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Diagnostic Value of Mean Platelet Volume in Prediction of Acute Myocardial Infarction

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ABSTRACT

Background: Acute myocardial infarction (AMI) is one of the main etiologies of death globally. In AMI, platelets were demonstrated to play a main role in thrombotic processes that limit the patency of the recanalized, infarct-related coronary artery and contribute to reperfusion injury. Platelet volume is an important indicator for platelet function and activation.

Objective: The aim of the present study was to evaluate mean platelet volume (MPV) as an early and independent predictor for AMI in patients with acute chest pain.

Patients and methods: This retrospective observational analytical study included a total of 107 patients presenting with acute chest pain presenting to Mansoura Emergency Hospital within the period from January 2019 to January 2020. Of them, 36 cases were diagnosed as stable coronary artery disease and 71 cases were diagnosed as AMI.

Results: There were statistically significant differences among both groups regarding age, female sex and BMI being increased in AMI group. Hypertension, DM and dyslipidemia were significantly increased among AMI cases compared to stable coronary artery disease ones. Stable coronary artery disease cases demonstrated significant increase in platelet count and significant decrease in MPV compared to AMI ones. Hypertension, DM, dyslipidemia, family history of CAD, prior MI, prior PCI and MPV could be used as significant predictors for AMI.

Conclusion: The current study concluded that larger platelet volumes may be used as predictor for AMI as well as ischemic complications.

Keywords: Diagnostic Value of Mean Platelet Volume, Acute Myocardial Infarction

INTRODUCTION

Acute myocardial infarction (AMI) is becoming the leading cause of morbidity and mortality in developing countries. The spectrum of presentation is wide from unstable angina to acute myocardial infarction. Despite advances in the acute coronary syndrome (ACS) diagnosing and therapies, physicians hospitalize more numbers of low-risk patients with suspected myocardial ischemia **(1).**

Troponin and creatine kinase-MB (CK-MB) are the routine biochemical markers, which are used to detect AMI. Troponin, as the most sensitive and tissue-specific cardiac marker, is now considered as the gold-standard biochemical tool for AMI risk stratification; however, it is undetectable in about 40-60% of patients suffering from an ACS. Therefore, using a multimarker approach may be of benefit to diagnose ACS **(1).**

It has been previously demonstrated that the atherosclerosis is a chronic inflammatory disease. Atherosclerotic lesions in large and medium-sized arteries can contribute to the ischemia of the heart, brain or extremities leading to infarction. Plaque rupture and thrombosis are the important complications of the atherosclerotic lesions resulting in ischemia. Some factors have been investigated regarding this issue, and one of the main suspected factors is platelet circulating in the blood flow.

Platelets are a source of inflammatory mediators, and they are being influenced in contact with artery surface **(2).**

The activated platelets release the mediators, and then, platelet adhesion and its athero-thrombotic potential can lead to the release of mediators, the progression of inflammatory process, and the propagation of intracoronary thrombus predisposing to thrombotic events **(2).**

Mean platelet volume (MPV), which is a component of complete blood count, is the most common and reliable index to identifying the platelet size and its activation status.

An increased MPV is associated with known cardiovascular risk factors, including diabetes mellitus (DM), hypertension (HTN), hypercholesterolemia, and obesity. Some investigations have demonstrated the correlation between elevated MPV and AMI, and also the association between increased MPV and percutaneous coronary intervention outcomes, including mortality and stent restenosis.

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However, some studies have also shown that MPV cannot be considered as either a marker of platelet activation or a cardiovascular risk factor [3]. The aim of the present study was to evaluate MPV as an early and independent predictor for AMI in patients with acute chest pain.

PATIENTS AND METHODS

This retrospective observational analytical study included a total of 107 patients presenting with acute typical chest pain, attending at Mansoura Emergency Hospital, and admitted in Cardiology Department in Medical Specialized Hospital. This study was conducted between January 2019 to January 2020.

Of 107 patients, 36 cases were diagnosed as stable coronary artery disease and 71 cases were diagnosed as AMI,

Inclusion criteria: Patients with acute chest pain typical for myocardial ischemia, age group above 18 years old, and both genders.

Exclusion criteria: Age group below 18 years old, severe hepatic or renal impairment, malignancy, myeloproliferative disorders, and patients who refuse to participate in the study.

The studied patients were divided into: Group 1 (Stable coronary artery disease, SCAD): patients presented with acute chest pain but without evidence of myocardial ischemia in ECG, Troponin I and ECHO, and **Group 2 (AMI):** patients presented with acute typical chest pain with ischemic changes of myocardial infarction in ECG and ECHO and elevated Troponin I values.

Files of patients admitted to Mansoura Specialized Hospital with chest pain from January 2019 to January 2020 were collected and all data were conducted including:

I) **Full history taking:** Personal history, history of the present illness, and present medical history including hypertension, diabetes mellitus, and coronary artery disease.

II) **Examination:** Vital signs, heart, neck, chest abdomen, lower limb, and ECG (12-lead ECG looking for signs of myocardial ischemia).

III) **Investigations:**

- **Laboratory:** Complete blood count, cardiac enzyme (Troponin I), coagulation profile, and random blood glucose levels.
- **Radiological:** Echocardiography in Cardiology Department for assessment of wall motion abnormality and left ventricular ejection fraction (LVEF).

Ethical consideration:

The study was approved by the Ethics Board of Mansoura University. The research objectives were explained to the participants' relatives individually and in groups. The researcher was available throughout the study. Informed written consent was obtained from each participant's relatives sharing in the study. Confidentiality and personal privacy were respected in all levels of the study. The relatives were ensured that the participation was completely voluntarily, and withdrawal from the study had no effect on their evaluation process. Collected data was not used for any other purposes.

Statistical analysis

Data were fed to the computer and analyzed using IBM Statistical Package for the Social Sciences (SPSS) Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp. Qualitative data were described using number and percent. Quantitative data were described using mean, standard deviation for parametric data after testing normality using Kolmogrov-Smirnov test. Significance of the obtained results was judged at the (0.05) level. Chi-Square test for comparison of 2 or more groups. Fischer Exact test was used as correction for Chi-Square test when more than 25% of cells have count less than 5 in 2*2tables . Student t-test was used to compare 2 independent groups. The Pearson product-moment correlation is used to determine the strength and direction of a linear relationship between two normally distributed continuous variables. Receiver Operating Characteristic (ROC) curve analysis: The diagnostic performance of a test or the accuracy of a test to discriminate diseased cases from non-diseased cases is evaluated using Receiver Operating Characteristic (ROC) curve analysis. Sensitivity and Specificity were detected from the curve and PPV, NPV and accuracy were calculated through cross tabulation.

Binary stepwise logistic regression analysis was used for prediction of independent variables of binary outcome. Significant predictors in the Univariate analysis were entered into regression model using forward Wald method /Enter. Adjusted odds ratios and their 95% confidence interval were calculated.

RESULTS

Concerning demographic characteristics of the studied patients, there were statistically significant differences among both groups regarding age, female sex and BMI being increased in AMI groups **(P<0.05),** with no significant difference regarding marital status **(P>0.05).**

Concerning the platelet count and MPV value among studied patients, there was statistically significant increase in platelet count and statistically

significant decrease in MPV in stable coronary artery disease cases compared to AMI ones (P<0.05).

Table (1): Demographic characteristics and platelet count and MPV value among of the studied patients.

	Stable coronary artery disease (N=36)	AMI N=71	Test of significance
Age/years Mean±SD	53.08±5.96	59.61±12.19	t=3.08 p=0.003*
Sex n(%) Male Female	33(91.7) 3(8.3)	51(71.8) 20(28.2)	χ^2=5.57 p=0.018*
Marital status n(%) Not married Married	5(13.9) 31(86.1)	22(31.0) 49(69.0)	χ^2=3.70 p=0.054
BMI (kg/m^2) Mean± SD	27.44±1.69	31.0±5.19	t=4.01 p<0.001*
Platelet count Mean±SD	262.06±16.39	225.80±18.38	t=2.37 p=0.02*
MPV(**fl**) Mean±SD	7.51±1.52	9.33±1.29	t=4.89 p<0.001*

t: Student t test , χ^2= Chi-Square test , *statistically significant if p<0.05

At cut off 7.45, MPV could be used as a significant predictor **(P=0.001)** in the differentiation between AMI from stable coronary artery disease cases with sensitivity, specificity, PPV, NPV and accuracy of 95.8, 72.2, 87.2, 89.7 and 87.9 respectively**.**

Table (2): Validity of MPV in differentiating AMI from stable coronary artery disease groups.

	AUC (95%CI)	P -Value	cut off point	Sensitivity%	Specificity%	PPV%	NPV%	Accuracy %
MPV(fl)	0.827 (0.730-0.925)	0.001*	7.45	95.8	72.2	87.2	89.7	87.9

PPV: Positive predictive value , NPV: Negative predictive value, AUC: area under curve

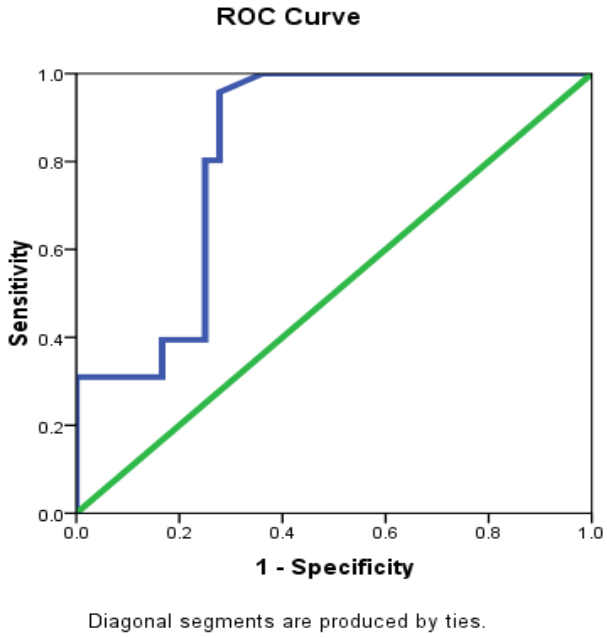

Figure (1): Receiver Operating characteristics curve of MPV in differentiating AMI group.

Regarding the correlation between MPV value and demographic characteristics among AMI group, AMI cases were demonstrated to be significantly associated with advanced age as well as higher BMI **(P<0.05),** while sex and marital status not **(P>0.05).** As regard the correlation between MPV value, medical and family history among AMI group, DM, dyslipidemia, smoking, family history of CAD, prior MI and prior PCI were demonstrated to be significantly increased among AMI cases **(P<0.05),** while HTN not **(P>0.05).**

Regarding, the correlation between MPV value and laboratory, treatment and outcome among AMI group, there were no statistically significant correlations among MPV and all parameters **(P>0.05),** except stenosis which demonstrated significant correlation **(P<0.05).**

Table (3): Correlation between MPV value and demographic characteristics, medical, family history, laboratory, treatment and outcome among AMI group.

	AMI N=71	**Test of significance**
	MPV(fl) Mean± SD	
Age/years	r= 0.298	p=0.012*
Sex		
Male	9.18±1.23	t=1.56
Female	9.71±1.41	p=0.124
Marital status		
Not married	9.08±1.28	t=1.04
Married	9.43±1.31	p=0.303
BMI (kg/m^2)	r= 0.650	p<0.001*
Hypertension		
-ve	9.29±1.56	t=0.166
+ve	9.35±1.05	p=0.869
DM		
-ve	8.89±1.17	t=4.56
+ve	10.22±1.095	p<0.001*
Dyslipidemia		
-ve	8.92±1.12	t=5.41
+ve	10.53±1.04	p<0.001*
Smoking		
Non smoker	8.93±0.90	t=2.20
Current	9.60±1.46	p=0.03*
Family history of CAD		
-ve	7.74±0.33	t=6.84
+ve	9.75±1.12	p<0.001*
Prior MI		
-ve	7.60±0.48	t=3.69
+ve	9.48±1.23	p<0.001*
Prior PCI		
-ve	7.78±0.31	t=6.13
+ve	9.71±1.15	p<0.001*
Platelet count	r=-0.183	p=0.127
Troponin		
-ve	9.30±1.52	t=0.09
+ve	9.33±1.21	p=0.929
Thrombolytic		
-ve	9.35±1.29	t=0.344
+ve	9.24±1.34	p=0.732
Stenosis		
-ve	8.98±1.21	t=3.20
+ve	9.89±1.26	p=0.004*
Death		
-ve	9.17±1.37	t=1.43
+ve	9.63±1.10	p=0.155

r: Pearson correlation coefficient (for correlation continuous parametric variables), t: Student t test , P: probability

At cut off 7.45, MPV could predict death among AMI cases with sensitivity, specificity, PPV, NPV and accuracy of 95.8, 72.2, 87.2, 89.7 and 87.9 respectively.

Table (4): Validity of MPV in predicting death among AMI cases

	AUC (95%CI)	P Value	cut off point	Sensitivity%	Specificity%	PPV%	NPV%	Accuracy %
MPV(fl)	0.587 (0.456-.718)	0.231	7.45	95.8	72.2	87.2	89.7	87.9

PPV: Positive predictive value, NPV: Negative predictive value AUC: area under curve

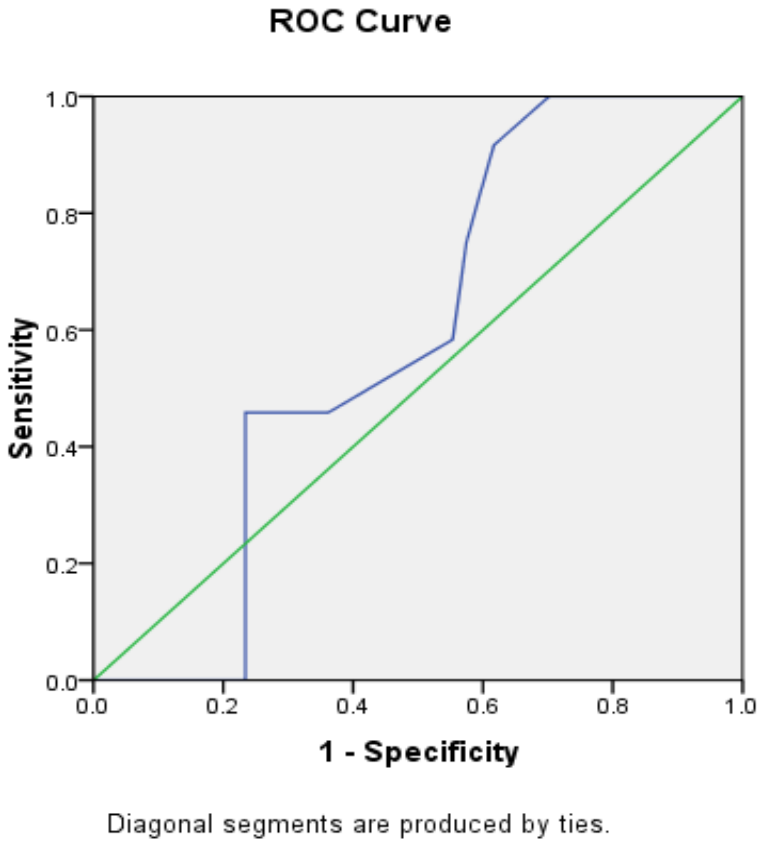

Figure (2): Validity of MPV in predicting death among AMI cases.

Regarding the predictors of acute myocardial infarction, hypertension, DM, dyslipidemia, family history of CAD, prior MI, prior PCI and MPV could be used as significant predictors for AMI **(P<0.05),** while MPV not **(P>0.05).**

Table (5): Predictors of acute myocardial infarction among studied patients.

	β	P-Value	Odds ratio (95%CI)
Hypertension	0.217	0.02*	4.805(1.124-6.23)
DM	1.464	0.04*	2.15(1.05-4.7)
Dyslipidemia	5.951	0.02*	5.2(1.24-8.97)
Family history of CAD	2.526	0.02*	4.9(2.5-9.7)
Prior MI	8.217	0.039*	3.58(2.18-8.9)
Prior PCI	9.474	0.01*	5.9(2.4-8.7)
Platelet count	0.0	0.774	undefined
MPV(fl)	0.917	<0.001*	2.55(1.74-3.74)
Overall % predicted=87.9%			

DISCUSSION

Concerning the demographic characteristics of the studied groups, there were statistically significant difference among both groups regarding age (53.08±5.96 in stable coronary artery disease versus 59.61±12.19 in AMI), sex (Male to female ratio was 91.7/8.3 in stable coronary artery disease and 71.8/28.2 in AMI) and BMI (27.44±1.69 in stable coronary artery disease versus 31.0±5.19 in AMI) being increased in AMI groups (P<0.05), with no significant difference regarding marital status (P>0.05).

It was demonstrated that, the incidence of AMI was eight to nine times greater in men and women aged 55 to 64 years [(4)], while **Doughty *et al.*** [(5)] demonstrated that, 4 to 10 percent of patients with AMI were ≤40 or 45 years of age.

In terms of the medical history, HTN, DM and dyslipidemia were significantly increased among AMI cases compared to (SCAD) ones (P<0.05), while smoking was associated with higher incidence of AMI but not reaches the statistical significance (P>0.05).

Balgobin *et al.* [(6)] displayed that, AMI is associated with modifiable and non-modifiable risk factors such as known hypertensive syndromes, known CAD, hyperlipidemia, thrombophilia states, substance abuse history, smoking history, obesity, multiple comorbidities, Medicaid insurance status, and Black race.

In the same line, **Pandit** [(7)] demonstrated that uncontrolled DM and HTN were commonly associated with the development of AMI.

Concerning smoking, **Banks *et al.*** [(8)] demonstrated that current smoking increases the risk of virtually all CVD subtypes, at least doubling the risk of many, including AMI and heart failure. Paroxysmal tachycardia is a newly identified smoking-related risk.

The current study revealed that, family history of CAD, prior AMI, prior PCI, drug history (β-blocker and Dual anti-platelet) and sudden death were demonstrated to be significantly increased among AMI cases compared to (SCAD) ones (P<0.05), while thrombolytic and stenosis were significantly increased among unstable angina ones (P<0.05).

The present study displayed that, there was statistically significant decrease in platelet count and statistically significant increase in MPV in AMI cases compared to unstable angina ones (P<0.05).

This came in accordance with **Khode *et al.*** [(9)] who conducted their study on 128 subjects; 39 patients with acute myocardial infarction (AMI), 24 patients with stable coronary artery disease (SCAD) and 65 controls. They demonstrated that, the MPV was significantly higher in patients with AMI (9.65 ± 0.96) as compared to SCAD (9.37 ± 0.88) and controls (9.21 ± 0.58).

In the same line, **Chu *et al.*** [(10)] performed a systematic review and meta-analysis investigating the correlation between MPV and AMI, all-cause mortality following myocardial infarction, and restenosis following coronary angioplasty. Pooled results from 16 cross-sectional studies involving 2809 patients investigating the correlation of MPV and AMI indicated that MPV was significantly higher in those with AMI than those without AMI (P<0.001).

In subgroup analyses, significant differences in MPV existed between subjects with AMI, subjects with stable coronary disease (P<0.001), and stable controls (P<0.001), but not vs. those with unstable angina (P=0.24) [(10)].

Pooled results from three cohort studies involving 3184 patients evaluating the risk of death following AMI demonstrated that an elevated MPV increased the odds of death as compared with a normal MPV (P=0.012). Pooled results from five cohort studies involving 430 patients who underwent coronary angioplasty revealed that MPV was significantly higher in patients who developed restenosis than in those who did not develop restenosis (P<0.001) [(10)].

MPV seems to play a role in mediating reperfusion injury. In patients with STEMI scheduled for PCI, MPV at admission may be a valuable discriminator of a higher-risk patient subgroup, and a useful guide when deciding whether adjunctive therapy may be necessary to improve outcomes [(11)].

In harmony with the current study, **Mercan *et al.*** [(12)] displayed that, MPV was detected to be significantly lower instable angina group, compared to unstable angina and AMI groups (p<0.001, p<0.001) respectively, whereas neither a significant difference was determined between stable angina and AMI groups in terms of MPV (p=0.126) nor was it between unstable angina and AMI groups (p=0.999). Thus, they concluded that; mean platelet volume was detected to be increased in patients with acute coronary syndrome. In addition, some investigators have suggested that an increase in MPV over time denote high residual platelet reactivity after conventional dual antiplatelet therapy in patients who have undergone PCI [(13)].

Chu *et al.* [(14)] observed that, the ACS group had significantly higher MPV values (10.8±0.86 fl vs 9.8±0.76 fl) compared to non-ACS group (p<0.001). Moreover, they found that the MPV values were higher in patients with AMI) (n=28 as compared with patients with Sable coronary artery disease (SCAD) (n=41) (11.0±0.79 fl vs 10.6±0.87 fl, p=0.027). Moreover, **Choi *et al.*** [(15)] suggested that MPV was

superior to platelet function testing in terms of predicting cardiac death or cardiovascular events in patients who had undergone PCI, particularly those in an acute coronary syndrome subgroup.

There are also studies conflicting with above-mentioned results implying a correlation between increased platelet volume and AMI. **Halbmayer et al.** [16] in their study comparing those patients waiting for coronary artery bypass graft surgery to the control group, did not detect any difference in terms of MPV. Thus, they claimed that MPV could not be used as risk indicator for coronary artery disease or AMI. Likewise, no significant difference was found between unstable angina pectoris and control arms in terms of MPV, in the study consisted of 54 patients with unstable AP and performed by **Butkiewicz *et al.*** [17]**.** In addition, increased platelet volume has been shown to be more reactive with greater production of thromboxane A2 and serotonin [18]**.** Finally, larger platelets are more often reticulated, and this is an independent predictor of poor response to dual antiplatelet therapy [19]**.**

The present study displayed that, MPV could be used as a significant predictor **(P=0.001)** in the differentiation between AMI from SCAD cases (at cut off 7.45) with sensitivity, specificity, PPV, NPV and accuracy of 95.8, 72.2, 87.2, 89.7 and 87.9 respectively.

In the same line, **Khode *et al.*** [9] revealed that, the best cut-off values for MPV when predicting AMI and SCAD in patients were 9.25 fl (sensitivity 56.4%; specificity 45.9%) and 9.15 fl (sensitivity 54.2%; specificity 42.23%), respectively.

In addition, **Yasar *et al.*** [20] demonstrated that, Patients with insufficient TIMI flow had a significantly higher mean admission MPV (9.8+/-1.5 fl vs. 8.6+/-1.4 fl; p<0.001). In addition, the receiver operating characteristic analysis yielded a cutoff value of 8.885 fl for MPV to predict insufficient TIMI flow, with sensitivity and specificity being 70.4% and 66.1%, respectively.

Regarding the correlation between MPV value and demographic characteristics among AMI group, AMI cases were demonstrated to be significantly associated with advanced age as well as higher BMI (P<0.05). With regard to the correlation between MPV value, medical and family history among AMI group, DM, dyslipidemia, smoking, family history of CAD, prior AMI and prior PCI were demonstrated to be significantly increased among AMI cases.

In terms of the correlation between MPV value and laboratory, treatment and outcome among AMI group, there were no statistically significant correlation among MPV and all parameters (P>0.05), except stenosis which demonstrated significant correlation.

Concerning the correlation between MPV and mortality in AMI cases, the current study displayed that, MPV could predict death among AMI cases (At cut off 7.45,) with sensitivity, specificity, PPV, NPV and accuracy of 95.8, 72.2, 87.2, 89.7 and 87.9 respectively.

This was in accordance with **Huczek *et al.*** [21] research who performed Kaplan-Meier survival analysis which showed six-month mortality rate of 12.1% in patients with high MPV versus 5.1% in low MPV group (p = 0.0125). After adjusting for baseline characteristics, high MPV remained a strong independent predictor of no-reflow (p < 0.0001) and mortality (p = 0.0084). They concluded that, MPV is a strong, independent predictor of impaired angiographic reperfusion and six-month mortality in STEMI.

In addition, **Mohammed *et al.*** [18] **and Lekston *et al.*** [22] displayed that, MPV is a strong independent predictor of impaired angiographic reperfusion, in-hospital major adverse cardiovascular events, and 30-day, 6-month, 12-month, and 2-year mortality from STEMI treated via primary PCI.

Also, **Avci *et al.*** [23] displayed that, high MPV was an independent predictor of all-cause mortality ((HR: 1.301 [1.070–1.582], p=0.008). Rising MPV during hospitalization in STEMI patients treated with pPCI was associated with long-term mortality.

CONCLUSION

It could be concluded that, platelet counts were decreased, and mean platelet volumes were increased in AMI patients. Based on these findings, the current study concluded that larger platelet volumes may be used as predictor for AMI as well as ischemic complications. For this purpose, we think that MPV measurement, which is a non-invasive and easy-to-perform method, may be a tool for the follow-up of these patients.

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