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CLINICO-PATHOLOGICAL CORRELATION OF HEMATOLOGICAL AND BIOCHEMICAL PROFILES IN PATIENTS WITH CHRONIC LIVER DISEASE: A CROSS-SECTIONAL ANALYSIS

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ABSTRACT

Background: Chronic liver disease (CLD) is characterized by progressive hepatic injury culminating in cirrhosis and hepatic decompensation. Hematological and biochemical parameters are vital in evaluating the extent of liver dysfunction and predicting clinical outcomes.

Materials and Methods: This cross-sectional observational study was conducted in the Department of General Medicine, RVM Institute of medical sciences and research Center, over one year from March 2024 to February 2025. A total of 150 adult patients diagnosed with CLD were enrolled. Detailed clinical histories were taken, and hematological parameters (hemoglobin, platelet count, TLC) and biochemical parameters (AST, ALT, bilirubin, albumin, INR) were analyzed. Disease severity was graded using the Child-Pugh classification. Statistical correlations between laboratory parameters and disease class were evaluated using SPSS software.

Results: The study cohort was male predominant (62.7%) and aged between 41–60 years. Alcoholic liver disease (41.3%) and NAFLD (19.3%) were the predominant etiologies. Anemia and thrombocytopenia were the most common hematological manifestations. Biochemical derangements included elevated AST, ALT, bilirubin, and INR, and reduced albumin levels. Platelet count, serum albumin, bilirubin, and INR significantly correlated with Child-Pugh class.

Conclusion: Routine hematological and biochemical investigations provide essential insights into CLD severity and prognosis. Their integration into clinical practice is crucial for early risk stratification and guiding therapeutic strategies.

Keywords: Chronic liver disease, Biochemical parameters, Hematology, Child-Pugh classification, Thrombocytopenia.

INTRODUCTION

Chronic liver disease (CLD) includes constellation of progressive hepatic disorders characterized by gradual destruction of liver architecture due to sustained inflammation and fibrosis. Over time, such persistent hepatic insult leads to cirrhosis and hepatic decompensation, culminating in complications like portal hypertension, ascites, coagulopathy, and hepatic encephalopathy. Globally, chronic liver disease constitutes a significant public health burden, being the 11th leading cause of mortality and 15th leading cause of disability-adjusted life years (DALYs) worldwide.[1]

Etiological factors contributing to CLD include chronic alcohol abuse, chronic viral hepatitis (notably hepatitis B and C), non-alcoholic fatty liver disease (NAFLD), autoimmune hepatitis, cholestatic liver diseases, and metabolic liver disorders.[2] In developing countries like India, viral hepatitis and alcohol-related liver disease dominate as leading causes, whereas NAFLD is increasingly recognized

in association with increasing rates of metabolic syndrome.[3]

Liver functions are intricately linked to hematological and biochemical homeostasis. The liver synthesizes plasma proteins, including albumin and various clotting factors, as well as the metabolism of bilirubin, cholesterol, glucose, and ammonia. Therefore, liver dysfunction invariably affects these parameters, often serving as clinical indicators of disease severity and prognosis.[4] Elevated transaminase levels generally reflect hepatocellular injury, while hypoalbuminemia and prolonged prothrombin time signify impaired synthetic capacity of the liver.[5]

Hematological abnormalities are equally common in CLD and are frequently multifactorial in origin. Anemia, leukopenia, and thrombocytopenia may arise due to hypersplenism secondary to portal hypertension, nutritional deficiencies, bone marrow suppression, and chronic gastrointestinal blood loss.[6] Platelet count, in particular, serves as a surrogate marker for assessing portal hypertension and splenomegaly.[7]

Several studies have emphasized the importance of assessing hematological and biochemical parameters in CLD for staging disease severity and predicting complications. For instance, serum albumin levels and INR are integral components of the Child-Turcotte-Pugh (CTP) score, while bilirubin, creatinine, and INR are used to calculate Model for End-Stage Liver Disease (MELD) score.[8] Hence, these biomarkers do not merely aid in diagnosis but also guide therapeutic decision-making and prognostication.

Moreover, specific patterns of derangements in these parameters can also help differentiate between underlying etiologies. For instance, a predominant elevation in ALT over AST may be suggestive of NAFLD in early stages, whereas AST predominance is typical in alcoholic liver disease.[9] Similarly, elevated ALP and GGT levels are more common in cholestatic and infiltrative liver disorders.[10]

Despite the widespread availability of these tests, regional and institutional variations persist in terms of disease presentation, etiological profiles, and laboratory abnormalities. Hence, periodic evaluation of hematological and biochemical patterns in local populations with CLD is essential to improve diagnostic accuracy and optimize management strategies. This study was conducted to assess the derangements in hematological and biochemical profiles in CLD and assess their clinical relevance in disease monitoring and management was the aim of this study.

MATERIALS AND METHODS

This cross-sectional observational study was done in the General Medicine department, RVM Institute of medical sciences and research Center, over one year, from March 2024 to February 2025. 150 adults with CLD were included in the study based on predefined inclusion and exclusion criteria. Patients were included into the study with their informed written consent, and approval from the institutional ethical board was obtained prior to the initiation of this study.

Diagnosed CLD patients (irrespective of etiology) of >6 months aged >18 years, who were willing to participate were included. Diagnosis of CLD was made based on clinical features, laboratory evidence, ultrasonography findings suggestive of coarse hepatic echotexture, splenomegaly, ascites, and/or endoscopic evidence of portal hypertension.

Exclusion criteria comprised patients with acute liver injury, hepatocellular carcinoma, end stage renal disease, hematological malignancies, those on cytotoxic therapy, and patients who were non-compliant or had incomplete medical records.

A detailed clinical history was obtained from each patient, focusing on presenting symptoms, duration of illness, history of alcohol intake, comorbidities, and prior hospitalizations. General physical examination and systemic examination were performed with special emphasis on signs of liver cell failure.

Blood samples were collected under aseptic precautions and processed in the hospital's central laboratory. The hematological profile included hemoglobin (Hb), total leukocyte count (TLC), differential count (DC), platelet count, and peripheral smear examination. All hematological parameters were analyzed using an automated hematology analyzer.

Biochemical parameters included serum bilirubin (total and direct), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), serum albumin, serum globulin, prothrombin time (PT), international normalized ratio (INR), fasting blood glucose, urea, creatinine, and serum electrolytes. These were analyzed using standard enzymatic and colorimetric methods in a fully automated biochemistry analyzer.

In addition, serological testing for HBsAg, HCV, and autoimmune markers (where relevant) was conducted to determine the etiology. Abdominal ultrasonography was done to assess hepatic echotexture, liver span, splenic dimensions, and evidence of ascitic fluid or other signs of portal hypertension. Upper gastrointestinal endoscopy was performed in selected patients to document the presence of esophageal or gastric varices.

All patients were classified based on the Child-Turcotte-Pugh (CTP) score into Class A, B, or C. The MELD score was also calculated using serum bilirubin, INR, and creatinine levels to assess disease severity. Statistical analysis was performed using SPSS software version 25.0. Quantitative variables were expressed as means and qualitative variables were represented as frequencies. Significance was taken if p-value was less than 0.05.

RESULTS

Table 1: Gender and age profile (n = 150).

Age (Years)	Male (n = 94)	Female (n = 56)	Total (n = 150)	Percentage (%)
18–30	10	6	16	10.7%
31–40	19	10	29	19.3%
41–50	24	15	39	26.0%
51–60	22	13	35	23.3%
>60	19	12	31	20.7%

Table 2: Etiological Profile of CLD

Etiology	Frequency (n)	Percentage (%)
Alcoholic liver disease	62	41.3%
Chronic Hepatitis B	28	18.7%
Chronic Hepatitis C	14	9.3%
Non-alcoholic fatty liver disease (NAFLD)	29	19.3%
Autoimmune hepatitis	5	3.3%
Cryptogenic	12	8.0%

Table 3: Distribution of Haematological and Biochemical Parameters

Parameter	Mean ± SD	Reference Range
Hemoglobin (g/dL)	10.4 ± 1.9	13–17 (M), 12–15 (F)
Platelet count ($\times 10^9$ /L)	98.2 ± 34.6	150–450
Total bilirubin (mg/dL)	3.4 ± 2.1	0.2–1.2
ALT (U/L)	62.8 ± 29.4	7–56
AST (U/L)	78.1 ± 31.6	10–40
Serum albumin (g/dL)	2.8 ± 0.6	3.4–5.4
INR	1.68 ± 0.41	0.8–1.2

Table 4: Child-Pugh Classification

Child-Pugh Class	Frequency	Percentage (%)
Class A	38	25.3%
Class B	56	37.3%
Class C	56	37.3%

Table 5: Correlation of Key Parameters with Child-Pugh Class (CPC)

Parameter	Class A (Mean ± SD)	Class B (Mean ± SD)	Class C (Mean ± SD)	p-value
Platelet count	135.3 ± 22.8	101.4 ± 18.2	78.6 ± 17.1	<0.001
Serum albumin	3.3 ± 0.3	2.9 ± 0.4	2.4 ± 0.5	<0.001
Total bilirubin	1.6 ± 0.8	3.2 ± 1.3	5.4 ± 1.9	<0.001
INR	1.21 ± 0.10	1.65 ± 0.21	2.12 ± 0.37	<0.001

Out of the 150 patients with CLD, a male predominance (62.7%) was observed and majority of the patients were in the age group of 41-60 years. The most common etiology was alcoholic liver disease (41.3%), followed by NAFLD (19.3%) and chronic hepatitis B (18.7%).

Hematological abnormalities were widespread, with mean hemoglobin levels below normal (10.4 ± 1.9 g/dL) and significant thrombocytopenia (mean platelet count: 98.2 ± 34.6 $\times 10^9$ /L), suggestive of hypersplenism and chronic inflammation. Biochemically, elevated transaminase levels were observed (ALT: 62.8 U/L, AST: 78.1 U/L), with marked hyperbilirubinemia and hypoalbuminemia indicating advanced hepatocellular dysfunction.

Most of the patients belonged to Class B (37.3%) and Class C (37.3%) of CPC, thus highlighting a predominance of moderate to severe liver dysfunction. Notably, a statistically significant worsening of key biochemical parameters (serum bilirubin, albumin, INR) and platelet count was observed with advancing Child-Pugh class ($p<0.001$), supporting their utility as indicators of disease severity.

DISCUSSION

CLD is a multisystem disorder that results in fibrosis, portal hypertension, and hepatic decompensation. The clinical and laboratory derangements reflect the functional decline of hepatocytes and are crucial in prognostication and monitoring.

In the present study, the peak incidence of CLD was seen in 40–60-years which is consistent with findings by Singh et al. who reported similar age profile in CLD patients in eastern India.[11] The male predominance (62.7%) in our cohort mirrors global observations. Vento et al. attributed this to higher rates of alcohol consumption and hepatitis B exposure among males.[12]

Anemia and thrombocytopenia were notable findings in our study, with mean hemoglobin and platelet levels well below normal. Tripathi et al. reported that thrombocytopenia in CLD is frequently due to hypersplenism secondary to portal hypertension.[13]

Afdhal and Nunes also demonstrated that low platelet count serves as a surrogate marker for cirrhosis of liver.[14]

Marked elevations in AST and ALT, along with hypoalbuminemia and elevated INR, were consistent with chronic hepatocellular dysfunction. Kwo et al. noted that in cirrhosis, transaminase levels may not always reflect severity, but hypoalbuminemia and INR prolongation remain robust indicators of poor synthetic capacity.[15] Wang et al. further emphasized that hypoalbuminemia independently predicts mortality in chronic liver disease.[16]

We observed a statistically significant decline in platelet counts and serum albumin, along with rising bilirubin and INR, across worsening Child-Pugh classes (A → C). D'Amico et al. confirmed that worsening synthetic dysfunction is directly associated with clinical decompensation and poorer survival.[17] Kim et al. showed similar gradients in INR and bilirubin across MELD score strata, underscoring their diagnostic utility.[18]

Alcoholic liver disease was the most common etiology in our population, followed by NAFLD and chronic viral hepatitis. Sarin et al. documented similar trends in an Indian multicentric study, with alcohol contributing to 40–45% of cirrhosis cases.[19] NAFLD's emergence as the second leading cause may reflect a shift toward metabolic syndrome–associated liver injury, as emphasized by Younossi et al.[20]

Our findings reiterate the prognostic value of routine laboratory tests in CLD. Parameters such as serum albumin, platelet count, INR, and bilirubin can aid in disease staging, timing of interventions, and transplantation decisions. As Kamath and Kim pointed out, these variables form the basis of MELD, now a cornerstone in liver transplant prioritization.[21]

CONCLUSION

Present study findings emphasize on utility of hematological and biochemical parameters in the evaluation and monitoring of chronic liver disease. Significant abnormalities such as anemia, thrombocytopenia, hypoalbuminemia, hyperbilirubinemia, and coagulopathy were observed and found to correlate directly with the severity of liver dysfunction. Among these, platelet count, serum albumin, INR, and total bilirubin emerged as critical markers in stratifying disease severity. The predominance of alcohol-related liver disease and the rising contribution of NAFLD highlight the changing etiological landscape and call for targeted preventive strategies.

These findings support the integration of routine laboratory parameters in clinical decision-making, especially in resource-constrained settings where access to advanced diagnostic tools may be limited. Future multicentric longitudinal studies with dynamic monitoring are warranted to validate these correlations and improve prognostic models for chronic liver disease patients.

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