International Journal of Advances in Medicine
Biradar SB et al. Int J Adv Med. 2016 May;3(2):349-352
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20161089

Research Article

Platelet volume indices in acute coronary syndrome - a case control study

Satish Basanagouda Biradar[1], Sangappa Virupaxappa Kashinakunti[2]*, Manjula R.[3]

[1]Department of General Medicine, [2]Department of Biochemistry, [3]Department of Community Medicine, S. Nijalingappa Medical College, Navanagar, Bagalkot, Karnataka, India

Received: 27 January 2016
Accepted: 27 February 2016

***Correspondence:**
Dr. Sangappa Virupaxappa Kashinakunti,
E-mail: drsvkashinakunti@yahoo.co.in

ABSTRACT

Background: Acute coronary syndrome (ACS) is a set of signs and symptoms due to rupture of a plaque which are a consequence of platelet- rich coronary thrombus formation. The aim of study was to compare the platelet volume indices (PVI) in acute coronary syndrome with healthy controls.
Methods: The study was carried out on 100 cases diagnosed with acute coronary syndrome and 50 controls from June 2012 to September 2014.
Results: All platelet volume indices i.e. mean platelet volume (MPV), platelet distribution width (PDW) and platelet large cell ratio (PLCR) were significantly raised in patients with acute myocardial infarction (AMI) and unstable angina (UA), (MPV 9.2 fl, PDW 11.3 fl, PLCR 19.3 fl,) compared to those with control group (MPV 8.6 fl, PDW 10.2 fl, PLCR 15.8 fl).
Conclusions: PVI are simple investigations that can be used for predicting impending acute cardiovascular events.

Keywords: Platelets, Unstable angina, ST segment, Myocardial infarction

INTRODUCTION

Acute coronary syndrome (ACS) is a set of signs and symptoms due to rupture of a plaque which are a consequence of platelet- rich coronary thrombus formation. The Thrombus leads to partial or complete coronary artery occlusion which in turn leads to myocardial ischemia and various clinical manifestations ranging from unstable angina (UA) to acute myocardial infarction (AMI).[1,2]

Platelets play a crucial role in pathogenesis of atherosclerotic complications, contributing to the thrombus formation after plaque rupture.[3,4] Increased platelet reactivity as well as shortened bleeding time is associated with increased platelet volume.[5] Large platelets that contain more dense granules are metabolically and enzymatically more active than small platelets and they have higher thrombotic potential.[6] They also express higher level of procoagulatory surface proteins such as p-selectin and glycoprotein III a.[7-9]

Platelets activation is a hallmark of ACS. It has been shown that platelet size when measured as mean platelet volume (MPV) is a marker of platelet function and is positively associated with indicators of platelet activity. An increased MPV an indicator of large and more reactive platelets has been associated with myocardial damage in ACS and has been found to be predictive of an unfavourable outcome, among survivors of AMI.[10,11] Previous studies have documented ethnic difference in MPV level.[12-15] Association of higher MPV values with ACS has been mostly studied among Caucasian patients.[16] A few reports have revealed larger MPV values in Indian patient with ACS compared to healthy control.[17] However there are less reports in comparison with stable coronary artery disease.

Platelet distribution width (PDW) and platelet large cell ratio (PLCR) are extended panel of PVI. The PDW is the platelet distribution width measured at 20% relative height of the total height of the curve depicting the distribution of them. An increased PDW is an indicates anisocytosis of platelets. The normal range for PDW is 9 to 14 fL. The PLCR indicates the percentage of large platelets with a volume >12 fL. The percentage of large platelets normally ranges from 15 to 35%. An increase in PLCR shows the presence platelet aggregates, micro erythrocytes and giant platelets.[18] Rechciński T et al in their study found that PDW and PLCR, can serve as useful prognostic factors for long-term mortality in patients after acute MI. PDW was found to be one of the independent risk factors of cardiac mortality, as well as of the occurrence of either death, recurrent MI or need for another revascularization procedure.[18]

Thus platelets indices are important simple effortless and cost effective tool that can be used and explored extensively especially in countries such as India for predicting the possibility of impending acute coronary events. Hence present study was undertaken to compare the platelet indices viz, MPV, platelet distribution width (PDW), platelet large cell ratio (PLCR) in UA patients and ST segment elevated myocardial infarction (STEMI) with healthy controls.

METHODS

The study was carried out on 100 patients admitted at Hanagal Shri Kumareshwara hospital Bagalkot, from June 2012 to September 2014. Ethical clearance was obtained from institutional ethics committee. Informed consent was obtained from all the participants. Patients more than 18 years of age diagnosed with UA, STEMI were included in the study. Patient having any platelet disorder, bleeding or clotting disorder were excluded from the study. Age and sex matched having a normal ECG and no past history of ischemic heart disease were considered for the study as controls.

Three groups were studied, group A: patients with STEMI; group B: patient with UA; group C: controls. Each consisting of 50 participants.

Method of collection of data

The study was carried out on patient presenting with ACS within 24 hours. All subjects were interviewed as per the predesignated proforma and complete clinical examination was done. Under aseptic precautions, 5 ml blood sample was collected in EDTA vaccutanier from the antecubital vein. The sample was run within 2 hours of vein puncture. Using the 3 part differentiated automated hematologyanalyzer (sysmex KX-21) and complete count analysis of the sample was made including the platelet indices (MPV, PDW, PLCR). Relevant investigations like ECG and cardiac enzymes were analysed for confirmation of the diagnosis. Troponin I sensitive kit was used for diagnosis of myocardial injury.

SPSS for window version; SPSS, 11.5 Inc., Chicago IL was used for statistical analysis. All the values were expressed in mean±SD.

RESULTS

The mean age of the participants in our study was 55±10 years. Majority of the patient diagnosed as ACS belonged to 6th decade of life (51 patients, 34%) followed by 7thdecade (44 patients, 29%) of life. In the present study 61.33% were males and 38.66% were females. The number of males in ACS group was 62 (41.33%) and females in ACS group were 39 (26%). In risk factors, smoking was highest in cases followed by diabetes mellitus and hypertension (Table 1).

Table 1: Risk factors in cases and controls.

Risk factors	Cases	Controls
Smoking	26	13
Alcohol consumption	1	0
Diabetes mellitus	20	8
Hypertension	18	9

Table 2: Comparison of platelet indices between STEMI and controls.

Parameter	STEMI	Controls	t	p
MVP fl	9.2±0.7	8.6±0.7	4.6	0.000
PDW fl	11.3±1.5	10.2±1.5	3.5	0.000
PLCR fl	19.2±5.4	15.8±4.5	3.3	0.001

STEMI: ST Segment elevated myocardial infraction, MPV: Mean platelet Volume, PDW: Platelet distribution width, PLCR: Platelet large cell ratio.

Table 3: Comparison of platelet indices between unstable angina and controls.

Parameter	UA	Controls	t	p
MVP fl	9.2+1.0	8.6+0.7	3.3	0.001
PDW fl	11.3+2.1	10.2+1.5	2.81	0.006
PLCR fl	19.4+7.7	15.8+4.5	2.7	0.008

UA: Unstable angina, MPV: Mean platelet volume, PDW: Platelet distribution width, PLCR: Platelet large cell ratio.

Comparison of platelet indices between STEMI and control showed statistically significant increase in all the platelet indices (MPV, PDW, PLCR, p value0.000, 0.000, 0.001 respectively) in STEMI compared to controls (Table 2). Similarly comparison between UA and control showed statistically significant increase in MPV, PDW, PLCR, p value 0.001, 0.006, 0.008 respectively (Table 3).

Comparison of platelet indices in UA and STEMI showed statistically non-significant differences in all parameters (MPV, PDW, P-LCR, p value 0.860, 0.947, 0.899 respectively) (Table 4).

Table 4: Compassion of platelet indices in unstable angina and STEMI.

Parameter	UA	STEMI	t	p
MVP fl	9.1±1.0	9.1 ±0.6	0.177	0.860
PDW fl	11.2±2.0	11.3±1.4	0.067	0.947
PLCR fl	19.3±7.6	19.2±5.3	0.133	0.899

UA: Unstable angina, STEMI: ST Segment elevated myocardial infraction, MPV: Mean platelet volume, PDW: Platelet distribution width, PLCR: Platelet large cell ratio.

DISCUSSION

The current study showed increased PVI among UA and STEMI compared to controls. The MPV, PWD, PCLR levels were significantly higher among patients with UA compared to controls. Similarly MPV, PWD, PCLR levels were significantly higher among patients with STEMI compared to controls. But there was no significant difference of PVI, when compared between UA and STEMI.

Yaghoubi A et al[1], study the mean age of 63.08±13.65 years, which was 8 years more than the mean age of the patients in the Vakili H study in Iran in 2009, but in the current study mean age all the participants was 55±10 years.[19,20] Besides 61.33% were males these finding were similar to Yaghoubi A et al 64.84% were men.[19] Distribution of risk factors was similar to that in another study carried out specifically on the relation between the risk factors of ACS and MPV.

In Yaghoubi A et al study, MPV increased significantly in MI patients compared to the controls, Varol E et al, Cemin R et al and Yilmaz et al also found that MPV was significantly higher in patients with ACS groups than controls.[19,21-23] In the present study also, MPV was statistically significantly increased in STEMI and UA compared to the controls, hence the current is in accordance with all above studies. Yaghoubi A et al[19] study showed that difference in MPV between MI patients and UA patients was not significant, like Khandekar et al in India.[19,24] In present study also there is no significant difference in MPV in STEMI and UA, but contrary to Yilmaz et al.[23]

Our study reveals that the PDW was significantly higher in patients with ACS than controls; these results are in accordance with Pervin S et al Nandwani S et al and Khandekar MM et al described in their studies all platelet volume indices including MPV, PDW and PLCR were increased significantly in patients with ACS than controls, our study also all the platelets indices were increased in STEMI and UA compared to the controls.[24-26] In case of PDW and MPV, Pervin S et al significant differences between the groups were found.[25] There are two hypotheses that described the increase in these parameters. First, when platelets are activated they change their size and shape (metamorphosis). Second, after platelet activation aggregation of more platelet this leads to release of younger platelet from bone marrow. These suggest that MPV and PDW are indirect indicators of platelet activation and their association with ACS. Among the platelet parameters PDW was most significant than MPV. These findings lead to the hypothesis that larger platelets as determined by their volumes, MPV and PDW may be useful markers in patients with ACS.[25,27]

A retrospective case-control study on ACS patients, conducted by Bhayana A et al showed MPV was same for both cases and controls (8.04) and no significant statistical difference was found between mean PDW of cases and controls.[28]

Further studies are needed to evaluate whether MPV provides added value in identifying patients at enhanced clinical risk and whether therapeutic modification of this marker may lead to improved cardiovascular care.

In conclusion the increased platelet indices may contribute to the pre-thrombotic state in acute ischemic syndromes.

ACKNOWLEDGEMENTS

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Khode V, Sindhur J, Kanbur D, Ruikar K, Nallulwar S. Mean platelet volume and other platelet volume indices in patients with stable coronary artery disease and acute myocardial infarction: A case control study. J Cardiovasc Dis Res. 2012;3:272-5.
2. Abrol S, Sharma R, Badgal A, Kundal V, Chowdhary S. Mean Platelet Volume in Acute Coronary Syndrome: A prospective observational study. J of Evolution of Med and Dent Sci. 2015;4(10):1606-10.
3. Corash L, Chen HY, Levin J, Baker G, Lu H, Mok Y. Regulation of thrombopoiesis: effects of the degree of thrombocytopenia on megakaryocyte ploidy and platelet volume. Blood. 1987;70(1):177-85.
4. Davis MJ, Thomas A. Thrombosis and acute coronary lesion in sudden cardiac ischaemic death. N Eng J Med. 1984;310:1137-40.
5. Martin JF, Trowbridge EA, Salmon G, Plumb J. The biological significance of platelet volume: in relationship to bleeding time, thrombaxane b2 production and megakaryocytic nuclear DNA concentration. Thromb Res. 1983;32:443-60.
6. Eldor A, Avizour M, Or R, Hanna R, Penchas S. Production of haemorrhagic diathesis in thrombocytopenia by mean platelet volume. Br Med J. 1982;285:397-400.

7. Haver VM, Gear ARL. Functional fractionation of platelets. J Lab Clin Med 1981;97:187-204.
8. Thompson CB, Eaton KA, Priciotta SM, Kushkin CA, Valeri CR. Size dependent platelet subpopulations: relationship of platelet volume to ultrastructure, enzymatic activity and function. Br J Haematol. 1982;50:509-19.
9. Thompson CB, Jakubowski JA, Quinn PG, Deykin D, Valeri CR. Platelet size as a determinanat of platelet function. J Lab Clin Med. 1981;101:205-13.
10. 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;11:387-92.
11. Endler G, Klimesch A, Sunder-Plassmann H, Exner M, Mannhalter C, Jordanova N 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.
12. Farkouh ME, Smars PA, Reeder GS, Zinsmeister AR, Evans RW, Meloy TD, et al. A clinical trial of a chest-pain observation unit for patients with unstable angina. Chest Pain Evaluation in the Emergency Room (CHEER) Investigators. N Engl J Med. 1998;339:1882-8.
13. Schull MJ, Vermeule MJ, Stukel TA. The risk of missed diagnosis of acute myocardial infarction association associated with emergency department volume. Ann Eme Arg Med. 2006;48:647-55.
14. Lee TH, Rouan GW, Weisberg MC, Brand DA, Acampora D, Stasiulewicz C, et al. Clinical characteristics and natural history of patients with acute myocardial infarction sent home from the emergency room. Am J Cardiol. 1987;60:219-24.
15. Puleo PR, Meyer D, Whthen C, Tawa CB, Wheeler S, Hamburg RJ et al. Use of a rapid assay of forms of creatione kinase MB to diagnose or rule out acute myocardial infarction. N Engl J Med. 1994;331:561-6.
16. Bath PM, Buttrerworth RJ. Platelet size: measurent, physiology and vascular disease. Blood Coagul Fibrinolysis. 1996;7:157-61.
17. Ranjith MP, Divya R, Metha VK, Krishnan MG, KamalRaj R, Kavishwar A. Significance of platelet volume indices and platelet count in ischaemic heart disease. J Clin Pathol. 2009;62:830-3.
18. Rechciński T, Jasińska A, Foryś J, Krzemińska-Pakuła M, Wierzbowska-Drabik K, Plewka M et al. Prognostic value of platelet indices after acute myocardial infarction treated with primary percutaneous coronary intervention. Can J Cardiol. 2013;20(5):491-8.
19. Yaghoubi A, Golmohamadi Z, Alizadehasl A, Azarfarin R. Role of platelet parameters and haematological indices in myocardial infarction and unstable angina. JPMA. 2013;63:1133-7.
20. Vakili H, Kowsari R, Namazi MH, Motamedi MR, Safi M, Saadat H et al. Could mean platelet volume predicts impaired reperfusion and in hospital major adverse cardiovascular event in patients with primary percutaneous coronary intervention after ST elevation myocardial infarction? J The univ Heart Ctr. 2009;4:17-23.
21. Varol E, Icli A, Ozaydin M, Erdogan D, Arslan A. Mean platelet volume is elevated in patients with myocardial infarction with normal coronary arteries, as in patients with myocardial infarction with obstructive coronary artery disease. Scand J Clin Lab Invest. 2009;69(5):570-4.
22. Cemin R, Donazzan L, Lippi G, Clari F, Daves M. Blood cells characteristics as determinants of acute myocardial infarction. Clin Chem Lab Med. 2011;49(7):1231-6.
23. Yilmaz MB, Cihan G, Guray Y, Guray U, Halil LK, Sasmaz H, Korkmaz S. Role of mean platelet volume in triaging acute coronary syndrome. J thromb thrombolysis. 2008;26;49-54.
24. 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;59:146-9.
25. Pervin S, Islam SM, Ferdoushi S, Hossain M, Sultana T, Hoque MH, et al. Platelet distribution width is an early indicator of acute coronary syndrome. University Heart Journal. 2013;9(1):3-8.
26. Nandwani S, Bhatnagar M. Study of Platelet volume Indices in Platelet of Acute Coronary Events. JIAG. 2011;7:22-4.
27. Pervin S, Ferdousy S, Hossain M, AI J, Sultana T. Elevated mean platelet volume is a marker of acute coronary syndrome. Bangladesh Med J. 2013;42(2):45-50.
28. Bhayana A, Joshi D. Is large platelet size a risk factor for acute coronary syndrome: a retrospective case-control study. J MGIMS. 2009;14(ii):52-5.

Cite this article as: Biradar SB, Kashinakunti SV, Manjula R. Platelet volume indices in acute coronary syndrome - a case control study. Int J Adv Med 2016;3: 349-52.