International Journal of Research in Medical Sciences
Shankar M et al. Int J Res Med Sci. 2023 Jan;11(1):xxx-xxx
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20223298

Original Research Article

Clinicodemographic, radiological, and immunological profiles of allergic bronchopulmonary aspergillosis in a tertiary care institute in Eastern India

Manish Shankar, Satyadeo Choubey*, Arshad Ejazi

Department of Pulmonary Medicine, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

Received: 25 November 2022
Revised: 02 December 2022
Accepted: 03 December 2022

***Correspondence:**
Dr. Satyadeo Choubey,
E-mail: chestsd@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Allergic bronchopulmonary aspergillosis (ABPA) is a type of hypersensitivity reaction to mold *Aspergillus fumigatus*, especially common in patients with bronchial asthma. However, systemic data regarding the clinicodemographic, radiological, and immunological profiles of patients is sparse, particularly from the eastern India.
Methods: This is a prospective observational study done over a year, from January 2021 to December 2021, in patients visiting the pulmonary medicine outpatient department (OPD) with symptoms similar to ABPA. Following the clinical examination, routine blood investigations, serum total immunoglobulin E (IgE) test, *Aspergillus*-specific IgE test, spirometry, and chest radiology (chest X-ray/high resolution CT scan) were done. ABPA was diagnosed using the 2013 ISHAM-ABPA working group criteria. Data were analyzed using statistical package for the social sciences (SPSS) software version 2021 against different categories of eosinophil count.
Results: A total of 99 patients, 74 male and 25 female, were diagnosed with ABPA. The mean age±standard deviation (SD) was 37.66±15.411. Most of the patients (93) were asthmatic. Chest radiology was normal in 52.5% of the cases. The absolute eosinophil count (mean±SD) was 3963.61±5333.363. The mean±SD serum IgE and *Aspergillus*-specific IgE levels were 8061.07±8374.274 and 7.5826±12.693, respectively.
Conclusions: There was no significant association between lung function abnormalities and eosinophil count. Serology variant (ABPA-S) was the most common finding among ABPA patients. A high eosinophil count was associated with a high incidence of bronchiectasis and hyper attenuated mucus (HAM), although the severity of bronchiectasis was not directly related to a higher peripheral eosinophil count. There may be some correlation between the peripheral eosinophil count and the total IgE but not between the peripheral eosinophil count and the *Aspergillus*-specific IgE.

Keywords: High attenuating mucus, Central bronchiectasis, Eosinophilia, Ige, Bronchial asthma, Allergic bronchopulmonary *Aspergillosis*

INTRODUCTION

As per a recent epidemiological study, the prevalence of bronchial asthma is increasing day by day worldwide, including in India.[1] It affects all age groups, including children.[2] One of the primary reasons for the poor control of asthma is allergic bronchopulmonary aspergillosis (ABPA). *Aspergillus fumigatus (A. fumigates)* is a ubiquitous fungal organism that can easily colonize the respiratory tract. ABPA is associated with complex immunological phenomena leading to eosinophilic inflammation, immunoglobulin E (IgE) production, mast cell degranulation, mucus production, and bronchiectasis.[3] As per various studies, its prevalence in India ranges from 6.9% to 22.3%.[4-7] This study was conducted to determine and assess the clinicodemographic, radiologic,

spirometric, and immunological profiles of the patients at the pulmonary medicine department of our institute.

METHODS

The study was conducted after approval from the ethics committee of the Indira Gandhi Institute of Medical Sciences (IGIMS), Shiekhpura, Patna, vide their letter number 1898/IEC/IGIMS/2020**.** Patients visiting the pulmonary medicine outpatient department (OPD) in IGIMS, Patna, were screened for bronchial asthma after their written consent. A total of 99 patients were included in our study conducted between January 2021 and December 2021. The study excluded pregnant patients or patients with a history of systemic steroid intake for more than five days in the preceding four weeks. After taking a detailed clinical history (such as any history of bronchial asthma, cough, hemoptysis, ATT intake, or smoking) and demographic data, patients were clinically examined for the presence of crepts, wheezing, or respiratory distress or failure. After that, they underwent routine blood biochemistry testing, a chest X-ray or high-resolution computed tomography (HRCT) of the thorax as needed, and spirometry with bronchodilator reversibility in accordance with the 2019 American Thoracic Society criteria. ABPA was diagnosed based on the 2013 ISHAM-ABPA working group criteria. The criteria included a predisposing condition of bronchial asthma, an obligatory criterion of *A. fumigates*-specific IgE level >0.35 kUA/l and total IgE level >1000 IU/ml, and at least any two of the following: peripheral blood eosinophil count >500/µl, pulmonary shadow consistent with ABPA, and presence of precipitins (IgG) against *A. fumigates*. Patients with a history and clinical examinations suggestive of bronchial asthma or patients with chronic cough (after ruling out tuberculosis (TB) or infective pneumonia) were subjected to spirometry, absolute eosinophil count, and total IgE level. The total IgE and *Aspergillus*-specific IgE levels were calculated using the ImmunoCap fluorescence enzyme immunoassay (FEIA) method with laboratory instruments at PhadiaTM, Uppsala, Sweden. The test allergen, covalently coupled to ImmunoCap, was first reacted with the specific IgE in the patient sample. After washing away the non-specific IgE, enzyme-labelled antibodies against IgE were added to form a complex. Following incubation, the unbound enzyme-anti-IgE was washed away, and the bound complex was then incubated with a developing agent. After stopping the reaction, the fluorescence of the eluate was measured. To evaluate the test results, the fluorescence from patient samples was compared to a standard curve.

Patients with a history of bronchial asthma or chronic cough with an eosinophil count >500/µl or *Aspergillus*-specific IgE more than 0.35 kUA/l were subjected to a chest X-ray. Patients who were diagnosed as being a case of ABPA and whose chest X-ray showed any opacity (such as consolidation, fleeting opacities, collapse, fibrosis, or bronchiectasis) were subjected to an HRCT scan of the thorax. In the HRCT scan, we looked for consolidation, central bronchiectasis (CB) hyperattenuating mucus (HAM), chronic pleuro-pulmonary fibrosis (CPF) and other radiological findings (ORF). On the basis of spirometry, bronchial asthma was graded as mild, moderate, or severe as follows: FEV1 >70% as mild, 50–69% as moderate, and <50% as severe.[8]

Statistical analysis was performed using statistical package for the social sciences (SPSS) software version 2021 (SPSS Inc.; IBM, USA). Descriptive analysis was done using the values of the mean with standard deviation and the median with range or number (percentage). The differences between the variables were analyzed using the Kruskal-Wallis test where required. The variables were compared using the Chi-square test for p values. Differences with a p-value lower than 0.05 were considered to be significant. A scatter plot was constructed to determine the relationship between the total eosinophil count and the serum total IgE and *Aspergillus*-specific IgE levels. A linear regression line was constructed to identify where the points were fitted using the r-square method.

RESULTS

A total of 99 patients with ABPA were included in the study conducted between January 2021 and December 2022. Of these, 74 (74.7%) were male, and 25 (25.3%) were female (Table 1). The minimum age was 10 years, and the maximum was 75 years. The mean age was 37.66, with a standard deviation (SD) of 15.411. Out of the total, 93 (93.9%) patients were asthmatic on examination. Furthermore, 58 (58.6%) patients belonged to rural areas, while the rest 41 (41.4%) patients were from urban areas, indicating that the disease is widely prevalent in villages, where it primarily remains underdiagnosed. Thirty (30.3%) of the total patients either had history of tuberculosis (TB) or were misdiagnosed as having TB. Cough was the predominant complaint in 89 (89.9%) patients. Radiology was normal in the majority of patients, likely because of the increased awareness of ABPA among physicians and earlier diagnosis of the disease when there is the least destruction in lung structures. This group of patients was designated as ABPA-S.

Out of the total patients, 52 (52.5%) were diagnosed as ABPA-S, 17 (17.2%) as ABPA-CB, two (2%) as ABPA-CB-HAM, four (4%) as ABPA-HAM, and the rest as ABPA-ORF (centrilobular nodules, consolidation, fleeting opacities, and fibrosis) (Table 2).

Spirometry was restrictive in three (3%) and normal in seven (7.1%) patients, likely due to the use of inhalers in the recent past. When the severity of obstruction was correlated with different levels of eosinophil count, no significant correlation was found between rising eosinophil count and lung function (p value=0.680). The median levels of total IgE and *Aspergillus*-specific IgE were analyzed against seven different categories of absolute eosinophil count (Table 3).

Table 1: Clinico-demographic, radiological and immunological profile.

Basic characteristics	Results (%)
Age (years) (mean±SD)	37.66±15.411
Sex	
Male	74 (74.7)
Female	25 (25.3)
Demographic profile	
Rural	58 (58.6)
Urban	41 (41.4)
Clinical parameter	
H/O bronchial asthma	93 (93.9)
H/O smoking	9 (9.1)
H/O haemoptysis	14 (14.1)
H/O ATT	30 (30.3)
Cough	89 (89.9)
Respiratory distress	44 (44.4)
Respiratory failure	9 (9.1)
Crepts	33 (33.3)
Wheezing	55 (55.6)
Spirometric findings	
Mild obstruction	26 (26.3)
Moderate obstruction	41 (41.4)
Severe obstruction	22 (22.2)
Restriction	3 (3.0)
Normal	7 (7.1)
AEC (cells/µl)	
Mean (SD)	3963.61 (5333.363)
Median (range)	1477 (20-27120)
Serum total IgE (IU/ml)	
Mean (SD)	8061.07 (8374.274)
Median (range)	5920 (803-58729)
Serum specific IgE (kUA/l)	
Mean (SD)	7.5826 (12.693)
Median (range)	0.99 (0.36-57.60)

ATT- Anti tubercular therapy, AEC- absolute eosinophil count, IgE- immunoglobulin E

Table 2: Radiological findings.

Radiological features	Frequency	Percent
Central bronchiectasis	17	17.2
Central bronchiectasis + HAM	2	2.0
Centrilobular nodules	7	7.1
Consolidation	10	10.1
Fleeting opacities	6	6.1
HAM	4	4.0
Right upper lobe fibrosis	1	1.0
Normal	52	52.5
Total	99	100.0

HAM- High attenuating mucus

There were significant differences between IgE values against different categories of eosinophil count (p value=0.001), especially in those with values of more than 5000 cells/µl. The AEC and serum total IgE values were converted into their logarithmic values, and linear regression was applied to determine the strength of their relationship, yielding an r^2 value of 0.13. This was proved by analyzing the wide distribution of the scattering of the data points around the fitted regression line, thus, showing their weak relationship (Figure 1). *Aspergillus*-specific IgE levels were not significantly different (p value=0.052) between different groups of eosinophils, as confirmed by linear regression analysis (r^2=0.04) (Figure 2).

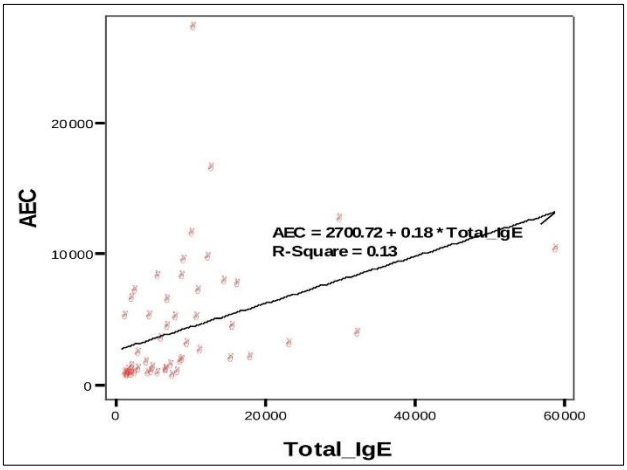

Figure 1: The strength of the relationship between AEC and serum total IgE is shown by this scatter graph by converting both into their corresponding logarithmic values and plotting them against a regression line. The r^2 value is 0.13, demonstrating the weak relationship between the two variables.

AEC- Absolute eosinophil count, IgE- immunoglobulin E

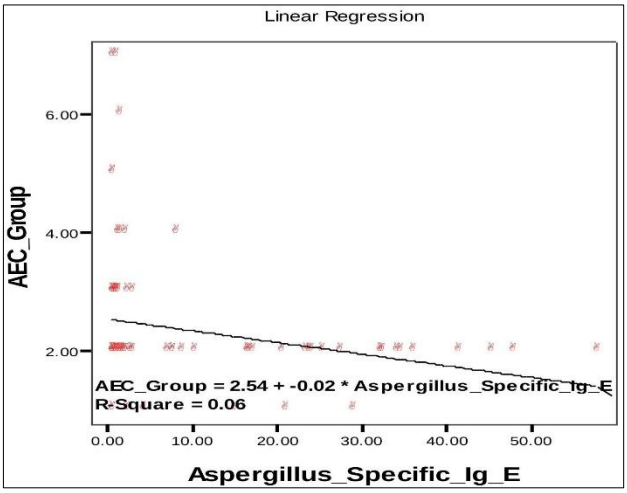

Figure 2: The strength of the relationship between AEC and serum *Aspergillus fumigatus* -specific IgE is shown by this scatter graph by converting both into their corresponding logarithmic values and plotting them against a regression line. The r^2 value is 0.06, demonstrating the weak linear correlation between the two variables.

AEC- Absolute eosinophil count, IgE- immunoglobulin E

Table 3: Spirometric, immunological findings in different categories of absolute eosinophil count.

AEC (cells/µl) n (%)=99 (100%)	<500 n=7 (7.1)	501-5000 n=65 (65.7)	5001-10000 n=18 (18.2)	10001-15000 n=4 (4)	15001-20000 n=2 (2)	20001-25000 n=1 (1)	>25000 n=2 (2)	P value
Spirometry								
Mild obstruction, n=26 (26.3%)	3 (3)	18 (18.2)	4 (4)	0	1 (1)	0	0	0.680
Moderate obstruction, n=41 (41.4%)	3 (3)	26 (26.3)	10 (10.1)	2 (2)	0	0	0	
Severe obstruction, n=22 (22.2%)	1 (1)	13 (13.1)	2 (2)	2 (2)	1 (1)	1 (1)	2 (2)	
Restriction, n=3 (3%)	0	2 (2)	1 (1)	0	0	0	0	
Normal, n=7 (7.1%)	0	6 (6.1)	1 (1)	0	0	0	0	
Immunological findings								
Total serum IgE IU/ml; median (IQR)	194 (1280 -4580	4924 (803-34865)	9872 (1100-16259)	24253 (10032-58729)	11390 (10028-12753)		10629 (10344-10914	0.001
Serum specific IgE kUA/l; median (IQR)	4.14 (0.45-28.8)	1.34 (0.36-57.6)	0.8350 (0.38-2.76)	1.56 (0.99-7.93)	0.3850 (0.37-0.40)		0.67 (0.49-0.85)	0.052

AEC- Absolute eosinophil count, IgE- immunoglobulin E

DISCUSSION

Aspergillus is a ubiquitous mold found in indoor and outdoor environments.[9] There are many species of *Aspergillus*. Among these, the most significant cause of human infections is *A. fumigatus*, which causes various lung conditions. One of the most common lung conditions is *Aspergillus*-induced TH2-mediated inflammatory response and hypersensitivity reaction, especially in patients with underlying conditions of bronchial asthma and cystic fibrosis, which lead to poor control of the disease.[3] In our study, we screened for ABPA among patients with a diagnosis of bronchial asthma visiting the pulmonary medicine OPD in our tertiary care institute. Among the previously published literature, the most common age group of ABPA mentioned in India is 20–40 years.[9] The mean age in our study was 37.66 years, which is comparable with earlier studies.

In our study, ABPA was more prevalent in males (74.7%) than in females (25.3%). This is probably because males have greater access to healthcare than females, who find it hard to access proper treatment due to poor social conditions. The disease was more prevalent in rural areas (58.6%), likely because of increased environmental exposure and sensitization to molds. Bronchial asthma patients showed a tendency toward increased colonization, sensitization, and lung structural damage caused by *Aspergillus*. A previous meta-analysis study showed the prevalence of ABPA in bronchial asthma patients ranged from 2% to 32%, with a pooled prevalence of 12.9%.[10] As we included only ABPA patients in our study, almost 93.9% of the patients were found to have bronchial asthma. Thus, bronchial asthma is the most common predisposing condition for developing ABPA. H/O smoking was seen in 9.1% of the patients, while hemoptysis was observed in 14.1% of the total. In various previous review articles, hemoptysis has been observed in 34%-85% of the cases. In one of the studies, the authors identified hemoptysis in 28.6% of the patients.[11]

Studies show that almost 50% of ABPA patients are incorrectly diagnosed as having pulmonary TB.[12] Of all the patients in our study, 30.3% had either undergone previous H/O TB treatment or were wrongly diagnosed and treated for TB. On examination of the chest, wheezing and diffuse crepts are the common findings in previous studies, but tachypnea, cyanosis, and respiratory distress and failure may also be seen.[9] It was also observed in our study.

The clinical diagnosis of ABPA requires a high degree of suspicion as the majority of the patients present with cough, sputum production, and dyspnea, thus, mimicking pneumonia.[9] Similarly, the majority of our patients presented with cough (89.9%) and respiratory distress (44.4%), while patients in advanced stages of the disease presented with respiratory failure (9.1%). On clinical examination, crepitation was present in 33.3% of the cases and wheezing in 55.6%. The most common radiological finding in our patients was a normal chest radiograph (52.5%). This was probably a result of maintaining a high index of suspicion for ABPA in each patient with bronchial asthma who visited our OPD, allowing for early detection of cases in the sensitizing stage, or ABPA-S stage, using clinical, biochemical, and radiological workup. Central bronchiectasis was found in 17.2% of the cases, HAM in 4%, and ABPA-CB-HAM in 2% of the cases that had reached the advanced stages of the disease.

Bronchiectasis, centrilobular nodules, and HAM are the main radiological features of ABPA.[13] Proximal bronchiectasis is seen in about 40% of ABPA patients.[14]

HAM occurs due to an intense inflammatory response with recurrent relapse. The lower occurrence of HAM in our study may be due to some genetic predisposition or mucus impaction and the formation of high-density mucus.[1] The presence of HAM has been linked to stronger inflammatory responses and is also associated with recurrent relapses.[15]

Other important radiological findings were consolidation, fleeting opacities, centrilobular nodules, and fibrosis in the rest of the patients.

It is still unclear whether obstructive spirometric abnormalities are due to *Aspergillus* sensitization or underlying bronchial asthma.[1] Most of our patients on spirometry had moderate obstruction (41.4%), and restriction was present in 3% of the patients, most likely due to bronchiectasis and fibrosis as a result of long-standing ABPA. Of the total, 7.1% of the patients were normal on spirometry. Although this is a relatively small amount, using inhalers or not having a history of bronchial asthma could be the cause. Obstruction was graded on spirometry against different levels of eosinophil count. It was found that there was no significant association between lung function abnormalities and the eosinophil count (p value=0.680). This is in unison with previous findings.[16]

The mean eosinophil count with SD was 3963.61±5333.363, and the median count with IQR was 1477 (20–27120) cells/µl. The distribution of the eosinophil count is shown in Table 3. Most (65.7%) patients had an eosinophil count between 500 and 5000. A count >500 is the cut-off for making a diagnosis of ABPA. Of the total, 7.1% of the patients' peripheral blood eosinophil count was <500. In ABPA patients, eosinophil accumulation is far higher in the lungs than in the peripheral blood, although there may be little correlation between the two.[17-21] This may be the reason for the low peripheral eosinophilia in some patients.

Apart from normal chest radiology, central bronchiectasis and HAM were most common in the absolute eosinophil count (AEC) group of more than 500. The 500–5000 AEC group had the greatest number of radiological changes discovered (30.3%), as the highest number of patients fell in this group. As per previous studies, patients with a high eosinophil count have a high prevalence of central bronchiectasis (CB) and HAM.[16-18]

Similarly, in our study, patients with an AEC of more than 500 were more susceptible to developing CB and HAM. However, as the eosinophil count moved to higher brackets, there was no significant increase in the prevalence of CB and HAM, indicating that the severity of bronchiectasis is not directly related to a higher (> 5000) peripheral eosinophil count.[21-22] This may also be because very few patients fell in the >5000 AEC group in this study.

The high level of IgE in ABPA is due to *A. fumigates*-induced synthesis of IL4 and IL5, which stimulates the differentiation and recruitment of eosinophils.

Previous studies did not find any correlation between peripheral eosinophilia and total and *Aspergillus* specific-IgE levels.[16-19] However, in our study, there was a significant difference between serum IgE levels in the different eosinophil groups (p value=0.001). As the eosinophil count increased, the level of serum total IgE also increased, but there was no significant difference between the rising peripheral eosinophil count and the *Aspergillus*-specific IgE level (p value=0.052). Thus, we may say that eosinophil and *Aspergillus*-specific IgE are independently related to a common source, i.e., *A. fumigates*.

Limitations

We could not include the *Aspergillus*-specific IgG antibody test in our study due to a lack of access. The test could have provided better insights for our study. The study also had a small sample size and was single-centered, which limited the clinical data available to us, causing a hindrance in the accurate representation of the demographics of ABPA patients in this region.

CONCLUSION

ABPA is a disease caused by hypersensitivity to *A. fumigates*, a species of the mold *Aspergillus*, in susceptible hosts. The most prevalent condition that can lead to ABPA is bronchial asthma. Difficult-to-treat asthma cases, in particular, need further evaluation. It may also present with pneumonia or TB-like symptoms. Thus, a high degree of suspicion is required to reach an ABPA diagnosis. In addition, the drug of choice for ABPA, gluco-corticosteroid, may cause these diseases to flare up, particularly in areas where they are endemic. Apart from clinical suspicion and chest imaging, simple blood tests like absolute eosinophil count and serum immunoglobulin E, both total and specific to *A. fumigates*, play a role in the diagnosis. If treated early, i.e., in the ABPA-S stage, a patient may be prevented from further progression to evident radiological lesions of HAM, central bronchiectasis, and eventually fibrosis. A high eosinophil count is associated with higher chances of developing radiological lesions, though they may not directly correlate with the severity of the lesion, the lung function abnormalities on spirometry, or the IgE levels.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Nath A, Khan A, Hashim Z, Patra JK. Prevalence of Aspergillus hypersensitivity and allergic

bronchopulmonary aspergillosis in patients with bronchial asthma at a tertiary care center in North India. Lung India. 2017;34:150-4.
2. Jat KR, Vaidya PC, Mathew JL, Jondhale S, Singh M. Childhood allergic bronchopulmonary aspergillosis. Lung India. 2018;35:499-507.
3. Knutsen AP, Slavin RG. Allergic bronchopulmonary aspergillosis in asthma and cystic fibrosis. Clin Dev Immunol. 2011;843763.
4. Maurya V, Gugnani HC, Sarma PU, Madan T, Shah A. Sensitization to Aspergillus antigens and occurrence of allergic bronchopulmonary aspergillosis in patients with asthma. Chest. 2005;127:1252-9.
5. Kumar R, Gaur SN. Prevalence of allergic bronchopulmonary aspergillosis in patients with bronchial asthma. Asian Pac J Allergy Immunol. 2000;18:181-5.
6. Agarwal R, Gupta D, Aggarwal AN, Saxena AK, Saikia B, Chakrabarti A, Jindal SK. Clinical significance of decline in serum IgE levels in allergic bronchopulmonary aspergillosis. Respir Med. 2010;104:204-10.
7. Ghosh T, Dey A, Biswas D, Chatterjee S, Haldar N, Maiti PK. Aspergillus hypersensitivity and allergic bronchopulmonary aspergillosis among asthma patients in eastern India. J Indian Med Assoc. 2010;108:863-5.
8. Pellegrino R, Viegi G, Brusasco V, Gustafsson P, Hankinson J, Jensen R, Johnson DC, MacIntyre N, McKay R, Miller MR, Navajas D, Pedersen OF, Wanger J. Interpretative strategies for lung function tests. Eur Respir J. 2005;26:948-68.
9. Prasad R, Kacker R, Gupta N. Allergic bronchopulmonary aspergillosis: Indian scenario. Indian J Allergy Asthma Immunol. 20192022;33:63-9.
10. Agarwal R, Aggarwal AN, Gupta D, Jindal SK. Aspergillus hypersensitivity and allergic bronchopulmonary aspergillosis in patients with bronchial asthma: systematic review and meta-analysis. Int J Tuberc Lung Dis. 2009;13:936-44.
11. Prasad R, Garg R, Sanjay, Dixit RP. Prevalence of Allergic Broncho Pulmonary Aspergillosis in Patients With Asthma Attending Allergy Clinic in a North West Indian Tertiary Care Institute. Int J Biomed Adv Res. 2016;7(5):230-4.
12. Ward S, Heyneman L, Lee MJ, Leung AN, Hansell DM, Müller NL. Accuracy of CT in the diagnosis of allergic bronchopulmonary aspergillosis in asthmatic patients. AJR Am J Roentgenol. 1999;173:937-42.
13. Kumar R. Mild, moderate, and severe forms of allergic bronchopulmonary aspergillosis: a clinical and serologic evaluation. Chest. 2003;124:890-2.
14. Agarwal R, Gupta D, Aggarwal AN, Saxena AK, Chakrabarti A, Jindal SK. Clinical significance of hyperattenuating mucoid impaction in allergic bronchopulmonary aspergillosis: an analysis of 155 patients. Chest. 2007;132:1183-90.
15. Agarwal R, Khan A, Aggarwal AN. Clinical relevance of peripheral blood eosinophil count in allergic bronchopulmonary aspergillosis. J Infect Public Health. 2011;4:235-43.
16. Murali PS, Dai G, Kumar A, Fink JN, Kurup VP. Aspergillus antigen-induced eosinophil differentiation in a murine model. Infect Immun. 1992;60:1952-6.
17. Wark PA, Saltos N, Simpson J, Slater S, Hensley MJ, Gibson PG. Induced sputum eosinophils and neutrophils and bronchiectasis severity in allergic bronchopulmonary aspergillosis. Eur Respir J. 2000;16:1095-101.
18. Kurup VP, Choi H, Murali PS, Coffman RL. IgE and eosinophil regulation in a murine model of allergic aspergillosis. J Leukoc Biol. 1994;56:593-8.
19. Agarwal R, Gupta D, Aggarwal AN. Allergic bronchopulmonary aspergillosis: lessons from 126 patients attending a chest clinic in north India. Chest. 2006;130(2):442-8.
20. Agarwal R, Khan A, Gupta D, Aggarwal AN, Saxena AK, Chakrabarti A. An alternate method of classifying allergic bronchopulmonary aspergillosis based on high-attenuation mucus. PLoS One. 2010;15:15346.
21. Agarwal R, Hazarika B, Gupta D, Aggarwal AN, Chakrabarti A, Jindal SK. Aspergillus hypersensitivity in patients with chronic obstructive pulmonary disease: COPD as a risk factor for ABPA? Med Mycol. 2010;7:988-94.
22. Agarwal R, Noel V, Aggarwal AN, Gupta D, Chakrabarti A. Clinical significance of Aspergillus sensitisation in bronchial asthma. Mycoses. 2011;54:531-8.

Cite this article as: Shankar M, Choubey S, Ejazi A. Clinicodemographic, radiological, and immunological profiles of allergic bronchopulmonary aspergillosis in a tertiary care institute in Eastern India. Int J Res Med Sci 2023;11:xxx-xx.