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A REVIEW ON RHINOSPORIDIOSIS

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ABSTRACT

Rhinosporidiosis is a chronic granulomatous infection of the mucous membranes that usually manifests as vascular friable polyps that arise from the nasal mucosa or external structure of the eye. Rhinosporidiosis is a non-contagious, sporadic, chronic granulomatous infection of the mucocutaneous tissue caused by rhinosporidium seeberi, yet an unisolated and unclassified fungus. Polyps mainly occurs in nasal, ocular regions, cutaneous and disseminated forms are relatively rare. R. seeberi is generally acquired by bathing in ponds contaminated by animal feces. Tropical and subtropical regions are considered endemic areas. The largest number of rhinosporidiosis cases was noted in India and Srilanka, followed by South American and African countries. This study also describes the diagnosis and management of rhinosporidiosis.

KEYWORDS: Rhinosporidiosis, Rhinosporidium seeberi, nasal polyps, diagnosis and management.

INTRODUCTION

Rhinosporidiosis is a chronic granulomatous infection of the mucous membranes that usually manifests as vascular friable polyps that arise from the nasal mucosa or external structure of the eye. Clinically, the lesions are seen as friable polyps are papillomas, classically described as strawberry-like in appearance. So, it is called as Strawberry nasal mass.

Rhinosporidiosis is a chronic infection of the mucous membrane that is caused by the mesomycetozoan Rhinosporidium seeberi. The lesions present as a polypoid or vascular mass, sometimes pedunculated, in the nose about 78%, nasopharynx about 68%, tonsil about

3%, eye about 1% and in skin very rare. In 1892 Malbran observed the organism in the nasal polyp.

Etiological Agent

Rhinosporidiosis is a chronic granulomatous clinical condition characterized by fungal infection of mucous membrane in humans, caused by Rhinosporidium seeberi. Rhinosporidium seeberi, was discovered by Guillermo Seeber in 1900 in Argentina, as a protozoan parasite which produces nasal polyp. In 1903, O'Kineley described its histology. In 1905, Minchin & Fantham studied O'Kineleys tissue and named the organism as Rhinosporidium kinealyi. In 1913, Z Schokke reported similar organism in horses and named it rhinosporidium equi. In 1923, J. Ashworth described that rhinosporidiosis is caused by a fungus and he named it Rhinosporidium seeberi in the honor of G seeber. In 1924, Forsyth described skin lesion. In 1924, Thirumoorthy reported the first female patient. In 1936, Cefferi established the identity of R.Seeberi and R.Equi. In 1953, Demellow described the mode of its transmission. R.Seeberi belongs to the class Rspherical cell is transformed into a single sporangium. Sporangia of various sizes are located in the stroma of the polyp. The overlying mucosa may show squamous metaplastic changes. The largest of these are usually immediate sub epithelial in location which may or may not show evidence of rupture. Mature sporangia are 100 to 450 micrometer in diameter with a thick chitinous wall and contain sporangospores in different stages of development. R.Seeberi is visualized by fungal stains such as PAS, Grocott methenimine silver and also the combination of two dyes-eosin and haematoxylin(Fig-1).

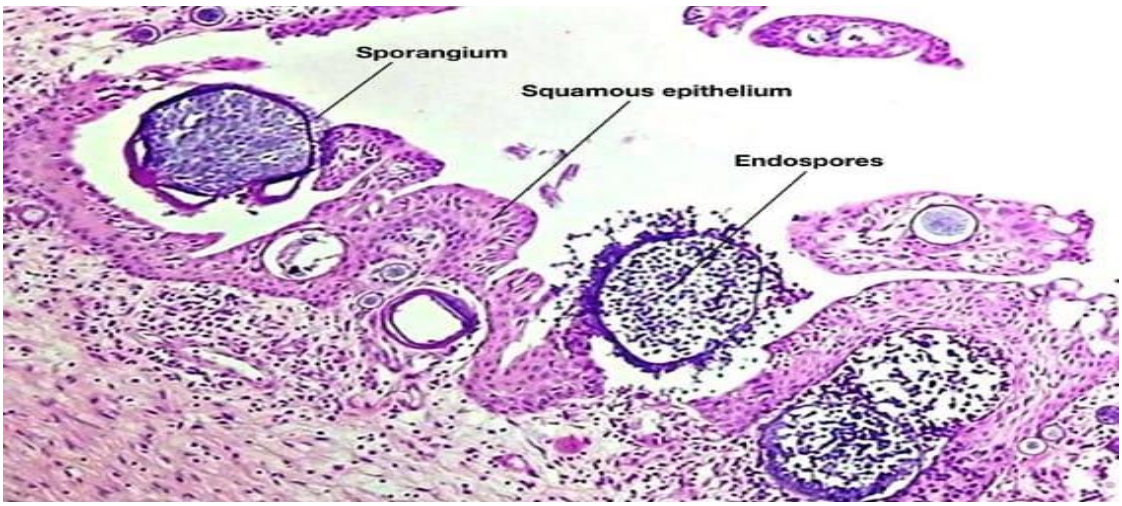

Fig 1: Pathology of Rhinosporidiosis.

Clinical Classification

Nasal rhinosporidiosis is the most commonly occurring form of rhinosporidiosis, characterized by epistaxis and development of sessile, pink to purple, peduncular polyps like nasal obstruction which can be unilateral or bilateral mostly in the upper respiratory tract(Fig-2).

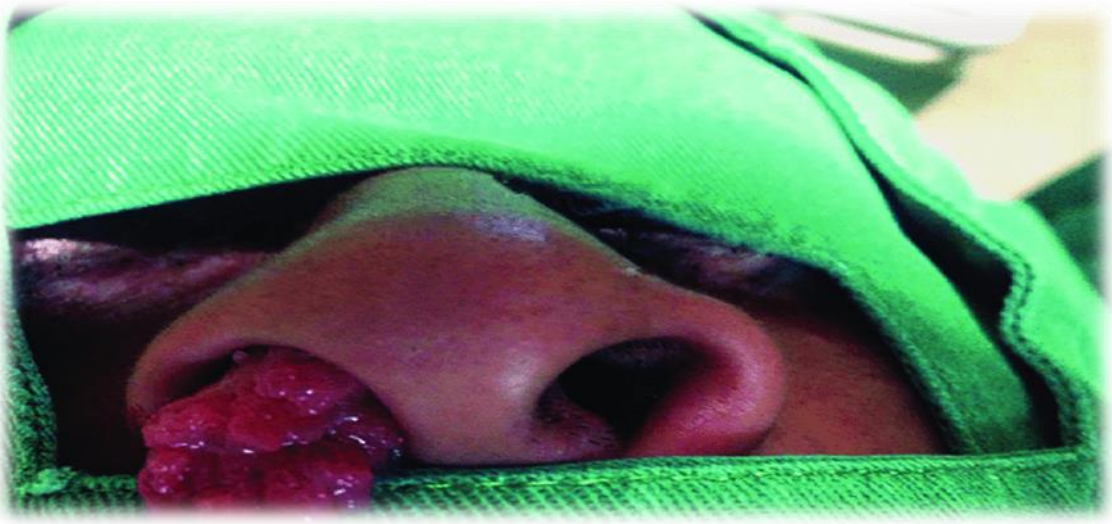

Fig 2: Nasal rhinosporidiosis.

Nasopharyngeal rhinosporidiosis chiefly presents with polypoid tumor like masses in affected mucosal sites. Because of the friable and pendulous nature of these lesions, presenting complaints are mainly of nasal symptoms of rhinorrhea, epistaxis or ultimately obstruction(Fig-3).

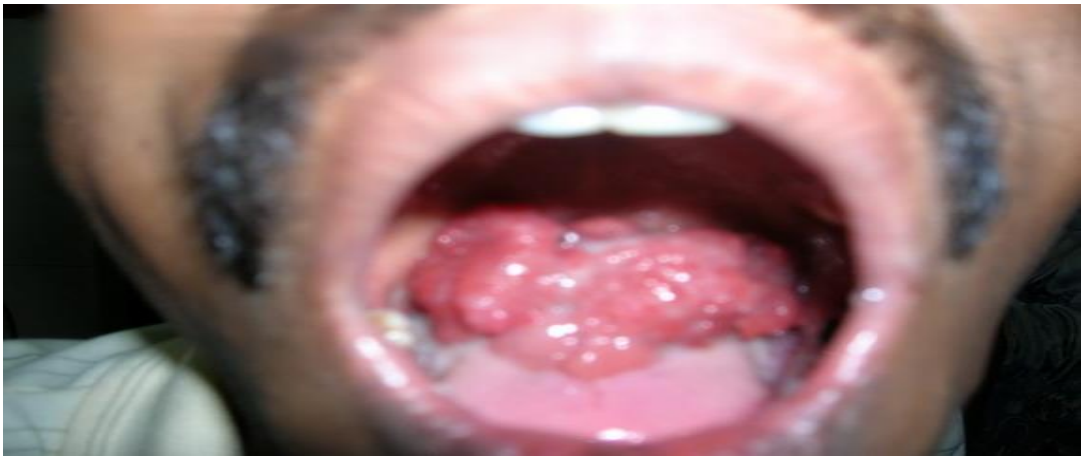

Fig 3: Nasopharyngeal Rhinosporidiosis.

Cutaneous rhinosporidiosis present as warty papules and nodules with whitish spots, crusting, lesions and bleeding on the surface. Three types of lesions are seen like satellite lesion, generalised cutaneous lesions and primary cutaneous lesions(Fig-4).

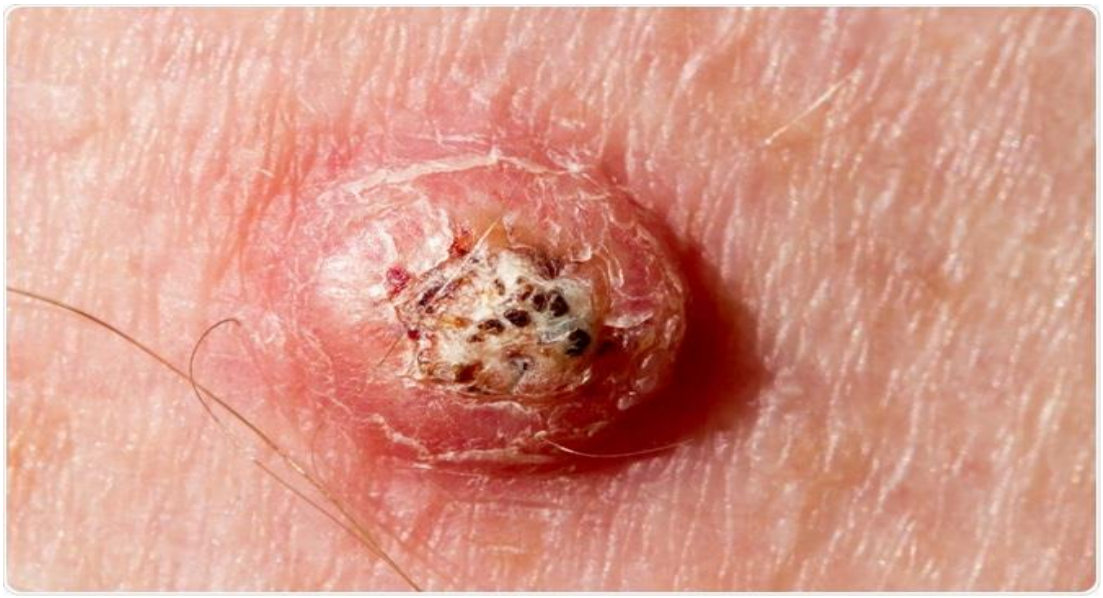

Fig 4: Cutaneous Rhinosporidiosis.

Ocular rhinosporidiosis begins as a sessile growth, which worsen to friable peduncular polyps in the eye. As per the size outgrowth symptoms of tearing, discharge, redness of eye, lid eversion, photophobia, and conjuctival infection may appear.

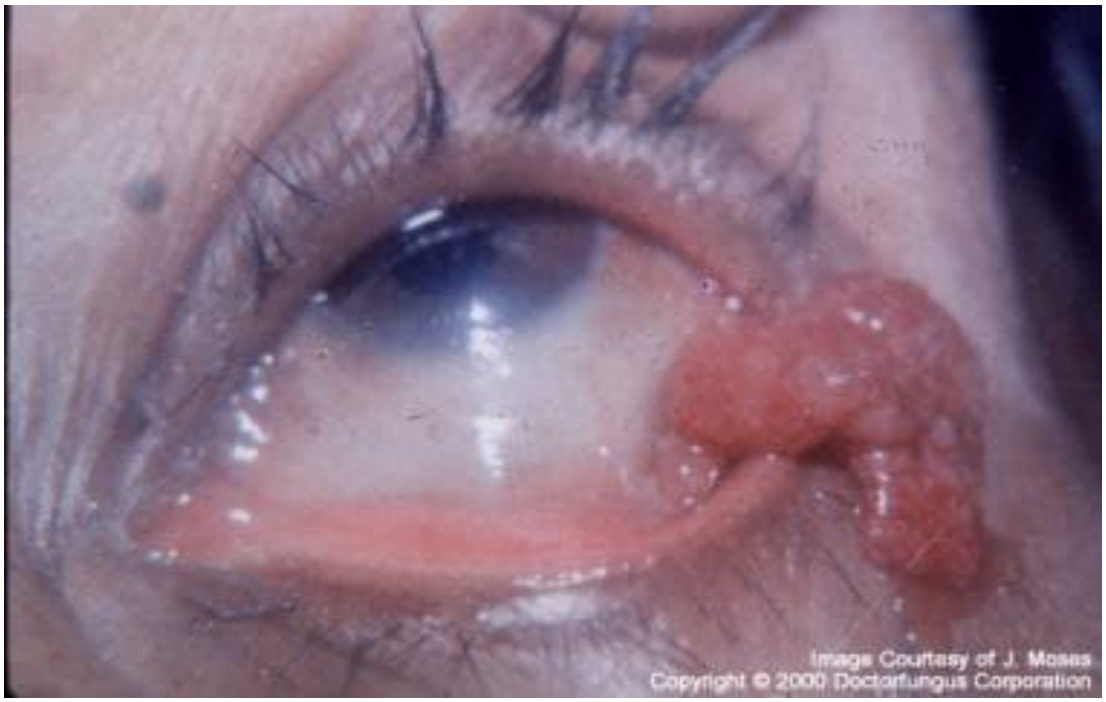

Fig 5: Ocular Rhinosporidiosis.

Epidemiology

Rhinospordiosis has been reported from about 70 countries with diverse geographical features although the highest incidence has been from India and Srilanka. Tropical and subtropical regions are considered endemic areas. In Europe and the United States, rhinosporidiosis is rarely seen in humans. In India first case were reported from Bihar in the State of Maranhao(Fig-6). The State of Maranhao, located in a tropical region, is the most rural State of Brazil and its geography has a wide diversity of ecosystems. Therefore, the large number of river and lakes and the high presence of riparian populations are strong predisposing factors for rhinosporidiosis. Mostly seen in humans of 20-40 years of age group and males are effected 4 times more frequently than females. This incidences in humans are related with ABO Blood group also and O+ blood group individuals are more about 70% prone to disease followed by AB+ for the disease as compared to other blood groups in INDIA.

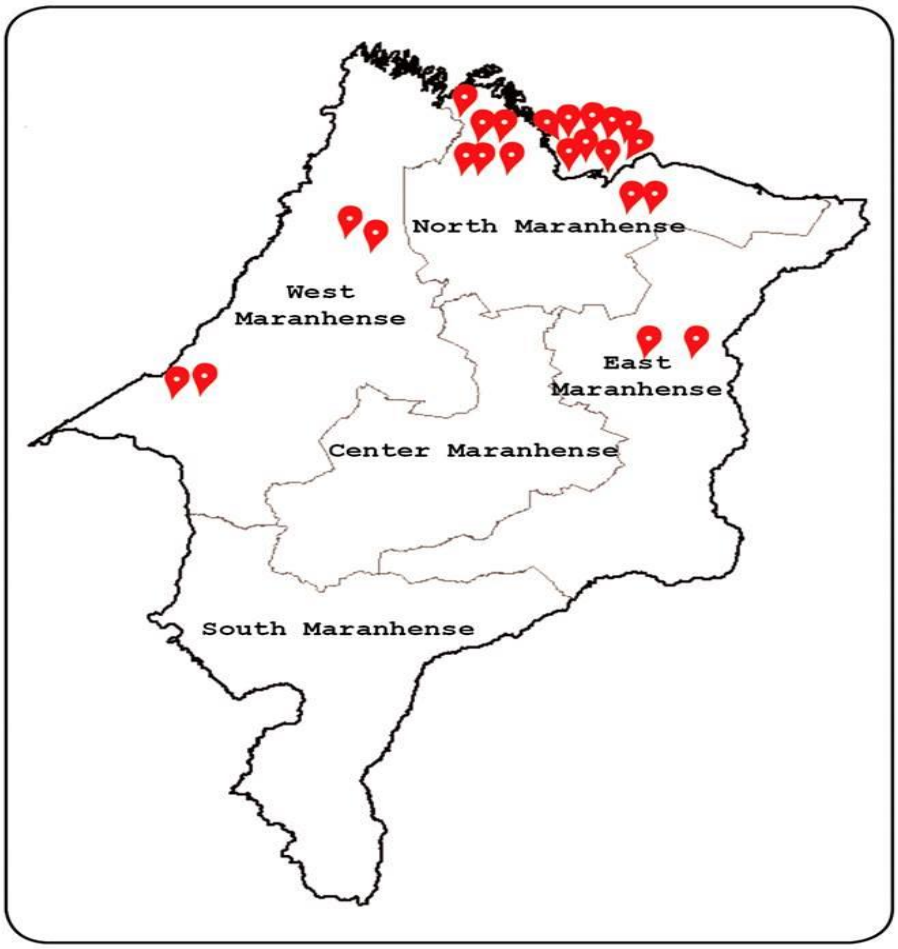

Fig 6: Distribution of rhinosporidiosis in the State of Maranhao, Brazil.

Transmission

Rhinosporidiosis is an infection caused by a fungi Rhinosporidium seeberi. It is generally acquired by bathing in ponds contaminated by animal feces, but still there is no proven theory about the complete life cycle of the organism. R.seeberi is a natural inhabitant of contaminated water and dust particles harbouring spores. Soil and water harbor the spores of these pathogen and hence soil and water act as reservoir for this pathogen. While drinking water, abraded nasal mucosa may get the infection about 70% cases and through dust fomites conjuctiva may give rise to ocular form about 15% of disease.

Diagnosis

Definitive diagnosis of rhinosporidiosis depends on histological examination with immunohistochemistry. This often requires excisional biopsy, scraping of superficial lesions or fine needle aspiration. Endoscopic examination involving rhinoscopy and CT scan. CT scan is helpful in identifying appropriate lesions of tissue over growth. CT scan helps in confirming the soft friable mass without bone involvement either in nasal cavity or at the affected site.

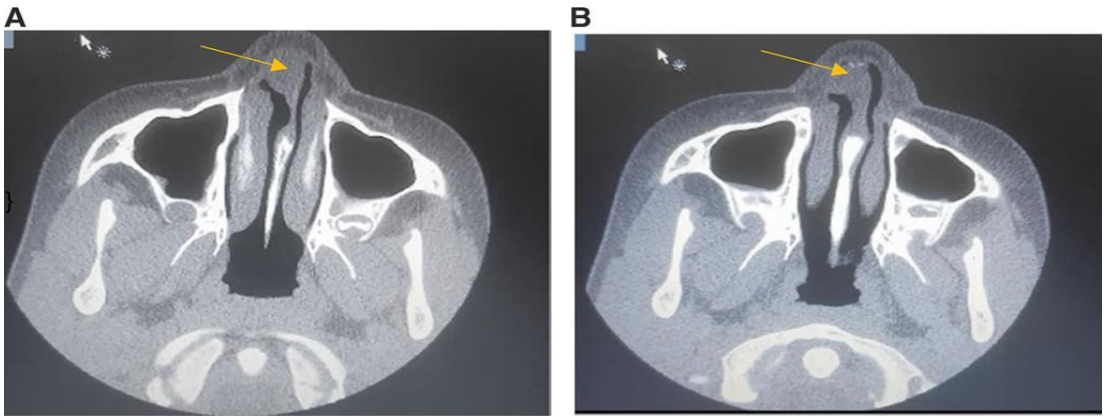

Fig 7: Computerized tomography(CT scan).

A simple computerised tomography of the head reveales a mass with nodular appearance in the nostril of soft tissye characteristics (Figure 7A and 7B).

Management

Rhinosporidiosis infection is treated primarily with surgical intervention. Medical management has rarely been reported as adjunct treatment to surgery. While several anti-fungal drugs have been tested clinically, the only drug which was found to have some anti-rhinosporidial effect is dapsone(4,4-diamino diphenyl sulfone). which appears to arrest the

maturation of the sporangia and to promote fibrosis in the stroma, when used as an adjunct to surgery. Medical management of rhinosporidiosis has not been strongly advised in several cases. Currently, dapsone is an adjuvant drug to surgery. Total excision of the polyp or conjuctiva is recommended, and electrocautery is most commonly utilized as it restricts free bleeding. The procedure should remove the entirety of the entire sac, and post operative dapsone therapy is usually recommemded. During removal of the polyp, spillage of the endospores into the surrounding mucosa may occur which can be contained by electrocautery. Despite the rare case of recurrence, regeneration or dissemination, the prognosis is generally very favourable. It typically follows a benign, prolonged course without treatment with morbidity.

CONCLUSION

Rhinosporidiosis is a chronic granulomatous infection caused by a fungus rhinosporidium seeberi. Here discussed in brief regarding possible mode of transmission, epidemiology, diagnostic and remedial approaches against this disease. Prevention will be the best option to be safe from this oraganism R.seeberi as the disease takes achronic course which makes diagnosis difficult. Hence swimmers and people who are frequent visitors to water bodies should have safety precautions as this organism get transferred through cut wounds.

REFERENCE

1. Morelli L, Polce M, Piscioli P et.al. Human nasal rhinosporidiosis: an Italian case report. Diagn. Pathol, 2006; 1: 25.
2. Mendoza L, Herr RA, Ajello L. Causative agent of rhinosporidiosis. J.Clin. Microbiol, 2001; 39(1): 413-415.
3. Madke B, Mahajan S, Kharkar V et.al. Disseminated cutaneous with nasopharyngeal rhinosporidiosis: light microscopy changes following dapsone therapy. Australas. J. Dermatol, 2011; 52: 4-6.
4. Das S, Kashyap B, Barua M et.al. Nasal rhinosporidiosis in humans: new interpretations and a review of the literature of this enigmatic disease. Med. Mycol, 2011; 49: 311-315.
5. Branscomb R. Rhinosporidiosis update. Lab. Med, 2002; 8(33): 631-633.
6. Arun BN, Manjula BV, Ravi CN et.al. Endoscopic removal of nasooropharyngeal rhinosporidiosis: a report. Internet J. Otorhinolaryngol, 2009; 9: 1-2.

7. Arseculeratne SN, Panabokke RG, Atapattu DN. Lymphadenitis, trans-epidermal elimination and unusual histopathology in human rhinosporidiosis. Mycopathologia, 2002; 52: 57-69.
8. Ahmed NA, Mohammed S, Raj G. Rhinosporidiosis: An Epidemiological study. J. Evo. Med. Dental. Sci, 2013; 38: 7227-7233.
9. Sinha A, Phukan JP, Bose K et.al. Clinicopathological study of rhinosporidiosis with special reference to cyto diagnosis. J Cytol, 2012; 29: 246-9.
10. Kundu AK, Jain M, Srivastava RK. Osseous involvement in rhinosporidiosis. Indian J Orthop, 2013; 47: 523-5.
11. Salim T, Komu F. Varied presentations of cutaneous rhinosporidiosis: a report of three cases. Indian J Dermatol, 2016; 61: 209-12.
12. John D, Selvin SS, Irodi A et.al. Disseminated rhinosporidiosis with conjuctival involvement in an immunocompromised patient. Middle east Afr Ophthamol, 2017; 24: 51-3.
13. Costa EF, Pinto LM, Campus MAG et.al. Partial regression of large anterior scleral stephyloma secondary to rhinosporidiosis after corneoscleral graft-a case report. BMC Ophthalmol, 2018; 18: 61.
14. Almeida F.A, Teixeira- junior A, Pinho JD. Evaluation of diagnosed cases of eye rhinosporidiosis in a public hospital of Maranhao, Northeast Brazil. BMC Ophthamol, 2019; 19: 218.
15. Ahluwalia KB. Culture of the organism that causes rhinosporidiosis. J. Clin. Microbiol, 2001; 39: 413-415.