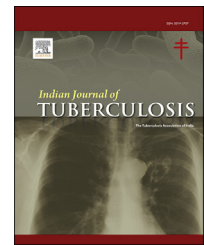


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Correspondence

A rare case of coexistence of allergic bronchopulmonary aspergillosis (ABPA) and active pulmonary tuberculosis- Role of CBNAAT in ABPA evaluation

Sir,

Allergic bronchopulmonary aspergillosis (ABPA) first described by Hinson et al in 1952 is an immune-mediated inflammatory disease caused by hypersensitivity to *Aspergillus fumigatus* that occurs predominantly in patients with underlying chronic airway disorders like bronchial asthma & cystic fibrosis [1,2]. In India pulmonary tuberculosis (PTB) is one of the most common chronic pulmonary infections. Among tubercular patients most of the reported cases of ABPA are among previously treated patients [3] and the others are cases of ABPA misdiagnosed as pulmonary tuberculosis [4]. The occurrence of ABPA in patients with active pulmonary tuberculosis has been rarely reported. Here we present a rare case of 80 years old male patient in which active pulmonary TB and ABPA were present simultaneously. This was possible only because of use of Cartridge based nucleic acid amplification test (CBNAAT) to rule out active pulmonary TB during ABPA evaluation.

A 80 year old male farmer presented to our chest clinic with complaints of cough with brownish expectoration, chest pain, and dyspnea on exertion (mMRC grade I progressing to mMRC grade III) since last 6 months. He was a non smoker and denied any past h/o antitubercular therapy, diabetes mellitus or any other significant medical illness. He also denied any drug intake, addiction or recent travel history. On examination patient was normotensive with heart rate of 96/min and respiratory rate 20/min. On chest auscultation there were bilateral diffuse rhonchi. Examination of other systems was unremarkable. A chest radiograph revealed opacity in the right lower lobe (Fig. 1). His hemogram revealed eosinophilia with absolute eosinophil count of 3500 cells/microL. A sputum sample for acid fast bacilli (AFB) was negative. His spirometry had moderate obstruction with significant post bronchodilator reversibility suggestive of bronchial asthma. Considering his clinical picture and high local prevalence of ABPA, he was further evaluated. Intradermal test with *A.fumigatus* elicited strong type I hypersensitivity reaction. The total serum Ig E and *Aspergillus fumigatus* specific Ig E levels were 9880IU/mL

(normal <295IU/mL) and 9.92kUA/L (normal <0.1kUA/L) respectively. HRCT of the thorax showed centrilobular nodules and tree in bud opacities in bilateral lower lobes (Fig. 2). Patient was thus diagnosed as a case of ABPA. As a part of routine institutional practice for all patients with abnormal chest radiology & negative sputum smear for AFB, his sputum sample was sent for Cartridge based nucleic acid amplification test (CBNAAT)/ gene Xpert analysis. The CBNAAT detected presence of rifampicin sensitive mycobacterium tuberculosis. The sputum was sent for culture which later confirmed the growth of *Mycobacterium tuberculosis*. The patient was started on antitubercular treatment with 4 drugs namely

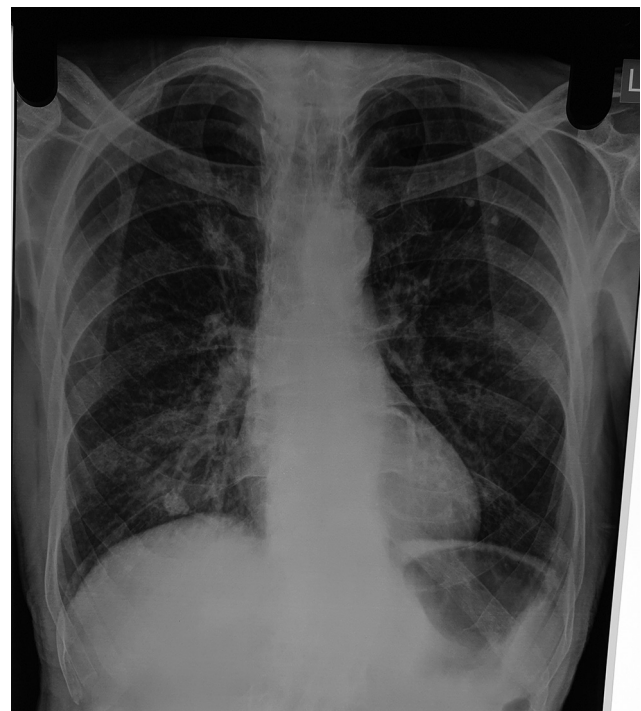


Fig. 1 – Chest X ray PA showing opacity in right lower zone.

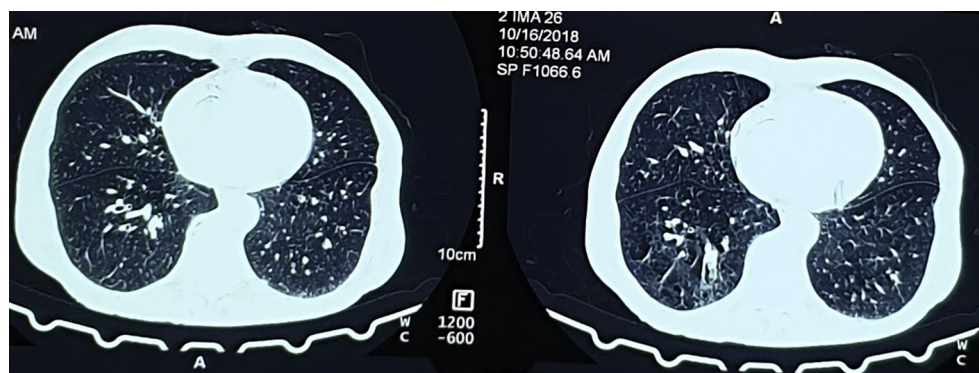


Fig. 2 – HRCT Thorax showing centrilobular nodules & tree in bud opacities in bilateral lower lobes.

isoniazid, rifampicin, pyrazinamide & ethambutol according to weight for PTB. He was also prescribed prednisolone at dose of 0.75 mg/kg for 6 weeks, 0.5 mg/kg for next 6 weeks, then tapered by 5 mg every 6 week with a plan to continue for a total duration of 9 months for ABPA. The patient showed marked clinical improvement in his condition. The total serum IgE after 3 months of treatment decreased to 3964 IU/mL and peripheral blood eosinophilia resolved. The patient is under regular follow up at our chest OPD.

This case brings into focus the importance of satisfactorily ruling out PTB while evaluating cases of ABPA. ABPA which was once considered rare is now being frequently diagnosed because of increased awareness among physicians & availability of better technology. However before starting steroids for treatment of ABPA, pulmonary tuberculosis needs to be ruled out satisfactorily. A simple sputum smear examination for AFB is not enough as it may miss paucibacillary disease. CBNAAT for MTB which was once very expensive and rarely available test that can detect very low MTB load (as low as 130–150 colony forming units per ml) especially in sputum smear negative patients, is now available free of cost at all district hospitals under RNTCP. It should be used in all ABPA cases to rule out active PTB before starting steroids to prevent dissemination of underlying PTB.

ABPA frequently complicates the disease process in bronchial asthma and cystic fibrosis patients. There are few case reports of ABPA developing in old treated PTB patients but it has been rarely reported in patients with active pulmonary TB. To our knowledge this is second case reported in which active PTB and ABPA are present concurrently. The only other case was reported by Min et al. [5]. With increasing awareness about ABPA & widespread availability of CBNAAT for MTB detection we expect more such cases will be reported in future.

To conclude, ABPA is a relatively common but under-diagnosed disease which may be coexistent with active pulmonary TB & is not restricted to old treated tubercular patients. Pulmonary tuberculosis should be carefully evaluated in patients with ABPA, particularly in high TB prevalent country like ours. CBNAAT for MTB should be used to rule out PTB in all ABPA patients before starting treatment with corticosteroids.

Conflicts of interest

The authors have none to declare

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