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A STUDY ON THE PREVALENCE OF ANEMIA IN PATIENTS WITH LIVER CIRRHOSIS IN A TERTIARY CARE CENTRE

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ABSTRACT

Background: Anemia is one of the most common hematological abnormalities observed in patients with liver cirrhosis. It develops due to multiple mechanisms, including chronic gastrointestinal blood loss, nutritional deficiencies, hypersplenism, bone marrow suppression, and altered iron metabolism. The presence of anemia is associated with increased disease severity, poor quality of life, and adverse clinical outcomes. However, data regarding its prevalence among patients with liver cirrhosis remain limited in many tertiary care settings.

Aim: To determine the prevalence of anemia among patients with liver cirrhosis attending a tertiary care center.

Materials and Methods:A hospital-based observational cross-sectional study was conducted in the Department of General Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Ambala, Haryana, during 2024–2025. A total of 100 adult patients with liver cirrhosis diagnosed by clinical examination, ultrasonography, and biochemical investigations were included. Demographic characteristics, hematological parameters, peripheral blood smear findings, iron profile, Child–Pugh classification, and etiological factors were recorded. Anemia was diagnosed according to the World Health Organization criteria. Statistical analysis was performed to determine prevalence and assess associations between anemia severity and clinical variables.

Results: Among the 100 patients, 77% had anemia, while 23% had normal hemoglobin levels. Moderate anemia (40%) was the most frequent category, followed by severe (22%) and mild anemia (15%). The mean hemoglobin level was 9.99 ± 2.99 g/dL. Macrocytic morphology was the predominant peripheral smear finding (31%). Child–Pugh Class B constituted the largest group (47%). Significant associations were observed between anemia severity and splenomegaly ($p = 0.002$), etiology of liver disease ($p = 0.038$), and Child–Pugh class ($p < 0.01$). Hemoglobin levels declined significantly with increasing severity of liver disease.

Conclusion: Anemia is highly prevalent among patients with liver cirrhosis and is strongly associated with disease severity. Routine hematological evaluation and early identification of anemia are essential for optimizing patient management and improving clinical outcomes.

KEYWORDS: Liver cirrhosis; Anemia; Hemoglobin; Child–Pugh classification; Splenomegaly; Iron profile; Chronic liver disease; Prevalence.

INTRODUCTION

The last common pathway of chronic liver damage brought on by a variety of etiologies, such as autoimmune hepatitis, alcohol-associated liver disease, chronic viral hepatitis, non-alcoholic fatty liver disease (NAFLD), and metabolic diseases, is liver cirrhosis. It is characterized by increasing fibrosis, distortion of hepatic architecture, and the production of regenerative nodules, ultimately leading to decreased liver function and portal hypertension. Cirrhosis continues to be a significant cause of morbidity and mortality worldwide, contributing significantly to hospital admissions and healthcare costs. Cirrhosis complications continue to negatively impact patients' quality of life and survival despite advancements in treatment therapies and preventive measures. Anemia is one of the most common but sometimes overlooked hematological abnormalities seen in clinical practice among these consequences. [1,2]

Patients with liver cirrhosis experience anemia, a multifactorial illness brought on by a complex interaction of processes. One of the main causes is still persistent gastrointestinal blood loss brought on by esophageal varices, portal hypertensive gastropathy, gastric antral vascular ectasia, and peptic ulcer disease. Inadequate dietary intake, malabsorption, persistent alcoholism, and altered metabolism also frequently result in nutritional deficits affecting iron, folate, and vitamin B12. While chronic inflammation inhibits erythropoiesis through cytokine-mediated pathways, hypersplenism linked to portal hypertension adds to excessive red blood cell sequestration and destruction. Anemia in these patients can also be made worse by bone marrow suppression brought on by medicines, viral infections, alcohol poisoning, or severe liver dysfunction.[3, 4]

Studies have shown that the prevalence of anemia in people with liver cirrhosis ranges from roughly 50% to over 75%, depending on the demographic and stage of the disease. Worsening hepatic dysfunction and higher scores on prognostic indices like the Child–Pugh classification and the Model for End-stage Liver Disease (MELD) score typically result in more severe anemia. Increased portal hypertension, repeated gastrointestinal bleeding, extended hospital stays, increased transfusion needs, and a poor prognosis have all been linked to hemoglobin decline. As a result, anemia is becoming more widely acknowledged as a significant indicator of cirrhosis severity and clinical outcomes rather than just a laboratory anomaly.[5, 6]

Due to the frequent coexistence of numerous etiologies, the examination of anemia in cirrhotic patients necessitates a comprehensive approach. Complete blood counts, red blood cell indices, peripheral blood smear examinations, iron studies, serum ferritin, vitamin B12, folate levels, reticulocyte counts, and assessments for occult or overt gastrointestinal bleeding are all part of the assessment. Determining the primary cause is crucial because management approaches vary significantly based on the underlying mechanism. Correcting nutritional deficiencies, controlling portal hypertensive bleeding, endoscopic procedures, pharmacological therapy, blood transfusions, erythropoiesis-stimulating agents in certain patients, or, if necessary, definitive management through liver transplantation are all examples of appropriate treatment.[7, 8]

Understanding anemia's prevalence in patients with liver cirrhosis is essential for enhancing patient treatment because of the condition's high frequency and important clinical ramifications. Early detection improves quality of life and may increase longevity by enabling prompt diagnostic investigation, focused treatment, and the avoidance of problems. However, the prevalence of anemia in cirrhosis patients varies by geographic location, liver disease aetiology, nutritional status, and access to healthcare. In order to produce local epidemiological data that can direct clinical practice, hospital-based research continue to be crucial. In order to better understand this prevalent but clinically relevant consequence, the current study was conducted to assess the hematological profile of patients with liver cirrhosis and to ascertain the prevalence of anemia in these individuals.[9, 10]

The purpose of this study was to evaluate the burden of anemia in this patient population and ascertain the prevalence of anemia among liver cirrhosis patients receiving treatment at a tertiary care facility. Estimating the prevalence of anemia in patients with liver cirrhosis was the main goal.

MATERIALS AND METHODS

Study Design: Hospital-based observational cross-sectional study.

Study Population:A total of 100 adult patients (>18 years) diagnosed with liver cirrhosis based on clinical examination, ultrasonography, and biochemical investigations, attending the Outpatient Department (OPD), General Medicine wards, and Intensive Care Unit (ICU).

Sample Size:100 patients with liver cirrhosis.

Study Duration:2024–2025.

Study Place: Department of General Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Ambala, Haryana.

Inclusion Criteria:

- Patients aged ≥18 years.
- Patients diagnosed with liver cirrhosis based on clinical examination, ultrasonography, and biochemical investigations.
- Patients admitted to the General Medicine wards or ICU, or attending the OPD.

Exclusion Criteria:

- Patients with malignancy.
- Pregnant women.
- Patients with a previous history of hematological or coagulation disorders unrelated to chronic liver disease.
- Patients already receiving treatment for anemia before the diagnosis of liver cirrhosis.
- Patients with chronic kidney disease (CKD).

Statistical Analysis: The collected data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. The prevalence of anemia was calculated as the proportion of patients with hemoglobin values below the World Health Organization (WHO) diagnostic criteria. Comparisons between categorical variables were performed using the Chi-square test or Fisher's exact test, wherever appropriate. Differences in the mean values of continuous variables between two groups were assessed using the independent Student's t-test, whereas comparisons among more than two groups were performed using one-way analysis of variance (ANOVA) followed by appropriate post hoc analysis when required. A p-value of <0.05 was considered statistically significant. Results were presented in the form of tables and graphs for appropriate interpretation.

RESULT

Table 1. Demographic Characteristics of the Study Population

Variable	Category	Frequency (n)	Percentage (%)
Gender	Male	79	79
	Female	21	21
Age Group (years)	18–30	5	5
	31–40	23	23
	41–50	28	28
	51–60	27	27
	>60	17	17
Mean Age (years)		49.19 ± 12.46	

Table 2. Prevalence, Severity of Anemia and Hematological Profile

Variable	Category/Parameter	Value
Anemia Status	Present	77 (77.0%)
	Absent	23 (23.0%)
Severity of Anemia	Normal	23 (23.0%)
	Mild	15 (15.0%)
	Moderate	40 (40.0%)
	Severe	22 (22.0%)
CBC Parameters	Hemoglobin (g/dL)	9.99 ± 2.99
	RBC ($\times 10^6/\mu L$)	3.32 ± 1.00
	MCV (fL)	88.24 ± 11.09
	MCHC (g/dL)	32.95 ± 2.92
	Platelet Count ($\times 10^3/\mu L$)	145.41 ± 117.88
Peripheral Blood Film	Normal	18 (18.0%)
	Microcytic	28 (28.0%)
	Macrocytic	31 (31.0%)
	Normocytic	17 (17.0%)
	Dimorphic	6 (6.0%)

Table 3. Iron Profile and Child–Pugh Classification

Variable	Category/Parameter	Value
Iron Profile	Serum Iron (µg/dL)	57.32 ± 47.96
	TIBC (µg/dL)	261.40 ± 109.53
	Serum Ferritin (ng/mL)	215.08 ± 218.90
	Transferrin Saturation (%)	14.60 ± 8.12
Child–Pugh Class	A	35 (35.0%)
	B	47 (47.0%)
	C	18 (18.0%)

Table 4. Association of Anemia Severity with Clinical Variables

Variable	Mild	Moderate	Severe	Normal	P-value
Splenomegaly Present	9	34	14	6	**0.002**
Splenomegaly Absent	6	6	8	17	
Alcoholic CLD	8	25	16	8	**0.038**
Viral CLD	7	15	6	15	

Table 5. Relationship Between Child–Pugh Class and Anemia

Variable		Mean ± SD	P-value
Hemoglobin according to Child–Pugh Class	Class A	10.67 ± 1.96	**<0.01**
	Class B	9.50 ± 2.65	
	Class C	7.83 ± 1.58	
Mean Child–Pugh Score according to Anemia Severity	Normal	5.67 ± 1.96	**<0.01**
	Mild	7.50 ± 2.65	
	Moderate	7.83 ± 1.58	
	Severe	8.33 ± 1.88	

Figure: 1. Association of Anemia Severity with Clinical Variables

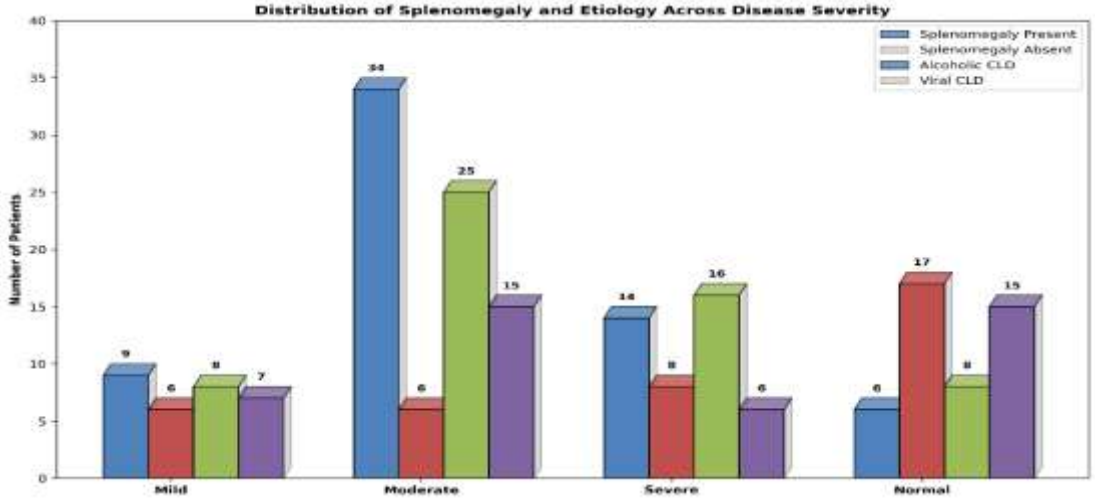
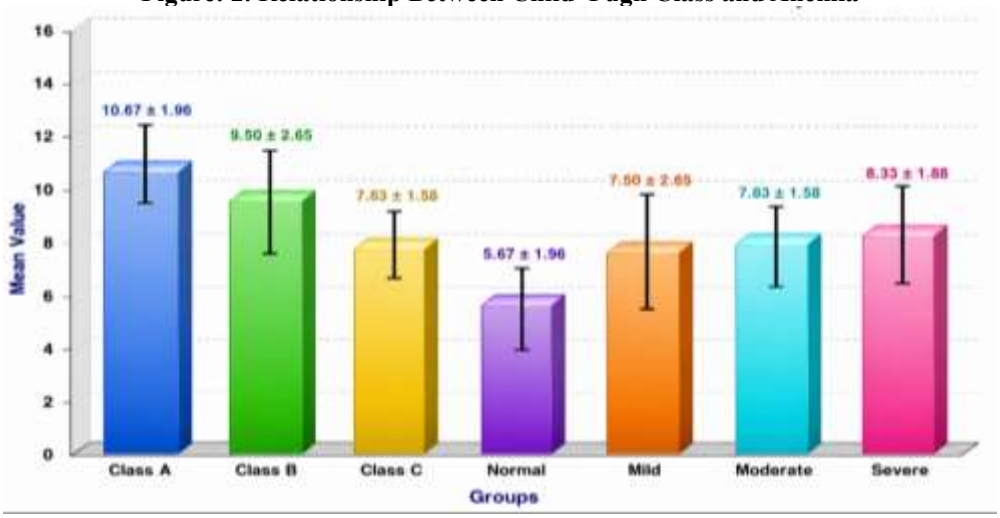


Figure: 2. Relationship Between Child–Pugh Class and Anemia



A total of 100 patients with liver cirrhosis were included in the present study. Among them, 79 (79.0%) were males and 21 (21.0%) were females, demonstrating a marked male predominance with a male-to-female ratio of approximately 3.8:1. Regarding age distribution, the largest proportion of patients belonged to the 41–50 years age group (28, 28.0%), followed closely by the 51–60 years age group (27, 27.0%). Patients aged 31–40 years accounted for 23 (23.0%), whereas 17 (17.0%) patients were older than 60 years. Only 5 (5.0%) patients belonged to the 18–30 years age group. The mean age of the study population was 49.19 ± 12.46 years, indicating that liver cirrhosis predominantly affected middle-aged and older adults in the present study.

Among the 100 patients with liver cirrhosis, 77 (77.0%) were diagnosed with anemia, while only 23 (23.0%) had normal hemoglobin levels, demonstrating a high prevalence of anemia in the study population. Based on WHO classification, 15 (15.0%) patients had mild anemia, 40 (40.0%) had moderate anemia, and 22 (22.0%) had severe anemia, whereas 23 (23.0%) patients were non-anemic. Thus, moderate anemia emerged as the most common category, and nearly two-thirds (62.0%) of the study population had moderate-to-severe anemia.

The hematological profile showed a mean hemoglobin level of 9.99 ± 2.99 g/dL, with values ranging from 3.1 to 17.9 g/dL, reflecting considerable variation in anemia severity. The mean RBC count was 3.32 ± 1.00 $\times 10^6/\mu L$, indicating reduced erythrocyte counts in many patients. The mean MCV was 88.24 ± 11.09 fL, suggesting the coexistence of both microcytic and macrocytic red cell populations. The mean MCHC was 32.95 ± 2.92 g/dL, indicating relatively preserved hemoglobin concentration within red blood cells. The mean platelet count was 145.41 ± 117.88 $\times 10^3/\mu L$, with a wide variation ranging from 20 to 619 $\times 10^3/\mu L$, consistent with thrombocytopenia commonly associated with liver cirrhosis.

Peripheral blood film examination demonstrated that macrocytic morphology was the most common finding, observed in 31 (31.0%) patients, followed by microcytic morphology in 28 (28.0%) patients and normocytic morphology in 17 (17.0%) patients. A dimorphic blood picture was present in 6 (6.0%) patients, while 18 (18.0%) patients had a normal peripheral blood smear.

The iron profile demonstrated substantial variability among patients with liver cirrhosis. The mean serum iron level was 57.32 ± 47.96 µg/dL, whereas the mean total iron-binding capacity (TIBC) was 261.40 ± 109.53 µg/dL. The mean serum ferritin concentration was 215.08 ± 218.90 ng/mL, reflecting a broad spectrum of iron stores among the patients. The mean transferrin saturation (TSAT) was 14.60 ± 8.12%, suggesting reduced iron availability in a considerable proportion of patients and indicating disturbed iron metabolism associated with chronic liver disease.

Assessment of liver disease severity using the Child–Pugh classification revealed that 47 (47.0%) patients belonged to Class B, making it the most frequent category. Thirty-five (35.0%) patients were classified as Child–Pugh Class A, whereas 18 (18.0%) patients were categorized as Class C, indicating that the majority of patients had moderate hepatic dysfunction.

The association between splenomegaly and anemia severity showed a statistically significant relationship ($p = 0.002$). Among patients with mild anemia, 9 had splenomegaly and 6 did not. In the moderate anemia group, splenomegaly was present in 34 patients compared with only 6 patients without splenomegaly. Similarly, among patients with severe anemia, 14 had splenomegaly and 8 did not, whereas among patients with normal hemoglobin levels, only 6 had splenomegaly while 17 had no splenomegaly. These findings suggest that worsening anemia is significantly associated with the presence of splenomegaly in patients with liver cirrhosis.

The severity of anemia also varied significantly according to the etiology of chronic liver disease ($p = 0.038$). Among the 57 patients with alcoholic liver disease, 8 had mild anemia, 25 had moderate anemia, 16 had severe anemia, and only 8 had normal hemoglobin levels. In comparison, among the 43 patients with viral liver disease, 7 had mild anemia, 15 had moderate anemia, 6 had severe anemia, and 15 had normal hemoglobin levels. Severe anemia was therefore more common in alcoholic liver disease (28.1%) than in viral liver disease (14.0%), whereas normal hemoglobin levels were observed more frequently among patients with viral liver disease.

A significant association was observed between liver disease severity and hemoglobin concentration. Patients belonging to Child–Pugh Class A had the highest mean hemoglobin level (10.67 ± 1.96 g/dL), followed by Class B (9.50 ± 2.65 g/dL), while the lowest hemoglobin level was observed among patients in Child–Pugh Class C (7.83 ± 1.58 g/dL). The progressive decline in hemoglobin concentration with increasing Child–Pugh class was statistically significant ($p < 0.01$), indicating that anemia becomes more pronounced as hepatic dysfunction advances.

Similarly, the mean Child–Pugh score increased progressively with worsening anemia severity. Patients without anemia had the lowest mean Child–Pugh score (5.67 ± 1.96), whereas patients with mild, moderate, and severe anemia had progressively higher scores of 7.50 ± 2.65, 7.83 ± 1.58, and 8.33 ± 1.88, respectively. This association was highly statistically significant ($p < 0.01$) and suggests that patients with more severe anemia tend to have more advanced liver disease.

DISCUSSION

Anemia is a very prevalent consequence that affects over three-fourths of the study group, according to the current study, which assessed the prevalence and hematological profile of anemia among patients with liver cirrhosis. The results also show a strong correlation between the severity of anemia and the advancement of liver disease, highlighting the significance of regular hematological evaluation in patients with long-term liver illness.

With a male-to-female ratio of 3.8:1 and a mean age of 49.19 ± 12.46 years, the study population was primarily male (79%). The majority of patients were between the ages of 41 and 60, suggesting that middle-aged individuals are the main demographic affected by liver cirrhosis. Similar findings were observed by Gkamprela et al., who discovered a distinct male predominance among cirrhotic patients, which was mostly due to the higher incidence of alcohol-related liver disease in men.[11] Similarly, Qamar and Grace found that men make up the majority of hospitalized patients due to higher exposure to alcohol and other risk factors, and that chronic liver disease typically manifests in the fifth and sixth decades of life.[12] Thus, the current study's demographic results are in line with earlier research.

One of the study's primary conclusions was that just 23% of patients with liver cirrhosis had normal hemoglobin levels, whereas 77% of patients had anemia. Nearly two-thirds of patients had moderate-to-severe anemia, and moderate anemia was the most common category (40%). These findings are comparable with those reported by Les et al., who observed anemia in around 75% of patients with advanced cirrhosis and concluded that its prevalence increased with disease progression.[13] Similarly, Paternostro et al. showed that anemia, which affects roughly 66–75% of patients with cirrhosis, is one of the most common hematological abnormalities. This is mainly due to gastrointestinal blood loss, hypersplenism, nutritional deficiencies, chronic inflammation, and impaired erythropoiesis.[14] Anemia is a significant clinical issue in cirrhotic patients, as confirmed by the prevalence found in this study, which is within this reported range.

The hematological profile indicated a mean hemoglobin content of 9.99 ± 2.99 g/dL, accompanied by lower mean RBC counts and substantial variability in platelet counts, confirming the coexistence of anemia and thrombocytopenia. Maruyama et al. observed similar results, stating that decreased platelet and hemoglobin counts are typical haematological signs of portal hypertension and hypersplenism in cirrhosis.[15] Similarly, portal

hypertension induces splenic sequestration of both erythrocytes and platelets, and poor hepatic synthetic function further leads to bone marrow suppression and altered hematopoiesis, which is why thrombocytopenia and anemia often coexist, according to Afdhal et al.[16] These observations closely reflect the findings of the present investigation.

Peripheral blood smear examination in the current study indicated that macrocytic anemia was the most common morphological pattern (31%), followed by microcytic (28%) and normocytic anemia (17%). Ballard found similar morphological distributions and identified macrocytosis as a typical characteristic of chronic liver illness, especially in patients with alcohol-associated cirrhosis because of direct toxic effects on erythroid precursors and related folate insufficiency.[17] Normocytic anemia, on the other hand, represents anemia of chronic illness, while microcytic anemia is typically linked to iron deficiency and persistent gastrointestinal blood loss. Thus, the complex pathophysiology of anemia in liver cirrhosis is supported by the coexistence of diverse red cell morphologies in this study.With significantly fluctuating serum ferritin contents, iron profile analysis revealed comparatively low mean serum iron and transferrin saturation. Rather than an isolated iron deficiency, this pattern points to disrupted iron metabolism. Darshan and Anderson published similar results, emphasizing that despite functional iron deficit, serum ferritin may stay increased in cirrhosis due to prolonged inflammation and hepatocellular damage.[18] Additionally, they found that many cirrhotic individuals had lower transferrin saturation, which is indicative of poor iron use rather than a reduction in the body's total iron reserves. Thus, the current results highlight how difficult it is to interpret iron studies in patients with chronic liver disease.

When the Child-Pugh classification was used to assess the severity of liver disease, Class B made up the biggest group (47%), followed by Class A (35%) and Class C (18%). Similar distributions have been shown by Hung et al., who noted that hospitalized cirrhosis patients most often fall into Child-Pugh Class B due to the fact that patients in this stage often experience problems that necessitate admission to tertiary care.[19] Their results are in line with the current investigation and show that the majority of hospitalized cirrhotic patients had moderate hepatic impairment.

Splenomegaly and anemia severity were shown to be statistically significantly correlated ($p = 0.002$), with splenomegaly being much more common in patients with moderate and severe anemia. This result confirms the known part hypersplenism plays in the etiology of anemia in patients with cirrhosis. Peck-Radosavljevic also showed that when liver illness progresses, portal hypertension-induced splenic enlargement causes excessive sequestration and early erythrocyte destruction, which significantly contributes to anemia.[20] Thus, the current results offer more proof that hypersplenism plays a significant role in determining the severity of anemia in liver cirrhosis.The severity of anemia was also influenced by the cause of chronic liver illness. The percentage of severe anemia was substantially higher in patients with alcoholic liver disease than in those with viral liver disease ($p = 0.038$). Because long-term alcohol use aggravates anemia through direct bone marrow suppression, folate insufficiency, gastrointestinal bleeding, and macrocytosis, this discovery is medically feasible. Numerous clinical trials have confirmed similar findings, with alcohol-related cirrhosis consistently linked to lower hemoglobin concentrations than viral etiologies.[17, 20]The current investigation also showed that the mean hemoglobin concentration significantly decreased as the Child-Pugh class increased. Child-Pugh Class A patients had the highest mean hemoglobin levels, while Class C patients had the lowest values ($p < 0.01$). On the other hand, as anemia severity worsened, mean Child-Pugh scores climbed gradually, suggesting that individuals with severe anaemia had more advanced liver impairment. Les et al. have reported similar correlations, finding that haemoglobin concentration independently declines as hepatic decompensation advances and is linked to worse clinical outcomes.[13] Similarly, Paternostro et al. found that anaemia is a significant indicator of the severity of the illness and is linked to higher rates of hospitalization, complications, and death in liver cirrhosis patients.[14] These results support the current investigation and imply that anaemia is a significant prognostic factor in chronic liver disease.

Overall, the results of this study demonstrate that anaemia is a very common and clinically significant consequence of liver cirrhosis and are consistent with recent research. Its complex aetiology, close correlation with Child-Pugh severity, and strong link with hypersplenism and alcoholic liver disease highlight the importance of thorough haematological screening and early treatment intervention in patients with chronic liver disease.

CONCLUSION

The present study demonstrated that anemia is a highly prevalent hematological complication among patients with liver cirrhosis, affecting 77% of the study population. Moderate anemia was the most common presentation, and the severity of anemia increased progressively with advancing liver dysfunction. Significant associations were observed between anemia severity and Child–Pugh class, splenomegaly, and the etiology of chronic liver disease, particularly alcoholic liver disease. Macrocytic anemia was the predominant morphological pattern, while iron profile abnormalities indicated disturbed iron metabolism in a substantial proportion of patients. These findings emphasize that anemia in liver cirrhosis is multifactorial and reflects both the underlying hepatic dysfunction and associated complications such as portal hypertension and hypersplenism. Routine evaluation of hemoglobin levels, peripheral blood smear, and iron studies should therefore be incorporated into the comprehensive

assessment of patients with liver cirrhosis. Early identification and appropriate management of anemia may improve functional status, reduce complications, enhance quality of life, and contribute to better overall clinical outcomes in this patient population.

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