Genus Rhinosporidium

Rhinosporidium seeberi

Rhinosporidium seeberi *(R. seeberi)* causes rhinosporidiosis '**Water mold**', which is manifested as **tumor-like polyps** developing primarily in the nostrils and conjunctiva in human and animals. This disease is characterized by the presence of large, round-shaped mature stage and small endospores with **resistance to culturing**.
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R. seeberi was first reported in 1900 as a **sporozoan parasite**, but later classified as lower fungi, although its morphological similarity with aquatic parasites were also noticed. According to 18S small-subunit ribosomal DNA sequencing, *R. seeberi* belongs to a group of fish parasite DRIP clade located between the animal and fungal divergence.

Although once thought to be a fungus, molecular methods have identified this organism as an **aquatic protistan parasite** *(class **Mesomycetozoea***) that is **taxonomically located where animals and fungi diverge** and is closely related to pathogens of fish. True taxonomic identity of *R. seeberi* has been controversial.

Seeber was the first to address *R. seeberi 's* complex life cycle in the tissue of its infected hosts. Because it resists culture, for more than 100 years.

Epidemiology
The first report of rhinosporidiosis was Published by Seeber 88 and occurred in a 19-year-old Argentinean patient with breathing difficulties caused by a polyp that obstructed his nasal passages. In his thesis Seeber noted also that in 1892 Malbram, in Buenos Aires, Argentina, was the first to diagnose the disease in humans, but he did not publish his finding. Although the disease has been frequently diagnosed in the Americas and other areas of world, rhinosporidiosis is more prevalent in India and Sri Lanka than in any other geographic locations. The disease affects mucous membranes and rarely the skin and/or the internal tissues of its infected hosts.

Host:
Rhinosporidiosis has been most widely reported in humans and dogs, but cats, horses, and other mammalian and avian species may also be affected.
The overwhelming majority of affected dogs have been young adult to middle-aged (with a range of 1 to 13 years of age), otherwise healthy, large-breed hunting dogs such as Labrador and golden retrievers, Doberman pinschers, Siberian huskies, German shepherds, and Rhodesian ridgebacks. No clear sex predisposition has been identified, but of cases reported in the literature, males have out numbered females. Affected cats have been outdoor cats.

Considering that rhinosporidiosis is associated with exposure to water and the agent belongs to a branch of aquatic parasites, it has been proposed that aquatic animals are the natural hosts and that the mammalian hosts acquire infection by contacting contaminated water. Therefore, there is a need for the investigation of the infection in fish besides mammalian animals as reservoirs as well as to conduct screening of antiparasitic drugs with infected fish or infected cell lines with the nearest phylogenetic relatives of *R. seeberi*.

Transmission

Mode of transmission of *R. seeberi* is not precisely understood, disease often occurs in **association with contact with aquatic or marshy environments**. It is thought that spores are released into the environment, where they come into contact with susceptible human and animal hosts. Spores may be introduced into tissues after trauma to the mucous membranes, where they form sporangia that produce new spores. Because disease in humans also occurs in arid regions, airborne spore transmission has also been suggested (Sykes, 2021).

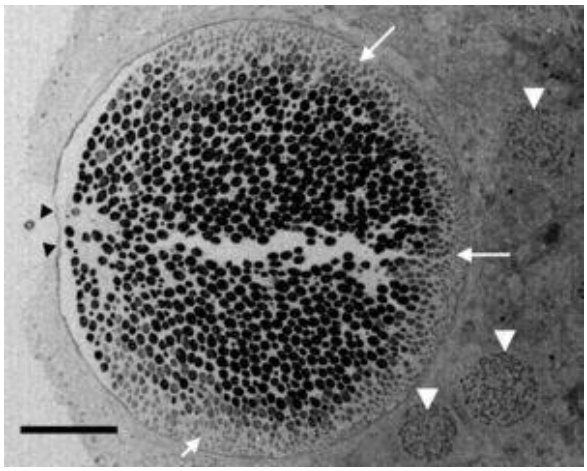
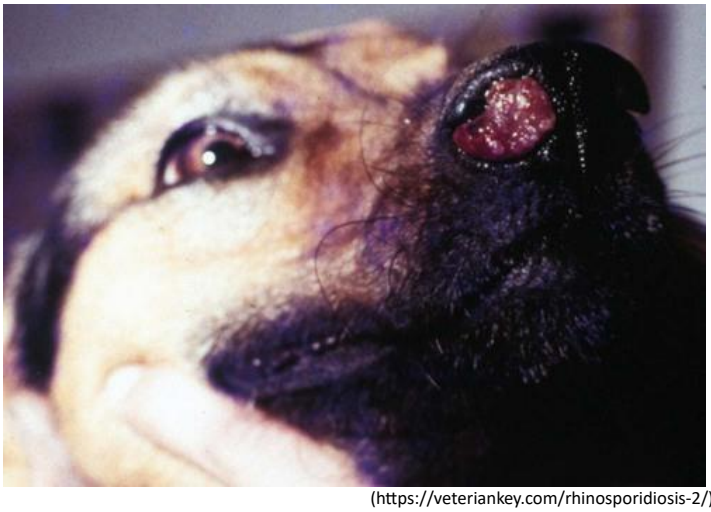


Fig. 1. Electron microscopy tissue section showing a *Rhinosporidium seeberi* mature sporangium containing hundreds of endoconidia, one already outside the sporangium and the others in the process of being expelled through a pore (arrow heads)



(https://veteriankey.com/rhinosporidiosis-2/)

Fig. 2 Two-year-old male dog of mixed German shepherd breeding from Argentina, with a bright red sessile growth in the right nostril. Rhinosporidiosis was diagnosed after histologic examination.

Clinical Signs

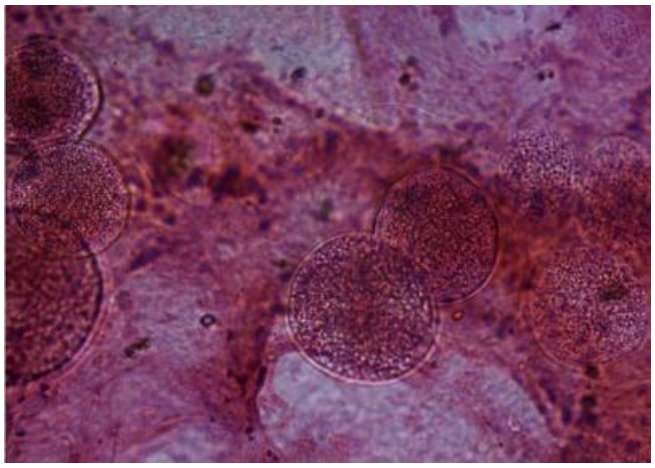
Tumor-like polyps developing primarily in the nostrils and conjunctiva in human and animals. This disease is characterized by the presence of large, round-shaped mature stage and small endospores with **resistance to culturing.**

Nasal rhinosporidiosis may present with unilateral nasal obstruction or epistaxis. Other symptoms may include **local pruritus, coryza with sneezing, rhinorrhea, and postnasal discharge (drip) with cough**.

Diagnosis

Cytodiagnosis on aspirates from rhinosporidial lumps or on smears of secretions from the surfaces of accessible polyps and fine-needle aspirates from lumps provide, with suitable stains, distinctive diagnostic features. The various developmental stages of sporangia can be readily identified by special fungus stains such as the **Gomori methenamine silver, Gridley's, and the periodic acid-Schiff stains**, although the identification of the stages can also be made with the routine haematoxylin and eosin stain.

On direct examination, the cytological smears show **spherules** as well-circumscribed, globular structures with several endospores within. The diameter of spherules ranges from 30 to 300 microns. The endospores may be confused with epithelial cells. The PAS stain is used to discriminate between endospores and epithelial cells, in which the residual cytoplasm and large nuclei can sometimes simulate the residual mucoid sporangial material around the endospores and the endospores themselves. The endospores stain markedly magenta while the epithelial cells are PAS-negative. Histological examination is thus necessary for the definitive diagnosis of rhinosporidiosis.



Rapid Hematoxylin & Eosin (H&E) and Periodic acid Schiff (PAS) stained smears revealed numerous globular sporangia containing spores along with many free lying spores inflammatory cells.

Rhinosporidium seeberi should be distinguished from another microorganism, *Coccidioides immitis*. This latter has similar mature stages represented by large, thick-walled, spherical structures containing endospores, but the spherules are smaller (diameter of 20–80 μm versus 50–1000 μm) and contain small endospores (diameter of 2–4 μm). Moreover, *Coccidiodes* does not stain with the mucicarmine.

Treatment

The first line of treatment is usually **total surgical excision and electro-cauterization of the polyp base**. Treatment for rhinosporidiosis is by excision of the lesions. Surgical margins should be wide to avoid recurrence. Freezing the entire lesion or the base of the lesion after excision may be effective (authors' experience). Therapeutic results have been obtained in dogs, when surgical excision was not possible, by administering dapsone (diaminodiphenylsulfone), a drug used for treatment of leprosy.

Among the drug therapies attempted, **remission has been reported** in some patients who received only Dapson treatment. Common in history of patients bathing in stagnant water.

Reference

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