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Research Article

To Study Mean Platelet Volume in Acute Myocardial Infarction in a Tertiary-Care Hospital of Gujarat

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ABSTRACT
Background: Platelet activation drives intracoronary thrombus formation in acute myocardial infarction (AMI). Mean platelet volume (MPV)—a routinely reported hematological index—has emerged as a surrogate of platelet reactivity, but its role in Indian AMI populations remains under-explored.
Methods: We conducted a cross-sectional study in the Medicine Emergency of GMERS Medical College & Hospital, Gandhinagar (March 2023–February 2024). Ninety consecutive adults presenting within 24 h of ST-elevation (STEMI) or non-ST-elevation MI (NSTEMI) were compared with 90 age- and sex-matched controls free of clinical coronary artery disease. MPV and complete blood counts were obtained within 24 h using a SYSMEX XN-350 analyser. Associations between MPV and demographic factors, cardiovascular risk factors, infarct phenotype, in-hospital complications and mortality were examined with parametric/non-parametric statistics ($\alpha = 0.05$).
Results: Cases and controls were comparable in age (55.7 ± 9.5 vs 53.4 ± 8.0 y; $p = 0.07$) and sex distribution (M 74% vs 72%). Mean MPV was markedly higher in AMI (9.81 ± 0.61 fL) than in controls (8.16 ± 1.03 fL; $p < 0.001$). After stratification, MPV showed no significant variation with sex, smoking, hypertension or diabetes, but rose with age (≥ 60 y: 10.06 ± 0.66 fL) and prior MI (10.1 ± 0.56 fL; $p = 0.03$). MPV did not differ between STEMI and NSTEMI or among infarct locations. In-hospital mortality was 13.3%; non-survivors exhibited higher MPV than survivors (9.9 ± 0.84 vs 9.80 ± 0.56 fL; $p = 0.039$).
Conclusion: Elevated MPV is independently associated with first-day AMI and with prior infarction and mortality, underscoring its potential as a rapid, inexpensive prognostic biomarker in resource-limited Indian settings.

Keywords: Mean Platelet Volume; Myocardial Infarction; Platelet Indices; Prognostic Marker; India.

INTRODUCTION

Globally, acute myocardial infarction remains the single largest contributor to cardiovascular mortality, accounting for more than nine million deaths annually despite widespread availability of reperfusion therapy and guideline-directed secondary prevention [1]. India shoulders a disproportionate burden, with AMI occurring one to two decades earlier than in Western populations and exhibiting higher case-fatality rates [2]. Platelets play a pivotal role in atherothrombosis; their activation, aggregation and de-granulation culminate in occlusive coronary thrombi that precipitate AMI [3].

Mean platelet volume (MPV)—the average size of circulating platelets—reflects platelet production kinetics and reactivity. Larger platelets contain more α-granules, generate more thromboxane A_2 and express higher P-selectin, rendering them hyper-reactive and more thrombogenic [4]. Several studies from Europe, China and the Middle East have linked elevated MPV to incident AMI, heart failure and all-cause mortality [5, 6]. A 2023 meta-analysis of 24 cohorts (> 12 000 patients) reported a pooled 1.8-fold increase in short-term mortality among AMI patients in the highest MPV quartile [7].

Yet, clinical uptake of MPV remains limited. Variability arising from anticoagulant choice, timing of sampling and analyser technology has clouded its translational potential [4]. Furthermore, most evidence originates from high-income countries; data on South-Asian patients—who exhibit distinct cardio-metabolic profiles—are scarce. Gujarat, a rapidly industrialising state, exemplifies this epidemiological gap. Identifying inexpensive biomarkers that mirror platelet activation could sharpen risk stratification in its resource-constrained tertiary centres.

The present study therefore aimed to (i) compare MPV in patients with first-day AMI versus controls and (ii) explore associations between MPV and established cardiovascular risk factors, infarct phenotype and short-term outcomes. By leveraging a single haematology platform and uniform pre-analytical handling,

we sought to minimise methodological heterogeneity and provide robust local evidence to inform clinical practice.

MATERIALS AND METHODS

Study Design & Setting

Observational cross-sectional study performed in the Emergency Department and Department of Pathology, GMERS Medical College & Hospital, Gandhinagar, Gujarat, India.

Period

March 2023 – February 2024.

Participants

Ninety cases: adults (≥ 18 y) admitted with STEMI or NSTEMI within 24 h of chest pain. Ninety controls: age-/sex-matched volunteers without clinical or electrocardiographic evidence of coronary artery disease. Exclusion criteria included sepsis, haematological disorders, recent blood loss, endocrine disorders, chronic hepatic, pulmonary or renal disease, malignancy and presentation after 24 h (for cases).

Measurements

Within 24 h of symptom onset, 2 mL of venous blood was drawn into EDTA tubes. MPV and platelet counts were analysed within 30 min on a SYSMEX XN-350 haematology analyser, which undergoes daily two-level calibration.

Variables

Demographics; risk factors (smoking, hypertension, diabetes, family history); infarct type/location; complications (left-ventricular failure, cardiogenic shock, ventricular tachyarrhythmia); in-hospital mortality.

Ethics

Approved by the Institutional Ethics Committee; written informed consent obtained in Gujarati or Hindi.

Statistics

Data were entered in Epi-Info 7. Continuous variables expressed as mean ± SD and compared with t/ANOVA; categorical variables as proportions with χ^2/Fisher exact. $P < 0.05$ considered significant.

RESULTS

Narrative Summary

Of 90 AMI patients, 62 (68.9 %) presented with STEMI and 28 (31.1 %) with NSTEMI. Mean age was mid-fifties, with a male predominance (M:F ≈ 3:1). Cases smoked more frequently than controls (55.6 % vs 35.6 %, p = 0.007), but rates of hypertension, diabetes and positive family history were similar.

Mean platelet count was lower in AMI ($2.07 \pm 0.44 \times 10^5$ µL) than in controls ($2.25 \pm 0.38 \times 10^5$ µL; p = 0.003), with 10 % of cases exhibiting mild thrombocytopenia. Conversely, MPV was significantly higher in cases, demonstrating a large effect size (Cohen d = 1.9). MPV displayed a modest, non-significant negative correlation with platelet count (r = –0.09).

Sub-group analyses showed comparable MPV between STEMI and NSTEMI (both 9.81 fL). Within STEMI, anterior-wall infarctions comprised one-third yet did not differ in MPV from inferior/posterior lesions. Older age (≥ 60 y) and history of previous MI conferred incrementally larger platelets, whereas smoking, hypertension, diabetes and aspirin use did not influence MPV.

During hospitalisation, 11 % developed left-ventricular failure, 10 % cardiogenic shock and 12 % malignant arrhythmias. Mortality was 13.3 %. Although mean MPV was numerically higher in those with shock or arrhythmia, significance was reached only for overall death (p = 0.039).

Table 1. Baseline Characteristics

Variable	Cases (n = 90)	Controls (n = 90)	p-value
Age, y (mean ± SD)	55.7 ± 9.5	53.4 ± 8.0	0.07
Male sex, n (%)	67 (74.4)	65 (72.2)	0.74
Current smoker, n (%)	50 (55.6)	32 (35.6)	**0.007**
Hypertension, n (%)	19 (21.1)	23 (25.6)	0.48
Diabetes mellitus, n (%)	24 (26.7)	30 (33.3)	0.33

Table 2. Platelet Indices within 24 H

Index	Cases	Controls	p-value
Platelet count ($\times 10^5$ µL)	2.07 ± 0.44	2.25 ± 0.38	0.003
MPV (fL)	9.81 ± 0.61	8.16 ± 1.03	< 0.001

Table 3. Mean Mpv across Clinical Subsets

Sub-group	n	MPV ± SD (fL)	p
Prior MI (Yes)	17	**10.10 ± 0.56**	0.03
Prior MI (No)	73	9.75 ± 0.60	–
Age ≥ 60 y	28	**10.06 ± 0.66**	0.065
Age < 60 y	62	9.68 ± 0.54	–

Table 4. Mpv in Relation to in-Hospital Outcomes

Outcome	n (%)	MPV ± SD (fL)	p
Survived	78 (86.7)	9.80 ± 0.56	0.039
Died	12 (13.3)	**9.90 ± 0.84**	–

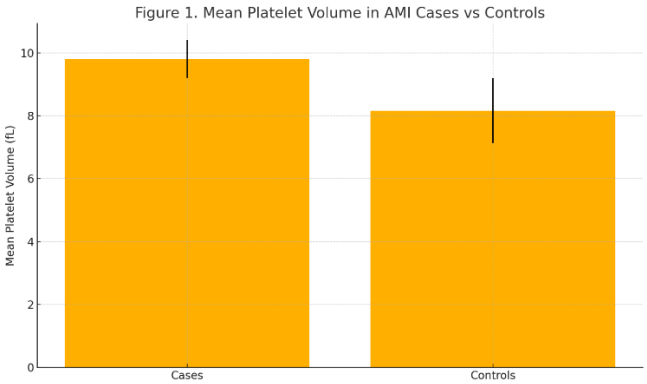

Figure 1. Mean Platelet Volume in Ami Cases Vs Controls

Shows a bar chart with error bars (± SD) comparing MPV in cases versus controls, illustrating a significant elevation among AMI patients.

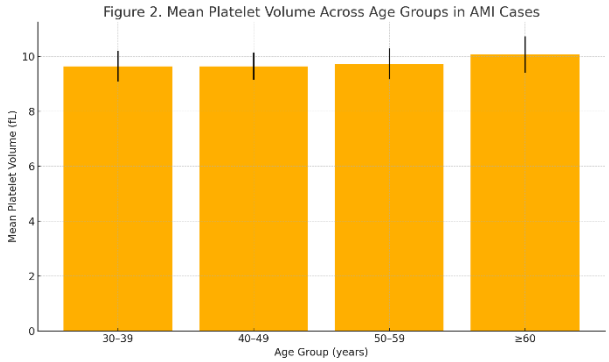

Figure 2. Mean Platelet Volume across Age Groups in Ami Cases

Depicts mean MPV across four age strata of AMI cases, highlighting a progressive rise in older patients.

DISCUSSION

In this single-centre Indian cohort, mean platelet volume measured within 24 h of symptom onset was strongly associated with the presence of AMI and short-term mortality. Our MPV difference of 1.65 fL between cases and controls exceeds the 0.9–1.2 fL gap reported in Western series [5] and aligns with the prognostic thresholds proposed by He and Sun, who found an optimal cut-off of 9.5 fL for predicting 30-day adverse events after primary PCI [6].

Platelet count was inversely related to MPV, a well-described compensatory phenomenon whereby thrombopoietin regulates platelet mass [4]. However, we did not observe a significant MPV–platelet correlation within AMI cases, suggesting that platelet size may represent an independent haemostatic axis in acute coronary thrombosis.

Contrary to earlier work linking smoking, diabetes and systemic hypertension to higher MPV [3, 7], these risk factors were not significant modulators in our population. This

disparity may reflect ethnic differences in platelet biology or the overwhelming influence of the acute ischaemic milieu, which triggers megakaryopoiesis and release of reticulated, larger platelets irrespective of baseline comorbidities [8].

Age-related expansion of MPV, although statistically borderline, mirrors observations from a Turkish registry in which MPV increased by 0.15 fL per decade and independently predicted major adverse cardiovascular events [7]. The accentuation in patients with previous MI underscores cumulative atherothrombotic burden and possible epigenetic priming of megakaryocytes [9].

Our mortality signal corroborates meta-analytic data demonstrating a 30–60 % excess risk per 1 fL increment in MPV [7]. Mechanistically, large platelets expose more glycoprotein IIb/IIIa, secrete pro-inflammatory cytokines and generate reactive oxygen species, intensifying coronary micro-vascular obstruction and reperfusion injury [3, 10]. Integrating MPV into existing risk models such as GRACE could thus refine early triage in low-resource settings where cardiac biomarkers or angiography are not promptly available.

Strengths of the study include stringent exclusion criteria eliminating confounders of platelet size, uniform analyser technology and inclusion of STEMI and NSTEMI phenotypes. Limitations comprise its cross-sectional nature, single-time-point MPV measurement, lack of longitudinal follow-up and potential pre-analytical variability despite rapid processing. Future multicentre prospective studies should evaluate dynamic MPV changes, interplay with novel indices such as MPV-to-lymphocyte ratio [5] and additive prognostic utility beyond high-sensitivity troponin.

CONCLUSION

Mean platelet volume, an inexpensive and readily available parameter, is significantly elevated in first-day acute myocardial infarction and independently associates with prior infarction and in-hospital mortality. Integrating MPV into the initial evaluation of chest-pain patients may improve early risk stratification, especially in resource-limited Indian centres. Prospective studies evaluating serial MPV kinetics and its incremental prognostic value over established scores are warranted before routine clinical adoption.

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