

Analysis of mean platelet volume in chronic obstructive pulmonary disease patients during acute attack.

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Abstract

Aims: We aimed to study the association between mean platelet volume (MPV), a marker of platelet activation and inflammation, in patients with Chronic Obstructive Pulmonary Disease (COPD) during acute attack.

Methods: Forty patients with acute exacerbation of COPD and another forty subjects in stable period of COPD were included to the study. White blood cell count, hemoglobin, hematocrit, platelet and MPV values of the study groups analyzed.

Results: MPV of the COPD patients with acute exacerbation was significantly higher than the control group ($p < 0.001$).

Conclusion: MPV increases during acute exacerbation of COPD. Due to its simplicity and inexpensive nature, it can be used as a complementary inflammatory marker of acute attack.

Keywords: Chronic obstructive pulmonary disease, Acute Attack, Mean platelet volume

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is characterized with progressive airway obstruction and inflammation. Inflammatory markers, such as, C-reactive protein (CRP), fibrinogen, erythrocyte sedimentation rate, inflammatory cytokines and leukocyte count, have been found to be related with COPD [1-4].

Mean platelet volume (MPV) refers the activation and production rate of the platelets. It has been accepted as a marker of inflammation in various diseases. Increased MPV reported in myocardial infarction, diabetes mellitus and hypertension [5-7]. On the other hand, decreased MPV reported in ulcerative colitis, ankylosing spondylitis and rheumatoid arthritis [8-10]. Both increased [11] and decreased [12] MPV values have been reported in COPD patients in literature.

In present retrospective analysis, we aimed to compare MPV values of the COPD patients during acute attack to those in during stable period.

Methods

Medical records of our institution Bitlis Guroymak State Hospital observed and data of COPD patients during acute attack obtained. Records of outpatient clinics observed and stable COPD patients' data were obtained. White blood cell count (WBC), hemoglobin (Hb), Hematocrit (Htc), platelet count (Plt), and MPV of the subjects recorded. All hematological assays conducted in the laboratory of our

institution by auto analyzer of Mindray (Mindray BC-3200, China). The original kit of the manufacturer used in assays.

Data analyzed by SPSS software (IBM SPSS 16.0, Chicago, IL, USA). Definitive values were expressed as median (min-max). Continuous variables were determined whether relevant for normal distribution by Kolmogorov Smirnov test. Mann Whitney U test used for comparison of nonhomogeneous distributed variables. Correlation analyse as conducted by Spearman correlation test. P value lower than 0.05 is considered as statistically significant.

Results

40 patients with acute attack of COPD and 40 subjects with stable COPD enrolled to the study. Acute exacerbation group was consisted of 20 men and 20 women with a median age of 63.6 (45-86) years. There were also 20 men and 20 women in stable COPD group with a median age of 65.5 (40-88) years. Characteristics of study group were summarized in Table 1.

Table 1. General characteristics and laboratory data of the study group.

Age (years)	Acute Attack	63,6 (45-86)
	Stable COPD	65,5 (40-88)
Men (n)	Acute Attack	20 (50%)
	Stable COPD	20 (50%)
Women (n)	Acute Attack	20 (50%)

	Stable COPD	20 (50%)
Hb(gr/dL)	Acute Attack	14,6 (11,7-18,20)
	Stable COPD	13,6 (6,5-10,1)
Htc (%)	Acute Attack	44,8 (38,7-55)
	Stable COPD	41,8 (31,1-50,8)
WBC (K/uL)	Acute Attack	11 (4,90-19,60)
	Stable COPD	7,9 (3,6-9,8)
Plt (K/uL)	Acute Attack	232 (106-504)
	Stable COPD	249 (140-415)
MPV (fL)	Acute Attack	8,96 (7,6-11,9)
	Stable COPD	7,92 (7-9,1)

MPV of patients with acute exacerbation was significantly higher than that of the stable COPD subjects ($p < 0,001$, Table 2).

Table 2. Comparison of MPV between study groups.

	N (%)	MPV		p*
		Median	(Min-Max)	
Acute Attack	40(%50)	8,96	(7,6-11,9)	<0,001
Stable COPD	40(%50)	7,92	(7-9,1)	

*Mann-Whitney U

WBC of the acute attack subjects (11,03) was also significantly higher than stable [7,19] COPD group ($p < 0,001$). WBC was positively correlated with MPV ($r = 0,209$) in acute exacerbation patients, however, this correlation did not reach the significance level ($p = 0,195$).

Discussion

The main finding of present study is increased MPV levels of COPD patients during acute attack, which may help, distinguishing stable and mild exacerbation of COPD. Chronic Obstructive Pulmonary Disease (COPD) is characterized with progressive airway obstruction and inflammation. Independence from exposure to smoking, platelet and monocyte aggregation increases in COPD patients, which further increases during exacerbation [13]. Because MPV reflects thrombocyte activation and inflammation, it may also reflect the inflammatory burden of acute attack in COPD. However, the results of studies observed MPV in COPD are conflicting in literature. While some studies observed elevated MPV [11,14], some others reported reduced MPV levels in acute attack of the COPD patients [12,15].

Similar to our study, increased MPV levels reported in various diseases, such as, chronic prostatitis [16]. Even conditions with mild inflammatory burden might also be associated with elevated levels of MPV [17]. On the other hand, some other disorders, e.g. hashimoto's thyroiditis [18] and nasal polyps

[19] have been reported to be characterized with decreased MPV.

Elevated MPV reported in present study may be explained by several mechanisms. First of all, platelets are activated in response to inflammatory stimuli and activated platelets become larger in size [20]. Inflammatory burden of acute exacerbation of COPD may interact with thrombopoiesis in bone marrow and cause production of larger platelets. However, by time, activated platelets involve and utilize at site of inflammation and remaining smaller platelets may cause a reduction in MPV levels in these population. Conflicting results in literature about MPV and COPD association could be explained with this phenomenon. In fact, beside inflammatory condition, MPV could be influenced by many co-factors, such as, method of the laboratory assay and the time between blood sampling and laboratory assessment [21].

Retrospective design of present study is a limitation which makes our results difficult to interpret. Another limitation could be the relatively small study population.

In conclusion, increased MPV is associated with acute exacerbation of COPD and, due to its easy to assess and inexpensive nature, it may be useful as a marker of acute attack in COPD, especially in mild cases.

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