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ORIGINAL ARTICLE

Interrelationship of Platelet Indices, Pulmonary Artery Diameter, and Severity of COPD and Pulmonary Hypertension

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ABSTRACT

Objective: The study aimed to evaluate how platelet indices and main pulmonary artery diameter correlate with the severity of chronic obstructive pulmonary disease and pulmonary hypertension.

Methodology: The methodology consisted of a cross-sectional analytical study that included 180 adults with stable COPD. Airflow limitation was graded by post-bronchodilator FEV1 percentage predicted (GOLD 1, 4). Main pulmonary artery diameter was measured on axial chest CT at the level of the pulmonary artery bifurcation; pulmonary artery systolic pressure (PASP) was estimated by transthoracic echocardiography. Platelet count, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) were recorded. The association with pulmonary hypertension, defined in this study as an estimated pulmonary artery systolic pressure (PASP) of ≥35 mmHg, was assessed using correlation analysis and multivariable logistic regression.

Result: Participants had a mean age of 65.1 ± 7.8 years. Among 180 participants, pulmonary hypertension was identified in 115 (63.9%). Participants with pulmonary hypertension had a greater mean pulmonary artery diameter than those without pulmonary hypertension (31.24 ± 2.57 mm vs. 27.08 ± 2.13 mm, *p* <.001). The pulmonary-hypertension group also exhibited higher values for MPV (8.67 ± 0.78 vs 7.74 ± 0.69 fL, p<.001), PDW, platelet count, and PCT. Pulmonary artery diameter showed a positive correlation with PASP (r=.694, p<.001) and an inverse correlation with FEV1% predicted (r=−.710, p<.001). MPV correlated positively with PASP (r=.625, p<.001) and inversely with FEV1% predicted (r=−.575, p<.001). Multivariable analysis identified higher MPV, larger pulmonary artery diameter, and lower FEV1% predicted as independent predictors of pulmonary hypertension.

Conclusion: Increased severity of COPD was associated with the presence of enlarged pulmonary arteries, high levels of pulmonary pressure estimation, and changes in platelet parameters. MPV and pulmonary artery size were the parameters that showed the most significant relationship to pulmonary hypertension and can be used as additional parameters for the evaluation of pulmonary vessel pathology in COPD. The evaluation of routine platelet parameters, CT pulmonary artery size, severity of COPD, and pulmonary pressure estimation is a practical multimodal approach to assessing pulmonary risk..

Keywords: COPD; pulmonary hypertension; pulmonary artery diameter; mean platelet volume; platelet distribution width; plateletcrit; FEV1.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous respiratory disorder characterized by persistent respiratory symptoms and airflow limitation. Current GOLD guidance recommends spirometric confirmation of diagnosis alongside systematic assessment of airflow obstruction severity, symptom burden, exacerbation history, and multimorbidity. Pulmonary vascular disease is a significant extrapulmonary and cardiopulmonary complication, especially in patients with advanced airflow limitation.[1,3,4]

Pulmonary hypertension (PH) in COPD carries significant clinical importance, as elevated pulmonary vascular pressure increases right-ventricular afterload, can restrict exercise capacity, and is linked to poor outcomes. Right-heart catheterization is the reference standard for definitive hemodynamic diagnosis, though noninvasive tools are widely used to identify patients who warrant further evaluation. Echocardiography estimates pulmonary artery systolic pressure, whereas chest computed tomography (CT) displays structural details of pulmonary vascular enlargement.[3,4,6,7]

Main pulmonary artery (PA) diameter and the ratio of PA diameter to ascending aortic diameter have been studied as imaging markers of pulmonary vascular disease in COPD. Earlier research has shown that pulmonary artery enlargement correlates with invasive pulmonary pressures and can flag patients facing higher risk of exacerbation or adverse outcomes. A meta-analysis demonstrated clinically valuable diagnostic performance for the CT-derived PA:A ratio in COPD-associated pulmonary hypertension; but, this measure cannot replace definitive hemodynamic assessment.[2–7]

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COPD is associated with systemic inflammation, endothelial dysfunction, and platelet activation. MPV, PDW, platelet count, and PCT, all obtained through a complete blood count test, have been explored as economical biomarkers. The relationship between MPV and platelets in COPD and pulmonary hypertension still remains inconsistent, as although some studies show abnormal or increased results, systematic studies have shown significant heterogeneity within diseases, instruments, and environments.[8–16]

Novelty of the Study: Previous literature on the issue mostly explored parallel research avenues with CT measurement of the pulmonary arteries serving as a proxy marker for pulmonary vascular disease and pulmonary hypertension, while platelet indices were considered mostly in relation to the availability of inexpensive surrogate markers of systemic inflammation, platelet activation, COPD severity, or pulmonary hypertension.[3–7,8–16] The novelty of the current study lies in the analysis of both these aspects in one cohort combining hematological parameters, structural parameter (main pulmonary artery diameter), physiological parameter (post-bronchodilator FEV1% predicted), and hemodynamic parameter (PASP measured via echocardiography). It allows the researcher to explore whether hematological parameters add value to imaging and lung function tests that can be done in a routine clinical practice to diagnose pulmonary vascular disease without substituting any other parameter.

LITERATURE REVIEW

The activation of platelets is a biologically plausible mechanism in COPD patients as it could be affected by chronic hypoxemia, systemic inflammation, oxidative stress, and damage to the

endothelium. A meta-analysis of 27 studies has shown that stable COPD patients have elevated platelet count and platelet-to-lymphocyte ratio compared to non-COPD control subjects, although MPV and PDW were not consistently associated with COPD.[13] There are also studies which have confirmed that the presence of platelet activation in blood samples is associated with respiratory symptoms and clinical features of COPD.[15]

Independent research has demonstrated that MPV is related to pulmonary vascular complications or clinical outcomes. In a multicenter cross-sectional analysis of patients with stable COPD, MPV was found to be elevated among patients with pulmonary hypertension, with increasing MPV observed with worsening of PH.[11] Another independent study found a positive association between platelet indices and pulmonary heart disease.[14] Research involving patients with exacerbated COPD has also involved the examination of MPV regarding clinical outcome, although results were inconsistent.[16] Not one platelet index appears to work consistently in any population of COPD patients, making a case for studying these parameters jointly with other physiologic and imaging parameters.[9,12-16]

CT-derived pulmonary artery measurements bear a stronger and more consistent association with pulmonary vascular pressure. In severe COPD, the pulmonary artery-to-aorta (PA:A) ratio has demonstrated a correlation with mean pulmonary artery pressure as determined by right-heart catheterization. Pulmonary artery diameters are larger among COPD patients with pulmonary hypertension. Systematic review evidence supports the PA:A ratio as a useful screening marker rather than a standalone diagnostic test.[3–7]

Pulmonary artery enlargement does not depend solely on pulmonary pressure. Hyperinflation, altered lung mechanics, hypoxic vasoconstriction, vascular remodeling, left-heart disease, age, and body size can influence vascular dimensions. Similarly, smoking, infection, corticosteroid therapy, cardiovascular disease, renal disease, and hematologic conditions can also affect platelet indices. Researchers investigating this interrelationship must set up rigorous exclusion criteria and apply multivariable adjustment.[9,12,13]

In addition to spirometry and echocardiogram parameters for assessing pulmonary pressures, adding a blood marker in conjunction with a common imaging test could potentially help pinpoint a subgroup of COPD patients who have a greater likelihood of developing pulmonary vascular disease. Both pulmonary artery measurements on CT and platelet parameters have individually been explored, but their combination in conjunction with spirometry and echocardiography of pulmonary pressures has not been studied extensively.[3-7,11,13-15] The current comprehensive method thus emphasizes complementation rather than replacement: the role of platelet parameters is in systemic inflammation and thrombosis, while that of pulmonary artery dilatation in structural changes.

Objectives

- The study aimed to determine the relationship between platelet indices, platelet count, mean platelet volume, platelet distribution width, and plateletcrit, and COPD severity.
- The study aimed to evaluate the relationship between main pulmonary artery diameter and COPD severity.
- Platelet indices and pulmonary artery diameter were assessed for their associations with pulmonary hypertension.
- This study identifies independent predictors of pulmonary hypertension by evaluating clinical, spirometric, hematologic, and imaging variables.

METHODOLOGY

Study Design and Setting: A cross-sectional analytical study was conducted at Kamal Surgical Complex from January 2023 to September 2023. This study evaluated the relationship among platelet indices, main pulmonary artery diameter, COPD severity, and pulmonary hypertension in adults with stable COPD.

Study Population: The study enrolled 180 adults aged 45 years or older who had spirometrically confirmed COPD. Participants represented all GOLD spirometric grades, spanning from mild to very severe airflow obstruction. Patients were enrolled during periods of clinical stability.

Inclusion and Exclusion Criteria: Participants were eligible if they were at least 45 years old, exhibited post-bronchodilator airflow obstruction consistent with COPD, and possessed complete blood count, chest CT, spirometry, and transthoracic echocardiography data collected within a clinically relevant interval. Exclusion criteria encompassed acute exacerbation or infection, diagnosed hematologic disease, active malignancy, chronic thromboembolic disease, interstitial lung disease, severe left-sided valvular disease, recent major surgery. The use of pharmacological agents with a documented substantial impact on platelet indices.

Assessment of COPD Severity: Airflow limitation was classified into four GOLD grades based on post-bronchodilator FEV1 percentage predicted: GOLD 1 (≥80%), GOLD 2 (50, 79%), GOLD 3 (30, 49%), and GOLD 4 (<30%). FEV1% predicted was also analyzed as a continuous variable. [1]

Platelet Indices: Platelet count, MPV, PDW, and PCT were analysed as standard complete-blood-count parameters reported in routine clinical practice. In clinical practice, MPV measurements require analysis on a single hematology analyzer within a standardized post-venipuncture interval, as values can fluctuate with anticoagulant exposure and processing delays.[9,12,13]

Pulmonary Artery Diameter: The main pulmonary artery diameter was measured on an axial CT image perpendicular to the vessel's long axis, taken at the level of the pulmonary artery bifurcation. Researchers recorded measurements in millimetres using consistent anatomical landmarks for every participant.[3,5–7]

Assessment of Pulmonary Hypertension: Pulmonary pressure was represented by echocardiographically estimated PASP. To classify participants as having pulmonary hypertension in the study analysis, a pulmonary artery systolic pressure (PASP) of ≥35 mmHg was used as the criterion. The same threshold was applied to all cohort members to enable statistical comparison.[3,6,7]

Statistical Analysis: We performed statistical analysis with IBM SPSS Statistics version 27.0 (IBM Corp. Armonk, NY, USA). Continuous variables are summarized as mean ± standard deviation, while categorical variables are reported as frequency and percentage. Between-group differences were evaluated with independent-samples t tests or one-way ANOVA, depending on the number of groups compared. The relationships among platelet indices, pulmonary artery diameter, PASP, and FEV1% predicted were assessed using Pearson correlation coefficients. A multivariable logistic regression analysis was conducted to identify factors independently associated with pulmonary hypertension. Statistical significance was set at $p < .05$.

RESULTS

One hundred and eighty participants were included in the study. The mean age was 65.1 years (SD = 7.8), and male participants comprised 69.4% of the sample. The mean post-bronchodilator FEV1 measured 49.1 ± 21.3% of the predicted value. Based on GOLD spirometric grading, 25 participants (13.9%) were classified as grade 1, 55 (30.6%) as grade 2, 60 (33.3%) as grade 3. 40 (22.2%) as grade 4. Pulmonary hypertension, defined by the study's PASP threshold, was identified in 115 participants (63.9%).

Progressive airflow limitation was associated with an enlarged pulmonary artery diameter and elevated pulmonary arterial systolic pressure (PASP). Across GOLD grades, mean MPV, PDW, platelet count, and PCT showed a progressive increase, with peak values observed in GOLD 4. As spirometric severity worsened, a greater proportion of participants met the pulmonary-hypertension threshold.

The main pulmonary artery diameter showed the strongest bivariate association with PASP ($r=0.694$, $p<.001$). The pulmonary

artery diameter showed a strong inverse correlation with FEV1% predicted ($r = -0.710$, $p < 0.001$). MPV showed a moderate positive correlation with PASP ($r=0.625$, $p<.001$) and a moderate inverse correlation with FEV1% predicted ($r=-0.575$, $p<.001$).

With correction for FEV1 and age, increased MPV and pulmonary artery diameter were independently related to pulmonary hypertension. Increased FEV1% predicted was independently related to a lower likelihood of pulmonary hypertension; however, age was not statistically significant in this model. The most important aspect of this analysis is that an everyday blood test measure and a CT marker remained relevant in the same multivariable analysis.

Table 1. Baseline characteristics of the study population

Characteristic	Value
Age, years	65.1 ± 7.8
Male sex, n (%)	125 (69.4)
Smoking exposure, pack-years	41.6 ± 18.1
FEV1, % predicted	49.1 ± 21.3
SpO2, %	91.8 ± 2.3
Main PA diameter, mm	29.74 ± 3.15
Estimated PASP, mmHg	37.4 ± 9.9
Platelet count, ×10⁹/L	294.7 ± 54.7
MPV, fL	8.34 ± 0.83
PDW, fL	12.08 ± 1.44
PCT, %	0.247 ± 0.062
Pulmonary hypertension, n (%)	115 (63.9)

Table 2. Selected variables according to GOLD spirometric grade

Grade	n	FEV1 %	PA diameter mm	PASP mmHg	MPV fL	PDW fL	PCT %	PH %
GOLD 1	25	86.2	25.92	23.1	7.55	10.92	0.205	0.0
GOLD 2	55	62.0	28.22	32.9	8.04	11.83	0.223	41.8
GOLD 3	60	38.4	31.06	41.6	8.48	12.34	0.256	86.7
GOLD 4	40	24.2	32.23	46.3	9.01	12.78	0.292	100.0

Table 3. Comparison according to pulmonary-hypertension status

Variable	PH present (n=115)	PH absent (n=65)	p value
FEV1, % predicted	37.5 ± 14.1	69.7 ± 15.7	<.001
PA diameter, mm	31.24 ± 2.42	27.08 ± 2.49	<.001
PASP, mmHg	43.6 ± 5.8	26.4 ± 4.6	<.001
Platelet count, ×10⁹/L	301.2 ± 57.5	283.2 ± 47.7	0.025
MPV, fL	8.67 ± 0.73	7.74 ± 0.64	<.001
PDW, fL	12.53 ± 1.30	11.29 ± 1.34	<.001
PCT, %	0.263 ± 0.063	0.219 ± 0.051	<.001
SpO2, %	90.8 ± 2.0	93.5 ± 1.6	<.001

Table 4. Correlations with estimated PASP and FEV1% predicted

Variable	r with PASP	p	r with FEV1	p
MPV	0.625	<.001	-0.575	<.001
PDW	0.465	<.001	-0.407	<.001
Platelet count	0.192	0.010	-0.311	<.001
PCT	0.400	<.001	-0.488	<.001
PA diameter	0.694	<.001	-0.710	<.001

Table 5. Multivariable logistic regression for pulmonary hypertension

Predictor	Adjusted OR	95% CI	p value
MPV (per 1 fL)	3.84	1.61–9.20	0.002
PA diameter (per 1 mm)	1.40	1.10–1.79	0.007
FEV1 (per 1% predicted)	0.92	0.89–0.96	<.001
Age (per year)	1.00	0.93–1.07	0.891

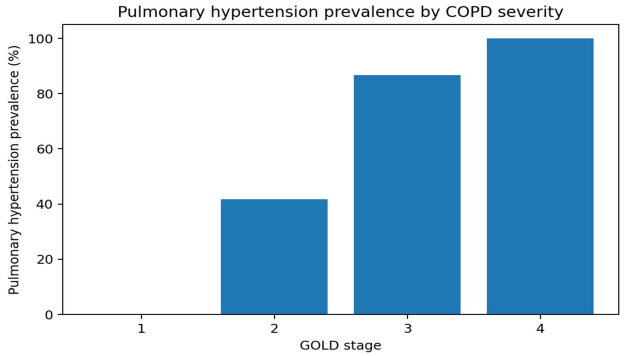


Figure 1. Pulmonary hypertension prevalence by GOLD grade.

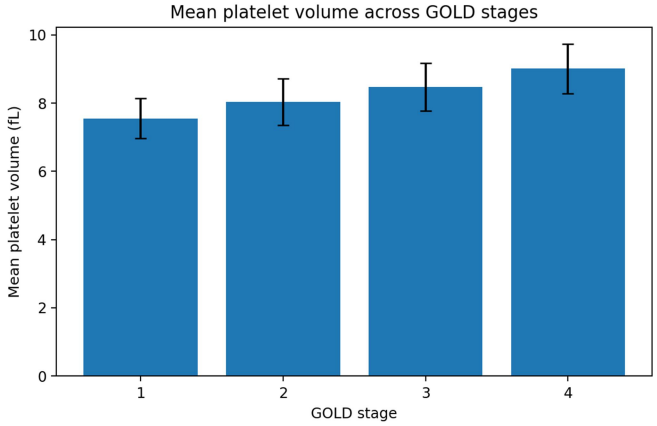


Figure 2. Mean platelet volume across GOLD grades (mean ± SD).

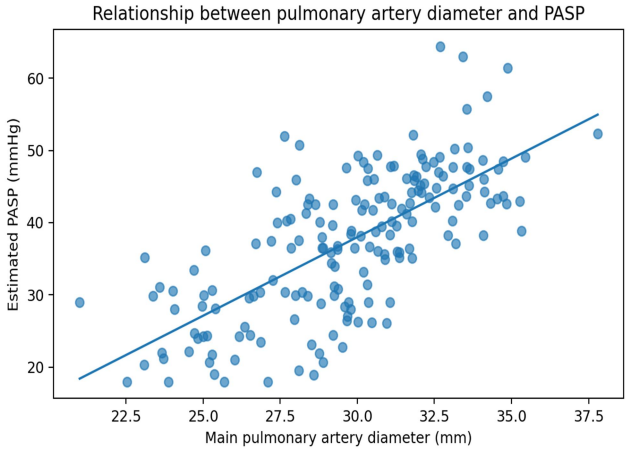


Figure 3. Relationship between main pulmonary artery diameter and estimated PASP.

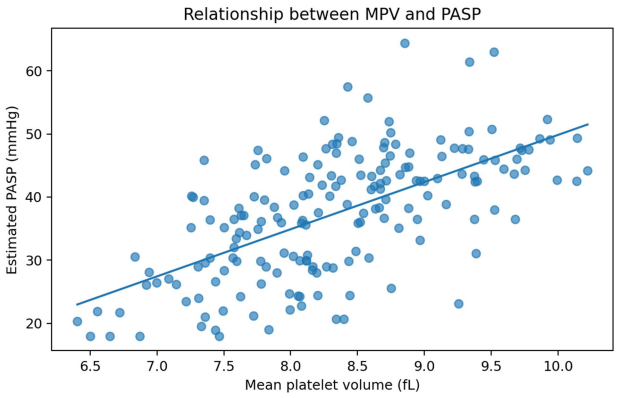


Figure 4. Relationship between mean platelet volume and estimated PASP.

DISCUSSION

There was an observed relationship between the severity of airflow obstruction, enlargement of the pulmonary arteries, increased pulmonary pressure, and platelet indices. The major pulmonary artery diameter was found to have a strong association with the PASP and a reverse correlation with FEV1% predicted. The MPV also showed correlation with PASP. Moreover, the MPV had an independent association with pulmonary hypertension on multivariate analysis. The primary importance of this study is the combined evaluation of routinely obtainable hematological, imaging, spirometric, and echocardiographic parameters in the same group of patients with COPD.

The pulmonary artery findings align with the direction established in previously published COPD research. Wells et al. Reported that the CT-derived PA:A ratio correlates with invasive mean pulmonary artery pressure in severe COPD, showing superior diagnostic accuracy compared to echocardiographic PASP within their selected cohort. Studies of advanced COPD have likewise identified enlarged pulmonary arteries and elevated PA:A ratios in patients with pulmonary hypertension. Subsequent systematic review and meta-analysis of the available evidence concluded that the CT-based pulmonary artery to aorta (PA:A) ratio is a valid screening tool for pulmonary hypertension associated with COPD.[3,4,6,7]

The progressive widening of pulmonary artery diameter across GOLD grades indicates that pulmonary vascular involvement intensifies with advancing COPD severity. Chronic hypoxic vasoconstriction, loss of the pulmonary vascular bed, inflammation, and endothelial dysfunction drive pulmonary vascular remodeling in COPD. Emphysema-related hyperinflation, left-heart disease, body size, and measurement technique can all influence CT vascular measurements. Interpretation of pulmonary artery diameter requires integration with clinical, physiological, and echocardiographic findings.[2–7]

The observed platelet data support some parts of the literature. MPV has been explored as an indicator of platelet activation, and one cross-sectional multicenter study found elevated MPV levels in COPD patients with stable condition and pulmonary hypertension.[11] There were other studies exploring altered platelet indices in COPD and pulmonary heart disease, and the results of systematic review revealed greater consistency of platelet count and platelet-to-lymphocyte ratio in comparison with MPV and PDW levels.[9,12–14] Research on platelet activation and the course of exacerbations provides additional proof for the existence of biological relationship between platelet activation and COPD phenotype.[15,16] This variability underlies the reason to interpret platelet indices as additional markers, not as diagnostic tools.

Disagreement among studies of platelet indices may stem from several factors. MPV is influenced by multiple pre-analytical factors, including sample handling, the type of anticoagulant used, and the timing of measurement, as well as clinical variables such as smoking status, medication exposure, active infection, cardiovascular comorbidity, and acute exacerbation status. COPD populations differ in age, hypoxemia, disease severity, and how often vascular comorbidities occur. These factors limit the use of a single universal platelet-index cut-off.[9,12,13]

The hematological and radiographic markers analyzed here seem to complement each other. Pulmonary artery dilation is an indicator of remodeling of the pulmonary vasculature or increased pressure within it. On the other hand, MPV and platelet indices may represent systemic inflammation and thrombosis. What makes the current paradigm valuable from a clinical perspective is that these commonly available markers are being correlated with FEV1% predicted and PASP in a single study. This multi-parametric correlation might be especially valuable in diagnosing COPD patients whose blood, CT, and spirometry findings suggest pulmonary vascular disease..

Because the design is cross-sectional, the observed associations do not establish temporal sequence and cannot support causal inference. Future prospective studies employing standardized imaging protocols, thorough evaluation of hypoxemia and comorbidities, and right-heart catheterization when clinically warranted would help substantiate the strength and clinical relevance of these associations.

Strengths and Limitations: A notable strength of this study is its integrated assessment of four complementary domains within a single COPD cohort: spirometric disease severity, CT-derived pulmonary artery diameter, echocardiographic pressure estimates, and routine platelet indices. Much of the available literature has evaluated these domains separately.[3–16] By placing them in the same analytical framework, the present study enables direct comparison of structural vascular change, pulmonary physiology, estimated hemodynamic burden, and systemic platelet-related changes. The use of routinely available measurements also enhances the practical relevance of the model for clinical settings where advanced pulmonary vascular testing may not be immediately available.

The study is not without limitations. The cross-sectional design precludes causal inference, and echocardiographic PASP cannot replace right-heart catheterization. Residual confounding from smoking exposure, hypoxemia, medication use, exacerbation history, cardiovascular comorbidity, and other inflammatory conditions may also have affected platelet and pulmonary vascular measurements.

CONCLUSION

As COPD severity increased, pulmonary artery systolic pressure rose, the main pulmonary artery diameter enlarged, and platelet indices showed progressive changes. Pulmonary artery diameter and mean platelet volume (MPV) showed especially strong associations with pulmonary hypertension, whereas a lower FEV1% predicted was independently linked to a higher likelihood of pulmonary hypertension. The main novelty of this study is not any single marker, but the integration of routine platelet indices with CT pulmonary artery measurement, spirometric severity, and echocardiographic pressure within one analytical model. This combined approach may provide complementary information for pulmonary vascular risk assessment in COPD and warrants prospective validation against definitive hemodynamic measurements and clinical outcomes.

REFERENCES

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for Prevention, Diagnosis and Management of COPD: 2023 Report. GOLD; 2023.
2. Wells JM, Washko GR, Han MK, et al. Pulmonary arterial enlargement and acute exacerbations of COPD. N Engl J Med. 2012;367:913–921.
3. Iyer AS, Wells JM, Vishin S, Bhatt SP, Wille KM, Dransfield MT. CT scan-measured pulmonary artery to aorta ratio and echocardiography for detecting pulmonary hypertension in severe COPD. Chest. 2014;145(4):824–832. doi:10.1378/chest.13-1422.

4. Shin S, King CS, Brown AW, et al. Pulmonary artery size as a predictor of pulmonary hypertension and outcomes in patients with chronic obstructive pulmonary disease. Respir Med. 2014;108(11):1626–1632.
5. Chung KS, Kim YS, Kim SK, et al. Functional and prognostic implications of the main pulmonary artery diameter to aorta diameter ratio from chest computed tomography in Korean COPD patients. PLoS One. 2016;11(5):e0154584. doi:10.1371/journal.pone.0154584.
6. Gašparović K, Pavliša G, Hrabak Paar M, et al. Diagnostic accuracy, sensitivity, and specificity of CT pulmonary artery to aorta diameter ratio in screening for pulmonary hypertension in end-stage COPD patients. Croat Med J. 2021;62(5):446–454. doi:10.3325/cmj.2021.62.446. PMID:34730884.
7. Chen X, et al. Diagnostic value of computed tomography-based pulmonary artery to aorta ratio measurement in chronic obstructive pulmonary disease with pulmonary hypertension: a systematic review and meta-analysis. Clin Respir J. 2022;16(4):276–283. PMID:35289083.
8. Ulasli SS, Ozyurek BA, Yilmaz EB, Ulubay G. Mean platelet volume as an inflammatory marker in acute exacerbation of chronic obstructive pulmonary disease. Pol Arch Med Wewn. 2012;122(6):284–290.
9. Steiropoulos P, Papanas N, Nena E, et al. Mean platelet volume and platelet distribution width in patients with chronic obstructive pulmonary disease: the role of comorbidities. Angiology. 2013;64(7):535–539. PMID:23052724.
10. Agapakis DI, Massa EV, Hantzis I, et al. The role of mean platelet volume in chronic obstructive pulmonary disease exacerbation. Respir Care. 2016;61(1):44–49. PMID:26604328.
11. Mohamed MF, Ali A, Abbas A, et al. Mean platelet volume as a predictor of pulmonary hypertension in patients with stable COPD. Int J Chron Obstruct Pulmon Dis. 2019;14:1099–1108. doi:10.2147/COPD.S176413. PMID:31213790.
12. Hlapčić I, Somborac-Bačura A, Popović-Grle S, et al. Platelet indices in stable chronic obstructive pulmonary disease—association with inflammatory markers, comorbidities and therapy. Biochem Med (Zagreb). 2020;30(1):010701. doi:10.11613/BM.2020.010701. PMID:31839721.
13. Zinellu A, Paliogiannis P, Sotgiu E, Mellino S, Fois AG, Carru C, Mangoni AA. Platelet count and platelet indices in patients with stable and acute exacerbation of chronic obstructive pulmonary disease: a systematic review and meta-analysis. COPD. 2021;18(2):231-245. doi:10.1080/15412555.2021.1898578. PMID:33929925.
14. Bai Y, Tao XN. Mean platelet volume combined red cell distribution width as biomarker of chronic obstructive pulmonary disease with pulmonary heart disease. Clin Respir J. 2020;14(12):1122–1130. doi:10.1111/crj.13248. PMID:32772499.
15. Fawzy A, Putcha N, Raju S, et al. Urine and plasma markers of platelet activation and respiratory symptoms in COPD. Chronic Obstr Pulm Dis. 2023;10(1):22-32. doi:10.15326/jcopdf.2022.0326. PMID:36367951.
16. Ziaei M, Sabaghzade M, Galavi M, Abdolrazaghnejad A. Relationship of mean platelet volume (MPV) and mean corpuscular volume (MCV) with the outcome of patients with acute exacerbation of COPD. Tanaffos. 2023;22(1):136-142. PMID:37920314.

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