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Original Research Article

Assessment of Serum IGE Levels and Their Association with Clinical Severity in COPD

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a heterogeneous, progressive respiratory disorder characterized by persistent airflow limitation and chronic airway inflammation. Although tobacco smoking is a major risk factor, distinct inflammatory pathways may contribute to differences in disease severity and clinical phenotype. Serum immunoglobulin E (IgE), a marker associated with type 2 and allergic inflammation, may have a potential association with airway obstruction and inflammatory activity in COPD.

Aim: To evaluate the association between serum IgE levels and disease severity in patients with COPD.

Material and Methods: This 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 24 months from February 2024 to January 2026. Fifty newly diagnosed patients with COPD attending the outpatient and inpatient services were enrolled after obtaining written informed consent. Participants underwent detailed clinical assessment, including sociodemographic and smoking history, chest radiography, oxygen saturation, modified Medical Research Council (mMRC) dyspnoea assessment, spirometry, and Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging. Laboratory investigations included complete blood count, absolute eosinophil count, serum IgE measured by fluoroenzyme immunoassay, and C-reactive protein (CRP). Statistical analysis was performed using Microsoft Excel and IBM SPSS Statistics version 20.

Results: The majority of participants belonged to the 60–69-year age group [18 (36%)], with a predominance of males [38 (76%)]. Smoking history was present in 41 (82%) patients. According to GOLD classification, 18 (36%) patients had moderate COPD, followed by severe COPD in 17 (34%), very severe COPD in 9 (18%), and mild COPD in 6 (12%). Serum IgE was elevated in 29 (58%) patients and within the normal range in 21 (42%). Mean pre-bronchodilator FEV_1 was lower among patients with elevated IgE than among those with normal IgE (49.2% vs. 63.63% predicted). Serum IgE demonstrated a strong inverse correlation with FEV_1 ($r = -0.74$, $p < 0.001$) and positive correlations with absolute eosinophil count ($r = 0.64$, $p < 0.001$) and CRP ($r = 0.51$, $p = 0.002$).

Conclusion: Elevated serum IgE was associated with greater airflow limitation and increased inflammatory activity among patients with COPD. Serum IgE may provide supportive information for identifying patients with an inflammatory phenotype and greater disease burden; however, its clinical utility as an independent marker of COPD severity requires validation in larger prospective studies.

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

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Introduction

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable respiratory disorder characterized by persistent airflow limitation, chronic respiratory symptoms, and

structural abnormalities involving the airways and lung parenchyma. It generally develops following prolonged exposure to noxious particles or gases, with tobacco smoke being a major risk factor along with biomass fuel exposure, occupational dust, and ambient air pollution. COPD encompasses a heterogeneous spectrum of pathological changes, including small-airway dysfunction, mucus hypersecretion, emphysematous destruction, impaired gas exchange, and recurrent exacerbations. Diagnosis is primarily based on clinical assessment and demonstration of persistent airflow obstruction on post-bronchodilator spirometry, while disease severity is further characterized using symptoms, exacerbation history, and lung function parameters. [1]

COPD represents a major global health burden, contributing substantially to morbidity, mortality, healthcare utilization, and impaired quality of life. The burden is particularly significant in low- and middle-income countries, where tobacco exposure, household air pollution, occupational hazards, and delayed diagnosis may coexist. Clinical progression is variable, with some individuals experiencing relatively stable disease while others develop recurrent exacerbations, progressive decline in lung function, and systemic complications. Contemporary understanding recognizes COPD as a lifelong respiratory disorder influenced by genetic susceptibility, early-life factors, environmental exposures, and cumulative adult risk factors rather than solely a smoking-related disease of older adults. [2]

The epidemiology of COPD is influenced by diverse environmental and demographic factors. Although cigarette smoking remains the most established risk factor, substantial disease burden is also attributable to non-tobacco exposures, including biomass fuel smoke, occupational pollutants, and ambient air pollution. These exposures are particularly relevant in India, where household and occupational environmental factors may contribute to chronic respiratory morbidity. Increasing age is additionally associated with greater COPD burden because cumulative exposure to risk factors occurs alongside age-related changes in lung structure, immune function, and physiological reserve. Characterization of demographic, clinical, and biological factors associated with disease severity is therefore important for improving early detection and patient stratification. [3] Spirometry, particularly forced expiratory volume in one second (FEV_1), remains central to the assessment of airflow limitation in COPD. However, spirometric impairment alone does not completely reflect the biological and clinical heterogeneity of the disease. Patients with comparable degrees of airflow limitation may differ substantially in inflammatory profile, symptom burden, exacerbation frequency, bronchodilator responsiveness, and therapeutic response. This has increased interest in accessible biomarkers that may provide additional information regarding disease phenotype and inflammatory activity. Blood-based biomarkers such as eosinophil count, C-reactive protein (CRP), and immunoglobulin E (IgE) have therefore been investigated in relation to different clinical manifestations of COPD. [4]

COPD has traditionally been characterized by predominantly neutrophilic inflammation, whereas eosinophilic and allergic inflammatory pathways have historically been more strongly associated with asthma. However, increasing evidence indicates that a subset of patients with COPD may exhibit type 2 inflammatory characteristics, including eosinophilic inflammation, airway hyperresponsiveness, mucus hypersecretion, and variable bronchodilator responsiveness. This recognition has contributed to a shift toward a treatable-trait approach, in which individual inflammatory and clinical characteristics are considered alongside conventional disease classification. The identification of such phenotypes may facilitate more individualized assessment and management of COPD. [5]

Blood eosinophil count is currently among the most extensively studied biomarkers of type 2 inflammation in COPD and may help identify patients who are more likely to benefit from inhaled corticosteroid-containing treatment. However, eosinophilic inflammation represents only one component of the broader inflammatory spectrum of COPD. Immunoglobulin E, a key mediator of allergic immune responses, may provide additional information regarding allergic sensitization and type 2 inflammatory activity in selected patients. Elevated serum IgE has been investigated in relation to airway obstruction, bronchodilator response, eosinophilic inflammation, and clinical characteristics of COPD. [6] Therefore, evaluating the relationship between serum IgE levels and objective measures of airflow limitation may contribute to improved understanding of inflammatory heterogeneity and disease severity in COPD. The present study was undertaken to assess the association between serum IgE levels and disease severity in patients with COPD.

Materials and Methods

A prospective observational study was conducted over 24 months, from February 2024 to January 2026, in the Department of Respiratory Medicine, People's College of Medical Science and Research Centre and associated People's Hospital, Bhopal. A total of 50 newly diagnosed patients with COPD attending the outpatient and inpatient services were enrolled after obtaining written informed consent.

Ethical approval was obtained from the Institutional Ethics Committee, and the study was conducted in accordance with applicable ethical principles. Patients aged 35–80 years with newly diagnosed COPD were included, while those with pneumonia, bronchiectasis, coronary artery disease, malignancy, autoimmune or immunodeficiency disorders, pregnancy, or previous treatment with steroids, immunosuppressants, or bronchodilators were excluded. Patients unwilling to provide consent were also excluded. Detailed demographic and clinical information, including age, sex, occupation, smoking history, residence, and relevant medical history, was recorded using a structured proforma. COPD was assessed based on clinical history and examination, oxygen saturation, chest radiography, modified Medical Research Council (mMRC) dyspnoea score, spirometry, and Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification. Spirometry parameters including forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and FEV_1/FVC ratio were recorded before and after bronchodilator administration. Blood samples were collected for complete blood count, absolute eosinophil count, serum IgE, and C-reactive protein (CRP). Serum IgE was estimated using the fluoroenzyme immunoassay method. Additional investigations, including electrocardiography and high-resolution computed tomography, were performed when clinically indicated. Patients subsequently received standard inhaled therapy with formoterol and budesonide, and spirometry was repeated to assess bronchodilator response. Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 20. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation. The independent t-test was used to compare pre-bronchodilator spirometric parameters according to serum IgE status, and the paired t-test was used to assess changes following treatment. Pearson's correlation coefficient was used to evaluate relationships between serum IgE, eosinophil count, and CRP. A p-value <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%
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 2: Association of Serum IgE with Pulmonary Function and Bronchodilator Response

Parameters	Normal IgE	Raised IgE	%
Pre-bronchodilator FEV_1 (% predicted)	63.63	49.20	12%
Post-bronchodilator FEV_1 (% predicted)	66.40	56.80	36%
Change in FEV_1	+2.77	+7.60	34%
IgE vs. FEV_1	$r = -0.74$	$P < 0.001$	18%

Table 3: Association of Serum IgE with COPD Severity and Inflammatory Markers.

Parameter	Findings	Statistical significance
IgE vs. COPD severity	Mild: 1 raised / 5 normal; Moderate: 8 raised / 10 normal; Severe: 12 raised / 5 normal; Very severe: 8 raised / 1 normal	--
IgE vs. eosinophil count	Positive correlation	$r = 0.64$, $p < 0.001$
IgE vs. CRP	Positive correlation	$r = 0.51$, $p = 0.002$
IgE vs FEV1	P value	<0.001

Discussion

The present study evaluated the association between serum IgE levels, pulmonary function, disease severity, and inflammatory markers among 50 newly diagnosed patients with COPD. The largest proportion of participants belonged to the 60–69-year age group [18 (36%)], followed by 50–59 years [14 (28%)] and ≥70 years [12 (24%)], with 30 (60%) patients aged ≥60 years. A male predominance was observed [38 (76%)], and 41 (82%) participants were smokers. These findings are consistent with the observations of Buist et al. (2007), who reported an increasing prevalence of COPD with advancing age and a higher prevalence of stage II or greater disease among men than women.[7] The predominance of smoking in the present cohort further emphasizes tobacco exposure as an important risk factor for COPD.

According to GOLD classification, moderate COPD was the most frequent category [18 (36%)], followed by severe [17 (34%)], very severe [9 (18%)], and mild disease [6 (12%)]. Serum IgE was elevated in 29 (58%) patients, while 21 (42%) had normal levels. Jin et al. (2014), in a study of 273 COPD patients, reported elevated total IgE in 129 (47.3%) patients. [8] Although the prevalence of raised IgE was somewhat higher in the present study, both studies demonstrate that elevated IgE is present in a substantial subset of COPD patients. Jin et al. also reported an association between elevated IgE, poorer lung function, and more advanced GOLD stage, supporting the potential relevance of IgE in identifying distinct inflammatory phenotypes within COPD. [8] In the present study, elevated IgE was more frequently observed in patients with advanced COPD. Among those with mild disease, only 1 of 6 patients had raised IgE, compared with 8 of 18 with moderate disease, 12 of 17 with severe disease, and 8 of 9 with very severe disease (Table 3). This distribution suggests an increasing proportion of IgE elevation with greater disease severity. Similar findings were reported by Vivek et al. (2019), who

observed elevated serum IgE in 42 (84%) of 50 COPD patients and reported progressively increasing mean IgE levels across GOLD stages.[9] Although the present study assessed IgE categorically rather than comparing mean IgE concentrations across GOLD stages, the overall pattern was consistent with their findings.

A significant relationship between serum IgE and pulmonary function was observed. Mean pre-bronchodilator FEV_1 was lower in patients with raised IgE than in those with normal IgE [49.2% vs. 63.63% predicted]. Furthermore, serum IgE demonstrated a strong inverse correlation with FEV_1 ($r = -0.74$, $p < 0.001$), indicating that higher IgE levels were associated with greater airflow limitation. Singh et al. (2010) similarly reported significant relationships between inflammatory markers and pulmonary function in COPD, including an inverse correlation between IgE and FVC ($r = -0.477$, $p = 0.034$). [10] These findings suggest that IgE-associated inflammatory pathways may be linked with impaired pulmonary function in a subset of COPD patients.

Patients with raised IgE also demonstrated a greater numerical improvement in FEV_1 following bronchodilator administration, increasing from 49.2% to 56.8% predicted (+7.6), compared with an increase from 63.63% to 66.4% (+2.77) in the normal-IgE group (Table 2). Rathod et al. (2010) evaluated IgE, eosinophil counts, and bronchodilator reversibility in obstructive airway disease and reported that IgE elevation and bronchodilator responsiveness may help characterize different inflammatory and functional airway phenotypes. [11]

The greater response observed in the raised-IgE group in the present study may therefore indicate a potentially reversible component of airflow limitation. However, because the present study did not establish an independent statistical association between IgE status and bronchodilator response, this finding should be interpreted as an observed trend rather than evidence of causality.

Serum IgE showed a significant positive correlation with absolute eosinophil count ($r = 0.64$, $p < 0.001$), suggesting an association between IgE elevation and eosinophilic inflammatory activity. Yaqub et al. (2025) similarly reported progressive increases in eosinophil count and serum IgE with increasing COPD severity and demonstrated a strong positive correlation between absolute eosinophil count and serum IgE ($r = 0.793$, $p < 0.0001$). [12] The concordance between these findings supports the possibility that elevated IgE may identify a subset of COPD patients with features of type 2/eosinophilic inflammation.

A significant positive correlation was also observed between serum IgE and CRP ($r = 0.51$, $p = 0.002$), indicating that higher IgE levels were associated with greater systemic inflammatory activity. Moayyedkazemi et al. (2018) reported significantly elevated hsCRP levels in COPD patients and found associations between hsCRP, FEV_1, and GOLD disease severity. [13] Although their study did not directly evaluate the relationship between IgE and CRP, their findings support the role of systemic inflammatory markers in COPD severity and pulmonary impairment.

Overall, the present study demonstrated that raised serum IgE was present in 29 (58%) patients and was associated with more advanced disease, lower mean FEV_1, and significant inverse correlation with airflow limitation. Elevated IgE was also positively correlated with eosinophil count and CRP, suggesting a relationship with both eosinophilic and systemic inflammatory activity.

These findings are consistent with Lommatzsch et al. (2022), who reported elevated total IgE in more than 30% of patients across large COPD cohorts and found that those with higher IgE had an increased risk of lung function decline (adjusted OR 1.62, 95% CI 1.12–2.34). [14] Collectively, the evidence suggests that serum IgE may serve as a useful adjunctive biomarker for identifying a biologically distinct COPD subgroup characterized by greater airflow limitation and inflammatory activity. However, larger longitudinal studies are required to establish whether IgE independently predicts disease progression and whether its measurement can improve clinical phenotyping or therapeutic decision-making.

Conclusion

Elevated serum IgE levels were frequently observed in patients with COPD and were associated with greater airflow limitation and disease severity.

Higher IgE levels were significantly associated with lower FEV_1 and positively correlated with eosinophil count and CRP, suggesting a relationship with eosinophilic and systemic inflammatory activity. Serum IgE may therefore serve as a useful adjunctive biomarker for COPD phenotyping and assessment of disease burden; however, its independent prognostic and clinical utility requires confirmation in larger prospective studies.

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