Research Article

TO INVESTIGATE THE LEVELS OF PLASMA sP-SELECTIN AND THE LINK BETWEEN PLASMA SP-SELECTIN LEVELS AND THE CIRCULATING WBC COUNT IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION (AMI)

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ABSTRACT

This study was aimed to investigate the levels of plasma sP-selectin and the link between plasma sP-select in levels and the circulating WBC count in patients with AMI.

A total of 274 adults (18 years above) patients with clinically suspected acute coronary syndrome who attended in cardiac emergency and admitted in coronary care unit in the department of Cardiology, King George's Medical University, India were enrolled in this study. Among them 248 patients had ECG changes (ST-elevation, ST- depression, T-inversion, appearance of Q-wave) with or without elevated troponin I and treated with anti-plate- let drugs. They were considered as cases (group I). The rest 26 Patients had normal ECG findings, normal cardiac troponin I and did not receive anti-platelet therapy. They were considered as controls (group II). Clinical history, medical reports, findings and information were documented in a pre-designed data sheet with informed and written consent. Blood samples for plasma levels of sP-selectin were obtained in the catheterization laboratory before coronary angiography. Descriptive statistical analyses were performed using SPSS software (version 20, 2008). The study samples were 274 patients, with a mean age of 59.29 ± 11.94 years in cases group and 40.54 ± 19.15 years in control group. Male patient proportion was higher than female in both groups. In cases group, correlation between sP selectin with WBC shows Pearson correlation coefficient (r), and their significance value, sP selectin were directly associated with WBC and demonstrate a significant positive relation (r=0.234, p=0.010). sP-selectin was markedly elevated in cases of AMI. sP-selectin may be involved in modulating the recruitment of circulating WBC during AMI. These findings raise the need for a prospective investigation of sP-selectin as a potential reliable clinical tool for rapidly diagnosing AMI.

INTRODUCTION

Acute myocardial infarction (AMI) is the main cause of death in patients hospitalized with cardiovascular disease in industrialized societies [1].The underlying cellular and molecular mechanisms of disease initiation and progression are quite complex [2-4].An early event in vessel injury is the adhesion of platelets and leukocytes to the damaged arterial wall [5-6].Growing evidence has demonstrated that an acute inflammatory process which results in platelet aggregation and leukocyte adhesion to the endothelium in the coronary circulation plays a crucial role in thrombus formationand subsequently in the development of AMI [7].P-selectin, a cellular adhesion molecule of platelets and endothelial cells stored in both the -granules of platelets and in the Weibel-Palade bodies of endothelial cells [8], is rapidly expressed on the surface of activated platelets and endothelial cells [9]. It is involved in mediating platelets and the rolling of leukocytes on activated endothelial cellsas well as in interactions of activated platelets with leukocytes [10]. Soluble P-selectin (sP-selectin), is a form of P- selectin which can be detected in plasma and represents a marker of platelet activation. Until recently, little data on plasma levels of sP-selectin were avail- able in patients with acute coronary syndromes [11].Only two recent studieshave investigated the plasma levels of sP-selectin following AMI. They demonstrated that the levels of sP-selectin are significantly higher in patients in the clinical setting of AMI who are undergoing thrombolytic therapy than in healthy control subjects. Furthermore, while leukocyte accumulation plays an important role in promoting fibrin deposition [7], the relationship between increased plasma levels of sP- selectin and the recruitment of circulating WBC in patients with AMI remains undetermined. Whether sP-selectin

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can be utilized as a marker for an early, rapid diagnosis of AMI is also uncertain. This study was proposed to investigate the levels of plasma sP-selectin and the link between plasma sP-selectin levels and the circulating WBC count in patients with AMI.

MATERIALS AND METHODS

This study was conducted in Department of medicine, in collaboration with Department of Cardiology, King George's Medical University. Total 274 adults (18 years above) patients with clinically suspected acute coronary syndrome who attended in cardiac emergency and admitted in coronary care unit in the department of Cardiology, King George's Medical University, India were enrolled in this study. Among them 248 patients had ECG changes (ST-elevation, ST- depression, T-inversion, appearance of Q-wave) with or without elevated troponin I and treated with anti-plate- let drugs. They were considered as cases (group I). The rest 26 Patients had normal ECG findings, normal cardiac troponin I and did not receive anti-platelet therapy. They were considered as controls (group II). Clinical history, medical reports, findings and information were documented in a pre-designed data sheet with informed and written consent. Blood samples for plasma levels of sP-selectin were obtained in the catheterization laboratory before coronary angiography and determined by auto- mated analyzer.

Statistical analysis

Descriptive statistical analyses were performed using SPSS software (version 20, 2008). Data were summarized as Mean±SD. Groups (Present vs Absent (controls) were compared by unpaired or independent Student's t test. A two-tailed p<0.05 was considered statistically significant.

RESULTS

The study samples were 274 patients, with a mean age of 59.29±11.94 years in cases group and 40.54±19.15 years in control group (Table 1).

Table 1 Differentiation of cases and controls according to age group

	Group	N	Mean	Std. Deviation	
Age (Years)	Cases	248	59.29	11.94	<0.001*
	Controls	26	40.54	19.15	

Table 2 Male patient proportion was higher than female in both groups

		Group		Total
		Cases	Controls	
Gender	Male	204	22	226
		82.3%	84.6%	82.5%
	Female	44	4	48
		17.7%	15.4%	17.5%
Total		248	26	274
		100.0%	100.0%	100.0%

Table 3 Hematological results in all studied groups

	Group	N	Mean	Std. Deviation	P value
WBC	Cases	248	8.78	3.35	0.064
	Controls	26	7.52	2.72	
Soluble P selectin	Cases	248	56.71	6.91	<0.001*
	Controls	26	38.15	4.79	

Applied unpaired t test for significance. *Significant.

The sex ratio in our study population showed that male patient proportion was higher than female in both groups (Table 2).

Regarding sP-selectin, it was significantly higher in the cases compared to the control ($P = 0.001$) (Table 3). In cases group, correlation between sP selectin with WBC shows Pearson correlation coefficient (r), and their significance value, sP selectin were directly associated with WBC and demonstrate a significant positive relation (r=0.234, p=0.010) (Table 4, Figure 1).

Table 4 Correlation between sP selectin with WBC

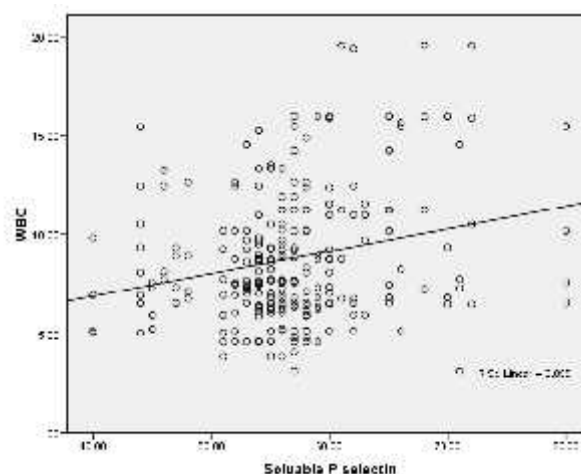|	Pearson correlation coefficient (r)	P value
WBC	0.234	0.010

Figure 1 Scatter diagram showing correlation between sP selectin with WBC

DISCUSSION

Platelet activation that results from coronary plaque rupture is vital in the pathogenesis of AMI. Soluble p-selectin (sP-selectin) is crucial in modulating leukocyte adhesion to both platelets and endothelial cells during inflammatory response and thrombus formation. This study was proposed to investigate the levels of plasma sP-selectin and the link between plasma sP-selectin levels and the circulating WBC count in patients with AMI. The study samples were 274 patients, with a mean age of 59.29±11.94 years in cases group and 40.54±19.15 years in control group. Male patient proportion was higher than female in both groups. Correlation analysis demonstrated that the increase in the plasma level of sP-selectin was significantly related to the circulating WBC count similar to other prospective cohort study of 142 consecutive patients with ST-segment elevated AMI of onset < 12 h who were undergoing primary percutaneous coronary intervention [12]. In another studya significant positive correlation was found between plasma sP-selectin and serum ferritin as well as total leucocytic count reflecting the interplay between platelet activation, iron free radicals and inflammation [13].

References

1. American Heart Association – 2000 Heart and Stroke Statistical Update. Dallas, TX: American Heart Association, 2000.

2. Hansson GK. Immune and inflammatory mechanisms in the development of atherosclerosis. *Br Heart J* 1993;69: S38-S42.

3. van der Wall AC, Becker AE, van der Loos CM, Das PK. Site of intimal rupture or erosion of thrombosed coronary atherosclerotic plaques is characterized by an inflammato- ry process irrespective of the dominant plaque morpholo- gy. Circulation 1994; 89:36-44.

4. Ross R. Atherosclerosis: an inflammatory disease. *N Engl J Med* 1999; 340:115-26.

5. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes. *New Engl J Med* 1992; 326:242-50.

6. Ross R. The pathogenesis of atherosclerosis: a prospec- tive for the 1990s. *Nature* 1993; 362:802-9.

7. Palabrica T, Lobb R, Furie BC, Aronovitz M, Benjamin C, Hsu YM, Sajer SA, Furie B. Leukocyte accumulation promoting fibrin deposition is mediated in vivo by P-selectin on adherent platelets. *Nature* 1992; 359:848-51.

8. Wagner DD. The Weibel-Palade body: the storage granule for von Willebrand factor and p-selectin. *Throm Haemost* 1993; 70:1-5.

9. Carlos TM, Harlan JM. Leukocyte-endothelial adhesion molecules. *Blood* 1994; 84:2068-101.

10. Gamble JR, Skinner MP, Berndt MC, Vadas MA. Prevention of activated neutrophil adhesion to endotheli- um by soluble adhesion protein GMP 140. *Science* 1990; 249:414-7.

11. Shimomura H, Ogawa H, Arai H, Arai H, Moriyama Y, Takazoe K, et al. Serial changes in plasma levels of soluble P-selectin in patients with acute myocardial infarction. *Am J Cardiol* 1998; 81:397-400.

12. Chiu C, Wu C, Yang C, Fang C, Hsieh Y, Hang C, Hung W, Chen C, Chen S, Yu T, Yeh KH. Levels and value of soluble P-selectin following acute myocardial infarction: evaluating the link between soluble P-selectin levels and recruitment of circulating white blood cells and the marker for the rapid diagnosis of chest pain. *Chang Gung medical journal*. 2005 Oct; 28(10):699.

13. Ebeid SA, Sadek NA, El-Benhawy SA, Mahmoud GN. Plasma substance P and soluble P-selectin as biomarkers of -thalassemia induced hypercoagulability. *Alexandria Journal of Medicine*. 2014 Jun 30; 50(2):107-11.

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