

Case Report

Nasal rhinosporidiosis: A clinicopathological study of 9 cases**Dr. Khade Archana L¹, Dr. Khare Manisha S², Dr. Saloni Karwa³, Dr. Akansha Gaajre⁴, Dr. Anumeha Chaturvedi⁵**¹Ex Senior resident, Department of Pathology, Lokmanya Tilak Medical college and General Municipal Hospital, Sion, Mumbai-400022²Professor, Department of Pathology, Lokmanya Tilak Medical college and General Municipal Hospital, Sion, Mumbai-400022³Ex Senior resident, Department of Pathology, Lokmanya Tilak Medical college and General Municipal Hospital, Sion, Mumbai-400022⁴Assistant Professor, Department of Pathology, HBT Medical College and Dr. R.N. Cooper Hospital, Juhu, Mumbai-400056⁵Resident, Department of Pathology, HBT Medical College and Dr. R.N. Cooper Hospital, Juhu, Mumbai-400056***Corresponding author**

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Abstract: To study incidence and clinicopathological features of nasal rhinosporidiosis, amongst inflammatory lesions of the nose. A Retrospective study of biopsy specimens from nose, diagnosed as rhinosporidiosis on histology, over a ten year period from January 2002 to October 2012, was carried out. The detailed clinical history was collected from clinical record. The tissues were studied with Haematoxylin and Eosin stain and Periodic acid Schiff stain (PAS). Total 09 cases of nasal rhinosporidiosis were studied. Rhinosporidiosis was most commonly seen in 2nd and 3rd decade with 3 (37.50%) cases in each. The youngest patient was 11 years old and oldest was 40 years old. All cases were seen in males. All the 9 patients of rhinosporidiosis presented with nasal mass, in addition epistaxis was seen in 6 (75%) cases. Rhinosporidiosis earlier thought to be protozoan now proved as of bacterial origin, often presenting as nasal mass with epistaxis in young adult males.

Keywords: Nose, infective lesion, polypoidal mass, rhinosporidiosis

INTRODUCTION

Rhinosporidiosis is a chronic granulomatous disorder of infective etiology previously thought to be caused by rhinosporidium seeberi but now a cyanobacterium, microcystis aeruginosa has been proposed as a causative agent [1,2]. The disease is endemic in India and Sri Lanka, however sporadic cases have also been reported from United States, South America, Italy, Iran, and Turkey [2-4]. Rhinosporidiosis most commonly involves nasal mucosa. In the nasal cavity septum, inferior turbinate, floor and lateral wall are commonly involved [5]. This study is carried out to find the incidence and clinicopathological features of nasal rhinosporidiosis in tertiary hospital.

MATERIAL AND METHODS

This was a retrospective 10 years study, conducted in the pathology department of a tertiary care

hospital. All cases diagnosed as rhinosporidiosis by histology were included in the study.

Detailed clinical data regarding age, sex and clinical presentations were collected from the clinical notes.

Histopathological samples were studied by routine paraffin embedding, slides were stained by hematoxylin and eosin (H and E) and periodic acid-Schiff (PAS) stains. Diagnosis was made by demonstration of thick-walled sporangia containing numerous endospores in a background of inflammatory infiltrate.

RESULT

During the study period, out of 220 cases of inflammatory lesions of nose, rhinosporidiosis was seen in 09(4.09%) cases. The age range of patient was

between 11 to 40 years. Most common age group affected was 2nd and 3rd decade with 3 (37.5%) cases in each. All the cases were seen in males. All the patients presented with reddish polypoidal nasal mass. Epistaxis was seen in 6 (75%) cases and nasal obstruction in 3 (37.50%) cases.

Histology showed polypoidal mass lined by squamous epithelium, stroma was edematous and was infiltrated by chronic inflammatory infiltrate comprising of lymphocytes and plasma cells. Characteristic sporangia were seen as globular cyst of varying sizes lined by well-defined thick cell wall containing numerous endospores which were PAS positive. (Fig.1&2)

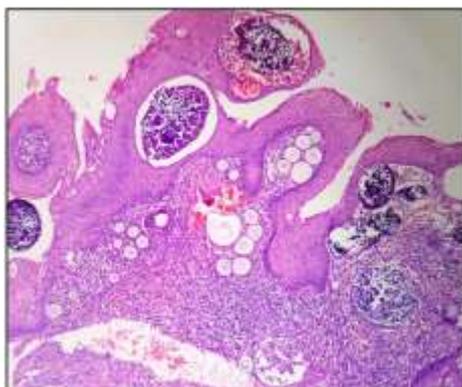


Fig-1: Shows polypoidal mass lined by squamous epithelium, stroma was edematous and was infiltrated by chronic inflammatory infiltrate comprising of lymphocytes and plasma cells with globular sporangia of varying sizes (H&E, 100X)

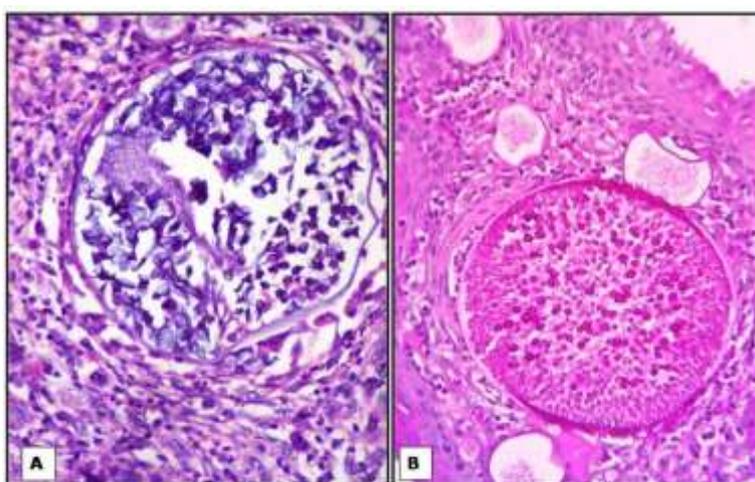


Fig-2: Shows sporangia filled with endospores (Fig 2A, H&E, 400X) which are PAS positive (Fig 2B, PAS 400X).

DISCUSSION

Rhinosporidium seeberi was first described as sporozoan by Malbran in 1892 in Argentina [1]. Seeber classified it as protozoan in 1900 [1]. Ashworth thought it to be phycomycetes and proposed the name rhinosporidium seeberi [1]. Some authors have also put it under a new class of aquatic protistans called mesomycetozoa along with other aquatic parasites that cause similar infections in amphibians and fish [6]. However, recent studies suggest a waterborne cyanobacterium microcystis aeruginosa as the causative organism of rhinosporidiosis. Ahluwalia *et al.* [2] has demonstrated large cells and nanocytes of microcystis

in round bodies of rhinosporidium by light and electron microscopy.

Rhinosporidiosis most commonly involves mucosa, particularly nasal mucosa followed by ocular involvement. There have been case reports of involvement of larynx, lips, palate, uvula, trachea, bronchus, ear, vulva, vagina, penis, rectum, and bone [7].

The disease is seen in all age group but often in early decades of life. Most series shows a predominance of male, though Gupta *et al.* found no significant sex difference [5]. The mode of transmission

is not clear but believed to be through dust, infective clothing, fingers, and bathing in stagnant water [1]. The possible pathogenesis is that spores enter traumatized nasal mucosa and multiply with formation of sporangia. These sporangia burst and releases spores. This results in reactive hyperplasia of the tissue and formation of polypoidal mass [8].

Nasal involvement presents with obstruction, discharge and epistaxis. Polypoidal masses within the nasal cavity may present at anterior nares.

Grossly, there is polypoidal granular masses often describe as having strawberry appearance. This is due to the highly vascular composition and presence of sporangia close to or actually on the surface of epithelium where they present as pale sharply defined dots [5].

Tondon *et al.* [9] studied 134 cases of lesions in nasal cavity, of which rhinosporidiosis was seen in 24% of cases. The age range was between 10 to 59 years. Age group commonly affected was 3rd decade with male predominance seen in 20 out of 24 cases. The presenting symptoms were nasal obstruction, discharge and epistaxis.

Makannavar and Chavan [10] studied 34 cases of rhinosporidiosis. Age range of patients was between 8 to 70 years. They observed maximum incidence in 2nd decade with male predominance seen in 31 (91.18%) cases, which is comparable with the findings in our study. According to them, male predominance seen in rhinosporidiosis could be due to more outdoor activity by males and as the disease transmission is via droplet infection and bathing in contaminated water which is usually done by males. Most common site was nose and nasopharynx with ocular involvement seen in 3 cases in their study.

Although histopathology is considered as gold standard in diagnosis of rhinosporidiosis, attempts at diagnosing by aspiration cytology and Papanicolaou staining have been documented [11]. The smear with 10% KOH or Giemsa stained imprint smears can also be used [11]. Histopathology shows characteristics sporangia with PAS positive endospores amidst dense chronic inflammatory cell infiltrate. However as the organism cannot be grown in culture, histopathology is the gold standard.

Treatment of rhinosporidial lesions is mainly surgical, however various medical therapies have been tried like griseofluvin, amphoterecin B and Dapson. Out of these only Dapson is found to be effective [12]. Total excision of the polyp, preferably electrocautery, is the recommended method of surgical therapy.

Recurrence may occur due to spillage of endospores in the surrounding mucosa during removal [13].

CONCLUSION

Rhinosporidiosis is a rare preventable condition which has to be considered in young males presenting as nasal mass with epistaxis.

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