

Mean platelet volume as an indicator of disease in patients with acute pulmonary embolisms

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Abstract. Mean platelet volume (MPV) is a simple and easy method of assessing platelet function in routine clinical practice. The data concerning MPV in pulmonary embolism are controversial. Therefore, the aim of this study was to evaluate MPV levels and platelet numbers in patients with acute pulmonary embolisms.

This retrospective study was conducted in the emergency department of the Medical Faculty Hospital of Yuzuncu Yil University between January 2010 and April 2012. The study enrolled 67 patients with acute PE (36 females and 31 males) and 53 healthy controls (31 females and 22 males). The platelet number and MPV values in patients with acute pulmonary embolism were reviewed.

There were no statistically significant differences between the acute PE patients and the controls with respect to the MPV values and platelet numbers (both, $p > 0.05$). The MPV values were inversely correlated with the platelet number in the patients with acute PE ($r: 0.388$; $p < 0.001$).

These results suggest that MPV is not a reliable indicator for diagnosing acute pulmonary embolism. Further studies are necessary to confirm these findings.

Key words: Pulmonary embolism, platelet, mean platelet volume

1. Introduction

Pulmonary embolism (PE) can be an acute, life-threatening emergency, and studies have suggested that advanced age is a risk factor for this condition. The mortality of acute PE is still higher in spite of the advances in diagnostic modalities and therapeutic options (1). PE occurs when the main pulmonary artery or one of its branches suddenly closes because of the presence of a thrombus deported from elsewhere in the body, usually from the deep veins of the leg (2). The etiopathogenesis of thrombosis has been revealed with the Virchow triad (3): blood flow changes, vessel wall changes and coagulation disorders.

Platelets are the smallest cells of the peripheral blood and form the cellular component of hemostasis. Platelets perform aggregation from the effect of their secreted mediators and then combine with another to form a hemostatic plug. Large platelets produce prothrombotic factors (e.g., thromboxane A_2) and are more active than small platelets. Thromboxane A_2 is a vasoconstrictor and also a substance that facilitates platelet aggregation (4, 5).

The mean platelet volume (MPV) is a laboratory marker obtained from complete blood count (CBC) analysis in routine clinical practice (6). MPV is one of the best indicators of platelet function and activation. Increased MPV values are associated with shortened bleeding times (7).

Several studies have investigated a possible link between MPV and several myocardial infarctions, cerebrovascular disease, diabetes, ulcerative colitis, acute pancreatitis, celiac disease and rheumatoid arthritis (8-14). Previous studies have investigated whether MPV can be used as a prognostic indicator in acute PE (15-17). However, the results of these studies are conflicting. Therefore, the aim of current study was to evaluate the MPV levels and platelet numbers in patients with acute PE.

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2. Materials and methods

2.1. Subjects

This retrospective study was conducted in the emergency department of the Medical Faculty Hospital of Yuzuncu Yil University between January 2010 and April 2012. 67 patients with acute PE (36 females and 31 males) and 53 healthy controls (31 females and 22 males) were enrolled for the study.

A PE diagnosis is based on the clinical likelihood, serum d-dimer levels, compression ultrasonography of the lower limb, ventilation-perfusion lung scans, and/or helical computed tomography (CT) (3), usually CT pulmonary angiography.

Patients with heart failure, peripheral vascular disease, cardiovascular disease, cancer, hematologic and hepatic disorders and a history of drug use (anticoagulant medication and oral contraceptives) were excluded from the study.

The control group consisted of 53 age- and sex-matched healthy that none had hematological disease and vascular disease and a history of drug use (anticoagulant medication and oral contraceptives). These subjects were asymptomatic with an unremarkable medical history and a normal physical examination.

All CBC analyses were performed in the hematology laboratory of our hospital. MPV was measured from a blood sample collected in dipotassium EDTA tubes. An automatic blood counter (Beckman-Coulter, LH 780, USA) was used for whole blood counts. MPV was measured within 30 minutes after sampling to prevent EDTA-induced platelet swelling.

The study protocol was conducted in accordance with the Helsinki Declaration as revised in 2000 and approved by the Yuzuncu Yil University Medical Faculty. All subjects were informed about the study, and written consent form was obtained from each patient.

2.2. Statistical analysis

The results were explained as the mean \pm standard deviation. The parametric variables were

compared using Student's t-test. The non-parametric continuous variables were compared with the Mann-Whitney U-test. Chi-squared or Fisher's exact tests were used for the categorical variables, as appropriate. All statistical calculations were performed using the program SPSS for Windows (version 16.0; SPSS, Inc., Chicago, IL, USA). The results were considered statistically significant when the p value was <0.05 .

3. Results

The study group consisted of two groups: acute PE patients (n=67) and healthy controls (n=53). The demographic characteristics and clinical data of the acute PE and control subjects are presented in table 1.

The mean age of the acute PE patients was 57 ± 18 years, and the mean age of the control subjects was 58 ± 13 years. Of the 67 acute PE patients, 36 were female and 31 were male. Of the 53 control subjects, 31 were female and 22 were male.

There were no statistically significant differences between the two groups with respect to age and gender ($p > 0.05$).

There were no statistically significant differences between the acute PE patients and the controls with respect to the MPV values and platelet numbers (both, $p > 0.05$) (Table 1).

The MPV values were inversely correlated with the platelet number in the patients with acute PE ($r = -0.388$; $p < 0.001$).

4. Discussion

In this study, we investigated the MPV values, an indicator of platelet activation in patients with acute PE. In addition, we reviewed the platelet numbers in patients with acute PE. However, we observed no statistically significant differences between the acute PE patients and the controls with respect to the MPV values and platelet numbers. In addition, we found a negative correlation between the MPV values and the platelet numbers in patients with acute PE.

Table 1. Demographic characteristics of the patients with pulmonary thromboembolism and the control subjects

Parameters	Controls(n=53)	Patients (n=67)	p
Age (years)	58 \pm 13	57 \pm 18	0.647
Sex (female/male)	31/22	36/31	0.602
Platelet ($10^3/\text{mm}^3$)	249.4 \pm 84.8	240.7 \pm 112	0.638
MPV (fL)	8.4 \pm 1.1	8.4 \pm 1.20	0.931

MPV: Mean platelet volume
Values are mean \pm SD;

Several investigators have assessed the role of MPV in PE patients (15-17). These results have been contradictory. In the Varol et al. study (15), the MPV was reported to be increased in the acute PE patients compared to the controls. Conversely, Kostrubiec et al. (16) reported that no significant differences were found in the MPV levels of the patients with acute PE compared to the controls. Similarly, Hilal et al. (17) assessed the effect of MPV in patients with acute PE. However, in that study, there was no significant difference in the MPV levels of patients with acute PE compared to the healthy controls. The authors also reported that acute PE was not an important determinant in the disease severity and diagnosis. In our study, similar to the results of Kostrubiec et al. (16) and Hilal et al. (17), there were no alterations in the MPV values of the acute PE patients compared to the controls. In addition, we found a negative correlation between the MPV values and platelet numbers in patients with acute PE.

MPV is considered to be a marker and determinant of platelet function because larger platelets are hemostatically more reactive than normal size platelets. Several studies have observed that MPV reveals the platelet activity (18,19). In the field of vascular injury, platelets are the most important blood component in the thrombus formation after aggregation. Platelet function is related to their metabolic and enzymatic activities. The prothrombotic potential of large volume platelets are greater (20). The MPV size, which is one of the best indicators of platelet function and activation, is related to the increased aggregation, thromboxane synthesis, beta-thromboglobulin release and increased expression of the adhesion molecules (18).

Increased MPV, a simple marker of platelet activation (7,21), was found to predict fatal outcomes in acute coronary syndromes and was related to the severity of acute ischemic cerebrovascular events (22-25). Increased MPV has been observed in patients with cardiovascular diseases and in patient groups with known coronary artery disease risk factors (26). Moreover, increased MPV also plays a prognostic role in cardiovascular diseases, for example, it is associated with increased mortality following myocardial infarction (26, 27).

It has been well documented that platelet activation plays an important role in atherothrombosis (26). There is a relationship between platelet activation and some diseases that are accompanied by thrombosis and inflammation (28-30). In recent studies, it has been suggested that there are common

pathophysiological links between venous thromboembolism and atherothrombosis (31-33). Platelets play an important role in the pathophysiology of atherothrombotic cardiovascular disease and in the formation of thrombus. It has been thought that platelets play an important role in the formation of coronary artery thrombosis (34) and that large platelets are younger (35) and more reactive (36). However, an inverse relationship between the platelet count and MPV has been observed in some studies (37-38).

Reportedly, the MPV values that reflect significant changes in platelet reactivity and aggregation are increased in arterial and venous thrombus and are risk factors for thrombosis (39). Han et al. (40) have reported that MPV is a significant risk factor for deep vein thrombosis. Another study reported that the increased MPV values observed in thrombosis were the result rather than the cause of thrombosis (41). Moreover, Acikgoz et al. (42) have reported that in Behcet's disease, increased thrombosis was associated with MPV.

Our study has several limitations. First, this study has a cross-sectional design. Second, the number of study patients was small, and these observations must be confirmed in larger number of patient samples.

In conclusion, there were no significant differences between the acute PE patients and the controls with respect to the MPV values. In addition, we found a negative correlation between the MPV values and the platelet numbers in patients with acute PE. These results suggest that MPV is not a reliable indicator for diagnosing acute pulmonary embolism. Further studies are necessary to confirm these findings.

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