

Rhinosporidiosis Presenting As Nasal And Urethral Polyp

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Citation

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Abstract

Rhinosporidiosis is a chronic granulomatous disease of mucous membranes and is endemic in South India and Sri Lanka. Although it predominantly affects nose and nasopharyngeal mucosa, other sites are also rarely affected. High index of suspicion and characteristic pathological feature are the mainstay of diagnosis for extra nasal infection.

We describe a case of 35 year old north Indian male who presented with urethral polyp. Otorhinolaryngological examination revealed a nasal mass as well. Biopsy from both the urethral and nasal mass showed features of rhinosporidiosis. The aetiopathogenesis, clinical & pathological picture and management of the disease have been discussed.

INTRODUCTION

Rhinosporidium seeberi predominantly affects the mucous membranes of the nose and nasopharynx. The organism thrives in hot, tropical climates, and endemic zones are South India and Sri Lanka.¹ It occasionally involves the lips, palate, uvula, conjunctiva, larynx, trachea, penis, vagina and bone.^{2,3} The diagnosis may be delayed when extranasal sites are involved. Occupational history and histopathology are helpful in arriving at a proper diagnosis.

CASE REPORT

A 35 yr old male resident of Uttar Pradesh, fisherman by occupation presented with a polypoid lesion at the urethral orifice. There was also a history of progressively increasing nasal obstruction on the left side for the past 1yr and spontaneous nasal bleeding for past 6 months.

Genital examination showed a polypoidal lesion 1.5 x 1.0x 0.5 cm present at the terminal urethra. Otorhinolaryngologic examination revealed a deep red, sessile, papillomatous lesion arising from lateral wall of right nasal cavity (Fig 1). The friable lesion resembled a strawberry, had yellow spots on undersurface, which bled on manipulation. The patient was subjected to contrast enhanced computerized tomography of nose and paranasal sinuses. Subsequently punch biopsy was performed from penile and nasal mass. Histopathologically the lesion contained unique round body structures of Rhinosporidium seeberi embedded in a fibromyxoid stroma. Diffuse infiltration of lymphocytes,

monocytes, and plasma cells was seen (Fig 2).

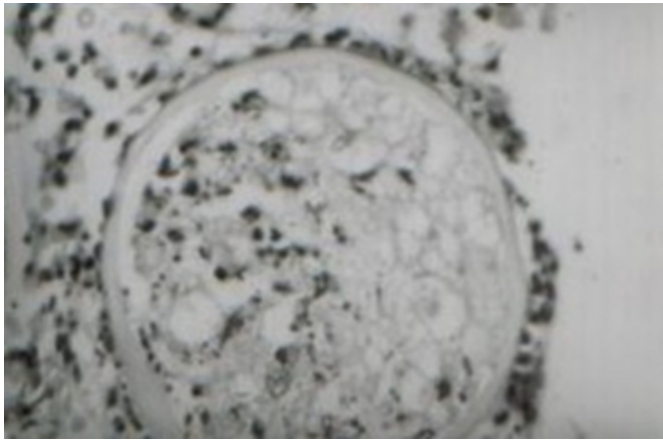
Figure 1

Figure 1: Clinical photograph of the patient showing nasal and oropharyngeal Rhinosporidiosis.



Figure 2

Figure 2: Microphotograph showing a sporangium containing numerous spores of *Rhinosporidium* species. (65 X, H & E stain).



The penile lesion was removed using sharp dissection along with wide excision of the nasal lesion with the help of cutting diathermy, the base of which was cauterized. Post operatively patient was advised to take Dapsone 100mg/day for a period of 3 months. The sites, from which the lesions were removed, healed with no evidence of recurrence up to a 6-month follow up period.

DISCUSSION

Rhinosporidiosis seeberi infection is endemic in South India and Sri Lanka with sporadic reports from various parts of world including the American Subcontinent.^{4,5} The disease has been found to be associated with animals like horses, mules, cats, dogs and wild ducks.⁶

Rhinosporidiosis is a prolonged painless disease with limited morbidity, four times more common in males, targeting individuals between 10-40 years.² Infection of nose and nasopharynx is observed in 85% of persons with rhinosporidiosis. Other sites involved are eye (9%), penile urethra, and external ear.² The instances of infection of parotid duct cyst⁷ and tibia have been reported.³

The typical lesion of Rhinosporidiosis is fleshy, vascular, polypoidal and granulomatous, grayish white dots, present on surface of lesion.⁵ On histopathology, large round chitinous structure filled with spore like bodies are seen.⁸ These contain endospores, which are released into the host tissues, each maturing in 10 days into a sporangium through an intermediate trophozoite stage. Epithelioid cell granuloma and giant cell response can be observed in about 47% of cases.³ Transepithelial migration of sporangia, a mechanism of endospore dispersal, can be observed in 76% of cases.

The organism can be observed by staining the smears with routine hematoxylin and eosin stain. The wall of young trophic forms are better delineated with the PAS stain and verhoeff van Gieson stain.³

Rhinosporidiosis is caused by Prototistan Mesomycetozoa.⁹ Recent 18S ribosomal ribonucleic acid (rRNA) gene analysis has placed *R. Seeberi* in a novel group of aquatic parasites.¹⁰ The etiologic agent *Rhinosporidiosis seeberi* has never been successfully propagated in vitro. Infection usually results from a local traumatic inoculation and is associated with water activities e.g. swimming in stagnant water. *Rhinosporidiosis* lives in soil and many authors agree that water is a necessary medium of transmission.² Disease progresses with the local replication of *Rhinosporidium seeberi* and associated hyperplastic growth of host tissue and a localized immune response. The chronicity, recurrences and systemic dissemination of infection have been related to the variable density and composition of host cell response.¹¹ Patients with rhinosporidiosis possess anti- *Rhinosporidiosis seeberi* IgG to an inner wall antigen expressed only during the mature sporangial stage. This finding suggests that the mapping of antigenic proteins may lead to important antigens with the potential as vaccine candidates.¹²

Wide local surgical excision with electrocoagulation of lesion base is the treatment of choice. Systemic Dapsone may be given in addition.¹³ Complication of the disease includes extremely rare life threatening dissemination, local secondary bacterial infection, and recurrence (10%).²

CORRESPONDENCE TO

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