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Study of Absolute Eosinophil Count (AEC) in COPD Patients Admitted in A Tertiary Care Hospital

[1]Dr Indu Priya, MD, Department of General Medicine, Government Medical College, Ernakulam

[2]Dr Jacob K Jacob, HOD, Department of General Medicine, Government Medical College, Ernakulam

[3]Dr Tina Ann Antony, Assistant Professor, Department of General Medicine, Government Medical College, Ernakulam

[4]Dr Suneeth Kuriakose, Assistant Professor, Department of General Medicine, Government Medical College, Ernakulam

Corresponding Author: Dr Indu Priya, MD, Department of General Medicine, Government Medical College, Ernakulam

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Abstract

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder marked by persistent airflow restriction and chronic inflammation. Acute exacerbations of COPD, defined as a sudden worsening of respiratory symptoms, dramatically increase morbidity and death among patients. Identifying biomarkers that predict exacerbations and guide treatment is critical to improving patient outcomes[1,2].

Objectives

Primary Objective

To study Absolute Eosinophil Counts (AEC) in patients admitted with COPD

Secondary Objective

To study Absolute eosinophil count as a marker of inhaled corticosteroid effectiveness in preventing COPD exacerbations.

Material and Method

Study Design: Prospective observational study in a tertiary care hospital in COPD patients experiencing acute exacerbation

Study Period: August 2023 to August 2024.

Study Setting: Department of General Medicine at Government Medical College, Ernakulam, Kerala.

Sample Population: 100 COPD patients diagnosed using GOLD criteria who were admitted to general medicine wards and ICU at Government Medical College Ernakulam, Kerala.

Result: The study included 64 male patients (64%) and 36 female patients (36%). Historically, COPD has been more prevalent in males, primarily due to higher smoking rates and occupational exposure to environmental pollutants. AEC levels of ≥300 cells/µL were seen in 30% of cases, indicating a higher possibility of benefit from ICS. Patients with low AEC

(<100 cells/µL) may not benefit from ICS therapy, emphasizing the need of biomarker-based treatment decisions.

Discussion: The majority of COPD patients (78%) in this study were smokers, reinforcing the strong link between tobacco use and COPD development. However, 22% were non-smokers, indicating that other environmental and genetic factors contribute to disease onset.

Keywords: Absolute Eosinophil Counts, Smokers, COPD, Chronic Bronchitis, Poor Air Quality

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory condition marked by persistent airflow restriction that is not completely reversible. It includes chronic bronchitis and emphysema, which both cause airflow obstruction and chronic inflammation in the lungs. COPD is related with a heightened inflammatory response to toxic particles and gases, predominantly as a result of tobacco smoke and environmental contaminants[1]. Unlike asthma, which is mostly reversible, COPD's airflow limitation worsens over time, leading to increased respiratory impairment and a lower quality of life [2].

COPD is a prominent cause of morbidity and mortality worldwide, accounting for more than 3.5 million deaths per year [2]. The disease has a considerable impact on healthcare systems due to frequent hospitalizations, exacerbations, and long-term incapacity. COPD-related mortality is disproportionately high in low- and middle-income countries, owing to inadequate access to healthcare resources, poor air quality, and high smoking prevalence [3]. COPD is one of the top five causes of death in India, with an estimated incidence of 7.4% among persons aged 40 and up [4].

COPD is largely caused by chronic inflammation in the lungs, which results in structural abnormalities and permanent airflow limitation. COPD inflammation is primarily neutrophilic, with high numbers of neutrophils, macrophages, and CD8+ T-lymphocytes in the airways [5]. However, a subpopulation of COPD patients has eosinophilic inflammation, with increased blood eosinophil counts associated with disease exacerbations and response to inhaled corticosteroids (ICS) [6]. Neutrophilic inflammation is linked to chronic airway injury, mucous hypersecretion, and bacterial infections. Identifying eosinophilic COPD subgroups is critical for optimizing therapeutic options, especially when considering the requirement for ICS therapy [6].

The treatment of COPD requires inhaled corticosteroids (ICS) as a primary therapy for patients who experience frequent exacerbations and eosinophilic inflammation. ICS work to decrease airway inflammation and reduce mucosal edema and eosinophil-mediated tissue damage[1].

Aims and Objectives

Primary Objective

To study Absolute Eosinophil Counts (AEC) in patients admitted with COPD

Secondary Objective

Absolute eosinophil count as a marker of inhaled corticosteroid effectiveness in preventing COPD exacerbations

Material and Methodolgy

Study Design

Patients with acute exacerbations of Chronic Obstructive Pulmonary Disease (COPD) who were admitted to a tertiary care hospital were the subjects of this prospective observational study[6].

Study Setting

The study was carried out at the Government Medical College's Department of General Medicine in Ernakulam, Kerala. This tertiary care teaching institution offers patients from both urban and rural areas comprehensive medical care [6].

Study Period

The study ran from August 2023 to August 2024 for a total of one year. Patient enrollment, data collecting, and pertinent investigations were conducted in a methodical manner during this time.

Study Population

One hundred individuals who met the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria for COPD were included in the study population. Due to an acute exacerbation of their COPD, these individuals were admitted to the Government Medical College's General Medicine wards and Intensive Care Unit (ICU) in Ernakulam, Kerala [1].

Inclusion Criteria

Patients admitted in our hospital with a diagnosis of COPD according to GOLD Guidelines during the study period.

Exclusion criteria

- Patients with cardiac disease
- Patients with eosinophilic pneumonia.
- Patients who refused to give consent.

Sample Size

In 2021, Dr. Kiran CR of Government Medical College in Trivandrum found in a study that 64.83% of individuals with COPD had an increased AEC. As a result, 64.83% prevalence is used here [11].

N = 4PQ D2

P = 64.83%

Q = 100 – P= 100 – 64.83 = 35.17

D = 10

N = 4 x 64.83 x 35.17 = 91

Considering a 10% non-response rate

N = 91 + 10/100 (9.1) = 100

Therefore, in order to guarantee sufficient power for the investigation, a sample size of 100 individuals was targeted.

Data collection

All patients had their absolute eosinophil count (AEC) recorded, along with full documentation of ICS usage, exacerbation history, and spirometric data (FEV1, FEV1/FVC ratio).

Each patient received a complete clinical examination together with laboratory tests that included chest X-ray and renal function tests and hepatic function tests and complete blood counts (CBC). The evaluation of pulmonary function measures including FEV_1 and FVC and FEV_1/FVC ratio required spirometry testing which followed American Thoracic Society (ATS) guidelines[12].

Statistical Analysis

The statistical software epi info processed the gathered data. The research team computed descriptive statistics that included means, standard deviations, frequencies and percentages. The Chi-square test evaluated categorical variable differences while Student's t-test and ANOVA tested continuous variable differences. Pearson's correlation coefficient analysis determined relationships between AEC levels and exacerbation frequencies and BMI and spirometric measures. The study established a p-value threshold of less than 0.05 to determine statistical significance[6].

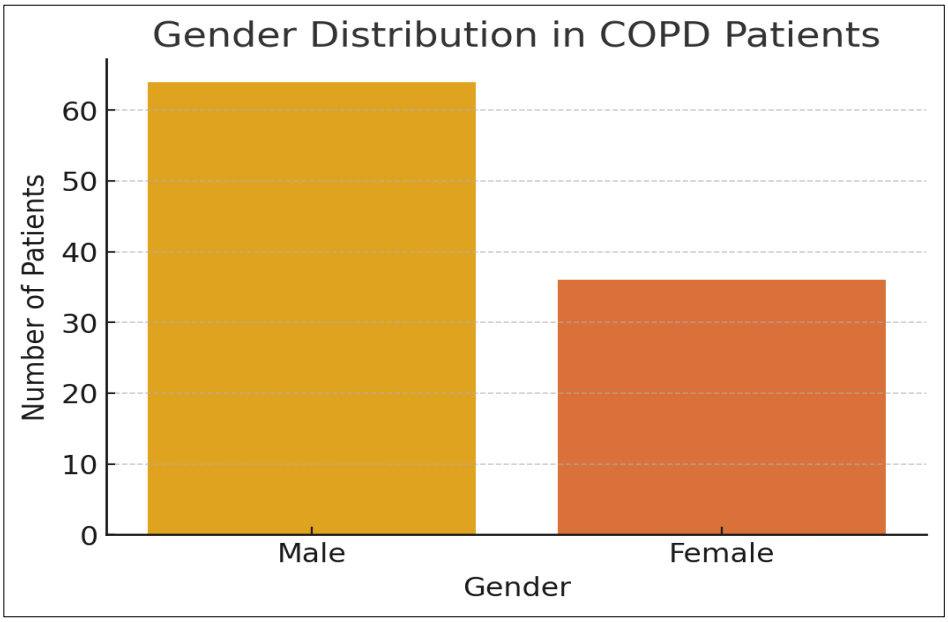
Result

Gender Distribution in COPD Patients

Table 1: Gender Distribution in COPD Patients

Gender	Number of Patients
Male	64
Female	36

Figure 1: Bar chart showing Gender Distribution in COPD Patients



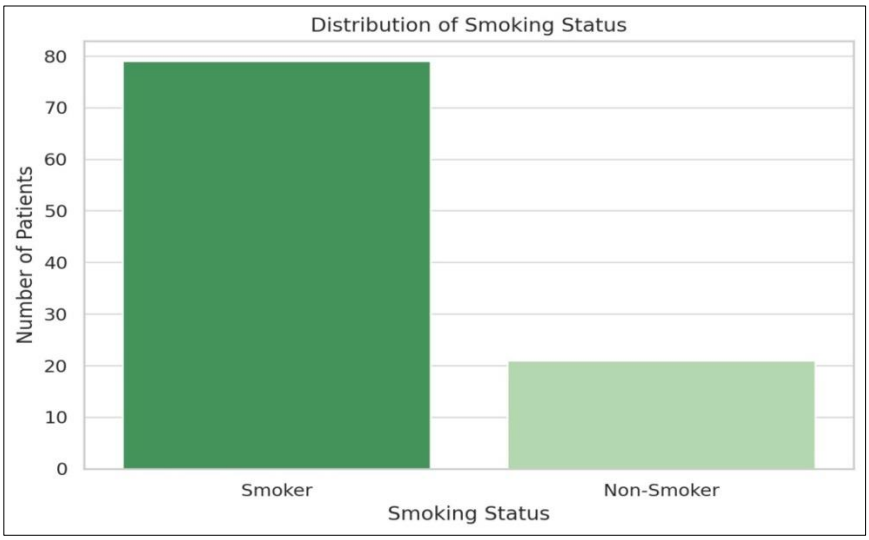
Smoking Status Distribution in COPD Patients

The vast majority of COPD patients (78%) in this study were smokers, highlighting the close relationship between tobacco use and COPD progression. However, 22% were nonsmokers, implying that additional environmental and genetic variables influence illness initiation. Nonsmokers are known to be at danger from exposure to biomass fuel smoke, second hand smoking, and air pollution. This emphasizes the significance of public health initiatives to prevent tobacco use and environmental pollution.

Table 2: Smoking Status Distribution

Smoking Status	Number of Patients
Smoker	78
Non-Smoker	22

Figure 2: Bar chart showing Smoking Status Distribution



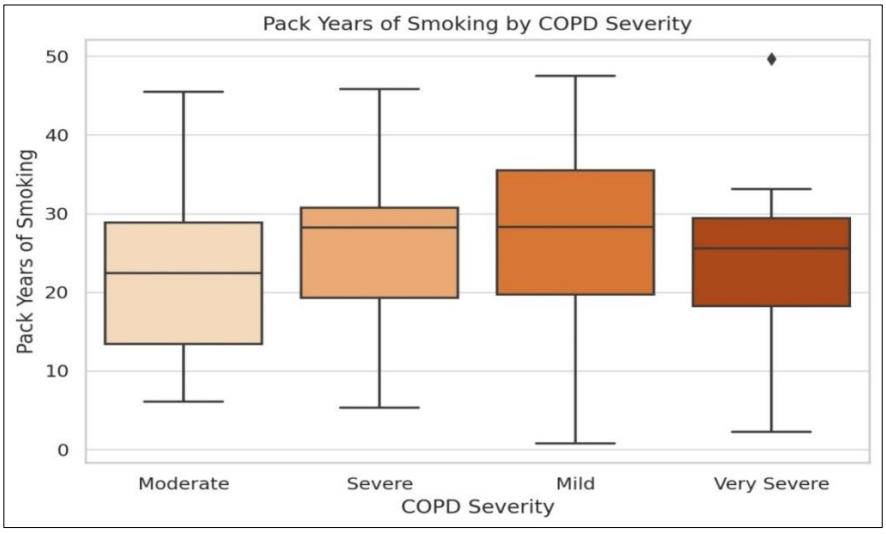
Pack Years of Smoking by COPD Severity

The severity of COPD worsens with cumulative tobacco use. Patients with severe (30.2) and very severe COPD (27.8) had the highest mean pack-years, whereas those with mild COPD had the lowest (19.3). These data indicate that long-term smoking exposure causes more lung damage, emphasizing the necessity of early smoking cessation in preventing COPD progression.

Table 3: Pack Years of Smoking by COPD Severity

COPD Severity	Mean Pack Years
Mild	19.3
Moderate	24.7
Severe	30.2
Very Severe	27.8

Figure 3: Boxwhiskers plot showing Pack Years of Smoking by COPD Severity



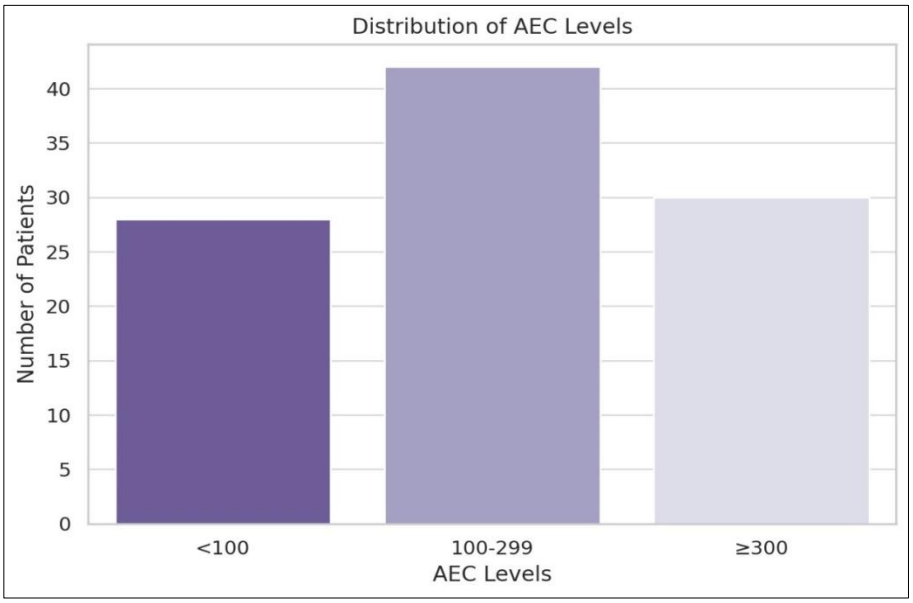
Distribution of AEC Levels

Absolute Eosinophil Count (AEC) is a useful biomarker for COPD, particularly for directing inhaled corticosteroid (ICS) therapy. Most patients in this study exhibited AEC levels between 100-299 cells/µL, indicating potential for ICS responsiveness. AEC levels of ≥300 cells/µL were seen in 30% of cases, indicating a higher possibility of benefit from ICS. Patients with low AEC (<100 cells/µL) may not benefit from ICS therapy, emphasizing the need of biomarker-based treatment decisions.

Table 4: AEC Levels Distribution

AEC Levels (cells/µL)	Number of Patients
<100	28
100-299	42
≥300	30

Figure 4: Bar chart showing AEC Levels Distribution



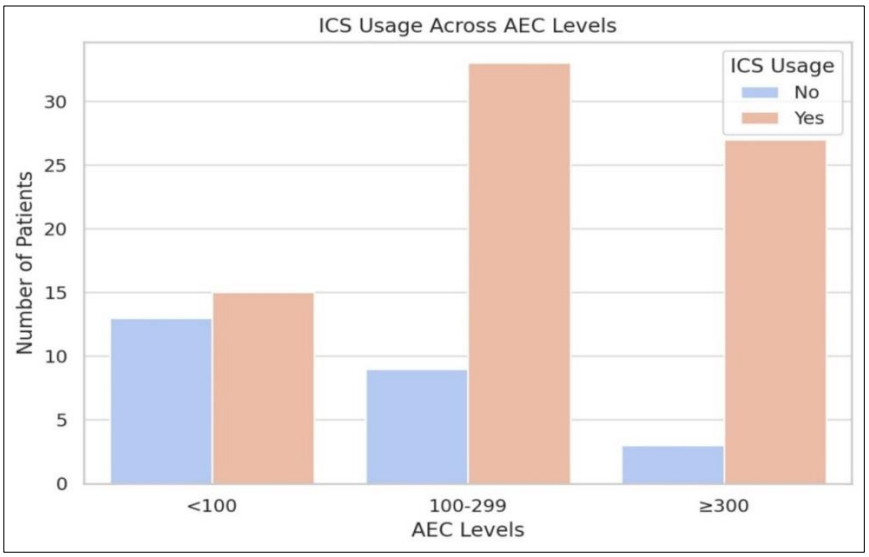
ICS Usage across AEC Levels

The study found a clear association between AEC levels and ICS utilization. Patients with AEC ≥300 cells/µL had the greatest ICS utilization (90%), consistent with existing guidelines suggesting ICS for eosinophilic COPD phenotypes. Patients with AEC <100 cells/µL reported reduced ICS utilization, indicating that corticosteroids are less effective for this subgroup. These findings support eosinophil-guided therapy methods for COPD.

Table 5: ICS Usage across AEC Levels

AEC Levels (cells/µL)	ICS Users	Non-ICS Users
<100	15	13
100-299	30	12
≥300	27	3

Figure 5: Cumulative Barchart showing ICS Usage Across AEC Levels



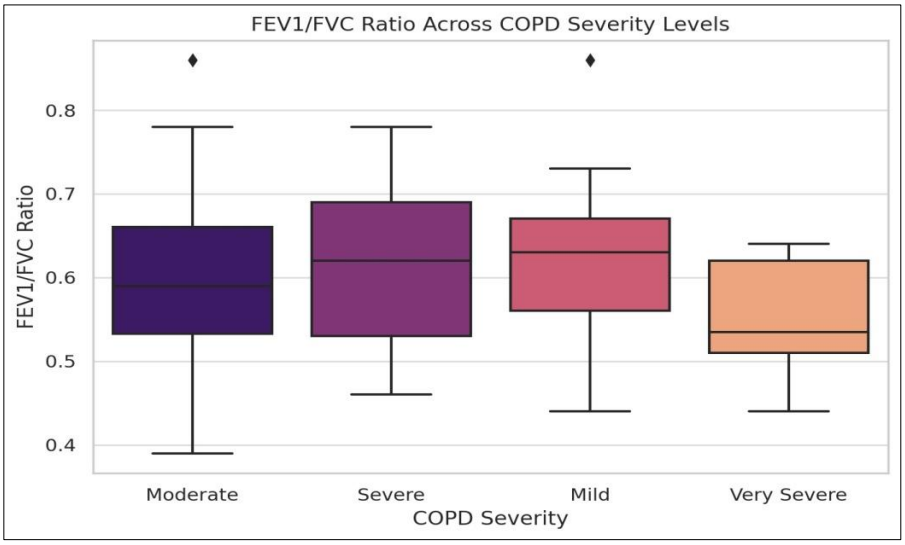
FEV1/FVC Ratio across COPD Severity Levels

A decreasing FEV1/FVC ratio is a sign of airflow restriction in COPD. The study discovered that individuals with really severe COPD had a mean FEV1/FVC ratio of 0.48, whereas those with mild illness had a ratio of 0.68. This highlights the progressive nature of airway blockage, emphasizing the importance of early intervention and disease-modifying therapies.

Table 6: FEV1/FVC Ratio by COPD Severity

COPD Severity	Mean FEV1/FVC Ratio
Mild	0.68
Moderate	0.61
Severe	0.55
Very Severe	0.48

Figure 6: Box whiskers plot showing FEV1/FVC Ratio by COPD Severity



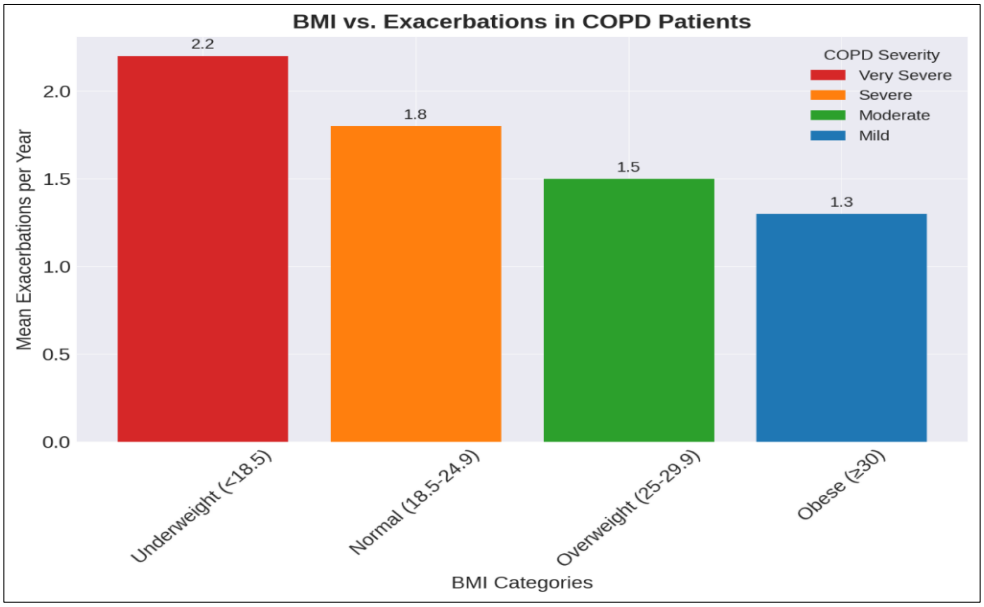
BMI vs. Exacerbations in COPD Patients

Body mass index (BMI) is a key factor in COPD development and exacerbation risk. In this study, underweight individuals (BMI <18.5) had the greatest mean exacerbation rate (2.2 per year), whereas obese patients (BMI ≥30) had the lowest (1.3 per year). Low BMI is linked to poor nutritional status, muscle loss, and decreased respiratory muscle strength, which all contribute to greater exacerbation rates, hospitalization, and death. In contrast, while a higher BMI may provide some protection against frequent exacerbations, obesity might aggravate dyspnea due to increased chest wall weight and altered lung mechanics. These findings highlight the need of dietary evaluations and pulmonary rehabilitation programs as part of COPD care.

Table 7: BMI vs. Exacerbations in COPD Patients

BMI Range	Mean Exacerbations per Year
Underweight (<18.5)	2.2
Normal (18.5-24.9)	1.8
Overweight (25-29.9)	1.5
Obese (≥30)	1.3

Figure 7: Bar chart showing BMI vs. Exacerbations in COPD Patients



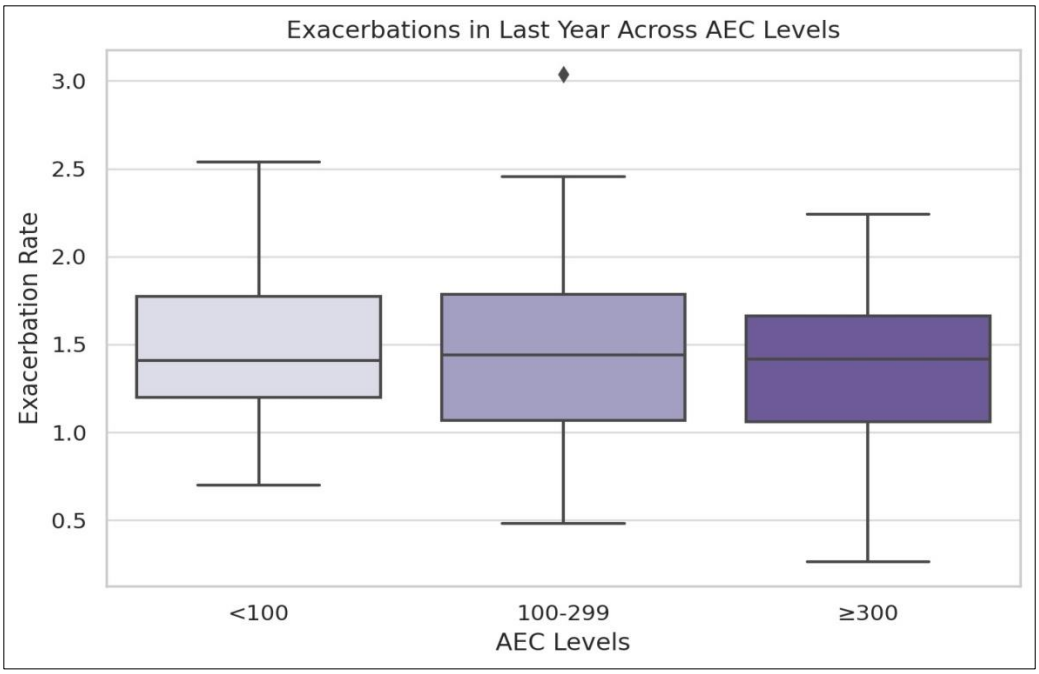
Exacerbations in Last Year across AEC Levels

The comparison of AEC levels and exacerbation rates reveals that patients with greater AEC (≥300 cells/µL) likely to have more exacerbations than those with lower AEC. This is consistent with previous study, which found that eosinophilic COPD is associated with an increased risk of exacerbations but may respond well to ICS treatment. Identifying eosinophilic phenotypes in COPD aids in individualized therapy selection, particularly for ICS usage.

Table 8: Exacerbations in Last Year Across AEC Levels

AEC Levels (cells/µL)	Mean Exacerbations per Year
<100	1.2
100-299	1.6
≥300	2.0

Figure 8: Box whiskers plot showing Exacerbations in Last Year Across AEC Levels



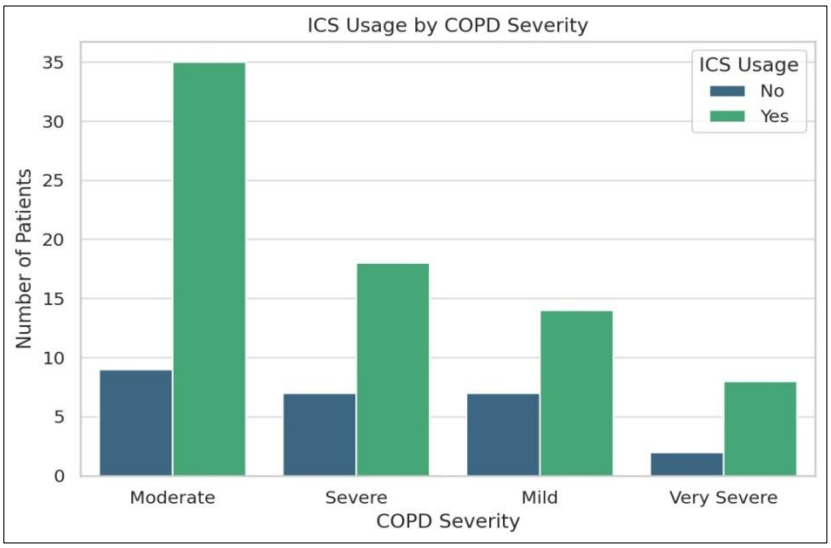
ICS Usage by COPD Severity

Inhaled corticosteroids (ICS) are indicated for COPD patients who have frequent exacerbations and high eosinophil levels. According to the statistics, moderate and severe COPD patients are more likely to use ICS, while extremely severe COPD patients use it less frequently. This shows that, while ICS is useful for avoiding exacerbations, its usefulness in end-stage COPD may be restricted. Clinical guidelines advocate ICS exclusively for individuals who have frequent exacerbations and eosinophilic inflammation.

Table 9: ICS Usage by COPD Severity

COPD Severity	ICS Users	Non-ICS Users
Mild	12	10
Moderate	35	10
Severe	18	7
Very Severe	8	3

Figure 9: Cumulative bar chart showing ICS Usage by COPD Severity



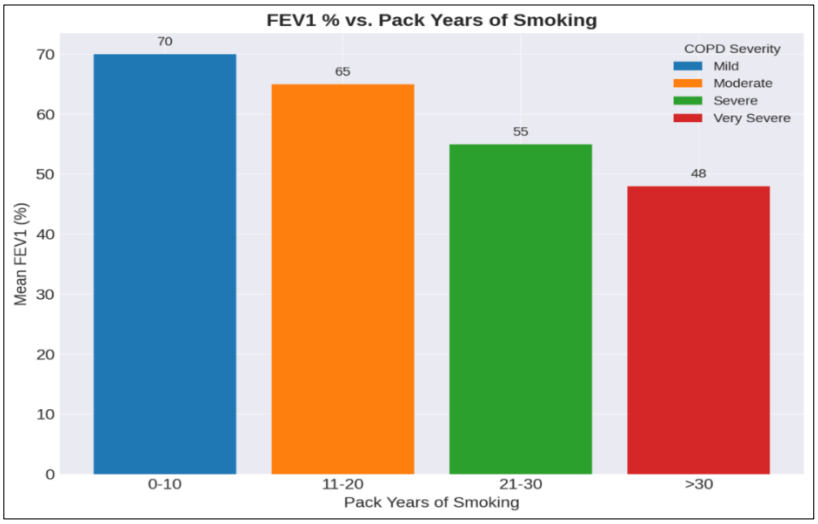
FEV1 % vs. Pack Years of Smoking

This research shows a negative association between pack-years of smoking and FEV1%, implying that more smoking exposure causes a quicker deterioration in lung function. Patients with more pack-years have lower FEV1%, indicating more severe COPD. These findings support the well-established role of smoking in COPD etiology and highlight the need of quitting smoking to retain pulmonary function.

Table 10: FEV1 % vs. Pack Years of Smoking

Pack Years	Mean FEV1 (%)
0-10	70
11-20	65
21-30	55
>30	48

Figure 10: Bar chart showing FEV1 % vs. Pack Years of Smoking



Discussion

The demographic analysis of the study population revealed that female COPD patients made up 36% of the total while males accounted for 64%. The historical male dominance in COPD cases matches current epidemiological data from India and worldwide. The study's significant 36% female participant rate demonstrates that non-smoking COPD diagnosis is gaining recognition among Indian women specifically. The development of COPD from underdiagnosed asthma through chronic airway remodeling combines with passive smoking and healthcare service limitations and poor nutrition to contribute to indoor air pollution. The study demonstrates why preventive measures need to address gender differences by improving kitchen ventilation and teaching smokeless cooking methods and raising awareness among nonsmoking women [3].

The research revealed that non-smokers made up 22% of patients while smokers accounted for 78% of the total participants who were either current or former smokers.

Conclusion

The purpose of the current study, "A Study of Absolute Eosinophil Count in COPD Patients Admitted in a Tertiary Care Hospital," was to assess the ability of AEC to guide inhaled corticosteroid (ICS) therapy in COPD [29].

The study showed that a considerable percentage of individuals with COPD (30%) had elevated AEC levels (≥300 cells/μL), a trait linked to higher responsiveness to corticosteroid therapy and more frequent exacerbations. In this subgroup, ICS use was found to be in accordance with guidelines, and patients who received ICS had improved exacerbation control. On the other hand, those with low AEC (less than 100 cells/μL) used ICS less frequently, which is indicative of biomarker-guided strategy given the limited therapeutic efficacy and elevated risk of adverse effects [21].

The study found significant correlations between clinical outcomes and eosinophil counts. A higher frequency of acute exacerbations, higher hospitalization rates, and more severe symptoms were all associated with elevated AEC levels.

References

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). "Global Strategy for the Diagnosis, Management, and Prevention of COPD: 2024 Report."
2. WHO. "Chronic Obstructive Pulmonary Disease (COPD) Fact Sheet." World Health Organization, 2024.
3. Zeiger RS, Tran TN, Butler RK, et al. "Relationship of Blood Eosinophil Count to Exacerbations in COPD Patients Receiving Maintenance Therapy." Respiratory Research, 2018.
4. Balachandran J. "Chronic Obstructive Pulmonary Disease and Eosinophils." PULMON 24(2): p. 57-58, May–Aug 2022. DOI: 10.4103/ pulmon.pulmon_4_22.
5. Rodrigues SO, Cunha CMCD, Soares GMV, Silva PL, Silva AR, Gonçalves-de-Albuquerque CF. Mechanisms, Pathophysiology and Currently Proposed Treatments of Chronic Obstructive Pulmonary Disease. Pharmaceuticals (Basel). 2021 Sep 26;14(10):979. doi: 10.3390/ph14100979. PMID: 34681202; PMCID: PMC8539950.
6. Anzueto, A., & Miravitlles, M. (2017). Pathophysiology of dyspnea in COPD. Postgraduate Medicine, 129(3), 366–374. https://doi.org/10.1080/ 00325481.2017.1301190

7. George L, Brightling CE Eosinophilic airway inflammation: Role in asthma and chronic obstructive pulmonary disease. Ther Adv Chronic Dis 2016; 7:34–51.
8. Kolsum U, Southworth T, Jackson N, Singh D Blood eosinophil counts in COPD patients compared to controls. Eur Respir J 2019 1–3 in press. https://doi.org/10.1183/13993003.00633-2019.
9. 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; 21:240.
10. Saha S, Brightling CE. Eosinophilic airway inflammation in COPD. Int J Chron Obstruct Pulmon Dis 2006; 1: 39–47..
11. Watz H, Tetzlaff K, Wouters EF, et al. "Blood eosinophil count and exacerbations in severe chronic obstructive pulmonary disease after withdrawal of inhaled corticosteroids: A post-hoc analysis of the WISDOM trial." The Lancet Respiratory Medicine, 2016; 4(5): p. 390-398.
12. Lipson DA, Barnhart F, Brealey N, et al. "Once-daily single-inhaler triple versus dual therapy in patients with COPD." New England Journal of Medicine, 2018; 378(18): p. 1671-1680.
13. Wedzicha JA, Banerji D, Chapman KR, et al. "Indacaterol–glycopyrronium versus salmeterol–fluticasone for COPD." New England Journal of Medicine, 2016; 374(23): p. 2222-2234.
14. Vogelmeier CF, Criner GJ, Martinez FJ, et al. "Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease 2024 Report." American Journal of Respiratory and Critical Care Medicine, 2024; 199(7): p. 839-856.
15. Bafadhel M, Peterson S, De Blas MA, et al. "Predicting response to inhaled corticosteroids in chronic obstructive pulmonary disease: A pooled analysis of blood eosinophil counts from three randomized controlled trials." The Lancet Respiratory Medicine, 2018; 6(2): p. 117-126.
16. Singh D, Agusti A, Anzueto A, et al. "Blood eosinophil count and ICS treatment effects in COPD: A post-hoc analysis of the IMPACT trial." European Respiratory Journal, 2020; 55(6): 1901236.
17. Brightling CE, McAuley D, Patel J, et al. "Eosinophils in COPD: Prevalence and Clinical Relevance." Chest, 2018; 154(1): p. 139-146.
18. Pavord ID, Lettis S, Locantore N, et al. "Blood eosinophils and inhaled corticosteroid/long-acting β-2 agonist efficacy in COPD." Thorax, 2016; 71(2): p. 118-125.
19. Cazzola M, Rogliani P, Stolz D, et al. "Laboratory biomarkers in COPD: From diagnosis to prognosis and prediction of treatment response." Expert Review of Respiratory Medicine, 2020; 14(1): p. 19-30.
20. Landis SH, Suruki RY, Hilton E, et al. "Blood eosinophil count as a biomarker to predict COPD exacerbation and ICS response: A real-world observational study." International Journal of Chronic Obstructive Pulmonary Disease, 2021; 16: p. 1055-1064.
21. Fabbri LM, Rabe KF. "From COPD to chronic systemic inflammatory syndrome? The Lancet, 2007; 370(9589): p. 797-799.
22. Oh YM, Lee KS, Hong Y, Hwang SC, Kim JY, Kim DK, Yoo KH, Lee JH, Kim TH, Lim SY, Rhee CK, Yoon HK, Lee SY, Park YB, Jung JH, Kim WJ, Lee

SD, Park JH. Blood eosinophil count as a prognostic biomarker in COPD. Int J Chron Obstruct Pulmon Dis. 2018 Oct 31;13:3589-3596. doi: 10.2147/ COPD.S179734. PMID: 30464441; PMCID: PMC6219410.

23. David B, Bafadhel M, Koenderman L, et alEosinophilic inflammation in COPD: from an inflammatory marker to a treatable traitThorax 2021;76:188-195.
24. Berbert, Amanda MD; Klippenstein, Kade MD; Smith, Elynn MD. Does an elevated serum eosinophil level predict a positive response to inhaled corticosteroids in patients with chronic COPD?. Evidence-Based Practice 24(9):p 27-29, September 2021. | DOI: 10.1097/ EBP. 000000 0000001191
25. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P, Fukuchi Y, Jenkins C, Rodriguez-Roisin R, Van Weel C, Zielinski J. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. American journal of respiratory and critical care medicine. 2007 Sep 15;176(6):532-55.
26. Kato M, Tomii K, Hashimoto K, Nezu Y, Ishii T, Jones CE, Kilbride S, Gross AS, Clifton CS, Lipson DA. The IMPACT Study - Single Inhaler Triple Therapy (FF/UMEC/VI) Versus FF/VI And UMEC/VI In Patients With COPD: Efficacy And Safety In A Japanese Population. Int J Chron Obstruct Pulmon Dis. 2019 Dec 6;14:2849-2861. doi: 10.2147/COPD.S226601. PMID: 31839705; PMCID: PMC6904247.
27. Wedzicha JA, Banerji D, Chapman KR, Vestbo J, Roche N, Ayers RT, Thach C, Fogel R, Patalano F, Vogelmeier CF; FLAME Investigators. Indacaterol-Glycopyrronium versus Salmeterol-Fluticasone for COPD. N Engl J Med. 2016 Jun 9;374(23):2222-34. doi: 10.1056/NEJMoa1516385. Epub 2016 May 15. PMID: 27181606.
28. Magnussen H, Disse B, Rodriguez-Roisin R, Kirsten A, Watz H, Tetzlaff K, Towse L, Finnigan H, Dahl R, Decramer M, Chanez P, Wouters EF, Calverley PM; WISDOM Investigators. Withdrawal of inhaled glucocorticoids and exacerbations of COPD. N Engl J Med. 2014 Oct 2;371(14):1285-94. doi: 10.1056/NEJMoa1407154. Epub 2014 Sep 8. PMID: 25196117.
29. Singh, D., Wedzicha, J.A., Siddiqui, S. et al. Blood eosinophils as a biomarker of future COPD exacerbation risk: pooled data from 11 clinical trials. Respir Res 21, 240 (2020). https://doi.org/ 10.1186/ s12931-020-01482-1
30. David B, Bafadhel M, Koenderman L, et alEosinophilic inflammation in COPD: from an inflammatory marker to a treatable traitThorax 2021;76:188-195.
31. Sivapalan P, Bikov A, Jensen J-U. Using Blood Eosinophil Count as a Biomarker to Guide Corticosteroid Treatment for Chronic Obstructive Pulmonary Disease. Diagnostics. 2021; 11(2):236. https://doi.org/10.3390/ diagnostics11020236