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PLATELET PARAMETERS IN ACUTE STEMI CASES WITH AND WITHOUT CO-MORBIDITIES.

Pathology

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ABSTRACT

INTRODUCTION: Coronary artery disease (CAD) is an epidemic and is the most common cause of mortality worldwide. Majority of deaths from CAD result from acute coronary syndrome (ACS). Generalised platelet activation occurs before an acute coronary event. Central to the pathogenesis of occlusive arterial disease is the activation of platelets at sites of vascular injury via pathologically exaggerated and deregulated versions of protective mechanisms involved in hemostasis.

AIM: To study the changes in platelet volume indices in ST-Elevated Myocardial Infarction (STEMI) and assess their usefulness in predicting coronary events.

MATERIALS AND METHODS: This is a cross sectional study conducted in tertiary care hospital and research center in Andhra Pradesh during a time period of one year (Jan 2018- Dec2018). All the patients diagnosed with acute STEMI were included in the study and their platelet indices were compared with those of controls having a normal electrocardiogram. A total of 77 cases and 77 controls were included in the study. Cases were again grouped into those with co-morbid conditions and those without co-morbid conditions.

RESULTS: A total of 77 cases with acute STEMI and 77 controls were included in the study.The mean age of patients was 61.3±10yrs with range from 45-80yrs. The mean age of control group was 62.3±10yrs. Out of 77 cases 78% were males, 22% were females. 65 cases have associated one or more co-morbid conditions considered in the sutdy, 12 cases were without any of them. The Mean Platelet Volume (MPV) was significantly higher in patients with STEMI (10.9±3) as compared to controls (8.5±0.9). The Platelet Distribution Width (PDW) was also significantly higher in cases compared to controls ($p<0.005$).

CONCLUSION: Platelet volume indices provides an important, simple, effortless and cost-effective tool which can be useful in predicting an impending acute coronary event.

KEYWORDS

STEMI, Platelet volume indices,MPV

INTRODUCTION:

Coronary artery disease (CAD) is an epidemic and is the most common cause of mortality worldwide. Majority of deaths from CAD result from acute coronary syndrome (ACS), acute myocardial infarction and unstable angina. Platelets play a pivotal role in atherothrombosis, the major cause of most ACS[1]). Central to the pathogenesis of occlusive arterial disease is the activation of platelets at sites of vascular injury via pathologically exaggerated and deregulated versions of protective mechanisms involved in hemostasis[1]. Platelets secrete and express a large number of substances that are crucial mediators of coagulation, inflammation, thrombosis and atherosclerosis (2,3).

Generalised platelet activation occurs before an acute coronary event. Platelet size has been shown to reflect platelet activity which is indirectly measured by the platelet parameters. Larger platelets are metabolically and enzymatically more active than small platelets[4]. Platelet indices correlate with functional status of platelets and is an emerging risk marker for atherothrombosis[5]. 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 substance or into leukotrienes which can amplify acute inflammatory response[6].

Patients with CAD fall into two large groups, patients with stable angina and patients with acute coronary syndrome including acute myocardial infarction (STEMI,NSTEMI) and unstable angina[7]. Platelet indices MPV,PDW,P-LCR have been well utilised for certain conditions like idiopathic thrombocytopenic purpura, aplastic anemia and other haemotological and myeloproliferative disorders to assess the prognosis but are underutilised for cardiovascular disorders[8].

Platelet indices are easily recorded by automated cell counter and are routinely available in most clinical laboratories. There is scope to make better use of the platelet parametrs generated, as patients with larger platelets can easily be identified during routine haemotological analysis and could possibly benefit from timely treatment [4].

AIM:

The study was undertaken to account the efficacy of platelet parameters in acute STEMI patients by comparing them with controls. To study the difference in platelet indices in STEMI cases with and wihout co-morbidities.

MATERIALS AND METHODS

This is a cross sectional study conducted in tertiary care hospital and research center in Andhra Pradesh during a time period of one year (Jan 2018- Dec2018). All the patients diagnosed with acute STEMI were included in the study and their platelet indices were compared with those of controls having a normal electrocardiogram. A total of 77 cases and 77 controls were included in the study. Cases were again grouped into those with co-morbid conditions and those without co-morbid conditions.

Co-morbidities in the present study were defined as those chronic conditions that were previously diagnosed or that may have been newly diagnosed during the patients hospital stay. Comorbidities included were hypertension, peripheral artery disease, diabetes mellitus, anemia. We adopted the most widely used definition of multimorbidity, that is, the coexistence of multiple chronic diseases and medical conditions in the same individual[9,10,11]. WHO definition of chronic disease which is health problems that require ongoing management over a period of years or decades[12].

Inclusion criteria:
Patients diagnosed with acute STEMI on their presenting electrocardiogram.

Patients more than 18yrs of age.

Exclusion criteria:
Patients with bleeding diathesis, previous stroke, major operations or significant trauma in past 2weeks

Patients less than 18yrs of age.

The blood samples of patients and controls were drawn from antecubital vein using a 5ml syringe and immediately mixed in EDTA vaccutainers. The sample was run within two hours of venepuncture using the 5 part differentiated automated hematology analyser and complete blood count analysis was made including platelet indices (MPV, PDW, P-LCR). Statistical analysis was performed by statistical package for the social sciences (SPSS) version 17. Student t-test was used to find the significance. Data expressed as mean ±SD. A $p<0.05$ was considered statistically significant.

RESULTS

A total of 77 cases with acute STEMI and 77 controls were included in the study.The mean age of patients was 61.3±10yrs with range from 45-80yrs. The mean age of control group was 62.3±10yrs. Out of 70 cases 78% were males, 22% were females. 65 cases have associated one or more co-morbid conditions considered in the sutdy, 12 cases were without any of them. (Table 1)

TABLE :1

variables	Controls (n=77)	Cases (acute STEMI) (n= 65+12)
Age: (mean ± SD) years	62.3±10yrs.	61.3±10yrs
Gender: Male (%)	79.6	78
Female (%)	20.4	22
Hypertension	----	55.2 (n=60)
Diabetes mellitus	----	58.5(n=60)
Peripheral vasular disease	----	15.0(n=60)

Mean MPV of cases with acute STEMI is significantly higher than that of controls. PDW of cases with acute STEMI is significantly higher than that of controls. Mean P-LCR of cases with acute STEMI is higher than that of controls but not statistically significant(table:2).

Among STEMI cases MPV,PDW and P-LCR were higher in STEMI with associated comorbid conditions than those without comorbid conditions. But statistical significance could not be made out because of small number of cases without morbidities.

TABLE :2

	Mean Platelet volume (Mean ± SD)	Plateletdistribution width (Mean ± SD)	P-LCR (Mean ±SD)
Acute STEMI cases (n=77)	10.9±3	17.0±3	20.09±4
Controls (n=77)	8.5±0.9	16.7±2	15.1±3.5
p- value	<0.001	<0.005	0.6

TABLE :3

	Mean Platelet Volume (Mean ± SD)	Plateletdistribution width (Mean ± SD)	P-LCR (Mean± SD)
STEMI with co-morbid conditions (n=65)	11.02±2	17.8±3	22.09±4.8
STEMIwithout comorbid conditions (n=12)	10.3±2	16.7±3	18.57±3.7

DISCUSSION

MPV evaluated in our study was higher in patients with acute STEMI compared to that of control group. This is in agreement with studies done by Khode et al, Chu et al, Lippi et al, Khandekar Et al.

Platelet reactivity is criticaly important in the formation and propagation of intracoronary thrombus[13]. MPV is one of the markers indicating the function of platelets[14]. Increased MPV was found to be associated with coronary artery disease, acute MI, congestive heart failure and hypertensive patients with evidence of target organ damage and cerebrovascular disease, an important complication of atherosclerosis[15].

The size of platelets has been found to associated with an increased number of megakaryocytes. The increased ploidy of megakaryocytes is correlated with megakaryocyte and platelet volume[16-19]. Elevated levels of CD40 ligands, which are expressed by activated platelets, have been found in atheromatous plaques[20]. This activation process results in signalling pathways that induce platelets to change their shape and size[21] and become more active in secreting thromboxane A2 and ADP into circulation. Larger platelets are more adhesive and tend to aggregate more than smaller ones[22] and contain more secretory granules and mitochondria[23].

Activated large platelets directly bind to the circulating coagulation protein fibrinogen via platelet integrin, glycoprotein IIb/IIIa[24,25]. Platelet-fibrinogen-platelet connection initiates the process of platelet aggregation[26] and thus leads to coronary thrombus formation. These findings led to the hypothesis that larger platelets as determined by their volumes, MPV, may be useful marker in patients with acute STEMI.

MPV was high in STEMI cases with associated co-morbid conditions than in those without co-morbid conditions. Significance could not be made out due to small number of cases without co-morbid conditions. Few studies showed increased MPV with increasing number of co-morbidities[27-29]. Patients with peripheral vascular disease particularly diabetics, have an altered megakaryocyte ploidy distribution, showing a shift towards higher ploidy in association with an increased platelet mass[30].

Effect of multimorbidity on MPV can be explained in part by the proximity of thrombosis and inflammation. Elevated MPV along with increased inflammatory biomarkers(CRP,IL-6) were reported in many conditions (hypertension) which are characterised by low grade inflammation[31]. More recently, it has become evident that platelet activation is also a hallmark feature in inflammation[32-34]. Platelet volume is determined both during megakaryopoiesis and thrombopoiesis. Megakaryocytic maturation, platelet production and platelet size could be regulated by cytokine such as IL-6, G-CSF,M-CSF.

CONCLUSION

Our study suggest that the increased platelet volume indices (PVI) contribute to the prothrombotic state in acute ischaemic syndromes and the larger platelets may play a specific role in infarction. Because larger platelets are haemostatically more active, the presence of larger platelets is probably a risk factor for developing coronary thrombosis and MI. Patients with larger platelets can easily be identified during routine haemotological analysis because PVI are generated as a byproduct of automated blood counts.

To conclude PVI provides an important, simple, effortless and cost-effective tool which can be useful in predicting an impending acute coronary event.

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