

Allergic Bronchopulmonary Aspergillosis with Aspergilloma Mimicking Fibrocavitary Pulmonary Tuberculosis

Anil K Agarwal, Rajesh Bhagat, Naresh Panchal and Ashok Shah

The spectrum of *Aspergillus* associated respiratory disorders can broadly be classified into three main clinical categories, viz, allergic aspergillosis, aspergilloma and invasive aspergillosis. Allergic bronchopulmonary aspergillosis (ABPA) is the most frequently recognised manifestation of allergic aspergillosis while allergic *Aspergillus* sinusitis (AAS) is a more recently described clinicopathological entity in which mucoid impaction akin to that of ABPA occurs in the paranasal sinuses.¹ Saprobic colonization of the bronchial tree leads to the formation of an aspergilloma in cavities. These three clinical categories usually remain mutually exclusive. Although cavitation is known to occur in ABPA, frequency of coexistent aspergilloma is rather uncommon²⁻⁵ while an association of ABPA and AAS is a rarity.^{6,7} The occurrence of ABPA, AAS and aspergilloma in a single patient has been documented only once.⁸

We report a patient of ABPA with an aspergilloma whose presentation mimicked a case of fibrocavitary pulmonary tuberculosis with an aspergilloma formation. The possibility of AAS, however, re-

SUMMARY A 47-year-old male, who presented with chronic fibrocavitary pulmonary disease, had received three courses of antituberculous therapy over a period of 17 years without relief. Four years prior to referral he had developed hemoptysis and rhinitis. Evaluation of the patient led to the diagnosis of allergic bronchopulmonary aspergillosis with coexistent aspergilloma, a rather uncommon association. Both his pulmonary and nasal symptoms showed a remarkable response to treatment with oral prednisolone. However, the possibility of concomitant allergic *Aspergillus* sinusitis remained open as the patient refused to undergo any invasive procedure.

mained open as the patient had rhinitis but refused to undergo a Caldwell-Luc operation or any other invasive procedure.

CASE REPORT

A 47-year-old male office clerk who had never smoked was referred to our Institute for evaluation of progressive pulmonary disease of 17 years duration. His clinical course was characterized by productive cough and wheezing dyspnoea which had increased during the last 4 years. In addition, recurrent hemoptysis had also occurred during the preceding 4 years. The patient also reported recurrent low-grade febrile episodes associated with malaise but there was no obvious history of loss of weight. Interrogation of the patient revealed an occasional passage of

brownish plugs along with sputum. A 4-year history of rhinorrhea and nasal blockage was also elicited. During these 17 years, on the basis of his symptomatological and radiological profile, the patient had received 3 courses of antituberculous therapy without relief. He was still on antituberculous drugs when he reported to us. During this entire period his sputum stains and cultures for *Mycobacterium tuberculosis* were persistently negative.

Physical examination revealed a thin, middle-aged man in no acute distress. Trachea was shifted to right with slight flattening of the right

From the Department of Clinical Research, V Patel Chest Institute, University of Delhi, PO-Box No. 2101, Delhi-110007, India.

Correspondence : Ashok Shah.

anterior chest. On auscultation, coarse crepitations were audible in both the left and right anterior chest along with bilateral polyphonic rhonchi. Low-pitched bronchial breathing was heard in left anterior upper chest. There was no cyanosis or clubbing. Nasal mucosa was erythematous with thick purulent secretions. Maxillary sinus tenderness was also present. Examination of other systems did not reveal any abnormality.

Laboratory evaluation showed a total leukocyte count of 8,200 cells/mm³ with 6% eosinophils. Several sputum stains and cultures were negative for *M. tuberculosis* and other pyogenic organisms. Spirometry was suggestive of severe impairment of lung functions.

A review of serial chest roentgenograms of the last 4 years revealed transient pulmonary infiltrates which culminated in marked fibrosis of the right upper lobe along with cavity formation in the left upper lobe within which, a well-defined density was also seen (Fig. 1). The mass within the cavity also demonstrated positional changes. A linear tomogram of the chest confirmed the presence of a fungal sphere in the cavity along with bilateral central bronchiectasis (Fig. 2). An X-ray of the paranasal sinuses showed bilateral haziness of the maxillary sinuses. Intradermal challenge with antigens of *Aspergillus fumigatus* elicited strong Type I and Type III hypersensitivity reactions while gel-diffusion studies detected strong bands of serum precipitins against *A. fumigatus*. Culture of sputum samples for pathogenic fungi repeatedly yielded *A. fumigatus*.

A diagnosis of ABPA with concomitant aspergilloma was established on the basis of 1) history of asthma, 2) history of occasional passage of brownish plugs along with sputum, 3) transient pulmonary infiltrates on review of

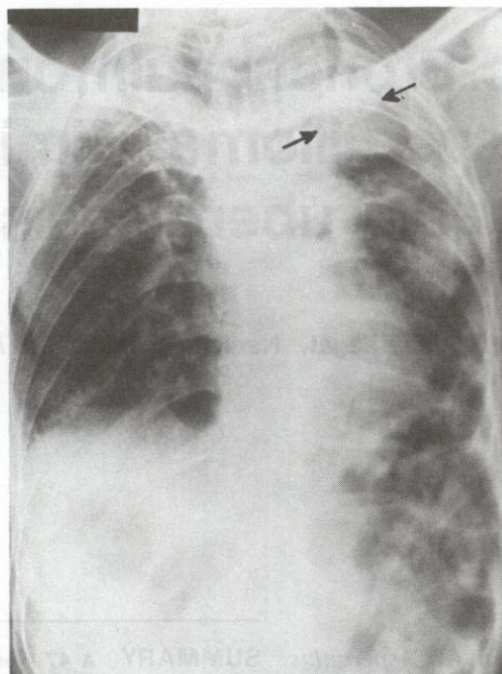


Fig. 1 Posteroanterior (PA) chest roentgenogram showing bilateral pulmonary infiltrates and marked fibrosis of the right upper lobe along with an aspergilloma in the left upper lobe cavity.

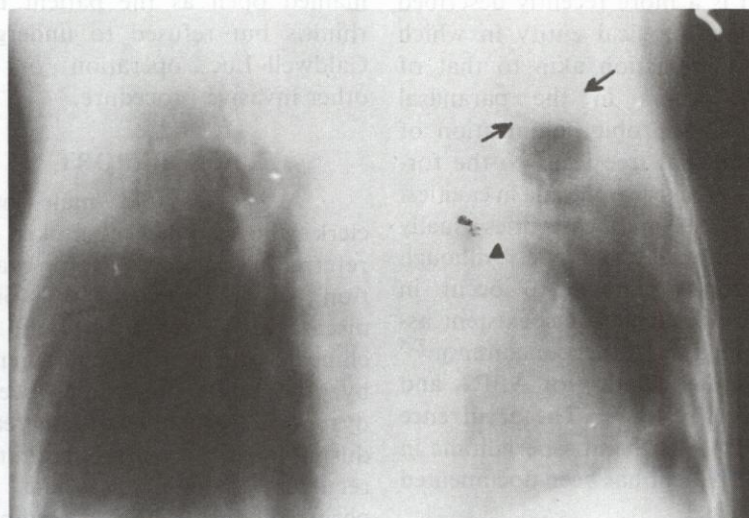


Fig. 2 Linear tomogram of the chest showing left upper lobe aspergilloma with central bronchiectasis.

previous chest roentgenograms, 4) fungal ball within the left upper lobe cavity on tomogram which also showed positional changes, 5) bilateral central bronchiectasis on tomography, 6) sputum culture positive for *A. fumigatus*, 7) bands of precipitins against *A. fumigatus*, 8) Type I and Type III hypersensitivity to intradermal challenge with extracts of *Aspergillus* spp.

Since the patient refused to undergo a Caldwell-Luc operation or any other invasive procedure, the possibility of coexistent AAS remained open.

Clinical course

Antituberculous drugs were stopped and the patient was initiated on oral prednisolone in the dose of 20 mg (0.5 mg/kg) once daily along with bronchodilators and other symptomatic therapy. The patient showed remarkable symptomatic improvement in 4 weeks. Both his pulmonary and nasal symptoms were relieved. Prednisolone was reduced to 20 mg on alternate days after 2 weeks and gradually tapered after 6 months. There was no further hemoptysis. Repeated sputum cultures for fungi and serum precipitins remained negative. Thereafter, the patient was lost to follow-up.

DISCUSSION

Initially though to be rare in India, ABPA is now recognized as an important emerging disorder.⁹ It is characterized by repeated episodes of exacerbations interspersed with periods of remission culminating, if untreated, in fibrotic lung disease which can resemble the chronic fibrocavitary disease of pulmonary tuberculosis. Even though chronic lung damage appears to provide a favourable environment for aspergilloma formation, aspergillomas are not frequently seen in patients with ABPA.²⁻⁵ Cavitation, however, is not a common feature of ABPA and may

occur in 30% of cases.¹⁰ In our case, since tuberculosis did not appear to be the cause of chronic fibrocavitary disease, it seems likely that chronic untreated ABPA resulted in progressive lung damage which led to development of multiple cavities in which aspergilloma formation occurred.

Another important feature in our patient was the subsequent development of nasal symptoms which were also abolished by therapy with oral prednisolone. Since the patient refused to undergo any invasive procedure, a strong suspicion of associated AAS however, remained.

Anderson *et al.*¹¹ reported a case of ABPA with bilateral apical aspergillomas which terminated in disseminated aspergillosis. They postulated that therapy with corticosteroids may have been the major predisposing factor in the dissemination of the disease. In contrast, our case showed both subjective and objective improvement with oral prednisolone which was in accordance with our earlier observations⁵ and those of Rosenberg and Greenberger⁴ who followed a patient with ABPA and aspergilloma for two years after initiation of corticosteroid therapy. However, exacerbation after a prolonged period of remission is also known.¹²

In developing countries, fibrocavitary disease along with fungal balls, as seen in our case, is often associated with inactive pulmonary tuberculosis. This has important clinical implications as these patients often receive antituberculous therapy for long periods while damage to the lungs continues to occur relentlessly. Our case highlights the fact that a high index of suspicion is required to establish a diagnosis of ABPA in a patient presenting with a fibrocavitary disease and also serves as a reminder that aspergillomas may occur with ABPA. Conservative medical management in such a case may show a remarkable response.

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