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Research Article

Haematological Abnormalities in Decompensated Chronic Liver Disease

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A B S T R A C T

Introduction: Decompensated chronic liver disease (DCLD) represents an advanced stage of hepatic dysfunction, often accompanied by significant hematological abnormalities. These include anemia, thrombocytopenia, and coagulation disorders, which not only reflect disease severity but also influence the risk of complications such as gastrointestinal (GI) bleeding. This study aimed to assess the prevalence, patterns, and clinical significance of hematological abnormalities in DCLD patients.

Materials and Methods: This study was conducted in the Department of General Medicine. Fifty adult patients diagnosed with DCLD were enrolled based on predefined inclusion and exclusion criteria. Relevant clinical history, physical examination, and laboratory investigations including complete blood count, peripheral smear, liver function tests, serum iron profile, serum folate, prothrombin time (PT), and international normalized ratio (INR) were recorded and analyzed.

Results: Among 50 DCLD patients, 42 (84%) were males. Alcoholic cirrhosis (66%) was the most common etiology. Anemia was observed in 94% of patients, with normocytic morphology being the most frequent (57.4%). Thrombocytopenia was present in 72% of cases, and prolonged PT and elevated INR were noted in 68% and 74% of patients, respectively. A statistically significant association was found between thrombocytopenia and GI bleeding ($p = 0.047$), and also between GI bleeding and elevated PT ($p = 0.009$) and INR ($p = 0.012$).

Conclusion: Hematological abnormalities are highly prevalent in DCLD and show a strong association with bleeding complications. Early identification and appropriate management of these derangements are crucial to improving patient outcomes.

Keywords: Hematological, DCLD, Thrombocytopenia

Introduction

Decompensated chronic liver disease (DCLD) constitutes a critical stage in the natural history of chronic liver disease (CLD) and remains a major contributor to global morbidity and mortality. The progression from compensated to decompensated cirrhosis is defined by the development of life-threatening complications, including ascites, hepatic encephalopathy, variceal hemorrhage, and jaundice, all of which signify advanced hepatic insufficiency and portend a poor prognosis.[1–4] Histologically, cirrhosis—the terminal phase of CLD—is characterized by hepatocyte necrosis, fibrosis, and the formation of regenerative nodules, culminating in irreversible architectural distortion and functional decline of the liver.[5]

Globally, cirrhosis accounts for an estimated 800,000 deaths annually and affects more than 160 million individuals, underscoring the urgent need for comprehensive management strategies targeting both hepatic and extrahepatic manifestations of the disease.[6] Among the extrahepatic complications, haematological abnormalities are frequently encountered and play a pivotal role in the clinical course of DCLD. These include anemia, leukopenia, thrombocytopenia, and coagulopathies, each arising from multifactorial etiologies and contributing to increased morbidity in this patient population.[7–9]

Thrombocytopenia is the most common hematological abnormality in cirrhosis, with a reported prevalence ranging from 64% to 84% in affected individuals. The pathogenesis is multifactorial, encompassing diminished thrombopoietin (TPO) synthesis secondary to hepatic dysfunction, hypersplenism-induced splenic sequestration, and immune-mediated platelet destruction.[10,11] As TPO is primarily produced by hepatocytes and sinusoidal endothelial cells, progressive liver fibrosis results in a marked decline in circulating TPO levels, further aggravating thrombocytopenia.[12,13]

Anemia is another frequent hematological complication observed in DCLD, attributable to chronic gastrointestinal blood loss, nutritional deficiencies (iron, folate, vitamin B12), bone marrow suppression, and hemolysis. Morphologically, normocytic normochromic anemia predominates in cirrhotic patients.[4016] Leukopenia is also prevalent, primarily due to portal hypertension-induced hypersplenism and bone marrow suppression.

In addition, coagulopathy in DCLD results from impaired hepatic synthesis of clotting factors, leading to prolongation of prothrombin time (PT) and elevation of the international normalized ratio (INR). These parameters, while traditionally interpreted as indicators of bleeding risk, do not reliably predict hemorrhagic complications in cirrhotic patients. Emerging evidence suggests that the risk of bleeding is modulated by a complex interplay of factors, including infection, renal dysfunction, systemic hypotension, and endothelial abnormalities. Furthermore, despite coagulopathy, patients with cirrhosis remain susceptible to thrombotic events, particularly in advanced disease stages such as Child-Pugh Class C.[16]

Hematological abnormalities in DCLD not only reflect the severity of hepatic dysfunction but also impact clinical outcomes, including susceptibility to infections, bleeding, and overall survival. Timely recognition and appropriate management of these abnormalities are essential for improving patient outcomes and guiding therapeutic interventions.

The present study was undertaken to assess the prevalence, distribution patterns, and underlying mechanisms of haematological abnormalities in patients with decompensated chronic liver disease. The findings aim to enhance understanding of the hematological spectrum in DCLD and provide evidence to support targeted clinical management in this high-risk population.

Materials and Methods

This study was conducted in the Department of General Medicine The primary objective was to evaluate the prevalence and patterns of haematological abnormalities among patients diagnosed with decompensated chronic liver disease (DCLD) admitted to the inpatient wards of the department.

Study Population and Sampling

The study population included all consecutive patients diagnosed with DCLD and admitted during the study period. A total of 50 patients meeting the predefined eligibility criteria were enrolled using the census sampling method. Inclusion criteria encompassed adult patients aged ≥18 years with a confirmed diagnosis of decompensated chronic liver disease of more than six months' duration. Cases with underlying etiologies such as alcoholic cirrhosis, post-necrotic cirrhosis, and metabolic liver diseases were included.Patients were excluded if they had hepatocellular carcinoma, any known malignancy, acute liver failure, primary coagulation disorders, or known hemostatic abnormalities. Further exclusion criteria included anemia attributable to non-hepatic causes and the presence of chronic end-stage medical conditions, such as chronic kidney disease (CKD), chronic obstructive pulmonary disease (COPD), coronary artery disease (CAD), or congestive cardiac failure.

Data Collection

Detailed patient information was recorded using a structured case proforma. Demographic data including age, sex, occupation, and residential details were documented.

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A comprehensive clinical history was obtained with particular focus on presenting complaints, symptom duration, history of decompensating events, prior medical or surgical interventions, comorbid conditions, and ongoing medications. A thorough general and systemic examination was carried out, with documentation of vital signs and relevant physical findings.

Investigations

All patients underwent laboratory evaluation after obtaining informed consent. The investigations included complete blood count (CBC), peripheral blood smear examination, serum iron profile, serum folate levels, liver function tests (LFTs), and coagulation parameters including prothrombin time (PT) and international normalized ratio (INR). These investigations were aimed at identifying hematological abnormalities and assessing hepatic synthetic function and coagulation status.

Statistical Analysis

Data were compiled and analyzed using Statistical Package for the Social Sciences (SPSS) software, version 21.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize baseline characteristics. Continuous variables were expressed as mean ± standard deviation (SD) and analyzed using the Student's t-test. Categorical variables were presented as frequencies and percentages, and associations between variables were assessed using the Chi-square test. A p-value < 0.05 was considered statistically significant.

Results

A total of 50 patients diagnosed with decompensated chronic liver disease (DCLD) were included in the study. Among these, 42 (84%) were male and 8 (16%) were female, resulting in a male-to-female ratio of 5.3:1. The most common etiology identified was alcohol-related liver disease, accounting for 33 (66%) cases. Non-alcoholic steatohepatitis (NASH) was observed in 11 (22%) patients, followed by hepatitis B virus (HBV) infection in 3 (6%) cases and hepatitis C virus (HCV) infection in 2 (4%) cases. Less common causes included Wilson's disease and autoimmune hepatitis, each reported in 1 (2%) patient. These findings reflect the predominance of alcohol as the principal etiological factor in DCLD in the study cohort.

Anemia Profile

Anemia was present in 47 out of 50 patients (94%). Based on hemoglobin levels, anemia was categorized into mild (Hb 10–11.9 g/dL), moderate (Hb 7–9.9 g/dL), and severe (Hb <7 g/dL) forms. Of the anemic patients, 11 (23.4%) had mild anemia, 23 (48.9%) had moderate anemia, and 13 (27.7%) were found to have severe anemia (Table 1). Morphologically, normocytic anemia was the most frequent subtype, observed in 27 (57.4%) patients, followed by microcytic anemia in 14 (29.8%) and macrocytic anemia in 6 (12.8%) patients.

Table 1.Severity of Anemia in DCLD Patients

Anemia Severity	Number of Patients (n)	Percentage (%)
Mild (10–11.9 g/dL)	11	23.4
Moderate (7–9.9 g/dL)	23	48.9
Severe (<7 g/dL)	13	27.7

Thrombocytopenia and Coagulation Abnormalities

Thrombocytopenia (defined as platelet count <150,000/µL) was observed in 36 (72%) patients, while 14 (28%) had normal platelet counts. Based on severity, 7 (14%) patients had platelet counts below 50,000/µL, 18 (36%) had counts between 50,000 and 100,000/µL, and 11 (22%) had counts between 100,000 and 150,000/µL.In terms of coagulation profile, prolonged prothrombin time (PT) was noted in 34 (68%) patients, and elevated international normalized ratio (INR) values were observed in 37 (74%) patients, indicating significant coagulation dysfunction among individuals with DCLD.

Association with Gastrointestinal Bleeding

A significant association was found between thrombocytopenia and gastrointestinal (GI) bleeding. Among patients with platelet counts <150,000/µL, 20 (55.6%) experienced GI bleeding, compared to 5 (35.7%) in the group with normal platelet counts. This association was statistically significant (p = 0.047) (Table 2).

Table 2.Association Between Platelet Count and GI Bleeding

Platelet Count	GI Bleeding Present (n)	GI Bleeding Absent (n)	p-value
<150,000/µL	20	16	0.047
150,000–450,000/µL	5	9	—

Additionally, GI bleeding was significantly associated with deranged coagulation parameters. Patients with GI bleeding had a mean PT of 18.3 ± 4.9 seconds, compared to 11.1 ± 2.0 seconds in those without bleeding (p = 0.009). Similarly, the mean INR in patients with GI bleeding was 2.49 ± 0.28, while it was 1.68 ± 0.12 in those without bleeding (p = 0.012), confirming a strong correlation between bleeding risk and impaired coagulation (Table 3).

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Table 3.Relationship Between GI Bleeding and Coagulation Parameters

Coagulation Parameter	GI Bleeding Present (Mean ± SD)	GI Bleeding Absent (Mean ± SD)	p-value
Prothrombin Time (sec)	18.3 ± 4.9	11.1 ± 2.0	0.009
INR	2.49 ± 0.28	1.68 ± 0.12	0.012

Discussion

Decompensated chronic liver disease (DCLD) is frequently associated with hematological abnormalities such as anemia, thrombocytopenia, and coagulation disturbances. These derangements contribute significantly to the clinical manifestations and complications, including gastrointestinal (GI) bleeding. The present study evaluated the prevalence, severity, and morphological patterns of these hematological abnormalities in 50 patients with DCLD and compared the findings with previously published literature.In our study, anemia was present in 47 out of 50 patients (94%), with a mean hemoglobin level of 8.2 ± 1.8 g/dL. Among them, 23.4% had mild anemia, 48.9% had moderate anemia, and 27.7% had severe anemia. These findings are consistent with those of Joeimon J et al. [16], who reported an anemia prevalence of 90%, with 14% of patients having hemoglobin levels below 6 g/dL. Similarly, Frijo J et al. [17] observed anemia in 86.8% of their study population, with normocytic normochromic anemia being the most prevalent morphological type (39.4%).Morphologically, normocytic anemia was the most common in our study (57.4%), followed by microcytic (29.8%) and macrocytic (12.8%) types. This distribution is comparable to the findings of G. Anbazhagan et al. [18], who also found normocytic anemia to be the most frequent pattern in CLD. However, E. Krithiga et al. [19] reported macrocytic anemia in 40% of patients, likely reflecting a higher incidence of nutritional deficiencies such as vitamin B12 and folate in their cohort. Kumar EH et al. [20] similarly noted moderate anemia in 41% of DCLD patients, predominantly of the normochromic type, reinforcing the high burden of normocytic anemia in chronic liver disease.

Thrombocytopenia was present in 36 patients (72%) in our study, with 14% having platelet counts below 50,000/µL. These findings are aligned with the study by Shetty V et al. [21], who reported a thrombocytopenia prevalence of 56.6% in cirrhotic patients. The pathogenesis of thrombocytopenia in DCLD is multifactorial, involving splenic sequestration secondary to portal hypertension, decreased thrombopoietin synthesis, and bone marrow suppression. Qamar AA et al. [9] also documented a high prevalence of thrombocytopenia in CLD patients, further supporting our observations.

Coagulation dysfunction was another prominent abnormality observed in our study. Elevated prothrombin time (PT) was noted in 34 patients (68%), and elevated INR in 37 (74%). These findings are consistent with those of Solomon RT et al. [23], who reported prolonged PT in 72% of patients with liver disease. Bhatia G et al. (2016) [24] similarly observed prolonged PT in 62% of their cases. Sharma A et al. [25] and Patil Amita Yatish et al. [26] reported prolonged PT and activated partial thromboplastin time (aPTT) in 63% and 55% of patients, respectively. In our study, 65.8% of patients with prolonged PT had at least one episode of hematemesis, highlighting the clinical impact of coagulation abnormalities.

A statistically significant association was observed between thrombocytopenia and GI bleeding in our study. Among patients with low platelet counts (<150,000/µL), 60% had GI bleeding, compared to 40% in those with normal counts (p = 0.047). These results are in agreement with Nagarajaiah RB et al. [28], who identified GI bleeding as the most common clinical presentation in patients with DCLD. Coagulation abnormalities were also significantly associated with bleeding risk. Patients with GI bleeding had a mean PT of 18.3 ± 4.9 seconds and a mean INR of 2.49 ± 0.28, both significantly higher than those without bleeding (PT: 11.1 ± 2.0 seconds; INR: 1.68 ± 0.12). These differences were statistically significant (p = 0.009 for PT; p = 0.012 for INR), indicating a strong correlation between coagulopathy and bleeding episodes. Selvamani S et al. [28] also documented prolonged PT in 46% of their patients, consistent with our findings.In terms of etiology, alcohol-related liver disease was the most common cause in our study (66%), followed by NASH (22%). Hepatitis B virus (HBV) and hepatitis C virus (HCV) infections were responsible for 6% and 4% of cases, respectively. These findings are comparable to those reported by Bhattacharyya M et al. [29], who observed HBV in 8.9% and HCV in 3.2% of CLD patients. Similarly, Ahmed S et al. [27] found that 11% of their DCLD cases were due to HBV and HCV infections. These results underscore the continued relevance of viral hepatitis as a significant but less common cause of DCLD in the current era, likely due to improved vaccination and treatment efforts.

The most common presenting complaints in our study were jaundice, abdominal distension, and bleeding manifestations. This clinical spectrum aligns with the

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findings of Nagarajaiah RB et al. [27], who also reported GI bleeding as the most frequent presentation in patients with decompensated cirrhosis. Gastrointestinal hemorrhage remains a serious and often life-threatening complication of DCLD, largely attributed to coagulopathy and complications of portal hypertension.

Conclusion

Overall, our study corroborates the findings of several previously published studies, establishing anemia, thrombocytopenia, and coagulopathy as common and clinically significant hematological abnormalities in DCLD. These parameters not only reflect the severity of hepatic dysfunction but also have important prognostic implications, particularly in predicting the risk of GI bleeding. Early detection and management of these abnormalities are essential to improving clinical outcomes in this vulnerable patient population.

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This study was limited by its single-centre design and relatively small sample size, which may affect the generalizability of the findings. The cross-sectional nature of the study did not allow for evaluation of long-term outcomes. Additionally, detailed evaluation of nutritional deficiencies and bone marrow pathology, which could further explain the causes of anemia, was not performed.

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