ORIGINAL RESEARCH PAPER
Volume-9 | Issue-1 | January-2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

LEFT UPPER LOBE COLLAPSE WITH LEFT SIDED PNEUMOTHORAX (EX-VACUO): UNUSUAL PRESENTATION OF ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS.

General Medicine

Surender Kashyap Vice Chancellor, Atal Medical And Research University,himachal Pradesh

Abinav Dagar* Assistant Professor, Dept. Of Pulmonary Medicine, KCGMC, Karnal
*Corresponding Author

Sumit Bharti Senior Resident, Dept. Of Pulmonary Medicine,KCGMC,Karnal

Priya Sharma Senior Resident, Dept. Of Pulmonary Medicine,KCGMC,Karnal

ABSTRACT

Lobar or segmental collapse and secondary spontaneous pneumothorax are two unusual presentations of allergic bronchopulmonary aspergillosis (ABPA). Lobar or segmental collapse is found in acute and exacerbation phases of ABPA due to "mucoid impaction" into damaged bronchi, and secondary spontaneous pneumothorax is due to rupture of sub-pleural fibro-cystic spaces of pulmonary parenchyma, mainly in the fibrotic stage of ABPA. Clinical presentation of these two in the background of poorly controlled asthma in compliant individuals creates confusion in the diagnosis, and ABPA should be searched for. Here, we report a rare case of left upper lobe collapse and left sided pneumothorax occurring simultaneously in an 18-year-old Asthmatic Male with ABPA.

KEYWORDS

Allergic Bronchopulmonary Aspergillosis, Asthma, Oral Corticosteroids, Pneumothorax, Upper Lobe Collapse.

INTRODUCTION:

Aspergillus species is an ubiquitous fungus present in the environment causing Pulmonary diseases in healthy and immune-compromised individuals with diverse clinical presentations – Allergic Bronchopulmonary Aspergillosis (ABPA) in asthmatics, Hypersensitivity Pneumonitis in nonasthmatics, saprophytic colonization in the form of Aspergilloma in preexisting pulmonary cavities, Invasive disseminated Aspergillosis and chronic necrotizing pneumonia in immunosuppressed patients. ABPA is an immunologically mediated pulmonary disease caused mainly by immunoglobulin E (IgE)-mediated type I, but also immunoglobulin G (IgG)-mediated type III and cell mediated type IV hypersensitivity reactions are involved. Incidence of ABPA in asthmatics is around 2.5-12.9%[1,2]. Central bronchiectasis is an important radiological feature and Fleeting pulmonary infiltrates in patients with poorly controlled asthma is the main clinical presentations of ABPA. Here, we report a case of ABPA in an 18-year-old male asthmatic patient with two unusual clinical presentations namely left sided pneumothorax and left upper lobe collapse.

CASE REPORT

A 18-year-old nonsmoker male patient presented in the emergency department with the sudden increase of breathlessness and left sided pleuritic chest pain since a day. His pulse rate was 124 beats/min, respiratory rate 28 breaths/min, temperature 37°C and blood pressure 100/64 mmHg. Examination of the respiratory system revealed hyper-resonance percussion note, absent breath sound, and reduced vocal resonance on the left side suggestive of left sided pneumothorax. Diffuse wheezes were audible on the right side, along with few crackles. Chest Radiograph(Figure 1) was obtained after admitting the patient and it confirmed the presence of left sided pneumothorax and collapse of left upper lobe. Soon after intercostal drainage tube was inserted and patient improved symptomatically . Repeat Chest Radiograph showed Partial Resolution of Pneum othorax along with collapse of left upper lobe. Air column movement was present in ICD and Airleak was absent. On further enquiry patient revealed history of intermittent breathlessness, cough and wheezing since childhood, mostly in the early morning. Episodic increase in respiratory symptoms mainly just before the winter was noticed by the patient, but unlike the present one, all were sub-acute in onset. The patient had history of allergic rhino-conjunctivitis and family history of atopy or asthma was absent. Complete hemogram revealed an elevated absolute eosinophil count which was 1264/cumm. In view of the childhood history suggestive of Asthma Total IgE , specific IgE for Aspergillus and HRCT thorax was done. Total IgE was found to be 3094 IU/L, specific IgE for Aspergillus Fumigatus was 1.66 KU and HRCT thorax(Figure 2,3) was revealed central bronchiectasis, Left Pneumothorax and multiple mucus impactions (high attenuated mucus plugs). In view of lung remaining collapsed after ICD insertion a bronchoscopy was performed which revealed excessive endobr onchial thick secretion in left upper and lingular lobes. A thick mucous plug was found in left upper lobe completely blocking the opening of the bronchus.N-Acetyl Cysteine was instilled locally at the site of mucus plug through the bronchoscope and then bronchoscopy brush was used to remove the mucus by applying suction. After the procedure repeat Chest Radiograph showed complete expansion of the collapsed left upper lobe and pneumothorax was completely resolved . Subsequently Intercostal drainage tube was removed(Fig 3) During hospital stay The patient was treated with oxygen, nebulized bronchodilators and antibiotics. Spirometry showed obstructive ventilatory defect. Patient was started treatment for ABPA and discharged in stable condition.

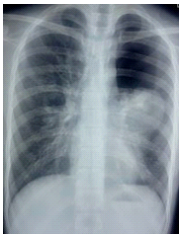

FIGURE 1

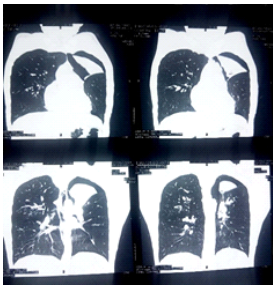

FIGURE 2

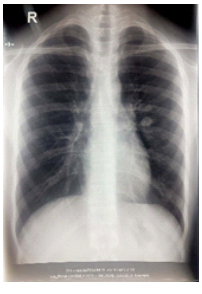

FIGURE 3

DISCUSSION

ABPA predominantly occurs in asthmatics, and also in patients with cystic fibrosis. Hinson et al. first described ABPA in asthmatics in 1952[3] It is a hypersensitivity reaction to the antigen of the fungus Aspergillus, most commonly A. fumigatus, that occurs mainly in atopic individuals. Diversity in clinical presentations results in a dilemma in the diagnosis of ABPA, and also delays the diagnosis. It should be suspected in patients of uncontrolled asthma inspite of possible optimal management. A study by Diwakar et al. showed that cough and dyspnea are the most common symptoms (99%), Expectoration is seen in 98% patients, wheezes in 97%, and hemoptysis in 47% patients . Fever is found in 80% cases, and nasal symptoms such as rhinorrhea, nasal congestion, sneezing is seen in 45% patients.[4] Radiological features of ABPA include fleeting pulmonary infiltrates, consolidation, "toothpaste" shadows and gloved-finger shadows due to "mucoid impaction" in dilated bronchi, central bronchiectasis, and upper zone fibrosis.[5] Segmental or lobar collapse is rarely seen in ABPA and pneumothorax is an unusual presentation of ABPA.[5] Diagnostic criteria of ABPA are as follows. Obligatory criteria being presence of Asthma, Elevated serum level of total IgE (>1000 IU/mL), Elevated serum level of IgE against A. fumigatus. Other criteria include Positive Aspergillus skin test (type I hypersensitivity reaction), presence of serum precipitins (IgG) against A. fumigatus, fleeting pulmonary infiltrates on chest X-ray, total eosinophil count in peripheral blood >1000 cells/mL, Central bronchiectasis on HRCT thorax. Presence of all obligatory criteria and three of five other criteria confirms the diagnosis.[6] In our case, the patient presented with uncontrolled asthma inspite of possible optimal management for it. Presence of elevated total IgE, Aspergillus – specific IgE, positive skin test to Aspergillus antigen, central bronchiectasis on CT, and peripheral blood eosinophilia confirms the diagnosis of ABPA. Besides that, two unusual clinical presentations of ABPA – pneumothorax and left upper lobe atelectasis were reported in our case. As per Patterson et al. there are five stages of ABPA: Stage I – acute, II – remission, III – exacerbation, IV – corticosteroid-dependent asthma, and V – fibrotic lung disease.[7] Cylindrical bronchiectasis of the central airways is the characteristic features of ABPA. Dilated segmental and sub-segmental airways, predominantly in upper lobes, may be occluded by impacted mucus containing Aspergillus hyphae, mainly in the acute and exacerbation stages. Complete obstruction of these affected bronchi leads to transient or persistent atelectasis of a segment or a lobe.

The walls of the affected airways are infiltrated with eosinophils, lymphocytes, and plasma cells. The airway lumen is occluded by mucus containing hyphal elements, and inflammatory cells, mainly eosinophils.Apart from central bronchiectasis, other permanent abnormality of ABPA includes fibro-cystic lesions; predominantly in the upper lobe. Although in most cases, bronchiectasis occurs proximally in ABPA patients, but bronchiectasis has been reported in the peripheral airways in some cases.[8,9] Rupture of these sub-pleural cystic spaces may result in secondary spontaneous pneumothorax.[10] Judson et al. described a case report of secondary spontaneous pneumothorax with bronchopleural fistula in ABPA, where they stated that sub-pleural cystic space may be formed due to air trapping distal to ball-valve obstruction of small airways by thick mucus dislodged from bronchiectatic segments, besides sub-pleural parenchymal fibrotic destruction.[11] Nowicka et al. have also reported left upper lobe collapse due to "mucoid impaction" in a case of ABPA.[12]

Secondary spontaneous pneumothorax due to ABPA is treated by intercostal tube drainage, antibiotic, and management of ABPA should be continued. On the other hand, segmental and lobar collapse due to "mucoid impaction" should be managed by nebulization with hypertonic saline (3%) to decrease the viscosity of mucus to ease its expectoration and urgent fibreoptic bronchoscopy should be done for removal of thick mucus plug from the segmental and sub-segmental bronchi.In our case too similar protocol was followed and thick mucus plugs were removed Bronchoscopically.

REFERENCES

1. Denning DW, Pleuvry A, Cole DC. Global burden of allergic bronchopulmonary aspergillosis with asthma and its complication chronic pulmonary aspergillosis in adults. Med Mycol 2013;51:361-70.
2. 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.
3. Hinson KF, moon aj, Plummer NS. Broncho-pulmonary aspergillosis; a review and a report of eight new cases. Thorax 1952;7:317-33.
4. Diwakar A, Panjabi C, Shah A. Allergic bronchopulmonary aspergillosis, allergic Aspergillus sinusitis and their co-occurrence. Open Allergy J 2008;1:52-61.
5. Shah A. Allergic bronchopulmonary and sinus aspergillosis: The roentgenologic spectrum. Front Biosci 2003;8:e138-46.
6. Agarwal R, Maskey D, Aggarwal AN, Saikia B, Garg M, Gupta D, et al. Diagnostic performance of various tests and criteria employed in allergic bronchopulmonary aspergillosis: A latent class analysis. PLoS One 2013;8:e61105.
7. Patterson R,Greenberger PA, Radin RC, RobertsM.Allergicbronchopulmonary asperg illosis: Staging as an aid to management. Ann Intern Med 1982;96:286-91.
8. Menzies D, Holmes L, McCumesky G, Prys-Picard C, Niven R. Aspergillus sensitization is associated with airflow limitation and bronchiectasis in severe asthma. Allergy 2011;66:679-85.
9. Agarwal R, Gupta D, Aggarwal AN, Behera D, Jinda SK. Allergic bronchopulmonary aspergillosis: Lessons from 126 patients attending achestclinic in North India. Chest 2006;130:442-8.
10. Agarwal R, Khan A, Garg M, Aggarwal AN, Gupta D. Pictorial essay: Allergic bronchopulmonary aspergillosis. Indian J Radiol Imaging 2011;21:242-52.
11. Judson MA, Marshall C, Beale G, Holt JB. Pneumothorax andbronchopleural fistula during treatment of allergicbronchopulmonary aspergillosis. South Med J 1993;8 6:1061-3.
12. Nowicka U, Wiatr E, Jakubowska L, Polubiec-Kownacka M. Allergic bronchopulmonary aspergillosis mimicking lung cancer in patients without bronchial asthma – Case report. PneumonolAlergol Pol 2012;80:77-81