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STUDY OF MEAN PLATELET VOLUME WITH ACUTE MYOCARDIAL INFARCTION

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Abstract:
Introduction: AMI stays a leading cause of death worldwide. The relationship between Mean Platelet Volume (MPV) and Acute Myocardial Infarction (AMI) has been the focus of several studies, which suggest that elevated MPV levels correlate with worse clinical outcomes in AMI patients.
Materials & Methods: This cace control study was planned to investigate the association between Mean Platelet Volume (MPV) levels and clinical outcomes in AMI patients.
Results: The total sample size comprised 168 participants, with 84 cases (50%) and 84 controls (50%). The mean age of the participants was 57.70 ± 6.52 years, with cases having a mean age of 60.90 ± 6.58 years, and controls having a mean age of 54.50 ± 4.65 years. The platelet count in the case group (2.02 ± 0.47 x10^5/µL) was significantly lower than in the control group (2.49 ± 0.56 x10^5/µL), with a p-value of 0.000. Similarly, the Mean Platelet Volume (MPV) was significantly higher in the case group (11.83 ± 1.16 fL) compared to the control group (8.23 ± 0.49 fL), with a p-value of 0.000, indicating a significant difference.In terms of Myocardial Infarction (MI) type, the case group had a variety of diagnoses, including AWMI (45.24%), IWSTEMI (39.29%), and NSTEMI (15.48%), with all controls being normal (p-value = 0.000). ECG changes in the case group included ST elevation (STE) in various leads, such as 2, 3, AVF (39.29%), V1-V3, V2-V5, and V1-V6, while controls presented with normal sinus rhythm (p-value = 0.000).
Conclusion: In conclusion, the elevated MPV in patients with AMI highlights its potential as a novel and easily measurable biomarker for identifying individuals at risk for myocardial infarction and other thrombotic cardiovascular events.

Keywords: Platelet, Mean platelet Volume, Myocardial Infarction, Acute MI

INTRODUCTION

Acute Myocardial Infarction (AMI) is a clinical condition characterized by the death of myocardial cells due to a prolonged interruption in coronary blood flow. This typically results from the rupture of an atherosclerotic plaque, leading to the formation of a thrombus that occludes the coronary artery, thereby depriving the heart muscle of oxygen. AMI can result in irreversible damage to the heart muscle, leading to various complications such as heart failure and arrhythmias **[1]**.

AMI is classified into two types: ST-Elevation Myocardial Infarction (STEMI) and Non-ST-Elevation Myocardial Infarction (NSTEMI). STEMI is characterized by a significant elevation of the ST segment on an electrocardiogram (ECG), showing full-thickness myocardial infarction due to complete occlusion of the coronary artery. NSTEMI, however, lacks ST-segment elevation and is typically caused by partial occlusion or transient blockage of the coronary artery [**2]**. While STEMI is often more severe, NSTEMI still carries substantial risk for adverse outcomes.

Global Epidemiology of AMI

AMI stays a leading cause of death worldwide. According to the World Health Organization (WHO), cardiovascular diseases, including AMI, account for over 17 million deaths annually, being approximately 31% of all global deaths. In developed countries, advancements in healthcare have led to a stabilization or slight decline in AMI incidence. However, developing countries are experiencing rising rates of AMI, driven by the adoption of unhealthy lifestyles, poor diet, and insufficient physical activity.

The incidence of AMI is rising in developing countries, particularly due to increasing rates of smoking, unhealthy diets, and a sedentary lifestyle. Concurrently, the global aging population contributes to a higher prevalence of AMI, as older adults are more prone to coronary artery disease due to age-related changes in the cardiovascular system. As such, AMI stays a major public health challenge, especially in low- and middle-income countries where healthcare resources may be limited.

Role of Platelets in AMI

Platelets, also known as thrombocytes, are small, anucleate cell fragments derived from megakaryocytes in the bone marrow. They play a crucial role in hemostasis, which is the process of blood clot formation to prevent excessive bleeding after vascular injury. Upon their release into circulation, platelets have a lifespan of 7-10 days (about 1 and a half weeks). Their primary function is to form a temporary plug at sites of vascular injury and to stabilize the thrombus through activation and aggregation.

Platelet activation is a tightly regulated process involving a series of molecular events that include receptor-ligand interactions and signaling cascades, ultimately leading to platelet aggregation and clot formation. Upon activation, platelets undergo shape change, release granules, and express surface receptors that mediate the aggregation process. In the context of Acute Myocardial Infarction (AMI), platelet activation is a critical part in the pathophysiology of thrombus formation. AMI occurs primarily due to the rupture of an atherosclerotic plaque, which exposes its pro-thrombotic contents to circulating platelets. This triggers platelet adhesion, activation, and aggregation at the site of plaque rupture, leading to the formation of a thrombus that occludes the coronary artery and disrupts blood flow to the heart muscle.

Relationship Between MPV and AMI

The relationship between Mean Platelet Volume (MPV) and Acute Myocardial Infarction (AMI) has been the focus of several studies, which suggest that elevated MPV levels correlate with worse clinical outcomes in AMI patients. MPV, reflecting platelet size and activation, is a key indicator of platelet reactivity, which plays a crucial role in the pathogenesis of AMI. Larger platelets are more reactive and prone to aggregation, which can worsen thrombus formation in coronary arteries, leading to coronary artery occlusion and myocardial ischemia **[3]**

Several studies have shown that high MPV levels are associated with larger infarct sizes and a worse prognosis in AMI patients. Elevated MPV has been linked to increased risk of complications, such as reinfarction and ventricular arrhythmias. The increased thrombotic potential of larger platelets promotes the formation of coronary thrombi, which obstructs blood flow to the heart muscle, worsening myocardial injury **[4]**. Therefore, MPV can serve as a reflection of increased platelet activation and a predictor of poor clinical outcomes in AMI patients.

MPV as a Prognostic Tool in AMI

MPV has appeared as a potential prognostic marker in AMI, offering valuable insights into the severity and potential outcomes of the event. High MPV levels have been associated with increased mortality and morbidity in AMI patients, suggesting that MPV can help find those at a higher risk of adverse events. Elevated MPV reflects intensified platelet activation, which correlates with more extensive coronary thrombosis, larger infarct sizes, and a higher risk of post-infarction complications such as heart failure, arrhythmias, and recurrent myocardial infarction.

MPV has been suggested to complement traditional biomarkers of myocardial injury, such as troponin and creatine kinase-MB (CK-MB), in assessing AMI severity and prognosis. Troponin and CK-MB are critical for confirming myocardial injury, while MPV provides more information about platelet activation and the thrombotic process involved in AMI. The integration of MPV with these biomarkers enhances the accuracy of risk stratification, helping clinicians find high-risk patients who may receive help from more aggressive treatment.

Recent clinical studies have increasingly focused on the role of Mean Platelet Volume (MPV) as a biomarker in Acute Myocardial Infarction (AMI), particularly in terms of its potential for early diagnosis, prognosis, and risk stratification. High MPV levels, showing larger, more reactive platelets, are linked with increased thrombotic potential and poorer clinical outcomes in AMI patients. Studies have shown that MPV is not only an indicator of platelet activation but also reflects the severity of myocardial injury and coronary thrombosis.

In early diagnosis, elevated MPV levels have been associated with increased infarct size and higher troponin levels, suggesting that MPV could serve as a complementary biomarker to traditional markers like troponin and CK-MB. For example, a study **[5]** showed that high MPV was a reliable indicator of myocardial injury in patients presenting with chest pain, enhancing diagnostic accuracy when combined with clinical symptoms and ECG findings.

In terms of prognosis and risk stratification, studies have highlighted that elevated MPV is associated with a higher incidence of ventricular arrhythmias, reinfarction, and mortality. As an independent risk factor, MPV could help find patients who are more likely to experience adverse events during hospitalization or post-AMI recovery, thus guiding therapeutic decisions.

Comparative studies have also explored MPV dynamics before and after therapeutic interventions in AMI patients. A study showed a reduction in MPV levels following angioplasty and fibrinolysis, correlating with improved coronary blood flow and reduced platelet activation. These findings emphasize the utility of MPV as a dynamic marker of treatment success and reperfusion in AMI patients. Although there are many limitations with MPV as predictor as lack of standardization in MPV measurement techniques in its widespread clinical use. Differences in blood collection methods, laboratory equipment, and processing techniques can lead to variability in MPV results. There is a pressing need for the establishment of uniform guidelines for MPV measurement, along with well-defined thresholds for its interpretation in clinical practice. Studies have emphasized the need for standardization to enhance the reliability and comparability of MPV as a clinical biomarker. This study was planned to investigate the association between Mean Platelet Volume (MPV) levels and clinical outcomes in AMI patients.

AIM & OBJECTIVES OF THE STUDY

Primary Objectives

The primary aim of this study is to investigate the association between Mean Platelet Volume (MPV) levels and clinical outcomes in AMI patients.

Secondary Objectives

In addition to the primary aim, the study has the following secondary obectives:

1. To evaluate the relationship between MPV and infarct size, complications (arrhythmias, heart failure), and mortality in AMI:
2. To assess the correlation between MPV and other cardiovascular risk factors (e.g., lipid profile, blood pressure):

MATERIALS AND METHODS

Study design:
This was a case-control type of study.

Study area:
The study has conducted in department of General Medicine & Cardiology at National Institute of Medical Sciences & Research, Jaipur, Rajasthan.

Study population:
Population of 18 years and above age of both sex present in National Institute of Medical Sciences & Research, Jaipur.

Study period:
18 Months (1st June 2023 to 30th November 2024).

Inclusion criteria:-
- Age 18 years and above of both sex.
- Diagnosis of acute MI
- Patients presents within 24-48 hr of onset of symptoms.

Exclusion criteria:-
- Patients With history of heart failure .
- With known case of congenital heart disease
- With known case of renal or liver disease.
- Receiving anti arrhythmic drugs.
- Receiving anti platelet drugs.
- With known Platelet disorders.
- Pregnant, lactating and menopausal females.
- Patients refuse to give informed written consent.

Sample size and Sampling technique:-
Sample size was calculated using following formula :-

$$n = \left(\frac{1+r}{r}\right)^2 X \frac{(Z_{\alpha/2} + Z_{1-\beta})^2 X (\sigma_1^2 + \sigma_2^2)}{(\mu_2 - \mu_1)^2}$$

$$= \left(\frac{1+1}{1}\right)^2 X \frac{(1.96 + 0.84)^2 X (0.72^2 + 0.95^2)}{(2.74 - 2.01)^2}$$

$$= 83.6 \cong 84 \text{ samples/group}$$

Where,
$Z\alpha_{/2}$: Inverse probability of standard normal distribution at 95% confidence interval
$Z_{1-\beta}$: Inverse probability of standard normal distribution at 80% power of the test
σ_1 & σ_2: Standard deviation of platelet count of AMI (Acute Myocardial Infarction) & control group
$\mu 1$ & $\mu 2$: Mean value of platelet count of AMI & control group
r: Sampling ratio

Sampling Technique: Purposive sampling technique

RESULTS & OBSERVATIONS

The total sample size comprised 168 participants, with 84 cases (50%) and 84 controls (50%). The mean age of the participants was 57.70 ± 6.52 years, with cases having a mean age of 60.90 ± 6.58 years, and controls having a mean age of 54.50 ± 4.65 years.

Table 1. Distribution of age of subjects enrolled in the study among both the groups.

PARAMETER	CASE	CONTROL	Total (%)
Sample	84 (50%)	84 (50%)	168 (100%)
Age	60.90 ± 6.58	54.50 ± 4.65	57.70 ± 6.52
Sex			
Female	22 (26.19%)	33 (39.29%)	55 (32.74%)
Male	62 (73.81%)	51 (60.71%)	113 (67.26%)
All the data is presented in Number , Percentage & Mean ± Standard Deviation.			

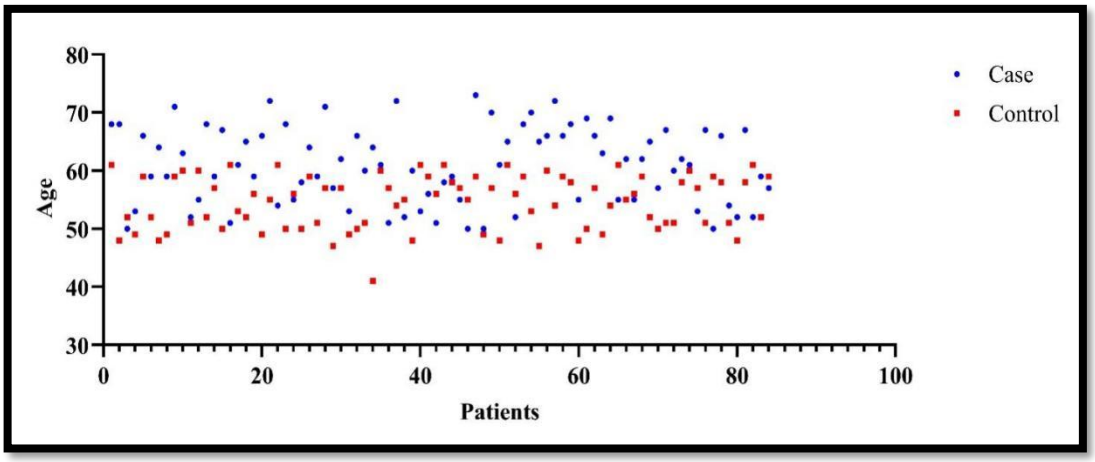

Figure 1. Scatter Plot showing the age of subjects enrolled in the study groups.

The sex distribution of the participants was 55 females (32.74%) and 113 males (67.26%). In the case group, there were 22 females (26.19%) and 62 males (73.81%), while in the control group, there were 33 females (39.29%) and 51 males (60.71%).

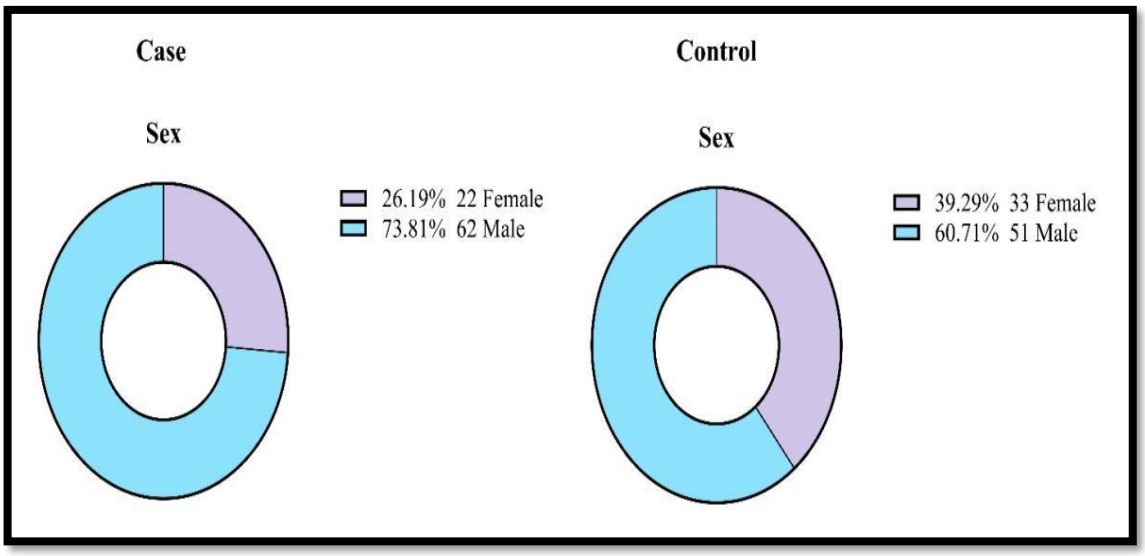

Figure 2. Donut Chart showing the gender distribution of subjects enrolled in the study among both the groups.

Regarding the chief complaints, all cases (100%) presented with chest pain, while no controls (0%) exhibited this symptom. In contrast, all controls (100%) were normal, with no cases reporting normal findings (p-value = 0.000).

For the history of present illness, the case group exhibited a variety of symptoms. Fifteen cases (17.86%) reported ghabrahat (nervousness), while none in the control group presented this symptom (p-value = 0.000). Other symptoms in the case group included profused sweating (7.14%), shortness of breath (SOB) in 49 cases (58.33%), and sweating with ghabrahat in 14 cases (16.67%). The control group had no such symptoms.

Table 3. Distribution of the history of present illness of the study subjects

PARAMETERS	CASE	CONTROL	TOTAL	P-VALUE
Chief Complaints				
Chest Pain	84 (100.00%)	0 (0.00%)	84 (50.00%)	**0.000***
Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
History of Present Illness				
Ghabrahat (Nervousness)	15 (17.86%)	0 (0.00%)	15 (8.93%)	**0.000***
Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
Profused Sweating	6 (7.14%)	0 (0.00%)	6 (3.57%)	
SOB	49 (58.33%)	0 (0.00%)	49 (29.17%)	
Sweating with Ghabrahat	14 (16.67%)	0 (0.00%)	14 (8.33%)	
Vitals				
Systolic BP (mmHg)	121.345 ± 14.881	136.429 ± 52.857	128.887 ± 94.551	0.303
Diastolic BP (mmHg)	78.619 ± 10.464	80.000 ± 4.265	79.310 ± 7.996	0.264
All the data is presented in Number , Percentage & Mean ± Standard Deviation. **The variance is significant at p-value<0.05.*				

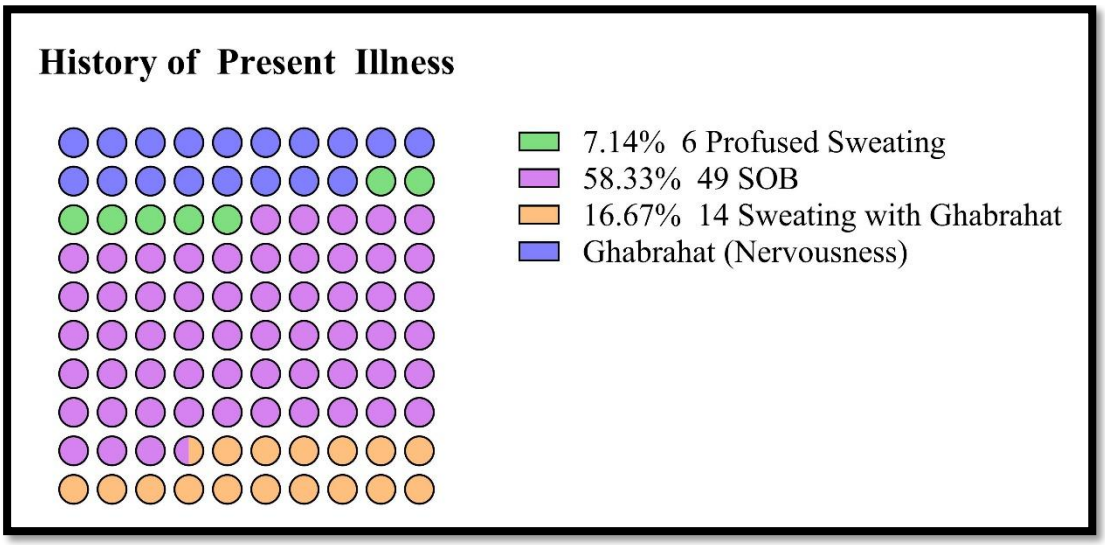

Figure 3. Dot Plot showing the history of present illness of the subjects enrolled in the study.

In terms of vitals, the systolic blood pressure (BP) in the case group was 121.35 ± 14.88 mmHg, which was lower than the control group, where the systolic BP was 136.43 ± 52.86 mmHg. The mean systolic BP for the total sample was 128.89 ± 94.55 mmHg, with a p-value of 0.303, indicating no statistically significant difference between the groups. Similarly, the diastolic BP was 78.62 ± 10.46 mmHg for cases and 80.00 ± 4.27 mmHg for controls, with the total sample having a mean diastolic BP of 79.31 ± 7.99 mmHg. The p-value for diastolic BP was 0.264, also indicating no significant difference between the groups. The variance in the data is significant where applicable, with the p-value < 0.05.

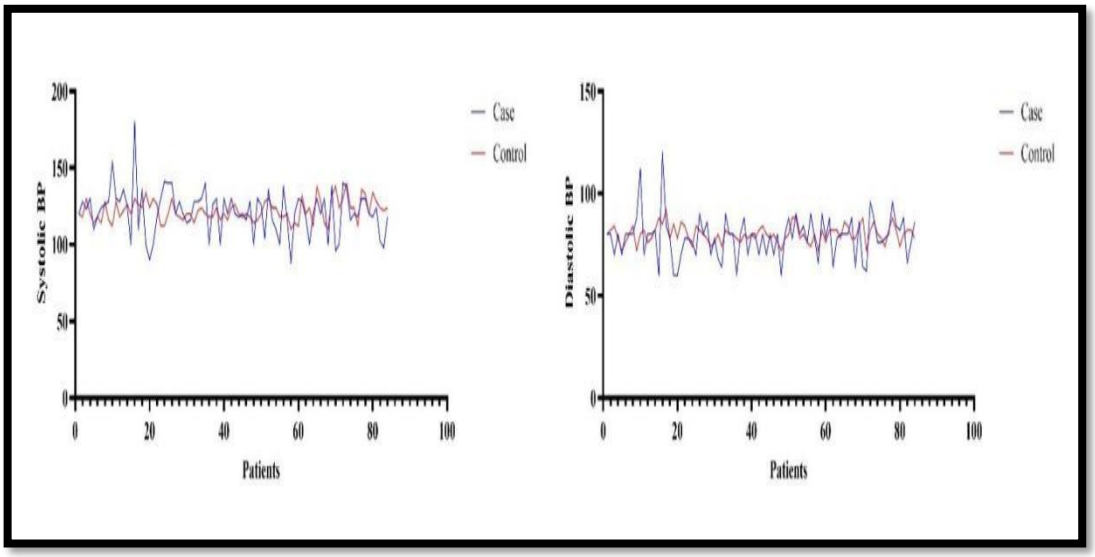

Figure 4. line graph showing the Blood Pressure of the study subjects enrolled in the study among both the groups.

DIAGNOSTIC PARAMETERS

Hemoglobin (Hb) levels were similar between the case group (12.61 ± 1.31 gm/dL) and the control group (12.86 ± 0.52 gm/dL), with no statistically significant difference (p-value = 0.108). However, White Blood Cell (WBC) counts were significantly higher in the case group (13.70 ± 2.68 x10^3/µL) compared to the control group (6.24 ± 0.80 x10^3/µL), with a p-value of 0.000. The platelet count in the case group (2.02 ± 0.47 x10^5/µL) was significantly lower than in the control group (2.49 ± 0.56 x10^5/µL), with a p-value of 0.000. Similarly, the Mean Platelet Volume (MPV) was significantly higher in the case group (11.83 ± 1.16 fL) compared to the control group (8.23 ± 0.49 fL), with a p-value of 0.000, indicating a significant difference.

Table 5. Distribution of Laboratory Parameters of subjects enrolled in the study among both the groups.

PARAMETERS	CASE	CONTROL	TOTAL	P-VALUE
Hb (gm/dL)	12.607 ± 1.306	12.855 ± 0.515	12.731 ± 0.99	0.108
WBC (x10^3/µL)	13.698 ± 2.680	6.237 ± 0.803	9.968 ± 4.229	**0.000**
Platelet (x10^5/µL)	2.021 ± 0.468	2.492 ± 0.557	2.256 ± 0.564	**0.000**
MPV (fL)	11.832 ± 1.159	8.226 ± 0.493	10.029 ± 2.01	**0.000**
Heart Sounds				
S1S2 Heard Abnormal	84 (100.00%)	0 (0.00%)	84 (50.00%)	**0.000**
S1S2 Heard Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
MI Type				
AWMI	38 (45.24%)	0 (0.00%)	38 (22.62%)	**0.000**
IWSTEMI	33 (39.29%)	0 (0.00%)	33 (19.64%)	
Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
NSTEMI	13 (15.48%)	0 (0.00%)	13 (7.74%)	
ECG Changes				
Normal Sinus Rhythm	0 (0.00%)	84 (100.00%)	84 (50.00%)	**0.000**
QS in V1-V3	2 (2.38%)	0 (0.00%)	2 (1.19%)	
QS in V2-V5	1 (1.19%)	0 (0.00%)	1 (0.60%)	
ST Elevation in V1, V3, V4	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in 2,3,AVF	33 (39.29%)	0 (0.00%)	33 (19.64%)	
STE in V1-V3	2 (2.38%)	0 (0.00%)	2 (1.19%)	

STE in V1-V4	10 (11.90%)	0 (0.00%)	10 (5.95%)	
STE in V1-V5	7 (8.33%)	0 (0.00%)	7 (4.17%)	
STE in V1-V6	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V1, V3	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V2-V4	4 (4.76%)	0 (0.00%)	4 (2.38%)	
STE in V2-V5	3 (3.57%)	0 (0.00%)	3 (1.79%)	
STE in V2-V6	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V2, V3	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V3-V6	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V4-V6	1 (1.19%)	0 (0.00%)	1 (0.60%)	
STE in V5-V6	2 (2.38%)	0 (0.00%)	2 (1.19%)	
Trop I	3 (3.57%)	0 (0.00%)	3 (1.79%)	
Trop T, CPKMB	10 (11.90%)	0 (0.00%)	10 (5.95%)	
Cardiac Biomarkers				
Elevated	84 (100.00%)	0 (0.00%)	84 (50.00%)	**0.000**
Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
Final Diagnosis				
Acute Myocardial Infarction	84 (100.00%)	0 (0.00%)	84 (50.00%)	**0.000**
Normal	0 (0.00%)	84 (100.00%)	84 (50.00%)	
All the data is presented in Number , Percentage & Mean ± Standard Deviation. The variance is significant at p-value<0.05.				

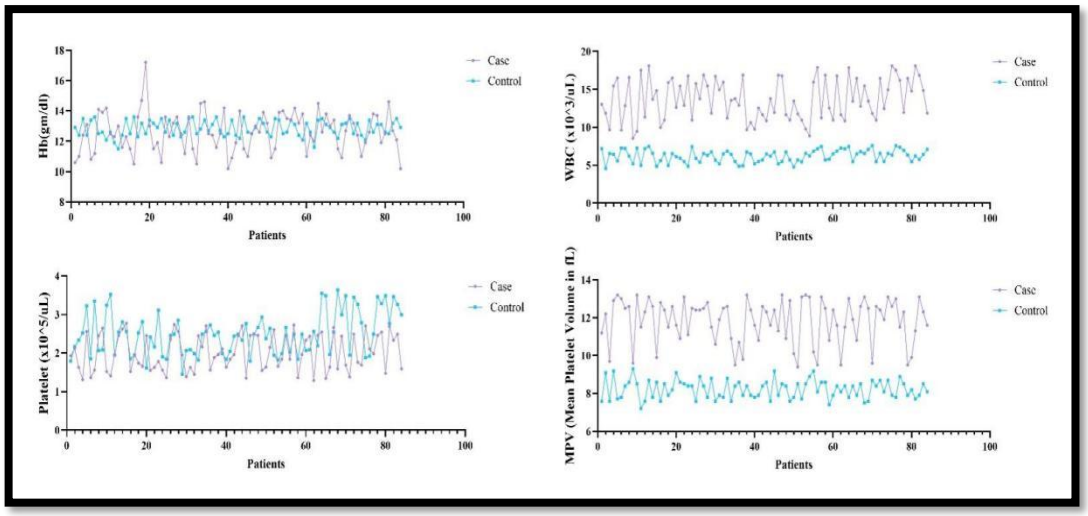

Figure 5. Line Graph showing the Laboratory Parameters of study subjects

For heart sounds, all cases (100%) exhibited S1S2 heard, while controls showed no such findings (p-value = 0.000). In terms of Myocardial Infarction (MI) type, the case group had a variety of diagnoses, including AWMI (45.24%), IWSTEMI (39.29%), and NSTEMI (15.48%), with all controls being normal (p-value = 0.000). ECG changes in the case group included ST elevation (STE) in various leads, such as 2, 3, AVF (39.29%), V1-V3, V2-V5, and V1-V6, while controls presented with normal sinus rhythm (p-value = 0.000).

Cardiac biomarkers were elevated in all cases (100%), while all controls had normal levels (p-value = 0.000). Finally, the final diagnosis for all cases was Acute Myocardial Infarction, with no cases in the control group (p-value = 0.000). The overall variance in these diagnostic parameters was found to be significant with a p-value < 0.05, reflecting clear differences between the case and control groups

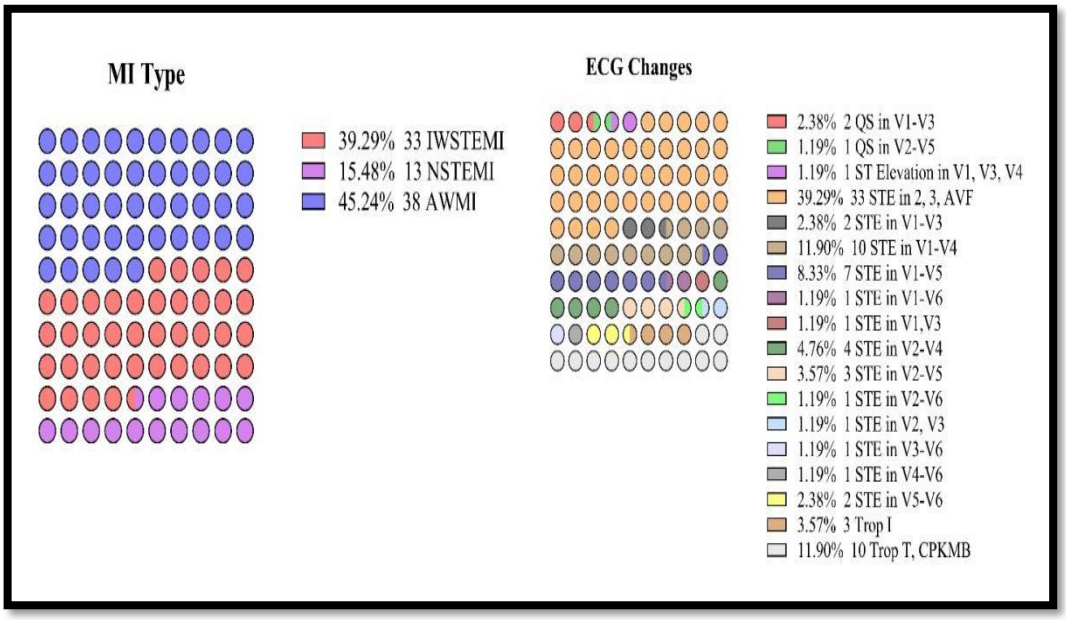

Figure 6. Dot Plot showing the type of Mi and ECG changes of the study subjects

DISCUSSION

The study comprised 168 participants, equally divided into 84 cases (patients diagnosed with Acute Myocardial Infarction, AMI) and 84 controls (healthy individuals). The mean age of the participants was 57.70 ± 6.52 years, with the case group having a mean age of 60.90 ± 6.58 years and the control group having a mean age of 54.50 ± 4.65 years. The sex distribution was 55 females (32.74%) and 113 males (67.26%), with 22 females (26.19%) and 62 males (73.81%) in the case group, and 33 females (39.29%) and 51 males (60.71%) in the control group.

In terms of clinical parameters, the chief complaints revealed that all cases (100%) presented with chest pain, while no controls (0%) exhibited this symptom, and all controls (100%) were normal. Regarding the history of present illness, 17.86% of cases reported ghabrahat (nervousness), with no controls experiencing this symptom. Other symptoms in the case group included profuse sweating (7.14%), shortness of breath (SOB) in 58.33%, and sweating with ghabrahat in 16.67%. The control group had no such symptoms.

Regarding vital signs, systolic blood pressure (BP) was lower in the case group (121.35 ± 14.88 mmHg) compared to the control group (136.43 ± 52.86 mmHg), and the mean systolic BP for the total sample was 128.89 ± 94.55 mmHg, with no significant difference between the groups (p-value = 0.303). Diastolic BP was also similar, with the case group having a mean of 78.62 ± 10.46 mmHg and the control group having 80.00 ± 4.27 mmHg, with no significant difference (p-value = 0.264).

The diagnostic parameters showed significant findings. Hemoglobin (Hb) levels were similar between the case group (12.61 ± 1.31 gm/dL) and the control group (12.86 ± 0.52 gm/dL) with no significant difference (p-value = 0.108). However, White Blood Cell (WBC) counts were significantly higher in the case group (13.70 ± 2.68 $x10^3/\mu L$) compared to the control group (6.24 ± 0.80 $x10^3/\mu L$), with a p-value of 0.000, indicating increased inflammation or immune response in AMI patients. The platelet count in the case group (2.02 ± 0.47 $x10^5/\mu L$) was significantly lower than in the control group (2.49 ± 0.56 $x10^5/\mu L$), and the Mean Platelet Volume (MPV) was significantly higher in the case group (11.83 ± 1.16 fL) compared to the control group (8.23 ± 0.49 fL), with both differences showing p-values of 0.000.

Larger platelets are associated with an increased thrombotic potential and may play a crucial role in the development of coronary thrombosis and MI. This study found that the higher MPV in the AMI group suggests that larger platelets are involved in infarction and may be a risk factor for developing coronary thrombosis. MPV can be easily identified through routine hematological analysis, where Platelet Volume Index (PVI) values are generated as a byproduct of automated blood counts. The

significant difference in MPV between the case and control groups highlights its potential as a cost-effective tool to identify individuals at higher risk for cardiovascular events, such as MI.
These findings align with previous research, which suggests that MPV could serve as a reliable biomarker for atherosclerotic or thrombotic tendencies in the body **[6][7][8]**
Regarding heart sounds, all cases (100%) exhibited S1S2 heard, while no controls exhibited such findings. Myocardial Infarction (MI) types in the case group included AWMI (45.24%), IWSTEMI (39.29%), and NSTEMI (15.48%), with all controls being normal. ECG changes in the case group included ST elevation (STE) in various leads, such as 2, 3, AVF (39.29%), V1-V3, V2-V5, and V1-V6, while controls presented with normal sinus rhythm.
Cardiac biomarkers, such as Trop I and Trop T, were elevated in all cases (100%), while all controls had normal levels. The final diagnosis for all cases was Acute Myocardial Infarction, with no cases in the control group. The significant differences in the diagnostic parameters and final diagnosis further validate the findings, with p-values < 0.05.
The study demonstrates that the increased MPV in patients diagnosed with AMI is significantly higher than in the control group. Larger platelets may contribute to infarction, serving as a risk factor for coronary thrombosis and myocardial infarction. The measurement of MPV during routine hematological analysis can be used as a cost-effective tool for identifying patients with atherosclerotic or thrombotic tendencies, potentially enabling early identification of those at risk for cardiovascular events. This supports the notion that MPV is a valuable biomarker for assessing the risk of coronary artery disease and may play a key role in predicting the onset of MI **[9][10]**

CONCLUSION

The study demonstrates that the increased Mean Platelet Volume (MPV) in patients diagnosed with Acute Myocardial Infarction (AMI) is significantly higher compared to the control group. This finding suggests that larger platelets may play a crucial role in the development of myocardial infarction (MI) by contributing to thrombosis formation within coronary arteries. The presence of larger platelets is often associated with a heightened thrombotic potential, which could increase the likelihood of plaque rupture and subsequent clot formation, potentially leading to an AMI event. Given this association, MPV could serve as an important biomarker for early identification of individuals at risk for coronary artery disease (CAD).
In conclusion, the elevated MPV in patients with AMI highlights its potential as a novel and easily measurable biomarker for identifying individuals at risk for myocardial infarction and other thrombotic cardiovascular events. By integrating MPV measurement into routine clinical practice, healthcare providers could improve early detection and implement preventative strategies to reduce the burden of cardiovascular diseases. This study contributes to a growing body of evidence supporting the use of MPV as a valuable tool for the management and prevention of coronary artery disease and its associated complications

Conflict of Interest: None

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