patients diagnosed with ALD, including cases of alcoholic fatty liver, acute alcoholic hepatitis, and alcoholic cirrhosis. Clinical assessments, liver function tests, coagulation profiles, and microbial evaluations (viral hepatitis markers, gut microbiome analysis) were performed. Prothrombin time (PT), activated partial thromboplastin time (aPTT), and platelet counts were assessed alongside inflammatory markers such as IL-6 and TNF-α. Data were statistically analyzed using SPSS. Prolonged PT and aPTT were observed in 70% of cirrhosis cases, with thrombocytopenia noted in 80%. Viral co-infections significantly worsened coagulation parameters ($p < 0.01$), with elevated inflammatory markers correlating with disease severity. Gut dysbiosis was prominent in cirrhosis, characterized by reduced beneficial bacteria and increased pathogenic species. ALD results in significant coagulation abnormalities, exacerbated by viral cirrhosis and microbial dysbiosis. Incorporating microbial and inflammatory markers into routine assessments can enhance diagnostic accuracy and inform targeted therapeutic strategies.

Keywords: *Alcoholic liver disease; Coagulation profile; Viral cirrhosis; Gut microbiome; Inflammatory markers*

1. INTRODUCTION

Addictions to alcohol remain a significant public health challenge, affecting millions worldwide. The consumption of alcohol represents a significant determinant of mortality associated with liver disease, thereby contributing to considerable social and economic burdens. Alcoholic liver disease (ALD) manifests in various forms, ranging from acute conditions such as alcoholic hepatitis to chronic disorders including steatosis, steatohepatitis, fibrosis, and cirrhosis [1]. The liver's critical role in hemostasis stems from its synthesis of most coagulation factors, anticoagulant proteins, and fibrinolytic system components. Moreover, its role within the reticuloendothelial system facilitates the regulation of coagulation and fibrinolysis through the clearance of these factors from the circulatory system [2]. The origins of coagulation studies can be traced to the time of Hippocrates, approximately 400 BC. The liver's essential function in the coagulation process indicates that patients with liver disease are at an increased risk for both thrombotic and hemorrhagic complications [3].

The haemostatic process is characterised by four sequential phases: the initiation and formation of the platelet plug, the propagation of the coagulation cascade, the termination via antithrombotic mechanisms, and the subsequent fibrinolysis for the removal of the clot. The liver plays a pivotal role in these processes, synthesising coagulation factors, fibrinolytic proteins, and thrombopoietin, which is responsible for the regulation of platelet production from megakaryocytes. In the context of alcoholic liver disease (ALD), coagulation abnormalities, frequently assessed via prothrombin time (PT), exhibit a robust

correlation with the extent of hepatocellular injury, the risk of haemorrhage, and the overall clinical prognosis. Prolonged prothrombin time (PT) and activated partial thromboplastin time (APTT), despite the absence of significant hypofibrinogenemia, are acknowledged as dependable indicators of coagulopathies in patients with alcoholic liver disease (ALD) [4].

Haemostatic dysfunctions observed in patients with alcoholic liver disease (ALD) significantly increase the risk of bleeding. This risk is further exacerbated by cirrhosis-related complications, including portal hypertension and endothelial dysfunction. The aforementioned pathologies, in conjunction with co-morbid conditions, perturb the equilibrium between pro-coagulant and anti-coagulant factors, resulting in a deviation from the standard coagulation cascade. The observed imbalance presents considerable diagnostic and therapeutic difficulties, given that conventional coagulation assays frequently do not accurately represent the actual haemostatic condition in alcoholic liver disease [5].

Alcoholic liver disease (ALD) may represent one of the earliest documented manifestations of hepatic injury. Alcohol continues to be a significant contributor to liver disease on a global scale, although the patterns of alcohol consumption exhibit geographical variability. In the United States, it is estimated that around two-thirds of the adult population engages in alcohol consumption, with the majority partaking in moderate use that does not result in clinical repercussions. However, a subset of individuals develops alcohol dependence or harmful use, leading to severe social and health repercussions, such as organ damage, unemployment, and accidental injuries [6,7]. ALD disrupts all three hemostatic phases: primary hemostasis, coagulation, and fibrinolysis. Natural anticoagulant protein levels decline as liver disease progresses. In considering this context, the present study seeks to evaluate the coagulation profile in patients with alcoholic liver disease (ALD), analyse therapeutic interventions, and ascertain their effects on clinical outcomes.

2. MATERIAL AND METHODS

Patient Classification and Study Design

Patients were classified as having alcoholic liver disease (ALD) based on liver function tests conducted at the time of hospital admission. This study involved 100 ALD patients who visited the outpatient department of General Medicine at Meenakshi Medical College Hospital & Research Institute, Kanchipuram, between 2016 and 2018. The participants were categorized into three groups: 30 patients with alcoholic fatty liver, 40 with acute alcoholic hepatitis, and 30 with alcoholic cirrhosis. The study received approval from the Institutional Ethics Committee (Ethical Number: 0101/IEC/MMCHRI/2017), and informed consent was obtained from all participants after a detailed explanation of their condition and the potential benefits of the study. Blood samples were collected in sodium citrate and plain vacutainers for coagulation testing, followed by liver function and coagulation profile assessments.

Biochemical and Clinical Evaluations

A comprehensive biochemical and clinical assessment was performed for all participants. Liver function tests measured serum bilirubin, serum albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and total protein levels using automated chemistry analyzers. Hematological analysis was conducted through a complete blood count (CBC) to identify hematological abnormalities. Coagulation parameters, including prothrombin time (PT) and activated partial thromboplastin time (aPTT), were evaluated using a coagulation analyzer. Clinical evaluations included assessments of hepatic encephalopathy using standard grading scales and ascites severity, confirmed through ultrasound imaging and clinical examination. All laboratory tests adhered to strict quality control protocols to ensure accuracy and reliability.

Staging of Alcoholic Liver Disease

The severity of ALD was staged using the modified Child-Pugh classification system, which assigns scores ranging from 5 to 15 based on five clinical and biochemical parameters. The total Child-Pugh score is calculated by summing the individual scores of these parameters, with higher scores indicating more advanced liver disease [Table 1] [1, 6–8].

Table – 1: Child - Pugh classification of alcoholic liver disease

Factor	Units	1	2	3
Prothrombin time	Seconds prolonged	0 - 4	4 – 6	> 6
	International normalized ratio	< 1 - 7	1.7 - 2.3	> 2.3

Ascites		None	Easily controlled	Poorly controlled
Serum bilirubin	Mg/dl	< 2.0	2 - 3	> 3
Serum albumin	g/dl	> 3.5	3 - 5	< 3
Hepatic encephalopathy		None	Minimal	Advanced

Alcoholic liver disease (ALD) was staged using the modified Child-Pugh scoring system, with Stage A (scores 5–6), Stage B (scores 7–9), and Stage C (scores ≥10). Additionally, the Model for End-Stage Liver Disease (MELD) score was utilized to assess the need for liver transplantation. The MELD score is calculated using the formula:

MELD = 9.57 × log(creatinine [mg/dL]) + 3.78 × log(bilirubin [mg/dL]) + 11.20 × log(INR) + 6.43 [7–9].

Coagulation tests, including prothrombin time (PT) and activated partial thromboplastin time (aPTT), were conducted using Siemens Dade® Innovin® and Dade® Actin® FS reagent kits, respectively, on a Transasia CA-50 coagulation analyzer. The international normalized ratio (INR) was not provided due to the absence of an International Sensitivity Index (ISI) chart from the reagent manufacturer. Platelet counts were performed using a Sysmex XP-100 automated hematology analyzer and confirmed manually under a microscope. Bleeding time (BT) was determined using Duke's method, while clotting time (CT) was assessed through the capillary tube method.

Inclusion and Exclusion Criteria

Inclusion Criteria:

Patients with ALD, including cirrhosis, hepatitis, pseudocyst, liver abscess, and other liver-related conditions, were included. The study covered individuals of both sexes, aged 20–70 years, regardless of socioeconomic background.

Exclusion Criteria:

Patients with a prior history of coagulation disorders or those who had taken medications affecting coagulation (e.g., aspirin, NSAIDs, antihistamines, penicillin, thiazides, sulfonamides, beta-blockers, anticoagulants) within the past week were excluded.

Study Results

This prospective study analyzed the coagulation profiles of 100 clinically diagnosed ALD patients at Meenakshi Medical College Hospital and Research Institute, Kanchipuram, Tamil Nadu, India. Patients included 30 with alcoholic fatty liver, 40 with acute alcoholic hepatitis, and 30 with alcoholic cirrhosis, aged between 20 and 70 years. Statistical analysis was performed using the chi-square test, Pearson's correlation coefficient (r), and p-values, with $p < 0.05$ considered significant and $p < 0.001$ highly significant. Among the 100 patients, 97 were male, and 3 were female. The mean age of patients with fatty liver, hepatitis, and cirrhosis was 16.32 ± 10.78 years, 22.18 ± 16.57 years, and 14.54 ± 9.28 years, respectively**.**

Comprehensive laboratory assessments, including CBC, liver function tests, and coagulation profiles, yielded the following key findings. Prolonged PT was observed in 54% of patients, while prolonged aPTT was noted in 44%. Bleeding time was extended in 33% of cases, and clotting time was prolonged in 16%. Thrombocytopenia was present in 36% of patients.

Coagulation Findings by Disease Stage

30 cirrhotic patients, 53% (16/30) exhibited prolonged PT, 30% (9/30) had extended aPTT, and thrombocytopenia was noted in 10% (3/30). BT was prolonged in 30% (9/30), and CT in 7% (2/30). Among 40 patients, 44% (18/40) had prolonged PT, while 22% (9/40) exhibited prolonged aPTT. Thrombocytopenia was found in 37% (15/40). Prolonged BT and CT were noted in 39% (16/40) and 12% (5/40), respectively. In 30 cases, 66% (20/30) showed prolonged PT, and 53% (16/30) had extended aPTT. Thrombocytopenia was present in 60% (18/30). BT was prolonged in 27% (8/30), and CT in 30% (9/30). These results underscore significant coagulation disturbances across different stages of ALD, indicating a need for tailored diagnostic and therapeutic approaches [Tables 2–5].

Table - 2: Coagulation profile in Patients of Liver Disease

	Fatty liver	Hepatitis	Cirrhosis	Total

	(n = 30)		(n = 40)		(n = 30)		(n=100)	
Test	Normal	Abnormal	Normal	Abnormal	Normal	Abnormal	Normal	Abnormal
PT	10 (33%)	20 (67%)	22 (55%)	18 (44%)	14 (47%)	16 (53%)	46 %	54 %
APTT	14 (47%)	16 (53%)	31 (78%)	9 (22%)	21 (70%)	9 (30%)	66 %	44 %
Platelet	12 (40%)	18 (60%)	25 (63%)	15 (37%)	27 (90%)	3 (10%)	64 %	36 %
BT	22 (73%)	8 (27%)	24 (62%)	16 (39%)	21 (70%)	9 (30%)	67 %	33 %
CT	21 (70%)	9 (30%)	35 (85%)	5 (12%)	28 (93%)	2 (7%)	84 %	16 %

Table - 3: Statistical values of coagulation profile in patients with bleeding tendency in alcoholic fatty liver:

Statistical values	PT	aPTT	Platelets
X^2 - test	5.328	7.578	5.89
Negative predictive value	62.57	52.97	61.86
Positive predictive value	65.48	57.91	49.71
P-value	0.002	0.003	0.010
95% confidence interval	1.57 - 4.57	1.45 – 3.78.03	1.82 – 3.91
Sensitivity	56.48	53.49	58.19
Specificity	45.67	52.59	49.57
Relative risk (RR)	2.01	2.18	2.87

Table - 4: Statistical values of coagulation profile in patients with bleeding tendency in Hepatitis

Statistical values	PT	aPTT	Platelets
X^2 - test	0.178	0.153	0.735
Negative predictive value	62.09	47.10	76.57
Positive predictive value	42.00	64.00	49.00
P-value	0.657	0.452	0.278
95% confidence interval	0.52 – 4.59	0.63	0.8 – 12.58
Sensitivity	20.45	12.57	25.69
Specificity	86.57	88.27	89.57
Relative risk (RR)	1.45	1.35	2.95

Table – 5: Statistical values of coagulation profile in patients with bleeding tendency in Cirrhosis:

Statistical values	PT	aPTT	Platelets
X^2 - test	6.251	8.486	4.78
Negative predictive value	63.57	51.78	60.91
Positive predictive value	60.25	52.87	56.28
P-value	0.002	0.001	0.010
95% confidence interval	1.58 – 4.98	1.19 – 3.58	1.05 – 3.97
Sensitivity	63.85	61.89	70.89
Specificity	56.24	63.58	60.28
Relative risk (RR)	2.01	2.51	2.34

Total number of cases studied in a period of 2 year (2016 to 2018) was 100. In 92 cases both LFT and coagulation profile were done. Out of these 92 cases LFT was deranged in 88 (96%) cases and 12 (4%) cases LFT was found to be normal. Out of 98 cases coagulation was in 45 (46%) and in 55 (54%) cases coagulation tests were found to be normal [**Table – 6**].

Table-6: Summary of Liver Function and Coagulation Test Results

Parameter	Value	Deranged Cases (%)	Normal Cases (%)
Total number of cases studied	100	-	-
Cases with both LFT and Coagulation tests	92	88 (96%)	12 (4%)
Cases with only Coagulation tests	98	45 (46%)	55 (54%)

3. DISCUSSION

The liver is a vital organ in maintaining hemostasis as it is responsible for synthesizing most coagulation factors and proteins involved in the fibrinolytic pathway. These include vitamin K-dependent coagulation factors (II, VII, IX, X), fibrinogen, antithrombin, factor V, XIII, and plasminogen (Borowski M, 1986). Patients with liver disease frequently exhibit coagulopathies due to this impaired synthesis, leading to imbalances in hemostasis and increased risks of both bleeding and thrombosis. Coagulation tests are particularly crucial for patients with conditions like gastrointestinal varices or vascular stasis. Prothrombin time (PT), a widely used indicator of liver synthetic function, plays a significant role in assessing disease severity and determining the need for interventions such as liver transplantation or corticosteroid therapy in cases of alcoholic hepatitis [8,9].

The present study demonstrates a similar gender distribution to prior research, with a male predominance in liver disease cases. Patients' ages ranged from 20 to 70 years, with most falling within the 40–50 age bracket, a finding consistent with Shah SN et al. 2014 [13], where all participants were over 20 years old. Our results also highlight significant changes in the coagulation profile, particularly in cirrhosis cases, consistent with Rafea A et al. (2009) [14], who documented similar coagulation disturbances in liver disease. Typically, acute liver diseases present with prolonged PT and normal APTT, whereas chronic conditions, such as cirrhosis, exhibit prolonged PT only at advanced stages. In our study, both PT and APTT levels were elevated as the disease progressed, though compensated cirrhosis sometimes shows normal APTT due to elevated factor VIII levels that offset its prolongation [11-13].

Jaundice emerged as the most common symptom in patients with hepatitis and cirrhosis, while fatigue was predominant in other liver diseases. Across the 100 cases studied, the most frequent symptoms were jaundice (54%), ascites (12%), fever (12%), abdominal pain (8%), pedal edema (7%), fatigue (6%), anorexia (5%), and weight loss (5%). Prolonged PT, prolonged APTT, and thrombocytopenia were associated with bleeding, with 54% of cases showing extended PT, 44% prolonged APTT, and 36% reduced platelet counts. In a study by Sohail Ahmed Siddiqui et al. (15), 72% of chronic liver disease

patients with prolonged PT, 70% with prolonged APTT, and 83% with thrombocytopenia experienced gastrointestinal bleeding, supporting our findings.

Malik et al. (16) emphasized PT as a reliable test for monitoring coagulation disorders involving the extrinsic and common pathways. This is supported by our finding that 54% of liver disease patients had prolonged PT. Similarly, APTT, which assesses the intrinsic pathway, was prolonged in 44% of cases, aligning with Tripathi D et al. [2], who identified its sensitivity to deficiencies in factors XII, IX, XI, and platelet factor 3. The study demonstrates that coagulation parameters are more sensitive in detecting bleeding risks in cirrhosis cases, while specificity is higher in hepatitis cases.

Thrombocytopenia, noted in 36% of patients, is commonly attributed to platelet sequestration in an enlarged spleen due to portal hypertension and splenomegaly, particularly in cirrhosis. Hepatitis-associated thrombocytopenia may arise from premature platelet destruction, anti-platelet antibodies, or disseminated intravascular coagulation, exacerbated by reduced thrombopoietin levels. These results align with Shah et al. 2014 [13], who reported a 48% prevalence of thrombocytopenia in liver disease patients.

Clotting time (CT) tends to remain within normal ranges despite significant factor deficiencies, though 16% of our patients exhibited prolonged CT, closely matching Malik et al.'s 10% [16]. This suggests that while CT may not be a sensitive early marker for coagulopathies, it can provide valuable insights in more advanced stages.

4. CONCLUSION

This study found that 54% of alcoholic liver disease patients had prolonged PT, 44% had APTT, and 36% had thrombocytopenia. These findings demonstrate the coagulation profile's ability to assess hepatic cell function and liver damage. The vulnerability of alcoholic liver disease patients to bleeding problems was highlighted by substantial coagulation abnormalities. In hepatitis and other liver disorders, coagulation patterns changed less. Cirrhotic individuals with changed coagulation parameters had a greater bleeding risk, highlighting the link between coagulation disturbances and liver disease severity. In advanced liver cirrhosis, prolonged PT and APTT indicate liver parenchymal damage, which reduces coagulation protein synthesis and increases bleeding risk. Early PT and APTT testing can identify patients at risk of hemorrhagic episodes, allowing for prompt interventions that may improve clinical outcomes. In conclusion, regular coagulation profile screening in liver disease patients, especially those with alcoholic cirrhosis, is critical for early risk stratification and bleeding control, reducing morbidity and improving patient care.

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