

Research Article

Relationship of Thrombocytopenia, Serum Albumin, and Coagulation Abnormalities with the Severity of Portal Hypertension in Patients with Chronic Liver Disease

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ABSTRACT

Background: Chronic liver disease can lead to portal hypertension which is a significant complication and is responsible for high morbidity and mortality. Worsening hepatic dysfunction and rising portal pressure can be suggested by thrombolytic, hypoglycaemia and coagulation abnormalities.

Objective: To assess the association of thrombolytic, serum albumin and coagulation defects to the severity of the portal hypertension in the patients of chronic liver disease.

Place and Duration of Study

This study was carried out in the Department of Medicine of Punjab Rangers Teaching Hospital, Lahore from January 2026 to June 2026.

Methodology: This Cross sectional study included 160 patients diagnosed with chronic liver disease (CLD). According to the WHO sample size formula, the sample size was determined. The platelet count, serum albumin, thrombotic time and international normalized ratio were evaluated and correlated with the degree of the portal hypertension.

Results: Of the patients, 38.1% had severe portal hypertension. The prevalence of thrombolytic, hypoglycaemia and prolonged INR were 61.3%, 58.8% and 46.9% respectively.

Conclusion: Increased severity of portal hypertension was significantly correlated with thrombolytic, low serum albumin and coagulation abnormalities.

Keywords: Chronic Liver Disease, Portal Hypertension, Thrombolytic, Serum Albumin, Coagulation Abnormalities.

INTRODUCTION

Chronic liver disease is an important health issue in the world and is linked to significant morbidity, mortality, and cost of health-care. It is caused by chronic liver damage due to chronic viral hepatitis, metabolic-associated fatty liver disease, alcohol abuse, autoimmune diseases and other chronic liver diseases. Gradually, liver damage causes liver fibrosis, disruption of the normal liver structure (architecture), and cirrhosis (Fernández-Garibay et al., 2025). Patients with cirrhosis can develop several serious complications, one of which is portal hypertension, which is one of the most important complications (Abid et al., 2026).

Portal hypertension is when the portal venous system has a higher than normal blood pressure. The elevation is primarily due to the resistance to blood flow in the cirrhotic and structurally altered liver in patients with cirrhosis. The portal pressure can increase and produce the following clinical problems: Splenomegaly, ascites, gastroesophageal varices, variceal bleeding, hepatic encephalopathy and other decompensated liver disease features (Fierro-Angulo et al., 2024). Hence, the severity of portal hypertension is closely related to the severity of chronic liver disease and its prognosis. This is because the severity can be accurately determined and this will help identify higher risk patients for complications and plan

patients' monitoring and treatment (Al-Dholae et al., 2023).

The following hematological and biochemical abnormalities are commonly found in the patients with chronic liver disease. One of the most common haematological abnormalities is thrombocytopenia (Patil & Devareddy, 2025). May be caused by splenic sequestration caused by portal hypertension, decreased production of thrombopoietin by the diseased liver, bone marrow suppression and increased platelet destruction. As portal hypertension worsens, platelet sequestration can increase due to splenic enlargement, which leads to a further decrease in platelets. Therefore, platelet count can be useful to give some clues about the severity of portal hypertension (Sohail et al., 2023).

The other hepatic synthetic function marker is serum albumin. The liver makes albumin, and its level is usually reduced when the liver disease is chronic. Hypoalbuminaemia can be a marker of liver dysfunction, decreased nutritional status, systemic inflammation and end-stage liver failure (Zhang et al., 2024). Patients with more severe portal hypertension may have a lower level of albumin as portal hypertension is typically associated with more advanced liver structural and functional damage (Awal et al., 2025).

Another common association of chronic liver disease is coagulation abnormalities. The liver is key to producing several clotting factors. As the hepatic dysfunction increases, the prothrombin time and International normalized ratio (INR) increase (Yang et al., 2025). Haemostatic abnormalities in chronic liver disease, while complex and involving both procoagulant and anticoagulant pathways, are reflected in the use of conventional coagulation parameters, which have been widely accepted markers of compromised hepatic synthetic function, in clinical practice (Bukhari et al., 2025).

A simple and convenient method of evaluating the patient with chronic liver disease may be the combination of the platelet count, serum albumin and coagulation parameters. These are routinely available, relatively low cost laboratory tests and can be performed in most health care settings (Mohamed et al., 2025). An understanding of their relationship with the severity of portal hypertension could help better assess the risk of their disease in the early stages, and identify patients who need more intense clinical management, or further investigation and monitoring. Hence the

present study was planned to compare the thrombocytopenia and the serum albumin level, coagulation abnormalities with the severity of portal hypertension in chronic liver disease (Chen et al., 2023).

Objective

To evaluate the relationship between thrombocytopenia, serum albumin and coagulation abnormalities with the severity of portal hypertension in CLD patients.

METHODOLOGY

This study was done as a cross sectional study in the Department of Medicine A total of 160 patients with chronic liver disease were included in the study Punjab Rangers Teaching Hospital, Lahore from January 2026 to June 2026. The calculations for the sample size were based upon the WHO sample size formula. Predefined inclusion and exclusion criteria were used to select patients. Information was recorded relating to the demographic and clinical aspects (age, sex, duration and cause of liver disease). Laboratory tests performed were complete blood count (CBC), platelet count, prothrombin time (PT) and international normalized ratio (INR). Clinical, laboratory, radiological and endoscopic parameters were used to assess severity of the portal hypertension. Patients were divided into the following groups based on the severity of portal hypertension: group A (portal hypertension was absent), group B (portal hypertension was mild), and group C (portal hypertension was severe). The thrombocytopenia, serum albumin level, coagulation abnormalities and portal hypertension severity were analyzed statistically. A p value of <0.05 was deemed to be statistically significant.

Inclusion Criteria

Inclusion criteria for the study were patients aged 18 years or older with a known diagnosis of chronic liver disease. Patients who were clinically, laboratory, radiological or endoscopic evidence of portal hypertension were eligible. All male and female patients who gave informed consent and fulfilled the necessary clinical and laboratory evaluation were included.

Exclusion Criteria

Patients with acute liver failure, primary hematological disorders associated with thrombocytopenia, active malignancy, chronic kidney disease, sepsis, pregnancy and patients

on coagulation therapy were excluded. Other patients with conditions or medications which may have independently affected platelet count, serum albumin, prothrombin time or international normalized ratio were also excluded.

Data Collection

A structured proforma was used to gather data following informed consent of each participant. Demographic data - such as age, sex, duration of the chronic liver disease and underlying etiology - were recorded. Clinical findings associated with portal hypertension (ascites, splenomegaly and varices) were recorded. Samples of blood were drawn for complete blood count, platelet count, serum albumin, prothrombin time and INR. Relevant ultrasound and endoscopy was also documented (if present). Patients were categorized based on the severity of portal hypertension by the following clinical, laboratory, radiological and endoscopic criteria: Colour Doppler ultrasound appearance of portal venous flow, presence of splenoportal hypertension on portal angiography and/or endoscopic appearance of varices. All the data

collected was processed using a statistical software. Subsequent assessment of the relationship between the laboratory abnormalities and severity of portal hypertension was made.

RESULTS

An overall of 160 patients having chronic liver disease were taken in the study. Sixty-one patients (38.1%) had severe portal hypertension, 57 patients (35.6%) had moderate and 42 patients (26.3%) had mild portal hypertension. The prevalence of thrombocytopenia, hypoalbuminemia and prolonged INR were 61.3%, 58.8% and 46.9 % respectively in 98 patients. Such abnormalities were more prevalent in those patients with more severe portal hypertension. MPC and serum albumin were found to be decreased progressively with increasing severity while the prothrombin time and INR were found to be increased. The statistical analysis revealed significant correlation between thrombocytopenia, low level of serum albumin, coagulation abnormalities and severity of portal hypertension with p values < 0.05.

Table 1. Distribution of Patients According To Severity of Portal Hypertension

Severity of Portal Hypertension	Frequency n	Percentage
Mild	42	26.3%
Moderate	57	35.6%
Severe	61	38.1%
Total	160	100%

Severe portal hypertension was the most common category (61 patients 38.1%) among 160 patients. In 57 patients (35.6%), moderate portal hypertension was present and

in 42 patients (26.3%) there was only a mild disease. From these data, it was determined that a significant number of patients had moderate to severe portal hypertension.

Table 2. Frequency of Thrombocytopenia among Study Patients

Platelet Status	Frequency n	Percentage
Thrombocytopenia	98	61.3%
Normal platelet count	62	38.7%
Total	160	100%

98 patients (61.3%) had thrombocytopenia while 62 patients (38.7%) had normal platelet counts. Thrombocytopenia was a frequent

hematological abnormality since it was seen in a large number of patients with chronic liver disease and portal hypertension.

Table 3. Frequency of Hypoalbuminemia among Study Patients

Serum Albumin Status	Frequency n	Percentage
Hypoalbuminemia	94	58.8%
Normal serum albumin	66	41.2%
Total	160	100%

94 patients (58.8%) had hypoalbuminemia and 66 patients (41.2%) had normal serum albumin level. Patients with higher severity of portal hypertension had decreased levels of

albumin, reflecting progressive decreased hepatic synthetic function with increased chronic liver disease.

Table 4. Frequency of Coagulation Abnormalities

INR Status	Frequency n	Percentage
Prolonged INR	75	46.9%
Normal INR	85	53.1%
Total	160	100%

Longer INR was seen in 75 patients (46.9%) and INR was within normal range in 85 patients (53.1%). Patients with severe portal hypertension also had more coagulation

abnormalities, implying that the more severe the chronic liver disease the greater the hepatic synthetic dysfunction.

Table 5. Laboratory Abnormalities According To Portal Hypertension Severity

Laboratory Abnormality	Mild n=42	Moderate n=57	Severe n=61	p-value
Thrombocytopenia	14 33.3%	35 61.4%	49 80.3%	<0.001
Hypoalbuminemia	13 31.0%	33 57.9%	48 78.7%	<0.001
Prolonged INR	8 19.0%	25 43.9%	42 68.9%	<0.001

Thrombocytopenia, hypoalbuminemia and length of the INR were all progressive with the increasing severity of portal hypertension. All the abnormalities were most common in those with severe portal hypertension. All the associations had a significance level of less than 0.001, and were related to greater severity of portal hypertension.

DISCUSSION

This study examined the association of thrombocytopenia, serum albumin and coagulation abnormalities to the extent of portal hypertension in chronic liver disease (CLD) patients. The results demonstrated an increase in these laboratory changes with increasing severity of portal hypertension (Islam et al., 2022). This implies that the generally available haematological and biochemical values could offer valuable data on the course of chronic liver disease and the extent of portal hypertension (Kumar et al., 2026).

61.3% of the study population had thrombocytopenia, which was most prevalent in those with severe portal hypertension. In the advanced chronic liver disease, platelet counts usually fall due to several mechanisms. Splenomegaly due to portal hypertension may result in more platelet sequestration in the spleen. Also, less thrombopoietin is produced by the liver, which leads to less production of platelets (Wang et al., 2025). There may also be a bone marrow suppression and increased break down of platelets. The progressive

nature of the thrombocytopenia from mild, through moderate to severe portal hypertension in the current study makes this an indirect surrogate marker of increasing severity of portal hypertension.

Abnormal levels of hypoalbuminemia were present in 58.8% of patients and were significantly higher in patients with severe portal hypertension. The synthesis rate of liver albumin is a measure of the liver's synthetic function and is usually reduced in the later stages of chronic liver disease (Zhao et al., 2024). Low levels of albumin may also be caused by an inadequate nutritional status, systemic inflammation, increased vascular permeability, and fluid retention. With progressive liver disease, the decreased hepatocellular function leads to decreased production of albumin and to clinical problems like ascites and peripheral edema. Thus, hypoalbuminemia in patients with severe PH could be due to both liver dysfunction and disease severity (Rathi et al., 2026).

Coagulation abnormalities were also frequently found in the present study. The prevalence of prolonged INR was 46.9% and they were more common in patients who experienced severe portal hypertension. Several coagulation factors are produced in the liver and as liver function deteriorates progressive production can be affected (Jadaun & Saigal, 2022). This can result in an extended prothrombin time and/or elevated INR. The PT and INR are clinically useful markers of decreased hepatic synthetic function, despite

the complexity of coagulation in chronic liver disease, which includes alterations in procoagulant and anticoagulant factors (Nouh et al., 2025).

Of note, in this study, thrombocytopenia, hypoalbuminemia and prolonged INR were found to be statistically significant parameters associated with severity of portal hypertension. The frequency of all 3 abnormalities was significantly higher in those with severe than those with mild or moderate portal hypertension (Cao et al., 2024). Taken together, these data suggest that assessment of several laboratory parameters may prove useful in obtaining more clinical information than would single parameter alone.

Laboratory tests, especially routine tests, have many advantages: They are widely available, inexpensive, and can be repeated during follow-up (Li et al., 2026). Their use may be helpful for the clinician to identify patients who may need further evaluation, endoscopic screening, imaging or increased monitoring for the development of complications. These parameters should not be used as a direct substitutes to the established assessment parameters of portal hypertension, however.

Finally, the results of the present study indicate that thrombocytopenia, hypoglycaemia and coagulation abnormalities are closely related to the progression of the portal hypertension in chronic liver disease (Guo et al., 2025). Therefore, their combined evaluation may be used as an aid to clinical risk assessment and used to better identify the patients with more advanced disease.

CONCLUSION

The present study showed that the number of platelets, serum albumin and coagulation abnormalities were significantly correlated with the severity of portal hypertension of chronic liver disease. Patients with severe portal hypertension had higher rates of low platelet counts and hypoalbuminemia and longer INR than patients with mild or moderate portal hypertension. These routinely obtained laboratory parameters can then help the clinician to determine the risk of advanced PH. They should be used along with clinical, radiological and endoscopic findings for early risk stratification, more intensive monitoring and clinical management but they are of complementary use.

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