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Original Research Article

Association between Serum IGE Levels and Disease Severity in COPD Patients

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation and chronic airway inflammation. Although smoking remains the major risk factor, inflammatory biomarkers may play an important role in identifying disease severity and clinical phenotype. Serum immunoglobulin E (IgE), commonly associated with allergic and eosinophilic inflammation, may be linked with airway obstruction, bronchodilator response and severity of COPD.

Aim: The aim of the study was to assess the association between serum IgE levels and disease severity in patients with COPD.

Material and Methods: The present prospective observational study was conducted in the Department of Respiratory Medicine, People's College of Medical Science and Research Centre and associated People's Hospital, Bhopal, over a period of 24 months from February 2024 to January 2026. A total of 50 newly diagnosed COPD patients attending OPD and IPD services were enrolled after obtaining written informed consent. Patients were evaluated using detailed clinical history, sociodemographic profile, smoking status, chest X-ray, oxygen saturation, mMRC dyspnoea score, spirometry and GOLD staging. Blood investigations included complete blood count, absolute eosinophil count, serum IgE measured by fluoroenzyme immunoassay method and C-reactive protein. Data were analysed using MS Excel and IBM SPSS version 20.

Results: Most patients belonged to the 60–69 years age group, 18 (36%), with male predominance, 38 (76%). Smoking was present in 41 (82%) patients. According to GOLD classification, 18 (36%) patients had moderate COPD, 17 (34%) had severe COPD, 9 (18%) had very severe COPD and 6 (12%) had mild COPD. Raised serum IgE was observed in 29 (58%) patients, while 21 (42%) had normal IgE levels. Mean pre-bronchodilator FEV1 was lower in the raised IgE group (49.2%) compared with the normal IgE group (63.63%). Serum IgE showed a strong negative correlation with FEV1 ($r = -0.74$, $p < 0.001$) and positive correlation with eosinophil count ($r = 0.64$, $p < 0.001$) and CRP ($r = 0.51$, $p = 0.002$).

Conclusion: Raised serum IgE levels were associated with increased COPD severity, reduced lung function and inflammatory activity. Serum IgE may serve as a useful supportive biomarker for assessing disease severity and identifying inflammatory phenotype in COPD patients.

Keywords: COPD, serum IgE, GOLD staging, FEV1, eosinophils.

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Introduction

Chronic obstructive pulmonary disease is a common, preventable and treatable respiratory disorder characterized by persistent airflow limitation, chronic respiratory symptoms and progressive structural changes in the airways and lung parenchyma. The disease usually develops after long-term exposure to noxious particles or gases, especially tobacco smoke, occupational dust, biomass fuel smoke and environmental pollution. COPD is not limited to simple airway narrowing; it represents a complex inflammatory disorder involving small airway disease, mucus hypersecretion, emphysematous destruction, impaired gas exchange and repeated episodes of acute worsening. Diagnosis and assessment are commonly based on clinical history, symptom

burden, spirometry and severity grading, with the GOLD strategy emphasizing post-bronchodilator airflow obstruction as the key diagnostic feature. [1] COPD remains an important cause of morbidity, mortality, hospital admission and reduced quality of life throughout the world. The clinical burden is particularly relevant in developing countries, where smoking, indoor air pollution, occupational exposures and delayed diagnosis commonly coexist. Patients often present late, when dyspnoea, cough, sputum production and exercise limitation have already affected daily activities. The course of COPD is heterogeneous; some patients show slow progression, whereas others develop frequent exacerbations, rapid lung function decline and systemic complications. Recent literature has emphasized that COPD should be viewed as a lifelong lung-health disorder influenced by genetic susceptibility, early-life exposures, environmental insults and adult risk factors rather than only as a smoking-related disease of older age. [2]

The global epidemiology of COPD has drawn increasing attention because the disease affects individuals across different age groups, socioeconomic backgrounds and exposure patterns. Although tobacco smoking remains the most established risk factor, many patients develop COPD due to non-smoking exposures, including biomass fuel, occupational irritants and ambient air pollution. This is especially important in India, where household smoke exposure, outdoor pollution and occupational dust may contribute substantially to chronic respiratory disease. The burden of COPD also rises with increasing age, as cumulative exposure, immunosenescence and progressive decline in lung function interact over time. Therefore, understanding demographic, clinical and biological factors associated with COPD severity is essential for improving diagnosis and patient stratification. [3] COPD severity is conventionally evaluated using spirometric parameters, particularly forced expiratory volume in one second, along with symptom scores and exacerbation history.

However, spirometry alone does not fully capture the biological diversity of the disease. Patients with similar airflow limitation may differ widely in inflammatory profile, bronchodilator responsiveness, and exacerbation tendency and treatment response. This has encouraged the search for simple biomarkers that may help identify clinically meaningful phenotypes. Blood-based markers are especially attractive because they are less invasive, widely available and easier to repeat in routine practice. Among such markers, eosinophil count, C-reactive protein and immunoglobulin E have received increasing attention in relation to airway inflammation and disease expression. [4] Traditionally, COPD was considered predominantly a neutrophilic inflammatory disease, while eosinophilic and allergic inflammation were mainly associated with asthma. This distinction is now considered too simplistic. Many COPD patients show overlapping inflammatory patterns, and a subgroup demonstrates type 2 inflammatory features, including eosinophilic activity, airway hyperresponsiveness, mucus production and partial bronchodilator reversibility. Recognition of treatable traits has shifted attention from a single disease label to individual patient characteristics that can guide therapy. The Lancet Commission has also highlighted the need for earlier diagnosis, better phenotyping and prevention-focused approaches to reduce the long-term impact of COPD. [5]

Eosinophilic inflammation is one of the best studied treatable traits in COPD. Blood eosinophil count is increasingly used to estimate the likelihood of response to inhaled corticosteroid-containing therapy and to identify patients with a greater inflammatory component. Nevertheless, eosinophils do not explain the complete immunological spectrum of COPD. Some patients may have allergic sensitization or increased IgE-mediated immune activity, suggesting that serum IgE may provide additional information beyond spirometry and eosinophil count. Exploring IgE in COPD is therefore relevant because it may help clarify whether allergic or type 2 immune pathways contribute to disease severity in selected patients. [6]

Materials and Methods

The present study entitled “Association between serum IgE levels and disease severity in COPD patients” was conducted as a prospective observational study among outpatient and inpatient newly diagnosed COPD patients seeking care in the Department of Respiratory Medicine, People’s College of Medical Science and Research Centre and associated People’s Hospital, Bhopal. The study was carried out over a period of 24 months, from February 2024 to January 2026.

The study design adopted for the present study was a prospective observational study. All outpatient and inpatient newly diagnosed COPD patients attending the Respiratory Department, People’s Hospital, Bhopal, were considered as the source of data for the study.

A total sample size of 50 newly diagnosed patients of COPD attending the Respiratory Department during February 2024 to September 2025 were recruited for the study.

The study area included the outpatient department and inpatient department of Respiratory Medicine,

People's College of Medical Science and Research Centre and associated People's Hospital, Bhopal. All newly diagnosed patients of COPD were included in the study. Patients less than 35 years of age and more than 80 years, patients with associated co-morbidities such as pneumonia, bronchiectasis and CAD, patients already on steroids, immunosuppressants or bronchodilators, patients with any malignancy, patients with autoimmune disorder or immunodeficiency disorder, pregnant females and those not giving consent were excluded from the study.

Written consent was obtained from all the study participants in the consent form after explaining the nature and purpose of the study in their local language using the participant information sheet. The participants were assured that confidentiality would be maintained, and the option to withdraw from the study was kept open throughout the study period.

Initially, ethical clearance was obtained from the Institute's Ethical Committee. All patients with COPD satisfying the selection criteria and willing to participate in the study were enrolled, and written consent was obtained from them. Detailed history regarding sociodemographic variables including name, age, gender, socioeconomic status, and residence and contact details was recorded along with detailed clinical history in the study proforma. COPD cases were diagnosed based on history, clinical features, oxygen saturation, chest X-ray, and dyspnoea score using the Modified Medical Research Council scale, spirometry and GOLD staging.

Severity of symptoms was assessed using the Modified Medical Research Council dyspnoea scale. Grade 0 was assigned when the patient became breathless only with strenuous exercise. Grade 1 was assigned when the patient developed shortness of breath while hurrying on level ground or walking up a slight hill. Grade 2 was assigned when the patient walked slower than people of the same age on level ground because of breathlessness or had to stop for breath while walking at their own pace on level ground. Grade 3 was assigned when the patient stopped for breath after walking about 100 yards or after a few minutes on level ground. Grade 4 was assigned when the patient was too breathless to leave the house or became breathless while dressing or undressing.

Further, spirometry was performed and parameters including forced vital capacity, forced expiratory volume in one second and FEV1/FVC ratio were recorded. Based upon GOLD guidelines, the severity of COPD was assessed. Blood samples were collected from the patients by phlebotomy into a plain vial, and blood investigations were carried out. Complete blood count, absolute eosinophil count, serum IgE and inflammatory biomarker C-reactive protein were assessed. Serum IgE was measured by the fluoroenzyme immunoassay method. Other tests, when clinically indicated, such as chest radiography, electrocardiography and HRCT, were also performed.

Further, patients were managed using inhaled Formoterol and Budesonide, and spirometry was repeated to assess improvement in these patients.

The study proforma included demographic parameters such as name, gender, age, occupation, phone number and address. Clinical parameters recorded in the proforma included blood investigations such as haemoglobin, total leukocyte count, differential leukocyte count, serum IgE, pulmonary function test findings and COPD GOLD stage.

Data were compiled using MS Excel and analysed with the help of IBM SPSS software version 20. Categorical data were expressed as frequency and proportions, whereas continuous data were expressed as mean and standard deviation. Association between serum IgE level and pre-bronchodilator spirometry was assessed using independent t-test, and the impact of treatment on spirometry, that is bronchodilator response, was assessed using paired t-test. Correlation of serum IgE levels with eosinophil count and CRP was done using Pearson correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

Results

Table 1 presents the demographic and baseline characteristics of the 50 study participants. The majority of patients belonged to the 60–69 years age group, accounting for 18 (36%) participants, followed by the 50–59 years age group with 14 (28%) participants. Patients aged ≥70 years constituted 12 (24%), while the 40–49 years age group represented the smallest proportion with 6 (12%) participants. Gender-wise distribution revealed a clear male predominance, with 38 (76%) males and 12 (24%) females enrolled in the study. Assessment of smoking status demonstrated that 41 (82%) participants were smokers, whereas only 9 (18%) were non-smokers, indicating that smoking was the predominant risk factor among the study population.

Table 2 shows the distribution of COPD severity according to the GOLD classification and serum IgE levels among the study participants. Most patients had moderate COPD, comprising 18 (36%) cases, closely followed by severe COPD with 17 (34%) cases. Very severe COPD was observed in 9 (18%) patients, while mild COPD accounted for only 6 (12%) patients. Evaluation of serum IgE

levels demonstrated that 29 (58%) participants had raised serum IgE levels, whereas 21 (42%) had normal serum IgE levels. Table 3 compares pre-bronchodilator FEV1 values between patients with normal and raised serum IgE levels and evaluates the correlation between serum IgE and lung function. The mean pre-bronchodilator FEV1 was 63.63% among patients with normal serum IgE, whereas it was markedly lower at 49.2% among patients with raised serum IgE, suggesting poorer pulmonary function in patients with elevated IgE levels. Correlation analysis further demonstrated a strong negative correlation between serum IgE levels and FEV1 ($r = -0.74$), which was highly statistically significant ($p < 0.001$).

Table 4 illustrates the bronchodilator response among patients with normal and raised serum IgE levels. Patients with normal serum IgE showed an improvement in mean FEV1 from 63.63 before bronchodilator administration to 66.4 after treatment, resulting in a mean increase of 2.77 units. In contrast, patients with raised serum IgE demonstrated an increase in mean FEV1 from 49.2 to 56.8, corresponding to a greater improvement of 7.6 units following bronchodilator therapy. Table 5 demonstrates the relationship between serum IgE levels and inflammatory biomarkers. Serum IgE showed a moderately strong positive correlation with absolute eosinophil count ($r = 0.64$), which was highly statistically significant ($p < 0.001$). Similarly, serum IgE exhibited a moderate positive correlation with C-reactive protein (CRP) ($r = 0.51$), which was also statistically significant ($p = 0.002$).

Table 6 depicts the distribution of normal and raised serum IgE levels across different stages of COPD severity. Among patients with mild COPD, 5 had normal serum IgE levels while only 1 had raised serum IgE. In the moderate COPD group, 10 patients had normal IgE levels compared with 8 patients with raised IgE. However, among patients with severe COPD, raised serum IgE levels predominated, with 12 patients compared to 5 with normal IgE. Likewise, in the very severe COPD group, 8 patients had raised serum IgE levels whereas only 1 patient had normal serum IgE.

Table 1: Demographic and Baseline Characteristics of Study Participants (n = 50)

Variable	Category	n	%
Age group	40–49 years	6	12%
	50–59 years	14	28%
	60–69 years	18	36%
	≥70 years	12	24%
	Total	50	100%
Gender	Male	38	76%
	Female	12	24%
	Total	50	100%
Smoking status	Smokers	41	82%
	Non-smokers	9	18%

Table 2: COPD Severity Based on GOLD Classification and Serum IgE Level Distribution

Variable	Category	n	%
COPD severity	Mild	6	12%
	Moderate	18	36%
	Severe	17	34%
	Very Severe	9	18%
Serum IgE level	Normal	21	42%
	Raised	29	58%

Table 3: Pre-Bronchodilator FEV1 Comparison and Correlation between IgE and Lung Function

Parameter	Group/Comparison	Value
Mean FEV1	Normal IgE	63.63%
Mean FEV1	Raised IgE	49.2%
IgE vs FEV1	r value	−0.74
IgE vs FEV1	P value	<0.001

Table 4: Bronchodilator Response with Respect to Normal and Raised Serum IgE

Group	Pre FEV1	Post FEV1	Change
Normal IgE	63.63	66.4	+2.77
Raised IgE	49.2	56.8	+7.6

Table 5: Correlation between IgE and Inflammatory Markers

Marker	r	p-value
Eosinophils	0.64	p< 0.001
CRP	0.51	p= 0.002

Table 6: IgE Levels vs COPD Severity

Severity	Normal IgE	Raised IgE
Mild	5	1
Moderate	10	8
Severe	5	12
Very Severe	1	8

Discussion

The present study included 50 newly diagnosed COPD patients, with the highest proportion in the 60–69 years age group, 18 (36%), followed by 50–59 years, 14 (28%), and ≥70 years, 12 (24%). Thus, 30 (60%) patients were aged 60 years and above, showing that COPD was more common in older age groups. Male predominance was observed, with 38 (76%) males and 12 (24%) females, and 41 (82%) patients were smokers. These findings are comparable with Buist et al. (2007), who reported in the BOLD study that COPD prevalence increased with age, with an odds ratio of 1.94 per 10-year age increment, and stage II or higher COPD was more common among males than females, 11.8% versus 8.5%, respectively. The predominance of smokers in the present study also supports the role of tobacco exposure as a major contributor to COPD development. [7] In the present study, COPD severity based on GOLD classification showed that most patients had moderate COPD, 18 (36%), followed by severe COPD, 17 (34%), very severe COPD, 9 (18%), and mild COPD, 6 (12%). Serum IgE levels were raised in 29 (58%) patients and normal in 21 (42%) patients, indicating that more than half of the COPD patients had elevated serum IgE levels. Jin et al. (2014) evaluated 273 COPD patients and reported elevated total IgE in 129 (47.3%) patients, which was slightly lower than the 58% observed in the present study. They also found that patients with elevated total IgE had worse lung function and more severe GOLD staging, supporting the present finding that raised IgE is commonly present in COPD patients and may be linked with disease severity. [8] In the present study, raised serum IgE was more frequent in advanced COPD stages. Among mild COPD patients, only 1 patient had raised IgE and 5 had normal IgE, while in moderate COPD, 8 had raised IgE and 10 had normal IgE. However, in severe COPD, 12 patients had raised IgE compared with 5 patients with normal IgE, and in very severe COPD, 8 patients had raised IgE compared with only 1 patient with normal IgE. This trend is similar to the study by Vivek et al. (2019), who studied 50 COPD patients and found elevated serum IgE in 42 (84%) patients and elevated absolute eosinophil count in 29 (58%) patients. They reported that mean serum IgE increased progressively with GOLD stage, from 505.5 in stage I to 858.6 in stage II, 1291.3 in stage III and 2278.5 in stage IV, showing a direct relationship between IgE level and COPD severity. [9] The present study showed that patients with raised serum IgE had poorer lung function compared with those having normal IgE. The mean pre-bronchodilator FEV1 was 63.63% in the normal IgE group, whereas it was reduced to 49.2% in the raised IgE group. A strong negative correlation was also observed between serum IgE and FEV1, with $r = -0.74$ and $p < 0.001$, indicating that increasing IgE levels were significantly associated with declining lung function. Singh et al. (2010) similarly observed that serum IgE and IL-1β levels were significantly raised in COPD patients compared with controls, and inflammatory markers correlated with pulmonary function impairment. They reported a significant negative correlation between IL-1β and FEV1, $r = -0.624$, $p = 0.003$, and between IgE and FVC, $r = -0.477$, $p = 0.034$, which supports the association of inflammatory and IgE-mediated pathways with airflow limitation in COPD. [10] In the present study, bronchodilator response was greater among patients with raised serum IgE than those with normal serum IgE. Patients with normal IgE showed improvement in FEV1 from 63.63 to 66.4, with a change of +2.77, whereas patients with raised IgE showed improvement from 49.2 to 56.8, with a greater change of +7.6.

This suggests that patients with raised IgE, despite having poorer baseline lung function, may show a better bronchodilator response, possibly due to an allergic or eosinophilic component contributing to airway obstruction. Rathod et al. (2010) compared bronchodilator reversibility, IgE, eosinophil and neutrophil counts among asthma and COPD patients and observed that IgE levels were raised in obstructive airway disease, with bronchodilator reversibility helping to differentiate airway phenotypes. This supports the observation that IgE-related inflammatory patterns may influence reversibility in selected COPD patients. [11] The present study demonstrated a moderately strong

positive correlation between serum IgE and absolute eosinophil count, with r = 0.64 and p < 0.001. This indicates that patients with higher serum IgE also tended to have higher eosinophilic inflammation. Yaqub et al. (2025) studied 96 COPD patients and reported that absolute eosinophil count increased progressively from GOLD stage A, 468 ± 102.26, to stage B, 696.5 ± 234.59, and stage E, 832.24 ± 115.05, with p < 0.0001. They also found that serum IgE was significantly higher in stage E, 1641.84 ± 580.50, compared with stage A, 271.15 ± 86.44, and stage B, 778.86 ± 468.30, and reported a strong positive correlation between AEC and serum IgE, r = 0.793, p < 0.0001. These findings closely support the present result that serum IgE and eosinophilic inflammation are strongly associated in COPD patients. [12] The present study also found a moderate positive correlation between serum IgE and C-reactive protein, with r = 0.51 and p = 0.002, suggesting that increased IgE levels may be associated not only with eosinophilic inflammation but also with systemic inflammatory activity. Moayyedkazemi et al. (2018) reported that COPD patients had significantly higher hsCRP levels than controls, 7.6 mg/L versus 4.2 mg/L, p < 0.001. They also observed a negative correlation between hsCRP and predicted FEV1, p = 0.03, r = 0.32, and a positive correlation between hsCRP and GOLD severity, p = 0.04, r = 0.3. Although their study evaluated CRP in relation to COPD severity rather than IgE directly, it supports the present observation that inflammatory biomarkers such as CRP increase with worsening pulmonary impairment in COPD. [13] Overall, the present study showed that raised serum IgE was present in 29 (58%) patients and was associated with lower mean FEV1, 49.2% versus 63.63% in normal IgE patients, a strong negative correlation with FEV1, r = −0.74, p < 0.001, greater bronchodilator response, +7.6 versus +2.77, positive correlation with eosinophils, r = 0.64, p < 0.001, and positive correlation with CRP, r = 0.51, p = 0.002. Lommatzsch et al. (2022), in large COPD cohorts, reported that total IgE was elevated in more than 30% of patients in both COSYCONET and WISDOM, and patients in the highest tertile of total IgE had increased risk of lung function decline, adjusted OR 1.62, 95% CI 1.12–2.34. Their findings support the present study by showing that elevated IgE is not merely an allergic marker but may identify a COPD subgroup with worse lung function and progressive disease. [14]

Conclusion

The present study concluded that raised serum IgE levels were commonly observed among COPD patients and were associated with greater disease severity. Patients with elevated serum IgE had lower pre-bronchodilator FEV1 and showed a significant negative correlation between serum IgE levels and lung function. Raised IgE was also positively correlated with eosinophil count and CRP, suggesting its association with inflammatory activity. Therefore, serum IgE may be considered a useful supportive biomarker for assessing disease severity and inflammatory phenotype in COPD patients.

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