



Monaldi Archives for Chest Disease

eISSN 2532-5264

<https://www.monaldi-archives.org/>

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Monaldi Arch Chest Dis 2023 [Online ahead of print]

To cite this Article:

Iqbal N, Irfan M, Bin Ali Zubairi M, et al. **Allergic bronchopulmonary aspergillosis: radiological and microbiological profile of patients presented in an outpatient pulmonary clinic in a developing country.** *Monaldi Arch Chest Dis* doi: 10.4081/monaldi.2023.2803

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Allergic bronchopulmonary aspergillosis: radiological and microbiological profile of patients presented in an outpatient pulmonary clinic in a developing country

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Contributions: NI, MI, MBAZ, MA, KJ, made contributions to conception and design, interpretation of data, drafting the manuscript and revising it critically for important intellectual content; SA, contributed to the statistical analysis and interpretation of data; ABSZ, made contributions to conception and design and revised the manuscript critically for important intellectual content. All the authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: the authors declare that they have no competing interests, and all authors confirm accuracy.

Ethics approval and consent to participate: the study protocol was approved by the Ethical Review Committee of the Aga Khan University Hospital. ERC number assigned 2019-0901-2239.

Informed consent: the study is a retrospective chart review that does not require informed consent. The manuscript does not contain any individual person's data in any form.

Patient consent for publication: not applicable.

Funding: none.

Availability of data and materials: all data generated or analyzed during this study are included in this published article.

Abstract

There is limited data available about allergic bronchopulmonary aspergillosis (ABPA) in Pakistan. The aim of the study was to describe the radiological and microbiological profile of ABPA patients presenting to the outpatient pulmonary clinic of a tertiary care hospital in Karachi, Pakistan. A retrospective study was conducted on ABPA patients who presented to the pulmonary outpatient clinic at Aga Khan University Hospital, Karachi, Pakistan, from January 2017 to December 2019. Data was collected on microbiology and radiology features on predesigned proforma. A total of 7759 asthmatic patients presented at the outpatient pulmonology clinic during the study period. Of the 245 patients labeled as ABPA, 167 fulfilled the inclusion criteria, and 91 (54.5%) were female (mean age 41.9 ± 13.0 years). A high resolution computed tomography scan of the chest was available for 126 patients. Of these, 104 (82.5%) patients had bronchiectasis. Central bronchiectasis was noted in 98 (94.2%), mucus plugging in 71 (56.3%), and hyperinflation was seen in 30 (23.4%) patients. Microbiological testing was available in 103/167 (61.7%) patients. The most common bacterial pathogen was *Pseudomonas aeruginosa* 32 (31.1%), followed by *Hemophilus influenzae* 16 (15.5%), and *Moraxella catarrhalis* 7 (9.7%). *Aspergillus fumigatus* 17 (23.6%) was the most common mold, followed by *Aspergillus flavus* 16 (22.2%) and *Aspergillus niger* 11 (15.3%). Co-infection (bacterial and fungal) was found in 18 (17.45%) patients. Bronchiectasis was frequently observed in our cohort of patients with ABPA. *Pseudomonas*

aeruginosa was found to be common among bacterial pathogens. Isolation of fungus is not uncommon in these patients.

Key words: asthma, ABPA, microbiology, radiology.

Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity disease caused by an immunological reaction to the *Aspergillus* species, with *Aspergillus fumigatus* being the most commonly implicated pathogen [1]. The occurrence of ABPA is commonly seen among asthma and cystic fibrosis patients with a prevalence of 12.9% and 8.9%, respectively [1]. ABPA is generally diagnosed via a combination of clinical, serological and radiographic findings [2-4]. Previous diagnostic criteria has been modified by the international society of Human and animal mycology (ISHAM) [2-4] since APBA may be present without bronchiectasis and all criteria previously proposed are not required to establish the diagnosis of ABPA. Radiological findings of ABPA vary and include, fleeting pulmonary infiltrates, centrilobular nodules characterizing dilated and opacified bronchioles, bronchiectasis and mucoid impaction leading to bronchocoele formation (the finger in glove sign) [5]. ABPA is often misdiagnosed as tuberculosis (TB) or pneumonia, due to similar radiological presentations [6,7], leading to a considerable delay in the provision of appropriate treatment especially in a TB endemic country like Pakistan.

Clinically ABPA is divided into five stages, stage I with acute flare while stage v is advanced fibrotic disease. It is further classified into two categories, according to Patterson et al, namely ABPA-S called seropositive only in the absence of bronchiectasis and ABPA-CB (central bronchiectasis) if bronchiectasis is present. ABPA-CB is the more aggravated form of the disease [8].

A Japanese study found isolation of *Aspergillus* spp. in sputum in 59% patients, including *A. fumigatus* (33%), *A. niger* (6%), *A. terreus* (4%), unspecified *Aspergillus* spp. (16%) and *Schizophyllum commune* identified in 6% [9]. Sputum microscopy and culture was positive in 63% of patients in Indian study but overall data is limited on microbiology for both fungi

and bacterial pathogens among patients with ABPA [10]. However, the most frequently isolated organisms reported in patients with non-cystic fibrosis CF bronchiectasis are *Haemophilus influenzae*, *Pseudomonas aeruginosa*, and *Moraxella catarrhalis* while *Pseudomonas aeruginosa* was reported with more advanced bronchiectasis [11]. Another complication of ABPA is development of chronic pulmonary aspergillosis (CPA), which has an estimated burden of 411,000 patients, out of 4,837,000 ABPA patients. CPA is associated with significant morbidities, including potentially fatal hemoptysis [12].

A study estimates that, annually, 1661 cases of ABPA develop in the Pakistani population but data on radiological and microbiological profile of these patients are very limited [13]. The objective of this study is to determine the microbiological and radiographic profile of ABPA patients. We believe that an understanding of radiological and microbiological patterns among ABPA patients in a TB endemic country, may considerably improve diagnosis and prognosis in these patients.

Materials and Methods

This retrospective study was conducted on patients diagnosed to have ABPA at the adult outpatient pulmonary clinic of Aga Khan University Hospital, (AKUH) Karachi, Pakistan from January 2017 to December 2019. AKUH is the one of the largest tertiary care facilities of Karachi with 650-beds, and largest outpatient department that receives patients from wide socio-economic backgrounds from all over the city and outside from different cities of Pakistan as well. The study was approved by the Ethical Review Committee of Aga Khan University Hospital.

The patients of ABPA were initially identified through outpatient pulmonary clinic database. Medical records of all patients who were labelled as ABPA in database were reviewed. Those patients who fulfilled the inclusion criteria were then recruited for study.

Inclusion criteria

The inclusion criteria for ABPS were adopted from ISHAM 2013 [4], and included: i) age ≥ 18 years with underlying asthma; ii) serum IgE level ≥ 1000 IU/ml; iii) peripheral eosinophilia ≥ 500 cells/ μ L in steroid naïve patients; iv) radiographic pulmonary opacities

consistent with ABPA; v) a positive type I *Aspergillus* skin test (immediate cutaneous hypersensitivity to *Aspergillus* antigen); vi) active TB and other infections were ruled out by sputum microscopy, culture and Xpert MTB/Rif. We did not use *Aspergillus* skin test in all of our patients.

Exclusion criteria

i) Age <18 years; ii) patients with an incomplete record were excluded from this study.

We classified patients into five stages, stage 1 patients with acute flare, stage 2 patients who underwent remission after treatment and remain asymptomatic, stage 3 patients with recurrent exacerbations, stage 4 patients who were steroids dependent and stage 5 patients with advanced fibrotic lung disease. Sputum results were obtained during exacerbations and worsening of symptoms. Chest imaging was reviewed by two independent pulmonologists and imaging reports issued by radiologist were also reviewed. Data was collected on predesigned proforma which included demographics, co-morbid, duration of asthma and ABPA, stages of ABPA, presenting clinical symptoms, smoking status, serum IgE and eosinophilia levels and radiological and microbiology findings.

Statistical analysis

SPSS V.19.0 was used for data analysis. Descriptive statistics were done, and results are presented as mean and standard deviation for continuous variable and number and percentages for categorical variables. Number and percentages were reported of all radiological and microbiological features of patients with ABPA.

Results

A total of 7759 asthmatic patients presented at outpatient pulmonology clinic during study period, 245 (3.15%) were labeled as ABPA and 167/245(68.16%) patients fulfilled the inclusion criteria. The mean age of patients was 41.9 ± 13.0 years, and 91 (54.5%) were females. All patients had a long-standing history of asthma with a mean duration of 17.7 ± 13.1 years before they were diagnosed with ABPA. Out of 167 patients, 104 (62.3%) patients had ABPA-CB and 63/167 (37.2%) had ABPA-S. All five stages of ABPA were

observed among the patients and 87 (52.1%) had stage 3. Cough (78.4%), dyspnea (56.9%), wheezing (25.1%), chest pain (25.1%) and hemoptysis (18.6%) were the predominant presenting symptoms at clinic. All patients received systemic and inhaled corticosteroids while 135 (80.8%) patients were on itraconazole (Table 1).

The radiographic images available for these patients included chest X-ray in 167/167 (100%) and high-resolution CT (HRCT) in 126/167 (75.44%). Table 2 describes the radiological pattern of study population. Bilateral involvement on X-rays was found in 84.4%. The common X-ray findings were hyperinflation 91 (54.5%) and fleeting infiltrations 84 (50.3%). HRCT was available for 126/167 (75.44%) patients, with bilateral involvement in 92% and bronchiectasis being the most commonly observed finding present in 104/126 (82.5%) patients (Table 2).

Sputum culture results were available in 103 (61.7%) patients with bacterial growth in 66 (64.1%) of the cases. The microbiological profile is presented in (Table 3). The common pathogens identified were *Pseudomonas aeruginosa* (31.1%) and *Hemophilus influenzae* (15.5%). One or more fungi were isolated in 46 (44.6%) cases. *Aspergillus fumigatus* (23.6%) followed by *Aspergillus flavus* (22.2%) were commonly isolated fungi. . A fungal-bacterial co-infection was seen in 18 patients. Microorganisms isolation are more common in ABPA-CB (n=81/103, 78.64%) as compared to ABPA-S (n=22/103, 21.36%). Among the bacterial pathogens, a greater percentage of patients with *Pseudomonas aeruginosa* (33.3%) and *Hemophilus influenzae* (17.2%) were isolated from ABPA-CB than with ABPA-S while *Hemophilus parainfluenzae* (9%) and *Klebsiella pneumonia* (18.8%) were frequently isolated with ABPA-S.

Discussion

This is the first study from Pakistan that has highlighted radiographic and the microbiological features associated with ABPA. We have found hyperinflation on chest X-ray and central bronchiectasis on HRCT to be the most common radiological findings. Microbiologically, we found *Pseudomonas aeruginosa* to be the most common bacterial infection among ABPA-CB patients. Isolation of fungi, such as *Aspergillus fumigatus* was also seen in patients with ABPA.

In our study, more patients had ABPA-CB than ABPA-S which is in concordance with another study conducted in India where patients with more pronounced lung damage seen in the form of ABPA-CB [14]. On radiological evaluation, bilateral chest x-ray and HRCT observations were noted in 84.4% and 92.1% of the patients respectively. The most common HRCT observation was bronchiectasis which was seen in 77.8% of the patients. This observation was found to be consistent with similar studies carried out in China and India, which also reported bronchiectasis as the most common observation [15,16]. Agarwal et al. [15] reported 36.6% of patients with a normal HRCT whereas in our study none of the patients had normal HRCT this may be due to delayed diagnosis leading to advanced disease due to poor diagnostic facilities in primary care centers or referral bias of a tertiary care hospital as there is no insurance system and patients pay from their own pocket. Additionally, patients with minimal disease did not undergo HRCT chest, HRCT is performed when underlying parenchymal abnormality is suspected. We suggest early HRCT chest in all patients with ABPA. On the other hand, study from China reported central bronchiectasis as a common CT finding [16]. However, hyperinflation, consolidation and tree in bud appearance was not reported as seen in our study. The study also described the presenting clinical symptoms of cough, dyspnea, sputum, wheezing, chest pain, fever and hemoptysis in the diagnosed patients consistent with our study.

Chest X-ray findings associated with ABPA are variable, during early stages, chest X-ray images are usually normal or mimicking asthma [5]. In our study, X-ray showed fleeting infiltrations and hyperinflation to be present in more than half of the patients while lung consolidations, cavitation, fibrosis, nodules and cystic changes were also seen.

Laboratory testing showed an increase in mean serum IgE which was comparable with published data [17]. Normally a blood eosinophil level >1000 cells/mm³ is used as a diagnostic criterion [18]. However, 25% of ABPA patients are also reported to have an absolute eosinophil count of <500 cells/mm³ [17], which may possibly be attributed to the use of drugs prior to diagnosis which may have reduced blood eosinophil levels. Agarwal et al. reported that peripheral eosinophilia has limited utility in the diagnosis of ABPA [19]. In our study, we measured the differential eosinophil count which was found to have a mean value of $11.2 \pm 7.7\%$. The concept of the five stages of ABPA was first proposed by

Patterson et al [20]. The fibrotic stage represents the most advanced stage of the disease with complications and a poor disease prognosis. It has been reported by Greenberger et al. that patients frequently present in stage 3 or the exacerbation stage of the disease which is characterized by the presence of chest infiltrates on radiography [21]. Furthermore, the same study reported that the total serum IgE concentration in stage 3 patients is normally two times as high as the baseline IgE levels, while in the remainder of the stages the IgE levels may be elevated or even normal [21]. More than half (52%) of our patients were also presented in stage 3 of the disease, while 50% of the patients were seen to have fleeting infiltrates on radiography. The total mean serum IgE levels in our study were elevated most likely because the majority of the patients were in stage 3 of ABPA.

The fungal isolation in ABPA is known to act as both the infecting organism as well as an allergen, both of which are responsible for eliciting the symptoms of ABPA [22]. These allergens lead to an immune response only in already immunocompromised patients. Furthermore, fungal hyphae are responsible for the release of chemicals which further trigger the secretion of mediators such as certain interleukins and cytokines and inflammatory cells, including eosinophils [17]. *Aspergillus* species have been previously reported in other studies like Shah et al. to be a major cause for ABPA [23], however data on bacterial spectrum in ABPA patients is not available.

Limitations

A few limitations of our study included (1) the small sample size that was taken into account and the fact that the ABPA patients from only one tertiary care hospital were included which might have created a referral bias in our results. Therefore, we suggest for a large-scale study involving multiple hospitals throughout the country to be conducted. (2) HRCT chest was not done in all patients and was only done where suspicious of bronchiectasis was high so may be percentage of bronchiectasis is under reported. (3) We did not use *Aspergillus* skin test or precipitating and IgG antibodies to *Aspergillus* in all of our patients. due to limited availability.

Conclusions

Hyperinflation and fleeting infiltrates on chest X-rays and bronchiectasis on HRCT was the most common radiological finding in our ABPA patients. *Pseudomonas aeruginosa* and *A. fumigatus* was identified as a most common bacterial and fungal pathogens isolated respectively on sputum samples during exacerbation particularly with ABPA-CB. We suggest that both radiographic evaluation and microbiological profile should be identified early to reduce the extent of lung damage.

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Table 1. Characteristics of study population (n=167).

Variables	n (%)
Age, in years	41.9 ± 13.0
Gender	
Male	76 (45.5)
Female	91 (54.5)
Duration of Asthma	17.7 ± 13.1
Duration of ABPA,	4.8 ± 5.0
ABPA	
ABPA-S*	63 (37.2)
ABPA-CB*	104 (62.3)
Stage of ABPA	
1	15 (9.0)
2	39 (23.4)
3	87 (52.1)
4	24 (14.4)
5	2 (1.2)
Mean Serum IgE IU /ml	2455 ± 1600.3
Mean Eosinophil count %	11.2 ± 7.7
Smoking status	
Current	1 (0.6)
Non-smoker	159 (95.2)
Ex-smoker	7 (4.2)
Symptoms	
Cough	131 (78.4)
Dyspnea	95 (56.9)
Wheezing	42 (25.1)
Chest pain	42 (25.1)
Fever	37 (22.2)

Weight loss	36 (21.6)
Hemoptysis	31 (18.6)
Pallet of sputum	31 (18.6)
Exacerbation in 12 months	120 (71.9)
Treatment:	
Itraconazole	135 (80.8)
Systemic Steroids	167 (100)

ABPA-S, allergic bronchopulmonary aspergillosis seropositive; ABPA-CB, allergic bronchopulmonary aspergillosis central bronchiectasis.

Table 2. Radiographic features in patients with ABPA.

Variables	n (%)
Chest X-ray; n=167/167	
Unilateral	26 (15.6)
Bilateral	141 (84.4)
Consolidation	29 (17.4)
Nodules	47 (28.1)
Cavitation	22 (13.2)
Fleeting filtration	84 (50.3)
Fibrosis	16 (9.6)
Hyperinflation	91 (54.5)
Cystic changes	47 (28.1)
HRCT; n=126/167	
Unilateral	10 (7.9)
Bilateral	116 (92.1)
Bronchiectasis	104 (82.5)
Central Bronchiectasis	98/104 (77.8)
Non Central Bronchiectasis	6/104 (4.7)
Mucus plugging	71 (56.3)
Hyperinflation/emphysema	30 (23.8)
Tree in bud Appearance	12 (9.5%)
Consolidation	11 (8.7%)
Collapse	11 (8.7%)
Fibrosis	7 (5.5%)
Aspergilloma	7 (5.5%)

Table 3. Microbiological features in patients with ABPA.

Sputum culture available	103/167 (61.7%)	
<u>bacteria isolated</u>	66 (64.1)	
<i>Pseudomonas aeruginosa</i>	32 (31.1)	
<i>Haemophilus influenzae</i>	16 (15.5)	
<i>Haemophilus parainfluenzae</i>	3 (2.9)	
<i>Klebsiella pneumoniae</i>	8 (7.8)	
<i>Moraxella catarrhalis</i>	7 (9.7)	
<u>fungi isolated</u>	46 (44.6)	
<i>Aspergillus fumigatus</i>	17 (23.6)	
<i>Aspergillus niger</i>	11 (15.3)	
<i>Aspergillus flavus</i>	16 (22.2)	
<i>Aspergillus terreus</i>	2 (2.8)	
Fungal bacterial co-infection	18 (17.5)	
<u>Organisms identified</u>	<u>ABPA-S (n=22/103)</u>	<u>ABPA-CB (n=81/103)</u>
<i>Pseudomonas aeruginosa</i>	5 (22.7%)	27 (33.3%)
<i>Haemophilus influenzae</i>	2 (9.0%)	14 (17.2%)
<i>Haemophilus parainfluenzae</i>	2 (9.0%)	1 (1.2%)
<i>Klebsiella pneumoniae</i>	4 (18.8%)	4 (4.9%)
<i>Moraxella catarrhalis</i>	0	7 (8.6%)
<i>Aspergillus fumigatus</i>	1 (4.5%)	16 (19.7%)
<i>Aspergillus niger</i>	4 (18.8%)	7 (8.6%)
<i>Aspergillus flavus</i>	5 (22.7%)	11 (13.5%)
<i>Aspergillus terreus</i>	1 (4.5%)	1 (1.2%)

ABPA-S, allergic bronchopulmonary aspergillosis seropositive, ABPA-CB, allergic bronchopulmonary aspergillosis central bronchiectasis.