

RHINOSPORIDIOSIS

Naushaba Rahat¹ & Saleem Sadiq²

ABSTRACT:

Rhinosporidiosis is a rare chronic granulomatous infection that usually manifests as vascular friable polyps arising from the nasal mucosa or external structures of the eyes caused by *Rhinosporidium seeberi*, recognized as water mold-related fungus.

We report a case of rhinosporidiosis in a twenty six years old male presenting at the Department of ENT, Jinnah Postgraduate Medical Centre Karachi with the complaints of blockage of nose and bleeding from nose for four months. EUA and endoscopy revealed polypoid mass in nasopharynx and nose. CT scan revealed a soft tissue mass in the nasopharynx with the impression of a polyp or a neoplasm. Histological examination revealed variable sized cysts with a chitinous wall consistent with rhinosporidiosis.

KEY WORDS: *Rhinosporidium seeberi*, chronic granulomatous infection.

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INTRODUCTION

Rhinosporidiosis is a chronic granulomatous infection that usually manifests as vascular friable polyps arising from the nasal mucosa or external structures of the eyes. It was initially described by Seeber, in 1900 in an individual from Argentina¹. The etiological agent of rhinosporidiosis is *Rhinosporidium seeberi*, that has never been cultured. *R. seeberi*, traditionally have been believed to be a fungus. Recent 18s ribosomal ribonucleic acid (rRNA) gene analysis has placed *R. seeberi* into a novel group of aquatic parasites². Most persons with

rhinosporidiosis have had bathing or working exposure to stagnant water¹. It is endemic in India, Srilanka, Bangladesh with only sporadic occurrence in United States³. It is uncommon in Pakistan⁴. Thought to be a zoonotic organism because it is seen in cattle, horse and mules⁵. It is clinically characterized by formation of papillomatous or polypoid mass⁴.

CASE REPORT

We report a case of rhinosporidiosis, a rare infectious disease seen in the Department of Pathology, Basic Medical Sciences Institute of J.P.M.C. This is the second such case seen in the last 10 years in our Department.

A twenty six years old male presented at the Department of ENT, JPMC with complaint of bleeding from nose for four months, and blockage of nose. EUA and endoscopy revealed polypoid mass in nose and nasopharynx. CT scan of paranasal sinuses revealed a soft tissue mass in the nasopharynx, measuring 4 x 3 cm related to left side. Left maxillary, sphenoidal and frontal sinuses were clear. Both orbits were normal in limits. Bone structure was normal. Rigid nasal endoscopy and biopsy of the mass was done.

Examination revealed two irregular grey

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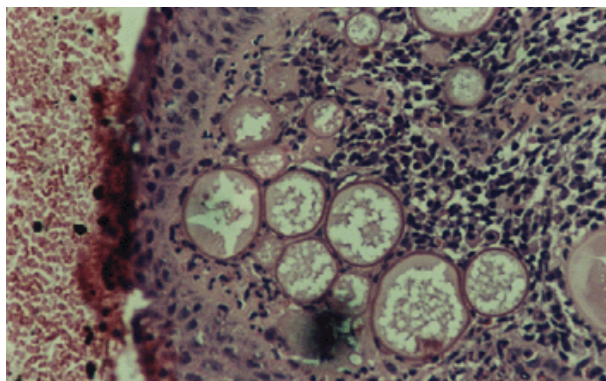


Figure-I: Photomicrograph of nasopharyngeal mucosa showing numerous cysts of *Rhinosporidium seeberi* (Haematoxylin and Eosin x 200)

white soft tissue pieces, larger measuring 2 x 1.5 x 1 cm and smaller 1.5 x 1.5 cm. Microscopic examination revealed polypoidal fragments of nasal mucosa with mucous glands. A mild to moderate chronic inflammatory infiltrate, and scattered cysts of varying sizes with an outer chitinous shell with an inner cellulose wall enclosing large numbers of spores, smaller trophic forms and a foreign body giant cell reaction (Fig-I).

DISCUSSION

Rhinosporidiosis is a chronic granulomatous infectious disease, characterized by hyperplastic polypoid lesions of the mucous membrane, predominantly nasal but, rarely of the skin and viscera⁵.

The condition was first observed by Professor Malbren, of Buenos Aires, in 1892. In 1896 Seeber re-examined this material and included it in his thesis for the M.D. degree, but failed to name it. Belou in 1903, designated the organism as *Coccidium seeberi*, though Wernicke apparently had suggested the name in 1900. The first case recognized in India was by O'Kinealyi in 1894. Minchin and Fantham in 1905 reviewed the material of O'Kinealyi's case and called the organism *Rhinosporidium kinealyi*, being unaware of the fact that it had earlier been named *Coccidium seeberi*. Ashworth after a study of *Rhinosporidium*, proved that it was not a sporozoa, but belonged

to the group phycomycetes in the sub-order of Chytridinea, and called it *Rhinosporidium seeberi*, which has become its accepted name⁶. Since then the microbe has been considered a fungus by most microbiologists, although its taxonomy has been debated. Using consensus polymerase chain reaction (PCR) approach, Fredrick and colleagues amplified a portion of the *R. seeberi* 18SrRNA gene directly from infected tissue. Analysis of the aligned sequence and inference of phylogenetic relationships showed that *R. seeberi* is a protist from a novel clade of Parasites that infect fish and amphibians and *R. seeberi* is not a classic fungus, but rather the first known human pathogen from the Drips clade, a novel clade of aquatic protistan parasites².

Majority of cases are reported from India, Srilanka and Bangladesh. It has been recognized in many other parts of the world, like South America, United States, England, Egypt, South Africa⁶. Predominantly male are involved (male to female ratio 4:1).¹ It is uncommon in Pakistan⁴. Mode of transmission is via water or dust, from which the endospores penetrate the nasal cavity mucosa, mature into sporangium within the submucosal compartment and after maturation burst with release of sporangia into surrounding tissue⁶. No immune deficiency has been associated with infection¹. Common sites of involvement are nose and nasopharynx. Other sites are larynx, tracheobronchial tree, esophagus, conjunctiva, ear, bone and skin⁶.

It usually presents as single or multiple, pedunculated or sessile masses, pink to deep red in colour, usually described as strawberry like appearance. They bleed easily with a history of nasal obstruction or epistaxis. In 1923, Ashworth described in detail the life cycle of the organism in tissue¹. Histologically the infected tissue reveals granulomatous reaction (mixed cell granuloma), pseudocystic abscesses and fibrosis around the causative organism⁴. The various stages in the life cycle of the organism may be found in the tissue, but more readily recognizable are the sporangia in varying stages of maturity and sizes, ranging from 120

to 300 microns in diameter and characterized by an outer chitinous shell with an inner cellulose wall enclosing thousands of spores⁷ (Fig 1). Diagnosis is established by observing the characteristic appearance of the organism in tissue biopsies¹. *R. seeberi* is clearly seen in hematoxylin and eosin stained section.

The wall of the maturing sporangium and the spores are stained positively by the Gridley's technique, periodic acid schiff (PAS), Gomori methenamine silver (GMS). The condition must be distinguished from the other polypoid lesions of the nasal cavity¹: both non-neoplastic and neoplastic.

The causes of non-neoplastic polyps include inflammatory polyps associated with chronic sinusitis, allergic rhinitis, tuberculosis, lepromatous leprosy, rhinoscleroma and inflammatory pseudotumours⁸. The causes of neoplastic polyps include squamous cell papilloma, juvenile angiofibroma, schwannoma, nasopharyngeal carcinoma, rhabdomyosarcoma, fibrosarcoma and malignant lymphoma⁹. Histologically inflammatory polyps are characterized by polypoid lesions, covered by respiratory epithelium, with a marked edematous, vascular myxomatous stroma, containing fibroblasts, eosinophils, plasma cells and lymphocytes⁵. Tuberculosis can produce polypoid mass in nasal cavity⁸, histologically characterized by caseating granulomas, giant cells of Langhans type and chronic inflammatory infiltrate. Lepromatous leprosy, histologically is characterized by extensive infiltration of *Mycobacterium leprae*-laden histiocytes in the submucosa⁸. Other differential include rhinoscleroma, which was

microscopically characterized by Mikulicz cells, the Russell bodies and bacillus *Klebsiella rhinoscleromatis*. Inflammatory pseudotumors are tumor like lesions, histologically heterogeneous consisting of edematous or hyalinized fibrous tissue infiltrated with varying number of plasma cells, Russell bodies and lymphocytes⁸.

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