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Research Article

RELATION OF PLATELET AGGREGATION RATIO IN MYOCARDIAL INFACTION

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ABSTRACT

Risk of platelet aggregation increases in hypercoagulable states like diabetic and dyslipidemic patients. We carried out a study in 100 patients with 30 patients as controls and compared platelet aggregation ratio in patients of MI with and without complication and compared it with control subjects. We came to a conclusion that platelet aggregation ratio was increased (lower PA ratio) in MI with complication as compared to MI without complication (0.50 to 0.75 mean of 0.61+-0.05 v/s 0.66 to 0.9 mean of 0.76+-0.02). Thus screening of this ratio in high risk IHD patients might lower the complications in acute events.
KEYWORDS: acute myocardial infarction, inflammation, platelet aggregation.

INTRODUCTION

Incidence of risk of development of unstable angina and coronary thrombosis is more in patients with CAD and PVD [1]. A hypothesis states that diabetics are high risk for CAD because diabetes itself predisposes to hypercoagulable state. The diurnal pattern of infarction has been associated with platelet aggregability,[2] and aggregation has been shown to be enhanced in the presence of factors relevant to an increased risk of infarction, such as a high intake of saturated fat [3] and diabetes[4]. Studies of the coronary arteries in AMI have shown that the unstable plaque formation is not just a local inflammatory phenomenon, but reflects the pan-coronary process of vulnerable plaque formation [5,6]. Upon activation, platelets degranulate and adhere to monocytes, a process mediated by the platelet surface expression of P-selectin (CD62P) [7] and [8], which binds to the constitutively expressed P-selectin glycoprotein ligand-1 (PSGL-1) on monocytes [9]. Firm attachment is subsequently mediated by monocyte CD11b/CD18-dependent platelet interactions [10]. Despite documented role of platelet aggregation in CAD, few methods are available for the detection of such aggregates.

Wu and hook introduced a simple method for detection of these aggregates by assessment of platelet aggregation ratio. As this method is simple and less time consuming we undertook this method for the assessment of platelet aggregates in myocardial infarction.

PLATELET AGGREGATION RATIO

Venous blood is mixed with formalin EDTA mixture in one tube and EDTA solution in another tube. These tubes are centrifuged to get platelet rich plasma (PRP). Platelet counts on both PRP samples are done and result is expressed as:

Platelet aggregate ratio = platelet counts in formalin/ platelet count in EDTA PRP

PRINCIPLE

Based on the principle that in the presence of formalin – EDTA mixture, platelet aggregate, when present would be fixed and centrifuged down and therefore the platelet count of PRP sample would be reduced. However platelet aggregates are broken in tube containing EDTA solution. Therefore the ratio approaches 1 in the absence of aggregate and drops below 1 when aggregates are present.

METHOD AND MATERIAL:

For the present study 100 cases of myocardial infarction were taken and 30 age and sex matched healthy subjects as controls.

These cases of MI were admitted within 12hrs of onset of MI out of 100 cases 60 were MI with complication and 40 MI without complication.

Other conditions known to increase platelet aggregation were excluded. None of these subjects was taking drugs known to alter platelet aggregation.

Platelet aggregation was determined by Wu and Hook method

RESULT

In 30 control subjects platelet aggregation ratio varied from 0.7 to 1.02 with the mean of 0.82 +/- 0.03. In cases of MI with complication, ratio varied from 0.50 to 0.75 with the mean of 0.61 +/- 0.05. This was a statistically significant difference when compared to control. (P value <0.001).

In cases of MI without complication the ratio varied from 0.66 to 0.9 with the mean difference of 0.76 +/- 0.02. This was a statistically significant difference as compared to controls (p value <0.001).

DISCUSSION

Estimation of platelet aggregation in an important factor in establishing a patient have coronary artery disease. As a proposed theory that contact of circulating platelet with damaged endothelium leads to platelet aggregation which in turn leads to the release of many platelet factors [11]. There is a positive correlation between PF4(platelet factor 4), and positive exercise test in CAD patients.[12] Increased plasma PF 4 levels have been reported in peripheral blood of patients with prinzmetal's angina.

In our study it showed that disturbance in PA ratio was a primary event and not the effect of infraction as it is showed that PA ratio was lower on the 1st day and in some cases in was lower within few hours of myocardial infarction. Thus increased level of circulating platelet aggregates predisposes a person to and acute event of myocardial infarction.

Platelet aggregation ratio was increased (lower PA ratio) in MI with complication as compared to MI without complication. This may be because of higher circulating levels of catecholamine's, sluggish circulation, prolonged bed rest and intramural thrombosis formation. Thus we suggest that patients with high risk for IHD should be screened for PA ratio and if found along with high risk factors anti platelet aggregation drugs should be considered.

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