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Cardiology

STUDY OF MEAN PLATELET VOLUME IN ACUTE MYOCARDIAL INFARCTION IN COMPARISON WITH HEALTHY INDIVIDUALS

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ABSTRACT **Background:** Ischemic heart disease is caused by atherosclerosis. Platelets and their aggregation and activation are involved in the onset of atherosclerotic lesions and the production of coronary thrombus. Platelets with a larger size are more enzymatically and metabolically active and have a higher thrombotic ability than platelets with a smaller size. **Aims And Objectives:** The purpose of this study was to look at the impact of mean platelet volume in acute myocardial infarction and compare MPV to controls. **Methodology:** This was a comparative study of 100 patients (50 with AMI and 50 age/gender-matched controls). Clinical variables, complete blood count, and lipid profile were analyzed. **Results:** The mean age of the AMI case group was 52.6±9.901yrs, and 53.38±10.458 years in control individuals (p=0.7032). In patients with AMI, the mean platelet count was 257608, the mean MPV was 12.63fl, and the mean PDW is 14.11fl. In the control population the mean MPV, PDW, and platelet were9.92fl, 11.15fl, and 252100/mm3. Patients with AMI had a substantially higher MPV (12.63±2.262fl) than control subjects (9.92±2.88fl) (p<0.001). In subgroup analysis within AMI patients, it was seen that MPV in those with hypertension (13.209±2.123fl) was higher than that in patients without hypertension (11.517±2.156f) with significant correlation (r= 0.128, p=0.0107). Hypertensive patients had significantly higher MPV than hypertensive controls.MPV in patients with AMI was found to be elevated with advancing age (pvalue<0.05, statistically significant), and Family History (p = 0.0356, statistically significant). The cut-off value for MPV in predicting acute myocardial infarction was 12.63 fl (sensitivity 75%; specificity 52%). The correlation of MPV with death or poor outcome has a negative connection (r - 0.265, p =0.01). The death due to AMI is depicted by the prognostic assessment of MPV at 15.8667fl. The AUC for MPV in predicting AMI was determined to be 0.650 after constructing a ROC curve. **Conclusion:** MPV is a sign of more reactive platelets that is strongly associated with acute myocardial infarction and predicts death or a bad prognosis. In individuals with AMI, larger platelets were easily detected during standard hematological tests. As a result, MPV is a straightforward, practical, and cost-effective variable that can be used in conjunction with normal biochemical cardiac indicators to predict an approaching cardiovascular event.

KEYWORDS :

INTRODUCTION

Globally, coronary atherosclerosis and myocardial infarction (MI) are the leading cause of morbidity and mortality.1Smoking, dyslipidemia, hypertension, diabetes mellitus, and a family history of CAD have a significant pathophysiological effect on risk. Acute coronary syndromes (ACS) are a group of symptoms caused by a decrease in blood flow in the coronary arteries, caused by the rupture of a plaque and the formation of platelet-rich coronary thrombus. Platelet activation is crucial in the progression of cardiovascular disease(CVD) to life-threatening clinical situations such as ischemic stroke and myocardial infarction (MI). The thrombus totally or partially obstructs the coronary arteries, causing clinical symptoms ranging from unstable angina to severe myocardial infarction.

Platelets with larger granules have more glycoprotein IIb/IIIa receptors, higher thromboxane A2 levels, and more collagen-active aggregates. Mean platelet volume (MPV) has been proposed as a measurement of platelet function and can be used to quantify platelet size. Large platelets are more enzymatically active and have a larger thrombotic potency than small platelets.MPV has been associated with CAD, unstable angina, and AMI in the previous research. Larger platelets released adhesion molecules, growth factors (platelet-derived growth factor, transforming growth factor, and basic FGF), and chemotactic factors (platelet factor 4, coagulation factors [factor V and factor XI], and cytokine-like factors) (interleukin-1 and CD40) in greater quantities. Unlike other prognostic indicators, MPV is automatically detected by a blood cell counter in an outpatient scenario at a very low cost.At the moment, we don't know the accuracy of MPV in detecting AMI in the Indian population suffering from severe chest pain.

AIMS AND OBJECTIVES

Aim:

This current study aims to study the significance of mean platelet volume in acute myocardial infarction and to correlate with MPV in healthy controls.

Objectives:

To compare platelet indices in AMI patients to those in healthy controls. To compare the average platelet volume of patients with acute myocardial infarction to healthy controls. To investigate the factors that influence MPV variation in AMI patients.

MATERIALS AND METHODS

Place Of Study

The study was carried out at Govt. General Hospital, Department of General Medicine, Kurnool Medical College in Kurnool, Andhra Pradesh.

Study Design

A prospective, observational case-control study.

Study Population

Patients with a confirmed diagnosis of acute myocardial infarction and satisfying the inclusion criteria were included. Typical rise and gradual fall (troponin) of biochemical markers of myocardial necrosis with at least one:

1.Ischemic symptoms
2.Development of pathologic Q waves on the ECG reading
3.ECG changes indicative of ischemia (ST-segment elevation/ depression).
4.New-onset LBBB

Sample Size=50

Study Duration

18 months duration from December 2019 to June 2021.

Inclusion Criteria:

1. Patients with acute myocardial infarction
2. Healthy Controls

Exclusion Criteria:

1. Sepsis, Immune thrombocytopenia
2. Severe hepatic and renal disease
3. Patients on anti-inflammatory agents
4. Anticoagulation &anti-platelet agents except for aspirin

METHOD OF COLLECTION OF DATA

For all the patients coming with chest pain first, 12 lead ECGs were taken within 10 min of arrival to the Emergency. The patient was connected to a cardiac monitor, Oxygen supplementation was given with a face mask if Spo2 < 94% on room air, IV line was secured. A detailed history of chest pain, palpitation, sweating, vomiting, dyspnea, giddiness was checked, past personal and family history were recorded if any. Patients were evaluated for risk factors such as

diabetes, hypertension, hypercholesterolemia, and smoking. The diagnosis of AMI was made based on clinical presentation, ECG changes, and serum cardiac biomarker (Troponin-T) levels. Serial ECG was taken.

2 ml of blood was collected in an EDTA container on admission. This sample was sent to the hematology to obtain the platelet indices by Beckman Coulter Auto analyzer.

RESULTS

The mean MPV of MI case group participants was 12.63 fl with a standard deviation of 2.2fl. The mean MPV of control was 9.92 with a standard deviation of 2.88fl. The mean PDW of cases was 14.11 fl with a standard deviation of 2.0021fl.

In patients with AMI, the mean platelet count was 2,57,608, the mean MPV was 12.63fl, and the mean PDW is 14.11fl.

Males were more than females; we compared the platelet indices in groups divided by gender. Platelet count was higher in females than males. No significant difference in MPV was seen between the male and female groups in the MI Cases.

Pearson correlation analysis used to identify the correlations between platelet count and platelet volume indices. Negative correlation observed between platelet count and MPV (r - 0.031, $p < 0.001$), platelet count and PDW (r- 0.108, $p < 0.001$).

There is a significant difference in MPV, PDW, and PLCR in AMI and control subjects.

MPV was significantly higher in patients with STEMI (13.71667±1.89fl) than patients without STEMI (10.10769±1.01347) ($p< 0.0001$). Similarly, MPV was higher in patients with STEMI (13.71667±1.89fl) than patients with NSTEMI (13.02142±1.780742fl) and those from unstable angina population (10.02±1.16fl) ($p<0.0001$).

The mean MPV in AMI patients with an ejection fraction of less than 29 percent is larger than in those with an EF of more than 50 percent (15.867± 0.8504 vs 12.355 ± 2.35024fl). ($P<0.001$) MPV was found to be greater in patients with hypertension (13.209 ± 2.123fl) than in patients without hypertension (11.517±2.156f), with a significant connection (r=0.128, p=0.0107) in subgroup analysis within AMI patients. MPV was observed to be higher in acute myocardial infarction cases with increasing age (p-value 0.05, statistically significant) and family history ($p = 0.0356$, statistically significant). There was a statistically significant association between MPV and a family history of heart disease. The association between aspirin intake and MPV was not statistically significant.

The prognostic measurement of MPV at 15.8667 ± 0.85049fl depicts the death in acute myocardial infarction cases.When predicting AMI in patients, the AUC of PDW, MPV, and P-LCR was found to be 0.852, 0.650, and 0.745 (95%CI). Logistic regression analysis of MPV quartiles, we observed>12.63 fL was a significant risk for developing AMI. The sensitivity of MPV in predicting AMI was higher than that of initial serum troponin I as the best cut-off value (78.6 percent vs 50.0%).

If the AUC of MPV in predicting AMI in patients with acute chest pain was greater than the initial troponin I level (0.855 vs 0.758). As a result, when combined with initial troponin I, the sensitivity and specificity of MPV for predicting AMI increased to 95% and 75%, respectively. We identified P-LCR was significantly higher in the AMI group, with>29.04%having a sensitivity and specificity of 70% and 50%. We show that PDW had an observed value of > 14.11 fL, with sensitivity and specificity of 75% and 55%, respectively.

DISCUSSION

Diabetes, systemic hypertension, smoking, and dyslipidemia were all found to significantly increase the risk of myocardial infarction. However, they only account for a percentage of the instances; additional risk factors must be observed. Large platelets are more reactive, create more thrombotic factors, and aggregate more readily than smaller platelets (Martin et al). (Haver and colleagues, 1981).57,58,59 Mean platelet volume (MPV) is an independent risk factor for acute myocardial infarction, according to Endler et al. (2002). MI had a greater MCV than controls in the current study. Patients with MI had significantly greater levels of MPV, PWD, and PCLR than controls. Similarly, STEMI patients had significantly greater MPV, PWD, and PCLR levels than NSTEMI and unstable angina patients.

In our study in the cases, there were 31 males and 19 females, while in the control group, there were 31 males and 17 females. The patient group was 52.6± 9.901 years old, while the control group was 53.38±10.458 years old. The prevalence of smoking (72%) systemic hypertension (66%), and diabetes mellitus (50%) were noticed in the AMI case group. Cases had a mean platelet count of 257608 ± 125218.2484/mm3, while controls had a mean platelet count of 2,52,100.00±1,15,902.89/mm3 (0.001, statistically significant). A significant association (r- 0.109, $p<0.001$) existed between those with acute myocardial infarction (12.63 ± 2.262fl) and those without acute myocardial infarction (Control) (9.92 ± 2.88fl). In subgroup analysis within AMI patients, it was observed that MPV in those with hypertension (13.209±2.123fl) was higher than that in patients without hypertension (11.517±2.156f) with significant correlation (r= 0.128, p=0.0107). In control participants with and without diabetes, there was no association ($p>0.05$). MPV was shown to be higher in individuals with acute myocardial infarction as they were older (p-value 0.05, statistically significant) and had a family history ($p = 0.0356$, statistically significant). Diabetes mellitus was not found to have any significant association with MPV in patients with AMI (12.6 ± 2.36572 fl vs. 12.668 ±2.2fl, $p = 0.9167$, not significant). Aspirin was not found to have any significant association with MPV (12.875±2.191fl vs 12.3±2.369, $p = 0.3798$, not significant). In neither cases nor control participants, subgroup analysis of smokers vs. nonsmokers failed to attain statistical significance.

When comparing patients with STEMI (13.7166±7.89fl) to patients without STEMI (10.10769±1.01347), MPV was substantially greater in STEMI patients (13.7166±7.89fl) ($p<0.0001$). When patients with STEMI (13.7±1667.89fl) were compared to patients with unstable angina cases (10.02±1.16fl), the MPV was significantly higher (13.7166±7.89fl) ($p<0.001$). Platelet count and MPV ($r = 0.031$, p 0.001) and platelet count and PDW($r= 0.108$, $p <0.001$) had negative correlations, according to Pearson correlation analysis. There was a statistically significant connection between higher platelet count and MPV (p-value 0.0001). 12.63 fl. oz. was the best cut-off value for MPV in predicting AMI (sensitivity 75%; specificity 52%). The sensitivity of MPV in predicting AMI was higher than that of initial serum troponin I at the optimum cutoff value (78.6% vs 50.0%). The sensitivity and specificity of MPV for predicting AMI can be raised to 95%and 75%when paired with initial troponin I. MPV in patients with acute myocardial infarction was found to be highly elevated in non-survival AMI cases than in survival cases (15.8667 ± 0.85049 fl vs. 12.42767±2.166912fl). The fatality due to acute myocardial infarction is depicted by the prognostic measurement of MPV at 15.8667 fl. As a result, MPV is an easily available laboratory variable that can be used to supplement the predictive value of initial troponin I for AMI (MPV >12.63 fl or initial troponin I >0.04 ng/ml, respectively).

CONCLUSION:

AMI patients had considerably greater MPV than controls. Platelet volume indicators can be used to forecast the onset of acute coronary events. This is a low-cost, straightforward, and reliable approach for detecting platelet activation and predicting the risk of acute MI. According to the findings, MPV can be used in conjunction with traditional risk variables to predict CAD and MI, allowing for more thorough examination and management.

REFERENCES:

1. Kumar V, Melhotra S, Ahuja Ret RC and Viash AK. Platelet and Acute Coronary Syndrome. J Fam Med. 2016; 3(4): 1063.
2. Jaya Manchanda, R M Potekar, Sharan Badiger, Abhishek Tiwari. The study of platelet indices in acute coronary syndromes. Annals of Pathology and Laboratory Medicine, Vol. 02, No. 01, Jan-Mar 2015.
3. Endler G, Klimesch A, Sunder-Plassmann H et al: Mean platelet volume is an independent risk factor for myocardial infarction but not for coronary artery disease. Br J Haematol, 2002; 117: 399–404
4. Dalby Kristensen S, Milner PC, Martin JF: Bleeding time and platelet volume in acute myocardial infarction – a 2 year follow-up study. Thromb Haemost, 1988; 59: 353–56.
5. Jakubowski JA, Adler B, Thompson CB et al: Influence of platelet volume on the ability of prostacyclin to inhibit platelet aggregation and the release reaction. J Lab Clin Med, 1985; 105: 271–76.
6. Halbmayer WM, Haushofer A, Radek J et al: Platelet size, fibrinogen and lipoprotein(a) in coronary heart disease. Coron Artery Dis, 1995; 6: 397– 402
7. Martin JF, Slater DN, Kishk YT, Trowbridge EA: Platelet and megakaryocyte changes in cholesterol-induced experimental atherosclerosis. Arteriosclerosis, 1985; 5: 604–12
8. Bessman JD: The relation of megakaryocyte ploidy to platelet volume. Am J Hematol,

1984; 16: 161–70
9. Brown AS, Hong Y, de Belder A et al: Megakaryocyte ploidy and platelet changes in human diabetes and atherosclerosis. Arterioscler Thromb Vasc Biol, 1997; 17: 802–7
10. Corash L, Chen HY, Levin J et al: Regulation of thrombopoiesis: effects of the degree of thrombocytopenia on megakaryocyte ploidy and platelet volume. Blood, 1987; 70: 177–85.