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Research Article

Role of Blood Eosinophils, Neutrophils and Serum IGE levels as Biomarkers in Chronic Obstructive Pulmonary Disease

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ABSTRACT
Aim: To predict the relationship of blood eosinophils, neutrophils & serum IgE levels in patients with chronic obstructive pulmonary disease, to modify disease progression, hospital admissions & prediction of COPD exacerbations. To correlate elevated blood eosinophils, neutrophils and serum IgE levels with exacerbation of COPD.
Materials and Methods: Cross sectional observational study of 140 patients at Department of Pulmonary Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar, suspected to have COPD
Results: This study showed a male predominance of 85% (119/140) and female 15% (21/140), mostly in the 50–70 years age group (83.5%). COPD was most common in Stage II (28.5%) and Stage III (41.4%). Exacerbations increased with staging: Stage I (38%), Stage II (60%), Stage III (67%), Stage IV (76%). Mean FEV1 declined from 54.3% in stable COPD to 48.9% in exacerbations. Absolute eosinophil counts rose from 305 (Stage I) to 1065 (Stage IV), neutrophils from 4650 to 5782/µL, and serum IgE from 790 to 3247 IU/mL. Smokers with >10 pack years had more exacerbations and elevated biomarkers. Elevated eosinophils (>300 cells/µL), neutrophils (>6000 cells/µL), and IgE (>150 IU/mL) were significantly higher in exacerbations (p=0.001), supporting biomarker-guided COPD management.
Conclusion: Elevated eosinophils, neutrophils, and IgE levels are observed in both stable and exacerbated COPD, with higher levels during exacerbations. These markers correlate with disease severity, smoking, and environmental exposures. Eosinophils also predict better ICS response. Recurrent exacerbations worsen lung function.

Keywords: COPD, Eosinophils, Exacerbation, Neutrophils, Sr.Ige.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death globally [(1).] It encompasses chronic bronchitis and emphysema. Chronic bronchitis is defined as a productive cough for at least three consecutive months over two years [(2,3).] Emphysema is characterized by abnormal airspace enlargement and alveolar wall destruction [(4,5),] which may result from small airway inflammation[(6)]. COPD affects approximately 9.7–10.3% globally [(7),] with Indian prevalence rates at 11.4% in males and 7.4% in females [(8)]. COPD is a heterogeneous condition defined by chronic respiratory symptoms and persistent airway and/or alveolar abnormalities [(9)]. Smoking is the leading cause [(10)]. Studies from Sweden and Denmark show over 74% of COPD cases are smoking-related [(11,12)].

In India, bidi smoking is more harmful than cigarettes [(13,14)]. Continued smoking accelerates FEV1 decline [(15)]. Environmental tobacco smoke also increases risk. Occupational exposures account for 15% of COPD cases [(16)]. Outdoor air

pollution and high traffic density contribute to COPD risk, especially in urban areas [17,18]. Indoor air pollution from biomass fuel is a major cause among rural Indian women. Other risk factors include alpha-1 antitrypsin deficiency (1–2% of cases) [9], childhood respiratory infections[19], genetic predisposition, and airway hyper-responsiveness.

Pathogenesis: Inflammatory cell recruitment (macrophages, neutrophils, eosinophils) damages lung tissue[9]. Protease-antiprotease imbalance and oxidative stress cause further injury. Neutrophil elastase and other enzymes degrade lung elastin, impairing recoil. Antioxidants like superoxide dismutase and vitamins A and E offer partial protection [9].

Exacerbations increase neutrophilic inflammation, though eosinophilic patterns are observed in 10–40% of cases and may respond better to corticosteroids. Eosinophilic airway inflammation is now recognized in COPD, underlining its heterogeneity and need for tailored treatments [20].

Aim and Objectives of the study

- To predict the relationship of blood eosinophils, neutrophils & serum IgE levels in patients with chronic obstructive pulmonary disease, to modify disease progression, hospital admissions &prediction of COPD exacerbations.
- To correlate elevated blood eosinophils, neutrophils and serum IgE levels with exacerbation of COPD.

MATERIALS AND METHODS

Study design: Cross sectional observational study done at Department of Pulmonary Medicine, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar. Duration of study was from September 2022- March 2024. Sample size**:** 140 Patients. After the informed written consent by the patients, study was proceeded with the approval of local ethical committee.

Inclusion Criteria

- Symptoms- breathlessness, cough with expectoration, wheeze
- Exposure to biomass and other inhalational injury
- Occupational exposure to dust & fumes
- Smokers and non-smokers

Exclusion Criteria

- Bronchial asthma patients
- Asthma COPD overlap syndrome
- Allergic to substances
- Allergic conditions which predispose to elevated blood eosinophils and serum
- IgE levels
- Exposure to pets
- Treated tuberculosis
- Family h/o bronchial asthma/atopic dermatitis
- Individuals using or used oral corticosteroids or ICS-containing products
- Regular drug intake other than bronchodilators& vitamin supplements.

Procedure

Patients clinically diagnosed with COPD were screened using inclusion and exclusion criteria and underwent baseline and post-bronchodilator spirometry. Those with $FEV_1/FVC < 0.7$ and no significant reversibility were included. Routine tests (CBC, LFT, RFT, sputum, chest X-ray) and additional investigations (CT chest, stool, peripheral smear, serum IgE) were done to exclude conditions like tuberculosis, parasitic infections, eosinophilic pneumonia, malignancy, and ACOS. A total of 140 patients were classified into GOLD stages I–IV and categorized as stable or exacerbation groups based on Anthonisen's criteria. Exacerbation frequency over the past year was recorded. Absolute eosinophil, neutrophil counts, and serum IgE were analyzed.

Statistical analysis

Collection of data was using SPSS software and $p < 0.05$ was considered significant.

RESULTS AND OBSERVATIONS

Distribution based on gender Out of 140 patients of study population, there are 119(85%) males and 21(15%) females, COPD Predominantly seen in males. In Distribution based on Age In this study population of 140 patients, maximum incidence is seen in age group of 50 -70 years.

Table 1: Comparison of Absolute Eosinophil Count in Stage I, II, III, IV

STAGE	AEC (mean)
Stage I	305.23
Stage II	612.3

Stage III	846.3
Stage IV	1065.3

There is an increase in AEC as the COPD stages advances, indicating a correlation between higher eosinophil counts and disease progression.

Table 2: Comparison of Neutrophil counts in COPD Stage I, II, III, IV

STAGE	NEUTROPHIL COUNT
Stage I	4650.14
Stage II	4830.03
Stage III	5583.43
Stage IV	5782.76

The data shows a steady increase in neutrophil counts from Stage I to Stage IV, suggesting that higher neutrophil levels are associated with more severe stages of COPD

Table 3: Comparison of Serum IgE in COPD Stage I, II, III, IV

Stage	Serum IgE
Stage I	789.9
Stage II	1724.4
Stage III	1988.3
Stage IV	3246.8

The results show a significant increase in serum IgE levels as the COPD stages progress, indicating higher IgE levels are linked to the worsening severity of COPD, Comparison of Neutrophil Count among Stable COPD & COPD.

Exacerbation: n=140, >6000 seen in both stable COPD & COPD exacerbation but comparatively higher in COPD exacerbation.

Table 4: Comparision of Neutrophil Count among Stable COPD & COPD Exacerbation

CUT OFF NEUTROPHIL COUNT (cells/µl)	STABLE COPD	COPD EXACERBATION	P- VALUE
<6000	4554.2	4209.9	<0.001
>6000	8788.2	9871.8	

n=140, >6000 seen in both stable COPD & COPD exacerbation but comparatively higher in COPD exacerbation

Table 5: Comparison of Serum IgE among Stable COPD & COPD Exacerbation

CUT OFF S.IgE (IU/ml) (mean)	STABLE COPD	COPD EXACERBATION	P- VALUE
<150	65.7	0	<0.001
>150	1728.8	2309	

n=140, >150 IU/ml seen in both stable COPD & COPD exacerbation but comparatively higher in COPD exacerbation.

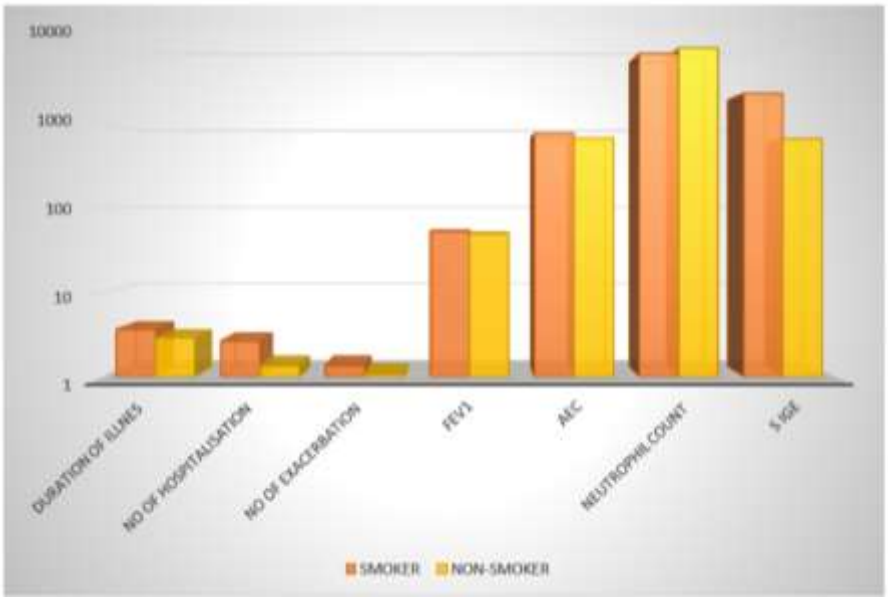

Chart 1: Comparision of Variables among Smokers & Non- Somkers

Table 6: Comparison of Variables among Smokers & Non- Smokers:

Exposure to smoke	Duration of illness	No of hospitalisation	No of exacerbation	FEV1	AEC	Neutrophil count	S IgE
SMOKER	3.61	2.63	1.36	51.57	747.25	6524.08	2214.13
NONSMOKER	2.88	1.35	1.08	48.67	660.62	7601.54	646.92

n=140, Duration of illness, No. of hospitalisations, Absolute eosinophil count, neutrophil count and serum IgE levels were higher among smokers compared to nonsmokers. Radiological Prevalence among the study population, most common radiological finding observed was Centriacinar& Paraseptal pattern. Irregular pattern seen the least.

DISCUSSION

This study investigates the significance of blood eosinophils, neutrophils, and serum IgE levels as biomarkers in patients with chronic obstructive pulmonary disease (COPD), assessing their role in both stable disease and during exacerbations. COPD is a progressive inflammatory airway disease characterized by airflow limitation and recurrent exacerbations, contributing significantly to global morbidity and mortality [6,8]. A total of 140 patients were studied, with a clear male predominance (85%) and the majority aged between 50–70 years (83.5%), consistent with epidemiological trends reported in previous studies [7,10,11].

COPD staging showed most patients in Stage II (28.5%) and Stage III (41.4%). As disease severity increased from Stage I to Stage IV, there was a corresponding increase in the number of exacerbations, illness duration, and decline in lung function (measured by FEV1), which aligns with the natural history of COPD progression described in earlier studies [5,18]. However, hospitalization rates did not differ significantly across stages. Bronchodilator use was nearly twice as high in exacerbation cases, and occupational or biomass exposure was more common in these patients, supporting evidence linking environmental and occupational exposures to COPD severity [12,15,16].

Biomarker analysis revealed a progressive rise in eosinophils, neutrophils, and serum IgE levels across disease stages. Mean values were significantly higher in exacerbation cases compared to stable COPD. These findings are consistent with previous studies that have

demonstrated the role of inflammatory biomarkers in COPD exacerbations [14,19].

The study applied clinically relevant biomarker thresholds: ≥300 cells/μL for eosinophils, ≥6000 cells/μL for neutrophils, and ≥150 IU/mL for serum IgE. All three biomarkers were elevated in both stable and exacerbation groups, but levels were notably higher during exacerbations. Smoking, including current, former, and passive exposure, especially in those with over 10 pack-years, was significantly associated with elevated biomarker levels, prolonged illness duration, and more frequent exacerbations, which is well supported in the literature [9,12,13].

The findings support existing literature. Elevated eosinophils were associated with better corticosteroid responsiveness, suggesting that eosinophil count can help identify patients likely to benefit from inhaled corticosteroids [19]. Similarly, elevated neutrophil counts were linked to disease severity and exacerbation risk, reflecting the neutrophil-driven inflammatory response characteristic of COPD [5,18].

High serum IgE levels indicated an allergic phenotype associated with more frequent exacerbations and suggest a potential role for targeted therapies in selected patients. This highlights the overlap between COPD and asthma-like inflammatory pathways, which has been increasingly recognized in recent years [2,18].

Integrating these biomarkers into routine clinical practice can aid early identification of high-risk patients, guide treatment decisions (e.g., ICS in eosinophilic COPD, antibiotics in neutrophilic exacerbations), and predict long-term outcomes. However, barriers remain, including the need for standardized cut-off values and broader access to testing. Future directions should focus on developing combined biomarker panels, conducting long-term studies on biomarker dynamics, and enhancing accessibility to enable personalized COPD management [8].

CONCLUSION

Elevated blood eosinophil counts, neutrophils & serum IgE levels were observed in both stable COPD and COPD exacerbation but comparatively was higher in COPD exacerbation. Hence, blood eosinophils and neutrophils can be used as prognostic biomarkers in COPD exacerbation. Biomarkers mainly elevated eosinophil counts were linked to higher exacerbation rates and better inhaled corticosteroid responses. Smoking is related with longer duration of illness, increased exacerbations, elevated blood eosinophils, neutrophils & serum IgE levels correlating with increased disease severity and exacerbation rates. The findings also show the impact of occupational exposure to dust and biomass fuels, especially in rural areas, on COPD exacerbations. Recurrent exacerbation of COPD is believed to accelerate disease progression and impairment of pulmonary function. Further, studies to be done to analyse biomarkers in COPD & ACOS. Larger study population would provide better outcome.

Ethical Approval: Approved

Conflict of Interest: The authors declare no conflict of interest.

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