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Mean Platelet Volume is Increased in Patients with Atrial Septal Aneurysm

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Abstract

Background: Systemic thromboembolism is a serious major complication in patients with Atrial Septal Aneurysm (ASA). However, the pathogenesis of thromboembolism in ASA is not fully understood. The aim of this study was to assess the Mean Platelet Volume (MPV), an indicator of platelet activation, in patients with ASA.

Patients and Methods: The study group consisted of 40 patients with ASA. An age, gender, and body mass index-matched control group consisted of 30 healthy volunteers. All patients and control participants underwent echocardiographic examination. We measured the serum MPV values in patients and control participants.

Results: Mean platelet volume was significantly higher among patients with ASA when compared with the control group (9.8 ± 1.1 vs 7.6 ± 0.8 fL, respectively; P < 0.001).

Conclusion: We have shown that MPV was significantly elevated in patients with ASA compared to control participant.

Keywords: Platelets; Arrhytmia; Atrial septal aneurysm; Mean platelet volume; Thromboembolism

Introductıon

Atrial Septal Aneurysm (ASA) is a risk factor for arterial embolism, as it frequently co-exists with an Atrial Septal Defect (ASD) or Patent Foramen Ovale (PFO). The incidence of ASA, depending on the imaging method, is 0.2-4 % in Transthoracic Echocardiography (TTE) and 2-8% in Transoesphageal Echocardiography (TEE). Despite prior reports concerning paradoxical embolism through a PFO, the magnitude of this phenomenon as a risk factor for a stroke remains undefined, because deep venous thrombosis is infrequently detected in such patients. atrial dysfunction and atrial arrhythmia, such as Atrial Fibrillation (AF) or platelet hyperactivity might represent an alternate and additional mechanism for arterial embolism in these patients [1-3].

Recent studies suggest that Mean Platelet Volume (MPV) is a potentially useful prognostic biomarker in patients with cardiovascular disease such as acute coronary syndrom, valvular heart disease pulmonary thromboembolism and hypertension [4-8].

For these reason the aim of this study was to assess the MPV , an indicator of platelet activation, in patients with ASA.

Methods

Selection of the patients

Forty patients were accepted in our study who had ASA as seen during echocardiography evaluation for chest pain et/or palpitation (15 males/25 females, mean age 54.4 ± 16.3 years) and 30 age-matched healthy volunteers were accepted as control groups (12 males/18 females, mean age 49.8 ± 11.2 years). Entry criteria included the presence of ASA by echocardiography and sinus rhythm on the surface ECG. A physical examination, the medical history of patients, and the blood biochemistry were evaluated in all groups. The subjects were defined as hypertensive if their blood pressure was ≥ 140/90 mmHg or if they were receiving any antihypertensive medication. Diabetes mellitus was defined as the presence of a history of antidiabetic medication usage or fasting glucose level above 126 mg/dl. Smoking status was classified as smokers or those who never smoked.

Patients with coronary artery disease, heart failure, valve disease, cardiomyopathy, hypertension, diabetes mellitus, chronic lung disease, thyroid dysfunction, anemia, malignancy, renal and hepatic insufficiency, chronic inflammatory disease, pregnancy, septicemia, cerebrovascular accident, and thrombocytopenia were exluded from the study. All of the patients were in sinus rhythm and none of them were taking cardioactive medications like antiarrhythmics, antiplatelet, antipsycotics, and antihistaminics. Every patient signed an informed consent form and the local ethics committee approved the study.

For the analysis of MPV, blood samples with K3 EDTA were analyzed after one hour of venipuncture by the Sysmex XT-2000i analyzer (Sysmex, Kobe, Japan).

Echocardiographic measurements

Two-dimensional, M-mode, pulsed and color flow doppler echocardioagraphic examinations of all subjects were performed by the same examiner with a commercially available machine (Vivid 7 pro, GE , Horten, Norway, 2-5 mHz phased array transducer). During echocardiography, a single-lead electrocardiogram was recorded continuously. ASA was detected by TTE. Patients who had an aneurysm with a base of > 15 mm and protrusion > 10 mm entered the study.

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M-mode measurements were performed according to the criteria of the American Society of Echocardiography [9,10].

The right atrium , Left Atrium (LA) diameter, LV end-systolic and end-diastolic diameters were measured.

LV ejection fraction (EF) was estimated by Simpson's rule.

Statistical analyses

The SPSS 16.0 statistical program (SPSS, Chicago, IL, USA) was used for the statistical study. Data were expressed as mean ± standard deviation (SD). Student *t*-test, one-way ANOVA- and chi-square test were used to compare the variables. A P value of less than 0.05 was considered significant.

Results

There was no statistically significant difference between patient group and the control with regard to age, gender, diameters of the left atrium and the left ventricle, pulmonary artery systolic pressure and body mass index (Table 1). Additionally there were no significant differences between the to groups with regard to lipid profile, fasting glucose levels,creatinin, white and red blood cell and platelet counts. However, MPV was found to be significantly higher in patients with ASA (9.4 ± 2.3 fl vs. 8.1 ± 2.0 fl, $p \leq 0.001$) (Table 2).

Discussion

ASA is a risk factor for arterial embolism. Potential mechanisms of stroke in patients with atrial septal abnormalities include paradoxical embolism from a venous source, direct embolization from thrombi formed within the aneurysm and the formation of thrombus as a result of atrial arrhythmias [11].

In previously published studies, the incidence of AF in ASA patients ranged from 0% to 23%. Similarly Janion et al. [12] reported that P wave dispersion (Pd) and paroxysmal AF was higher in ASA patients. In another study, it was reported that supraventricular arrhythmia Pd and atrial dysfunction was higher in ASA patients than in the control subjects [13]. Rigatelli et al., in a study of 98 patients with ASA, found that left atrial dysfunction was higher in the patient groups [3].

Recent studies suggest that MPV is a potentially useful prognostic biomarker in patients with cardiovascular disease such as acute coronary syndrome, valvular heart disease pulmonary and systemic thromboembolism and hypertension. However relationship between MPV and ASA is not defined.

The present study showed that MPV was significantly higher in patients with ASA compared to controls. It is known that platelets having dense granules are more active biochemically, functionally, and metabolically and are a risk factor for developing coronary and pulmonary thrombosis, leading to myocardial infarction. In previous studies, increased MPV was demonstrated in acute myocardial infarction [14,15], mitral and aortic stenosis [4,5], deep vein thrombosis [6], and hypertension [7]. The stroke in patient with ASA, platelet activation may play a considerable role. Because larger platelets secrete more prothrombogenic thromboxane A2, serotonin, beta-thromboglobulin, and procoagulant membrane proteins like P-selectin and glycoprotein IIIa [15].

	Patients (N=40)	Controls (N=30)	P-Value
Age (years)	54.4 ± 16.3	49.8 ± 11.2	NS
Male/female(n/n)	15/25	12/18	NS
LA diameter(mm)	33.5 ± 3.4	34.2 ± 3.6	NS
LV EDD (mm)	43.1 ± 4.1	44.2 ± 4,9	NS
LV ESD (mm)	22.6 ± 2.7	24.4 ± 2.2	NS
RA diameter (mm)	33.5 ± 3.3	32.8 ± 3.1	NS
LVEF (%)	64.2 ± 5.2	64.8 ± 5.3	NS
BSA (m2)	1.9 ± 0.5	1.8 ± 0.3	NS
Heart rate (bpm)	75.9 ± 8.2	69.3 ± 5.3	NS
SPAP (mmHg)	28.5± 3.8	25.4 ± 3,4	NS
SBP (mmHg)	121 ± 23	122.5 ± 25	NS
DBP (mmHg)	74.2 ± 11	80.3 ± 12	NS
BMI (kg/m^2)	25 ± 3.5	23 ± 3.1	NS
Smoking (n)	10	11	NS

LA: Left Atrium, LVEDD: Left Ventricular End-Diastolic Dimension, LVESD: Left Ventricular End-Systolic Dimension, RA: Right Atrium, LVEF: Left Ventricular Ejection Fraction, BSA: Body Surface Area, SPAP: Systolic Pulmoner Artery Pressure, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index

Table 1: Comparison of clinical and echocardiographic features of ASA patients and controls group.

	Patients (N=40)	Controls (N=30)	P-Value
Glucose(mg/dl)	97.1 ± 13.9	93.3 ± 13.0	0.989
Creatinin(mg/dl)	0.7 ± 0.2	0.8 ± 0.3	0.246
Total cholesterol(mg/dl)	195.9 ± 52	199.2 ± 56	0.356
Trigliserid(mg/dl)	124 ± 26	125 ± 29	0.112
LDL- cholesterol(mg/dl)	*125.3 ± 9.5*	*133.7 ± 15*	0.231
HDL- cholesterol(mg/dl)	45.8 ± 9.5	44.9 ± 8.6	0.141
White-blood cell count ($x10^3/mm^3$)	8.2 ± 2.5	8.8 ± 2.6	0.124
Hemoglobin (g/dl)	14.2 ± 2.5	14.3 ±2.6	0.168
Platelet count($x10^3$)	248.5 ± 62.1	282.3 ± 79.2	0.151
Mean platelet volume (fl)	9.8 ± 1.1	7.6 ± 0.8	< 0.001

Table 2: Comparison of biochemical parameters of ASA patients and controls group.

In this comprehensive study, we have demonstrated that MPV significantly higher in ASA patients than the control subjects and it is thought that this may be related to thromboembolism more common in those patients.

The most significant limitation of our study was the insufficient number of patients. The other limitations of our study was not to be prospective and not to include stroke patients.

In conclusion, our findings show that MPV is increased in patients with ASA, compared to controls. The increased MPV seems to predict an increase in the prevalence of thromboembolism and stroke in patients with ASA. For this reason effective anti-platelet therapy should be given in these patients. Further prospective studies are required to establish the clinical significance of increased MPV and to investigate the role of anti-platelet agents in ASA patients.

Conflict of Interest

There is not a financial or other conflict of interest between our work and that of the authors.

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