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Original Article

Haematological Abnormalities in Chronic Liver Disease: A Clinico-Pathological Study

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ABSTRACT

Background: Chronic liver disease (CLD) is frequently accompanied by a wide spectrum of haematological abnormalities arising from portal hypertension and hypersplenism, impaired hepatic synthetic function, nutritional deficiencies and bone-marrow suppression. This study was undertaken to characterise the pattern of these abnormalities and to correlate them with the clinical severity of liver disease.

Methods: This hospital-based observational clinico-pathological study evaluated 85 patients with established CLD over a 10-month period (January–October 2025) in the Department of Medicine, IQ City Medical College and Hospital, Durgapur. Detailed clinical assessment, complete haemogram with peripheral smear, coagulation profile and liver function tests were performed for each patient. Disease severity was graded using the Child-Pugh classification. Continuous variables were compared across Child-Pugh classes using one-way ANOVA or the Kruskal–Wallis test; categorical associations were assessed with the chi-square or Fisher exact test, and correlations with Spearman's rank coefficient. A p-value <0.05 was considered significant.

Results: The mean age was 49.2 ± 11.1 years with a slight male preponderance (M:F = 1.12:1). Anaemia was the commonest abnormality (88.2%), followed by thrombocytopenia (82.4%); leukopenia and pancytopenia each occurred in 21.2%. Moderate anaemia predominated (52.9%) and the peripheral smear was most often normocytic normochromic (56.5%) followed by macrocytic (36.5%). Haemoglobin, platelet count and total leucocyte count fell progressively from Child-Pugh class A to C, while INR and bilirubin rose (all $p<0.001$). Severity of anaemia increased significantly with Child-Pugh class ($\chi^2=56.9$, $p<0.001$). Thrombocytopenia was strongly associated with splenomegaly (OR 6.0, $p=0.004$), and pancytopenia with Child-Pugh class C (OR 33.2, $p<0.001$). Haemoglobin and platelet count correlated positively with serum albumin and negatively with bilirubin and INR (all $p<0.01$).

Conclusion: Haematological abnormalities are almost universal in CLD and worsen in parallel with hepatic decompensation. The haemogram, peripheral smear and coagulation profile together provide an inexpensive bedside index of disease severity and merit routine clinico-pathological correlation in the management of CLD.

Keywords: *Chronic liver disease; Anaemia; Thrombocytopenia; Pancytopenia; Child-Pugh classification; Peripheral blood smear.*

INTRODUCTION

Chronic liver disease (CLD) represents a continuum of progressive hepatic injury, fibrosis and ultimately cirrhosis, and constitutes a major cause of morbidity and mortality worldwide. Common aetiologies include alcohol-related liver disease,

chronic viral hepatitis (hepatitis B and C), non-alcoholic fatty liver disease (NAFLD/NASH), autoimmune hepatitis and metabolic disorders such as Wilson disease, while a substantial proportion remain cryptogenic.[1]

Haematological abnormalities are among the most frequent systemic manifestations of CLD and may involve any of the three cell lines. Anaemia in CLD is multifactorial, resulting from gastrointestinal blood loss, hypersplenism, haemodilution, nutritional deficiencies of folate and vitamin B12, and the direct toxic effects of alcohol on the marrow. Thrombocytopenia is largely attributable to portal hypertension with consequent splenic sequestration (hypersplenism) and to reduced hepatic synthesis of thrombopoietin. Leukopenia and, in advanced disease, pancytopenia reflect a combination of hypersplenism and bone-marrow suppression.[2]

In addition to cytopenias, impaired hepatic synthesis of coagulation factors produces prolongation of the prothrombin time and a raised international normalised ratio (INR), contributing to the bleeding tendency that characterises decompensated disease. These abnormalities are not merely incidental laboratory findings; their severity tends to mirror the degree of hepatic dysfunction and therefore carries prognostic significance.[3,4]

Despite their clinical importance, the haematological profile of CLD is often under-evaluated in routine practice. A systematic clinico-pathological assessment that links the haemogram, peripheral smear and coagulation indices to objective severity grading (Child-Pugh classification) can provide a readily available, inexpensive marker of disease progression. The present study was therefore designed to describe the spectrum of haematological abnormalities in patients with CLD and to correlate them with clinical and biochemical indices of disease severity.

Aims and Objectives

To study the spectrum and frequency of haematological abnormalities in patients with chronic liver disease.

1. To determine the prevalence and pattern of anaemia, thrombocytopenia, leukopenia and pancytopenia in CLD.
2. To characterise red-cell morphology on peripheral blood smear and classify the type of anaemia.
3. To correlate haematological parameters with Child-Pugh severity and with biochemical markers of hepatic function.

MATERIALS AND METHODS

Study design, setting and period

This was a hospital-based, observational, cross-sectional clinico-pathological study conducted in the Department of Medicine, IQ City Medical College and Hospital, Durgapur, West Bengal, over a period of 10 months from January 2025 to October 2025.

Study population

A total of 85 consecutive patients with a diagnosis of chronic liver disease, established on the basis of clinical, biochemical, ultrasonographic and/or endoscopic evidence, were enrolled after informed consent.

Inclusion criteria: adult patients (≥18 years) of either sex with established CLD of any aetiology.

Exclusion criteria: patients with a known primary haematological disorder, malignancy, chronic kidney disease, recent blood transfusion, or those on drugs known to cause cytopenias, were excluded to limit confounding.

Data collection and laboratory methods

Each patient underwent detailed history-taking and clinical examination, with documentation of pallor, icterus, pedal oedema, bleeding tendency, splenomegaly and ascites. Venous blood samples were analysed for a complete haemogram (haemoglobin, total and differential leucocyte counts, platelet count and red-cell indices — MCV, MCH, MCHC and RDW) on an automated analyser, with a manual peripheral blood smear examination in every case. The coagulation profile (prothrombin time, INR and activated partial thromboplastin time) and liver function tests (total bilirubin, AST, ALT, ALP, serum albumin and total protein) were also recorded. Disease severity was graded using the Child-Pugh classification (A, B and C).

Operational definitions. Anaemia was defined and graded by haemoglobin concentration; thrombocytopenia as a platelet count <1.5 lakh/mm³; leukopenia as a total leucocyte count <4000 cells/mm³; and pancytopenia as the simultaneous presence of anaemia, thrombocytopenia and leukopenia.

Statistical analysis

Data were compiled and analysed using standard statistical software. Continuous variables are expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Comparison of continuous parameters across the three Child-Pugh classes was performed using one-way analysis of variance (ANOVA), with the Kruskal–Wallis test applied to skewed variables. Associations between categorical variables were tested using the chi-square test, with the Fisher exact test where expected cell counts were small. Correlations between haematological and biochemical parameters

were assessed using Spearman's rank correlation coefficient. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Demographic and clinical profile

Of the 85 patients studied, the mean age was 49.2 ± 11.1 years (range 20–68 years), with the majority in the fourth to sixth decades of life. There was a slight male preponderance, with 45 males (52.9%) and 40 females (47.1%), giving a male-to-female ratio of 1.12:1. The median duration of illness was 31 months (interquartile range 20–47).

The commonest presenting clinical findings were pallor (70.6%) and icterus (64.7%), followed by splenomegaly and ascites (61.2% each), pedal oedema (41.2%) and a clinical bleeding tendency (20.0%). The distribution of patients by Child-Pugh class was A in 17 (20.0%), B in 39 (45.9%) and C in 29 (34.1%), indicating that two-thirds of the cohort had decompensated disease.

Table 1. Baseline demographic and clinical characteristics (n = 85)

Characteristic	Value	—
Mean age (years)	49.2 ± 11.1	20–68 (range)
Male / Female, n	45 / 40	1.12 : 1
Median duration of illness (months)	31	20–47 (IQR)
Pallor, n (%)	60 (70.6)	—
Icterus, n (%)	55 (64.7)	—
Splenomegaly, n (%)	52 (61.2)	—
Ascites, n (%)	52 (61.2)	—
Pedal oedema, n (%)	35 (41.2)	—
Bleeding tendency, n (%)	17 (20.0)	—
Child-Pugh A / B / C, n	17 / 39 / 29	20.0 / 45.9 / 34.1 %

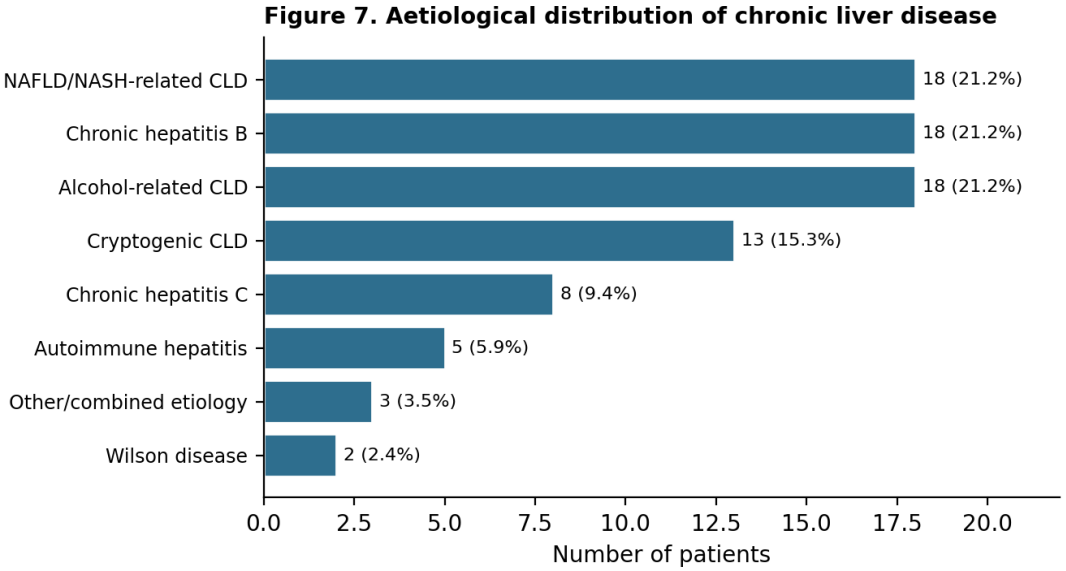

Figure 7. Aetiological distribution of chronic liver disease in the study cohort.

The aetiology of CLD was led equally by alcohol-related disease, chronic hepatitis B and NAFLD/NASH (18 patients, 21.2% each), followed by cryptogenic CLD (15.3%), chronic hepatitis C (9.4%), autoimmune hepatitis (5.9%), and a small number of cases of Wilson disease and combined/other aetiologies.

Spectrum of haematological abnormalities

Haematological abnormalities were near-universal in this cohort. Anaemia was the single most frequent finding, present in 75 patients (88.2%), followed closely by thrombocytopenia in 70 patients (82.4%). Leukopenia and pancytopenia each affected 18 patients (21.2%). Only 5 patients (5.9%) had no major haematological abnormality.

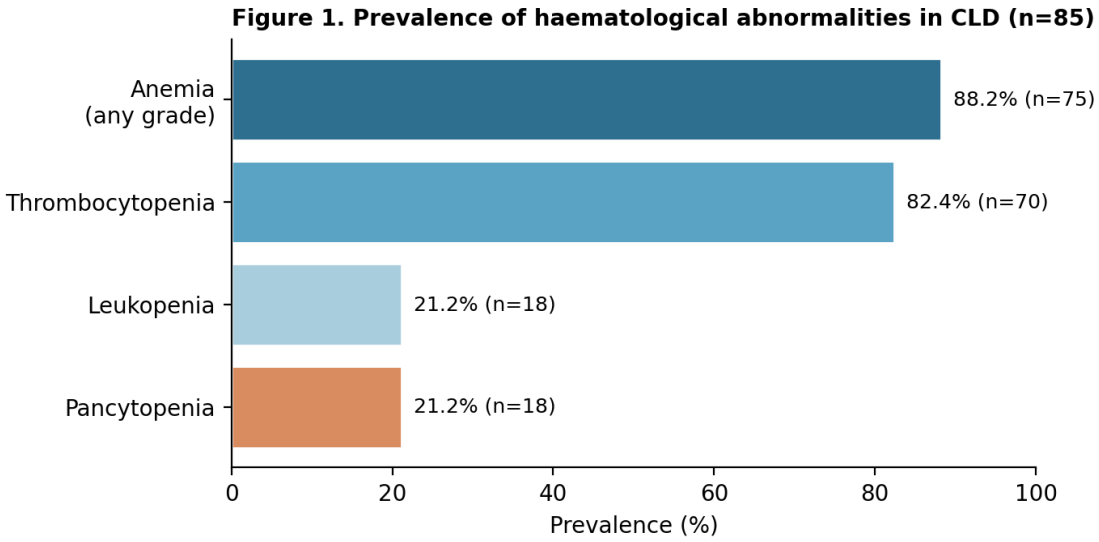

Figure 1. Prevalence of the principal haematological abnormalities in chronic liver disease.

When the abnormalities were considered in combination, the commonest composite pattern was anaemia with thrombocytopenia (55.3%), followed by pancytopenia (21.2%); isolated anaemia (11.8%) and isolated thrombocytopenia (5.9%) were less frequent.

Table 2. Pattern of haematological abnormalities (n = 85)

Abnormality / Pattern	**n**	**%**
Anaemia (any grade)	75	88.2
Thrombocytopenia (<1.5 lakh/mm³)	70	82.4
Leukopenia (<4000/mm³)	18	21.2
Pancytopenia	18	21.2
Composite patterns		
Anaemia + thrombocytopenia	47	55.3
Pancytopenia	18	21.2
Isolated anaemia	10	11.8
Isolated thrombocytopenia	5	5.9
No major abnormality	5	5.9

Severity and morphology of anaemia

Among the 85 patients, moderate anaemia was the predominant grade (45 patients, 52.9%), followed by mild anaemia (23, 27.1%) and severe anaemia (7, 8.2%); 10 patients (11.8%) were not anaemic. The mean haemoglobin for the cohort was 10.3 ± 1.8 g/dL.

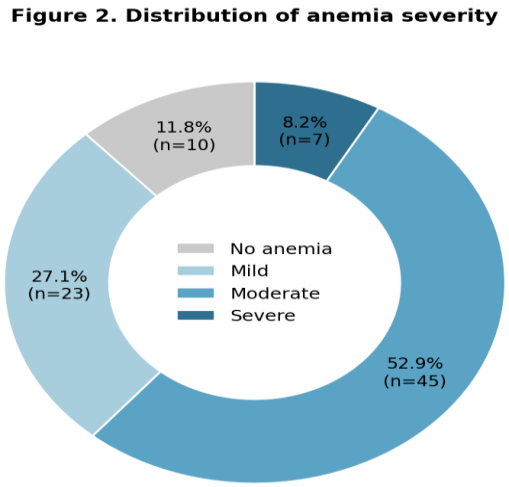

Figure 2. Distribution of anaemia severity in the study population.

Peripheral blood smear examination showed a normocytic normochromic picture in the majority (48 patients, 56.5%), consistent with anaemia of chronic disease and haemodilution, while a macrocytic picture — often with target cells and reflecting underlying liver disease and folate deficiency — was seen in 31 patients (36.5%). A microcytic hypochromic pattern, suggesting iron deficiency from chronic blood loss, was least common (6 patients, 7.1%).

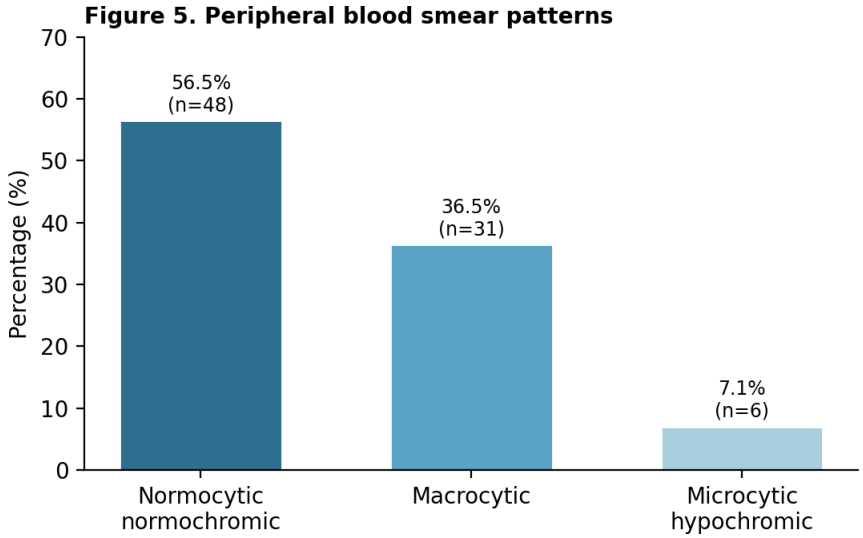

Figure 5. Distribution of peripheral blood smear morphological patterns.

Haematological parameters and disease severity

A consistent and statistically significant deterioration of all three cell lines was observed with advancing Child-Pugh class. Mean haemoglobin fell from 12.3 g/dL in class A to 10.6 g/dL in class B and 8.8 g/dL in class C; the platelet count declined from 1.66 to 0.88 and 0.55 lakh/mm³; and the total leucocyte count fell from 7384 to 5808 and 3849 cells/mm³ respectively. Conversely, INR and total bilirubin rose progressively while serum albumin declined. All these trends were highly significant ($p<0.001$).

Figure 3. Haematological parameters across Child-Pugh classes (p<0.001 for each)

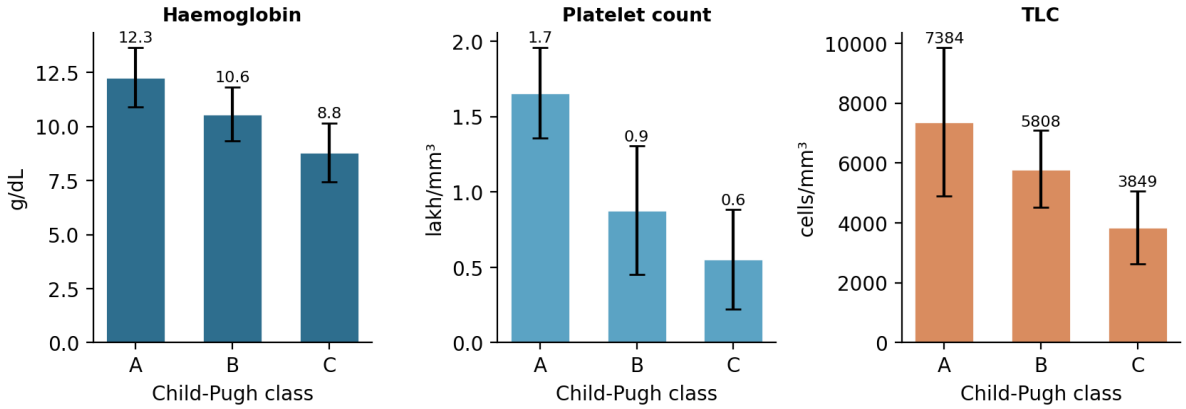

Figure 3. Mean haemoglobin, platelet count and total leucocyte count across Child-Pugh classes (error bars = SD).

Table 3. Haematological and biochemical parameters by Child-Pugh class (mean ± SD)

Parameter	Class A (n=17)	Class B (n=39)	Class C (n=29)	p-value
Haemoglobin (g/dL)	12.3 ± 1.4	10.6 ± 1.2	8.8 ± 1.4	<0.001
Platelet (lakh/mm³)	1.66 ± 0.30	0.88 ± 0.43	0.55 ± 0.33	<0.001
TLC (cells/mm³)	7384 ± 2486	5808 ± 1281	3849 ± 1225	<0.001
INR	1.17 ± 0.09	1.58 ± 0.24	2.15 ± 0.42	<0.001
Total bilirubin (mg/dL)	1.3 ± 0.6	3.0 ± 1.7	6.3 ± 3.2	<0.001
Serum albumin (g/dL)	3.41 ± 0.30	2.95 ± 0.34	2.38 ± 0.32	<0.001
MCV (fL)	92.7 ± 7.4	91.1 ± 9.8	92.8 ± 9.8	0.70 (NS)

NS = not significant. Comparisons by one-way ANOVA / Kruskal–Wallis test.

The severity of anaemia was also strongly related to Child-Pugh class. Whereas the majority of class A patients had no or only mild anaemia, moderate and severe anaemia dominated in classes B and C; all 7 cases of severe anaemia occurred in class C. This association was highly significant ($\chi^2 = 56.9$, $p<0.001$).

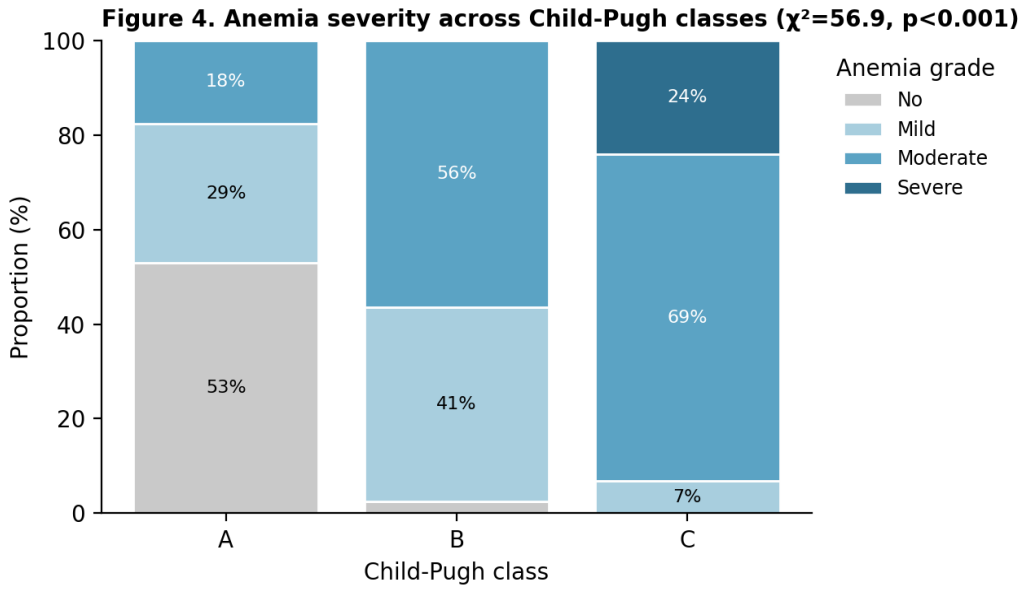

Figure 4. Distribution of anaemia severity across Child-Pugh classes.

Determinants of thrombocytopenia and pancytopenia

Thrombocytopenia was significantly associated with the presence of splenomegaly: 48 of 52 patients (92.3%) with splenomegaly were thrombocytopenic, compared with 22 of 33 (66.7%) without (OR 6.0, p=0.004), supporting hypersplenism secondary to portal hypertension as a principal mechanism. Thrombocytopenia also paralleled Child-Pugh severity (χ^2=50.6, p<0.001).

Pancytopenia clustered strongly in advanced disease. Sixteen of 29 class C patients (55.2%) were pancytopenic versus only 2 of 56 in classes A and B combined, yielding a markedly elevated odds ratio (OR 33.2, p<0.001). Pancytopenia was likewise more frequent in patients with splenomegaly (OR 4.05, p=0.033).

Table 4. Associations of thrombocytopenia and pancytopenia

Association	Exposed	Comparator	p-value (OR)
Thrombocytopenia × splenomegaly	48/52 (92.3%)	22/33 (66.7%)	0.004 (OR 6.0)
Thrombocytopenia × Child-Pugh	—	—	<0.001
Pancytopenia × Child-Pugh C	16/29 (55.2%)	2/56 (3.6%)	<0.001 (OR 33.2)
Pancytopenia × splenomegaly	15/52 (28.8%)	3/33 (9.1%)	0.033 (OR 4.05)

3.6 Clinico-pathological correlation

Haematological indices correlated significantly with markers of hepatic synthetic function. Haemoglobin showed a moderate positive correlation with serum albumin ($\rho = 0.55$, $p<0.001$) and negative correlations with total bilirubin ($\rho = -0.54$) and INR ($\rho = -0.51$). Platelet count correlated positively with albumin ($\rho = 0.44$) and negatively with INR ($\rho = -0.55$) and bilirubin ($\rho = -0.35$), and the total leucocyte count correlated positively with albumin ($\rho = 0.49$). These relationships indicate that worsening hepatic synthetic capacity is accompanied by progressive depression of all haematological lines.

Figure 6. Correlation of haematological parameters with hepatic synthetic function

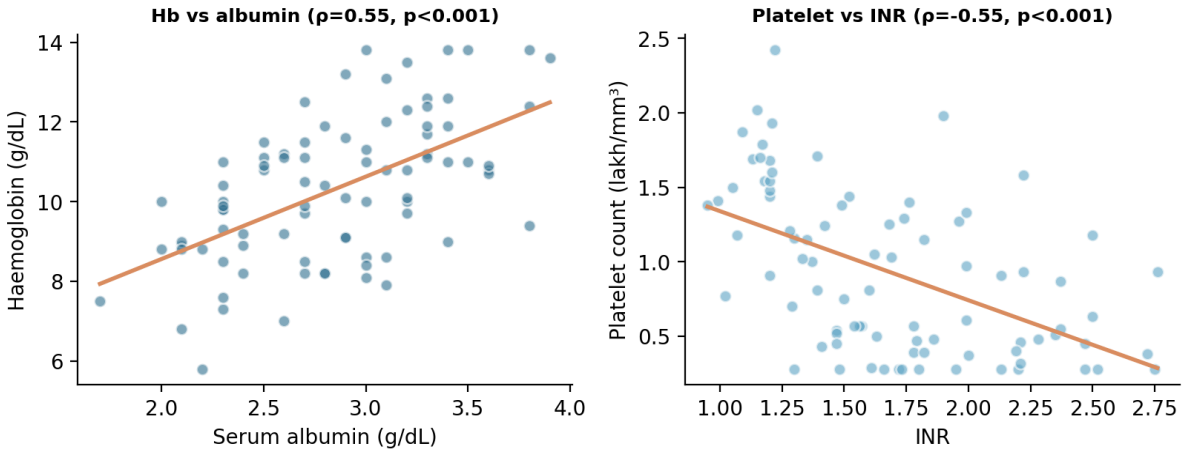

Figure 6. Correlation of haemoglobin with serum albumin and of platelet count with INR.

Table 5. Spearman correlations of haematological with biochemical parameters

Variable pair	ρ (rho)	p-value
Haemoglobin vs albumin	+0.55	<0.001
Haemoglobin vs bilirubin	−0.54	<0.001
Haemoglobin vs INR	−0.51	<0.001
Platelet vs INR	−0.55	<0.001
Platelet vs albumin	+0.44	<0.001
Platelet vs bilirubin	−0.35	0.001
TLC vs albumin	+0.49	<0.001

DISCUSSION

This clinico-pathological study of 85 patients confirms that haematological abnormalities are an almost invariable accompaniment of chronic liver disease and that their severity tracks closely with hepatic decompensation. Anaemia (88.2%) and thrombocytopenia (82.4%) were the dominant findings, with leukopenia and pancytopenia each present in roughly one-fifth of patients — a pattern broadly consistent with the published literature on cytopenias in cirrhosis.[5]

The predominance of moderate, normocytic normochromic anaemia in our cohort accords with the multifactorial origin of anaemia in CLD, in which anaemia of chronic disease, haemodilution from fluid retention, and hypersplenism act together.[6] The substantial proportion of macrocytic smears (36.5%) reflects the contribution of liver disease itself, alcohol and folate deficiency, while the small microcytic fraction points to superimposed iron deficiency from gastrointestinal blood loss. The peripheral smear therefore retains considerable diagnostic value in identifying the dominant mechanism of anaemia in an individual patient.

The strong, graded fall in haemoglobin, platelet count and leucocyte count from Child-Pugh A to C — together with the rising INR and bilirubin — provides clear evidence that the haemogram mirrors the severity of hepatic dysfunction. The highly significant association between thrombocytopenia and splenomegaly (OR 6.0) is in keeping with hypersplenism due to portal hypertension as the principal mechanism of low platelet counts, reinforced by reduced hepatic thrombopoietin production.[7] The dramatic concentration of pancytopenia in Child-Pugh class C (OR 33.2) similarly identifies advanced portal hypertension and marrow suppression as the substrate of trilineage cytopenia.[8-10]

The significant correlations of haemoglobin and platelet count with serum albumin, bilirubin and INR underscore the clinico-pathological unity of the syndrome: as synthetic liver function declines, so do the formed elements of blood. From a practical standpoint, these findings suggest that a simple, inexpensive haemogram and coagulation profile can serve as a surrogate index of disease severity, complementing formal Child-Pugh scoring, particularly in resource-limited settings.
The clinical implications are twofold. First, recognition of the type and severity of cytopenia can guide targeted evaluation — for example, iron studies and endoscopy for microcytic anaemia, or assessment of portal hypertension where thrombocytopenia and splenomegaly coexist. Second, the bleeding risk implied by progressive thrombocytopenia and coagulopathy warrants caution before invasive procedures in decompensated patients.

Limitations

This was a single-centre, cross-sectional study with a modest sample size, which limits the generalisability of the findings and precludes inference about causation or temporal change. Bone-marrow examination and specific haematinic assays

(serum ferritin, vitamin B12, folate) were not uniformly available, so the precise contribution of each mechanism to anaemia could not be quantified. Larger, multicentre, prospective studies with longitudinal follow-up would help confirm these associations and define their prognostic weight.

CONCLUSION

Haematological abnormalities are near-universal in chronic liver disease, with anaemia and thrombocytopenia being the most frequent. The severity of cytopenias increases significantly with worsening Child-Pugh class, thrombocytopenia is closely linked to splenomegaly, and pancytopenia is a marker of advanced decompensation. The strong correlation between haematological indices and hepatic synthetic function establishes the haemogram, peripheral smear and coagulation profile as a simple, inexpensive and clinically informative index of disease severity. Routine clinico-pathological correlation of these parameters should therefore form an integral part of the assessment and monitoring of patients with chronic liver disease.

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