Platelet count and MPV, routinely measured but ignored parameters used in conjunction with the diagnosis of acute coronary syndrome: single study center in Iranian population, 2010

Ali Zare Mirzaie[1], Maryam Abolhasani[2], Bina Ahmadinejad[3], Mahshid Panahi[4]

Department of Pathology, Rasoul-e-Akram Hospital, Tehran University of Medical Sciences, Tehran, Iran.

Abstract

Background: Myocardial infarction is the major cause of morbidity and mortality in industrialized countries. Platelet count and the mean platelet volume (MPV), a simple and reliable indicator of platelet size which correlates with platelet activation, might associate with troponin in acute chest pain.

Methods: We analyzed MPV of 851 patients who were admitted to Rasoul-e-Akram Hospital with acute chest pain during the year 2010. Two blood samples were taken from each patient within 4 hours of their arrival for routine hematology, including platelet count and MPV, and cardiac troponin T. Also, electrocardiograms of the patients were recorded. Association of MPV and platelet count with troponin was observed.

Results: The patients in troponin positive group, who had also ischemic electrocardiographic changes, had higher MPV values than non- acute coronary syndrome (ACS) patients with normal cardiac troponin T levels (9.9 *vs* 9.5 fl with $p < 0.001$). In troponin negative group, the mean of platelet count was higher than that in the positive group (221683 *vs* 198814/µl with $p < 0.001$).

Conclusion: MPV and platelet count are inexpensive laboratory tests which can be measured in association with other laboratory biomarkers in patients presenting with acute chest pain.

This could help to lower hospitalization rates and also avoid misdiagnosis and having complications of patients with ACS.

Keywords: MPV, Troponin, Platelet count, Chest pain.

Introduction

Myocardial infarction is the major cause of morbidity and mortality in industrialized countries (1). Acute coronary syndrome (ACS) is a set of signs and symptoms caused by rupture of an arterial plaque which provokes platelet–rich coronary thrombus formation. The thrombus leads to partial or complete coronary artery occlusion, which, in turn, results in myocardial ischemia and various clinical manifestations ranging from unstable angina (UA) to acute myocardial infarction (AMI) (2). Platelets are blood cells with diverse sizes and densities. In addition to aggregation, platelets modulate important pathophysiological processes, including inflammation and coagulation. Larger platelets generally contain more dense granules, express more glycoprotein llb/llla receptors, have a higher thromboxane A2 level and aggregate more rapidly with collagen, and thus are considered to be more active. It has been shown

1. Department of Pathology, Assistant Professor, Rasoul-e-Akram Hospital, Tehran University of Medical Sciences, Tehran, Iran. zaremir@gmail.com
2. **(Corresponding author)** Department of Pathology, Assistant Professor, Hasheminejad Kidney Center, Tehran University of Medical Sciences, Tehran, Iran. mar.abolhasani@gmail.com
3. Department of Pathology, General Practitioner in Rasoul-e-Akram Hospital, Tehran University of Medical Sciences, Tehran, Iran. drasal63@yahoo.com
4. Resident of pathology, Tehran University of Medical Sciences, Tehran, Iran. mahshid_panahi@yahoo.com

that platelet size, measured as mean platelet volume (MVP), is a marker of platelet function and is positively associated with platelet activity. An increased MPV is an indicator of larger and more active platelet, associated with myocardial damage in ACS, and is an unfavorable predictive factor in AMI survivors (3-4).

This study aimed to investigate whether there is an association between MPV and platelet count measured at admission with cardiac troponin T (cTnT) elevation in a large sample of patients with acute chest pain who were admitted to the emergency departments.

Methods

851 patients were selected with acute chest pain who were admitted to the emergency department of Rasoul-e-Akram Hospital during the year 2010. Two blood samples were taken from each patient in 4 hours of their arrival: one for routine hematologic tests and the other for cTnT. Peripheral venous blood for hematologic testing and cTnT measurements was collected in tubes containing K2–ethylenediaminetetraacetic acid and lithium heparin respectively. The cTnT samples were centrifuged at 1500g for 10 minutes at room temperature and immediately were analyzed by Roche cardiac reader. The samples for hematologic test, including platelet count and MPV, were assayed on cell counter of Sysmex kx 21. The cTnT results were stratified into two groups: troponin positive (troponin ≥0.1μg/L) and negative (troponin<0.1 μg/L). All patients underwent a standard

12-lead electrocardiogram (ECG), which was interpreted by the cardiologists of the hospital according to conventional criteria. Patients with ACS did not undergo coronary angiography. All the patients with acute chest pain who were admitted to the emergency department of our hospital during the year 2010 were included in the study. Patients without cTnT, platelet count or MPV in their laboratory results, or ECG due to any reasons were excluded from the study.

Statistical Analysis

Significance of differences and frequency distribution of values between the groups were assessed by the Mann-Whitney U-test. Statistical analyses were performed using statistical software SPSS. P<0.05 was considered statistically significant.

Results

The baseline characteristic of our study population and the main results of this investigation are shown in Table 1. A total of 851 were enrolled in the study. 169 (19.9%) of admitted patients had positive troponin and 682 (80.1%) had negative results.

MPV and platelet count are significantly higher and lower in troponin positive group than troponin negative group respectively.

In troponin negative group, the mean of MPV was 9.57±2.054 fl with max: 13.9 and min: 5.7. The mean age of the subjects was 62.62± 32.14 years with max: 100 and min: 18.The mean of platelet count was 221683±167032/μl with max: 939000 and min: 26000.

Table 1. MPV and platelet counts in troponin positive and negative groups.

Troponin	Patients with negative troponin (n=682)		Patients with positive troponin (n=169)		p
Patients' characteristics	Mean		Mean		
	Statistic	Standard Error	Statistic	Standard Error	
Age (yr)	62.62 (range:18-100)	0.62	68.59 (range:22-92)	1.07	<0.005
Platelet count (/μl)	221683 (range:26000-939000)	3198	198814 (range:26000-637000)	6776	<0.001
MPV (fl)	9.57 (range:5.7-13.9)	0.04	9.92 (range:7.5-13.4)	0.09	<0.001

In troponin positive group, the mean of MPV was 9.92± 2.37 fl with max: 13.4 and min: 7.5.The mean age of the subjects was 68.59± 27.92 years with max: 92 and min: 22.The mean of platelet count was 198814± 176166/µl with max: 637000 and min: 26000.

As it is shown in Fig. 1 and Table 1, in troponin positive group, MPV values are significantly higher than that those in troponin negative group (9.92 vs. 9.57 fl with p<0.001), (Fig. 2 and Table 1), and platelet count is significantly higher than that in troponin positive group (221683 vs 198814/µl with p< 0.001). Also the age of patients in troponin negative group was significantly lower than troponin positive group (62.62 vs. 68.59 with p value <0.005) (Table 1).

Discussion

Chest pain is a commonly presented symptom in hospitals. However, a minor group of patients admitted to the emergency department with chest pain are proved to have a cardiac etiology using electrocardiography and cTnT measurement (5). Current cardiac markers are not sensitive enough for early diagnosis of ACS (6), which is a leading cause of mortality. Physicians always search for rapid and independent markers for early and accurate diagnosis of ACS. Now the question need to be addressed whether these required markers are novel and expensive or they are ignored markers that could contribute to cells known to pathogenesis of thromboemboli.

Platelets play a pivotal role in atherogenesis (7). Platelet activation ultimately leads to the formation of thromboxane A_2, a potent vasoconstrictor and platelet aggregating substance, or leukotrienes, strong mediators of acute inflammatory response (8). Larger hyperactive platelets play an important role in intracoronary thrombus formation and acute thrombotic events (9). Decrease in platelet count can be due to participation of platelets due to thrombus process (10).
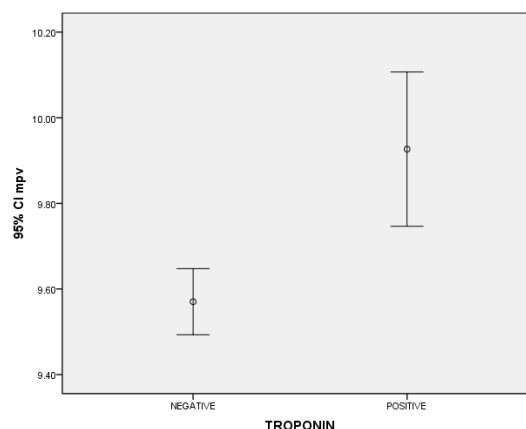
Recently, there has been some focus on

Fig. 1. Comparison of MPV in troponin positive and negative groups. MPV values were significantly higher in troponin positive group than troponin negative group.
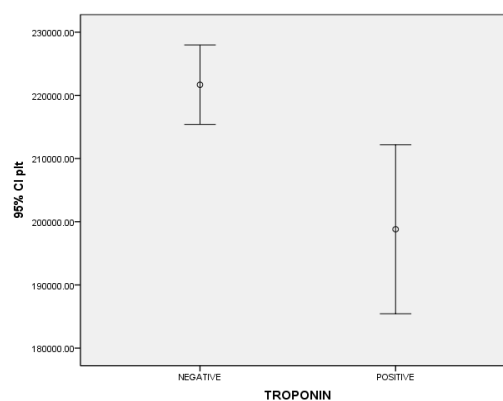

Fig. 2. Comparison of platelet count in troponin positive and negative groups. Platelet counts were significantly lower in troponin positive group than troponin negative group.

platelet count and MPV for aiding the diagnosis of the ACS. The reason is these simple, inexpensive tests available in routine laboratories and used for nearly every patient admitted to emergency room by a routine CBC test. If these parameters are proven to be valuable in the diagnosis of ACS, then medical cost would be far less for patients.

Previous studies have shown for diagnosis of various illnesses that increased MPV or a decrease in platelet count might be useful. They can be conjunct with other conventional biochemical cardiac markers understanding etiology for patients admitted with chest pain. (8,10-15). It has also been

reported that MPV measurement at admission might be valuable in the prediction of the infarct-related artery patency (16) and is also a useful hematological marker for early and easy identification of patients with stable CAD who are at a higher risk of post percutaneous coronary intervention low-reflow (17). Finally, elevated MPV and resistance to aspirin have proven to be prognostic factors, for death, MI and the composite endpoint (18).

To the best of our knowledge, MPV and PLT count had not been previously studied in Iranian population presenting with chest pain. Our study was performed on the patients who were admitted to the emergency department of Rasoul-e-Akram Hospital for chest pain. The findings indicated that MPV level at admission is significantly higher in patients who have elevated troponin levels than those with normal levels. Also, PLT count at admission was significantly lower in patients who have elevated troponin levels than those with normal troponin levels.

In our study, the electrocardiography of the patients was also recorded and interpreted by the cardiologists according to conventional criteria for ACS. The patients were diagnosed with ACS according to the presence of suggestive cardiac symptoms, ischemic electrocardiographic changes, and elevation of cTnT. Based on the obtained results, elevated MPV and low PLT count are noted in ACS.

The obtained data have confirmed the results of previous studies (8, 10-15). Our findings provide further evidence that platelet activation, measured by elevated MPV and low PLT count, may contribute to the pathogenesis of thrombosis-related complications in coronary artery disease (19).

These valuable tests may also have some other advantages. High MPV may show adverse prognosis (18, 20-21) and therapeutic needs (20). Patients with ACS having high pre-procedural MPV values might benefit from an intensified antiplatelet therapy after coronary interventions (20). MPV is also considered as a cost-effective tool that may be used even for predicting the possibility of acute coronary events (14).

These simple parameters which are already available in routine laboratories can help with accurate diagnosis of ACS without adding an economic burden. Also, it can give us some insight about prognosis and therapeutic management of patients and predicting the possibility of acute coronary events in patients who do not fulfill the criteria of ACS on arrival.

Conclusion

MPV and PLT count at admission are significantly higher and lower respectively in patients diagnosed with ACS than in those with chest pain of non-cardiac origin. Therefore, these simple and inexpensive laboratory measurements can be used in conjunction with other laboratory tests for patients admitted to the emergency department with chest pain. This could help to lower hospitalization and also misdiagnosis and having complications in patients with ACS. Nevertheless, further randomized clinical trials are needed to confirm the clinical usefulness of MPV and PLT count in risk stratification scoring systems that can predict myocardial infarction in admitted patients with ACS.

References

1. Mckarns SC, Smith. CJ, Payne VM. Doolittle DJ. Blood parameters associated with atherogenic and thrombogenic risk in smokers and nonsmokers with similar life-styles. Mod Pathol 1995 May; 8(4):434-40.

2. Chu H, Chen WL, Huang CC, Chang HY, Kuo HY, Gau CM, et al. Diagnostic performance of mean platelet volume for patients with acute coronary syndrome visiting an emergency department with acute chest pain: the Chinese scenario. Emerg Med J 2011 Jul; 28(7):569-74.

3. Kilicli-Camur N, Demirtunc R, Konuralp C, Eskiser A, Basaran Y. Could mean platelet volume be a predictive marker for acute myocardial infarction? Med Sci Monit 2005 Aug; 11(8):387-92.

4. Endler G, Klimesch A, Sunder-Plassmann H, Schillinger M, Exner M, Mannhalter C, et al, Mean platelet volume is an independent risk factor for myocardial infarction but not for coronary artery disease. Br J Haematol 2002 May; 117(2):399-404.

5. Sequist TD, Bates DW, Cook EF, Lampert S, Schaefer M, Wright J, et al. Prediction of missed myocardial infarction among symptomatic outpatients without coronary heart disease. Am Heart J 2005 Jan; 149(1):74-81.

6. Alan H. B. Wu. Cardiac markers, 2nd ed. Totowa, New Jersey: Humana Press; 2003.pp.344-350.

7. Yilmaz MB, Cihan G, Guray Y, Guray U, Kisacik HL, Sasmaz H, et al. Role of mean platelet volume in triagging acute coronary syndromes. J Thromb Thrombolysis 2008 Aug; 26(1):49-54.

8. Lippi G, Filippozzi L, Salvagno GL, Montagnana M, Franchini M, Guidi GC, et al. Increased mean platelet volume in patients with acute coronary syndromes. Arch Pathol Lab Med 2009 Sep; 133(9):1441-3.

9. Smith NM, Pathansali R, Bath PM. Platelets and stroke. Vasc Med 1999; 4(3):165-72.

10- Mathur A, Robinson MS, Cotton J, Martin JF, Erusalimsky JD. Platelet reactivity in acute coronary syndromes: evidence for differences in platelet behaviour between unstable angina and myocardial infarction. Thromb Haemost 2001 Jun; 85(6):989-94.

11. Huczek Z, Filipiak KJ, Kochman J, Michalak M, Roik M, Piatkowski R, et al. Baseline platelet size is increased in patients with acute coronary syndromes developing early stent thrombosis and predicts future residual platelet reactivity, A case-control study. Thromb Res 2010 May; 125(5):406-12.

12. Cemin R, Donazzan L, Lippi G, Clari F, Daves M. Blood cells characteristics as determinants of acute myocardial infarction. Clin Chem Lab Med 2011 Jul; 49(7):1231-6.

13. Chu H, Chen WL, Huang CC, Chang HY, Kuo HY, Gau CM, et al. Diagnostic performance of mean platelet volume for patients with acute coronary syndrome visiting an emergency department with acute chest pain: the Chinese scenario. Emerg Med J 2011 Jul; 28(7):569-74.

14. 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 Feb; 59(2):146-9.

15. Avramakis G, Papadimitraki E, Papakonstandinou D, Liakou K, Zidianakis M, Dermitzakis A, et al. Platelets and white blood cell subpopulations among patients with myocardial infarction and unstable angina. Platelets 2007 Feb; 18(1):16-23.

16. Maden O, Kacmaz F, Selcuk MT, Selcuk H, Metin F, Tufekcioglu O, et al. Relationship of admission haematological indices with infarct-related artery patency in patients with acute ST-segment elevation myocardial infarction treated with primary angioplasty. Coron Artery Dis 2007 Dec; 18(8):639-44.

17. Duygu H, Turkoglu C, Kirilmaz B, Turk U. Effect of mean platelet volume on postintervention coronary blood flow in patients with chronic stable angina pectoris. J Invasive Cardiol 2008 Mar; 20(3):120-4.

18. Aksu H, Ozer O, Unal H, Hobikoglu G, Norgaz T, Buturak A, et al. Significance of mean platelet volume on prognosis of patients with and without aspirin resistance in settings of non-ST-segment elevated acute coronary syndromes. Blood Coagul Fibrinolysis 2009 Dec; 20(8):686-93.

19. Yetkin, E. Mean platelet volume not so far from being a routine diagnostic and prognostic measurement. Thromb Haemost 2008 Jul; 100(1):3–4.

20. Yang A, Pizzulli L, Lüderitz B. Mean platelet volume as marker of restenosis after percutaneous transluminal coronary angioplasty in patients with stable and unstable angina pectoris. Thromb Res 2006; 117(4):371-7.

21. Goncalves SC, Labinaz M, Le May M, Glover C, Froeschl M, Marquis JF, et al. Usefulness of mean platelet volume as a biomarker for long-term outcomes after percutaneous coronary intervention. Am J Cardiol 2011 Jan; 107(2):204-9.