Research Article

Mean Platelet Volume As A Potential Prognostic Marker in Patients With Decompensated Liver Disease: A Prospective Observational Study

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Abstract

Background: Anemia, cytopenias and deranged coagulation are common in decompensated liver disease, yet the platelet indices reported with every automated blood count are rarely used for risk assessment. Mean platelet volume (MPV) reflects platelet size and turnover and is available without extra cost. This study examined whether MPV is related to thrombocytopenia and coagulopathy in patients with decompensated liver disease.

Methods: In this prospective observational study, 60 consecutive adults with decompensated chronic liver disease were enrolled in the Department of General Medicine of a tertiary teaching hospital in Chennai, India, between May 2024 and December 2025. Complete blood count with MPV, peripheral smear, liver and renal function tests, prothrombin time (PT), international normalized ratio (INR), fibrinogen and abdominal ultrasonography were recorded. Patients were grouped by MPV as <9, 9–11 and >11 fL. Categorical associations were tested with the chi-square test and the chi-square test for linear trend.

Results: Men formed 70.0% of the cohort and alcohol was the leading cause (53.3%). Anemia was present in 46 (76.7%), thrombocytopenia in 40 (66.7%) and leukopenia in 22 (36.7%) patients; PT was prolonged in 34 (56.7%). Thrombocytopenia rose from 42.9% in the lowest MPV band to 68.8% and 85.7% in the middle and highest bands (p for trend = 0.017). Coagulopathy increased in the same direction (42.9%, 56.3% and 71.4%), though not significantly (p for trend = 0.130). Anemia was more frequent in patients with coagulopathy (88.2% vs 61.5%; p = 0.015).

Conclusion: Higher MPV accompanied a greater frequency of thrombocytopenia in decompensated liver disease. MPV may help identify patients with more marked platelet abnormalities, but its prognostic value needs confirmation in larger outcome-based studies that include Child-Pugh and MELD scores.

Keywords: Mean platelet volume; Liver cirrhosis; Thrombocytopenia; Blood coagulation disorders; Anemia; Hypersplenism.

INTRODUCTION

Decompensation is the point at which the course of cirrhosis changes sharply. Once ascites, variceal hemorrhage, hepatic encephalopathy or jaundice develop, median survival drops from more than 12 years to roughly 2 years [1]. For this reason, current European guidance treats decompensated cirrhosis as a separate clinical stage that calls for closer monitoring and earlier intervention [2]. The burden is large: cirrhosis was responsible for about 1.32 million deaths worldwide in 2017, and South Asia contributed a considerable share of them [3].

Blood count abnormalities are among the most frequent findings in these patients. Anemia, leukopenia and thrombocytopenia may appear singly or in combination, and a fall in platelet count is often the earliest change [4]. Thrombocytopenia arises from pooling of platelets in an enlarged spleen, reduced hepatic production of thrombopoietin and, in alcohol-related disease, direct suppression of the marrow [5,6]. Hypersplenism secondary to portal hypertension further shortens the life span of all three cell lines [7]. Prolonged PT and raised INR reflect the reduced synthesis of clotting factors, although the hemostatic balance in cirrhosis is more complex than these conventional tests suggest [8]. Anemia affects about three quarters of patients with chronic liver disease and has several overlapping causes, including gastrointestinal blood loss, hypersplenism, folate deficiency and the toxic effect of alcohol on red cell production [9].

Mean platelet volume (MPV) is generated automatically with every complete blood count. Larger platelets are generally younger and more reactive, and MPV changes in conditions of increased platelet turnover and systemic inflammation [10]. Since it adds no cost, MPV is an attractive bedside marker in hospitals where elastography or hepatic venous pressure measurement are not routinely available. However, published findings on its role in

cirrhosis are inconsistent, and few studies have examined it in Indian patients who already have decompensated disease.

The present study was therefore undertaken to describe the hematological profile of patients with decompensated liver disease and to assess whether MPV is associated with thrombocytopenia and coagulopathy in this group.

MATERIALS AND METHODS

Study design and setting: This hospital-based prospective observational study was carried out in the Department of General Medicine, A.C.S. Medical College and Hospital, Dr. M.G.R. Educational and Research Institute, Maduravoyal, Chennai, Tamil Nadu, India, from May 2024 to December 2025.

Ethics: Approval for the study was obtained from the Institutional Ethics Committee of A.C.S. Medical College and Hospital, Chennai, before enrolment began. Written informed consent was obtained from every participant, and patient confidentiality was maintained throughout. No investigation was performed solely for research purposes.

Participants: Adults aged 18 years or more with chronic liver disease of more than six months' duration, abnormal liver function tests and ultrasonographic evidence of chronic liver disease were eligible if they had at least one feature of decompensation (ascites, variceal bleeding, hepatic encephalopathy or jaundice). Alcoholic, viral, non-alcoholic fatty and other causes were included. Patients with primary coagulation disorders, gastrointestinal or hematological malignancy, acute liver failure, pregnancy, sepsis unrelated to liver disease, or ongoing treatment for anemia of non-hepatic origin were excluded.

Sample size: Using the formula $n = Z^2P(1–P)/L^2$, with Z = 1.96, an expected prevalence of hematological abnormalities of 80% and an absolute precision of 10%, the required sample was about 61. A total of 60 patients were enrolled by consecutive sampling.

Data collection: A structured proforma was used to record age, sex, duration of illness, alcohol intake, previous viral hepatitis and presenting complaints, including abdominal distension, abdominal pain, leg swelling, jaundice, hematemesis and melena. Clinical examination documented ascites, splenomegaly and stigmata of chronic liver disease. Investigations included complete blood count with MPV, peripheral smear, serum bilirubin, transaminases, alkaline phosphatase, albumin, renal function tests, PT, INR, fibrinogen, bleeding time and clotting time. Reticulocyte count, iron studies, vitamin B12 and folate were measured in patients with anemia. Abdominal ultrasonography was done to look for cirrhosis, portal hypertension and splenomegaly.

Definitions: Anemia was defined according to World Health Organization criteria as hemoglobin <13 g/dL in men and <12 g/dL in women, and graded as mild, moderate or severe. Leukopenia was defined as a total leukocyte count <4,000 cells/mm^3. Thrombocytopenia was defined as a platelet count <150,000/mm^3 and graded as mild, moderate or severe according to laboratory reference ranges. Coagulopathy was defined as prolongation of PT or elevation of INR above the laboratory reference range. Gastrointestinal bleeding was defined as hematemesis or melena at presentation. MPV was grouped as <9 fL, 9–11 fL and >11 fL.

Statistical analysis: Data were entered in Microsoft Excel and analyzed with statistical software. Categorical variables are presented as frequency and percentage. The Pearson chi-square test was used for associations between nominal variables. Because MPV bands are ordered, their association with thrombocytopenia and coagulopathy was tested with the chi-square test for linear trend. For tables with sparse cells (etiology), an exact test based on Monte Carlo simulation was used. A p value <0.05 was considered statistically significant. The study workflow is summarized in Figure 1.

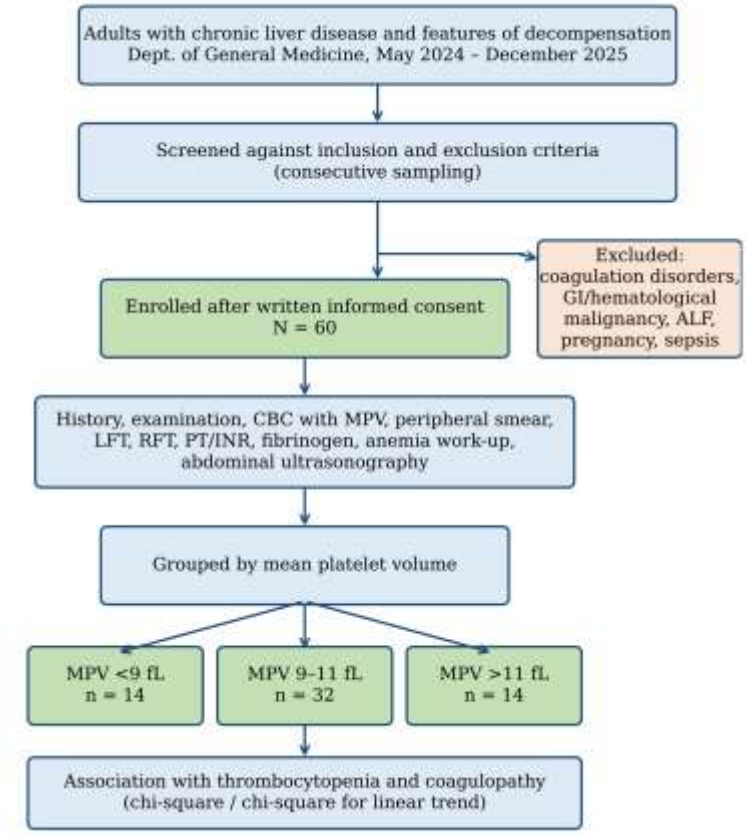


Figure 1. Flow diagram of patient selection, evaluation and analysis. MPV: mean platelet volume; CBC: complete blood count; LFT: liver function tests; RFT: renal function tests; PT/INR: prothrombin time/international normalized ratio; ALF: acute liver failure; GI: gastrointestinal.

RESULTS

Baseline profile: Sixty patients were studied. The largest age group was 41–50 years (26.7%), and 42 patients (70.0%) were men. Alcohol-related liver disease was the most common cause (53.3%), followed by viral hepatitis (23.3%) and non-alcoholic fatty liver disease (15.0%). Ascites (73.3%) and jaundice (63.3%) were the dominant clinical features, and 16 patients (26.7%) presented with hematemesis (Table 1; Figures 2 and 3).

Table 1. Baseline characteristics of the study participants (N = 60)

Variable	Category	n (%)
Age (years)	18–30	8 (13.3)
	31–40	12 (20.0)
	41–50	16 (26.7)
	51–60	14 (23.3)
	>60	10 (16.7)
Sex	Male	42 (70.0)
	Female	18 (30.0)

Variable	Category	n (%)
Etiology	Alcohol-related liver disease	32 (53.3)
	Viral hepatitis	14 (23.3)
	NAFLD	9 (15.0)
	Others (autoimmune/metabolic)	5 (8.3)
Clinical features	Ascites	44 (73.3)
	Jaundice	38 (63.3)
	Abdominal pain	28 (46.7)
	Pedal edema	22 (36.7)
	Hematemesis	16 (26.7)
	Melena	14 (23.3)

NAFLD: non-alcoholic fatty liver disease. Clinical features are not mutually exclusive.

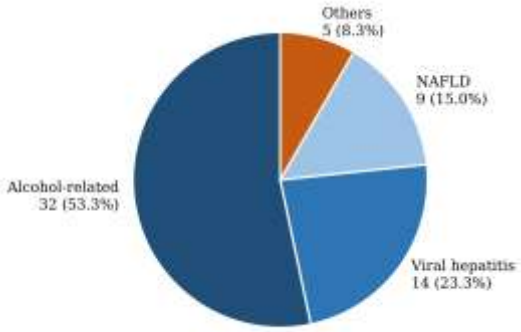


Figure 2. Pie chart showing the etiology of decompensated liver disease (N = 60). NAFLD: non-alcoholic fatty liver disease.

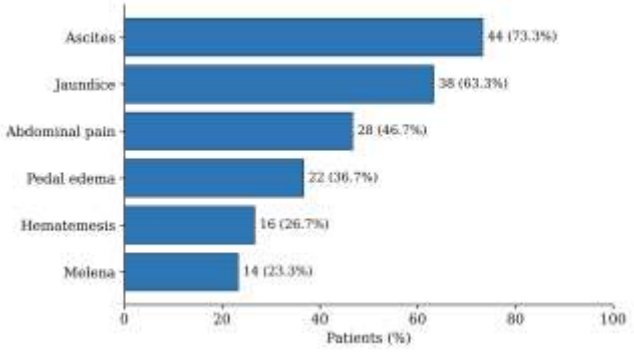


Figure 3. Bar diagram of presenting clinical features (N = 60). Features are not mutually exclusive.

Hematological and coagulation profile: Anemia was the most common abnormality (76.7%), and moderate anemia was the largest severity group (33.3%). Normocytic normochromic anemia was the predominant morphological type (36.7%), followed by macrocytic (23.3%) and microcytic hypochromic anemia (16.7%). Two thirds of the patients had thrombocytopenia, mostly of mild degree. PT was prolonged in 56.7% and INR in 50.0%. More than half of the patients (53.3%) had MPV between 9 and 11 fL (Table 2; Figures 4 and 5).

Table 2. Hematological, coagulation and MPV profile (N = 60)

Parameter	Category	n (%)
Cytopenias	Anemia	46 (76.7)
	Thrombocytopenia	40 (66.7)
	Leukopenia	22 (36.7)
Severity of anemia	Mild	14 (23.3)
	Moderate	20 (33.3)
	Severe	12 (20.0)
	No anemia	14 (23.3)
Type of anemia	Normocytic normochromic	22 (36.7)
	Macrocytic	14 (23.3)
	Microcytic hypochromic	10 (16.7)
Platelet count	Normal	20 (33.3)
	Mild thrombocytopenia	18 (30.0)
	Moderate thrombocytopenia	14 (23.3)
	Severe thrombocytopenia	8 (13.3)
Coagulation	Prolonged PT	34 (56.7)
	Prolonged INR	30 (50.0)
	Low fibrinogen	18 (30.0)
MPV	<9 fL	14 (23.3)
	9–11 fL	32 (53.3)
	>11 fL	14 (23.3)

MPV: mean platelet volume; PT: prothrombin time; INR: international normalized ratio.

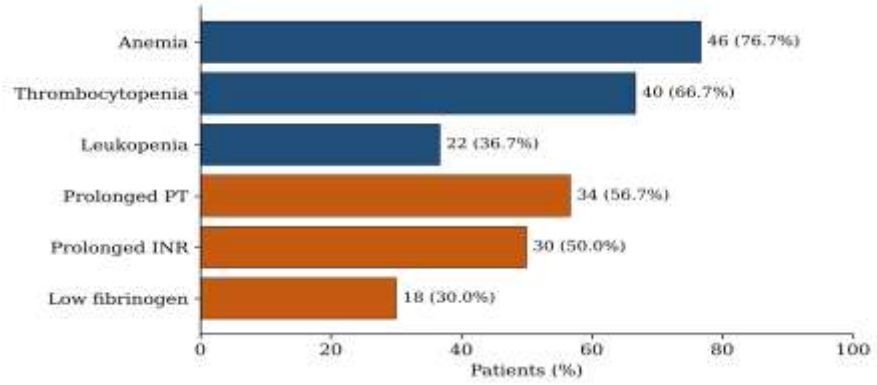


Figure 4. Prevalence of cytopenias (blue) and coagulation abnormalities (orange) among the study participants (N = 60). PT: prothrombin time; INR: international normalized ratio.

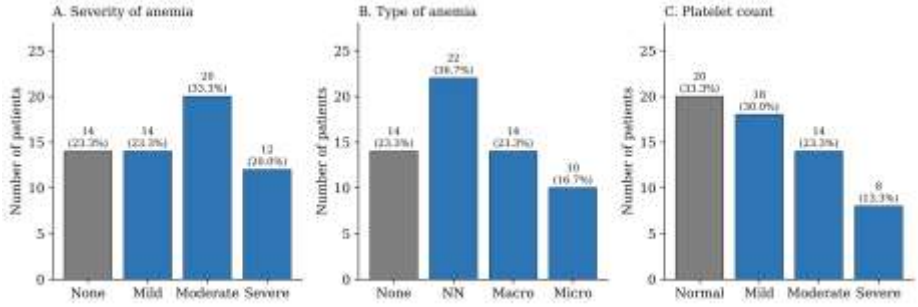


Figure 5. Distribution of (A) anemia severity, (B) morphological type of anemia and (C) platelet count category (N = 60). NN: normocytic normochromic; Macro: macrocytic; Micro: microcytic hypochromic.

MPV, thrombocytopenia and coagulopathy: The proportion of patients with thrombocytopenia increased steadily across MPV bands, from 42.9% (<9 fL) to 68.8% (9–11 fL) and 85.7% (>11 fL). This linear trend was statistically significant (χ^2 for trend = 5.69, p = 0.017). Coagulopathy showed a similar upward pattern (42.9%, 56.3% and 71.4%), but the trend did not reach significance (p = 0.130) (Table 3; Figure 6).

Table 3. Association of MPV with thrombocytopenia and coagulopathy

MPV (fL)	Total, n	Thrombocytopenia present, n (%)	Coagulopathy present, n (%)
<9	14	6 (42.9)	6 (42.9)
9–11	32	22 (68.8)	18 (56.3)
>11	14	12 (85.7)	10 (71.4)
Total	60	40 (66.7)	34 (56.7)
χ^2 for linear trend; p		5.69; **0.017**	2.29; 0.130
Pearson χ^2 (df = 2); p		5.90; 0.052	2.33; 0.312

Coagulopathy: prolonged PT or raised INR. Bold value indicates p <0.05.

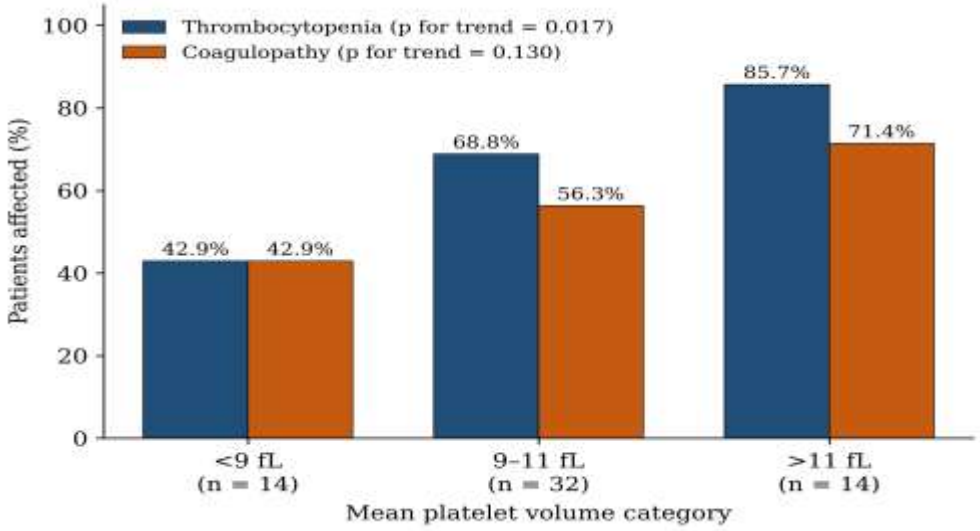


Figure 6. Clustered bar diagram showing the proportion of patients with thrombocytopenia and coagulopathy across MPV categories. Thrombocytopenia increased significantly with rising MPV (chi-square for linear trend, p = 0.017); the rise in coagulopathy was not significant (p = 0.130).

Other associations: Anemia was significantly more frequent in patients with coagulopathy (88.2% vs 61.5%; p = 0.015). Anemia was commonest in alcohol-related disease (87.5%), but differences across etiologies were not significant (p = 0.142); age and sex also showed no significant association. Thrombocytopenia was more frequent in patients with leukopenia (81.8% vs 57.9%; p = 0.058), and gastrointestinal bleeding occurred in 32.5% of thrombocytopenic patients compared with 15.0% of those with normal platelet counts (p = 0.148); neither difference was statistically significant (Table 4).

Table 4. Factors associated with anemia, thrombocytopenia and gastrointestinal bleeding

Variable	Category	Outcome present, n (%)	p value
Anemia			
Age	≤50 years (n = 36)	26 (72.2)	0.319
	>50 years (n = 24)	20 (83.3)	
Sex	Male (n = 42)	34 (81.0)	0.231
	Female (n = 18)	12 (66.7)	
Etiology	Alcohol-related (n = 32)	28 (87.5)	0.142[a]
	Viral (n = 14)	10 (71.4)	
	NAFLD (n = 9)	5 (55.6)	
	Others (n = 5)	3 (60.0)	
Coagulopathy	Present (n = 34)	30 (88.2)	**0.015**
	Absent (n = 26)	16 (61.5)	
Thrombocytopenia			
Sex	Male (n = 42)	30 (71.4)	0.232
	Female (n = 18)	10 (55.6)	
Etiology	Alcohol-related (n = 32)	24 (75.0)	0.588[a]
	Viral (n = 14)	8 (57.1)	
	NAFLD (n = 9)	5 (55.6)	
	Others (n = 5)	3 (60.0)	
Leukopenia	Present (n = 22)	18 (81.8)	0.058
	Absent (n = 38)	22 (57.9)	
GI bleeding			
Platelet status	Thrombocytopenia (n = 40)	13 (32.5)	0.148
	Normal (n = 20)	3 (15.0)	

Pearson chi-square test unless stated. [a]Exact test (Monte Carlo) for sparse cells. Bold value indicates p <0.05. GI: gastrointestinal.

DISCUSSION

In this cohort of 60 patients with decompensated liver disease, three of every four patients were anemic and two of every three had thrombocytopenia. The more useful observation concerns MPV: the share of thrombocytopenic patients climbed steadily across the three MPV bands, and this trend was statistically significant. Coagulopathy rose in the same direction, although the change was not large enough to be significant in a sample of this size.

Men made up 70.0% of our patients, and alcohol accounted for 53.3% of cases. In the multicentre Indian survey by Mukherjee et al., which covered 13,014 patients with chronic liver disease, 73% were men and alcohol was the most frequent cause of cirrhosis at 34.3% [11]. The higher alcohol share in our series probably reflects the case mix of a tertiary medical ward rather than a true regional difference. Behera and Dash, who studied 69 patients with cirrhosis, reported an even stronger alcohol predominance (92.75%), together with ascites in 84.06%, anemia in 95.65% and thrombocytopenia in 68.12% [12]. Our figures for ascites (73.3%) and thrombocytopenia (66.7%) are close to theirs, while anemia was less frequent in our patients (76.7%), possibly because fewer of them had alcohol-related disease. Joshi et al. found anemia in 85.3% of patients with chronic liver disease, with moderate anemia forming the largest group (55.3%) and normocytic normochromic morphology the commonest pattern (28.7%); thrombocytopenia was present in 60% [13]. The order of morphological types in our study was the same, although moderate anemia made up a smaller share (33.3%). In a much larger series of 1,979 hospitalized cirrhotic patients, Ren et al. reported anemia in 70.2% and showed that severe anemia independently increased 90-day mortality (HR 1.649) [14]. Twelve of our patients (20.0%) had severe anemia, a subgroup that deserves closer follow-up in light of these data. Anemia was also significantly more common in our patients with coagulopathy (88.2% vs 61.5%), which fits with the combined effect of reduced hepatic synthesis and ongoing blood loss.

Qamar et al., who followed 213 patients with compensated cirrhosis, showed that thrombocytopenia is usually the first cytopenia to appear and that baseline thrombocytopenia and leukopenia independently predicted death or liver transplantation after adjustment for hepatic venous pressure gradient and Child-Pugh score [15]. In our study, thrombocytopenia was present in 81.8% of leukopenic patients compared with 57.9% of those with normal leukocyte counts (p = 0.058). Sohail et al., in a multicentre South Asian series of 377 patients, found an overall thrombocytopenia prevalence of 64% and a similar gap between leukopenic and non-leukopenic patients (89.5% vs 52.5%; p = 0.003) [16]. Both studies point in the same direction and suggest hypersplenism as the common mechanism; our smaller sample is the most likely reason the difference fell just short of significance.

Gastrointestinal bleeding was about twice as common in our thrombocytopenic patients (32.5% vs 15.0%), but this difference was not significant. Biswas et al., analyzing 913 patients with acute variceal bleeding, observed that platelet count did not predict 42-day rebleeding (HR 0.999; p = 0.360) and that rebleeding rates were similar across platelet strata [17]. Variceal bleeding depends mainly on portal pressure rather than on platelet number, which may explain why the platelet count by itself is a weak marker of bleeding risk.

Several groups have reported larger platelets in cirrhosis. Suvak et al. recorded a mean MPV of 8.37 fL in cirrhotic patients against 7.88 fL in controls (p <0.001), with higher values in those with ascitic fluid infection (8.79 vs 8.05 fL) [18]. Karaman et al. found MPV to be higher in chronic hepatitis C patients with advanced fibrosis than in those with milder fibrosis (9.67 vs 8.91 fL; p = 0.015) [19]. In a surgical cohort, Iida et al. noted a higher MPV in cirrhotic than in non-cirrhotic livers (10.8 vs 10.2 fL), and the MPV-to-platelet ratio identified cirrhosis with an area under the curve of 0.78 [20]. Mohamed et al. showed that MPV correlated with INR, bilirubin and MELD score (r = 0.424; p = 0.008) in 114 Egyptian patients [21]. Our finding that thrombocytopenia rose from 42.9% to 85.7% across MPV bands agrees with these reports. A likely explanation is that peripheral destruction and splenic pooling of platelets drive the marrow to release younger, larger cells. The rise in coagulopathy from 42.9% to 71.4% across MPV bands is consistent with the correlation between MPV and INR described by Mohamed et al. [21], although in our sample this trend was not statistically significant.

Whether MPV predicts outcome is far less certain. Oikonomou et al., studying 525 patients with stable decompensated cirrhosis, found that the MPV-to-platelet ratio was only borderline on univariate analysis (HR 3.75; p = 0.05) and lost significance after adjustment [22]. Giannini et al. observed no difference in MPV across Child-Pugh classes (p = 0.438) or MELD strata (p = 0.978), and MPV was almost identical in Child-Pugh C patients who died and

those who survived (9.15 vs 9.10 fL; p = 0.794) [23]. Reinthong et al. likewise found no correlation between MPV and mortality (r = –0.066) [24], and Erdogan et al. reported no meaningful difference in MPV between cirrhotic patients with and without variceal bleeding (7.9 vs 8.6 fL; p = 0.46) [25]. Taken together, these studies suggest that MPV reflects platelet turnover more reliably than it forecasts survival. Our results fit this view: MPV tracked the frequency of thrombocytopenia, but without follow-up data we could not test whether it predicts death or further decompensation.

Strengths and limitations: The study was prospective, used standard WHO-based definitions and focused only on decompensated patients, a group often mixed with compensated cases in earlier reports. Its main limitations are the small sample and single-centre design, which reduced the power to detect moderate associations. MPV was analyzed in categories rather than as a continuous value, and Child-Pugh and MELD scores, spleen size and endoscopic findings were not correlated with MPV. There was no control group and no longitudinal follow-up, so the prognostic role of MPV could only be inferred indirectly from its association with thrombocytopenia.

CONCLUSION

Anemia, thrombocytopenia and coagulopathy were common in patients with decompensated liver disease. Higher MPV was associated with a progressively greater frequency of thrombocytopenia, and coagulopathy showed a similar but non-significant trend. As an inexpensive parameter already reported with the routine blood count, MPV may help clinicians recognize patients with more marked platelet abnormalities. Larger multicentre studies that measure MPV as a continuous variable, include Child-Pugh and MELD scores and follow patients for clinical outcomes are needed before MPV can be accepted as a prognostic marker.

DECLARATIONS

Ethical approval: Approval was obtained from the Institutional Ethics Committee, A.C.S. Medical College and Hospital, Chennai, prior to the commencement of the study.

Informed consent: Written informed consent was obtained from all participants.

Conflict of interest: The authors declare no conflict of interest.

Funding: None.

Author contributions: BDK: conception, data collection, statistical analysis and drafting of the manuscript; KS: study design, supervision and critical revision; RR: data interpretation and critical review; CS: overall supervision and final approval. All authors read and approved the final manuscript.

Data availability: The data supporting this study are available from the corresponding author on reasonable request.

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