

CASE REPORT

Nasal Septal Perforation in a Patient with Allergic Bronchopulmonary Aspergillosis and Rhinitis on Long Term Corticosteroids

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Symptoms of rhinitis are frequently encountered in patients with allergic bronchopulmonary aspergillosis (ABPA).¹ These patients are often prescribed intranasal corticosteroids along with oral corticosteroids, the cornerstone for the treatment of ABPA. In addition, such patients commonly receive orally inhaled corticosteroids. Intranasal corticosteroid sprays have now emerged as the gold standard in the management of allergic rhinitis (AR).² Nasal septal perforation is not a widely recognized adverse effect of intranasal corticosteroids and has rarely been described in the literature.³ We report a patient of ABPA with nasal symptoms who developed nasal septal perforation while on treatment with oral and inhaled corticosteroids along with nasal corticosteroid spray. This report was prompted by the absence of such a description in the literature.

CASE REPORT

A 22-year-old male, a non-

SUMMARY A 22-year-old male, referred to us as a case of multi-drug resistant tuberculosis was diagnosed as allergic bronchopulmonary aspergillosis (ABPA) after serological and computed tomography confirmation. He was initiated on oral as well as inhaled corticosteroids along with nasal corticosteroid spray for his nasal complaints. One year subsequently, he developed a nasal septal perforation. Biopsy taken from the site did not reveal any granulomatous or atrophic changes and cultures of the biopsy did not yield any organism. The septal defect, repaired surgically by Hazeltine's method healed completely within 6 weeks. There have been anecdotal reports of septal perforation in patients with rhinitis on intranasal corticosteroids but hitherto not in patients with ABPA. A periodic examination of the nasal septum should be undertaken in patients with ABPA and rhinitis on long term inhaled oral and intranasal corticosteroids along with oral corticosteroids.

smoker, was referred to our Institute as a suspected case of resistant pulmonary tuberculosis for evaluation. The patient had a progressive pulmonary disease characterized by a history of cough and mucoid expectoration along with frequent episodes of low grade fever, often accompanied by haemoptysis. Interrogation revealed that the patient had recurrent wheezing and dyspnoea since childhood. A history of nasal symptoms was also elicited in the form of blocked nose, runny nose and bouts of sneezing. There was no history of passage of

brownish plugs; neither in the sputum nor nasal secretions. In spite of repeated stains and cultures being negative for *Mycobacterium tuberculosis*, the patient had received several courses of anti-tubercular therapy without any relief. A review of six previous chest roentgenograms over the past 8 years

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showed transient pulmonary infiltrates. Roentgenogram of the paranasal sinuses was normal.

A diagnosis of ABPA was established on the basis of 1) history of asthma, 2) peripheral blood eosinophilia (9%), 3) transient pulmonary infiltrates seen on review of previous chest roentgenograms, 4) presence of central bronchiectasis with normal periphery on computed tomogram of thorax (Fig. 1), 5) strong bands of precipitins against *A. fumigatus* and *A. flavus*, 6) positive Type I and Type III hypersensitivity reactions to intradermal challenge with extracts of *Aspergillus* spp. 7) raised total IgE and 8) positive specific IgG and IgE to *A. fumigatus*. The possibility of concomitant allergic *Aspergillus* sinusitis was ruled out as the roentgenogram of the paranasal sinuses was normal. The patient's nasal symptoms were ascribed to associ-

ated AR. He was initiated on oral prednisone 30 mg (0.5mg/kg/day) once daily along with inhaled budesonide 400 µg twice daily and inhaled salmeterol 50 µg twice daily. Prednisone was reduced to 30 mg on alternate days after 2 weeks and gradually tapered. Within 3 weeks the patient showed remarkable response to the above therapy resulting in resolution of his pulmonary symptoms. Although his nasal symptoms had subsided, nasally inhaled corticosteroids had to be added subsequently to achieve complete control. The correct technique emphasizing the importance of directing the spray towards the lateral aspect of the nose was demonstrated and was reinforced on subsequent visits.

A year after the initiation of therapy, the patient complained of passage of a plug from the nose. Following which, he developed a

whistling sound emanating from the nose while breathing. Examination revealed a 15 x 15 mm perforation in the septum, about 1.5 cm posterior to columella (Fig. 2). Nasally inhaled corticosteroids were withdrawn immediately. Biopsy from the site did not reveal any granulomatous or atrophic changes, while cultures of the biopsy material did not yield any organism. Skin biopsy ruled out Hansen's disease as the cause of perforation. The septal perforation was repaired by modification of Hazeltine's method under local anaesthesia.⁴ The perforation healed completely within 6 weeks time.

DISCUSSION

Nasal septal perforations due to prolonged use of topical corticosteroids in patients with rhinitis have been described only anecdotally.⁵ Since local applica-



Fig. 1 Computed tomogram of thorax showing central bronchiectasis.



Fig. 2 Septal perforation (arrow) visible on nasal examination prior to surgery. A probe in the other nostril illuminating the lesion is also seen.

tion of corticosteroids may cause dermal atrophy, the possibility of mucosal atrophy by long term use of intranasal corticosteroids has been thoroughly investigated.⁶ In several studies, including chronic use of intranasal budesonide for periods up to 36 months, biopsy investigations did not show any effect on nasal mucosa.^{7,8}

Septal perforation in a patient of ABPA receiving nasally and orally inhaled steroids along with oral corticosteroids has not been documented. Certain features

distinguish our patient from the earlier reports of perforation in patients with rhinitis. In a recent review, treatment with steroids was thought to be an important risk factor in 11 of the 32 patients with nasal septal perforation.⁹ The septal defect in these patients occurred in the initial months of therapy. Nevertheless, our patient developed the perforation after more than 12 months of medication. In the earlier reports, epistaxis, crusting, dryness, itching, irritation or stinging sensation were reported frequently before the development of septal per-

foration.^{10,11,12} It is noteworthy that our patient did not have any warning symptom which could have suggested an impending perforation. The absence of some of these symptoms like irritation, crusting, itching and stinging could possibly be explained as these complaints have often been ascribed to the presence of the propellant, propylene glycol, in older formulations rather than the drug.⁶ It has also been suggested that regular antihistamine use may predispose to the development of perforation by compromising the blood supply to the nasal septum.¹³ However, our patient was not on any regular antihistamines.

Another unusual feature was the complete healing of the defect within 6 weeks of surgical repair in spite of the patient being on oral corticosteroids. In the previous reports, perforations secondary to intranasal corticosteroid sprays have been managed medically.^{5,9} Elsewhere it has been advocated that perforations where biopsy shows no specific abnormality should be observed and managed medically.¹⁴

This case serves as a reminder that the nasal septum should be periodically observed in patients with ABPA and rhinitis who are on long term intranasal corticosteroids. The effect of concomitant long term oral corticosteroids on the development and treatment of nasal septal perforation also needs to be evaluated. A surgical repair may be attempted in uncomplicated cases of perforation secondary to intranasal corticosteroids.

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