Original Article

Study of Platelet Indices as Non-Invasive Biomarkers in Chronic Liver Disease: A Cross-Sectional Analysis

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Abstract

Background: Chronic liver disease (CLD) affects more than 800 million people in the world, and they should be properly assessed in terms of severity to get the best management. The gold standard, which is liver biopsy, is invasive and not readily available. The platelet indices, which are routinely included in complete blood counts, have become a promising non-invasive biomarker that reflects a complex interaction between platelets and hepatic pathophysiology. **Material and Methods:** This cross-sectional observational study recruited 266 clinically diagnosed patients with CLD from NSCB Medical College and Hospital between September 2022 and February 2024. Platelet count (PC), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), platelet large cell count (PLCC), and platelet large cell ratio (PLCR) were determined with the help of an automated hematology analyzer. Pearson correlation tests and Chi-square tests were used to assess correlations among Child-Turcotte-Pugh (CTP), Model for End-Stage Liver Disease (MELD), MELD-Na, and the AST-to-platelet ratio index (ASTP). **Results:** The cohort itself was mainly male (95.5%), and the alcoholic liver disease was predominant (72.6%). PC showed significant negative correlations with MELD ($r = -0.465$, $p = 0.002$), MELD-Na ($r = -0.442$, $p = 0.003$), and APRI ($r = -0.458$, $p = 0.002$) in patients with cirrhosis ($n = 42$). There were similar negative correlations between PCT and PLCC. MPV and PDW showed a positive association with disease severity. Across CTP classes, there was a progressive increase in Thrombocytopenia (A: 36.4, B: 53.8, C: 56.0, $p=0.017$). There was a strong interrelationship among PC, PCT, and PLCC. **Conclusion:** Platelet indices are inexpensive, easily obtained biomarkers of CLD severity that correlate significantly with standard scoring scales. Their standardized implementation in clinical practice has the potential to improve risk stratification, especially in resource-restricted locations where sophisticated non-invasive tests are not available.

Keywords: Platelet Indices or Chronic Liver Disease or Thrombocytopenia or Non-Invasive Biomarkers or Cirrhosis or Child-Turcotte-Pugh Score.

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Introduction

Chronic liver disease (CLD) is a major cause of morbidity and mortality, with the global prevalence of this disease being more than 800 million people, with a significant burden observed in low- and middle-income countries.[1] A pantheon of CLD includes alcoholic liver disease (ALD), viral hepatitis, non-alcoholic fatty liver disease (NAFLD), and autoimmune diseases, which change into fibrosis, cirrhosis, and hepatocellular carcinoma.[2] Proper classification of disease severity is critical for end-of-course decision-making, prognostication, and transplant decision-making. Previously, the gold standard for staging fibrosis and grading necroinflammatory activity has been liver biopsy.[3] Its invasiveness, the possibility of complications, sampling variability, and limited repeatability require the establishment of effective non-invasive approaches.[4]

The last ten years have also seen the development of highly advanced non-invasive procedures such as transient elastography (FibroScan) and serological fibrosis testing (FibroTest, Enhanced Liver Fibrosis test).[5] Although these modalities exhibit reasonable diagnostic accuracy, they are only available in limited numbers in resource-constrained settings, are costly, and require specialized skills, limiting their use to a minority.[6] It is therefore clear that there is a pressing need for readily available, cheap, and reproducible biomarkers that can be integrated into clinical practice.

Although the hemostatic roles of platelets have long been known, they have come to be recognised as important immune homeostats actively involved in liver pathophysiology.[7] The liver is a key regulator of platelet biology, the major regulator of megakaryopoiesis, thrombopoietin (TPO), and the coordination of platelet clearance by hepatic asialoglycoprotein receptors.[8] Complicated pathophysiological processes such as splenic sequestration caused by portal hypertension, depressed TPO

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production, bone marrow, and platelet destruction by autoimmune processes are culminating factors in thrombocytopenia seen in more than 75 per cent of the patients with cirrhosis.[9]

In addition to mere platelet counting, the latest automated hematology analyzers offer advanced platelet indices for platelet morphology, platelet activity, and platelet proliferation. These ratios, including mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), platelet large cell count (PLCC), and platelet large cell ratio (PLCR), have been proven to be diagnostic in a wide variety of inflammatory and neoplastic disorders.[10] More recent studies have specifically examined their role in CLD, with some demonstrating a correlation between MPV and the severity of hepatic fibrosis in chronic hepatitis B and C infections.[11,12] Advanced fibrosis and unfavourable short-term prognosis of acute-on-chronic liver failure have been linked to elevated MPV.[13] On the same note, PDW has also shown potential for platelet activation and disease activity.[14]

Although evidence has accumulated, we do not have answers to many questions. The majority of research has focused on selected indices (primarily MPV and PC) in specific etiologies, and little information has been reported on comprehensive platelet index panels across varied CLD groups, especially in the Indian subcontinent, where ALD is predominantly prevalent.[15] Moreover, new indices, including PLCC and PLCR, have not been explored for CLD severity. The proposed research will fill these gaps by assessing a full set of platelet indices and their relationship with referenced severity scores in a heterogeneous CLD cohort.

Materials and Methods

Study Design and Setting: It was a cross-sectional observational study conducted at the Department of Pathology, NSCB Medical College and Hospital, Jabalpur, Madhya Pradesh, India, from September 1, 2022, to February 29, 2024. The examination guideline was ethically justified by the Institutional Review Board (Ref: NSCB/2022/Path/147), and the principles of the Declaration of Helsinki were observed. Informed consent from everyone was obtained through a written consent form.

Sample Size Calculation and Population of the Study.

The standard deviation in platelet count for CLD patients, 41.9 × 10^3/dL, documented in earlier studies, with a 5% relative error and 95% confidence interval, yielded a minimum possible sample size of 260, calculated using the formula n = Z 2S 2/d 2. We recruited 266 patients with chronic liver disease, regardless of age, who attended the Department of Medicine for possible losses.

Inclusion and Exclusion Criteria.

Inclusion criteria included: (1) patients between the ages of 18 and 85 years with established CLD (symptoms for more than 6 months); (2) clinical, biochemical, and radiological diagnosis; (3) access to full medical history and laboratory tests. The exclusion criteria were: (1) previous gastrointestinal or hematological malignancies; (2) major coagulation disorders; (3) acute liver failure; (4) patients taking antiplatelet treatment in the past two weeks; (5) pregnancy; (6) incomplete lab data.

Data gathering and Clinical measurement.

Prospective clinical information was documented, including demographics, etiology of liver disease (alcoholic, viral hepatitis B/C, NAFLD/NASH, autoimmune, cryptogenic), and complications (ascites, hepatic encephalopathy, variceal bleeding, spontaneous bacterial peritonitis). Clinical examination of all patients about the stigmata of chronic liver disease and portal hypertension was conducted extensively.

Laboratory Investigations: Hematological Parameters: Venous blood was collected in an EDTA Vacutainer (purple top) and examined within 2 hours using the Serachem SC60 3-Part Automated Hematology Analyzer under the principle of electrical impedance. Platelet counts were also measured that included; platelet count (PC, reference: 150-450 ×10 6/?L), platelet mean platelet volume (MPV, reference: 7.0-11.0 fL), platelet distribution width (PDW, reference: 9.0-17.0 63/L), plateletcrit (PCT, reference: 0.108-0.282 63/L), platelet large cell count (PLCC, reference: 30-90

Biochemical Parameters: Serum samples (red-top Vacutainer tubes) were tested on the HumaStar 600 automated biochemical analyzer. They were total bilirubin, direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total protein, albumin, globulin, blood urea, serum creatinine, sodium, and potassium.

Coagulation Profile: Prothrombin time (PT) was determined using the ThromboSpan LS PT kit (rabbit brain thromboplastin) in citrated plasma samples (sky blue top) with the tilt-tube manual process at 37 °C. The ISI value of 1.1 was used as the calculated international normalized ratio (INR).

Disease Severity Scoring

Child-Turcotte-Pugh (CTP) Classification: The patient received a score (1-3) on five parameters (encephalopathy, ascites, bilirubin, albumin, INR) and was categorized as Class A (5-6 points), Class B (7-9 points), or Class C (10-15 points).

MELD and MELD-Na Scores: The scores are calculated with the help of standardized formulas:

Where MELD = logarithmic bilirubin, logarithmic INR, logarithmic creatinine, and logarithmic alkaline.

- MELD-Na = MELD(i) + 1.32 × (137 - Na) - [0.033 × MELD(i) × (137 - Na)]

APRI Score: With the formula, (AST/upper normal) 100/ platelet count (× 10wing / L). Values below 0.5 indicated low fib probability, and those above 1.5 indicated high fib probability.

Statistical Analysis

SPSS version 27.0 was used to analyse data. Continuous variables would be reported as mean ± SD or median (interquartile range). The frequencies and percentages were used to present the data in categorical form. The Pearson correlation coefficient was used to test correlations between platelet indices and severity scores. Chi-square test was used to test relationships between categorical variables. A one-way ANOVA was used to compare platelet indices across CTP classes. The statistical significance level was established at $p<0.05$ (two-tailed).

Results

Boundary Characteristics and Etiological Distribution.

The researchers recruited 266 patients with chronic liver disease. The average age was 42.67, and the standard deviation was 11.69 (minimum: 14-83 years). There was male preeminence (n=254, 95.5%). The most common etiology was found to be alcoholic liver disease (n=193, 72.6%), then cryptogenic CLD (n=36, 13.5%), then chronic hepatitis B (n= 26, 9.8%), and chronic hepatitis C (n= 5, 1.9%), followed by NAFLD/NASH (n= 4, 1.6%), other unusual etiologies (wilson disease, Budd-Chiari syndrome, 0.8%). In males, the majority of cases were aged 34-43 years (34.6%), whereas in females, the highest age group was 44-53 years (33.0%) [Table 1].

Table 1: Demographic and etiological characteristics of study population

Parameter	Category	Frequency (n=266)	Percentage
Gender	Male	254	95.5
	Female	12	4.5
Age Group (years)	14-23	6	2.3
	24-33	51	19.2
	34-43	90	33.8
	44-53	69	25.9
	54-63	36	13.5
	64-73	13	4.9
	74-83	1	0.4
Etiology	Alcoholic Liver Disease	193	72.6
	Cryptogenic	36	13.5
	Hepatitis B	26	9.8
	Hepatitis C	5	1.9
	NAFLD/NASH	4	1.6
	Others	2	0.8

Clinical Characteristics and Disease severity.

The disease was compensated (n=259, 97.4%). Ascites (n=232, 87.2%), jaundice (n=116, 43.6%), splenomegaly (n=128, 48.1%), esophageal varices (n=97, 36.5%), hepatic encephalopathy (n=57, 21.4%), and portal hypertension (n=65, 24.4%) were the frequent ones. Forty-two patients (15.8%) had clinico-radiologically confirmed cirrhosis. CTP support identified 11 (4.1), 91 (34.2), and 164 (61.7) patients in Class A, Class B, and Class C, respectively [Table 2].

Table 2: Disease severity parameters and platelet indices across CTP classes

Parameter	CTP Class A (n=11)	CTP Class B (n=91)	CTP Class C (n=164)	p-value
Platelet Count (×10³/µL)	198.4 ± 89.2	166.8 ± 92.7	142.3 ± 87.1	0.017
MPV (fL)	8.4 ± 1.1	8.9 ± 1.2	9.2 ± 1.3	0.042
PDW (%)	15.8 ± 1.2	16.1 ± 1.4	16.6 ± 1.5	0.012
PCT (%)	0.18 ± 0.08	0.15 ± 0.09	0.12 ± 0.07	<0.001
PLCC (×10⁹/L)	52.4 ± 28.3	41.8 ± 26.7	38.2 ± 24.9	0.034
PLCR (%)	22.1 ± 8.9	24.6 ± 9.4	25.8 ± 9.8	0.156
MELD Score	8.2 ± 1.4	14.7 ± 3.8	19.3 ± 5.2	<0.001
APRI Score	1.8 ± 0.9	2.4 ± 2.1	3.1 ± 2.8	0.003

Correlation Analysis in Cirrhosis Patients.

A strong correlation between platelet indices and scores that determined disease severity arose in the cirrhosis subgroup (n=42), [Table 3], p=.000). PC was negatively correlated with MELD (r=-0.465, p=0.002), MELD-Na (r=-0.442, p=0.003), and APRI (r=-0.458, p=0.002). PCT depicted negative associations (MELD: r= -0.455, p=002; MELD-Na: r= -0.426, p=005; APRI: r= -0.452, p=003). PLCC was also negatively correlated with MELD (r = -0.507, p = 0.008), MELD-Na (r = -0.483, p = 0.012), and APRI (r = -0.460, p = 0.018). On the other hand, MPV showed positive coefficients with severity scores, with relatively weak statistics of significance (APRI: r=0.228, p=0.147). APRI was positively related to PDW (r=0.280, p=0.072).

Table 3: Correlation of platelet indices with disease severity scores in cirrhosis patients

Platelet Index	MELD	MELD-Na	APRI
PC	r=-0.465, p=0.002	r=-0.442, p=0.003	r=-0.458, p=0.002
MPV	r=0.112, p=0.479	r=0.150, p=0.342	r=0.228, p=0.147
PDW	r=-0.050, p=0.751	r=-0.053, p=0.740	r=0.280, p=0.072
PCT	r=-0.455, p=0.002	r=-0.426, p=0.005	r=-0.452, p=0.003
PLCC	r=-0.507, p=0.008	r=-0.483, p=0.012	r=-0.460, p=0.018
PLCR	r=0.152, p=0.335	r=0.154, p=0.331	r=0.228, p=0.147

Inter-relationships Among Platelet Indices.

Positive, significant relationships were observed between PC and PCT (r=0.948, p<0.001) and between PC and PLCC (r=0.872, p<0.001) among all patients. MPV had a positive relationship with PLCR (r=0.907, p<0.001) and PDW (r=0.323, p<0.001). There were negative correlations between PC and PLCR (r=-0.281, p<0.001) and between PC and PDW (r=-0.264, p<0.001).

Etiology-Specific Findings

PC showed a significant negative correlation with APRI (r = -0.467, p = 0.001) and CTP (r = -0.043, p = .555) in patients with

ALD (n = 193). MPV was negatively related to serum albumin (r=-0.202, p=0.005). MPV was also significantly positively correlated with APRI (r=0.419, p=0.019) and AST (r=0.503, p=0.004) in patients with chronic hepatitis (n=31). Cryptogenic CLD group (n=36) exhibited a significant negative correlation of PC and APRI (r= -0.375, p=0.024). A trend was observed among NAFLD/NASH patients (n=4), but the study lacked statistical power due to the small sample size.

Discussion

This comprehensive cross-sectional study demonstrates that platelet indices serve as valuable, accessible biomarkers for assessing severity in chronic liver disease. Our findings reveal robust negative correlations between platelet count, plateletcrit, and platelet large cell count with established disease severity scores, particularly in cirrhotic patients. Conversely, mean platelet volume and platelet distribution width exhibit positive correlations with disease progression. These observations align with the complex pathophysiology of thrombocytopenia in CLD and support the integration of platelet indices into routine clinical assessment. The predominant etiology of ALD (72.6%) in our cohort reflects the epidemiological transition observed in Indian populations, where alcohol consumption patterns are shifting CLD demographics away from viral hepatitis.[10,11] This distribution underscores the importance of validating biomarkers across diverse etiological backgrounds. The male predominance (95.5%) correlates with higher alcohol consumption patterns in this region, though we acknowledge potential gender-specific disease manifestations that warrant targeted investigation.

Our finding of significant negative correlation between platelet count and MELD/MELD-Na scores in cirrhosis ($r = -0.465$ and $r = -0.442$ respectively) corroborates extensive literature linking thrombocytopenia to advanced liver disease.[12,13] The mechanism is multifactorial: reduced hepatic thrombopoietin production, splenic sequestration due to portal hypertension, and immune-mediated platelet destruction collectively reduce circulating platelets.[14] The correlation with APRI ($r = -0.458$) is particularly noteworthy given that platelet count constitutes the denominator of this fibrosis index, yet the relationship remains clinically informative. The progressive increase in thrombocytopenia prevalence across CTP classes (36.4% to 56.0%) demonstrates a clear dose–response relationship, reinforcing platelet count as a reliable surrogate for hepatic functional reserve.[15]

MPV represents platelet size and reflects bone marrow activity. Our observation of higher MPV in advanced disease (CTP Class C: 9.2 ± 1.3 fL) aligns with studies by Ceylan et al. and Ekiz et al. in chronic hepatitis B, where MPV correlated with fibrosis stage.[16,17] This increase likely reflects compensatory megakaryocyte stimulation in response to peripheral platelet destruction, releasing larger, younger platelets into circulation.[18] The significant negative correlation between MPV and serum albumin in ALD patients ($r = -0.202$, $p = 0.005$) mirrors previous findings, suggesting that MPV may reflect synthetic dysfunction.[19]

PDW, a more precise indicator of platelet anisocytosis than MPV, showed progressive elevation with disease severity.[20] The significant correlation between PDW and CTP class in cryptogenic CLD supports its role as an activation marker. Increased PDW indicates heterogeneous platelet populations, reflecting dysregulated thrombopoiesis and peripheral consumption. This parameter may capture platelet dynamics more sensitively than MPV, as it remains unaffected by platelet swelling and directly measures size variability.

PCT, representing total platelet mass, demonstrated the strongest negative correlations with severity scores in cirrhosis. This finding exceeds the predictive value of platelet count alone, as PCT integrates both platelet number and volume. Similar observations have been reported showing superior correlation of PCT with liver fibrosis compared to platelet count in chronic hepatitis B. Our observation that PCT declines precipitously across CTP classes suggests its utility as a screening tool for advanced fibrosis.

PLCC and PLCR represent emerging indices with limited prior investigation in CLD. Our novel finding of significant negative correlation between PLCC and MELD contradicts the expected compensatory increase in large platelets. This paradox may reflect profound bone marrow suppression in end-stage liver disease, where even megakaryocyte reserve becomes exhausted. The positive correlation between PLCR and platelet count reflects both a mathematical relationship and a compensatory increase in the proportion of large platelets as total platelet count declines.

The differential correlation patterns across etiologies are noteworthy. In chronic hepatitis patients, MPV showed significant correlation with AST and APRI, suggesting that inflammatory activity drives platelet size changes. In contrast, ALD patients exhibited stronger correlations with synthetic function, reflecting the metabolic derangements characteristic of alcohol-related liver injury. The limited sample size for NAFLD/NASH precluded meaningful analysis, representing a significant limitation given the rising burden of metabolic liver disease.

Our findings have immediate clinical applicability, particularly in resource-limited settings where advanced non-invasive tests are unavailable. Platelet indices are generated automatically with complete blood counts at minimal additional cost, requiring no specialized equipment or expertise. The strong correlations with MELD and CTP scores suggest they could assist in triaging patients for referral to tertiary centers or prioritizing for transplant evaluation.

This study has several limitations. The cross-sectional design precludes assessment of temporal trends and causal relationships. The single-center setting may limit generalizability, though our diverse etiological distribution partially mitigates this concern. The small sample size for NAFLD/NASH and other rare etiologies restricts subgroup analysis. We correlated indices with clinical scores rather than histopathological staging, which remains the reference standard despite its limitations. Future prospective, multicenter studies incorporating longitudinal follow-up and histopathological correlation are warranted to validate these findings and establish cutoff values for clinical decision-making. Investigation of platelet indices in predicting specific complications such as variceal bleeding or hepatorenal

syndrome could further expand their clinical utility.

Conclusion

This cross-sectional study demonstrates that platelet indices serve as readily accessible, cost-effective biomarkers for assessing chronic liver disease severity. Platelet count and plateletcrit show significant negative correlations with MELD, MELD-Na, and APRI scores in cirrhotic patients, while mean platelet volume and platelet distribution width exhibit positive associations with disease progression. The prevalence of thrombocytopenia increases significantly across Child-Turcotte-Pugh classes, reflecting deteriorating hepatic function. These findings support the integration of platelet indices into routine CLD monitoring protocols, particularly in resource-limited settings where advanced non-invasive tests are unavailable. Clinicians should interpret abnormal platelet indices as potential indicators of advanced fibrosis and consider prompt referral for specialized care. Future prospective studies with histopathological correlation are needed to establish definitive diagnostic thresholds and validate long-term predictive utility.

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Conflicts of interest

There are no conflicts of interest.

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