Journal of Basic Research in Medical Sciences

Journal HomePage : https://jbrms.medilam.ac.ir

Ilam University of Medical Sciences Publications

Frequency of Peripheral Blood Eosinophilia in Chronic Pulmonary Diseases with Obstructive Pattern in Spirometry

Bahareh Soltani [1], **Ayat Ahifar** [1]✉, **Reza Ghaderi** [1], **Vahan Moradians** [1]

[1] Department of internal medicine, Hazrat Rasoul Hospital, School of Medicine, Iran University of Medical Sciences, Tehran, Iran

Article Info

Article type:
Original Article

Article History:
Received: Oct. 01, 2025
Revised: Oct. 04, 2025
Accepted: Oct. 14, 2025
Published Online: Apr. 06, 2026

✉ ***Correspondence to:***
Ayat Ahifar
Department of internal medicine, Hazrat Rasoul Hospital, School of Medicine, Iran University of Medical Sciences, Tehran, Iran

Email:
a.ahifar@gmail.com

A B S T R A C T

Introduction: Eosinophils are inflammatory cells that perform diverse functions and play roles in both homeostasis and disease in various tissues. Evidence indicates that many patients with chronic respiratory conditions, including asthma and COPD, exhibit persistent eosinophilic airway inflammation. This study aimed to evaluate the prevalence of blood eosinophilia in patients with obstructive patterns on spirometry.

Materials & Methods: This cross-sectional study was conducted on patients attending the Hazrat Rasoul Akram Hospital, affiliated with Iran University of Medical Sciences. Patients who underwent spirometry and had peripheral blood tests were included in the study. Information regarding age, gender, body mass index, underlying diseases, smoking habits, medication use, spirometry results, and complete blood count (CBC) was collected and analyzed statistically.

Results: A total of 103 participants were selected based on the inclusion criteria and participated in the study. In the current study, 8 patients (7.8%) were eosinophilic-positive and 95 patients (92.2%) were eosinophilic-negative. The mean FEV1 in the eosinophilic-negative group was 63.25% (with a standard deviation of 20.585), while in the eosinophilic-positive group it was 60.00% (with a standard deviation of 14.203). Statistical tests confirmed no significant difference between the two groups, suggesting that reduced FEV1 was not associated with blood eosinophilia in this population. Additionally, the mean FEV1/FVC ratio in the eosinophilic-negative group was 80.72% (with a standard deviation of 17.10), and in the eosinophilic-positive group it was 77.33% (with a standard deviation of 15.62), which also showed no statistically significant difference.

Conclusion: This study found no significant association between blood eosinophilia and demographic or pulmonary function variables in patients with obstructive patterns on spirometry. Future studies with more precise designs and larger sample sizes may clarify the role of eosinophilia in prognosis and treatment response.

Keywords: Pulmonary Disease, Chronic Obstructive, Eosinophilia

➢ **Cite this paper**
Soltani B, Ahifar A, Ghaderi R, Moradians V. Frequency of Peripheral Blood Eosinophilia in Chronic Pulmonary Diseases with Obstructive Pattern in Spirometry. *J Bas Res Med Sci*. 2026; 13(2):40-47.

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

© Author(s) retain the copyright

Publisher: Ilam University of Medical Sciences

Introduction

Pulmonary function increases until roughly 15–20 years of age and begins a gradual decline from about 25 years onward (1). Inadequate attainment of peak lung function and an accelerated subsequent decline each contribute to reduced respiratory capacity later in life (2, 3). Cigarette smoking is the principal driver of this deterioration and frequently culminates in chronic obstructive pulmonary disease (COPD), which is characterized by persistent reductions in forced expiratory volume in one second (FEV1) and in the FEV1/forced vital capacity (FVC) ratio (4). A similar downward trajectory is observed in some individuals with asthma, where airway remodeling—marked by basement-membrane thickening, excess mucus production, smooth-muscle hyperplasia, and epithelial shedding—can result in fixed airflow obstruction. Although COPD is classically associated with neutrophilic inflammation, a subset of patients exhibits eosinophilic inflammation that differs mechanistically from the eosinophil-driven pathways typical of asthma (5).

Nevertheless, in both asthma and COPD, airway eosinophilia—identified by elevated sputum and/or blood eosinophil counts—affects a substantial proportion of patients and underlies responsiveness to anti-interleukin therapies (6, 7). Eosinophils, versatile immune effector cells, participate in both tissue homeostasis and disease. Observational data indicate that 20–40% of patients with stable COPD display eosinophilic airway inflammation, and sputum eosinophilia is present in 28% of acute exacerbations (8). Corresponding Chinese studies reported airway eosinophilia in 38% of acute COPD exacerbations and blood eosinophil counts (BEC) ≥ 2% in more than 40% of hospitalized patients (8).

Eosinophil recruitment to the airways is orchestrated by chemokines such as CCL11 and CCL5, together with cytokine signalling—particularly interleukin-5—which promotes eosinophil maturation and activation through epithelial and immune-cell interactions (9, 10). Increased eosinophil numbers in both central and peripheral airways have been demonstrated via biopsy, bronchoalveolar lavage, and induced-sputum analysis, alongside broader type-2 (T2) inflammation that may represent a therapeutic target in eosinophilic COPD (11, 12).

Several studies link high peripheral eosinophil levels to lower lung function in both general and asthmatic populations, yet findings on their influence on lung-function growth or decline remain inconsistent (13-15). Investigations into the relationship between BEC and exacerbation risk in COPD are similarly mixed: while some studies report higher exacerbation rates in patients with BEC ≥ 150 – 300 cells/μL (16-18), others—including the SPIROMICS, CHAIN, and BODE cohorts—find no significant association (19-21).

Emerging evidence suggests that temporal variability in eosinophil counts may predict clinical outcomes more accurately than single measurements. Patients with pronounced fluctuations experience more frequent exacerbations (21, 22). Moreover, persistently high eosinophil counts during hospitalization appear to signal an increased risk of future exacerbations, whereas persistently low counts may be linked to poorer survival (12). Whether eosinophilic inflammation contributes to lung-function decline solely in asthma or also in COPD remains uncertain. Accordingly, the present study aims to determine the prevalence of blood eosinophilia among patients with obstructive spirometric patterns.

Materials and methods

Study Design

This cross-sectional investigation was carried out in the pulmonary clinic of Hazrat Rasool Akram Hospital, Tehran, between April 2023 and April 2024.

Setting and Participants

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

Patients were eligible if they had completed both spirometry and peripheral blood analyses; those with incomplete records were excluded. A consecutive, non-probability sampling strategy was used.

Sample Size

Relying on the 52.8% prevalence of elevated eosinophilia reported by Mogensen et al., we calculated a required sample size of 84 patients (α = 0.05, power = 80%) and set a recruitment target of 90 to account for possible attrition.

Measurements & Validity and Reliability

Demographic and clinical information—including age, sex, body-mass index, comorbidities, smoking status, current medications, spirometric indices, and complete blood counts—was collected with a standardized checklist.

Statistical and Data Analysis

After obtaining ethics approval and institutional permission, medical charts were accessed by record number, and the data were entered into SPSS v26. Descriptive statistics (means, variances, standard deviations, and absolute and relative frequencies) were calculated. Continuous variables were compared using independent-samples t-tests or one-way ANOVA, while categorical variables were analyzed with the χ^2 test. A p-value < 0.05 was considered statistically significant.

Results

A total of 103 patients were included in the analysis, and their demographic and clinical profiles are presented in Table 1.

The cohort's mean age was 59.61 ± 13.59 years (range, 22–85 years). Women comprised 42 participants (40.8%), while men accounted for 61 (59.2%). With respect to smoking history, 47 individuals (45.6%) reported previous or current smoking, whereas 56 (54.4%) had never smoked.

Anthropometric assessment revealed a mean height of 165.29 ± 8.91 cm, an average weight of 74.26 ± 18.79 kg, and a mean body mass index (BMI) of 27.24 ± 6.59 kg/m². Pulmonary function testing showed a mean forced expiratory volume in one second (FEV_1) of 63.00 ± 20.13 % predicted, a mean forced vital capacity (FVC) of 77.72 ± 18.98 % predicted, and an average FEV_1/FVC ratio of 80.45 ± 16.94 %. Peripheral blood analysis yielded a mean eosinophil count of 176.67 ± 178.38 cells/μL and an average eosinophil percentage of 2.04 ± 1.89 %. Using the threshold of ≥500 cells/μL, eosinophilia was detected in 8 patients (7.8%), whereas 95 patients (92.2%) fell below this cutoff. A bronchodilator response of ≥12 % was observed in 35 participants (34.0%); the remaining 68 (66.0%) exhibited <12 % change, with a mean post-bronchodilator improvement of 11.77 ± 12.72 % (range, 0–75 %) (Table 1).

Table 2 summarizes eosinophilia distribution by sex and smoking status. Of the eosinophilic subgroup, 75 % were male and 25 % were female, a difference that did not reach statistical significance (p = 0.344). Similarly, 62.5 % of eosinophilic patients were smokers compared with 44.2 % in the non-eosinophilic group, again with no significant association (p = 0.319).

Comparative analyses of demographic variables, anthropometric measures, and spirometric indices between eosinophilic and non-eosinophilic patients are detailed in Table 3. No significant differences emerged for age (p = 0.695), height (p = 0.399), weight (p = 0.317), BMI (p = 0.643), FEV_1 (p = 0.565), FVC (p = 0.954), or FEV_1/FVC ratio (p = 0.574).

Table 1. Demographic, Anthropometric, Spirometric, and Eosinophil Characteristics (N = 103).

Variable	Minimum	Maximum	Mean ± SD
Age (years)	22	85	59.61 ± 13.59
Height (cm)	144	186	165.29 ± 8.91

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

Weight (kg)	44	144	74.26 ± 18.79
BMI (kg/m²)	15.19	43.36	27.24 ± 6.59
FEV1 (%)	19	99	63.00 ± 20.13
FVC (%)	32	116	77.72 ± 18.98
FEV1/FVC (%)	35.00	134.38	80.45 ± 16.94
Eosinophil count (cells/μL)	6	920	176.67 ± 178.38
Eosinophil percentage (%)	0.05	8.60	2.04 ± 1.89
Eosinophilia ≥500 cells/μL (%)	-	-	8 (7.8%) patients
Bronchodilator response ≥12% (%)	-	-	35 (34.0%) patients
% Change post-bronchodilator (mean ± SD)	0	75	11.77 ± 12.72

Table 2. Eosinophilia Distribution by Sex and Smoking Status.

Variable		Eosinophilia Negative (<500/μL)	Eosinophilia Positive (≥500/μL)	Total	p-value
Sex	Female	40 (42.1%)	2 (25.0%)	42	0.344
	Male	55 (57.9%)	6 (75.0%)	61	
Smoking	Negative	53 (55.8%)	3 (37.5%)	56	0.319
	Positive	42 (44.2%)	5 (62.5%)	47	

Table 3. Comparison of Demographics and Spirometry Indices Between Eosinophilic and Non-Eosinophilic Groups.

Variable	Negative (<500/μL) (n=95)	Positive (≥500/μL) (n=8)	p-value
Age (years)	59.80 ± 13.42	57.38 ± 16.41	0.695
Height (cm)	164.98 ± 8.56	169.00 ± 12.47	0.399
Weight (kg)	73.78 ± 19.02	80.00 ± 15.63	0.317
BMI (kg/m²)	27.16 ± 6.69	28.16 ± 5.56	0.643
FEV1 (%)	63.25 ± 20.59	60.00 ± 14.20	0.565
FVC (%)	77.69 ± 19.43	78.00 ± 13.42	0.954
FEV1/FVC (%)	80.72 ± 17.10	77.33 ± 15.62	0.574

Discussion

Eosinophils are pivotal inflammatory mediators in obstructive airway diseases such as asthma and COPD. While their contribution to asthma pathogenesis is well documented, the clinical relevance of peripheral blood eosinophilia in COPD remains contentious. This study quantified blood eosinophilia in patients with obstructive spirometric patterns and examined its relationship with demographic characteristics, smoking status, and lung function, with the goal of identifying patient subgroups that might benefit from personalized anti-inflammatory therapies. Using a threshold of ≥500 cells/μL, eosinophilia was present in 7.8% of the cohort (8/103), markedly lower than reported by studies applying a ≥300 cells/μL cutoff—e.g., the ECLIPSE cohort (23) found 37% and the SPIROMICS study (19) 28%.

Adopting the higher ≥500 cells/μL cutoff excluded individuals with moderate eosinophilia (300–500 cells/μL), yet Bafadhel et al. (24) have shown that such levels still predict responses to biologic therapy. Beyond threshold selection, disparities in population characteristics (e.g., ethnicity and geography) and study design (cross-sectional vs. longitudinal) further explain divergent findings. The

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

ECLIPSE study (23), for example, tracked eosinophil stability over time and linked it to exacerbation risk, whereas our analysis was limited to a single time point. Additional confounders—most notably systemic corticosteroid use (34% of patients)—may also have suppressed peripheral eosinophil counts. By endorsing a ≥300 cells/μL threshold, the GOLD 2023 guidelines (25) should improve cross-study comparability and aid in identifying candidates for targeted biologics. The low prevalence observed here highlights the need for standardized definitions, longitudinal monitoring, and consideration of environmental exposures such as air pollution in future investigations.

We also explored associations between blood eosinophilia (≥500 cells/μL) and demographic factors—age, sex, smoking status, and body mass index (BMI)—and found no statistically significant relationships. Mean age was 57.38 years in the eosinophilia-positive group versus 59.80 years in the negative group, mirroring findings by Casanova et al. (20). Although a higher proportion of men appeared in the positive group (75% vs. 57.9%), the difference was not significant, aligning with Singh et al. (21) and contrasting with Pignatti et al. (6). Similarly, a history of smoking was present in 62.5% of eosinophilia-positive patients without reaching significance, contrary to data from Brusselle et al. (26). BMI did not differ significantly between groups, consistent with Dixon et al. (27). The stringent eosinophil definition, limited eosinophilia-positive sample (n = 8), and unaccounted confounders (e.g., medications, comorbidities) likely attenuated detectable associations.

Pulmonary-function results mirrored these demographic findings. Mean FEV_1 values were 60.00% in eosinophilia-positive patients and 63.25% in eosinophilia-negative patients (P = 0.565), while FEV_1 /FVC ratios averaged 77.33% and 80.72%, respectively (P = 0.574). These observations diverge from studies reporting worse lung function with elevated eosinophils—e.g., Mogensen et al. (4) noted greater FEV_1 decline in asthma patients. In COPD, however, neutrophils and macrophages dominate airway inflammation, and eosinophils play a lesser role (28, 29), possibly explaining the lack of association here. The strict ≥500 cells/μL threshold and small eosinophilia-positive group reduced statistical power. Pascoe et al. (30) demonstrated that even ≥300 cells/μL can predict anti-IL-5 responsiveness and modest FEV_1 improvements. Moreover, systemic corticosteroids may have lowered blood eosinophil counts while airway eosinophilia persisted undetected. Incorporating sputum eosinophil or surrogate biomarkers such as fractional exhaled nitric oxide (FeNO) could yield a more comprehensive assessment of airway inflammation. Collectively, our findings suggest that peripheral blood eosinophil counts alone may be insufficient to predict lung-function impairment in patients with obstructive spirometric patterns unless combined with additional inflammatory markers (e.g., IL-5, IgE).

Limitation and Strengths

The best thing about this text is that it lays out a structured research roadmap for future studies on eosinophilia in obstructive airway diseases. It focuses on threshold standardization, long-term longitudinal cohorts, multi-omics approaches, targeted therapeutic trials, and AI-based predictive modeling, all of which can help with precision medicine and make the results more useful in the real world. A full demographic and clinical view is also shown by looking at environmental factors and foreign joint research at the same time. However, its main flaw is that it only makes suggestions and doesn't back them up with empirical data. It doesn't go into operational details like sample size, cost, feasibility in resource-limited settings, or how to actually run trials; it's more of a conceptual framework than a research plan that can be put into action right away.

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

Conclusion

Although the present study did not demonstrate a significant association between blood eosinophilia and demographic variables or pulmonary function in patients with obstructive lung disease, it underscores the importance of standardizing definitions and investigating underlying factors. These findings strengthen the case for a personalized approach to the management of obstructive pulmonary disease, particularly in cases where persistent eosinophilia or fluctuating levels are observed. Future studies with more rigorous study designs and larger sample sizes may help clarify the role of eosinophilia in prognosis and treatment response.

Funding

There is no financial conflicts of interest to disclose.

CRediT authorship contribution statement

Conceptualization, Methodology, Validation, Formal Analysis, Investigation, Resources, Software, Data Curation, Writing–Original Draft Preparation, Writing–Review & Editing, Visualization, Supervision, Project Administration: BS, AA, RG, VM.

Declaration of Generative AI and AI-assisted technologies in the writing process

No AI writing assistance was utilized in the production of this manuscript.

Conflict of interest

The authors have declared that no competing interests exist.

Ethical considerations

The study protocol was approved by the ethics committee of Iran University of Medical Sciences (IR.IUMS.FMD.REC.1402.560), and strict confidentiality was maintained; data were used solely for research purposes, and results are reported in aggregate to protect patient anonymity.

Data availability statement

The data that support the findings of this study are available on request.

Acknowledgements

The authors appreciate the assistance of PFT ward staff in Hazrat Rasoul Akram Hospital for collecting data.

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

References

1. Wang X, Mensinga TT, Schouten JP, Rijcken B, Weiss ST. Determinants of maximally attained level of pulmonary function. Am J Respir Crit Care Med. 2004;169(8):941-9. https://doi.org/10.1164/rccm.2201011
2. Lin CH, Cheng SL, Chen CZ, Chen CH, Lin SH, Wang HC. Current Progress of COPD Early Detection: Key Points and Novel Strategies. Int J Chron Obstruct Pulmon Dis. 2023;18:1511-24. https://doi.org/10.2147/COPD.S413969
3. Bui DS, Lodge CJ, Burgess JA, Lowe AJ, Perret J, Bui MQ, et al. Childhood predictors of lung function trajectories and future COPD risk: a prospective cohort study from the first to the sixth decade of life. Lancet Respir Med. 2018;6(7):535-44. https://doi.org/10.1016/S2213-2600(18)30100-0
4. Mogensen I, Vonk JM, Wijnant SRA, Zhou X, Boezen HM, Brusselle G, et al. Blood eosinophil level and lung function trajectories: cross-sectional and longitudinal studies in European cohorts. ERJ Open Res. 2020;6(4). https://doi.org/10.1183/23120541.00320-2020
5. DiLillo KM, Norman KC, Freeman CM, Christenson SA, Alexis NE, Anderson WH, et al. A blood and bronchoalveolar lavage protein signature of rapid FEV(1) decline in smoking-associated COPD. Sci Rep. 2023;13(1):8228. https://doi.org/10.1038/s41598-023-32216-0
6. Pignatti P, Visca D, Cherubino F, Zampogna E, Lucini E, Saderi L, et al. Do blood eosinophils strictly reflect airway inflammation in COPD? Comparison with asthmatic patients. Respir Res. 2019;20(1):145. https://doi.org/10.1186/s12931-019-1111-1
7. Pavord ID, Chanez P, Criner GJ, Kerstjens HAM, Korn S, Lugogo N, et al. Mepolizumab for Eosinophilic Chronic Obstructive Pulmonary Disease. N Engl J Med. 2017;377(17):1613-29. https://doi.org/10.1056/NEJMoa1708208
8. Tashkin DP, Wechsler ME. Role of eosinophils in airway inflammation of chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis. 2018;13:335-49. https://doi.org/10.2147/COPD.S152291
9. Yii ACA, Tay TR, Choo XN, Koh MSY, Tee AKH, Wang DY. Precision medicine in united airways disease: A "treatable traits" approach. Allergy. 2018;73(10):1964-78. https://doi.org/10.1111/all.13496
10. Cui Y, Chen Y. Blood eosinophils in chronic obstructive pulmonary disease: A potential biomarker. J Transl Int Med. 2023;11(3):193-7. https://doi.org/10.2478/jtim-2023-0096
11. Yang J, Yang J. Association Between Blood Eosinophils and Mortality in Critically Ill Patients with Acute Exacerbation of Chronic Obstructive Pulmonary Disease: A Retrospective Cohort Study. Int J Chron Obstruct Pulmon Dis. 2021;16:281-8. https://doi.org/10.2147/COPD.S289920
12. Cui Y, Zhang W, Ma Y, Zhan Z, Chen Y. Stability of blood eosinophils in acute exacerbation of chronic obstructive pulmonary disease and its relationship to clinical outcomes: a prospective cohort study. Respir Res. 2021;22(1):301. https://doi.org/10.1186/s12931-021-01888-5
13. Mogensen I, Alving K, Jacinto T, Fonseca J, Janson C, Malinovschi A. Simultaneously elevated FeNO and blood eosinophils relate to asthma morbidity in asthmatics from NHANES 2007-12. Clin Exp Allergy. 2018;48(8):935-43. https://doi.org/10.1111/cea.13137
14. Frette C, Annesi I, Korobaeff M, Neukirch F, Dore MF, Kauffmann F. Blood eosinophilia and FEV1. Cross-sectional and longitudinal analyses. Am Rev Respir Dis. 1991;143(5 Pt 1):987-92. https://doi.org/10.1164/ajrccm/143.5_Pt_1.987
15. Hancox RJ, Pavord ID, Sears MR. Associations between blood eosinophils and decline in lung function among adults with and without asthma. Eur Respir J. 2018;51(4). https://doi.org/10.1183/13993003.02536-2017
16. Müllerová H, Hahn B, Simard EP, Mu G, Hatipoğlu U. Exacerbations and health care resource use among patients with COPD in relation to blood eosinophil counts. Int J Chron Obstruct Pulmon Dis. 2019;14:683-92. https://doi.org/10.2147/COPD.S194367
17. Yun JH, Lamb A, Chase R, Singh D, Parker MM, Saferali A, et al. Blood eosinophil count thresholds and exacerbations in patients with chronic obstructive pulmonary disease. J Allergy Clin Immunol. 2018;141(6):2037-47.e10. https://doi.org/10.1016/j.jaci.2018.04.010
18. Zeiger RS, Tran TN, Butler RK, Schatz M, Li Q, Khatry DB, et al. Relationship of Blood Eosinophil Count to Exacerbations in Chronic Obstructive Pulmonary Disease. J Allergy Clin Immunol Pract. 2018;6(3):944-54.e5. https://doi.org/10.1016/j.jaip.2017.10.004
19. Hastie AT, Martinez FJ, Curtis JL, Doerschuk CM, Hansel NN, Christenson S, et al. Association of sputum and blood eosinophil concentrations with clinical measures of COPD severity: an analysis of the SPIROMICS cohort. Lancet Respir Med. 2017;5(12):956-67. 10.1016/S2213-2600(17)30432-0
20. Casanova C, Celli BR, de-Torres JP, Martínez-Gonzalez C, Cosio BG, Pinto-Plata V, et al. Prevalence of persistent blood eosinophilia: relation to outcomes in patients with COPD. European Respiratory Journal. 2017;50(5):1701162. https://doi.org/10.1183/13993003.01162-2017
21. Singh D, Wedzicha JA, Siddiqui S, de la Hoz A, Xue W, Magnussen H, et al. Blood eosinophils as a biomarker of future COPD exacerbation risk: pooled data from 11 clinical trials. Respir Res. 2020;21(1):240. https://doi.org/10.1186/s12931-020-01482-1
22. Han Y, Hua J, He B, Zhang J. Inhaled Antibiotics and Airway Bacterial Decolonization for Patients with Chronic Obstructive Pulmonary Disease: The Rationale and Future. J Transl Int Med. 2022;10(3):181-4. https://doi.org/10.2478/jtim-2022-0005
23. Agusti A, Calverley PM, Celli B, Coxson HO, Edwards LD, Lomas DA, et al. Characterisation of COPD heterogeneity in the ECLIPSE cohort. Respir Res. 2010;11(1):122. https://doi.org/10.1186/1465-9921-11-122
24. Bafadhel M, Pavord ID, Russell REK. Eosinophils in COPD: just another biomarker? Lancet Respir Med.

[Downloaded from demo.medilam.ac.ir on 2026-11-14]

2017;5(9):747-59. https://doi.org/10.1016/S2213-2600(17)30217-5
25. Tamondong-Lachica DR, Skolnik N, Hurst JR, Marchetti N, Rabe APJ, Montes de Oca M, Celli BR. GOLD 2023 Update: Implications for Clinical Practice. Int J Chron Obstruct Pulmon Dis. 2023;18:745-54. https://doi.org/10.2147/COPD.S404690
26. Brusselle GG, Maes T, Bracke KR. Eosinophils in the spotlight: Eosinophilic airway inflammation in nonallergic asthma. Nat Med. 2013;19(8):977-9. https://doi.org/10.1038/nm.3300
27. Dixon AE, Johnson SE, Griffes LV, Raymond DM, Ramdeo R, Soloveichik A, et al. Relationship of adipokines with immune response and lung function in obese asthmatic and non-asthmatic women. J Asthma. 2011;48(8):811-7. https://doi.org/10.3109/02770903.2011.613507
28. Janson C, Bjermer L, Lehtimäki L, Kankaanranta H, Karjalainen J, Altraja A, et al. Eosinophilic airway diseases: basic science, clinical manifestations and future challenges. Eur Clin Respir J. 2022;9(1):2040707. https://doi.org/10.1080/20018525.2022.2040707
29. Navabi B, Upton JE. Primary immunodeficiencies associated with eosinophilia. Allergy Asthma Clin Immunol. 2016;12:27. https://doi.org/10.1186/s13223-016-0130-4
30. Pascoe S, Locantore N, Dransfield MT, Barnes NC, Pavord ID. Blood eosinophil counts, exacerbations, and response to the addition of inhaled fluticasone furoate to vilanterol in patients with chronic obstructive pulmonary disease: a secondary analysis of data from two parallel randomised controlled trials. Lancet Respir Med. 2015;3(6):435-42. https://doi.org/10.1016/S2213-2600(15)00.

[Downloaded from demo.medilam.ac.ir on 2026-11-14]