

Case Study

Aspergillus tamaraii - a Rare Cause of Nasal Polyposis

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A B S T R A C T

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Aspergillosis of head and neck region, primarily affects the nasal cavity and paranasal sinuses. There are more than 185 species of *Aspergillus* and over 95% of all infections are caused by *A. fumigatus*, *A. flavus* and *A. niger*. Any type of Aspergillosis may progress to more aggressive disease illustrating the importance of early recognition of this increasingly encountered disease. We report here a case of a 11 year old immunocompetent male patient who presented with bilateral nasal obstruction due to polyps. The biopsy tissue was sent for histopathological examination which revealed features of allergic rhinosinusitis. Wet mount preparation revealed septate fungal hyphae about 4-8 μm in diameter with branching at acute angles. A diagnosis of *A. tamaraii* was made from wet mount and repeated isolation in culture. The patient responded to surgical excision of the polyps and oral itraconazole post operatively. The isolate in our case is *Aspergillus tamaraii* which is probably the first case reported. Thus, there is a need to develop greater understanding of the pathogenesis of the disease, formulate better and more sensitive diagnostic techniques, develop superior antifungal agents and increase awareness of disease among clinicians.

Introduction

Nasal polyposis is a chronic inflammatory disease of the mucous membrane in the nose and paranasal sinuses presenting as pedunculated smooth, gelatinous, semitranslucent, round or pear shaped masses of inflamed mucosa prolapsing into the nose (Bachert *et al.*, 1998). In the general population the overall prevalence rate of nasal polyposis ranges from 1–4%. It is more common in adults than in children under 10 years of age (Boeve, 2002). The underlying mechanisms of nasal polyposis are still largely unknown.

Several hypotheses have been put forward including chronic infection, aspirin intolerance, alteration in aerodynamics with trapping of pollutants, epithelial disruptions, epithelial cell defects/ gene deletions, inhalant or food allergies. Primary symptoms of nasal polyposis are nasal blockage, congestion, hyposmia or anosmia and if associated with chronic sinusitis a purulent nasal discharge. Secondary symptoms comprise post nasal drip, rhinorrhea, facial pain, headache, sleep disturbance and lowered quality of life

(Benoliel, 2001). Diagnosis can be made by history, clinical examination, radiography, nasal endoscopy and additional tests for allergy, aspirin sensitivity, bacteriology and pulmonary function tests (Chrostowski and Pongracic, 2002). The warm, moist environment of the upper respiratory tract is an ideal environment for the proliferation of fungi (Mygind, 1990). However, they are rarely pathogenic because host resistance is high except under favourable conditions in highly susceptible individuals. According to Corey JP research, persistence of allergic fungal sinusitis with recurrence of sinonasal symptoms (with or without polyposis) is common, particularly when there has been incomplete eradication of allergic fungal mucin (Corey *et al.*, 1995). Even when the patient is clinically disease free, recurrence can occur presumably from reexposure to fungal antigens. Therefore close clinical, endoscopic, and radiographic follow-up of nasal polyposis is important (Osguthorpe and Hadley, 1999).

Case report

A 11 year old male presented with history of nasal obstruction, mouth breathing and snoring since one year. The patient gave history of scanty seropurulent, odourless nasal discharge from nasal cavity. Onset was insidious starting on left side and gradual progression to bilateral total nasal blockage. Patient subsequently developed breathing through mouth. He denied epistaxis, excessive sneezing, headache or trauma to nose. History of recurrent upper respiratory infections including fever, cough, sputum production, ear discharge, earache, tinnitus, vertigo, ataxia and asymmetry of face or facial pain were negative. He had no history of tuberculosis, diabetes mellitus, asthma, use of corticosteroids, other immunosuppressants or prolonged antibiotic therapy. On general physical examination, he was afebrile with a blood pressure of

120/80 mm of Hg and pulse rate of 92 per minute. All other vital parameters were normal. Anterior rhinoscopy revealed bilateral pale white nasal polyps. Polyps were non-tender and probe could be moved all around the growth. Nasal mucosa was normal with no bleed on touch. Bilateral nares blockage was assessed by patency test. There was no abnormal finding on posterior rhinoscopy. Irregular, tender swellings were palpable on both sides medial to medial canthus of eyes with upward extension on the upper eyelids. Ethmoidal sinus was also tender. Clinical examination of ears and throat was normal. Computed tomography (CT) scan and magnetic resonance imaging (MRI) revealed bilateral pale white nasal polyps. Fiber-optic endoscopic sinus surgery (FESS) was done and biopsy tissue sent for histopathological and microbiological studies. Hematological parameters were not suggestive of inflammatory pathology. Laboratory values were notable for haemoglobin of 12 gm%. The white blood cell count was 10300/cmm with 72% segmented neutrophils, 22% lymphocytes, 4% monocytes, 2% eosinophils and no basophils. Random blood glucose of 110 mg% and fasting level of 70 mg% were recorded. Kidney function test and liver function tests were normal. Urine did not show any protein or glucose and culture did not show any growth. Blood culture was sterile. Chest X-ray was normal. The patient was presumed to be immunocompetent as the patient was not reactive for HIV and had no diabetes mellitus, neutropenia, evidence of hematologic or any other malignancy in the body, or any concurrent infections.

Culture of the biopsy polyp tissue on Sabouraud dextrose agar (SDA) with chloramphenicol was incubated at 37°C and 25°C. On day three of incubation, few white fluffy colonies with yellow to green centre were seen on the obverse with a yellow pigment on the reverse. (Figure 1).Colonies

turned grayish green by day ten. On Czapek's agar, yellow green colonies at the first 3 days of growth, shifted to brown or brownish green after 7 days without exudates and pinkish reverse. On Lactophenol cotton blue (LPCB) wet mount, conidial heads loosely radiating with subglobose vesicles were seen (Figure 2). Strigmas were uniseriate to biseriate at maturity covering the entire surface of the vesicles. Conidia were subglobose to cylindrical with average dimensions 3.21 to 3.32 μm . Colonies on malt extract were yellow-green to olivaceous brown, with gray reverse. This isolate followed the morphological and microscopical features of *A. tamarii* (Domsch *et al.*, 2007).

Patient was started on oral itraconazole. He was reviewed for endoscopic examination after one month. Assessment revealed few bilateral small nasal polyps which were excised by sphenoidectomy, uninectomy and posterior ethmoidectomy. Biopsy was taken from this sample and sent for histopathological and microbiological studies. The fungus was identified as *Aspergillus tamarii* and confirmed by Department of Medical Mycology, PGIMER, Chandigarh.

Discussion

It is important to remember that there is no single etiological factor that is responsible for the development of nasal polyposis, but that inflammation still remains to be the central major factor in all nasal polyps. Allergy, viral infection, bacterial infection, fungal infection, and environmental pollution have all been suggested as possible initial triggers that may up regulate inflammation of the lateral wall of the nose to develop nasal polyposis (Moretz, 1987). Nasal polyposis due to *Aspergillus* may present in two different forms. The acute life threatening form is seen in

immunocompromised patients. The typical patient of allergic nasal polyposis is an immunocompetent, atopic, young adult with a long standing history of allergic rhinitis, nasal congestion, headache, polyposis, asthma and/or recurrent sinusitis. On examination, polyps may be evident with associated mucopurulent discharge, enlarged turbinates, hyperplastic nasal mucosa and occasional facial pain (Milosev, 1966).

The role of superficial and saprophyte fungus which cause imbalance in local immunity of nasal mucus are more important than invasive fungus (Mirkin, 2002). Hyper-reactivity to fungal organisms could be one of the mechanisms underlying the development of nasal polyposis according to Ricchetti study (Panda *et al.*, 2004). The nose and paranasal sinuses have local factors which promote fungal infection including nasal polyps, recurrent bacterial infections, neutropenia and chronic rhinitis with stagnation of nasal secretions.

The usual fungus isolated is *Aspergillus*; other cause being phaeoid fungi like *Bipolaris*, *Exserohilum*, *Curvularia* and *Alternaria*. The positive results of wet mounts, LPCB mounts and repeated isolation on culture confirms the diagnosis of *Aspergillus tamarii* beyond doubt in the present case. Reports of histopathology are supportive evidence. Case reports of paranasal sinus mycosis from India and Sudan have reported *A. flavus* as the cause in majority of cases. Invasive aspergillosis of the paranasal sinuses was reported in immunocompetent hosts for the first time by Milosev and others in 1966 (Ricchetti, 2002). Since then, this condition was reported mainly from the tropical areas, including the Indian subcontinent (Shah and Deokule, 2007). The isolate in the present case is *Aspergillus tamarii* which is probably the first case reported.

There is a need to develop a greater understanding of the pathogenesis of nasal polyposis, formulate better and more sensitive diagnostic techniques, develop superior antifungal agents and increase an awareness of the disease among clinicians. More important is the need to develop antifungal susceptibility testing for

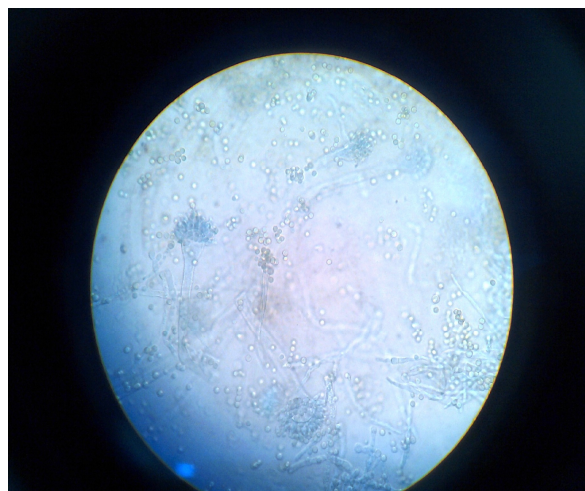
Aspergillus species and means to prevent occurrence of the disease especially in immunocompromised individuals.

Knowledge of local patterns of infection and antifungal susceptibility would prove useful in selecting empirical therapy and formulating prophylactic strategies.

Figure.1 Sabourauds dextrose agar showing growth of *A. tamarii*



Figure.2 Lactophenol cotton blue mount of culture showing hemispherical vesicle covered with biserial phialides bearing smooth conidia characteristic of *Aspergillus tamari*



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