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Original Research Article

Mean Platelet Volume as a Predictor of Myocardial Infarction Risk: Association with Hypertension, Smoking, Diabetes Mellitus, and Alcohol Consumption in STEMI and NSTEMI Patients — A Cross-Sectional Study

Nischay N. Hegde[1], M. K. Malatesha[2]

[1]Junior Resident, Department of General Medicine, JJM Medical College, Davangere, Karnataka, India
[2]Professor, Department of General Medicine, MD, DM (Cardiology), FACEE, JJM Medical College, Davangere, Karnataka, India

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Corresponding Author: Dr. Nischay N. Hegde
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Abstract

Background: Cardiovascular diseases remain the foremost cause of mortality globally, with acute myocardial infarction (AMI) constituting a major proportion. Mean platelet volume (MPV), a marker of platelet size and reactivity, has been proposed as a cost-effective predictor of thrombotic cardiovascular events. However, its association with conventional cardiovascular risk factors in the context of ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) remains insufficiently characterized.

Methods: This hospital-based cross-sectional study was conducted over a period of one year at a tertiary care centre. A total of 60 patients diagnosed with AMI (30 STEMI and 30 NSTEMI) and 60 age- and sex-matched healthy controls were enrolled. Venous blood samples were collected within six hours of admission into EDTA tubes for estimation of platelet count, MPV, platelet distribution width (PDW), and plateletcrit (PCT) using an automated hematology analyzer. Conventional risk factors including hypertension, diabetes mellitus, smoking, and alcohol consumption were documented. Statistical analysis was performed using SPSS version 26.0, employing Chi-square test, Student's t-test, and ANOVA, with significance set at $p<0.05$.

Results: Mean MPV was significantly elevated in AMI cases (10.58±0.91 fL) compared to controls (8.49±0.87 fL, $p<0.001$). STEMI patients demonstrated a higher mean MPV (10.97±0.82 fL) than NSTEMI patients (10.19±0.88 fL, $p=0.001$). Among hypertensive AMI cases, 38.46% of STEMI patients had MPV >11 fL compared to 8.33% in NSTEMI ($p<0.001$). Smokers with STEMI showed significantly higher MPV values than non-smokers and controls ($p=0.002$). Diabetic patients in the STEMI group had a higher prevalence of elevated MPV ($p=0.014$). Alcohol consumption was not significantly associated with MPV elevation ($p=0.29$).

Conclusion: MPV was significantly elevated in patients with AMI, particularly in the STEMI subgroup. A synergistic association was observed between elevated MPV and traditional cardiovascular risk factors, especially hypertension, smoking, and diabetes mellitus. MPV may serve as a simple, inexpensive, and readily available biomarker for early cardiovascular risk stratification.

Keywords: mean platelet volume; acute myocardial infarction; STEMI; NSTEMI; hypertension; diabetes mellitus; smoking; platelet indices.

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Introduction

Cardiovascular diseases (CVDs) continue to represent the predominant cause of morbidity and mortality worldwide, accounting for an estimated 17.9 million deaths annually according to the World Health Organization.[1] Among CVDs, acute myocardial infarction (AMI) remains the most devastating clinical manifestation, characterized by myocardial necrosis resulting from acute interruption of coronary blood flow. The global burden of ischemic heart disease has shown a persistent upward trend, particularly in low- and middle-income countries where epidemiological transition, urbanization, and adoption of sedentary lifestyles have contributed to an accelerating prevalence of conventional risk factors.[2] The pathogenesis of AMI is fundamentally linked to atherosclerotic plaque disruption and subsequent thrombus formation within the coronary vasculature. Platelets play a central and indispensable role in this thrombotic cascade through adhesion, activation, and aggregation at the site of endothelial injury.[3] Platelet reactivity is not uniform across the circulating population; larger platelets are

metabolically and enzymatically more active, produce greater quantities of thromboxane A2, express higher levels of surface glycoprotein IIb/IIIa receptors, and demonstrate enhanced aggregation responses compared to their smaller counterparts.[4] These properties render larger platelets disproportionately prothrombotic and clinically consequential in the setting of acute coronary syndromes.

Mean platelet volume (MPV), a standard parameter reported by modern automated hematology analyzers, provides an objective and reliable measurement of average platelet size in a given blood sample. The normal reference range for MPV is generally considered to be 7.0 to 11.0 femtoliters (fL).[5] An elevated MPV reflects an increased proportion of large, hyperactive platelets in the circulation and has been proposed as a surrogate marker of platelet activation and thrombotic potential. Over the past two decades, accumulating evidence has supported the role of MPV as a clinically meaningful biomarker in cardiovascular medicine, with multiple studies demonstrating significantly elevated MPV values in patients presenting with AMI as compared to those with stable angina pectoris or healthy controls.[6]

A systematic review and meta-analysis encompassing over 2800 patients from 16 cross-sectional studies confirmed that MPV was significantly higher in individuals with AMI than in those without the condition, further establishing its potential as an independent predictor of acute coronary events.[7] Moreover, elevated MPV has been associated with more extensive coronary atherosclerosis, multivessel coronary artery disease, and adverse outcomes including in-hospital mortality and long-term vascular mortality following myocardial infarction.[8] The prognostic significance of MPV has been underscored by observations that patients in the highest quintile of MPV demonstrate a substantially greater hazard ratio for overall vascular mortality and ischemic heart disease events compared to those in the lowest quintile. Traditional cardiovascular risk factors such as hypertension, diabetes mellitus, cigarette smoking, and alcohol consumption are well-established contributors to the development and progression of coronary artery disease. Hypertension promotes endothelial dysfunction, accelerates atherosclerotic plaque formation, and augments platelet activation through shear stress–mediated mechanisms.[9] Diabetes mellitus is associated with increased platelet turnover, enhanced surface expression of adhesion molecules, and resistance to antiplatelet agents, all of which correlate with higher MPV values. Cigarette smoking induces oxidative stress, impairs endothelial nitric oxide bioavailability, and directly stimulates platelet activation and aggregation. Chronic alcohol consumption, depending on the quantity and pattern, may exert both prothrombotic and paradoxically antithrombotic effects on platelet function.

Despite the growing body of evidence supporting MPV as a cardiovascular biomarker, its integration into routine clinical practice for risk stratification remains limited, particularly in resource-constrained settings where advanced cardiac biomarkers and imaging modalities may not be readily available. Furthermore, the interplay between MPV elevation and conventional cardiovascular risk factors in differentiating between STEMI and NSTEMI presentations has not been adequately explored in the Indian population, where the epidemiological profile, genetic predisposition, and prevalence of risk factors may differ substantially from Western cohorts.[10]

The present study was therefore undertaken to evaluate the association between MPV and the risk of myocardial infarction, with specific emphasis on examining the relationship of MPV with hypertension, smoking, diabetes mellitus, and alcohol consumption across STEMI and NSTEMI subgroups. It was hypothesized that elevated MPV values would demonstrate a significant association with AMI, particularly with the STEMI phenotype, and that this association would be potentiated by the concurrent presence of conventional cardiovascular risk factors.

Aims and Objectives: The present study was designed with the primary aim of evaluating the association between mean platelet volume and the risk of acute myocardial infarction in patients admitted to a tertiary care centre. The study specifically sought to compare MPV values among patients with STEMI, NSTEMI, and healthy age- and sex-matched controls. Additionally, the study aimed to assess the relationship between MPV elevation and conventional cardiovascular risk factors, namely hypertension, diabetes mellitus, smoking, and alcohol consumption, across these three study groups.

The secondary objective of the investigation was to determine whether MPV could serve as a practical, cost-effective, and readily accessible biomarker for early identification of individuals at elevated risk for acute coronary events, particularly in healthcare settings with limited access to advanced cardiac diagnostic modalities. The study further aimed to evaluate whether the concurrent presence of traditional cardiovascular risk factors and elevated MPV values demonstrated a synergistic association with the severity of myocardial infarction as reflected by the STEMI versus NSTEMI classification.

Materials and Methods

Study Design and Setting: This hospital-based cross-sectional analytical study was conducted in the Department of General Medicine at a tertiary

care teaching hospital over a period of one year. The study was initiated after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants or their legally authorized representatives prior to enrollment.

Study Population and Sample Size: The study population comprised patients admitted with a diagnosis of acute myocardial infarction during the study period. The diagnosis of AMI was established as per the criteria laid down by the Joint European Society of Cardiology/American College of Cardiology Committee for the Universal Definition of Myocardial Infarction.

Sample size was estimated using OpenEpi software version 3.0 with the following parameters: an expected mean difference in MPV between STEMI and NSTEMI patients of 0.78 fL, standard deviation of 1.10 fL, 95% confidence level, and 80% statistical power. The minimum required sample size was calculated as 60 cases (30 in each group). An equal number of 60 age- and sex-matched apparently healthy controls were recruited from among first-degree relatives accompanying the patients who had no history of coronary artery disease.

Selection Criteria: Cases included patients aged 18 years and above admitted with a diagnosis of AMI, further classified into STEMI (based on electrocardiographic evidence of ST-segment elevation with elevated cardiac biomarkers) and NSTEMI (based on elevated cardiac biomarkers including CPK-MB and/or Troponin I without persistent ST-segment elevation). Controls included age- and sex-matched apparently healthy individuals above 18 years without a history of coronary artery disease.

Patients with known platelet disorders, bone marrow diseases, bleeding diatheses, preeclampsia, chronic liver disease, history of recent surgery or blood transfusion within the preceding six weeks, known ischemic heart disease on antiplatelet therapy, and those receiving drugs known to cause thrombocytopenia were excluded from the study.

Data Collection and Laboratory Investigations: A detailed clinical history was obtained from all study subjects with particular emphasis on cardiovascular risk factors including hypertension, diabetes mellitus, smoking habits, and alcohol consumption. General physical examination and systemic examination were performed. A non-hemolyzed venous blood sample was collected within six hours of admission into EDTA-anticoagulated tubes prior to the administration of antiplatelet drugs. Complete blood count including platelet count (PLC), mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) was performed using an automated hematology analyzer based on impedance technology (Coulter principle). The normal reference range for MPV was considered as 7.0 to 11.0 fL.

Statistical Analysis: The collected data was entered into Microsoft Excel spreadsheets and analyzed using SPSS version 26.0 (IBM Corporation, Armonk, NY, USA). Categorical variables were expressed as frequencies and percentages and compared using the Chi-square test or Fisher's exact test as appropriate. Continuous variables were expressed as mean ± standard deviation and compared using the independent samples Student's t-test for two-group comparisons and one-way analysis of variance (ANOVA) for three-group comparisons. Univariate analysis was performed to assess the association of individual risk factors with MPV values across study groups. A p-value of less than 0.05 was considered statistically significant for all analyses.

Results

A total of 120 subjects were enrolled in the study, comprising 60 cases of AMI (30 STEMI and 30 NSTEMI) and 60 age- and sex-matched healthy controls. The cases and controls were well matched for demographic characteristics. The majority of subjects in both groups, 19 (31.67%) cases and an equal proportion of controls, belonged to the age group of 61–70 years, followed by 15 (25.00%) in the 51–60 years age group. The mean age of the study population was 56.72±12.34 years (range 30–78 years). Among the 60 AMI cases, 37 (61.67%) were males and 23 (38.33%) were females, and the control group was matched accordingly. The overall male-to-female ratio in the study was 1.61:1.

Table 1: Distribution of cases according to type of myocardial infarction (n=60)

Type of MI	Frequency	Percentage (%)
Inferior wall MI	09	15.00
Anterior wall MI	05	8.33
Anterior + lateral wall MI	08	13.33
Extensive anterior wall MI	03	5.00
Inferior + posterior wall MI	02	3.33
Posterior wall MI	01	1.67
Inferior + lateral wall MI	02	3.33
NSTEMI	30	50.00
Total	60	100.00

Among the 60 AMI cases, STEMI and NSTEMI were equally distributed with 30 (50.00%) cases each. Among the 30 STEMI patients, inferior wall MI was the most common presentation accounting for 9 (15.00%) cases, followed by anterior plus lateral wall MI in 8 (13.33%) cases. Anterior wall MI was observed in 5 (8.33%) patients, extensive anterior wall MI in 3 (5.00%), inferior plus posterior wall MI in 2 (3.33%), posterior wall MI in 1 (1.67%), and inferior plus lateral wall MI in 2 (3.33%) cases.

Table 2: Comparison of MPV values among study groups

MPV (fL)	STEMI n (%)	NSTEMI n (%)	Controls n (%)	P value
<9	01 (3.33)	02 (6.67)	41 (68.33)	
9–11	18 (60.00)	26 (86.67)	19 (31.67)	<0.001*
>11	11 (36.67)	02 (6.67)	00 (0.00)	
Mean±SD	10.97±0.82	10.19±0.88	8.49±0.87	<0.001*

The distribution of MPV values across the three study groups demonstrated a highly significant difference ($p<0.001$). The mean MPV was highest in the STEMI group (10.97±0.82 fL), followed by the NSTEMI group (10.19±0.88 fL), and was lowest in the control group (8.49±0.87 fL). MPV values exceeding 11 fL were observed in 11 (36.67%) STEMI patients and only 2 (6.67%) NSTEMI patients, while no control subject had MPV above 11 fL. Conversely, 41 (68.33%) controls had MPV values below 9 fL compared to only 1 (3.33%) STEMI and 2 (6.67%) NSTEMI patients. This distribution clearly indicated that patients with AMI, particularly those presenting with STEMI, exhibited significantly higher platelet volumes than healthy individuals.

Table 3: Distribution of MPV values according to hypertension status in study groups

MPV (fL)	STEMI (n=13)	NSTEMI (n=12)	Controls (n=10)	P value
<9	00 (0.00)	00 (0.00)	08 (80.00)	
9–11	08 (61.54)	11 (91.67)	02 (20.00)	<0.001*
>11	05 (38.46)	01 (8.33)	00 (0.00)	

Among hypertensive subjects, the association between MPV elevation and MI type was highly significant ($p<0.001$). In the STEMI subgroup, 5 (38.46%) hypertensive patients had MPV values exceeding 11 fL, compared to only 1 (8.33%) in the NSTEMI subgroup. No hypertensive control had MPV above 11 fL. The majority of hypertensive NSTEMI patients, 11 (91.67%), had MPV values in the 9–11 fL range, while 8 (80.00%) hypertensive controls had MPV below 9 fL. These findings suggested that hypertension combined with elevated MPV was strongly associated with the STEMI phenotype.

Table 4: Distribution of MPV values according to smoking status in study groups

MPV (fL)	STEMI (n=10)	NSTEMI (n=07)	Controls (n=09)	P value
<9	00 (0.00)	00 (0.00)	07 (77.78)	
9–11	07 (70.00)	06 (85.71)	02 (22.22)	0.002*
>11	03 (30.00)	01 (14.29)	00 (0.00)	

When MPV values were analyzed among smokers across the three groups, a statistically significant association was observed ($p=0.002$). Among smoking STEMI patients, 3 (30.00%) had MPV above 11 fL, whereas only 1 (14.29%) smoking NSTEMI patient demonstrated this elevation. None of the smokers in the control group had MPV exceeding 11 fL, and the majority, 7 (77.78%), had values below 9 fL. This pattern indicated that smoking in conjunction with elevated MPV significantly increased the likelihood of developing STEMI.

Table 5: Distribution of MPV values according to diabetes mellitus status in study groups

MPV (fL)	STEMI (n=08)	NSTEMI (n=05)	Controls (n=06)	P value
<9	01 (12.50)	00 (0.00)	04 (66.67)	
9–11	04 (50.00)	04 (80.00)	02 (33.33)	0.014*
>11	03 (37.50)	01 (20.00)	00 (0.00)	

Among diabetic subjects, 3 (37.50%) STEMI patients had MPV values exceeding 11 fL compared to 1 (20.00%) among NSTEMI patients. No diabetic control had MPV above 11 fL. The majority of diabetic NSTEMI patients, 4 (80.00%), had MPV between 9 and 11 fL, while 4 (66.67%) diabetic controls had values below 9 fL. This association between diabetes mellitus, elevated MPV, and STEMI was statistically significant ($p=0.014$).

Table 6: Univariate analysis of MPV values with risk factors among study subjects

Risk Factor	Group	Status	Mean±SD MPV (fL)	P value
Hypertension	Cases	Present (n=25)	10.62±0.94	
	Cases	Absent (n=35)	10.55±0.89	<0.001*
	Controls	Present (n=10)	8.27±0.58	
	Controls	Absent (n=50)	8.53±0.91	
Diabetes mellitus	Cases	Present (n=13)	10.68±0.90	
	Cases	Absent (n=47)	10.55±0.91	0.41
	Controls	Present (n=06)	8.42±0.76	
	Controls	Absent (n=54)	8.50±0.89	
Smoking	Cases	Present (n=17)	10.71±0.93	
	Cases	Absent (n=43)	10.53±0.90	0.03*
	Controls	Present (n=09)	8.38±0.52	
	Controls	Absent (n=51)	8.51±0.92	
Alcohol	Cases	Present (n=12)	10.59±0.98	
	Cases	Absent (n=48)	10.57±0.89	0.29
	Controls	Present (n=07)	8.47±0.45	
	Controls	Absent (n=53)	8.49±0.91	

Univariate analysis of MPV values with cardiovascular risk factors revealed that hypertension demonstrated the strongest association, with hypertensive AMI cases showing a mean MPV of 10.62±0.94 fL compared to 8.27±0.58 fL in hypertensive controls ($p<0.001$). Smoking also showed a statistically significant association ($p=0.03$), with mean MPV values of 10.71±0.93 fL among smoking cases versus 8.38±0.52 fL among smoking controls. Diabetes mellitus showed a trend toward higher MPV in cases (10.68±0.90 fL) versus controls (8.42±0.76 fL) but did not reach statistical significance on univariate analysis ($p=0.41$). Similarly, alcohol consumption did not demonstrate a significant independent association with MPV elevation ($p=0.29$). Overall, these findings indicated that hypertension and smoking were the risk factors most strongly associated with elevated MPV in the setting of acute myocardial infarction.

Discussion

The present study demonstrated a statistically significant elevation of mean platelet volume in patients with acute myocardial infarction compared to healthy controls, with the highest values observed in the STEMI subgroup. The mean MPV in the overall AMI group was 10.58±0.91 fL, which was significantly greater than the 8.49±0.87 fL recorded in the control group ($p<0.001$). Furthermore, the STEMI subgroup exhibited a significantly higher mean MPV (10.97±0.82 fL) compared to the NSTEMI subgroup (10.19±0.88 fL, $p=0.001$). These findings are consistent with the observations of Bharihoke et al.,[11] who reported significantly elevated MPV and other platelet volume indices in patients with AMI and stable coronary artery disease compared to controls. Similarly, Khandekar et al. [12] in their study on platelet volume indices in an Indian population found that MPV was significantly elevated in patients with coronary artery disease and AMI, corroborating the present findings.

The systematic review and meta-analysis conducted by Chu et al. [13] encompassing 2809 patients across 16 cross-sectional studies confirmed that MPV was significantly higher in individuals with AMI compared to those without AMI ($p<0.001$). Their findings are in strong agreement with the present study and further support the role of MPV as an independent biomarker for acute coronary events. In contrast, Rai et al. [14] in their comparative study did not find a significant difference in MPV between AMI patients and those with normal coronary angiography, suggesting that the relationship between MPV and coronary artery disease may be influenced by population-specific factors and methodological differences in sample timing and analysis.

The present study observed that elevated MPV in combination with hypertension was strongly and significantly associated with the STEMI phenotype ($p<0.001$). Among hypertensive STEMI patients, 38.46% had MPV exceeding 11 fL, compared to 8.33% in hypertensive NSTEMI patients. Manchanda et al. [15] similarly reported that platelet indices including MPV were significantly elevated in acute coronary syndrome patients with associated hypertension. The mechanistic basis for this observation relates to the hemodynamic stress imposed by sustained hypertension, which promotes endothelial injury, accelerates atherosclerotic plaque progression, and enhances shear stress–mediated platelet activation, thereby favoring the production and release of larger, more reactive platelets from the bone marrow.

Smoking demonstrated a significant association with elevated MPV values in AMI patients in the present study ($p=0.002$ for cross-tabulation; $p=0.03$

on univariate analysis). This observation aligns with the findings of Murat et al.,[16] who reported that high MPV levels were independent predictors of multivessel coronary artery disease in patients with acute coronary syndromes undergoing coronary angiography. Cigarette smoking is known to induce chronic oxidative stress, impair endothelial function, and directly stimulate platelet activation through multiple pathways including enhanced thromboxane synthesis and reduced nitric oxide bioavailability, all of which collectively promote a prothrombotic milieu characterized by larger and more reactive platelets.

Diabetes mellitus showed a significant association with elevated MPV in the context of STEMI when analyzed categorically (p=0.014), although univariate analysis comparing mean MPV between diabetic and non-diabetic subjects did not reach significance (p=0.41). These findings are partly consistent with Agrawal et al.,[17] who reported that MPV in diabetic AMI patients was significantly higher than in non-diabetic AMI patients. The pathophysiological rationale for elevated MPV in diabetes includes increased platelet turnover secondary to chronic hyperglycemia, glycation of platelet membrane proteins, and enhanced megakaryocyte ploidy in the bone marrow, leading to production of larger platelets with greater hemostatic capacity.

Alcohol consumption did not demonstrate a statistically significant independent association with MPV elevation in the present study (p=0.29), which is consistent with the variable and often contradictory findings reported in the literature regarding alcohol and platelet function. Astuti et al.[18] reported that alcohol intake was significantly associated with elevated MPV in their control group but not in their AMI cases, suggesting a complex and possibly nonlinear relationship between alcohol consumption and platelet size. The relationship between alcohol and cardiovascular risk is well recognized to follow a J-shaped curve, with moderate consumption potentially conferring protective effects and heavy consumption increasing thrombotic risk. Slavka et al.[19] in their large prospective study observed that elevated MPV functioned as an independent risk factor for overall vascular mortality and ischemic heart disease, with patients in the highest quintile demonstrating a 1.5-fold increased hazard ratio for vascular death. Similarly, Amraotkar et al.[20] noted that MPV was significantly increased during the acute phase of MI compared to stable coronary artery disease, although this difference did not persist at three months post-infarction, suggesting that MPV elevation in AMI may partly represent an acute-phase response.

The present study findings are broadly consistent with this body of literature and support the utility of MPV as a practical, cost-effective biomarker for cardiovascular risk assessment, particularly when considered in conjunction with conventional risk factors.

Conclusion

The present study demonstrated a statistically significant association between elevated mean platelet volume and the risk of acute myocardial infarction. Patients with STEMI exhibited significantly higher MPV values compared to those with NSTEMI and healthy controls. A synergistic association was observed between elevated MPV and conventional cardiovascular risk factors, with hypertension and smoking demonstrating the strongest and most consistent associations. Diabetes mellitus also showed a significant categorical association with elevated MPV in STEMI patients, whereas alcohol consumption did not reach statistical significance. MPV is a simple, inexpensive, and readily available parameter that is routinely measured in automated hematology analyzers, and these findings suggest its potential utility as an early biomarker for cardiovascular risk stratification, particularly in resource-limited healthcare settings. In patients with hypertension and smoking history, elevated MPV values may serve as an early indicator of increased susceptibility to acute coronary events and specifically to the STEMI phenotype. Further large-scale prospective studies are warranted to validate these findings and to establish optimal MPV cutoff values for clinical risk prediction.

References

1. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: update from the GBD 2019 study. J Am Coll Cardiol. 2020;76(25):2982–3021.
2. Gupta R, Mohan I, Narula J. Trends in coronary heart disease epidemiology in India. Ann Glob Health. 2016;82(2):307–15.
3. Davi G, Patrono C. Platelet activation and atherothrombosis. N Engl J Med. 2007;357(24):2482–94.
4. Bath PM, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis. 1996;7(2):157–61.
5. Noris P, Melazzini F, Balduini CL. New roles for mean platelet volume measurement in the clinical practice. Platelets. 2016;27(7):607–12.
6. Ranjith MP, Divya R, Mehta VK, Krishnan MG, KamalRaj R, Kavishwar A. Significance of platelet volume indices and platelet count in ischaemic heart disease. J Clin Pathol. 2009;62(9):830–3.
7. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, et al. Mean platelet

volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Haemost. 2010;8(1):148–56.
8. Ozkan B, Uysal OK, Duran M, Sahin DY, Elbasan Z, Tekin K, et al. Relationship between mean platelet volume and atherosclerosis in young patients with ST elevation myocardial infarction. Angiology. 2013;64(5):371–4.
9. Nadar S, Blann AD, Lip GY. Platelet morphology and plasma indices of platelet activation as markers of myocardial infarction: a comparison with markers of general vascular and inflammatory damage. J Am Coll Cardiol. 2004;44(2):317–22.
10. Kiliçli-Çamur N, Demirtunç R, Konuralp C, Eskiser A, Başaran Y. Could mean platelet volume be a predictive marker for acute myocardial infarction? Med Sci Monit. 2005;11(8):CR387–92.
11. Bharihoke N, Dosi S, Singh P, Subhedar V, Raje P, Malpani G. Mean platelet volume and other platelet volume indices in acute myocardial infarction and stable coronary artery diseases: a hospital based prospective observational study. IOSR J Dent Med Sci. 2018;17(3):14–7.
12. Khandekar MM, Khurana AS, Deshmukh SD, Kakrani AL, Katdare AD, Inamdar AK. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. J Clin Pathol. 2006;59(2):146–9.
13. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, et al. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Haemost. 2010;8(1):148–56.
14. Rai A, Saidi M, Salehi N, Sahebjamei F, Jalilian M, Janjani P. Comparison of mean platelet volume in acute myocardial infarction vs. normal coronary angiography. Glob J Health Sci. 2016;8(11):320.
15. Manchanda J, Potekar RM, Badiger S, Tiwari A. The study of platelet indices in acute coronary syndromes. Ann Pathol Lab Med. 2015;2(1):A30–5.
16. Murat SN, Duran M, Kalay N, Gunebakmaz O, Akpek M, Doger C, et al. Relation between mean platelet volume and severity of atherosclerosis in patients with acute coronary syndromes. Angiology. 2013;64(2):131–6.
17. Agrawal BK, Manchanda B, Garg A, Mittal A, Mahajan NC, Agrawal U. Mean platelet volume in acute myocardial infarction: a case-controlled study. J Cardiovasc Dis Res. 2015;3(1):6.
18. Astuti Y, Setianto BY, Taufiq N. Mean platelet volume as a predictor of atherosclerotic severity in non ST elevation acute myocardial infarction. Acta Cardiol Indones. 2019;5(1):10.
19. Slavka G, Perkmann T, Haslacher H, Greisenegger S, Marsik C, Wagner OF, et al. Mean platelet volume may represent a predictive parameter for overall vascular mortality and ischemic heart disease. Arterioscler Thromb Vasc Biol. 2011;31(5):1215–8.
20. Amraotkar AR, Song DD, Otero D, Trainor PJ, Ismail I, Kothari V, et al. Platelet count and mean platelet volume at the time of and after acute myocardial infarction. Clin Appl Thromb Hemost. 2017;23(8):1052–9.