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A STUDY OF HEMATOLOGICAL PROFILES IN CHRONIC LIVER DISEASE WITH SPECIAL REFERENCE TO ANEMIA, THROMBOCYTOPENIA, AND LEUKOPENIA

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ABSTRACT

Introduction:
Chronic liver disease (CLD) is a progressive disorder associated with significant morbidity and mortality due to hepatic and extrahepatic complications. Hematological abnormalities such as anemia, thrombocytopenia, and leukopenia are frequent in CLD and often correlate with disease severity. These simple, cost-effective parameters may provide valuable insight into prognosis and clinical outcomes, especially in resource-limited settings.

Aim and Objective:
This study aimed to evaluate the hematological profile of CLD patients, with special reference to the prevalence and patterns of anemia, thrombocytopenia, and leukopenia, and to assess their correlation with clinical features and disease severity.

Materials and Methods:
A prospective cohort study was conducted in the Department of General Medicine, G.R. Medical College, Gwalior, from April 2023 to September 2024. One hundred patients aged 18–60 years with CLD confirmed by ultrasonography were enrolled using purposive sampling. Patients with comorbidities, pregnancy, or prior anemia treatment were excluded. Hematological indices, peripheral smear findings, and biochemical parameters were recorded and statistically analyzed.

Results:
Moderate anemia was most common (37%), followed by severe (20%) and mild (16%). Normocytic normochromic morphology predominated (45%), with macrocytic and microcytic variants also present. Thrombocytopenia occurred in 52%, significantly associated with severe anemia ($p < 0.001$) and complications like hematemesis and ascites. Leukopenia was infrequent, while mortality reached 12%. Hypoalbuminemia showed strong correlation with advanced disease severity, underscoring its prognostic value.

Conclusion:
Anemia and thrombocytopenia are highly prevalent in CLD and correlate with clinical severity. Peripheral smear analysis remains a valuable, low-cost diagnostic adjunct. Hematological profiling should be integrated into routine CLD management for early risk stratification and prognostic evaluation.

Keywords: chronic liver disease; anemia; thrombocytopenia; leukopenia; peripheral smear

INTRODUCTION

Chronic liver disease (CLD) is a progressive condition marked by gradual deterioration of liver function over months to years, leading to cirrhosis, end-stage liver disease, or hepatocellular carcinoma. It arises from diverse causes including alcoholic liver disease, non-alcoholic fatty liver disease (NAFLD), viral hepatitis B and C, autoimmune disorders, and genetic conditions like hemochromatosis (1). Early stages are often asymptomatic, delaying diagnosis until complications occur. Lifestyle, genetic, and environmental factors influence its prevalence, with alcohol and viral hepatitis as major contributors and NAFLD rising with obesity and diabetes. WHO estimates attribute over two million annual deaths to liver disease, underscoring its global impact (2).

The progression of CLD carries substantial morbidity, characterized initially by nonspecific symptoms like fatigue, anorexia, or abdominal discomfort, before advancing to more severe manifestations including jaundice, ascites, pruritus, gastrointestinal hemorrhage, and hepatic encephalopathy (3). Cirrhosis, denoting advanced irreversible scarring, marks a critical stage with high risk for life-threatening complications such as portal hypertension, variceal bleeding, and hepatocellular carcinoma. The prognosis worsens with the onset of decompensation, where survival declines steeply. Liver cancer, a common sequela of cirrhosis, ranks among the leading causes of global cancer-related mortality. These outcomes underscore the severe consequences of unchecked CLD progression. Mortality is concentrated in those with advanced disease or severe complications, highlighting the importance of preventive and early intervention strategies (4).

Beyond individual health, CLD imposes considerable socioeconomic burdens. Patients often require frequent hospitalizations, specialized investigations, long-term medication, and complex interventions such as endoscopic therapy, transjugular intrahepatic portosystemic shunts, or even liver transplantation (5). The associated costs create heavy pressure on healthcare systems. Additionally, patients' quality of life is severely compromised by physical disability, psychological stress, and impaired work productivity. Families and caregivers experience long-term strain, and public health expenditure rises substantially, especially in low- and middle-income countries where resources are already limited. WHO predicts that unless preventive strategies are strengthened, global mortality from cirrhosis and related complications will continue to escalate in the coming decades (6).

The impact of CLD is not restricted to the liver; it exerts multisystem effects through mechanisms such as toxin accumulation, metabolic imbalance, and altered hemodynamics. Neurological impairment due to ammonia buildup results in hepatic encephalopathy, manifesting as confusion, personality changes, or coma. Renal impairment, often termed hepatorenal syndrome, develops from complex circulatory alterations. Disturbances in coagulation arise due to impaired synthesis of clotting factors, producing a delicate balance between bleeding and thrombosis. Susceptibility to infection is another hallmark, resulting from immune dysfunction (7). This systemic involvement complicates management, requiring multidisciplinary care from hepatologists, gastroenterologists, nephrologists, and critical care specialists. Early identification of complications such as portal hypertension or renal dysfunction allows timely interventions, improving both quality and expectancy of life (8).

Chronic liver disease (CLD) disrupts vital molecular functions including protein synthesis, detoxification, and metabolism. Reduced albumin and clotting factor production causes ascites, edema, and bleeding risks, while impaired detoxification leads to toxin accumulation and encephalopathy. Carbohydrate and lipid metabolic disturbances contribute to glucose imbalance, dyslipidemia, and cardiovascular vulnerability (9). Hematological abnormalities are frequent, with anemia arising from bleeding, deficiencies, hemolysis, and marrow suppression. Thrombocytopenia, driven by hypersplenism and reduced thrombopoietin, heightens bleeding risk, while leukopenia from splenic sequestration and marrow suppression increases infection susceptibility. Their coexistence

commonly indicates advanced disease and poor prognosis, complicating management and worsening patient outcomes (10).

The hematological changes in chronic liver disease arise from multifactorial mechanisms, including portal hypertension causing splenomegaly with sequestration of blood cells, nutritional deficiencies impairing hematopoiesis, chronic inflammation suppressing marrow, reduced thrombopoietin lowering platelet production, and coagulopathy from deficient clotting factors. These disruptions contribute to systemic consequences of hepatic failure (11). Anemia patterns in cirrhosis vary: normocytic normochromic from chronic gastrointestinal bleeding, microcytic hypochromic from iron deficiency, macrocytic from alcohol toxicity and folate deficiency, hemolytic from hypersplenism or autoimmunity, and anemia of chronic disease from impaired iron utilization. This multifactorial anemia necessitates individualized evaluation, nutritional support, bleeding control, and hypersplenism management (12).

Chronic liver disease (CLD) is a progressive condition with diverse causes such as alcohol, viral hepatitis, and NAFLD, leading to cirrhosis, cancer, and severe complications. It disrupts protein synthesis, detoxification, metabolism, and hematopoiesis, resulting in systemic effects (13). Hematological abnormalities like anemia, thrombocytopenia, and leukopenia serve as accessible, cost-effective markers of disease severity, portal hypertension, bleeding risk, and infection susceptibility. Monitoring these parameters is crucial for risk stratification, timely intervention, and guiding treatment, especially in resource-limited settings. Given its global burden and socioeconomic impact, CLD requires early recognition, multidisciplinary management, and continuous monitoring to improve outcomes and reduce mortality (14). nThe aim of this study is to evaluate the hematological profile in patients with chronic liver disease, focusing on the distribution and patterns of anemia. It also seeks to determine the prevalence of thrombocytopenia and leucopenia, which are common hematological abnormalities associated with disease progression. By analyzing these parameters, the study aims to provide insights into disease severity, complications, and their clinical significance in the overall management of chronic liver disease.

MATERIALS AND METHODS

This prospective cohort study was conducted in the Department of General Medicine, G.R. Medical College, Gwalior, from April 2023 to September 2024 to evaluate hematological profiles in 100 patients with chronic liver disease, particularly cirrhosis. Using purposive sampling, patients aged 18–60 years diagnosed by ultrasonography and consenting to participate were included, while those with comorbidities, pregnancy, or prior anemia treatment were excluded. The study focused on anemia, thrombocytopenia, and related abnormalities using standardized diagnostic criteria. Institutional Ethics Committee approval and written informed consent were obtained, ensuring confidentiality, patient rights, and adherence to ethical principles throughout the research process.

RESULTS

The study population was predominantly middle-aged, with 63% of cases between 41–60 years and 37% between 18–40 years, highlighting greater prevalence in the middle-aged group. Hemoglobin assessment revealed widespread anemia, as most patients had subnormal levels. The largest proportion (30%) fell in the 12.1–18 g/dL range, followed by 28% in the 8.1–10 g/dL range and 22% in the 6–8 g/dL range, reflecting varying degrees of anemia across the cohort.

Table 1: Classification Based on Anaemia Severity

Anaemia Category	Count	Percentage
Moderate Anaemia	37	37.0
Normal	27	27.0
Severe Anaemia	20	20.0
Mild Anaemia	16	16.0
Total	100	100.0

The table shows that moderate anaemia is the most common category (37%), followed by normal (27%) and severe anaemia (20%), with mild anaemia being the least prevalent (16%). This distribution highlights a significant burden of anaemia in the studied population, with nearly three-fourths of cases showing some degree of severity, underscoring its clinical importance.

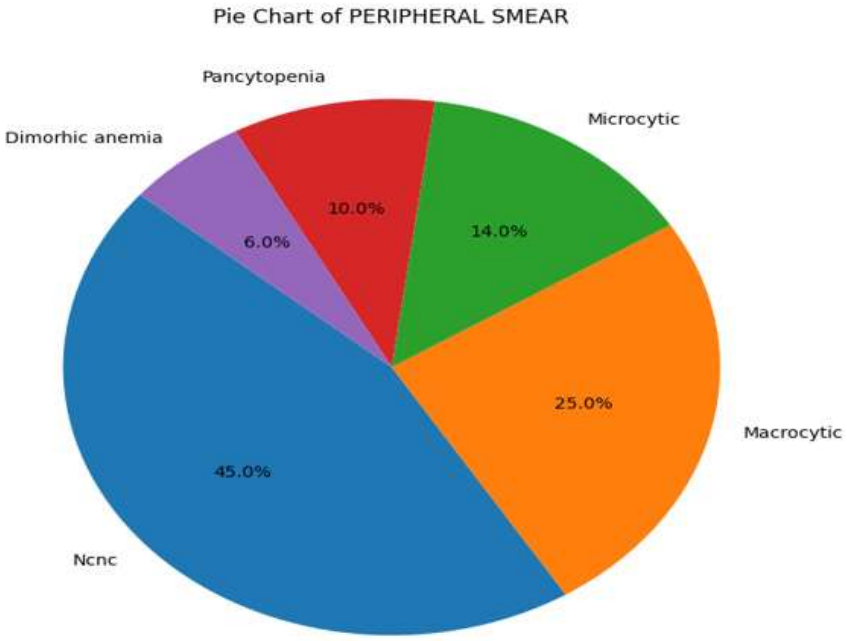

Figure 1: Distribution of Peripheral Smear Morphologies

The peripheral smear analysis reveals that normocytic normochromic (NCNC) morphology is most common (45%), followed by macrocytic (25%) and microcytic (14%) patterns, while pancytopenia (10%) and dimorphic anaemia (6%) are less frequent. This distribution indicates that although a large proportion of patients maintain near-normal morphology, a substantial subset shows pathological changes reflecting nutritional deficiencies, bone marrow suppression, or mixed etiologies. These findings emphasize the heterogeneity of haematological abnormalities in the studied population.

Table 2: Mortality Outcome Among Patients

DEATH	Count	Percentage
YES	12	12.0
No	88	88.0
Total	100	100.0

The data show that mortality occurred in 12% of patients, while 88% survived, reflecting a relatively favorable outcome in the majority. However, the observed death rate highlights the presence of a clinically significant risk subgroup, emphasizing the importance of early detection, appropriate interventions, and vigilant monitoring to reduce mortality.

Table 3: Descriptive Statistics of Clinical and Laboratory Parameters

Variable	Mean ± SD	Median	Range (Min - Max)
Age	44.10 ± 8.15	44.00	29.0 - 60.0
HEMOGLOBIN (gm/dl)	10.15 ± 2.88	10.00	4.0 - 16.0
PLATELETS	141.73 ± 70.36	107.50	30.0 - 337.0
WBC COUNT	6.68 ± 4.14	6.00	1.0 - 24.0
MCV	93.58 ± 20.56	88.50	52.0 - 137.0
T.BILIRUBIN	4.89 ± 5.46	2.00	0.0 - 34.0
D.BILIRUBIN	1.91 ± 2.43	1.00	0.0 - 11.0

AST	131.28 ± 162.62	67.50	22.0 - 1200.0
ALT	107.12 ± 203.76	45.50	11.0 - 1456.0
S. ALBUMIN(mg/dl)	2.49 ± 0.87	3.00	1.0 - 4.0
PT(Scc)	15.23 ± 2.51	15.00	11.0 - 23.0
INR(Scc)	1.01 ± 0.10	1.00	1.0 - 2.0

The descriptive data indicate a middle-aged cohort (mean age 44 years) with marked hematological and biochemical abnormalities. Hemoglobin and platelet counts are reduced, reflecting anemia and thrombocytopenia, while elevated bilirubin, AST, and ALT suggest significant hepatic dysfunction. Low serum albumin and prolonged PT/INR further highlight impaired synthetic liver function, consistent with advanced liver disease pathology.

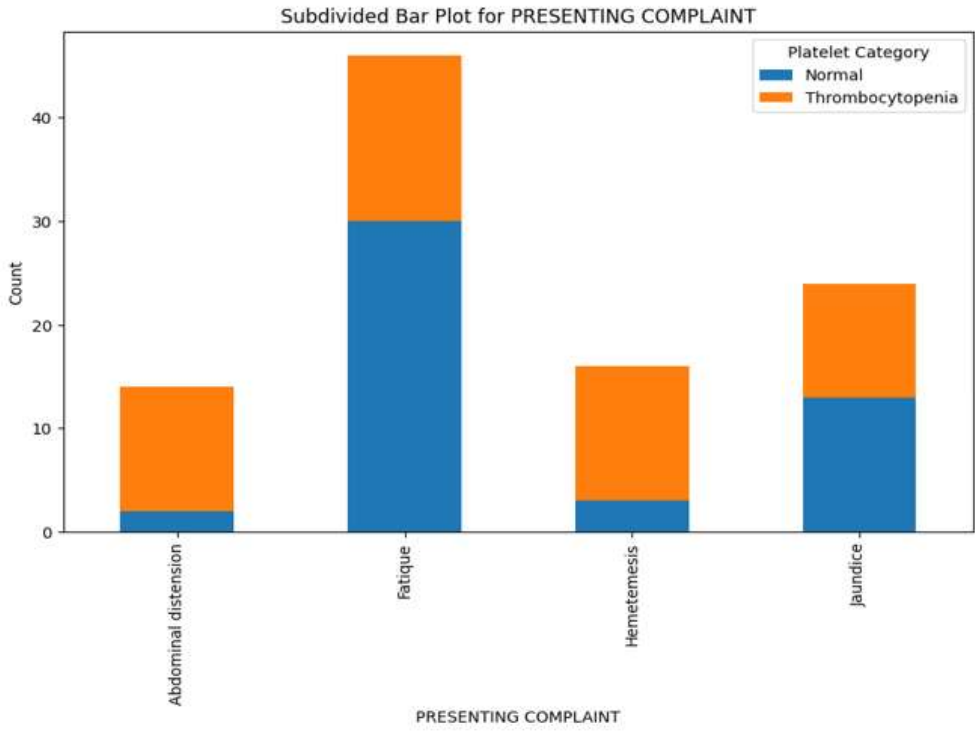

Figure 2: Platelet Count Variation Across Presenting Symptoms

The bar plot shows that fatigue was the most frequent presenting complaint, predominantly in patients with normal platelet counts, while abdominal distension and hematemesis were mainly associated with thrombocytopenia. Jaundice occurred in both groups but with a higher share of thrombocytopenic cases. This indicates that platelet abnormalities are closely linked with more severe clinical presentations such as bleeding and abdominal complications.

Table 4: Relationship Between Anaemia Severity and Platelet Status

Platelet Category	Normal	Thrombocytopenia	Total
Anaemia Category			
Mild Anaemia	8 (50.0%)	8 (50.0%)	16 (100.0%)
Moderate Anaemia	11 (29.73%)	26 (70.27%)	37 (100.0%)
Normal	25 (92.59%)	2 (7.41%)	27 (100.0%)
Severe Anaemia	4 (20.0%)	16 (80.0%)	20 (100.0%)
Total	48 (48.0%)	52 (52.0%)	100 (100.0%)
'Chi-square test statistic: 32.7661, P-value: 0.0000'			

The analysis shows a significant association between anaemia severity and platelet status ($\chi^2 = 32.77$, $p < 0.001$). While most patients with normal haemoglobin had normal platelet counts, moderate and severe anaemia were strongly associated with thrombocytopenia, reaching 70–80%. This indicates that worsening anaemia correlates with increased risk of platelet depletion, reflecting advanced disease severity.

Table 5: Comparative Analysis of Clinical and Laboratory Parameters Based on Platelet Status

	Platelet Category			
Variable	**Normal Mean +- SD Median Range**	**Thrombocytopenia Mean +- SD Median Range**	**Test Stat**	**P-Value**
Age	44.19 ± 8.66, 43.5, (29.0-59.0)	44.02 ± 7.74, 44.0, (30.0-60.0)	0.0003	0.9862
HEMOGLOBIN (gm/dl)	11.77 ± 2.63, 12.5, (6.0-16.0)	8.65 ± 2.23, 8.0, (4.0-15.0)	28.6176	0.0000
PLATELETS	207.02 ± 39.74, 200.0, (151.0-337.0)	81.46 ± 20.96, 89.0, (30.0-110.0)	74.2754	0.0000
WBC COUNT	6.5 ± 3.08, 6.0, (1.0-15.0)	6.85 ± 4.95, 6.0, (1.0-24.0)	0.2420	0.6228
MCV	92.21 ± 18.33, 89.0, (60.0-137.0)	94.85 ± 22.53, 87.0, (52.0-137.0)	0.0747	0.7846
T.BILIRUBIN	5.38 ± 6.88, 2.0, (0.0-34.0)	4.44 ± 3.72, 4.5, (0.0-14.0)	0.1929	0.6605
D.BILIRUBIN	1.71 ± 2.37, 1.0, (0.0-10.0)	2.1 ± 2.48, 1.0, (0.0-11.0)	1.5348	0.2154
AST	107.6 ± 87.22, 71.0, (27.0-433.0)	153.13 ± 208.12, 66.0, (22.0-1200.0)	0.1391	0.7092
ALT	64.98 ± 77.18, 46.5, (11.0-417.0)	146.02 ± 268.13, 44.5, (11.0-1456.0)	0.6092	0.4351
S. ALBUMIN(mg/dl)	2.92 ± 0.61, 3.0, (1.0-4.0)	2.1 ± 0.89, 2.0, (1.0-4.0)	23.0235	0.0000
PT(Scc)	15.21 ± 2.04, 15.0, (12.0-22.0)	15.25 ± 2.9, 15.0, (11.0-23.0)	0.0129	0.9096
INR(Scc)	1.0 ± 0.0, 1.0, (1.0-1.0)	1.02 ± 0.14, 1.0, (1.0-2.0)	0.9231	0.3367

The comparative analysis shows that patients with thrombocytopenia had significantly lower hemoglobin ($p < 0.001$), reduced serum albumin ($p < 0.001$), and markedly lower platelet counts ($p < 0.001$) compared to those with normal platelets. Other parameters, including liver enzymes, bilirubin, and coagulation indices, did not differ significantly. This suggests that thrombocytopenia in these patients is strongly linked with worsened anemia and impaired liver synthetic function, rather than generalized hepatic injury.

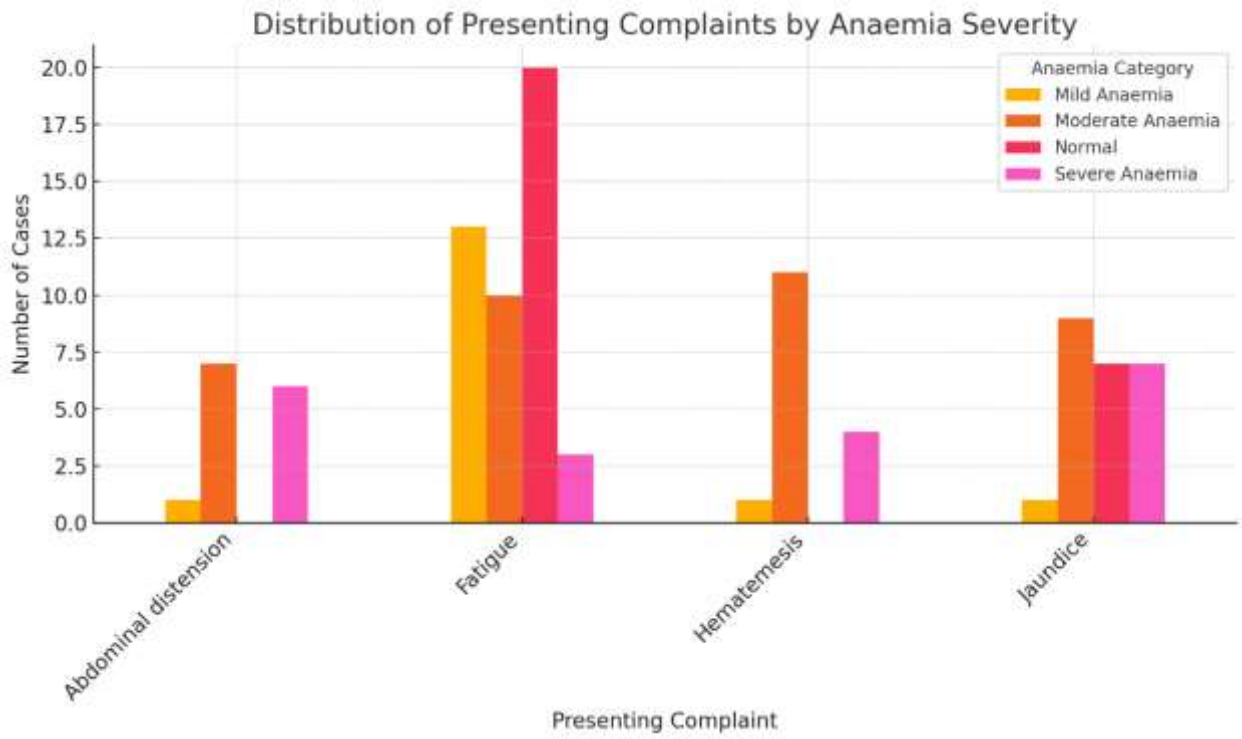

Figure 3: Distribution of Presenting Complaints by Anaemia Severity

The chart shows that fatigue was the most common presenting complaint across all anaemia categories, especially in moderate anaemia. Abdominal distension and hematemesis were more frequent in moderate and severe anaemia, while jaundice appeared consistently across different severities. This indicates that symptom burden intensifies with worsening anaemia, reflecting both systemic and hepatic involvement.

DISCUSSION

The present study highlights the hematological disturbances observed in patients with chronic liver disease (CLD) and demonstrates their significant association with clinical presentation and disease severity. Anemia was the most frequent abnormality, affecting nearly three-fourths of the cohort, with moderate anemia being the predominant category, followed by severe and mild forms. The etiology of anemia in CLD is multifactorial, encompassing iron deficiency, folate and vitamin B12 deficiency, gastrointestinal blood loss, hemolysis, alcohol-induced bone marrow suppression, and hypersplenism (15). A strong correlation was found between anemia severity and Child-Pugh (CP) class, with moderate and severe anemia clustering in CP Class B and C patients, while CP Class A patients mostly had mild or no anemia. These findings are consistent with those reported by Joshi et al., who documented that the majority of anemic patients were in CP Class B and C (16), and with Scheiner et al., who also observed a higher prevalence of anemia in decompensated cirrhosis compared with compensated cases (17). This underlines the fact that anemia severity reflects underlying hepatic dysfunction and can serve as a surrogate marker for disease progression.

Peripheral smear analysis further enriched these observations by providing morphological patterns of anemia. Normocytic normochromic anemia was most common, followed by macrocytic and microcytic types, while pancytopenia and dimorphic anemia, though less frequent, were significantly associated with moderate to severe anemia ($\chi^2 = 21.3787$, $P = 0.0451$). Macrocytosis was especially linked to alcoholism, a finding that corroborates the work of Patel and Shah, who demonstrated that macrocytic anemia predominated among alcohol-related CLD patients (18). In contrast, Joshi et al. found normocytic normochromic anemia with thrombocytopenia in 28.7% of cases, while Scheiner et al. observed normocytic patterns in 78% of their CLD cohort, with microcytic and macrocytic forms accounting for 13% and 9%, respectively (16, 17). Together, these studies reinforce that smear

morphology reflects the underlying pathophysiological mechanisms of liver disease and may guide clinical management.

Thrombocytopenia was another striking abnormality, seen in more than half of patients. Its prevalence correlated with clinical complications, being significantly associated with hematemesis (81.25%) and abdominal distension (85.71%) ($p = 0.0005$). Severe anemia was also linked with the highest rates of thrombocytopenia (80%, $p < 0.0001$), suggesting that combined cytopenias signal advanced disease. The pathogenesis of thrombocytopenia involves splenic sequestration, reduced hepatic production of thrombopoietin, and bone marrow suppression (19). These mechanisms explain the close relationship between low platelet counts and clinical bleeding manifestations in CLD.

The mortality outcome in this cohort was 12%, with 88% surviving during the observation period. Although hematological abnormalities were common and often severe, the relatively favorable survival may reflect timely diagnosis and supportive therapy. Descriptive statistics of laboratory parameters revealed reduced mean hemoglobin (10.15 g/dl), low platelet counts (141.73 $\times 10^3/\mu L$), and hypoalbuminemia (2.49 g/dl), alongside elevated bilirubin and transaminases, all of which confirm systemic involvement of the liver. Coagulation studies demonstrated prolonged prothrombin time, consistent with impaired synthesis of clotting factors. These findings are in line with those of Allison MG et al., who noted similar trends of progressive hematological and biochemical deterioration with worsening CP scores (20).

The comparison of hematological indices across CP score groups provided further insights. Hemoglobin and platelet counts declined significantly from CP A to CP B, while bilirubin and prothrombin time increased, and albumin levels fell. These results reinforce that hematological parameters can be integrated with CP classification for prognostic assessment. Serum albumin, in particular, emerged as a strong indirect marker of both nutritional status and liver dysfunction. The observations echo prior reports by Joshi et al. (16), Scheiner et al. (17), and Allison MG et al. (20), who emphasized the clinical utility of hematological markers in stratifying disease severity, this study reaffirms that anemia, thrombocytopenia, and their morphological correlates are not only frequent but are strongly associated with disease stage, clinical manifestations, and prognosis in CLD. Simple, cost-effective investigations such as complete hemogram, peripheral smear, and coagulation profile provide valuable insights comparable to complex scoring systems. The consistency of these findings with prior studies (15, 21) strengthens their validity and underlines the role of hematological profiling as an integral component of evaluation and management in chronic liver disease.

CONCLUSION

In this study, anemia was observed in nearly three-fourths of patients, most frequently in its moderate form, with normocytic normochromic patterns (45%) pointing to chronic disease rather than nutritional deficiencies. Thrombocytopenia was present in 52% and often coexisted with anemia, strongly linked to severe clinical events like hematemesis and ascites, underscoring their prognostic significance. Peripheral smear examination proved valuable as a simple, cost-effective diagnostic tool. Patients with advanced liver disease (CP Score C) exhibited marked hematologic derangements, including lower hemoglobin, platelets, and albumin with elevated bilirubin, highlighting albumin as a key surrogate marker for severity and nutritional compromise.

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