Correlation between Platelet indices and ischemic heart disease

Dr. Saransh Baranwal[1], Dr. Sujit Kumar (MD)[2]

[1]*Junior resident Department of Medicine MLN Medical College Prayagraj, UP, India, 211002*
[2] *Professor Department of Medicine MLN Medical College Prayagraj, UP, India, 211002*

Abstract:
Background: Evaluation of the correlation between various platelet indices and ischemic heart disease (IHD).
Materials and Methods: A cross-sectional study was done. 120 patients of IHD (40 patients of STEMI, 40 patients of NSTEMI and 40 patients of stable angina) diagnosed on the basis of clinical, electrocardiographic and other laboratory parameters were included in the study. Various platelet indices of ischemic heart disease patients were compared with 40 controls. Platelet indices included platelet count, mean platelet volume (MPV), platelet distribution width (PDW) and plateletcrit (PCT).
Results: The mean platelet count of the present study was 1.96±0.85 in cases and 1.40±0.55 in controls group (p value < 0.05). The mean MPV (FL) was 10.37±1.00 in cases and 8.22±0.69 in controls group (p value < 0.05). The mean PDW (%) was also significantly higher in cases as compared to controls (p <0.001). The mean plateletcrit (PCT) was 0.19±0.07 in cases and 0.18±0.06 in controls group. This was not significantly different in between cases and controls (p =0.188).
Conclusion: The occurrence of ischemic heart disease is significantly associated with various platelet indices including platelet count, MPV and PDW. Association between plateletcrit and ischemic heart disease was not found to be significant (p value=0.188). These findings are consistent with other previously done studies.
Key Word: Ischemic heart disease, Platelet indices, Platelet count

----

I.   Introduction

IHD is a condition in which the oxygen demand and supply to a segment of the myocardium are in imbalance[1]. As per World Health Organization estimates, 1 out of every 3 persons (31 percent of all fatalities) in affluent countries dies of cardiovascular disorders, and coronary artery disease is the most common cause among them. Cardiovascular diseases account for around a third of all fatalities globally[2]. During the occlusion and reperfusion phases of a myocardial infarction, platelets play an important role. They also play a key role in microvascular blockage, tissue perfusion, and vascular patency maintenance[3]. Also, platelet activation is an important hallmark of acute coronary syndromes (ACS)[4].

Various platelet indices include platelet count, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT).

MPV assesses the average size of platelets and is also considered a marker of platelet function and activation[5]. Larger platelets will have a greater content of granules and Platelets with more granules, higher thromboxane production, β-thromboglobulin, serotonin release, enhanced expression of P-selectin, glycoprotein IIb/IIIa, and fibrinogen receptors are more active and prone to aggregate than platelets with fewer granules[6].

PDW is a measurement of platelet size variability. During activation, the platelets' form changes from discoid to spherical. As a result, platelet indices such as MPV and PDW can be utilized to monitor platelet activation and serve as a useful predictor of ischemia events. More reactive platelets will undergo morphological changes in the cell, generating pseudopodia, and so contribute to the formation of thrombus[7].
PCT is the proportion of blood volume occupied by platelets[8].

II.   Material And Methods

A hospital based cross-sectional study was carried out in department of general medicine & cardiology in SRN Hospital, MLN medical college, Prayagraj, Uttar Pradesh, India over a period of 24 months in 2019-2021 after taking approval from the Scientific Research and Ethics Committee. A total of 160 adult subjects (both male and females) were included in the study.
Study Design: Observational cross-section study
Study Location: This was a tertiary care teaching hospital-based study done in Department of Medicine, at Swaroop Rani Nehru Hospital, MLN medical college, Prayagraj, Uttar Pradesh.

Study Duration: November 2019 to November 2021.
Sample size: 160 patients.
Subjects & selection method: The study population was selected from ischemic heart disease patients who presented to Swaroop Rani Nehru Hospital with ischemic heart disease between from November 2019 to November 2021. 40 patients each of STEMI, NSTEMI and stable angina were selected.

Inclusion criteria:
1.   Patients diagnosed as acute myocardial infarction (STEMI & NSTEMI),
2.   Patients diagnosed with stable angina.

Exclusion criteria:
1.   Patients with severe hepatic or renal impairment, patients taking oral anticoagulation medicine.
2.   Old CAD patients already on anti-platelet drugs,
3.   Any other patients on thrombocytopenia causing medications,
4.   Patients with history of any bleeding disorder,
5.   Febrile patients.

Procedure methodology
        The study was carried out at the department of internal medicine and cardiology of tertiary care hospital in Uttar Pradesh, India. After taking approval from the Scientific Research and Ethics Committee, patients who fulfilled the inclusion criteria were taken up for the study. First, the purpose of the study was explained to the study subjects attending OPD/IPD of Internal Medicine & Cardiology Department in the local language with the help of the information sheet. After taking written/informed consent, their parameters were filled in a pre- designed pro-forma.
History and examination -
Already diagnosed patients of STEMI, NSTEMI and stable angina based on their history, ECG changes, elevation in troponin I values, and tread-mill stress testing were selected for the study.
Detailed history regarding risk factors, duration, symptoms, signs and aggravating factors of ischemic heart disease were recorded. Hb, TLC, platelet count, MPV, PDW, SGOT, SGPT, serum urea and serum creatinine from first day of medical contact were also analyzed and recorded. 2D-echo was done in all the patients.
Electrocardiogram & Troponin-I -
ECG and troponin-I levels were recorded from all the patients with symptoms suggestive of ischemic heart disease.
Echocardiography -
Echocardiography was performed in all patients included in the study to assess the degree of cardiac dysfunction (LV ejection fraction) and any regional wall motion abnormalities (RWMA) using FUJI-FILM Sonosite M–Turbo echocardiography machine.
Tread-mill stress testing -
Tread-mill stress testing reports were reviewed from the patients labelled as stable angina. It was not done in other patients of acute myocardial infarction.
Data entry and Statistical analyses -
The collected data were transformed into variables, coded and entered in Microsoft Excel. Data were analyzed and statistically evaluated using SPSSPC-20 version. Quantitative data was expressed in mean, standard deviation, and difference between two comparable groups were tested by student's t-test (unpaired) or Mann Whitney 'U' test. Three or more groups mean was analyzed using one-way ANOVA or Kruskal Wallis H test followed by post-hoc test, while qualitative data were expressed in percentage. Statistical differences between the proportions were tested by chi square test or Fisher's exact test. Pearson correlation coefficient was used to see the correlation between various laboratory biochemical parameters and ischemic heart disease.
Ethical considerations -
1.   The protocol of the thesis was approved by the ethical committee of MLN Medical College, Prayagraj, Uttar-Pradesh India.
2.   All participants were told about the purpose of the study along with informed written consent.
3.   No pressure or coercion was exerted on subjects for participation in the study.
4.   Confidentiality and privacy was ensured at all stages of the study period.
5.   The subjects were free to leave the study at any time and no reasons for the same were asked. They were not debarred from getting any medical services from the hospital even if they refused to participate in the study.
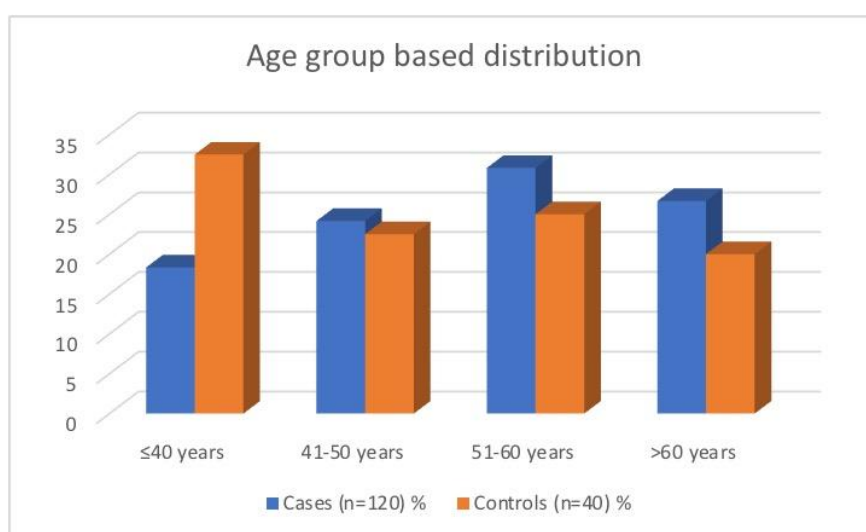6.   The data collected was used for the study purpose only.

III. Result

The percentage of ≤40 years, 41-50 years, 51-60 years, and >60 years age group were 18.33%, 24.17%, 30.83%, and 26.67% in cases and 32.50%, 22.50%, 25.00%, and 20.00% in control group. On the basis of age range, both cases and control groups were significantly different. The mean age was not significantly different in between cases (53.41±12.63) and control groups (49.55±12.55).

Distribution of participants according to ischemic heart disease (case group) and healthy (control group) are shown in Table 1 and Figure 1. Total 120 individuals are enrolled in this study. In which, 80 (75%) patients had ischemic heart disease (case group) including stable angina, STEMI & NSTEMI; and 40 (25%) were healthy (control group).

Table 1: Distribution of participants according to case and control numbers

	Group	Number	Percentage
IHD	Cases	120	40
Healthy	Controls	75	25
Total		160	

Fig.1 Distribution of patients according to case and control groups

Distributions of study population according to the age are shown in Table 2 and Figure 2. The percentage of ≤40 years, 41-50 years, 51-60 years, and >60 years age group were 18.33%, 24.17%, 30.83%, and 26.67% in cases and 32.50%, 22.50%, 25.00%, and 20.00% in control group. On the basis of age range, the both cases and control groups were significantly different. The mean age was not significantly different in between cases (53.41±12.63) and control groups (49.55±12.55).

Table 2: Distribution of study population according to different age group

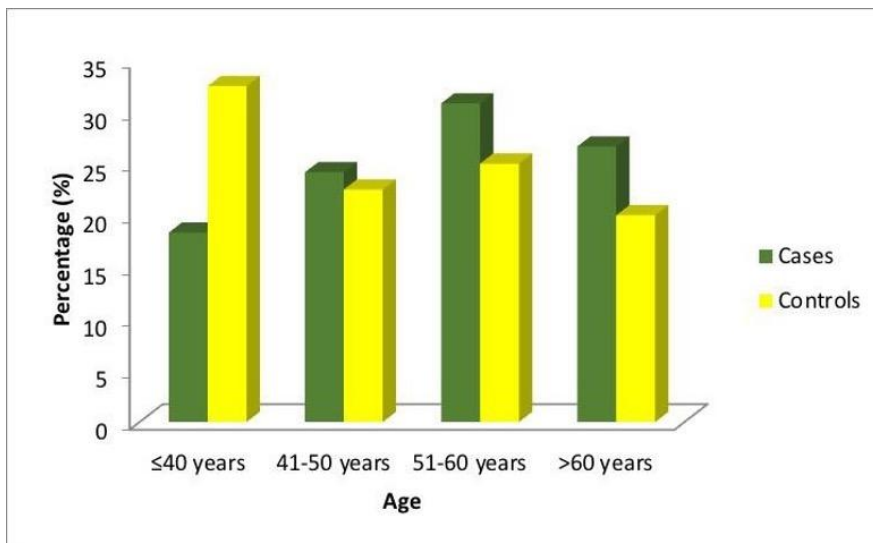|	Cases (n=120)		Controls (n=40)		Chi Sq.	[1]p-value
Age group	n	%	n	%		
≤40 years	22	18.33	13	32.50	3.67	0.300
41-50 years	29	24.17	9	22.50		
51-60 years	37	30.83	10	25.00		
>60 years	32	26.67	8	20.00		
Mean ± SD	53.41±12.63		49.55±12.55		p=0.096	

[1]=Chi-square test

Figure 2: Bar Chart shows the distribution of study population according to different age group

Distributions of study population according to the gender are shown in Table 3 and Figure 3. The percentage of males and females were 60.83% and 39.17% in cases and 55.0 and 45.0% in control group. On the basis of sex distribution, the both cases and control groups were not significantly different.

Table 3: Distribution of study population according to gender

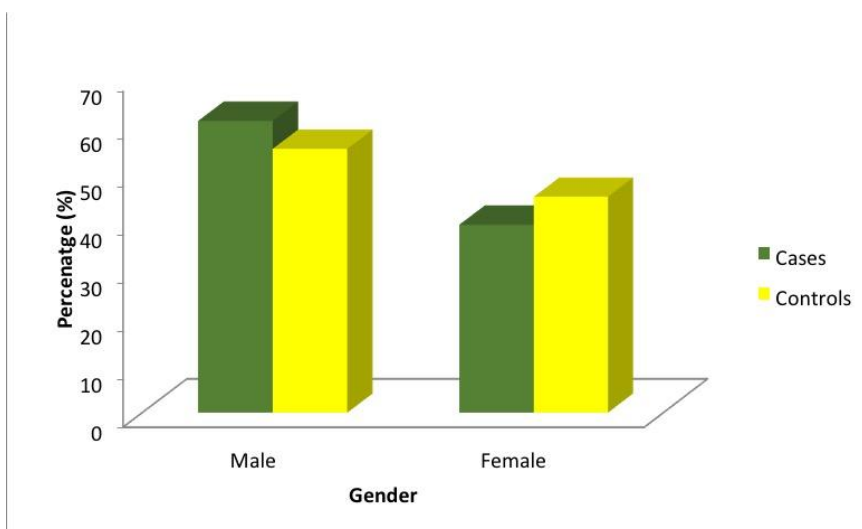|	Cases (n=120)		Controls (n=40)		Chi Sq.	[1]p-value
	n	%	n	%		
Male	73	60.83	22	55.00	0.22	0.642
Female	47	39.17	18	45.00		

[1]=Chi-square test

Fig. 3: Bar Chart shows the distribution of study population according to different age group

The comparisons of mean Platelet count (lac cells/mm3) in between cases and controls group are shown in Tables 4 and Figure 4. The mean Platelet count (lac cells/mm3) was 1.96±0.85 in cases and 1.40±0.55 in controls group. The mean Platelet count (lac cells/mm3) was significantly higher in cases as compared to controls (p <0.001).

Table 4: Comparison of mean Platelet count (lac cells/mm3) in between cases and controls group

	Cases (n=120)		Controls (n=40)		t	¹p-value
	Mean	±SD	Mean	±SD		
Platelet count (lac cells/mm3)	1.96	0.85	1.40	0.55	3.94	<0.001*

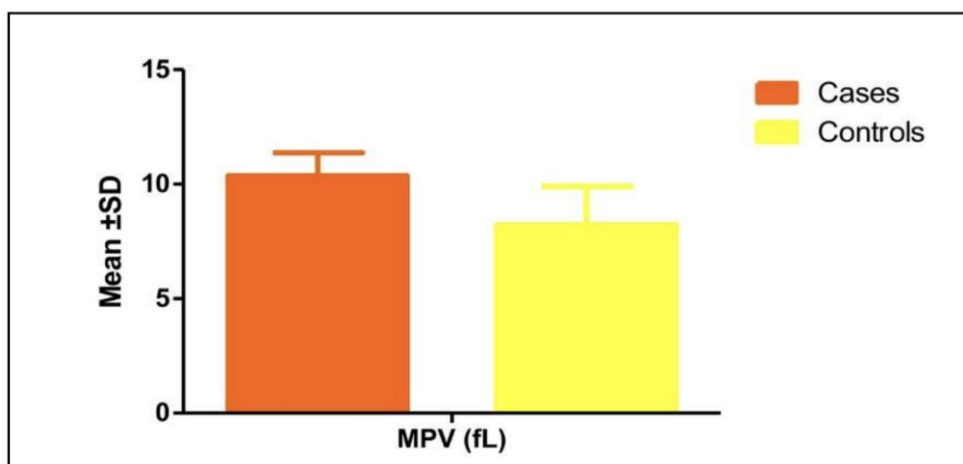*=Significant (p<0.05), ¹=Independent t test
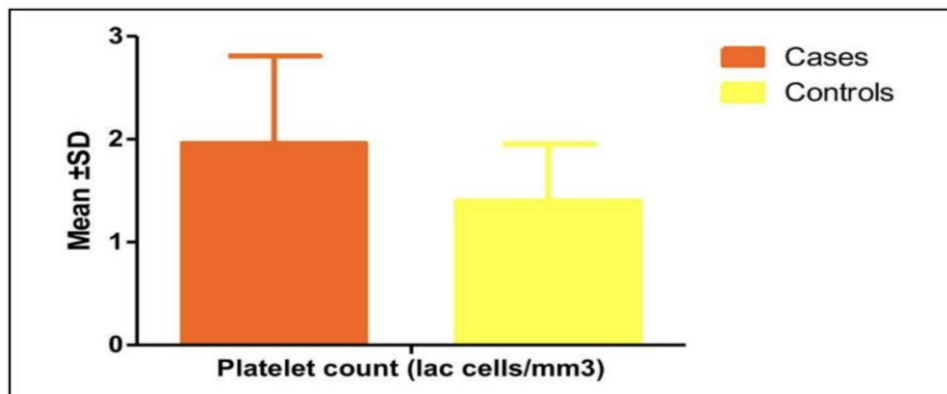
Fig. 4: Bar Chart shows the comparison of mean Platelet count (lac cells/mm3) in between cases and controls group

The comparisons of mean MPV (FL) in between cases and controls group are shown in Table 5 and Figure 5. The mean MPV (FL) was 10.37±1.00 in cases and 8.22±0.69 in controls group. The mean MPV (FL) was significantly higher in cases as compared to controls (p <0.001).

Table 5: Comparison of mean MPV (FL) in between cases and controls group

	Cases (n=120)		Controls (n=40)		t	¹p-value
	Mean	±SD	Mean	±SD		
MPV (fL)	10.37	1.00	8.22	1.69	9.73	<0.001*

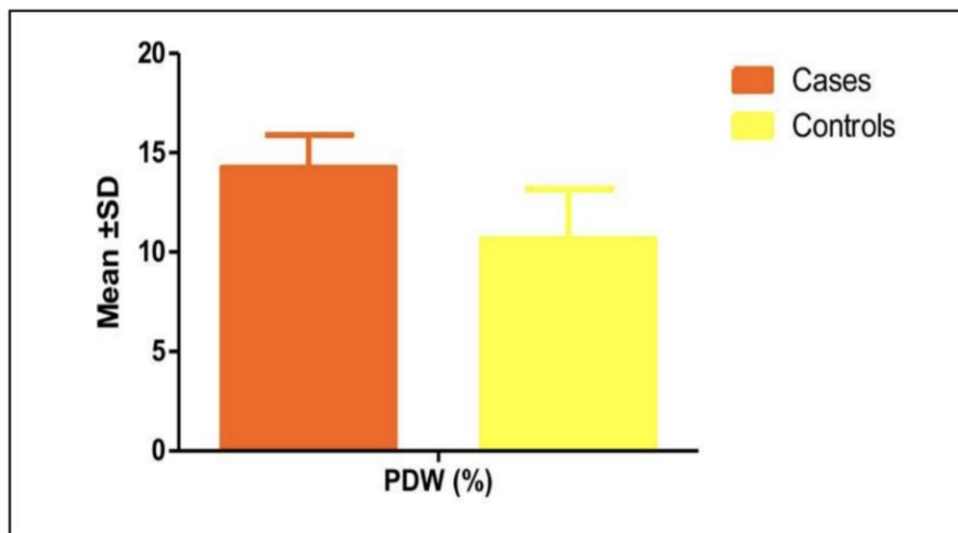*=Significant (p<0.05), ¹=Independent t test

Fig. 5: Bar Chart shows the comparison of mean MPV (FL) in between cases and controls group

The comparisons of mean PDW (%) in between cases and controls group are shown in Table 6 and Figure 6. The mean PDW (%) was 14.25±1.65 in cases and 10.64±2.54 in controls group. The mean PDW (%) was significantly higher in cases as compared to controls (p <0.001).

Table 6: Comparison of mean PDW (%) in between cases and controls group

|	Cases (n=120)		Controls (n=40)		t	[1]p-value
	Mean	±SD	Mean	±SD		
PDW (%)	14.25	1.65	10.64	2.54	10.35	<0.001[*]

[*]=Significant (p<0.05), [1]=Independent t test

Fig. 6: Bar Chart shows the comparison of mean PDW (%) in between cases and controls group

The comparisons of mean PCT in between cases and controls group are shown in Table 7 and Figure 7. The mean PCT was 0.19±0.07 in cases and 0.18±0.06 in controls group. The mean PCT was not significantly different in between cases and controls (p =0.188).

Table 7: Comparison of mean PCT in between cases and controls group

	Cases (n=80)		Controls (n=40)		t	[1]p-value
	Mean	±SD	Mean	±SD		
PCT	0.19	0.07	0.18	0.06	1.32	0.188

[1]=Independent t test
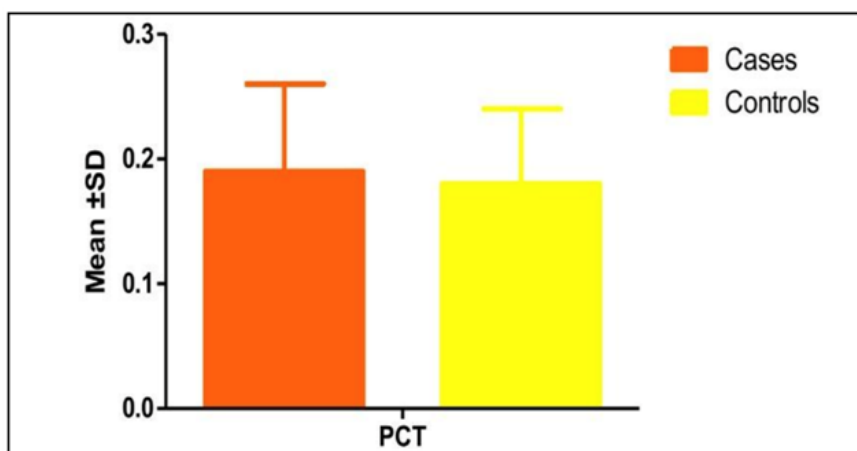
Fig. 7: Bar Chart shows the comparison of mean PCT in between cases and controls group

IV. Conclusion

The findings of the study are as follows:

1. Mean age of ischemic heart disease subjects was 53.41±12.63years with range of 27-88 years. The male to female ratio was 1.55:1.

2. Three-fourth subjects were of ischemic heart disease and rest were control. Out of all the cases, one-third patients were each of stable angina, NSTEMI and STEMI.

3. Out of 120 subjects with ischemic heart disease, 73(60.83%) were males and 47(39.17%) were females.

4. The mean platelet count(lac cells/mm3) of the cases was 1.96±0.85 and for the controls, it was 1.40±0.55. Platelet count was significantly related with ischemic heart disease ($p<0.001$).

5. The mean MPV (fl) was 10.37±1.00 among the cases and among the controls, it was 8.22±0.69. MPV was significantly related with ischemic heart disease ($p<0.001$).

6. The mean PDW (%) of the cases was 14.25±1.65 and for the controls, it was 10.64±2.54. PDW was found to be significantly associated with ischemic heart disease ($p<0.001$).

7. Mean PCT of the cases was 0.19±0.07; for controls, it was 0.18±0.06. There was no significant correlation between PCT and ischemic heart disease.

References

[1]. Bairey Merz C. Ischemia and No Obstructive Coronary Artery Disease (INOCA). Circulation. 2017;135(11):1075-1092.

[2]. Mozaffarian D, Benjamin E. Heart Disease and Stroke Statistics—2016 Update. Circulation. 2016;133(4):38-60.Meadows Ta, Bhatt Dl. Clinical Aspects Of Platelet Inhibitors And Thrombus Formation. Circ Res. 2007 May 11;100(9):1261-75.

[3]. Meadows T, Bhatt D. Clinical Aspects of Platelet Inhibitors and Thrombus Formation. Circulation Research. 2007;100(9):1261-1275.

[4]. VandeWerf F. Management of acute myocardial infarction in patients presenting with ST-segment elevation. European Heart Journal. 2003;24(1):28-66.

[5]. Kiliçli-Çamur1ABEF N, Demirtunç1B R, Konuralp2DEF C, Eskiser3F A, Başaran4A Y. Could mean platelet volume be a predictive marker for acute myocardial infarction?. Med Sci Monit. 2005;11(8):392.

[6]. Huczek Z, Kochman J, Filipiak KJ, Horszczaruk GJ, Grabowski M, Piatkowski R, Wilczynska J, Zielinski A, Meier B, Opolski G. Mean platelet volume on admission predicts impaired reperfusion and long-term mortality in acute myocardial infarction treated with primary percutaneous coronary intervention. Journal of the American College of Cardiology. 2005 Jul 19;46(2):284-90.

[7]. Sridharan S, Shanmugapriya M, Eswari V. A Comparative study of Platelet indices in Acute Myocardial Infarction and Stable Coronary Artery Disease patients. International Journal. 2019 May;2(3):108.

[8]. Chandrashekar V. Plateletcrit as a screening tool for detection of platelet quantitative disorders. Journal of Hematology. 2013 Jun 13;2(1):22-6.