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Original Research Article

The role of platelet indices in ischemic heart disease: a hospital based case control type of study

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ABSTRACT

Background: In developing countries, Ischemic heart diseases (IHD) is one of the leading causes of morbidity and mortality. The underlying pathology of CAD is atherosclerosis. When this atherosclerotic plaque ruptures, platelets play a crucial role in the prothrombotic events and forms a thrombus on this plaque and as a result coronary artery gets occluded causing ischemia and infarction. Platelet contains many chemokines, cytokines and growth factors. Release of these factors along with interaction with endothelial cells and leukocytes promotes inflammation and progression of atherosclerosis. We aimed to investigate the association between platelet volume indices in patients with diagnosis of Ischemic heart disease in comparison with control group.
Methods: By using automated cell counter platelet count and platelet volume indices - were compared with Normal healthy or non-cardiac chest pain patients with the use of unpaired t test.
Results: In the present study, we demonstrated that platelet count is significantly low and MPV and PDW are significantly high in Ischemic heart disease as compared to patients with noncardiac chest pain or healthy subjects. The correlation of MPV with PC revealed an inverse correlation between the patients of IHD and healthy or non-cardiac chest pain patients which is statistically significant.
Conclusions: The platelet volume indices are an important, simple, effortless and a cost-effective tool useful in predicting the development of an acute coronary event sometimes in the near future and therapeutic modification for improved patient's cardiovascular care.

Keywords: Atherosclerosis, Automated cell counter, Ischemic heart diseases, Platelet indices

INTRODUCTION

In developing countries, Ischemic heart diseases (IHD) is one of the leading causes of morbidity and mortality. The spectrum of presentation is wide and ranges from unstable angina to acute myocardial infarction.[1] Major risk factors for coronary artery disease (CAD) are age, family history, obesity, mental stress, diabetes mellitus (DM), hypertension (HTN), elevated low-density lipoproteins (LDL) and smoking. The underlying pathology of CAD is atherosclerosis. It initiates by second decade of life and progresses chronically to manifest as Acute Coronary Syndrome (ACS) in fourth and fifth decade of life. When this chronic atherosclerotic plaque ruptures, platelets play a crucial role in the prothrombotic events and forms a thrombus on this plaque and as a result coronary artery gets occluded causing ischemia and infarction. Approximately 70% of myocardial infarctions are caused by rupture of atherosclerotic plaque.[2] Platelets also plays important role in progression of atherosclerosis. Alpha granules and dense granules of platelet contains many chemokines,

cytokines and growth factors. Release of these factors along with interaction with endothelial cells and leukocytes promotes inflammation and atherosclerosis.[3] A casual role of platelet hyper-reactivity or of local platelet activation in an acute coronary event has been suggested.

Platelet products in plasma (beta-thromboglobulin, thromboxane and platelet factor) have been measured to determine platelet activation in patients with coronary artery disease. The results of these tests remain controversial and difficult to interpret. It has not yet been possible to select a platelet function test that can be used to detect platelet activation in relation to the complications of atherosclerosis. Platelets differ in size, density and activity. Increased platelet reactivity, as well as shortened bleeding time, is associated with increased platelet volume. Large platelets are metabolically and enzymatically more active than small platelets as assessed by in vitro aggregometry and have a higher thrombotic potential.[4,5]

They also express higher levels of pro-coagulatory surface proteins such as P-selectin and glycoprotein IIIa and also produce more thromboxane A2.[6,7] Emergence of automated cell counters provided speed, accuracy and feasible utilization of time. Automated cell counter uses the principle of electrical impedance. Along with RBC indices and WBC indices it also measures platelet count (PC) and platelet volume indices (PVI) - mean platelet volume (MPV), platelet distribution width (PDW) and plateletocrit (PCT). These parameters are now routinely available in almost all laboratories. These techniques may be complementary to peripheral blood smear (PBS) examination. The MPV can reflect changes in the level of platelet stimulation and by this it indirectly measures the platelet activation. It is important to find a platelet function test that is relevant to the risk stratification of patients with Ischemic heart disease so as to treat them in priority for better outcome. In present study, we aimed to investigate the association between platelet volume indices in patients with diagnosis of Ischemic heart diseases, in comparison with control group.

METHODS

This is a hospital-based case control type of study. It was done from June 2017 to June 2018 in Government medical college, Surat. Study was carried out in coordination between department of Pathology and Medicine. Total 200 patients were enrolled in this study. The patients were divided into two groups.

Group A (Test Group) - The first group comprised of 100 patients admitted to the intensive care unit of tertiary health care centre with an Ischemic heart disease(acute coronary, either unstable angina or acute myocardial infarction) diagnosed on the basis of symptoms of ischemia, characteristic electrocardiographic changes indicative of ischemia.

Group B (Control group) - The second group comprised of 100 normal persons attending Medicine out-patient department for a fitness check-up or patients with noncardiac chest pain. None of these patients had conventional cardiac risk factors and all had normal electrocardiograms with no prior history of any heart disease.

Exclusion Criteria

Patients with severe hepatic or renal impairment, or on oral anticoagulation therapy, malignancy or myeloproliferative disorders. In all patients detailed clinical history was collected.

Collection of samples

Blood sample was drawn from patients of acute coronary syndrome within 6 hours of arrival before administration of any anticoagulant, in EDTA bulbs and samples were analyzed within 2 hrs after collection. Similarly, blood samples from normal healthy subjects or from patients with non-cardiac chest pain was collected and analyzed. Analysis of the blood sample was done by 3-part Automated hematology analyser ABX Micros 60 - HORIBA.

Statistical analysis

Collected data was entered in Microsoft excel sheet and data was analysed using software Statistical Package for the Social Sciences (SPSS). Quantitative data were summarized in form of mean and S.D. (Standard Deviation) and the difference was analysed by using Mann-Whitney Unpaired Test. Pearson correlation was used to find correlation between PVI and PC.

RESULTS

A total of 200 patients were enrolled for the present study. They were divided into two groups.

Group A (Test Group) comprised of 100 patients with Ischemic heart disease {n=100}

Group B (Control group) comprised of 100 patients with non-cardiac chest pain or normal healthy subjects{n=100}

As described in figure 1, Group A comprised of 100 patients of Ischemic heart disease. Mean age of this group is 51 years with range of 27 to 95 yrs. The male to female ratio is 1.8:1 with 64 males and 36 females. Figure 2 shows age wise distribution of patient with ischemic heart disease.

As described in Figure 3, Group B comprised of 100 patients of healthy control group. Mean age of this group is 46.15 years with range of 28 to 75 yrs. The male to female ratio is 1.6:1 with 62 males and 38 females. Detailed history was taken followed by a thorough clinical examination. Most of the patients of acute

myocardial infarction and unstable angina presented with complaints of constricting type of chest pain while some either complained of palpitation, sweating, referred pain over left arm or back, dyspnea, nausea and vomiting.

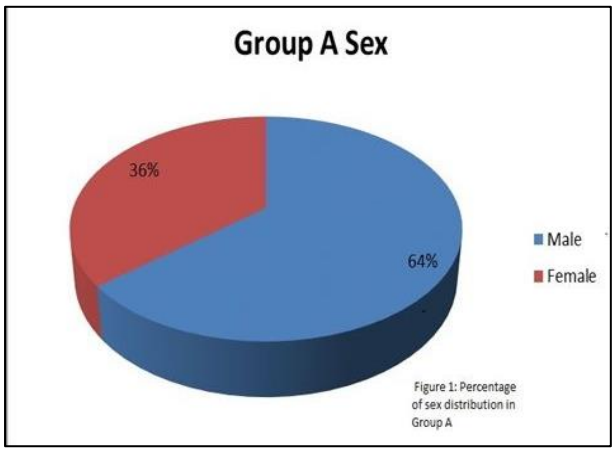


Figure 1: Percentage of sex distribution in group A.

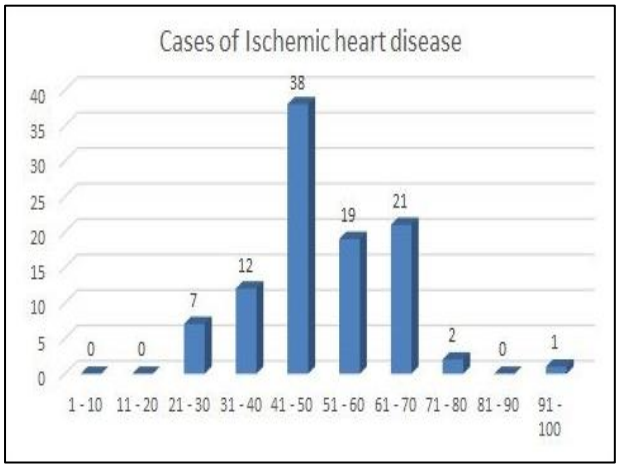


Figure 2: Age-group wise distribution of patients with IHD.

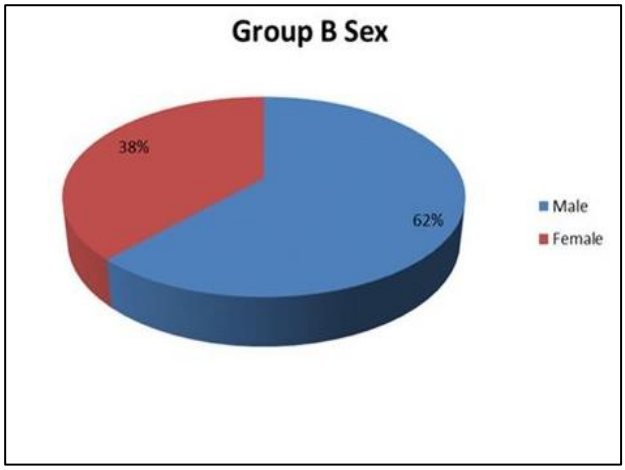


Figure 3: Percentage of sex distribution in Group B.

Comparison of platelet distribution width (PDW) and mean platelet volume (MPV) was done along with platelet count (PC) in both the groups. Median of platelet count, MPV, PDW are compared in both the groups and are shown in Table 1, 2, 3.

Table 1: Comparison of the median platelet count in both the groups.

Platelet indices	Group A	Group B
Median Platelet value ($x10^3/\mu l$)	216	259.5

Table 2: Comparison of the median MPV in both the groups.

Platelet indices	Group A	Group B
Median MPV (fl)	8.35	7.8

Table 3: Comparison of the median PDW in both the groups.

Platelet indices	Group A	Group B
Median PDW (fl)	16.705	16.2

Comparison of PVI and PC in both the groups by applying unpaired-t test. Results are described in table 4. We demonstrated that platelet count is significantly low in Ischemic heart disease (217.16±71.12 x103/µl) as compared to patients with non-cardiac chest pain or healthy subjects (262.2±97.26 x103/µl) and the difference was statistically significant($p<0.05$).

In the present study, we demonstrated that MPV is significantly high in Ischemic heart disease (8.39±1.01 fl) as compared to patients with non-cardiac chest pain or healthy subjects (7.96±0.93 fl) and the difference is statistically significant. PDW is significantly high in Ischemic heart disease (16.84±1.34 fl) as compared to patients with non-cardiac chest pain or healthy subjects (16.22±0.79 fl).

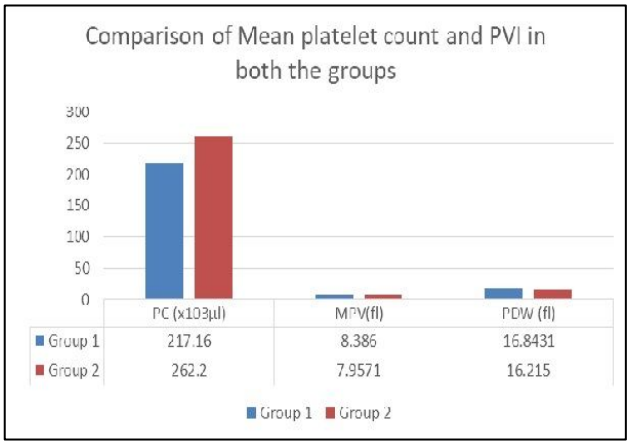


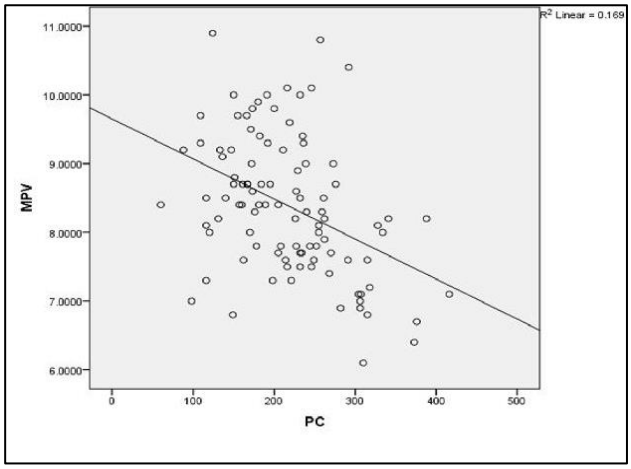
Figure 4: PC and PVI in both the groups.

Table 4: Comparison of PVI and PC in both the groups by applying unpaired-t test.

	Group A (Mean±S.D)	Group B (Mean±S.D)	p Value	Significance
PC (x10^3/µl)	217.16±71.12	262±97.26	0.001(<0.05)	Significant
MPV (fl)	8.39±1.01	7.96±0.93	0.002(<0.05)	Significant
PDW (fl)	16.84±1.34	16.22±0.79	0.000(<0.05)	Significant

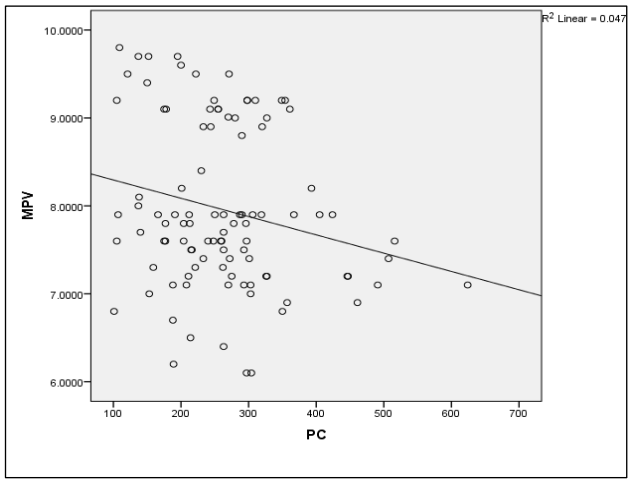
Table 5: Correlation between MPV and PC in Group A and B.

MPV with PC	Group A	Group B
Pearson Correlaton	-0.411	-0.217
p Value	0.000022 (<0.05) (Significant)	0.030(<0.05) (Significant)



R^2= 0.169; p= 0. 0.000022(Significant)

Figure 5: Correlation between PC and MPV in Group A. The scatter diagram shows Inverse correlation between MPV and PC Group A.



R^2= 0.047; p = 0.030 (Significant)

Figure 6: Correlation between PC and MPV in Group B. The scatter diagram shows Inverse correlation between MPV and PC Group B.

Correlation between MPV and PC in Group A and B is described in Table 5. The correlation of MPV with PC revealed an inverse correlation between the patients of IHD and healthy or non-cardiac chest pain patients which is statistically significant (Figure 5 and 6).

DISCUSSION

The present study of Platelet count and platelet volume indices (MPV & PDW) was performed on 100 cases of Ischemic heart disease and 100 cases healthy persons or non-cardiac chest pain were taken.

Large number of studies till now studied isolated platelet volume indices and platelet count in Ischemic heart disease and compared it to healthy control groups. Very few out of these have studied all the parameters of platelet indices like MPV, PDW and platelet count in Ischemic heart disease and compared them with healthy controls. Present study is one of these efforts in which we have compared Platelet parameters (Platelet count, MPV and PDW) between IHD patient and Control group with the other studies in literature.

In present study Group A(Test) patients were in the age group between 27 to 95 years with a mean age of 51±12.25 yrs. In group B ages of patients were ranging from 28 to 75 years, mean age was of 46.15±12.49 yrs.

In present study out of the 100 cases of group A, 64 were males and 36 were females. Thus, the male to female ratio was 1.8:1 while 1.6:1 in Group B with 62 males and 38females. Males predominated females in both the groups in our study and the sex ratio was comparable.

The main focus of present study was to look into changes in platelet count and platelet volume indices in Ischemic heart disease versus healthy controls or non-cardiac chest pain. *Mann-Whitney U test applied for platelet comparison in both the groups.

In the present study, we demonstrated that platelet count is significantly low in Ischemic heart disease (217.16±71.12 x10^3/µl) as compared to patients with non-cardiac chest pain or healthy subjects (262.2±97.26x10^3/µl) and the difference was statistically significant (p<0.05).

As described in table 6, our results are consistent with Cameron et al, which studied platelet size in myocardial

infarction compared 100 patients of AMI with 200 age matched controls and found that AMI patients have significantly lower platelet count (275 x10^3/µl) than controls (295 x10^3/µl).[8]

Table 6: Comparison of platelet count in IHD and Healthy subjects or non-cardiac chest pain in different studies.

Author	No	IHD PCx10^3/µl	No	Healthy subjects or non-cardiac chest pain PCx10^3/µl	p value
Cameron et al,	100	275 (SE=7)	200	295 (SE=5)	<0.05
Martin et al,	126	274±65	1590	276±68	NS
Pizzulli et al,	108	245±56	97	261±58	<0.05
MPRanjith et al,	60	201±13.01	60	256.65±25.49	<0.05
Kiliçli-Çamur et al,	35	223±64	60	222±52	NS
Khandekar et al,	94	232.84±88.8	30	270.77±75.2	<0.05
Prem Shanker Pipliwal et al,	60	231.25±67.27	60	276.38±120.86	<0.05
G.Ranjani et al,	50	AMI-210.84 UA - 221.76	25	227.4	NS
Present study	100	IHD 217.16±71.12	100	262.2±97.26	0.001 (<0.05)

A study by Martin et al, compared influence of platelet size on outcome after myocardial infarction.126 patients with AMI had lower platelet count 274±65 x10^3/µl than 1590 age matched controls 276±65 x10^3/µl and the difference was not statistically significant.[9]

Pizzulli et al, compared platelet count and platelet volume indices of 108 patients with unstable angina and 97 patients with noncardiac chest pain. Study found lower platelet count (245±56 x10^3/µl) in unstable angina group than (261±58 x10^3/µl) non cardiac chest pain and the difference was statistically significant.[10]

Other studies like M P Ranjith et al, compared the platelet count in 60 acute coronary syndrome patients with 60 age matched controls respectively and found to have significantly lower platelet count 201±13.01 x10^3/µl in ACS group than as compared 256.65±25.49 x10^3/µl in the noncardiac chest pain group.[11]

A study by Kiliçli-Çamur N et al, compared 35 patients of unstable angina with 35 age matched controls. Unstable angina group had slightly higher platelet count (223±64 x10^3/µl) than control group (222±52x10^3/µl) but the difference was not statistically significant.[12]

Other studies like Khandekar et al, compared the platelet count in 94 acute coronary syndrome patients with 30 age matched controls respectively and found to have significantly lower platelet count 232.84±88.8 x10^3/µl in ACS group than as compared 270.77±75.2 x10^3/µl in the noncardiac chest pain group.[13]

Pipliwal PS et al, compared mean platelet volume and other platelet volume indices in 60 patients with acute myocardial infarction. Study found lower platelet count (231.25±67.27 x10^3/µl) in MI patients as compared to 60 healthy controls (276.38±120.86 x10^3/µl) and the difference was statistically significant.[14]

G. Ranjani et al, done a study on platelet volume indices in acute coronary syndrome. Study comprised of 25 patients each with STEMI, with UA, with chronic stable angina and with noncardiac chest pain. Study found out lower platelet count (210.84x10^3/µl and 221.76x10^3/µl) respectively in STEMI and UA patients as compared to (227.04x10^3/µl) in control group but the difference was not statistically significant.[15]

In the present study, we demonstrated that platelet count is significantly low in Ischemic heart disease as compared to patients with noncardiac chest pain or healthy subjects.

Amongst the platelet volume indices all the variable like MPV and PDW were found to have increased in IHD when compared to healthy subjects or noncardiac chest pain patients. This indicates that along with atherosclerosis, acute coronary syndrome might be associated with a systemic increase in platelet destruction rate. This increase platelet destruction rate is not completely compensated for by an increase in platelet production rate. In addition, platelet consumption at the site of the coronary atherosclerotic lesion may contribute to further decrease in platelet count.

In the present study, we demonstrated that MPV is significantly high in Ischemic heart disease (8.39±1.01 fl) as compared to patients with non-cardiac chest pain or healthy subjects (7.96±0.93 fl) and the difference is statistically significant.

Similarly as described in table 7, Cameron et al, Martin et al, Martin et al, Pizzulli et al, Khanderkar et al, MP Ranjith et al, Prem shanker pipliwal et al, G. Ranjani et al, and Vitthal Khode compared MPV between IHD patients and control groups and found to have MPV 9.07, 7.3, 10.09±1.43, 9.4±1.23, 10.43±1.03, 10.97±0.58, 11.97±1.458, 9.8±0.86(AMI) - 9.5±0.84(UA) and 9.65±0.96 fl respectively, which are significantly higher in as compared to control groups.[8-1,13-17]

Table 7: Comparison of MPV in IHD and Healthy subjects or non-cardiac chest pain in different studies.

Author	No	IHD MPV (fl)	No	Healthy subjects or non-cardiac chest pain MPV (fl)	p value
Cameron et al,	100	9.07 (SE 0.08)	200	8.32 (SE=0.05)	<0.05
Martin et al,	15	7.3	22	6.32	<0.05
Martin et al,	126	10.09±1.43	1590	9.72±1.12	<0.05
Pizzulli et al,	108	9.4±1.23	97	8.2±0.95	<0.05
Kiliçli-Çamur Net al,	35	11.37±0.91	60	11.25±0.75	NS
Khandekar et ai,	94	10.43±1.03	30	9.20±0.91	<0.05
MP Ranjith et al,	60	10.97± 0.58	60	9.12±0.63	<0.05
Prem Shanker pipliwal et al,	60	11.97±1.458	60	10.72±0.94	<0.05
G.Ranjani et al,	50	AMI 9.8±0.86 UA 9.5±0.84	25	8.2±0.56	<0.05
Vitthal khode,	39	9.65±0.96	65	9.21±0.58	<0.05
Present study	100	8.39±1.01	100	7.96±0.93	0.002(<0.05)

A study by Kiliçli-Çamur N et al, also compared 35 patients of unstable angina with 35 age matched controls. Unstable angina group had slightly higher MPV (11.37±0.91) than control group (11.25±0.75) but the difference was not statistically significant.[12]

We interpret this as an acute process of generalized platelet activation in Ischemic heart disease. Due to high consumption of platelets at the site of coronary lesion, bone marrow is stimulated resulting in release of larger platelets in patients of ACS. However, since changes in platelet size are determined at thrombopoiesis and platelets circulate in man for 10 days it is likely that the large platelets were circulating at the initiation of symptoms. Our data suggest that the increased mean platelet volume contributes to the prethrombotic state in acute ischemic syndromes. They are consistent with previous findings that the mean platelet volume is increased at time of admission with an acute myocardial infarction. Furthermore, large platelets are a risk factor for myocardial infarction and death and activated megakaryocytes are present in the bone marrow at the time of sudden cardiac death.

Table 8: Comparison of PDW in IHD and Healthy subjects or non-cardiac chest pain in different studies.

Author	No	IHD PDW (fl)	No	Healthy subjects or non cardiac chest pain PDW (fl)	p value
Khandekar et al,	94	13.19±2.34	30	30	<0.05
MP Ranjith et al,	60	14.63±0.64	60	60	<0.05
Prem Shanker,	60	15.23±3.503	60	60	<0.05
G.Ranjani et al,	50	MI- 14.3±0.2 UA- 14.8±0.7	25	25	MI & control Not Significant UA and Control Significant
Present study	100	16.84±1.34	100	100	<0.05

In the present study, we demonstrated that PDW is significantly high in Ischemic heart disease (16.84±1.34fl) as compared to patients with non-cardiac chest pain or healthy subjects (16.22±0.79fl). As described in table 8, similar results were found by Khandekar et al, where 94 patients of ACS were compared to 30 healthy controls.[13] IHD patient had

significantly higher PDW (13.19±2.34fl) than healthy controls (10.75±1.42 fl).

Our results also match with another Study MP Ranjith et al, where PDW of 60 patients with ACS was compared with 60 non cardiac chest pain patients, it was found that PDW in ACS (14.63±0.64 fl) was significantly higher than noncardiac chest pain patients (12.01±0.55 fl).[11]

The platelet parameters, as they were high in patients of ACS, were compared with each to find out the correlation, it is found that there is statistically significant inverse relation between MPV and PC in ACS and control group (r=-0.520; p<0.001).Larger platelets, associated with a reduced count, have been observed in the early stage of ACS, and they are considered to be a risk factor for developing coronary thrombosis.

CONCLUSION

From the analysis of the present study, we conclude that platelet count was significantly lower in study group as compared to control group (normal healthy controls or non-cardiac chest pain patients). MPV and PDW were significantly higher in study group compared to control group. This change suggests the role of Platelets in Ischemic heart disease by Platelet activation and Coronary thrombosis leading to myocardial infarction.

However, study has some limitations. Normal reference range of Platelet count (150-450 $x10^9$/L), Mean platelet volume (8-10 fl) and Platelet distribution width (11.5-16.5 fl) are wide. In present study though difference in Platelet count, MPV and PDW were statistically significant between Test and Control group, the absolute difference was narrow and individual values were within the normal reference range.

So, as this study is small and cardiac enzyme, coronary angiography findings were not available, larger comprehensive studies are required for confirmation. If patient's own previous platelet parameters are available, then comparison of it may be beneficial for patients. In conclusion, the platelet volume indices are an important, simple, effortless and a cost-effective tool, generated as a by-product of automated blood counts, useful in predicting the development of an acute coronary event sometimes in the near future and therapeutic modification for improved patient's cardiovascular care.

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