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A COMPARATIVE STUDY OF PLATELETS BEHAVIOUR IN CONTROLS AND ACUTE MYOCARDIAL INFARCTION PATIENTS

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Abstract : Acute myocardial infarction is one of the leading vascular diseases. Platelet-endothelium plays a crucial role in its etiopathogenesis.

The present study was undertaken to compare the platelet aggregability in controls and acute myocardial infarction patients along with its effect on the peripheral platelet count.

The work was conducted in five patients from ICCU and 5 controls between the age group of 40–60 years by using Chronolog Dual Channel Aggrometer with ADP and epinephrine as agonists. The platelets were counted by haemocytometry.

The results were statistically analysed by Student`s `t` test which was found to be significant. There was increase in the aggregation index in patients compared to controls. It was also observed that the degree of increase was more with ADP than epinephrine. Relatively, there was tendency of low platelet count.

The increase in the aggregation index can be attributed to the hyperreactiveness of the platelets to ADP than epinephrine. The relative thrombocytopenia can be accounted to the sequestration of platelets in the coronary microvasculature. In conclusion, balance between EDRF and EDCF determines the platelet activation.

Key words: AMI, platelet aggregation, ADP, epinephrine, aggregation index, EDRF, EDCF

INTRODUCTION

AMI is one of the vascular diseases having varied number of modifiable and non-modifiable risk factors (1). Among them, modifiable factors are hyperlipidemia, hypertension, diabetes mellitus, sedentary life, stress, smoking, alcoholism, whereas non-modifiable factors are age, sex, and heredity (2). Platelet–endothelium interaction plays a crucial role in the pathogenesis of AMI (3). Pathologically, it is defined as the necrosis of the part or whole of the myocardium due to inadequate coronary blood flow to that particular part (4). Several studies have documented that the platelets became abnormally hyperreactive and their survival time has been decreased in AMI (4). The activated Platelets, by suitable agonists in optimum

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concentration, release many vasoactive substances which simultaneously cause vasospasm and induce platelet aggregation and their recruitment (5, 6, 7). Thus the platelets have dual role of normal hemostasis and abnormal intra-arterial thrombosis (8, 9).

The present work was undertaken to compare the platelet aggregability in controls and in AMI patients as well as correlate the platelet aggregability with peripheral platelet count.

METHODS

I. Selection of the patients

Five patients were selected from ICCU between the age group of 40–60 years on the following diagnostic criteria (2) :

1. History of chest pain suggestive of cardiac disease.
2. ST segment elevation in the corresponding ECG leads.
3. Elevation of specific serum cardiac enzymes.

II. Selection of controls

Five controls were chosen from college staff who were not diabetic, hypertensive and without any history suggestive of cardiac and haemostatic diseases.

Equipment

1. Aggregometer (Cronolog dual channel with Recorder) (10, 11) : This is a photometer modified to permit measurement of changes in optical density of a platelet suspension under conditions of constant temperature and continuous agitation, which is connected to a pen recorder.

2. Coulter counter (T-540) (12) : This is the instrument which works on the basis of Coulter principle wherein the platelets are counted electronically.

Two investigations were carried out by using citrated blood drawn aseptically in the proportion of 1:9 vol/vol.

1. Aggregation study was conducted by using Chronolog dual channel aggrometer after separating PRP and PPP by differential centrifugation, wherein ADP and epinephrine were used as agonists in the conc. of 0.05 mM and 0.5 micro M respectively (final concentration) (6, 7).

2. The platelet count was done by automated haemocytometry by using 1 ml of anticoagulated blood.

The graph of aggregation index was recorded by using PRP to which 8 micro L of ADP and epinephrine were added along with magnetic stirring separately (6, 7).

The graph paper moves at the speed of 2.5 cm per min horizontally. Vertically, each division of 1 cm corresponds to 10% aggregation (6). The height of the single oscillation is proportional to the size of the aggregate and the slope of the graph indicates rate of the aggregation (10). The graph is either single phase or double phase depending on the agonists and their concentrations. The triad of percentage, rate and size of aggregation is designated as "Aggregation Index" (11).

RESULTS

The statistical analysis was done by Student's 't' test which was found to be significant.

TABLE I : Showing mean +/- SD of aggregation indices by using ADP.

Parameters	*Cases*		*Controls*		*P-value*
	Mean	*SD*	*Mean*	*SD*	
Aggn. %	72.00	18.00	41.00	16.80	<0.05(S)
Aggn. rate	57.00	23.15	32.00	11.35	<0.05(S)
Aggn. size	9.60	3.26	3.80	1.46	<0.05(S)
Platelet count	2.10	0.08	2.80	0.16	<0.05(S)

TABLE II : Showing mean +/- SD of aggregation indices by using epinephrine.

Parameters	*Cases*		*Controls*		*P-value*
	Mean	*SD*	*Mean*	*SD*	
Aggn. %	64.00	30.06	34.00	14.62	<0.05(NS)
Aggn. rate	38.40	22.07	19.80	4.75	<0.05(NS)
Aggn. size	5.60	2.41	3.60	2.24	<0.05(NS)
Platelet count	2.10	0.08	2.80	0.16	<0.05(S)

DISCUSSION

The increase in the aggregation indices may be attributed to the platelet hyperreactiveness and adhesiveness which has been found to be hallmark of AMI and could be due to alteration in the platelet membrane phospholipid (3, 12). Relative thrombocytopenia may be accounted to the sequestration of the platelets in the coronary microvasculature (8). The non-significant result of epinephrine induced curve could be due to circadian variation and stress induced release of epinephrine (13, 14).

Goldenferb et al found significant increase in platelet aggregation induced by ADP in AMI (15), which has been accounted to the imbalance in the circulating endothelium derived relaxing (EDRF) and contracting (EDCF) factors (3).

Finally, it may be emphasized that the platelet behaviour can be considered as independent to the other risk factors according to the studies shown by Gerrard J.M. and Julius Sagel et al. (6,7). But platelet behaviour i.e. aggregability can be aggravated by any of the modifiable and non-modifiable risk factors (2).

CONCLUSION

With this, probably following conclusion may be derived.

1. The platelets are the main participants in the formation of microthrombi in the coronary vasculature which could be further promoted by the epinephrine release due to stress.

2. The *"in vitro"* study can be implemented on a large scale as predictive, diagnostic and prognostic aid in the vascular diseases.

3. Balance between the level of EDRF and EDCF is probably the determinant of platelet activity.

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