

Evaluation of Mean Platelet Volume Role in Predicting COPD Prognosis

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ABSTRACT

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Introduction: Platelets attract leukocytes to the site of inflammation, and these cells engage in several intracellular and intercellular processes that participate in subsequent thermogenesis. Mean platelet volume (MPV) was also found to be decreased in chronic obstructive pulmonary disease (COPD) patients due to the inflammatory burden manifested in exacerbations, extensive platelet destruction, or the use of larger platelets at the inflammation site during the intercellular interactions. This study aimed to evaluate the role of MPV in predicting COPD prognosis. **Patient and methods:** A retrospective cross-sectional study included 51 COPD patients from those attending the chest department at Benha University Hospital from December 2021 to January 2023. All patients were subjected to the following: written consent from the patients, full history taking, and clinical examination including BMI, CXR P-A view, CBC with differential, CRP and Pulmonary function tests. **Results:** A significant positive correlation was found between forced expiratory volume (FEV %) and MPV. Also, there was an inverse highly significant correlation between FEV% and C - reactive protein (CRP). A significant negative correlation was detected between FEV1% and neutrophil to lymphocyte ratio (NLR) and an inverse highly significant correlation was also detected between FEV1% and platelet to lymphocyte ratio (PLR). Regression analysis indicates that CRP and NLR were the most statistically significant indicators for FEV% level respectively. On the other hand, MPV was a significant positive predictor for FEV% level.

Conclusion: MPV may serve as a useful biomarker for predicting COPD severity, with lower MPV values associated with worse disease progression. CRP and NLR also demonstrated strong prognostic relevance.

Keywords: Mean platelet volume, COPD, forced expiratory volume.

Introduction

Platelets are the main mediators of hemostasis and thrombosis ^[1]. A new function of platelets in the initiation and maintenance of inflammation and immunity has been recognized ^[2]. Chronic obstructive pulmonary disease (COPD) is a multisystem disease, resulting in continued morbidity and mortality in patients. COPD is characterized pathologically by air passage inflammation, mucus hypersecretion, breakdown of tissue with impairment of repair, and remodeling of lung vessels of the lung ^[3].

Recent evidence suggests that the lung could be an essential organ for platelet biogenesis with the bone marrow as the primary site, thus implicating the potential role of platelet mediators in the development and progression of COPD ^[4]. Platelets participate in the pathogenesis of COPD by various mechanisms, including stimulation of elastase activity, formation of platelet aggregates, and modulating signaling pathways of hypoxia ^[5].

Neutrophil to lymphocyte ratio (NLR) value was significantly elevated in stable COPD patients compared with healthy subjects, which suggests that systemic inflammation is persistent in this population. This observation is consistent with other published data that showed an elevation of inflammatory markers such as IL-6 and CRP in patients with stable COPD compared with healthy controls ^[6]. Thus, this study aimed to evaluate the role of mean platelet volume in predicting COPD prognosis.

Patients and methods

This retrospective cross-sectional study included 51 COPD patients who attended the Chest Department at Benha University

Hospital and its outpatient clinics during the period from December 2021 to January 2023. The study was conducted after obtaining approval from the Research Ethics Committee at the Faculty of Medicine, Benha University {R.C.4.6.2024}. Written informed consent was obtained from all participants after providing a detailed explanation of the study's purpose. To ensure confidentiality, each patient was assigned a secret code number.

Inclusion criteria: All COPD patients included in the study were in a stable phase of the disease.

Exclusion criteria:(1) The presence of systemic inflammatory diseases, severe liver disease, renal failure, malignant diseases, bone marrow failure, active infection, diabetes with severe complications, lung diseases other than COPD, and iron deficiency anemia. (II) History of platelet-modifying drugs, including aspirin and Plavix.

Methods: All patients were subjected to the following; written consent from the patient, full history taking and clinical examination including BMI, CXR P-A view, CBC with differential and CRP, and Pulmonary function tests.

Statistical analysis:

Categorical variables were presented as n (%), whereas numerical variables were presented as mean \pm standard deviation (SD) or median (inter-quartile ranges Q1, Q3) for normally distributed and skewed data, respectively. The normality of distributions was checked with Kolmogorov–Smirnov test. Correlations were assessed using Spearman's correlation coefficients for skewed distributed variables. Linear regression analysis was used to detect predictors of FEV% level. Statistical significance was

established at $p \leq .05$. Data were analyzed using SPSS 26.0 for Windows (SPSS Inc., Chicago, IL, USA).

Results:

The studied population consisted of 51 patients; the mean age (\pm SD) was 60.41 ± 9.6 ranging from 40 to 78 years. All studied groups were males (100%). The mean BMI was 31.02 ± 6.8 ranging from 22 to 47. Regarding laboratory findings and pulmonary function tests, the median CRP was 6.2 [IQR 3, 10]. The median WBC was 7.6 [IQR 5.9, 9]. The median PLT was 206 [range 170, 283]. The median MPV was 8.7 [IQR 7.8, 9.4]. The median PLR was 106.8 [IQR 84.4, 146.7]. The median NLR was 1.8 [range 1.19, 2.4]. The median FEV1% was 67 [IQR 56, 98] (Table, 1).

Correlation analysis showed significant positive correlation between FEV% and MPV ($\rho = 0.332$, $p = 0.017$). Also, there was inverse highly significant correlation between FEV% and CRP ($\rho = -0.599$, $p = 0.000$). Significant inverse correlation was detected between FEV1% and NLR ($\rho = -0.356$, $p = 0.01$) and inverse highly significant correlation was also detected between FEV1% and PLR ($\rho = -0.392$, $p = 0.004$) (Table, 2 and figure, 1).

Regression analysis indicates that CRP and NLR were statistically significant predictors for FEV% level ($B = -1.64$, $p = 0.001$) and ($B = -11.95$, $p = 0.003$) respectively. On the other hand, MPV was significant positive predictor for FEV% level ($B = 8.344$, $p = 0.009$) (Table, 3 and figure, 2).

Table (1): Sociodemographic characteristics, Laboratory findings and Pulmonary function tests of cases (N=51).

Variable	Statistics (N = 51)	
Age (years)	Mean \pmSD (range) 60.41 ± 9.6 (40-78)	
Sex	Male (n, %)	51 (100%)
	Female (n, %)	0 (0%)
BMI (add measuring unit)	Mean \pmSD (range) 31.02 ± 6.8 (22-47)	
Laboratory findings and Pulmonary function tests	Median (Q1, Q3)	
CRP	6.2 (3, 10)	
WBC	7.6 (5.9, 9)	
PLT	206 (170, 283)	
MPV	8.7 (7.8, 9.4)	
PLR	106.8 (84.4, 146.7)	
NLR	1.8 (1.19, 2.4)	
FEV1%	67 (56, -98)	

BMI: body mass index, CRP: C - reactive protein, WBC: white blood cell counts, PLT: platelets, MPV: mean platelet volume, PLR: platelet to lymphocyte ratio, NLR: neutrophil to lymphocyte ratio, FEV1: forced expiratory volume in 1st second

Table (2): Spearman Correlation between FEV1% and other parameters (N=51).

Variable	Correlation coefficient (ρ)	p-value
MPV	0.332*	0.017
CRP	- 0.599**	0.000
NLR	-0.356*	0.01
PLR	-0.392**	0.004

MPV: mean platelet volume, CRP: C - reactive protein, NLR: neutrophil to lymphocyte ratio, PLR: platelet to lymphocyte ratio

***Correlation is significant at the 0.05 level (2-tailed). **Correlation is significant at the 0.01 level (2-tailed).**

Table (3): linear regression for predictors associated with FEV1%.

Variable	B	(95% CI)	P
CRP	-1.644	(- 2.56 – (-0.756))	0.001
MPV	8.344	(2.194 – 14.493)	0.009
NLR	-11.95	(-19.679 – (-4.221))	0.003
PLR	-0.50	(- 0.178 – 0.077)	0.429

CRP: C - reactive protein, MPV: mean platelet volume, NLR: neutrophil to lymphocyte ratio, PLR: platelet to lymphocyte ratio

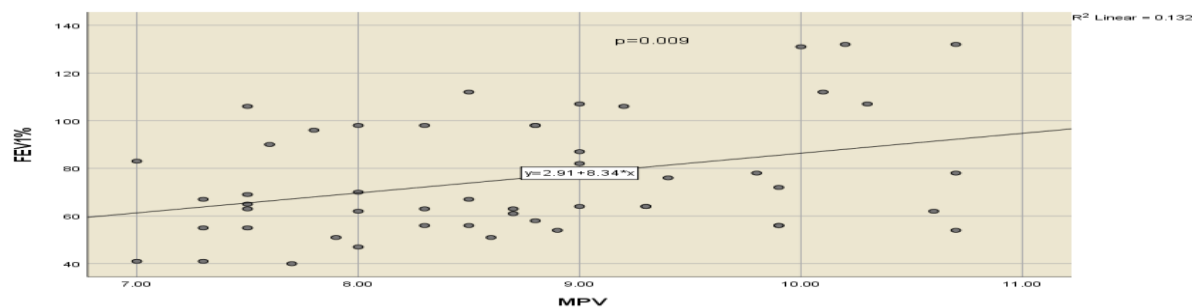


Figure (1): Significant positive correlation between MPV and for FEV1% level.

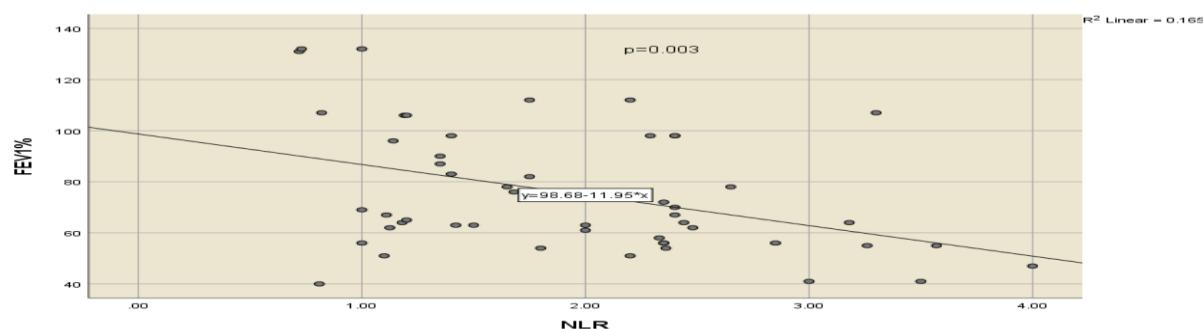


Figure (2): Significant inverse correlation between NLR and for FEV1% level.

Discussion

Increased inflammation in the lungs is associated with raised inflammatory markers in circulation leading to an increase in systemic inflammation as well [4]. Several biomarkers of inflammation are found to be raised in COPD including; C - reactive protein (CRP), Interleukin-8 (IL-8), Tumour Necrosis Factor (TNF- α), leptin, endothelin-1, fibrinogen, IL-6, Leukotriene (LT) B4 and E4 [7]. Many inflammatory indicators such as SII, NLR, PLR, PPN and LMR, can be used to estimate the severity of systemic inflammatory state, reflecting disease ranking and progression. Systemic

inflammation is linked with several diseases, including depression, hypertension, acute ischemic stroke [8], and rheumatoid arthritis. Furthermore, systemic inflammatory markers may also be closely associated with the pathogenesis of COPD. Increases in the NLR and PLR were noted in COPD patients, particularly during exacerbation, which are also linked to the negative outcomes of the disease [9]. Mean platelet volume (MPV) is increased in patients at risk for athero-thrombotic disease, and measurement of PLT volume has been suggested as an indicator of PLT activation [10]. MPV has been investigated

as an indicator of inflammation in different diseases ^[11], and MPV has also been noted to be increased in cardiovascular disease, peripheral artery disease, and cerebrovascular disease ^[12]. Recent studies have found that an increased MPV was a predictor of unprovoked venous thromboembolism and arterial thrombosis ^[13]. Other studies show that chronic obstructive airway disease patients have altered PLT functions and clotting system activation. In particular, it has been shown that there is a shortened PLT half-life, increased PLT size, increased PLT aggregation, high levels of blood fibrinogen, and in vitro and in-vivo PLT activation in these patients ^[14]. MPV is one of the PLT function indices. It reflects the PLT production rate and stimulation ^[15]. MPV is a parameter generated by routine complete blood count (CBC) test that is usually overlooked by clinicians ^[16].

The current study, showed a significant positive correlation between FEV% and MPV. Also, there was an inverse highly significant correlation between FEV% and CRP. A significant inverse correlation was detected between FEV1% and NLR and an inverse highly significant correlation was also detected between FEV1% and PLR. Regression analysis indicates that CRP and NLR were statistically the most significant predictors for FEV%. On the other hand, MPV still has a significant positive predictive value for FEV% level. In agree with our result, **Forsheh et al.**, showed that COPD patients had a significantly lower MPV than the non-COPD group. They found that, only MPV among the platelet indices was significantly associated with the disease severity and that MPV significantly decreased with increasing severity of COPD in the GOLD

classification, but no such difference was observed in the CAT classification ^[17]. On the other hand, **Hlapčić et al.**, found that MPV and PDW were lower in the COPD group ^[5].

Onder et al., reported that MPV was significantly increased in patients with COPD compared with healthy controls (18). **Bansal et al.**, noted the impact of decreased oxygen tension on the platelets in COPD patients and they concluded that MPV was significantly raised in COPD patients with hypoxia compared with non-hypoxic subjects and controls ^[19].

Ulasli et al., demonstrated that MPV is decreased during exacerbation period of COPD, while it is increased during the stable period [20]. **Wang et al.**, confined those patients with COPD have a lower MPV during acute exacerbation ^[21]. **Karadeniz et al.**, reported that MPV increased once patient has recovered from exacerbation ^[22].

Neutrophil to lymphocyte ratio (N/L) is an established marker of inflammation and has been shown to increase in several inflammatory conditions ^[23]. A positive linear correlation was found between NLR and COPD severity in the stable group ^[24]. Some authors found a moderate correlation between NLR and FEV1, mMRC, and BODE index. These findings suggest that NLR could be used as a clinical performance marker in COPD patients ^[25]. In the same estimated that $NLR \geq 2.7$ (determined by ROC analysis) could predict moderate/severe AECOPD over 3 years ^[26]. NLR was significantly higher in stable COPD patients, when compared to controls and even higher in AECOPD

when compared to stable COPD and controls ^[27].

Gonzalez et al., found that NLR correlates with clinical and functional parameters in patients with COPD and they concluded that it could be used as a putative marker associated with COPD severity and relevant mortality ^[28]. **Uslu and Gülşen** concluded that NLR could reflect the severity of COPD, and can be used as a potential useful marker ^[29]. Our results revealed that NLR and PLR are promising markers in predicting COPD severity and future acute exacerbation ^[30].

CRP could be an important marker in determining COPD prognosis, and raised levels are in both acute exacerbations and chronic, stable disease, implying a continuing low-level, inflammatory activity. Elevated levels could also be associated with decreased exercise tolerance due to disordered energy metabolism, increased cardiac disease, and all-cause mortality ^[31]. Also, **Aksu et al.**, found that CRP levels are raised in stable COPD patients independent of smoking behaviour and history of biomass exposure. They also demonstrated that higher CRP levels were related to low FEV1% predicted, SpO2 and 6MWD and to high m-MRC levels ^[32].

Conclusion

MPV could be used in addition to other markers of inflammation like CRP, N/L ratio and P/L as good predictors of COPD severity. Limitations of the study; MPV was not studied in COPD exacerbations; small size of the study population and PDW was not studied.

No conflict of interest

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