http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v8i7.31

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Chronic Fungal Rhinosinusitis due to Aspergillosis: A Case Report

Authors
Lekshmi Vijayamohanan, Sarita Asotra*, Madhuri Dhadwal, Ankita Dheer
IGMC Shimla
*Corresponding Author
Sarita Asotra

Abstract
Aspergillosis is a ubiquitous mold causing allergic bronchopulmonary aspergillosis in healthy people and invasive disease, pneumonia and serious sinusitis in immunocompromised patients. It's characteristic morphologic features distinguish it from other fungi and aid diagnosis in an otherwise unremarkable case of nasal polyposis. We report a case of a 59 year old lady with chronic rhino sinusitis caused by Aspergillosis, where histopathology played a key role in diagnosis.
Keywords: *Aspergillus, rhinosinusitis, histopathology, Humans, Female, Middle Aged, Young Adult, Adult, Aspergillosis, Allergic Bronchopulmonary.*

Introduction

Fungal rhinosinusitis refers to a spectrum of disease encompassing invasive and non invasive forms, which can extend to invasion of the orbit and brain.

A progressive rise in incidence of fungal sinusitis has been observed in both normal and immunocompromised individuals, with Aspergillus and Zygomycetes being the commonest causative organisms. A propensity for arterial invasion and distal embolization points to the importance of a rapid and early diagnosis.[(2)]

Case History

A 59 year old lady presented to our hospital with complaints of recurrent episodes of left nasal obstruction since 4 years with increasing severity since the past 8 months, along with nasal discharge which was scanty, clear to greenish with no foul odor.

Headache, facial pain, protrusion of the eyes or other ophthalmic complaints were absent.

No history of diabetes, steroid intake, or surgery was noted to indicate immunosuppression.

On Clinical Examination: Vitals were within normal limits. Nasal examination revealed deviation of nasal septum with S shaped configuration; left fronto-nasal recess was blocked by a mass-like mucosal thickening. Concha bullosa of the right turbinate was seen. No facial disfigurement or proptosis was present; other signs of atopy were absent. Other systems: within normal limits.

Lab Investigations: Eosinophilia (13%) with absolute eosinophil count of 650. Viral markers such as HIV were negative, blood glucose was within normal range.

CT Paranasal Sinuses: mucosal thickening in left maxillary, ethmoid, sphenoid and frontal sinus were noted with blocking of the osteomeatal unit, predominantly in the maxillary and ethmoid sinus.

Patchy hyper density was seen in the left maxillary sinus due to retained secretions.

An endoscopic guided biopsy of the left nasal mass was performed under anaesthesia.

Greyish white to dark brown tissue material was removed and tissue samples were sent to our histopathology department.

A 10% KOH Mount of the tissue revealed narrow, branched septate hyphae with globose vesicles with phialides and conidia.

Gross Examination: We received multiple grey white to grey brown soft tissue pieces; measuring 1cmx1cmx1cm.

Microscopic Examination: revealed tightly packed acute branching hyphae with septation, necrosis, inflammatory exudate of neutrophils, plasma cells, lymphocytes and eosinophils; with abundant Charcot Leyden crystals, round calcification along with normal lining epithelium and mucous glands.

A PAS staining proved confirmatory; while **Gomori-methenamine silver (GMS) stain** showed septate hyphae and acute angle branching, thus a diagnosis of Chronic Fungal Rhinosinusitis (Aspergillosis) was rendered.

An SDA (Sabouraud dextrose agar) culture & LCB (Lactose Cotton Phenol Blue) mount: provided affirmation of the same.

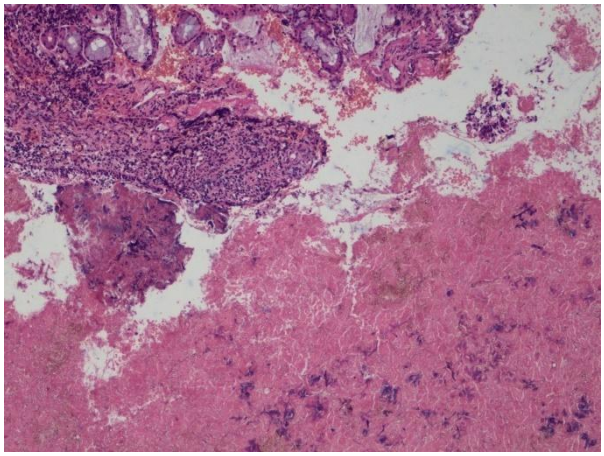

Fig 1 Photomicrograph showing normal lining mucosa, mucus glands with fungal mass. 100x[H&E]

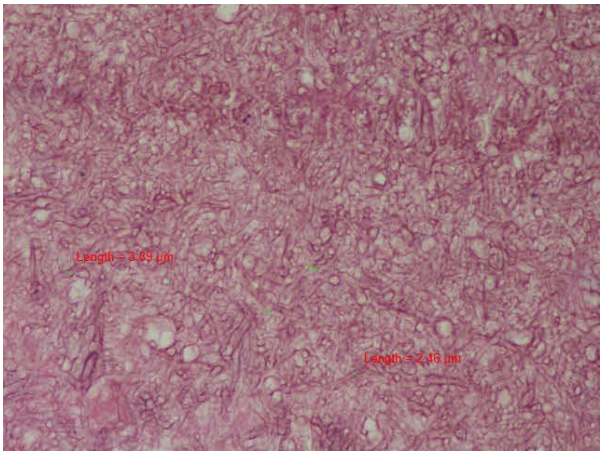

Fig 2 Photomicrograph showing acute branching hyphae measuring less than 4µm in diameter 400x[H&E]

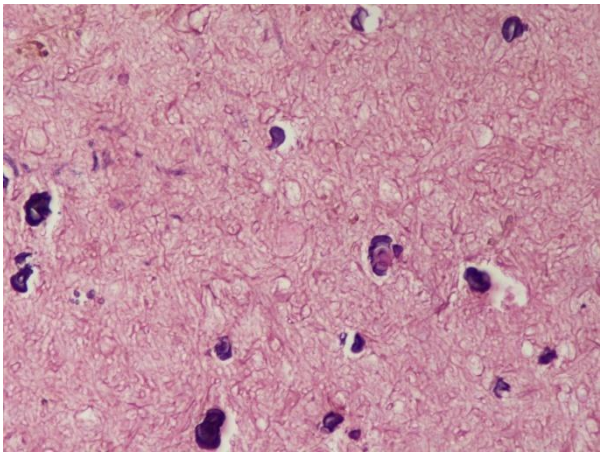

Fig 3 Photomicrograph showing numerous rounded calcification admixed in hyphae 400x[H&E]

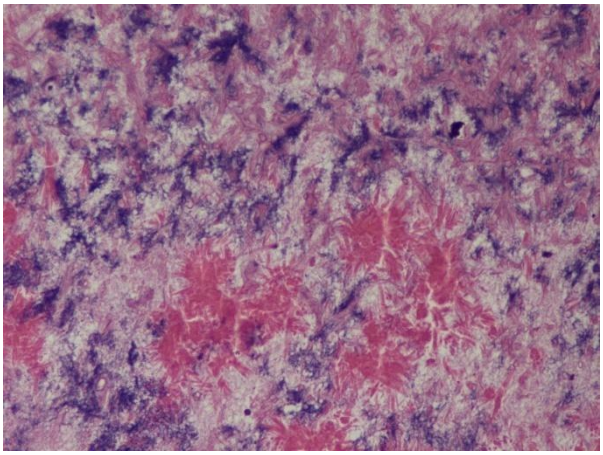

Fig 4 Photomicrograph showing abundant Charcot crystals with fungal hyphae 400x[H&E]

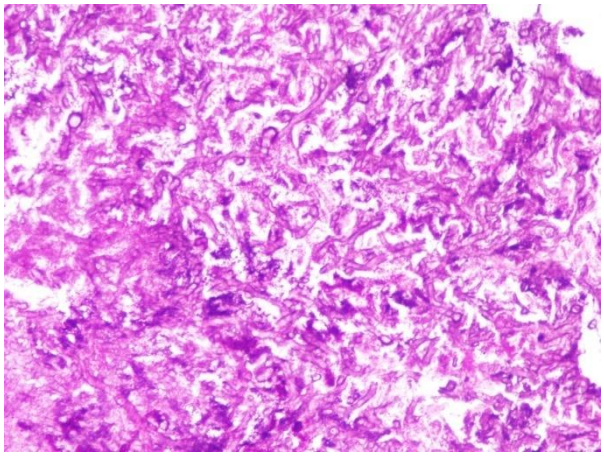

Fig 5 Photomicrograph showing acute branching hyphae as Y shaped patter 400x [PAS Stain]

Discussion

Fungal sinusitis was first described in 1791AD by Plaignaud in a 22 year old soldier as a "fungal tumor" in the maxillary sinus.(1)

Aspergillus is the most common fungus in histologically verified CNS mycosis from India, and presents with focal neurological signs and symptoms.[3] The most common species is Aspergillus fumigates. (11)

Allergic Fungal Rhinosinusitis (AFRS) affects 1%–2% of the world's population with regional variation in incidence.(7)(8)

Major conditions increasing predisposal are neutropenia and corticosteroids.(11)

Aspergillus spreads to the CNS by direct inoculation by trauma or surgery, or direct extension from paranasal sinuses or eye, or invasion of arteries and veins and fungemia.(5)

It is a saprophytic fungus present throughout the world, infecting following inhalation of the conidia or mycelial fragments on soil, decaying matter and vegetation; often leading to allergic bronchopulmonary aspergillosis, paranasal granuloma, fungal ball, invasive aspergillosis and endocarditis.(6)

The 2-3 micron size spores reach the alveoli, where alveolar macrophages recognize beta-1,3 glucan through Toll-like receptor-2 and lectin dectin-1. These receptors activate phagocytes to kill the conidia; however in immunosuppressed states they germinate into hyphae; which invade tissues.

Virulence factors produced by Aspergillus include adhesins, antioxidants, enzymes and toxins, as well as mannitol, catalases, superoxide dismutase, and melanin. Aspergillum growing on the surface of peanuts produce the carcinogenic Aflatoxin; may contribute to hepatocellular carcinoma in Africa.(11)

Currently, patients are said to have AFRS on fulfilling the following criteria by Bent and Kuhn:

type I hypersensitivity, nasal polyposis, characteristic findings on CT scan, presence of fungi on direct microscopy or culture, and allergic mucin containing fungal elements without tissue invasion.(9) The "peanut butter" or "cottage cheese" like mucus from sinuses of patients with AFRS cannot be differentiated from those of Allergic Bronchopulmonary Aspergillosis; termed "allergic" or "eosinophilic mucin", the color ranges from tan, green, brown to black and is composed of degenerated eosinophils, Charcot Leyden crystals, scant hyphae and mucus. Eosinophils, plasma cells and lymphocytes are seen in the adjoining sinus mucosa.(12)

While colonizing aspergillosis (aspergilloma) refers to growth of fungus in pulmonary cavities and nose with minimal invasion, often forming brownish "fungal balls" in the cavities, invasive aspergillosis is an opportunistic infection primarily involving the lung and often disseminating to the heart valves and brain. The necrotizing pulmonary lesions are often referred to as "target lesions", with sharp round grey foci and hemorrhagic borders. (11)

Aspergillus grows by budding or branching. It forms fruiting bodies, in pulmonary cavities particularly and septate filaments 5-10 microns thick, branching at acute angles (40 degrees). The conidia are 1–3 μm in diameter, carried by air and easily inhaled in the lungs.(11)(10)

A key differential is Mucormycetes, which forms aseptate hyphae with varying width (6-50um), frequent right angle branching.(11)

Conclusion

The rising incidence of Aspergillosis as a cause of fungal rhinosinusitis calls for a high degree of suspicion, particularly in asthmatic people. Histopathological examination complemented by simple stains and assisted by microbiological tests aid a rapid diagnosis and initiation of therapy.

References

1. Plaignaud X. Observation sur un fongus du sinus maxillarie. J Clin 1791;1:111.
2. A. Alam, B. N. Chander, G. S. Sabhikhi, and M. Bhatia, "Sinonasal mucormycosis: diagnosis using computed tomog- raphy," *Medical Journal Armed Forces India*, vol. 59, no. 3, pp. 243–245, 2003.
3. A. Chakrabarti, A. Das, J. Mandal et al., "The rising trend of invasive zygomycosis in patients with uncontrolled diabetes mellitus," *Medical Mycology*, vol. 44, no. 4, pp. 335–342, 2006.
4. S. Malhotra, S. Duggal, N. K. Bhatia, N. Sharma, and C. Hans, "Rhinocerebral zygomycosis with pulmonary aspergillosis in a non-HIV-infected patient: an unusual case report from India," *Journal of Medical Microbiology*, vol. 58, no. 1, pp. 146–150, 2009.
5. E. Abedi, A. Sismanis, K. Choi, and P. Pastore, "25 years' expe- rience treating cerebro-rhino-orbital mucormycosis," *Laryn- goscope*, vol. 94, no. 8, pp. 1060–1062, 1984.
6. J.E. Bennett, "Aspergillosis," in*Harrison's Principles of Internal Medicine*, K. J. Isselbacher, J. Wilson, A. Fauci, E. Braunwald, J. Martin, and D. Kasper, Eds., pp. 855–862, McGraw Hill, New York, NY, USA, 2005.
7. Allphin AL, Strauss M, Abdul-Karim FW. Allergic fungal sinusitis: problems in diagnosis and treatment. *Laryngoscope*. 1991;101:815-820.
8. Collins MM, Nair SB, Wormald PJ. Prevalence of noninvasive fungal sinusitis in South Australia. *Am J Rhinol*. 2003;17:127-132.
9. Bent JP, Kuhn FA. Diagnosis of allergic fungal sinusitis. *Otolaryngol Head Neck Surg*. 1994;580-588.
10. Sharma OP, Chwogule R. Many faces of pulmonary aspergillosis. Eur Respir J. 1998;12:705–15.
11. Kumar, V., Abbas, A. and Aster, J., 2013. *Robbins Basic Pathology*. 9th ed. Elsevier Saunders, pp.388,389.
12. deShazo RD, Chapin K, Swain R. Fungal sinusitis. N Eng J Med 1997;337:254-59.

Abbreviations

AFRS: Allergic Fungal Rhinosinusitis
CNS: Central nervous System.