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Correlation of allergic serological markers with bronchiectasis severity in high-resolution computed tomography chests of patients with allergic bronchopulmonary aspergillosis: A retrospective study

Padamati Advytha[1], Karniha Balasubramanian[2], Ancila P[3], Sowmya P[4], Nalini Jayanthi Nagesh[5]

[1,3]Postgraduate Resident, [2]Assistant Professor, [4]Senior Resident, [5]Professor and Head, Department of Respiratory Medicine, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India

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ABSTRACT

Background: Allergic bronchopulmonary aspergillosis is a hypersensitivity reaction to *Aspergillus fumigatus* that leads to airway inflammation and bronchiectasis in susceptible individuals. Elevated immunoglobulin E (IgE), specific IgE, and eosinophilia correlate with disease severity; however, their precise relationship with high-resolution computed tomography (HRCT) findings remains unclear. **Aims and Objectives:** This study aimed to correlate allergic serological markers with the severity of bronchiectasis in HRCT chest using a bronchiectasis score on patients with allergic bronchopulmonary aspergillosis (ABPA). **Materials and Methods:** This retrospective observational study included data from 50 patients at SRM Medical College, Hospital and Research Centre, Kattankulathur, from June 2022 to June 2024. The medical records of patients with ABPA were reviewed for symptoms, history of asthma, steroid use, allergies, and comorbidities. The data included absolute eosinophil count (AEC), total IgE, specific IgE for *A. fumigatus*, and HRCT findings. The correlation between serological markers and bronchiectasis severity was analyzed using the bronchiectasis score. **Results:** The highest proportion (36%) of patients were aged ≤30 years, with a male predominance (58%). Cough was the most common symptom (96%), and hemoptysis was seen in 8% of study subjects. Asthma severity and serological markers were correlated with the bronchiectasis score, where asthma severity showed a strong correlation with the bronchiectasis score ($r = 0.807$, $P < 0.05$). AEC ($r = 0.473$), total IgE ($r = 0.447$), and specific IgE ($r = 0.504$) levels showed moderate positive correlations. **Conclusion:** A good correlation exists between allergic serological markers and bronchiectasis severity in patients with ABPA, as observed on HRCT. Higher AEC, total IgE, and specific IgE levels against *A. fumigatus* were associated with more severe bronchiectatic changes.

Key words: Allergic bronchopulmonary aspergillosis; Bronchiectasis; Serological markers; Immunoglobulin E; Eosinophilia; *Aspergillus fumigatus*

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INTRODUCTION

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction to *Aspergillus fumigatus* occurring in patients with asthma or cystic fibrosis and is characterized by airway inflammation, mucus plugging, and lung structural damage, frequently developing into bronchiectasis.[1] High-resolution computed tomography (HRCT) of the

Address for Correspondence:
Dr. Karniha Balasubramanian, Assistant Professor, Department of Respiratory Medicine, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India. **Mobile:** +91-8056197690. **E-mail:** karniha93@gmail.com

chest is currently the standard reference for determining bronchiectasis severity, revealing in-depth airway abnormality imaging, including bronchial dilation, thickening of walls, and mucus impaction. Determining serological markers associated with disease severity on HRCT can enhance early diagnosis, direct treatment, and predict disease progression.[2]

Several immunological markers are important in ABPA pathogenesis, which are diagnostic and prognostic. Raised total serum immunoglobulin E (IgE) levels are a characteristic feature of ABPA, representing an amplified immune response to *A. fumigatus* antigens in patients with ABPA. Specific IgE and immunoglobulin G (IgG) concentrations to *A. fumigatus* are similarly usually elevated, indicating allergic sensitization and chronic antigenic stimulation, respectively.[3] Peripheral eosinophilia, another important serological marker, reflects the presence of an ongoing Th2-driven immune response that is responsible for airway inflammation and fibrosis. More importantly, cytokines such as interleukin (IL-4) and IL-5 are recognized to induce eosinophilic inflammation and further worsen lung injury.[4]

The severity of bronchiectasis in ABPA is extremely variable between individuals and depends on the severity of airway obstruction, mucous plugging, and the frequency of infection.[5] Scoring systems using HRCT assess bronchial dilatation, bronchial wall thickening, and mucous plugging to quantify the burden of disease. More recent studies have indicated that greater levels of IgE and eosinophils may correlate with more severe bronchiectatic changes, but the exact correlation between serological markers and HRCT abnormalities is not well-defined.[6]

A high correlation would potentially enable patients to be stratified according to disease severity to receive personalized treatment strategies, including corticosteroids, antifungal drugs, and IL-4/IL-5 pathway-targeted biologics.[7] Identification of important serological predictors for severe bronchiectasis may also enable earlier intervention, reducing long-term morbidities such as pulmonary fibrosis and respiratory failure.

Aims and objectives

To correlate allergic serological markers (AEC, total IgE, and specific IgE for *A. fumigatus*) with the severity of bronchiectasis in HRCT chest using a bronchiectasis score in patients with ABPA.

MATERIALS AND METHODS

This retrospective observational study included data from 50 patients at SRM Medical College, Hospital, and Research Centre, Kattankulathur, from June 2022 to June 2024.

The medical records of patients diagnosed with ABPA according to the International Society for Human and Animal Mycology (ISHAM) criteria (Table 1) at SRM Medical College Hospital and Research Centre who met the inclusion criteria were reviewed for this study. Data on clinical symptoms such as cough, expectoration, and hemoptysis were collected. The patient's past medical history, including a history of bronchial asthma or obstructive airway disease, allergies, previous respiratory admissions, and comorbidities, were collected from discharge summaries. Details such as smoking (measured in pack-years), atopy/allergy, and a family history of asthma were also recorded. Treatment history included inhaler use (inhaled corticosteroids [ICS]) and anti-tuberculosis therapy.

Laboratory data on absolute eosinophil count (AEC), serum total IgE levels, and specific IgE for *A. fumigatus* were recorded accordingly for all study subjects. HRCT chest reports were retrieved from radiology records. Images were analyzed for the presence and degree of bronchiectasis, the number of bronchial segments involved, axial distribution,

Table 1: Revised International Society for Human and Animal Mycology-ABPA Working Group consensus criteria for diagnosing allergic bronchopulmonary aspergillosis

Predisposing conditions (asthma, cystic fibrosis, chronic obstructive lung disease, bronchiectasis) or compatible clinical-radiological presentation
Essential components
A. fumigatus-specific IgE ≥0.35 kUA·L
Serum total IgE ≥500 IU·mL
Other components (any two)
Positive IgG against *A. fumigatus*
Blood eosinophil count ≥500 cells·µL (could be historical)
Thin-section chest computed tomography consistent with ABPA (bronchiectasis, mucus plugging, and high-attenuation mucus) or fleeting opacities on chest radiograph consistent with ABPA
Important considerations
Expectoration of mucus plugs, finger-in-glove, and fleeting opacities on chest radiograph, lung collapse, and others
A positive type 1 skin test is acceptable when *Aspergillus*-IgE is unavailable
Serum total IgE <500 IU·mL may be acceptable if all other criteria are fulfilled
A. fumigatus-specific IgG can be detected using lateral flow assays or enzyme immunoassays. The cutoffs for *A. fumigatus*-specific IgG must be developed for specific populations (e.g., ≥27, ≥60, and ≥40 mgA·L for India, Japan, and the UK, respectively). In the absence of population-specific cutoffs, we suggest using manufacturer recommendations
High-attenuation mucus is pathognomonic of ABPA and confirms ABPA diagnosis even if all other criteria are not fulfilled
Elevated IgE against rAsp f1, f2, and f4 supports the diagnosis of ABPA and could be used as another component for diagnosing ABPA.

ABPA: Allergic bronchopulmonary aspergillosis, IgE: Immunoglobulin E, IgG: Immunoglobulin G, *A. fumigatus*: *Aspergillus fumigatus*; rAsp: Recombinant *A. fumigatus*

Table 2: Bronchiectasis score

Category	o	1	2	3
Severity of bronchiectasis	Absent	Mild (luminal diameter greater than the adjacent vessel)	Moderate (luminal diameter two to three times the diameter of the adjacent vessel)	Severe (luminal diameter more than 3 times the diameter of the adjacent vessel)
Extent of bronchiectasis	Absent	1–5 segments	6–9 segments	>9 segments
Axial distribution	Absent	Inner third	Up to the middle third	In to outer third
Bronchial wall thickening	Absent	Present more than 0.5×compared to adjacent pulmonary artery		

and bronchial wall thickening. Using these parameters, a bronchiectasis score was calculated (Table 2).

After calculating the bronchiectasis score (Table 2) for all the HRCT chest scans of study subjects, the score was correlated to the following parameters:

1. Asthma severity (according to Global Initiative for Asthma guidelines)
2. AEC levels
3. Serum total IgE
4. Specific IgE for *A. fumigatus*.

Inclusion criteria

Patients diagnosed with ABPA according to the modified ISHAM criteria (Table 1) and who had undergone HRCT chest at SRM Medical College Hospital, whether as inpatients or outpatients, were included in the study.

Exclusion criteria

Patients with a history of extensive pulmonary tuberculosis (with the possibility of post-tuberculosis [PTB] sequelae) and who had evidence of active PTB infection and those who had other possible causes for bronchiectasis like chronic aspiration, etc., were also excluded from the study. All those patients who had any concomitant medical condition that could affect total IgE level were also excluded from the study.

Statistical analysis

The data were put into Microsoft Excel and analyzed with the Statistical Packages for the Social Sciences version 25.0. Categorical variables were presented as frequencies and percentages, while continuous variables were described using the mean and standard deviation. Pearson's Correlation Coefficient was employed to assess the strength and direction of linear relationships between bronchiectasis scores and continuous variables. A $P<0.05$ was considered statistically significant.

RESULTS

Most patients (36%) were 30 years old or younger, followed by 26% in the 41–50 age group, 22% in the 31–40 age group, and 16% older than 50 years. There were more males (58%) than females (42%). Cough was the most common symptom, reported by 96% of the patients. Phlegm production was observed in 38% of patients. Haemoptysis was seen in 8% of patients. Regarding asthma severity, 42% had mild persistent asthma, followed by mild intermittent asthma in 36%, moderate persistent in 16%, and severe persistent asthma in 6% (Table 3).

The mean AEC was 983.96 ± 423.75 cells/μL, and the mean total IgE level was 2328.84 ± 1969.79 IU/mL, showing high variability between participants. The mean specific IgE level was 4.3444 ± 8.15 kUA/L, indicating a significant variation (Table 4).

Asthma severity had the strongest correlation ($r=0.807$) with the bronchiectasis score, and AEC showed a moderate positive correlation ($r=0.473$) with the bronchiectasis score. Total IgE and specific IgE levels also had moderate positive correlations with bronchiectasis scores ($r=0.447$, $r=0.504$), and all $P<0.05$, indicating significant correlations (Table 5 and Figures 1-4).

DISCUSSION

Our study shows the correlation between the bronchiectasis severity score and multiple variables. It was observed that asthma severity had the strongest correlation ($r=0.807$) with bronchiectasis score, indicating that patients with more severe asthma tend to have worse bronchiectasis and that asthma could exacerbate bronchiectasis progression. AEC showed a moderate positive correlation ($r=0.473$), suggesting that eosinophilic inflammation might be involved in disease progression. Total IgE and specific IgE levels also had moderate positive correlations with bronchiectasis score ($r=0.447$, $r=0.504$), respectively, reinforcing the role of allergic inflammation in bronchiectasis progression. Since all $P<0.05$, these correlations are statistically significant, meaning they are unlikely to be due to chance.

Our results align with those of Neyaz et al., who reported a significant positive association between asthma and

Table 3: Distribution of demographic and clinical characteristics

Patient characteristics	n (%)
Age (years)	
≤30	18 (36)
31–40	11 (22)
41–50	13 (26)
> 50	8 (16)
Gender	
Male	29 (58)
Female	21 (42)
Cough	
Yes	48 (96)
No	2 (4)
Expectoration	
Yes	19 (38)
No	31 (62)
Hemoptysis	
Yes	4 (8)
No	46 (92)
Severity of asthma	
Mild intermittent	18 (36)
Mild persistent	21 (42)
Moderate persistent	8 (16)
Severity of asthma	3 (6)

Table 4: Mean values of markers

Parameter	Mean±SD
Absolute eosinophil count	983.96±423.75
Serum total IgE	2328.84±1969.79
Specific IgE for *Aspergillus fumigatus*	4.3444±8.15

IgE: Immunoglobulin E

Table 5: Correlation between bronchiectasis score and multiple variables

Parameter	Correlation coefficient (r)	P-value
Asthma severity	0.807	<0.0001
Absolute eosinophil count	0.473	0.005
Total IgE	0.447	0.001
Specific IgE	0.504	0.002

IgE: Immunoglobulin E

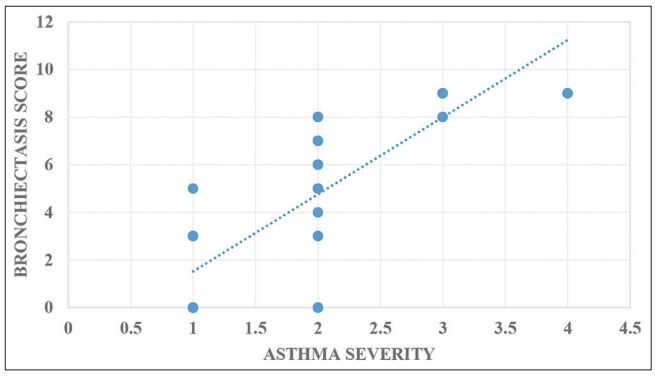

Figure 1: Correlation between asthma severity and bronchiectasis score. (where, 1 - mild intermittent, 2 - mild persistent, 3 - moderate persistent and 4 - severe persistent)

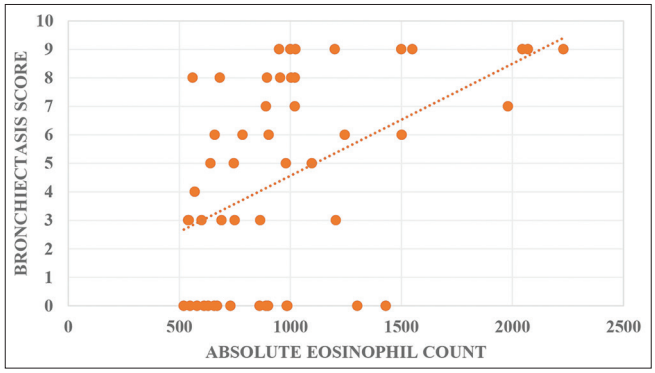

Figure 2: Correlation between absolute eosinophil count and bronchiectasis score

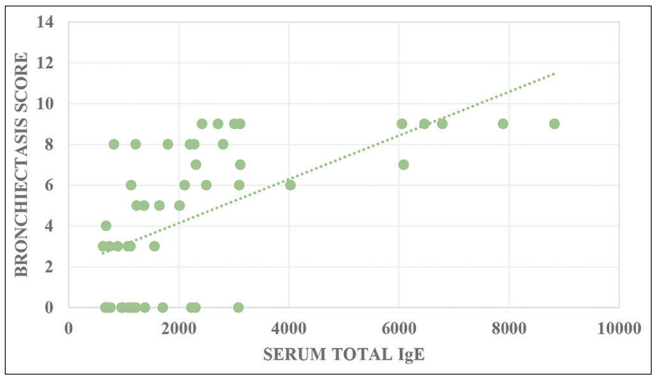

Figure 3: Correlation between serum total immunoglobulin E and bronchiectasis score

bronchiectasis severity on HRCT. Their study also demonstrated a strong relationship between specific IgE levels and bronchiectasis extent, further supporting the link between allergic sensitisation and airway structural changes.[8] However, our study expands upon these findings by providing a more detailed quantitative assessment of serological markers, including AEC and total IgE levels, emphasising their role in disease progression.

Similarly, Ren et al., reported that higher total IgE and eosinophil counts were associated with more extensive bronchiectasis, higher Smith and Bhalla scores, and increased mucous plugging. They also identified a correlation between fractional exhaled nitric oxide (FeNO) and allergic inflammation, further underscoring the role of type 2 immunity in ABPA-related bronchiectasis.[9] Although our study did not assess FeNO levels, our findings support these conclusions by demonstrating a strong association between AEC and bronchiectasis severity.

We found a significant correlation between *A. fumigatus*-specific IgE and bronchiectasis severity, supporting the role of fungal sensitization in airway damage. Agarwal et al., evaluated the diagnostic performance of various tests

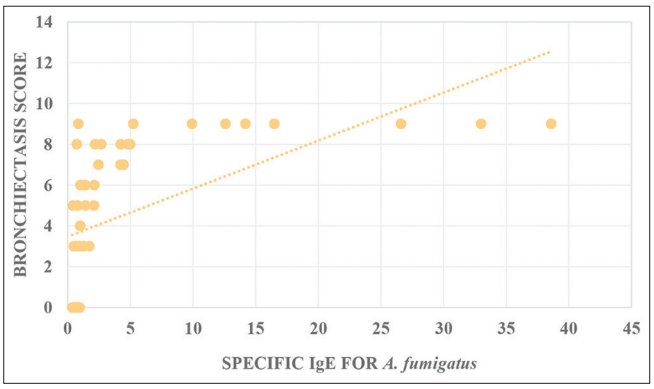

Figure 4: Correlation between specific immunoglobulin E for *Aspergillus fumigatus* and bronchiectasis score

for ABPA and emphasized the significance of *Aspergillus*-specific IgE and eosinophil counts. They described *A. fumigatus*-specific IgE >0.35 kUA/L as having a sensitivity of 100% and a specificity of 69.3% for the diagnosis of ABPA.[10] Agarwal et al., presented additional evidence for the role of immunological markers in the diagnosis and classification of ABPA, including the significance of *Aspergillus*-specific IgE and high-attenuation mucus.[11]

Sehgal et al., reported that severe or extensive bronchiectasis in ABPA was associated with decreased lung function, higher immunological markers, and more exacerbations. In their study, individuals with severe bronchiectasis had markedly high total IgE and *Aspergillus*-specific IgE levels. Moreover, their multivariable model revealed that extensive bronchiectasis increased the risk of ABPA exacerbation (aRR: 1.51), further elucidating the clinical significance of worsening bronchiectasis on outcomes of the disease.[12]

Similarly, Pollock et al., demonstrated that ABPA patients with *Aspergillus* sensitisation or elevated *Aspergillus*-specific IgG levels had more severe disease, worse lung function, and a higher frequency of exacerbations. Their study shows the vital role of allergic serological markers in bronchiectasis progression.[13] Kalaiyarasan et al., reported a high prevalence of ABPA among patients with asthma and found that those with ABPA had significantly higher AEC and total IgE levels than non-ABPA asthmatics.[14] Their findings emphasize the importance of early screening for ABPA in patients with asthma, particularly those with higher allergic markers, to prevent bronchiectasis progression.

Variability in the presentation of bronchiectasis in patients with ABPA has been emphasized in previous studies. Bhankhur et al., found that ABPA-associated bronchiectasis was present in only 31.4% of patients with severe asthma, suggesting that bronchiectasis is not a universal consequence of ABPA.[15]

Kalaiyarasan et al., found that central bronchiectasis on HRCT was a frequent feature of ABPA.[14] This aligns with our findings, as we observed that worsening allergic markers correlated with more severe bronchiectasis, emphasizing the role of allergic inflammation in structural lung injury.

Few studies have investigated the correlation between asthma severity, specific IgE levels with bronchiectasis severity using detailed HRCT scores, incorporating multiple parameters such as mucoid impaction, centrilobular nodules, mosaic perfusion, consolidation/atelectasis, and the presence of bullae and emphysema, along with bronchiectasis in ABPA patients. These studies found no significant correlation between specific IgE levels and HRCT scores, suggesting that HRCT findings—other than bronchiectasis—do not determine serological severity. Similarly, Agarwal et al., observed that HRCT findings, apart from central bronchiectasis and hyperattenuating mucus, likely represent the fibrotic, end-stage phase of the disease and do not influence serological severity.[10]

Limitations of the story

1. This study was conducted in a single center, limiting the generalizability of findings in broader populations
2. The sample size, though adequate for statistical analysis, may not fully capture the diverse spectrum of ABPA
3. Confounding factors such as environmental allergen exposure and prior antifungal treatments, were not explicitly controlled, which could have impacted the observed associations.

CONCLUSION

Our retrospective study demonstrated a significant correlation between allergic serological markers and bronchiectasis severity in patients with ABPA, as assessed using HRCT. Higher AEC, total IgE, and specific IgE levels for *A. fumigatus* were associated with increased bronchiectasis changes, indicating a potential role for allergic inflammation in disease progression. Asthma severity showed the strongest correlation with bronchiectasis scores, suggesting that poorly controlled asthma exacerbates structural lung damage. Early identification of high-risk patients using serological biomarkers could enable timely intervention to prevent disease progression. Corticosteroids remain the mainstay of ABPA treatment, effectively reducing inflammation and controlling exacerbations. Antifungal therapy, such as itraconazole or voriconazole, may be beneficial in reducing fungal load and inflammation, particularly in patients with recurrent exacerbations. Biologic therapies targeting type 2 inflammation, such as omalizumab (anti-IgE) and

dupilumab (anti-IL-4/IL-13), hold promise for patients with severe disease who are refractory to conventional treatment. Optimizing asthma management through ICS s and bronchodilators is also essential in reducing the risk of bronchiectasis progression.

Future recommendations

Further research is needed to refine risk stratification and establish standardized treatment algorithms integrating allergic and inflammatory markers. Prospective multicenter studies with larger cohorts should evaluate the long-term impact of targeted biological therapies and antifungal agents on bronchiectasis progression. Additionally, exploring the role of emerging biomarkers such as FeNO and periostin could provide deeper insights into disease mechanisms. Early screening for ABPA in asthmatics, particularly those with high IgE and eosinophil counts, should be emphasized to enable timely intervention and reduce the burden of irreversible lung damage. By advancing our understanding of the immunopathology of ABPA and bronchiectasis, we can develop more effective, individualized treatment strategies aimed at improving patient outcomes and preventing long-term complications such as pulmonary fibrosis.

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Authors' Contribution:
PA- Definition of intellectual content, prepared first draft of manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation and submission of article; **KB-** Literature survey, design, clinical protocol, editing, and manuscript revision; **AP-** Data collection, statistical analysis, preparation of figures; **SP-** Review manuscript, literature survey; **NJ-** Coordination and manuscript revision.

Work attributed to:
SRM Medical College Hospital and Research Institute, Kattankulathur-603203, Tamil Nadu, India.

Orcid ID:
Dr. Padamati Advytha - https://orcid.org/0009-0009-8254-2754
Dr. Karniha Balasubramanian - https://orcid.org/0009-0002-4292-4597
Dr. Ancila P - https://orcid.org/0009-0000-1549-8655
Dr. Nalini Jayanthi Nagesh - https://orcid.org/0000-0002-4672-0578

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