

Research Article

Evaluation of platelet count and platelet indices in patients with coronary artery disease

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Abstract

Background: Ischemic heart disease is mainly caused by atherosclerosis and its complications. Platelets and their activity have an important role in initiation of atherosclerotic lesions and coronary thrombus formation. Larger platelets are enzymatically and metabolically more active and have a higher potential thrombotic ability as compared with smaller platelets.

Aims: To study the changes in platelet volume indices and platelet count in ischaemic heart disease and assess their usefulness in predicting coronary events.

Methods: This was a comparative study of 180 patients (60 patients with stable angina, 60 with acute coronary syndrome and 60 with non-cardiac chest pain). Blood venous sample were drawn from all subjects after admission and collected in EDTA tubes. Platelet count and volume indices were assayed within 30 min of blood collection, using Nihon Kohden autoanalyzer.

Results: All platelet volume indices—mean platelet volume (MPV), platelet distribution width (PDW), and platelet concentrate (PCT)—were significantly raised in patients with AMI and UA. In patients with myocardial infarction, the mean values of MPV, PDW, platelet count and PCT were 11.02fL, 17.85%, 2.61 lac/cumm and 0.34% respectively. In patients with unstable angina were 10.31fL, 16.75%, 2.3 lac/cumm and 0.36% respectively while in normal healthy control the mean values of these indices were 7.98 fL, 10.70%, 2.66 lac/cumm and 0.24% respectively.

Conclusions: Patients with acute coronary syndrome had higher platelet volume indices and lower platelet counts compared with those with stable angina and the normal population. Measurements of platelet volume indices and platelet count may be of some benefit in detecting those patients at higher risk for acute coronary events.

Keywords: Platelet count, platelet indices, mean platelet volume, platelet distribution width, platelet concentrate, Acute myocardial infarction, unstable angina

1. Introduction

Platelet activation plays a central role in the transformation of atherosclerotic cardiovascular disease (CVD) into its potentially major adverse clinical events, such as ischemic stroke and myocardial infarction (MI)¹⁻³. Increased platelet activation may also represent the net pathophysiological effects of a number of CVD risk factors, such as smoking and raised cholesterol, thus representing a broad marker of CVD risk⁴. Platelets play a key role in the development and progression of cardiovascular disease, with increased aggregation and activation occurring in patients with chronic stable angina and acute coronary syndrome (ACS)⁵. Circulating platelets are heterogeneous with respect to their size, density and reactivity. It is generally accepted that large platelets are metabolically and enzymatically more active than small ones^{6,7}. It has been postulated that large platelets may be an indicator of platelet activation, and thus be related to the extent and also clinical presentation of coronary artery disease (CAD)⁸. The degree of platelet activation may be assessed by platelet indices such as platelet count, mean platelet volume (MPV) and platelet distribution width (PDW). It is also unclear whether these parameters can be considered risk factors for CAD.

The WHO has drawn attention to the fact that CAD is a modern epidemic more in geriatric population i.e. people above 60 years of age. Large increase in CAD is projected and now it is the most common cause of death⁹. Platelets play a crucial role in pathogenesis of atherosclerotic complications, contributing to thrombus formation or apposition after plaque rupture^{10,11,12}. After rupture of arteriosclerotic plaque in coronary arteries, platelets hyperactivity and local platelets activation have been suggested to play a causal role in prothrombotic events leading to MI¹³. An increased platelet reactivity and shortened bleeding time are associated with increased platelet volume, therefore; platelet size has been considered to reflect platelet level of activity as the large platelets are more active than small platelets and they have a higher thrombotic potential due to high concentration of thromboxane A₂¹⁴.

2. Material and Method

This study was done in the laboratories of Dhiraj Hospital of Smt. B. K. Shah Medical Institute and Research Center for the period between June 2013 to August 2014. The subjects were divided into three groups:

- (1) First group includes Forty patients were admitted to the coronary care unit, Dhiraj Hospital with diagnosis of acute myocardial infarctions (AMI).
- (2) Second group includes Forty patients were admitted to coronary care units diagnosed as having unstable angina.
- (3) Third group includes Forty healthy control groups.

Data were translated into a computerized database structure. An expert statistical advice was sought for Statistical analyses were computer assisted using SPSS version 20 (Statistical Package for Social Sciences). The outcome quantitative variables were normally distributed and conveniently described by mean, SD (standard deviation) and SE (standard error), and the parametric statistical tests of significance were used. P value less than the 0.05 level was considered statistically significant.

2.1 Criteria of inclusion of the subjects

Patients with newly diagnosed acute myocardial infarction [STEMI and NSTEMI (cardiac enzyme activity positive)] and unstable angina (some patients had previous acute myocardial infarction).

Sample collection:

Blood samples were taken from both groups of patients and control. 2.0 ml of blood were withdrawn in syringes by clean venipuncture; blood was dispersed in ethylene diaminetetraacetic acid (EDTA) tube.

2.2 Methods

Blood sample (320 microliter) from EDTA tube were put in automated analyzer Nihon Kohden for hematology (analysis system).

Nihon Kohden analysis system give data like:

RBC, HGB, HCT, MCV, MCH, MCHC, RDW-CV, RDW-SD, PLT, PDW, PCT, MPV, Lym%, Neu%, Mix%, etc.

3. Results

Table No. 1: Age wise distribution of cases

Cases	Age (Mean \pm SD)
MI (40)	56.45 \pm 7.14
UA (40)	52.2 \pm 5.40
Control (40)	49.60 \pm 5.54
Total (120)	52.92 \pm 0.96

MI - Myocardial infarction; UA- Unstable angina

A total of 120 subjects were analyzed, 40 healthy controls with a mean age of 49 year (\pm 5.5) and 40 MI cases with a mean age 56.4 (\pm 7.14) and 40 cases with unstable angina with a mean age of 52.7 (\pm 5.40) and mean age of all cases 52.9 (\pm 0.96).

Table No. 2: Age and Sex wise distribution of cases

Cases	Sex	Age (Mean \pm SD)
MI	Male	29 (72.5%) 57.13 \pm 7.9
	Female	11 (27.5%) 54.63 \pm 4.0
UA	Male	23 (57.5%) 54.08 \pm 6.05
	Female	17 (42.5%) 50.82 \pm 3.76
Control	Male	25 (62.5%) 50.82 \pm 3.76
	Female	15 (37.5%) 48.12 \pm 5.13
Total		120 (100%)

Male constituted 62.5% of healthy control, 72.5% of MI and 57.5% of cases with unstable angina. No important differences were observed between 3 study groups. Female constituted 37.5% of healthy control, 27.5% of MI and 42.5% of cases with unstable angina. No important differences were observed between 3 study groups. Males have higher incidence of MI and UA than females.

In control group a total of 40 subjects were analyzed mean age of male 50 year (\pm 3.76) and for female mean age 48 year (\pm 5.13). In Cases of MI a total of 40 subjects were analyzed mean age of male 57.13 year (\pm 7.9) and for female mean age 54 year (\pm 4.0). In Cases of unstable angina a total of 40 subjects were analyzed mean age of male 54 year (\pm 6.05) and for female mean age 50 year (\pm 3.76).

Table no. : 3 Comparison of platelet count and platelet indices in all cases

Cases	Platelet Count (Mean \pm SD)	Mean Platelet volume (Mean \pm SD)	Platelet distribution width (Mean \pm SD)	PCT (Mean \pm SD)
MI	2.61 \pm 1.0	11.02 \pm 2.38	17.85 \pm 3.30	0.34 \pm 0.12
UA	2.30 \pm 1.0	10.31 \pm 2.11	16.75 \pm 3.9	0.36 \pm 0.13
Control	2.66 \pm 0.87	7.98 \pm 2.57	10.70 \pm 2.07	0.24 \pm 0.07
Total	2.52 \pm 0.96	9.44 \pm 2.59	14.67 \pm 4.40	0.31 \pm 0.12
p	NS	P<0.05	P<0.05	P<0.05

ANOVA analysis

The difference in mean platelet indices between the 3 study groups is shown in table 3 that was significantly higher in MI and UA groups compared to healthy control.

Mean platelet count in control group is 2.66 Lac (\pm 0.96), cases of MI 2.61 Lac (\pm 1.0) and in cases of unstable angina 2.30 Lac (\pm 1.0). Platelet count is higher in control group than other group.

Mean platelet distribution width in control group is 10.70 (\pm 2.07), in cases of MI 17.85 (\pm 3.30) and in cases of unstable angina 16.75 (\pm 3.9). Platelet distribution width is significantly higher in cases of MI and unstable angina than control group.

Mean platelet volume in control group is 9.78 (\pm 2.57), in cases of MI 11.02 (\pm 2.38) and in cases of unstable angina 10.31 (\pm 2.11). Platelet volume is significantly higher in cases of MI and unstable angina than control group.

Mean platelet concentrate in control group is 0.24 (\pm 0.07), in cases of MI 0.34 (\pm 0.12) and in cases of unstable angina 0.36 (\pm 0.13). Mean platelet concentrate is significantly higher in cases of MI and unstable angina than control group.

4. Discussion

Platelets are heterogeneous blood elements with diverse sizes and densities. Platelet activation is a hallmark of acute coronary syndrome. 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 larger and more reactive platelets, has been associated with myocardial damage in ACS and has been found to be predictive of an unfavorable outcome among survivors of AMI^{1,2}.

Platelet activation leads to the formation of free arachidonic acid, which can be transformed into prostaglandins, such as thromboxane A₂, one of the most potent vasoconstrictor and platelet-aggregating substances, or into leukotrienes, which can amplify the acute inflammatory response¹⁰. Platelet function and size correlate because larger platelets, produced from activated megakaryocyte in the bone marrow, are likely to be more reactive than normal platelets because large platelets contain more secretory granules and mitochondria and are known to be more active than small platelets¹⁴. Consequently, larger and hyperactive platelets play a vital role in accelerating the formation and propagation of intracoronary thrombus, leading to the occurrence of acute thrombotic events¹⁵.

Martin et al¹⁶ (India) found that MPV was significantly higher in those patients who have MI, compared with healthy group, which is confirmed by Senaran et al¹⁷ (India), both studies are similar to our study that MPV was significantly higher in patients of MI.

Mathur et al¹⁸ (Turkey) found platelet counts to be significantly lower and MPV to be higher in patients with unstable angina pectoris and MI. Different to our study, platelet count is increased significantly in MI and UA. MPV, in our study, is higher in both MI and UA which is similar to Mathur et al study.

In an Indian study conducted by Khandekar et al¹⁹, the authors suggested that all platelet volume indices are significantly raised in patients with acute myocardial infarction and unstable angina patients as compared to those with stable coronary artery disease.

Khode²⁰ found the mean platelet volume 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).

5. Conclusions

Larger platelets are haemostatically more active and are a risk factor for developing coronary thrombosis and subsequent acute coronary events (myocardial infarction and unstable angina). Patients with larger platelets can easily be identified during routine hematological analysis and could possibly benefit from preventive treatment.

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