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Research Article

CIGARETTE SMOKING INCREASES THE MEAN PLATELET VOLUME IN ELDERLY PATIENTS WITH RISK FACTORS FOR MI

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ABSTRACT

This study was proposed to study the effects of cigarette smoking and myocardial Infarction on platelet size.

A total of 298 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 50 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 mean platelet volume (MPV) were obtained in the catheterization laboratory before coronary angiography and determined by auto- mated analyzer.

The mean age of controls was 40.9±17.9 and that in cases was 59.2±11.9. Among them 72.2% were smokers in cases groups while 60% were nonsmokers in control group. The mean platelet in case group was 301.01±177.1 and in control group 304.7±132.5. While the mean MPV in case group was 8.5.0±1.2 fl and in control group 7.8±0.4 fl ($p<0.001$). In Platelet; statistically no significant difference was observed between smoking status (smoker or nonsmoker for both groups cases and controls) (p value > 0.05). It was observed that at all these groups higher mean was observed in smoker in cases groups than other groups i.e. 307.6±192.72. In MPV; statistically significant difference was observed between smoking status (smoker or nonsmoker for both groups cases and controls) (p value < 0.05). It was observed that at all these groups higher mean was observed in smoker in cases groups than other groups i.e. 8.58±1.41.

These results suggest that smoking may increase platelet consumption in atherosclerotic vessels and that subsequently megakaryocytes are activated to produce larger platelets, which are more active. Thus, an increase in MPV due to smoking may also contribute to the acceleration of atherosclerosis and should be considered as a risk factor for atherosclerotic disease

INTRODUCTION

According to the WHO, about six million deaths worldwide each year occur owing to tobacco use (smoking and smokeless) [1]. In spite of the fact that tobacco smoking is positively associated with many diseases, increasing prevalence of smoking among young people is still a problem of severe concern for health professionals. A cigarette smoke contains over 4000 chemicals [2] and a cigarette smoker is exposed to harmful substances including nicotine, free radicals, carbon monoxide, and other gaseous products [3]. It is well known that smokers have higher risk for cardiovascular diseases, hypertension, inflammation, stroke, clotting disorder, and respiratory disease [4-8].

Platelets, also called thrombocytes, are a component of blood whose function (along with the coagulation factors) is to stop bleeding by clumping and clotting blood vessel injuries. Platelets are important for hemostasis, wound healing, and inflammation [9]. The increased platelet activity and thrombus formation and thromboembolic diseases are among the major cause of mortality in developed countries. Successful management of these diseases relies on early detection of progressive activation of coagulation.

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Mean platelet volume (MPV) is a machine-calculated measurement of the average size of platelets found in blood and is typically included in blood tests as part of the CBC. Since the average platelet size is larger when the body is producing increased numbers of platelets, the MPV test results can be used to make inferences about platelet production in bone marrow or platelet destruction problems. Larger platelets are more active than the smaller ones. These platelet parameters are estimated routinely by automated blood counters. As the point that platelet activation results in morphologic variations of platelets is known, a sequence of platelet parameters measured by hematology analyzers have been applied by several researchers.

The MPV is perhaps the most widely studied platelet activation parameter [10-11]. Mean platelet component and platelet component distribution width are the new indices that are assessed recently as potential platelet activation markers [12]. Nevertheless, these indices are not assessed by all the hematology analyzers. There are a very few studies relating the effect of smoking on platelets. In addition, many of the studies have not compared the data with those of the nonsmoking control groups [13-14]. Kario *et al*. [15] found elevated MPV in smoking patients, which reduced after the patients stopped smoking. However, Butkiewicz *et al*. [16] studied the impact of smoking on platelet activation and few other morphological indices including MPV and found no effect on MPV by smoking. Thus, studies on this have reported conflicting results. Hence, this work was undertaken to study the effect of cigarette smoking on platelet parameters (mean platelet volume) in elderly patients with risk factors for myocardial infarction.

MATERIALS AND METHODS

This study was conducted in Department of medicine, in collaboration with Department of Cardiology, King George's Medical University.

Total 298 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 50 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 Mean platelet volume (MPV) 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 cohort consisted of 248 patients diagnosed with MI and 50 healthy volunteer controls. The mean age of controls was 40.9±17.9 and that in cases was 59.2±11.9. There were 204 males in case group and 45 in control group.

Table 1 Association of smoking status within groups

Smoking Status		Group		Total
		Cases	Controls	
	Smoker	179	20	199
		72.20%	40.00%	66.80%
	Non-smoker	69	30	99
		27.80%	60.00%	33.20%
Total		248	50	298
		100.00%	100.00%	100.00%

P=<0.001

The association of smoking status with groups are evaluated and summarized in Table 1. It shows higher percentage was obtained in cases groups are smoker (72.2%) while 60% were obtained in control group are non-smoker. Statistically this association was significant (P<0.05).

Table 2 Mean platelet volume and platelet in the patients in case and control groups

	Group	N	Mean	Std. Deviation	P value
Platelet	Cases	248	301.01	177.12496	0.887
	Controls	50	304.76	132.54904	
MPV	Cases	248	8.551	1.21672	<0.001*
	Controls	50	7.892	0.47158	

Applied unpaired t test for significance.

The mean platelet in case group was 301.01±177.1and in control group 304.7±132.5. While the mean MPV in case group was 8.5.0±1.2 fl and in control group 7.8±0.4 fl (p<0.001)(Table 2).

In Platelet; statistically no significant difference was observed between smoking status (smoker or nonsmoker for both groups cases and controls) (p value > 0.05). It was observed that at all these groups higher mean was observed in smoker in cases groups than other groups i.e. 307.6±192.72

In MPV; statistically significant difference was observed between smoking status (smoker or nonsmoker for both groups cases and controls) (p value < 0.05). It was observed that at all these groups higher mean was observed in smoker in cases groups than other groups i.e. 8.58±1.41.

DISCUSSION

MPV is a platelet marker which can be obtained as a part of complete blood count(CBC) using an automated hematology counter which measures the average size of platelets present in the blood. In this study MPV was significantly higher in patients with AMI in comparison to the control subjects. This finding was in accordance with the observation by Yekelar *et al* [17]. AMI occurs due to coronary atherosclerosis and thrombus formation. Platelets play a significant role in atherosclerosis as well as thrombosis [18,19]. There are various studies where higher MPV has been correlated with metabolic syndrome,

hypertension, increasing age, and hyperlipidemia [20-21] but contradictory studies also exist [22].

Table 3 Correlation in cases and control groups.

		N	Mean	Std. Deviation	95% Confidence Interval for Mean		P value
					Lower Bound	Upper Bound	
Platelet	Smoker in cases	179	307.6	192.72	279.1702	336.0231	0.732
	Non-smoker in cases	69	283.93	127.76	253.2372	314.6179	
	Smokers in controls	20	321.3	148.71	251.7034	390.8966	
	Non-smoker in controls	30	293.73	121.99	248.1787	339.2879	
	Total	298	301.64	170.27	282.229	321.0515	
MPV	Smoker in cases	179	8.5885	1.40553	8.3812	8.7958	0.001
	Non-smoker in cases	69	8.4536	0.43911	8.3481	8.5591	
	Smokers in controls	20	8.2	0.29736	8.0608	8.3392	
	Non-smoker in controls	30	7.6867	0.45693	7.516	7.8573	
	Total	298	8.4404	1.15269	8.309	8.5718	

Applied one-way ANOVA

In the present study risk factors like smoking did not show correlation with platelet in either cases or controls. But in MPV; statistically significant difference was observed between smoking status (smoker or nonsmoker for both groups cases and controls) (p value < 0.05). It was observed that at all these groups higher mean was observed in smoker in cases groups than other groups i.e. 8.58±1.41.

CONCLUSION

MPV is a very low cost investigation and can be obtained easily in most health care settings. This study corroborates others observations that MPV is higher in patients with AMI.

These results suggest that smoking may increase platelet consumption in atherosclerotic vessels and that subsequently megakaryocytes are activated to produce larger platelets, which are more active. Thus, an increase in MPV due to smoking may also contribute to the acceleration of atherosclerosis and should be considered as a risk factor for atherosclerotic disease. However, we propose that it may be useful as a marker of myocardial infarction in an appropriate clinical situation. Further study may be tested in a larger cohort of patients with AMI to confirm its use as an adjunct to diagnosis.

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