Allergic bronchopulmonary aspergillosis (ABPA) as a cause of middle lobe syndrome has been documented only once before [1]. Allergic Aspergillus sinusitis (AAS) in the paranasal sinuses, is also an Aspergillus-related hypersensitivity phenomenon [2], and is akin to the mucoid impaction of ABPA, but it is rarely associated with ABPA [3]. We describe a case of ABPA with co-existent AAS, in which the patient presented as a case of middle lobe syndrome.

Case history

A 55 year old male shopkeeper, a nonsmoker, was referred to our Institute for evaluation of "non-resolving consolidation of the right lung". His predominant complaints were right lower chest pain, productive cough and fever for the previous two weeks. A 12 yr history of rhinorrhoea, nasal blockage, and occasional passage of brownish plugs from the nostrils was elicited. Three years earlier, he had undergone a septoplasty for a deviated nasal septum, without relief. Over the past 2 yrs, he had experienced episodic wheezing dyspnoea and had occasionally expectorated brownish plugs along with sputum. One of his sons had rhinitis. In spite of sputum smears and cultures being negative for Mycobacterium tuberculosis, the patient had received a full course of antitubercular chemotherapy, on the basis of infiltrates present on his chest roentgenogram.

Examination revealed a febrile middle-aged man in no acute distress. There was no cyanosis or clubbing. Chest examination showed restricted movements, increased vocal fremitus, impaired percussion note, and high-pitched bronchial breathing, along with whispering pectoriloquy over the right mammary region. Bilateral rhonchi were audible. Nasal mucosa was erythematous, with thick mucopurulent secretions. Maxillary sinus tenderness was also present.

The total white blood cell count was 7.7x10^9/l with 26% eosinophils. Repeated sputum smears and cultures were negative for M. tuberculosis and other pyogenic organisms. Spirometry was suggestive of moderately severe airflow limitation.

Chest roentgenograms (figs 1 and 2) revealed a right lower zone opacity, suggestive of middle lobe syndrome. A review of four previous chest roentgenograms over the past 2 yrs revealed transient pulmonary infiltrates. A bronchographic study demonstrated bilateral central bronchiectasis with normal peripheral filling. A roentgenogram of the paranasal sinuses showed bilateral haziness of the maxillary sinuses.

On fibrebronchoscopy a narrowed but patent right middle lobe bronchus with profuse secretions was visualized. Cultures of the bronchial aspirate yielded no organisms.

Intradural challenge with antigens of A. fumigatus and A. flavus elicited strong Type I and Type III hypersensitivity reactions, whilst gel-diffusion studies detected strong bands of serum precipitins against the same antigens. Histological examination of the biopsy material from the maxillary sinuses revealed an inspissated inflammatory exudate with mucin, extensive coagulative necrosis, collection of eosinophils enclosing fungal hyphae in places
and Charcot-Leyden crystals, similar to the mucoid impaction of ABPA. Culture of the pathological material from the maxillary sinuses yielded a pure and heavy growth of *A. fumigatus*.

On the basis of these findings, together with the history of asthma, occasional passage of brownish plugs from nostrils and in sputum, peripheral blood eosinophilia, transient pulmonary infiltrates, bilateral central bronchectasis, and haziness of both the maxillary sinuses, the diagnosis of ABPA with middle lobe syndrome and AAS was established.

![Chest roentgenogram posteroanterior (PA) view, showing right-sided lower zone opacity, with loss of right heart border silhouette.](image1)

**Fig. 1.**

![Chest roentgenogram, right lateral view, showing middle lobe infiltrations.](image2)

**Fig. 2.**

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**Treatment and course of disease**

Within two weeks of therapy with 30 mg of prednisolone, (0.5 mg·kg⁻¹) daily, the patient's nasal and pulmonary symptoms were abolished. Chest roentgenogram obtained one month after therapy showed a reinflated right middle lobe.

**Discussion**

Of the 933 patients with middle lobe syndrome reviewed by WAGNER and JOHNSTON [4] in 1983, ABPA was incriminated in only one case [1]. The rarity of ABPA presenting as middle lobe syndrome is perhaps surprising, as collapse due to mucoid impaction is not uncommon and, moreover, the middle lobe often collapses in isolation.

Another notable feature in our patient was the concomitant occurrence of AAS and ABPA, a rather rare association. In a recent review of 71 cases of allergic fungal sinusitis [5], concomitant occurrence of ABPA was reported in only five cases. Although our patient presented with acute manifestation of ABPA, the patient's nasal symptoms preceded his pulmonary symptoms, which suggests that AAS may have triggered the subsequent development of ABPA.

Prednisolone to which our patient had a remarkable response remains the cornerstone of treatment of ABPA and AAS, which suggests that common immunopathological processes are involved.

Our case highlights the fact that ABPA can also present as a middle lobe syndrome. Furthermore, AAS should be sought for in cases of ABPA with nasal symptoms. Early diagnosis of AAS itself could alter the course of the disease and prevent the subsequent development of associated ABPA.

**References**