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Original Article

Radiology Section

Allergic Bronchopulmonary Aspergillosis (ABPA)-The High Resolution Computed Tomography (HRCT) Chest Imaging Scenario

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ABSTRACT

Introduction: Allergic bronchopulmonary aspergillosis (ABPA) is a progressive disease which can lead to recurrent exacerbations, bronchiectatic changes and end-stage fibrosis. Early diagnosis and treatment prevents its progression and alleviate its clinical manifestations. High resolution CT of the chest has emerged as a promising investigation for its diagnosis.

Aims and Objectives: To review the high resolution computed tomography (HRCT) chest manifestations in ABPA patients.

Materials and Methods: This study included 110 patients with ABPA who had undergone HRCT of the chest in the routine diagnostic workup for ABPA. The scans were assessed for changes in bronchi, parenchyma and pleura and findings consistent with ABPA were evaluated.

Results: HRCT chest was normal in 24 patients. 86 patients demonstrated central bronchiectasis with predilection for upper and middle lobes. Centrilobular nodules with or without linear opacities (tree in bud pattern), mucoceles and high-attenuation mucus were seen in 86%, 59% and 36% patients respectively.

Conclusion: Central bonchiectasis combined with centrilobular nodules and mucus impaction (especially high attenuation mucus) strongly favour the diagnosis of ABPA.

Keywords: Bronchiectasis, Mucoceles, High attenuation mucus

INTRODUCTION

Aspergillosis is a mycotic disease caused by *Aspergillus species*, usually *A fumigatus*. *Aspergillus* is a saprophytic, aerobic fungus that develops on dead or decaying organic matter and produces airborne spores that can be inhaled by man [1]. Pulmonary aspergillosis can be subdivided into five categories: (a) Aspergilloma, (b) Hypersensitivity reaction (ABPA), (c) Semi-invasive (chronic necrotizing) aspergillosis, (d) Airway-invasive aspergillosis (e) angioinvasive aspergillosis [2]. *Aspergillus*-specific IgE-mediated Type I hypersensitivity reaction and specific IgG-mediated Type III hypersensitivity reactions are believed to play an important role in the pathogenesis of ABPA [3].

ABPA was first described in 1952 from the United Kingdom by Hinson et al., [4]. Even after five decades of research this disorder is under diagnosed. In the developing countries, one-third of cases with ABPA are still misdiagnosed as pulmonary tuberculosis [5]. Though asthma is the most common contributing factor, ABPA is also seen in patients with cystic fibrosis and other underlying bronchiectatic diseases. Novey and colleagues estimated that ABPA occurs in 0.25%-11% of patients with asthma [6]. Major and minor criteria have been established for diagnosis of ABPA [7].

Patients with all five major criteria are considered to have ABPA-CB (ABPA with central bronchiectasis). These individuals should be identified early in the course of disease to mitigate and prevent the repetitive infections that may exacerbate the central bronchiectasis observed in this disorder. The presence of all criteria in the absence of central bronchiectasis suggests a diagnosis of ABPA-S (seropositive ABPA) [8]. Minor criteria, while not required, are often supportive of the diagnosis.

Mucoid impaction of underlying bronchiectatic airway is a characteristic finding in ABPA and typically occurs distal to the diseased central airways. Tubular branching opacities extend from the hilum and form a “gloved-finger” appearance. Mucoid impactions may be associated with distal atelectasis or air trapping [7]. Pleural involvement is less common but can occur, including effusions, pleural thickening, and calcifications. Fibrotic changes and even end-stage fibrosis may develop.

Radiological investigations are used to establish the initial diagnosis of ABPA and to assess the pathologic sequel at different stages of the disease. Diagnosis of APBA is frequently missed on chest radiographs. The chest radiograph is normal in almost 50% of the cases [9]. HRCT of the chest allows better assessment of the pattern and distribution of bronchiectasis and of other abnormalities that are not apparent on chest radiography. HRCT appearance of the parenchymal abnormalities reflects the macroscopic pathologic findings [10].

OBJECTIVES

The objective of our present study was to review the high HRCT chest manifestations in patients with ABPA and to reiterate the importance of HRCT chest in diagnosis of ABPA.

MATERIALS AND METHODS

A total number of 1,370 patients of asthma were referred to our department from the TB, Chest and Medicine Departments of our tertiary care teaching hospital from July 2007 to June 2013 for HRCT chest. An informed consent was taken from all patients. The study was approved by the Ethics Committee of our Medical college.

HRCT of the chest was performed on a single row (GE CT/e) and 16-row, multiple detector CT scanner (Siemens Somatom Emotion, Germany) with a 512 matrix size. The scan parameters were-130 kilovolts; 100 mAs; window width-1200 Hounsfield units (HU); and window level-600 HU. The scans were obtained in the supine position at full end-inspiration from lung apex to base. The image acquisition was spaced and the images were reconstructed (0.7mm) using the high-spatial-frequency algorithm. Prone scans were taken in ten patients in whom aspergilloma was suspected in bronchiectatic cavities. The scans were analyzed at the lobar as well as segmental level. Right lung was divided into 10 segments, and left into eight segments. Bronchial, parenchymal, pleural and mediastinal abnormalities were evaluated.

Bronchial changes-Central or peripheral bronchiectasis with lobar predominance was evaluated. On HRCT of the chest, a bronchus is considered to be dilated if the broncho-arterial ratio (internal

diameter of the bronchus divided by the external diameter of its accompanying artery) is > 1 [11]. The bronchiectasis was considered to be central if it was present in medial two-thirds of lung parenchyma. Predominant type of bronchiectasis (tubular, cystic or varicose) was established. Mucoceles (opacified dilated bronchi giving gloved finger appearance) were evaluated. Presence or absence of high attenuation mucus (HAM) (70-90 HU) with attenuation higher than skeletal muscles was observed. Other bronchial abnormalities such as bronchial wall thickening, air-fluid level in dilated bronchi were looked for.

Parenchymal changes-Collapse/consolidation of lung lobes/ segments, fibrosis, centrilobular nodules, linear opacities and mosaic attenuation were evaluated. Bronchiolitis is characterized by centrilobular nodules and branching linear or nodular areas of increased attenuation ("tree-in-bud" pattern).

Patients were then evaluated for immediate skin reactivity to *Aspergillus*, total eosinophil count, total serum IgE levels, IgE levels specific for aspergillosis and were classified as having ABPA if they fulfilled the criteria in the [Table/Fig-1].

Patients were classified radiologically as ABPA-S, ABPA-CB and ABPA-CB-HAM based on the presence or absence of CB and HAM.

OBSERVATIONS

It was observed that 110 patients of asthma fulfilled the criteria for ABPA.

ABPA had predominance in female patients with 68% female and 32 % male patients. The youngest patient was 10-years and the oldest patient was 63-years.

The mean eosinophil count of patients was 620/mm^3.

The serum IgE levels in observed patients were > 380 IU/L (SRL/ Ranbaxy). The IgE levels specific for aspergillosis were > 0.1 KUA (SRL/Ranbaxy).

Major criteria
History of asthma (regardless of severity)
Central (proximal bronchiectasis on chest radiographs)
Immediate skin reactivity to *Aspergillus*
Elevated total serum IgE (> 1000ng/ml)
Elevated IgE or IgG to *Aspergillus*
Minor criteria
Serum eosinophilia > 500/ mm 3
Precipitating antibodies to *A fumigatus*
Pulmonary opacities/infiltrates
Mucus plugging
Broncholiths
Bronchial culture positive for *Aspergillus*

[Table/Fig-1]: Diagnostic criteria for Allergic Bronchopulmonary Aspergillosis [7]

A total of 1,980 segments of lungs were evaluated for bronchial and parenchymal changes on HRCT of the chest and the imaging findings were reviewed [Table/Fig-2].

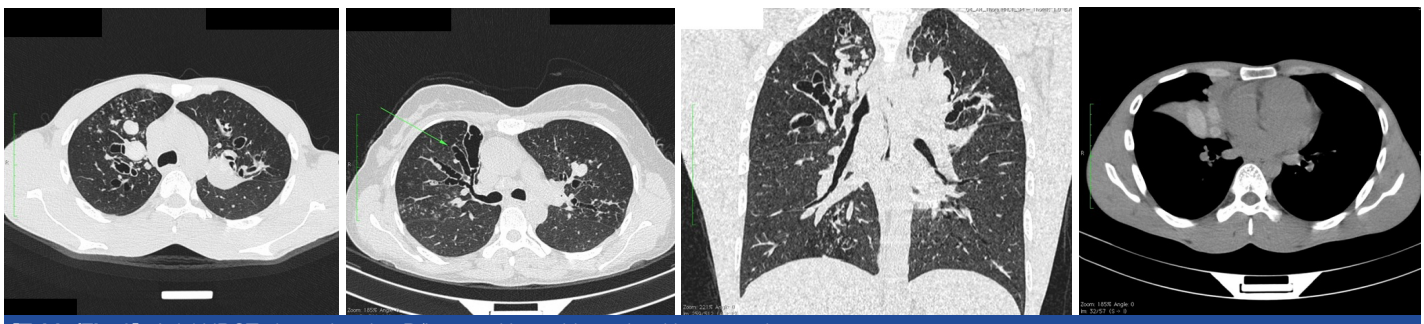
HRCT chest was normal in 24 patients and bronchiectasis (cystic, tubular or varicose) was observed in 86 patients. Central bronchiectasis [Table/Fig-3] was seen in 1,554 segments. Peripheral along with central bronchiectasis was observed in 30 patients in 115 segments [Table/Fig-4]. Centrilobular nodules with branching linear opacities [Table/Fig-5], mucoid impaction, High attenuation mucus (HAM) in the dilated bronchi [Table/Fig-6] and mosaic attenuation suggesting concomitant small airway disease [Table/Fig-7] was seen in 95, 65,40 and 20 patients respectively. Aspergillomas, which moved to dependent positions on prone scans,were noted in dilated bronchiectatic cavities in left upper lobe in six and left lower lobe in four patients.

DISCUSSION

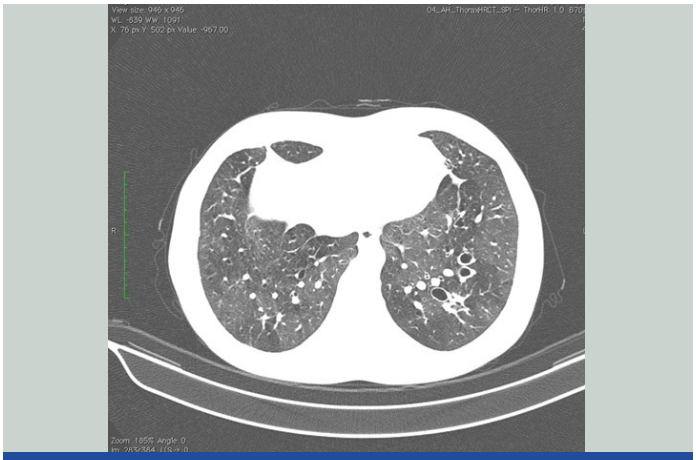
HRCT findings in ABPA consist primarily of bronchiectasis and mucoid impaction involving predominantly the segmental and sub-segmental bronchi of the upper lobes, along with centrilobular nodules or branching linear structures [12]. Mucoid impaction, which is a common finding, is filling of the dilated airways by mucoid secretions. The bronchial mucus plugging in ABPA is generally hypodense, but may also have high CT attenuation values. High-attenuation mucus (HAM) is said to be present if the mucus plug is visually denser than the para-spinal skeletal muscle. Goyal et al., were the first to describe HAM in ABPA [13]. Although this radiological diagnosis was missed for long periods before [14] and probably even after the description of this finding [15,16], numerous reports have since described the finding of HAM in ABPA [17–19]. Currently, the presence of HAM is considered pathognomonic of ABPA [20]. The reason for the hyper attenuating mucus is probably similar to allergic fungal sinusitis and is currently attributed to the presence of calcium salts and metals (the ions of iron and manganese) [21] or desiccated mucus [22]. Our study showed mucoid impaction in the

HRCT Finding	No. of Patients	%
Normal	24	21.8
Bronchiectasis Central Peripheral	 86 30	 78.2 28
Mucoceles HAM	65 40	59 36
Air-Fluid Level In Bronchus	25	23
Centrilobular Nodules	95	86
Consolidation	40	36
Aspergilloma	10	9
Mosaic Attenuation	20	18
Fibrosis	40	36
Enlarged Lymph Nodes	35	32

[Table/Fig-2]: Shows the HRCT chest findings in patients with ABPA



[Table/Fig-3]: Axial HRCT chest showing B/L central bronchiectasis with mucoceles
[Table/Fig-4]: Axial HRCT chest showing central and peripheral bronchiectasis
[Table/Fig-5]: Coronal HRCT chest image showing B/L central bronchiectasis with mucoceles and centrilobular nodules in right upper lobe
[Table/Fig-6]: Axial HRCT chest image showing high attenuation mucus (HAM) in dilated bronchus with adjacent collapsed segment of right middle lobe



[Table/Fig-7]: Axial HRCT chest image showing B/L bronchiectasis (signet ring appearance) with B/L mosaic attenuation

dilated bronchi in 59% patients. High-attenuation mucus (80–110 HU) was observed in 36% of patients with ABPA which is consistent with observation by Franquet T et al., [2] that in approximately 30% of patients, the impacted mucus is highly opaque or demonstrates frank calcification at CT.

Agarwal et al., [9] recommended that ABPA should be classified as ABPA-S (serologic), ABPA-CB (central bronchiectasis), and ABPA-CB-HAM (Central bronchiectasis-High attenuation mucus). Based on the presence or absence of HAM it should be graded as ABPA-S (mild), ABPA-CB (moderate), and ABPA-CB-HAM (severe).

Our study showed that HRCT chest may be normal in patients with ABPA which categorizes them into ABPA-S. Central bronchiectasis which is a radiologically characteristic feature of ABPA was seen in 78% patients which were then categorized into ABPA-CB and ABPA-CB-HAM. Considering Agarwal's classification, our study had 24 patients with ABPA-S, 46 patients with ABPA-CB (moderate) and 40 patients with ABPA-CB-HAM (severe).

In ABPA, centrilobular nodules are often seen as branching opacities (tree-in-bud pattern). The finding of centrilobular nodules is more common in central bronchiectasis associated with ABPA than in central bronchiectasis associated with asthma [23]. In our study, 95 patients had central bronchiectasis associated with centrilobular nodules in tree in bud pattern. Mosaic attenuation noted in 20 patients in 14 segments is due to the concomitant small airways disease.

Aspergilloma was observed in bronchiectatic cavities in 10 patients which shifted to dependent position on prone scans. The presence of aspergilloma in dilated bronchiectatic cavities has been documented [24].

HRCT chest of 40 patients in our study showed bronchiectasis, centrilobular nodules and high attenuation mucus impaction which is considered as highly suggestive of ABPA [23].

CONCLUSION

The present study showed that HRCT chest is normal in patients with ABPA-S but shows typical findings of ABPA in patients with ABPA-CB and ABPA-CB-HAM which helps it to differentiate from other pulmonary infections like tuberculosis. Central bonchiectasis combined with centrilobular nodules and mucus impaction (especially high attenuation mucus) strongly favor the diagnosis of ABPA.

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