International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-1-12*

A Study of Hematological Abnormalities in Chronic Liver Disease Patients in a tertiary care hospital of central India

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Received: **November 18, 2025**
Revised: **December 15, 2025**
Accepted: **January 03, 2026**
Published: **January 22, 2026**

Abstract
Background: Chronic liver disease (CLD) is associated with multiple systemic manifestations, including a wide spectrum of hematological abnormalities, which significantly contribute to morbidity and mortality. Despite their clinical importance, the hematologic alterations in CLD patients remain under-characterized in certain regional populations.***Aim and objective:*** To determine the types and prevalence of anemia and other hematological abnormalities in CLD patients admitted to a tertiary care hospital in Central India.***Methods:*** A cross-sectional hospital-based observational study was conducted over two years in 60 CLD patients aged >18 years. Data collection included clinical evaluation, laboratory investigations (CBC, LFT, coagulation profile, peripheral smear), and imaging. Inclusion and exclusion criteria were applied as per protocol. Statistical analysis was performed using SPSS.***Results:*** The mean age was 49.7 ± 8.2 years, with a male predominance (58.33%). Alcohol was the most common etiology (56.66%), followed by hepatitis B (18.33%), hepatitis C (10%), and others (15%). Anemia was present in 56.7% of patients, with microcytic hypochromic type being the most common (52.9%). Moderate thrombocytopenia was observed in 65% of patients, and leukopenia in 26.7%. Prolonged prothrombin time was noted in 88.3% and hypoalbuminemia in 38.33%.***Conclusion:*** Hematological abnormalities, particularly anemia, thrombocytopenia, and coagulation derangements, are common in CLD patients and correlate with disease severity. Early detection and management can improve clinical outcomes.

Keywords: Anemia, Chronic liver disease, Coagulation abnormalities, Hematological abnormalities, Thrombocytopenia

INTRODUCTION
Chronic liver diseases (CLDs) are a set of diseases characterized by decreased hepatic function as a result of chronic inflammation or insult to the liver for more than 6 months. At their most advanced stage, CLDs often lead to the development of cirrhosis, defined as the irreversible distortion of the liver architecture by fibrosis, scar, and abnormal nodules. With cirrhosis comes the risk for progressive liver dysfunction and complications of portal hypertension.1 Chronic liver disease with or without liver failure is associated with considerable morbidity and mortality and affects the quality of life as usually they are progressive and often non-reversible.2

Liver performs numerous and vital roles in maintaining homeostasis and health. It plays a major role in carbohydrate, lipid and protein metabolism. Liver is the major storage site for iron, vitamin B12 and folic acid. The liver is involved in or is responsible for various haematological abnormalities due to its portal circulation and its synthetic (clotting factors, thrombopoietin) and immune functions. Primary liver problems like cirrhosis can lead to haematological abnormalities and primary haematological diseases can in turn affect the liver and its functioning.3- 5

Chronic liver disease (CLD) is one of the major causes of death that has a year-on-year rising incidence and in the western countries is now the fifth leading cause of early loss of life. Major causes of CLD are alcohol abuse, obesity/metabolic disease, autoimmune hepatitis, and viral hepatitis (HBV and HCV); furthermore, the majority of people dying from CLD are under the age of 70.6, 7

Abnormalities in haematological parameters are common in patients with cirrhosis. The pathogenesis of abnormal haematological indices (HIs) in cirrhosis is multifactorial and includes portal hypertension-induced sequestration, alterations in bone marrow stimulating factors, viral- and toxin-induced bone marrow suppression. Abnormalities in haematological indices

are associated with an increased risk of complications including bleeding and infection.Splenic sequestration and destruction of platelets, white blood cells (WBCs) and red blood cells (RBCs) in the portal hypertension-induced enlarged spleen is defined as hypersplenism.In patients with cirrhosis, there is a redistribution of platelets, with up to 90% of the circulating platelet mass located in the enlarged spleen.3

Since the liver plays a major role in iron balance, it is obvious that liver diseases of different aetiology, and especially advanced CLD with portal hypertension, are directly related to abnormalities in iron homeostasis.Iron is a micronutrient of vital importance for human oxygenation, given its critical role in oxygen transport via Hemoglobin (Hb) and myoglobin. It also plays an important role in multiple metabolic pathways, such as DNA synthesis and mitochondrial function. The concentration of iron in the body is 2-4 g, and over 80% is contained in the Hb of red blood cells.8- 10

The liver performs a major role in iron homeostasis. It is the main organ for the production of the iron regulatory hormone hepcidin, expressed in iron excess conditions as well as in cases of inflammation, blocking the absorption of iron from the enterocytes. The role of hepcidin in liver diseases, with or without cirrhosis, is still under investigation, but is probably one of the contributing factors to the anaemia of chronic disease present in a variety of liver conditions. Iron deficiency (ID), with or without anaemia, is associated with many symptoms and complications that have a significant and negative impact on patients. It can increase cardiovascular morbidity and mortality, impair cognition, and decrease quality of life11. The impact on quality of life in particular is substantial and can affect patients physically and emotionally, impairing their cognition and their ability to work. ID can also induce a variety of metabolic changes in the liver.11- 14

Haemolysis also represents a common cause of anaemia in patients with liver disease, especially of alcoholic cause, and is usually attributed to spur-cell anaemia. It is related to abnormal cholesterol loading of the red blood cell membrane, which results in spiculated erythrocytes with a short lifespan, called acanthocytes.15- 17

Aim and Objectives

To study the different types and hematological abnormalities of anemia in patients with chronic liver disease.

MATERIALS AND METHODS

This cross-sectional hospital-based observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital in Central India over a period of two years using systematic random sampling, involving 60 chronic liver disease (CLD) patients aged above 18 years who were admitted to the department. Patients included had symptoms persisting for more than six months and CLD due to alcoholic, post-infective, or metabolic causes, while those with gastrointestinal malignancy, acute liver failure, primary coagulation disorders, drug-induced hepatitis, or age below 18 years were excluded. After obtaining written informed consent, detailed history taking, clinical examination, and relevant laboratory investigations including complete blood count, liver function tests, and coagulation profile, along with abdominal ultrasonography, were performed. Data were analyzed using SPSS software.

RESULTS

Table 1:Distribution of patients according to age (n=60)

Age group (years)	Number	Percentage (%)
18-30	1	1.66
31-40	6	10
41-50	23	38.33
51-60	19	31.66
>60	11	18.33
Gender		
Males	35	58.33
Females	25	41.66

Out of 60 patients 38.33 % (n = 23) were belonging to 41 to 50 years while 31.6 % (n = 19) were belonging to 51 to 60 years, 18.33 % (n= 11) were belonging to more than 60 years while 10% (n = 6) were belonging to 31 to 40 years. Only 1 was (1.66%) belonging to 18 to 30 years. Out of 60 patients 58.33 % (n = 35) were males while 41.66% (n = 25) were females.

Table 2: Distribution of patients according to aetiology (n=60)

Aetiology	Number	Percentage (%)
Alcohol	34	56.66
Hepatitis B	11	18.33
Hepatitis C	6	10
Others	9	15
Total	**60**	**100%**

Out of 60 patients Alcohol was the etiologic agent in 56.66% (n = 34) while Hepatitis B for 18.33% (n = 11) cases. Hepatitis C was the etiological agent in 10% (n = 6) while 15% (n = 9) were other aetiologies for CLD.

Graph 1: Distribution of patients according to haemoglobin levels (gm/dl) and severity of anemia (n=34)

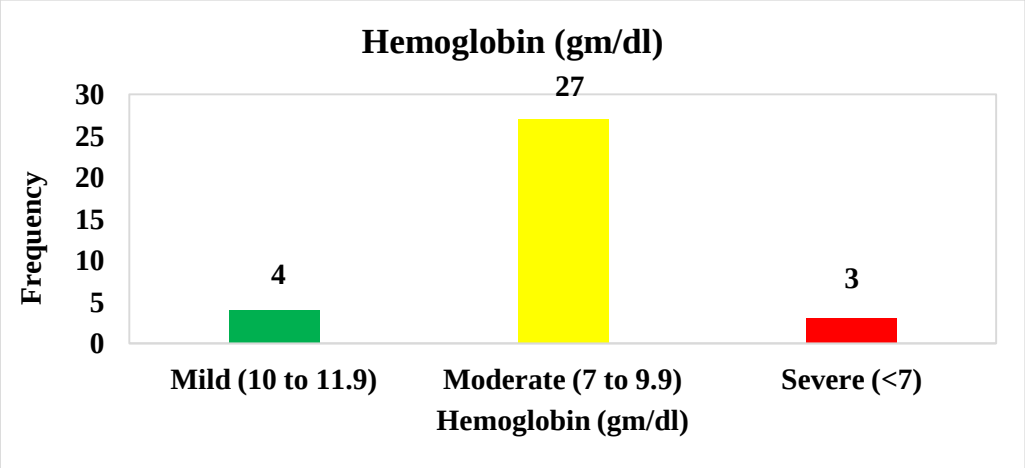

Table 3. Distribution of patients according to type of anemia (n=34)

Type of anaemia	Number (n)	Percentage (%)
Microcytic hypochromic anaemia	18	52.9
Normocytic normochromic anaemia	14	41.2
Macrocytic anaemia	2	5.9
Total	**34**	**100**

Out of 34 patients with anaemia, microcytic anaemia was found to be present in 52.9% (n = 18) of the cases. In contrast, macrocytic anaemia and normocytic anaemia was present in 5.9 % (n = 2) and 41.2% (n = 14) of the patients respectively.

Table 4: Distribution of Total Leucocyte Count and Platelet Count in Chronic Liver Disease Patients (n=60)

Total leucocyte count (per cumm)	Severity	Frequency (n=60)	Percentage (%)
<4000	Leukopenia	16	26.7
4000 to 11,000	Normal	44	73.3
> 11,000	Leucocytosis	0	0.0
Platelet count (per µl) (n=60)			
> 1,50,000	Normal	7	11.7
1,50,000 to 1,00,000	Mild Thrombocytopenia	9	15.0
1,00,000 to 50,000	Moderate Thrombocytopenia	39	65.0
< 50,000	Severe Thrombocytopenia	5	8.3

Table shows out of 60 CLD patients, leukopenia (<4,000/cumm) was seen in 16 (26.7%), normal counts (4,000–11,000/cumm) in 44 (73.3%), and leucocytosis (>11,000/cumm) in 0 (0%). Platelet counts were normal (>1,50,000/µl) in 7 (11.7%), mildly low (1,50,000–1,00,000/µl) in 9 (15.0%), moderately low (1,00,000–50,000/µl) in 39 (65.0%), and severely low (<50,000/µl) in 5 (8.3%)

Table 6: Showing distribution of coagulation profile, biochemical parameters, hepatic encephalopathy, and child-turcotte-pugh score in chronic liver disease patients (n=60)

INR	Frequency (n=60)	Percentage (%)
<1.1	18	30.0
1.2 to 1.5	21	35.0
1.6 to 2	18	30.0
≥ 2.1	3	5.0
Prothrombin Time		
9 to 12 seconds	7	11.7
Prolonged	53	88.3
Albumin (g/dL)		
< 2.8	23	38.33
2.8 to 3.5	37	61.67
> 3.5	0	0
Total Bilirubin(mg/dL)		
< 2mg/dL	40	66.7
2 – 3mg/dL	17	28.3
> 3mg/dL	3	5.0
Hepatic Encephalopathy		
Grade 1	51	85.0
Grade 2	7	11.7
Grade 3	2	3.3
Child-Turcotte-Pugh Score		
A	1	1.7
B	47	78.3
C	12	20

Table shows that among 60 CLD patients, **INR** was <1.1 in 18 (30.0%), 1.2–1.5 in 21 (35.0%), 1.6–2.0 in 18 (30.0%), and ≥2.1 in 3 (5.0%). **Prothrombin Time** was normal (9–12 seconds) in 7 (11.7%) patients and prolonged in 53 (88.3%). **Albumin** levels were <2.8 g/dL in 23 (38.33%) and 2.8–3.5 g/dL in 37 (61.67%), with none having levels >3.5 g/dL. **Total Bilirubin** was <2 mg/dL in 40 (66.7%), 2–3 mg/dL in 17 (28.3%), and >3 mg/dL in 3 (5.0%). **Hepatic Encephalopathy** was Grade 1 in 51 (85.0%), Grade 2 in 7 (11.7%), and Grade 3 in 2 (3.3%). Based on the **Child-Turcotte-Pugh Score**, 1 (1.7%) patient was Class A, 47 (78.3%) were Class B, and 12 (20.0%) were Class C.

CONCLUSION

As per the study conducted among 60 Chronic liver disease patients, we inferred many conclusive results regarding the hematological and hemostatic abnormalities in chronic liver disease patients. Most of the patients were males and most of them were within age group of 41-50. Alcohol was found to be most common cause of chronic liver disease.The complete blood count had shown that the majority of patients had anaemia, leukopenia, and thrombocytopenia. The overall incidence of anaemia was 56.6%, which was found to be mild in 11.8% patients, moderate in 79.4% patients, and severe in 8.8 patients. Microcytic hypochromic anaemia and normocytic normochromic anaemia were the most common and second most common finding respectively in chronic liver disease as inferred from the study. Leucopenia was present in 26.7% patients. Majority of the patients had thrombocytopenia of moderate severity with prolonged PT in most of the patients. In this study 100% of patients had low albumin levels. Majority of patients had grade 1 hepatic encephalopathy. As per Child-Turcotte-Pugh score 78.3% patients were under class B category. Assessing the severity and type of anaemia is a useful tool for early initiation of the treatment in patients of CLD for reducing the morbidity and mortality. Early detection and treatment of haematological changes can prevent complications and reduce the mortality in CLD patients.

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