Rhinosporidiosis

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Rhinosporidium seeberi is the etiologic agent of a chronic, and usually painless, localized granulomatous infection of the mucous membranes of the sinonasal tract, conjunctiva, and urethra. Endemic in India and Sri Lanka, the disease is becoming more significant as a result of migration. R seeberi has not been definitively cultured but is thought to be a blue-green algae, with the infectious agent being a thick-walled sporangium containing endospores. It is passed to humans from animals (cats, free-grazing horses) or possibly fomites, identified in water or soil contaminated by waste.

Common symptoms are nonspecific and include nasal obstruction, epistaxis, and rhinorrhea. Nasal and urethral infections have a male predominance, while conjunctival infections have a female predominance. All ages are affected, although there is a peak in the third and fourth decades.

Surgical treatment is the mainstay of therapy for rhinosporidiosis, but there is a 10% recurrence rate. While dapsone may be helpful, antibiotics for the most part are ineffective.

The lesions clinically form single or multiple polypoid, friable, red-to-pink masses, frequently mistaken clinically for neoplasia. Rhinosporidiosis is characterized histologically by a well-developed acute and chronic inflammation that surrounds round, thick-walled sporangia (up to 300 μm) (figure 1) filled with hundreds of small (2 to 9-μm endospores/algae) (figure 2). There may be overlying surface hyperplasia (pseudoepitheliomatous hyperplasia). The cyst walls are thick and birefringent, stained with hematoxylin and eosin (deeply magenta), Grocott Gomori methenamine silver stain (GMS), digested periodic acid–Schiff, and Mayer mucicarmine. The sporangia are below the surface, not within the epithelium.

The clinical and pathology differential diagnosis includes squamous papilloma, oncocytic type; Coccidioidomycosis immitis; and other infectious agents.

Suggested reading

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