

Fungal Foes: *Rhinosporidium seeberi*

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Rhinosporidiosis is a chronic mucocutaneous disease associated with exposure to pond water. Lesions occur principally on the face but also may involve other sites such as the genitalia.

Pathogenesis

Molecular studies have shown that *Rhinosporidium seeberi* is related to protistan fish pathogens and shows relationships to both protists and fungi.^{1,2} The organism is now classified as a protist in the class Mesomycetozoa that represents the animal-fungal boundary, and molecular evidence suggests the presence of several host-specific strains of the organism.³

Incidence

The disease may occur worldwide but is particularly prevalent in India and Sri Lanka. An Indian study of 462 participants with rhinosporidiosis found that the disease was most common in men aged 21 to 30 years.⁴ The most common sites of involvement were the nose and nasopharynx, followed by ocular tissue. Generalized rhinosporidiosis was noted in 7 participants.⁴ In a report of 54 patients with ocular involvement, 91% (49/54) had conjunctival involvement, the male to female ratio was approximately 3 to 1, and approximately half of the patients were 10 years and younger. Total excision of the fungal mass was generally effective, though recurrence occurred in 2 patients between 9 and 12 months following intervention.⁵

Clinical Manifestations

Parotid duct involvement may be accompanied by ulceration and mucopurulent discharge.⁶ Urethral involvement may be misdiagnosed as condyloma

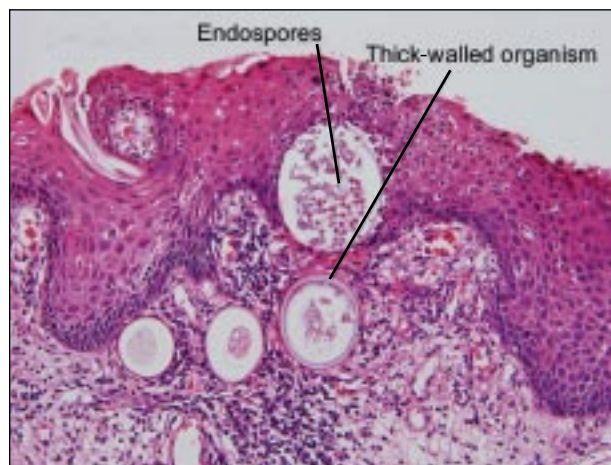
acuminatum. Tracheal involvement is particularly dangerous and may present with severe respiratory distress.⁷ Pulmonary lesions and lytic bone lesions may occur.⁸

The disease also may manifest with isolated large subcutaneous tumors.⁹ Disseminated cutaneous involvement often produces tumorlike swelling, and these patients often have mucosal involvement as well. While some patients with disseminated rhinosporidiosis are immunocompromised, widespread disease also may occur in immunocompetent adults.¹⁰

In addition to affecting humans, rhinosporidiosis occurs in animals such as dogs, swans, mules, and horses. The disease has been imported to nonendemic areas through international movement of livestock.^{11,12} Human disease has been reported in temperate climates as far north as Canada.¹³

Pathology

Rhinosporidiosis is diagnosed by examining biopsy specimens. Microscopic examination reveals thick-walled spherules with granular cytoplasm and a small central nucleus. Large sporangia that contain maturing endospores typically are present (Figure).



Histopathology of rhinosporidiosis (H&E, original magnification $\times 200$).

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The author reports no conflict of interest.

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Immune Response

An antibody response to the organism occurs, but even high titers of antibodies do not cause metabolic inactivation or morphologic damage to the endospores in vitro.¹⁴ There is evidence of cell-mediated immunity to the organism and suppression of cell-mediated immunity in chronic disease.^{15,16}

Treatment

Surgical excision of isolated lesions is the mainstay of therapy. Dapsone has been used as adjunctive therapy. Disseminated disease presents a particular challenge and a variety of agents have been tried with variable success. A 7-minute exposure to 70% ethanol, chlorhexidine, cetrimide-chlorhexidine, povidone-iodine, or silver nitrate inactivates spores in vitro, and topical therapy may play an adjunctive role in treatment.¹⁷

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