ORIGINAL ARTICLE

Mean platelet volume as an inflammatory marker in acute exacerbation of chronic obstructive pulmonary disease

Sevinc S. Ulasli[1], Berna A. Ozyurek[2], Eylul B. Yilmaz[2], Gaye Ulubay[2]

1 Afyon Kocatepe University, Faculty of Medicine, Department of Pulmonary Diseases, Afyon, Turkey
2 Baskent University, Faculty of Medicine, Department of Pulmonary Diseases, Ankara, Turkey

KEY WORDS

acute-phase reactant, chronic obstructive pulmonary disease, exacerbation, mean platelet volume

ABSTRACT

INTRODUCTION Mean platelet volume (MPV) is inversely correlated with inflammation in inflammatory bowel diseases, rheumatoid arthritis, and ankylosing spondylitis as shown in the previous studies. It has been reported that elevated values of MPV are associated with cardiovascular diseases and stable chronic obstructive pulmonary disease (COPD). However, MPV values in acute exacerbation of COPD have not been investigated so far.

OBJECTIVES This retrospective study was conducted to investigate the relationships between MPV and acute phase reactants and functional parameters during COPD exacerbation.

PATIENTS AND METHODS The study included 47 patients with COPD with mild to very severe airway obstruction and 40 age-matched healthy subjects. C-reactive protein levels and complete blood count were analyzed and compared in patients during the stable period and during exacerbation of COPD.

RESULTS MPV values were 9.3 ±1.4 and 8.6 ±1.0 fl during stable period and during acute exacerbation, respectively. Mean MPV values in the control group were 9.3 ±0.8 fl. MPV values were significantly lower in patients during acute exacerbation than in those during the stable period of COPD and in control subjects (both, $P < 0.001$).

CONCLUSIONS The results suggest that assessment of MPV in COPD exacerbation may indicate systemic inflammation. Thus, MPV may be used as a negative acute-phase reactant in COPD exacerbation.

Correspondence to:
Sevinc S. Ulasli, MD, Afyon Kocatepe University, Faculty of Medicine, Department of Pulmonary Diseases, Ahmet Necdet Sezer Arastirma ve Uygulama Hastanesi, Ali Cetinkaya Kampusu, Izmir Karayolu 7. Km., 03200 Afyon, Turkey,
phone: +90-505-307-36-58,
fax: +90-272-246-33-22,
e-mail: sevincsarinc@gmail.com
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INTRODUCTION Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality in the world and is primarily characterized by the presence of airflow limitation resulting from inflammation and remodeling of the airways.[1] Increasing evidence indicates that COPD is a complex disease involving more than airflow obstruction.[2] It has been established that stable COPD is associated with low-grade systemic inflammation as demonstrated by an increase in blood leukocytes, acute-phase proteins such as C-reactive protein (CRP), and inflammatory cytokines.[3-5] COPD exacerbations are an important outcome in COPD because patients with exacerbations have impaired health status, reduced physical activity, and accelerated decline of lung function. COPD exacerbations are associated with an increase not only in airway inflammation but also in systemic inflammation.[6-8] Thus, an increase in inflammatory markers, such as blood leukocytes, CRP, erythrocyte sedimentation rate, and inflammatory cytokines, is observed and is most likely associated with lung function decline.[8,9]

Mean platelet volume (MPV) is one of the platelet function indices. It reflects the platelet production rate and stimulation.[10] MPV is a parameter generated by routine complete blood count test that is usually overlooked by clinicians.[11] Previous studies showed inverse correlations between MPV and disease activity in inflammatory bowel diseases, rheumatoid arthritis, and ankylosing spondylitis.[12-15] Two studies have demonstrated elevated MPV values in COPD patients.[16,17] However, to our knowledge, MPV values in acute exacerbation have not been investigated so far. Based

on this background, we sought to evaluate the relationships between MPV and acute-phase reactants and to clarify the decrease of MPV values during COPD exacerbation.

PATIENTS AND METHODS

Study population

We retrospectively enrolled 47 patients with the diagnosis of COPD exacerbation who were admitted to our outpatient and emergency clinics at the Pulmonary Diseases Department of the Baskent University, Faculty of Medicine, Ankara, Turkey, between the years 2008–2009. We recorded the follow-up data of the same patients in the stable period 3 months after an acute exacerbation. COPD was diagnosed according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, based on past smoking history, clinical evaluation, and pulmonary function tests, showing irreversible airflow obstruction.

An exacerbation of COPD was defined as sustained (48 hours or more) worsening of dyspnea, cough, or sputum production leading to an increase in the use of maintenance medications and/or supplementation with additional medications.[18,19]

Medical history, CRP levels, and complete blood count of 47 patients with acute COPD exacerbation were recorded. Complete blood count and CRP measurements were taken at first administration of medications for exacerbation and before initiation of any additional therapies such as systemic corticosteroids, antibiotics (oral/intravenous), and theophylline (intravenous). Diagnosis of a COPD exacerbation was based on a thorough review of the signs and symptoms such as sustained worsening of dyspnea, cough, or sputum production and/or purulence. Three months after an acute exacerbation, pulmonary function tests (PFTs), complete blood cell count, and measurement of CRP levels were performed in the same patients in the stable period of COPD. All patients with COPD were ex-smokers.

The control group included 40 age-matched healthy subjects without smoking history (men/women, 26/14; mean age, 68.7 ±9.2 years). Complete blood cell count was also performed in controls.

In our study population, patients with mild COPD were taking short-acting β_2-agonists and/or anticholinergic agents, patients with moderate COPD were on inhaled long-acting β_2-agonists and/or anticholinergic agents, and those with severe-to-very-severe COPD – additional inhaled corticosteroids and/or oral theophylline in the stable period.

Patients with acute exacerbation were treated depending on disease and exacerbation severity. β_2-agonists, the addition of anticholinergics (or an increase in dosage), the intravenous administration of corticosteroids, antibiotic therapy when indicated, and the intravenous administration of methylxanthines such as theophylline were used.

Patients who had another exacerbation in the 3-month period, patients with hypoxia, acute and/or chronic pulmonary thromboembolism, obstructive sleep apnea, cor pulmonale, coronary artery disease, connective tissue and inflammatory bowel diseases, and noncompliant patients were excluded from the study.

Pulmonary function testing

A clinical spirometer (SensorMedics Vmax Spectra 229, Bilthoven, Netherlands) was used for all assesments, and a laboratory technician demonstrated each respiratory maneuver for each subject before testing. Patients were instructed to perform forced expirations until 3 acceptable measurements were obtained according to the European Respiratory Society criteria.[20,21] Each recorded result was expressed as a percentage of the predicted value for that parameter. Predicted values were calculated according to the system developed by Quanjer et al.[22] These results were used to define disease severity according to the GOLD classification, namely, Stage I (mild): forced expiratory volume in 1 second to forced vital capacity ratio (FEV_1/FVC) <70% and FEV_1 ≥80% predicted; Stage II (moderate): FEV_1/FVC <70% and 50% ≤FEV_1 <80% predicted; Stage III (severe): FEV_1/FVC <70% and 30% ≤FEV_1 <50% predicted, Stage IV (very severe): FEV_1/FVC <70% and FEV_1 <30% predicted or FEV_1 <50% predicted plus chronic respiratory failure.[1]

Laboratory measurements

Complete blood cell counts were measured by an automatic blood counter (A Cell-Dyn 3700, Abbott, Illinois, United States). Blood was collected in potassium-ethylenediaminetetraacetic acid tubes and measured within 1 hour after venipuncture The expected MPV values in our laboratory ranged between 7.0 and 12.0 fl.

CRP levels were determined by the immunoturbidimetric method. Complete blood cell count and CRP in patients were recorded both for the stable period and exacerbation.

Statistical analysis and ethical committee

All statistical analyses were assessed using the SPSS program (SPSS version 15.0; SPSS Inc., Chicago, Illinois, United States). The χ^2, Kruskal-Wallis, and Mann-Whitney tests were used to compare the parameters of the patients in the stable period and in exacerbation and control subjects. All parameters were expressed as mean values ± standard deviation. The Pearson's correlation analysis was used to investigate the relationship between the parameters. A P value less than 0.05 was considered statistically significant.

The study was approved by the Institutional Review Board of the Baskent University. The research protocol complies with the 2000 Declaration of Helsinki.

RESULTS

The characteristics of patients with COPD and the results of PFTs and complete blood

TABLE 1 Demographics, functional parameters, and laboratory results of patients during the stable period and controls

Parameters	COPD patients (n = 47)	Control group (n = 40)
age, y	70.6 ±10	68.7 ±9.2
BMI, kg/m²	26 ±4.8	27.8 ±4.6
sex, male/female	37/10	26/14
smoking, pack years	19.5 ±7	–
FEV_1/FVC	53 ±12.7	78.9 ±4.8
FEV_1, %	54.5 ±24.5	123 ±14.2
FEV_1, l	1.37 ±0.7	3.6 ±0.7
FVC,%	78.6 ±27.5	131.5 ±15.7
FVC, l	2.57 ±1	4.6 ±0.9
IC, l	1.91 ±0.7	4.2 ±1.1
Hb, g/l	141 ±17	142 ±15
hematocrit	0.42 ±0.04	0.42 ±0.04
WBC count, /µl	7524 ±1918	7530 ±1727
neutrophil, %	61.3 ±7.1	57.4 ±5.9
platelets, ×10³/µl	301 ±30	258 ±43
MPV, fl	9.3 ±1.3	9.3 ±0.8
CRP, mg/l	6.7 ±4.2	2.5 ±2.4

Data are presented as mean ± standard deviation.

Abbreviations: BMI – body mass index, COPD – chronic obstructive pulmonary disease, CRP – C-reactive protein, FEV_1 – forced expiratory volume in 1 second, FVC – forced vital capacity, Hb – hemoglobin, IC – inspiratory capacity, MPV – mean platelet volume, WBC – white blood cell

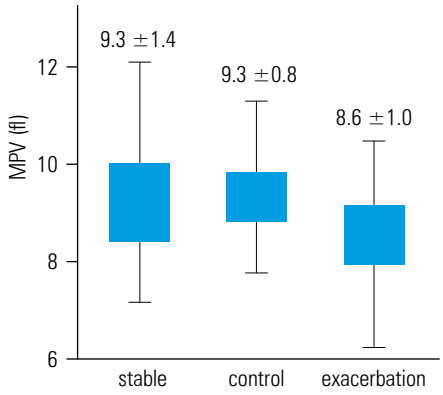

FIGURE 1 Mean platelet volume (mean ± standard deviation) in the study population (stable patients, controls, and patients with exacerbation) Abbreviations: see TABLE 1

cell counts are presented in TABLE 1. All COPD patients were ex-smokers (mean pack years, 19.5 ±7.0).

The mean MPV values of COPD patients were 9.3 ±1.4 fl in the stable period and 8.6 ±1 fl in exacerbation. The mean MPV values of age-matched controls were 9.3 ±0.8 fl. The MPV values of all groups are presented in FIGURE 1. Mean MPV values in exacerbation were significantly lower than those in the stable period or in the control group (P <0.001, P <0.001, respectively). There was no statistically significant difference in MPV values between patients with stable COPD and controls (P = 0.86).

There were no significant correlations between MPV and platelet count either in patients with stable COPD or those with exacerbation (r = –0.13, P = 0.36 and r = –0.20, P = 0.17, respectively). There was a significant correlation between the body mass index (BMI) and MPV in stable COPD (r = 0.31, P = 0.035).

CRP levels and complete blood cell count results were also assessed in 4 stages according to airflow limitation (TABLE 2). There were no significant correlations between CRP and complete blood cell count and disease severity either in patients with stable COPD or those with exacerbation.

CRP levels and complete blood cell count were compared between patients with stable COPD and those with exacerbation. CRP levels, white blood cell (WBC) count, and neutrophil percentages were increased and MPV values were decreased in patients with exacerbation (TABLE 3).

The mean CRP levels were 6.7 ±4.2 in the stable period and 46.3 ±29.2 in COPD exacerbation. There were statistically significant correlations between CRP and WBC count and between CRP and neutrophil percentage in patients with COPD exacerbation (r = 0.45, P = 0.001 and r = 0.29, P = 0.046, respectively; FIGURES 2 and 3).

We observed significant negative correlations between inspiratory capacity (IC) and CRP levels in the stable period and in exacerbation (r = –0.48, P = 0.02 and r = –0.54, P = 0.008, respectively; FIGURE 4) although there were no correlations between FEV_1 (% and l) and CRP levels during a stable and exacerbation period. Moreover, there were no significant correlations between FEV_1 results (% and l) and WBC count and neutrophil percentage in patients with stable COPD and those with exacerbation.

CRP levels and BMI did not correlate significantly either in patients with stable COPD or exacerbation (r = –0.05, P = 0.73 and r = –0.06, P = 0.71, respectively).

There was a statistically significant correlation between neutrophil percentage and MPV in patients with exacerbation (r = –0.4, P = 0.013). No statistically significant correlations were observed between MPV and FEV_1 (r = 0.06, P = 0.64) or between MPV and other PFT results. There were no correlations between MPV and CRP either in patients with stable COPD or exacerbation (r = 0.08, P = 0.58 and r = 0.07, P = 0.68, respectively).

DISCUSSION COPD should be considered a complex systemic disease involving several organs and systems (musculoskeletal, cardiovascular, endocrine) as well as metabolic abnormalities leading to weight loss.[2,23] Recent studies have shown that COPD is associated with low-grade systemic inflammation including systemic oxidative stress, activation of circulating inflammatory cells, and increased levels of inflammatory cytokines. The levels of inflammatory proteins such as CRP, fibrinogen, and proinflammatory cytokines are increased in the systemic circulation of patients with stable COPD.[2,3,24] During exacerbation periods, this inflammatory state becomes worse and higher levels of interleukin 6, CRP, fibrinogen, and lipopolysaccharide-binding protein

TABLE 2 Complete blood cell count and C-reactive protein during the stable period and during exacerbation according to the Global Initiative for Chronic Obstructive Lung Disease classification of disease severity

		Stage I (n = 7)	Stage II (n = 18)	Stage III (n = 16)	Stage IV (n = 6)	*P*
Hb, g/l	stable	148 ±18	141 ±17	137 ±17	145 ±18	0.75
	exacerbation	142 ±15	138 ±22	140 ±17	144 ±21.9	0.91
WBC, /µl	stable	7922 ±1271	7589 ±2289	7397 ±1840	7195 ±1873	0.76
	exacerbation	9065 ±3132	9744 ±4539	9173 ±3916	10 805 ±7105	0.84
neutrophil, %	stable	59 ±6	63 ±6.9	60.6 ±8.4	59.8 ±3.8	0.51
	exacerbation	62 ±17	66.9 ±11.6	67.0 ±8.26	68.4 ±10.9	0.63
platelets, × 10^3/µl	stable	254 ±54	272 ±103	378 ±50,7	237 ±29	0.98
	exacerbation	273 ±53	270 ±98	273 ±113	251 ±41	0.88
MPV, fl	stable	8.9 ±1.04	9.1 ±1.5	9.14 ±1.3	9.37 ±1.34	0.81
	exacerbation	8.2 ±1.1	8.9 ±1.02	8.3 ±0.7	8.2 ±1.5	0.19
CRP, mg/l	stable	6.2 ±4.7	5.9 ±4.1	6.2 ±4.5	6.0 ±4.5	0.99
	exacerbation	44.5 ±7.4	38.5 ±8.6	59.3 ±8.5	53.8 ±6.9	0.96

Abbreviations: see TABLE 1

TABLE 3 Complete blood count and C-reactive protein during the stable period and during exacerbation

	Stable period	Exacerbation	*P*
Hb, g/l	141 ±17	140 ±19	0.6
hematocrit	0.42 ±0.04	0.42 ±0.05	0.9
WBC, /µl	7524 ±1918	9584 ±4454	<0.001
neutrophil, %	61.3 ±7.1	66.4 ±10.3	0.002
platelet, × 10^3/µl	301 ±30	269 ±91	0.479
MPV, fl	9.3 ±1.3	8.5 ±1.03	<0.001
CRP, mg/l	6.7 ±4.2	46.3 ±29.2	<0.001

Abbreviations: see TABLE 1

have been demonstrated, decreasing again during recovery.[25,26]

MPV is one of the most widely used surrogate markers of the platelet function and has been shown to reflect inflammatory burden in different chronic diseases.[12-15] Overproduction of proinflammatory cytokines and acute-phase reactants can suppress the size of platelets by interfering with megakaryopoiesis and a subsequent release of small-size platelets from the bone marrow.[27] Moreover, Becchi et al.[28] also found a negative MPV trend in sepsis. However, to our knowledge, MPV values in acute COPD exacerbation have not been assessed before our study. We observed that MPV decreased during COPD exacerbation compared with the stable period and the control group while serum leukocyte count and neutrophil percentage were increased. Moreover, a negative correlation betweeen MPV and neutrophil percentage was observed indicating increased systemic inflammation during COPD exacerbation. Therefore, we suggest that decreased MPV values could predict acute exacerbation of COPD similarly to other inflammatory markers.

MPV can also potentially provide useful clinical data and thus be incorporated in the risk assessment algorithm for venous thromboembolism and arterial thrombosis.[29,30] Larger platelets are probably younger, more reactive, and produce more thrombogenic factors.[31] Elevated MPV values have been associated with cardiovascular diseases, although a recent study showed no correlation between MPV and coronary artery disease.[32-36] Bansal et al.[16] reported that MPV values were significantly higher in 100 patients with COPD compared with 100 healthy subjects, which could be due to hypoxia causing bone marrow stimulation or increased sequestration of smaller platelets with larger platelets remaining in the circulation. Onder et al.[17] reported the effect of hypoxia on thrombocytes in COPD patients and found that MPV values were significantly increased in hypoxic patients with COPD compared with nonhypoxic subjects and controls. Biljak et al.[37] found that 109 patients with COPD in the stable period had significantly increased platelet count and reduced MPV compared with 51 healthy controls. However, the groups were not matched for age and had a different smoking status.

The above 3 studies in patients with COPD did not evaluate MPV values during acute COPD exacerbation.[16,17,37] In our study, MPV values in patients with stable COPD were also higher than those in the control group, and MPV values of the patients with stage IV COPD during the stable period were the highest; however, these results did not achieve statistical significance. These results

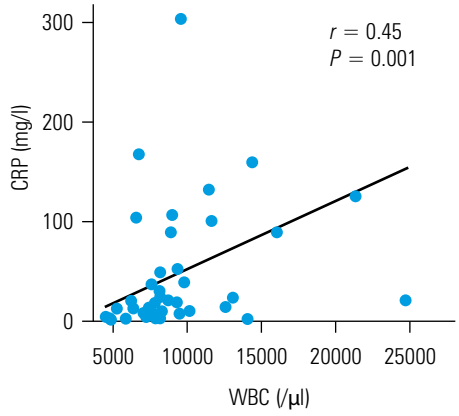

FIGURE 2 C-reactive protein levels and white blood cell count during exacerbation
Abbreviations see **TABLE 1**

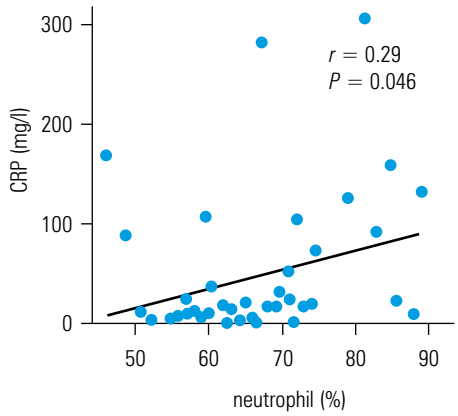

FIGURE 3 C-reactive protein levels and neutrophil percentage during exacerbation
Abbreviations see **TABLE 1**

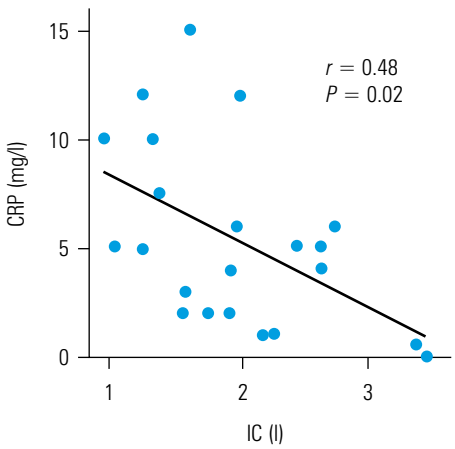

FIGURE 4 C-reactive protein levels and inspiratory capacity during the stable period
Abbreviations: see **TABLE 1**

might be due to the small sample size, lower number of patients at stage IV, and strict patient selection criteria as we excluded patients with hypoxia, acute or chronic pulmonary thromboembolism, and cardiovascular diseases.

CRP is an acute-phase protein synthesized predominantly by hepatocytes in response to tissue damage or inflammation. It reflects the total systemic burden of inflammation of individuals and has been shown to be increased in COPD in stable condition and during exacerbation.[24,38-40] In line with the available data, the mean CRP levels of our patients with exacerbation were increased, and statistically significant correlations between CRP levels and WBC count and between CRP levels and neutrophil percentage during exacerbation were revealed.[2] On the other hand, we did not observe a correlation between MPV values and CRP levels either in patients with stable COPD or exacerbation, which might be attributed to the baseline increase in CRP levels, high MPV levels in the stable period, and small sample size.

In the previous cross-sectional studies, CRP has been observed to be associated with FEV_1, but no association with a progressive decline in FEV_1 has been reported in longitudinal studies.[41] Dentener et al.[26] also did not find any correlations between CRP levels and lung function in patients with stable COPD. We did not observe a correlation between CRP levels in stable COPD and during exacerbation and FEV_1 in the stable period, and due to the limitation of this retrospective study we could not obtain PFT results in patients with COPD exacerbation so we were unable to analyze the association between FEV_1 decline and CRP levels. Torres et al.[42] reported that CRP levels correlated independently with other important prognostic clinical variables such as IC/total lung capacity (TLC) in stable COPD, but they did not investigate the association between IC and CRP levels.[42] They were the first to report an inverse correlation between lung hyperinflation as determined by the IC/TLC ratio and circulating CRP levels. In our study, IC in the stable period was negatively correlated with CRP levels in patients with stable COPD and with exacerbation. This result confirms the existing data supporting the fact that when lung function deteriorates, CRP levels increase.[43-45] MPV was not correlated with PFT results – either FEV_1 or IC. Moreover, MPV values were not significantly different between the stages of COPD in our study, which is in line with the results of Biljak et al.[37] These findings suggest that MPV could be used as an inflammatory marker in exacerbations independently from disease severity. Further prospective follow-up studies on a larger number of subjects are needed to investigate these relationships and validate the findings.

CRP levels have been significantly higher in COPD patients with low BMI in the previous studies.[46,47] In the present study, CRP was not correlated with BMI because of the differences in the study population as we included more patients with mild obstruction and fewer patients with severe and very severe obstruction, and the mean BMI of our patients was higher than that of the patients in other studies. On the other hand, we observed a correlation between MPV and BMI in the stable period. Although this positive correlation was previously demonstrated in different patient populations,[48-51] we are the first to report it in patients with COPD. Thus, we assumed that decreased MPV values might indicate malnutrition and increased inflammatory response in COPD. The correlation between BMI and MPV in COPD will have to be confirmed in further studies.

In conclusion, our results suggest that decreased MPV values might indicate increased systemic inflammation during COPD exacerbation.

Therefore, MPV could be used as a negative acute-phase reactant in acute COPD exacerbation. Measurement of changes in MPV values during follow-up may be considered as a quick and reliable tool in the assessment of inflammatory response.

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ARTYKUŁ ORYGINALNY

Średnia objętość płytek krwi jako marker zapalenia w fazie zaostrzenia przewlekłej obturacyjnej choroby płuc

Sevinc S. Ulasli[1], Berna A. Ozyurek[2], Eylul B. Yilmaz[2], Gaye Ulubay[2]

1 Afyon Kocatepe University, Faculty of Medicine, Department of Pulmonary Diseases, Afyon, Turcja
2 Baskent University, Faculty of Medicine, Department of Pulmonary Diseases, Ankara, Turcja

SŁOWA KLUCZOWE

parametr ostrej fazy, przewlekła obturacyjna choroba płuc, średnia objętość płytek krwi, zaostrzenie

STRESZCZENIE

WPROWADZENIE Średnia objętość płytek krwi (*mean platelet volume* – MPV) jest zależna w sposób odwrotnie proporcjonalny od stopnia intensywności procesu zapalnego w chorobach zapalnych jelit, reumatoidalnym zapaleniu stawów i zesztywniającym zapaleniu stawów kręgosłupa, jak donoszą dotychczasowe badania. Wykazano, że zwiększone wartości MPV wiążą się z chorobami sercowo-naczyniowymi i stabilną fazą przewlekłej obturacyjnej choroby płuc (POChP). Nie badano dotąd MPV w zaostrzeniu POChP.

CELE To badanie retrospektywne miało na celu prześledzenie związku między MPV a wskaźnikami ostrej fazy i parametrami funkcjonalnymi podczas zaostrzenia POChP.

PACJENCI I METODY Do badania zakwalifikowano 47 pacjentów z POChP o obturacji dróg oddechowych od łagodnej do bardzo ciężkiej oraz 40 zdrowych ochotników w podobnym wieku. Przeanalizowano stężenie białka C-reaktywnego oraz morfologię krwi i porównano oba parametry u pacjentów z POChP w fazie stabilnej i w zaostrzeniu choroby.

WYNIKI Wartości MPV wynosiły odpowiednio 9,3 ±1,4 fl w okresie stabilnym i 8,6 ±1,0 fl w czasie zaostrzenia POChP. Średnie wartości MPV w grupie kontrolnej wynosiły 9,3 ±0,8 fl. Wartości MPV były znamiennie niższe u pacjentów z POChP podczas zaostrzenia niż u osób w stabilnym okresie choroby i u osób z grupy kontrolnej (dla obu porównań $p < 0{,}001$).

WNIOSKI Wyniki badania wskazują, że ocena wartości MPV w czasie zaostrzenia POChP może wskazywać na ogólnoustrojowy proces zapalny. Z tego powodu MPV może być uważana za ujemny wskaźnik ostrej fazy podczas zaostrzenia POChP.

Adres do korespondencji:
Sevinc S. Ulasli, MD, Afyon Kocatepe University, Faculty of Medicine, Department of Pulmonary Diseases, Ahmet Necdet Sezer Arastirma ve Uygulama Hastanesi, Ali Cetinkaya Kampusu, Izmir Karayolu 7. Km., 03200 Afyon, Turcja,
tel.: +90-505-30-73-658,
fax: +90-272-24-63-322,
e-mail: sevincsarinc@gmail.com

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