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Rhinosporidiosis in Cattle and Buffaloes

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Abstract

Rhinosporidium seeberi is the causative agent of rhinosporidiosis, a granulomatous, mucocutaneous infection. The majority of recorded cases of eye infections have happened in hot, dry climates. The clinical features of a painless, friable, polypoid mass, which is typically unilateral, can aid in diagnosis, but histological results are the gold standard. The only method of treatment for rhinosporidiosis is surgical excision of the afflicted area with deep cauterization to avoid recurrence because antifungal and antibacterial medications are ineffective against *Rhinosporidium seeberi.*

Keywords: *Rhinosporidiosis*, *Rhinosporidium seeberi*, Granulomatous infection

Introduction

Rhinosporidiosis is a chronic granulomatous infection, noncontagious, sporadic, benign, usually non-fatal disorder caused by *Rhinosporidium seeberi* (Sefu and Fauzia, 2011). Guillermo Seeberi found *R. seeberi* in Argentina in 1990. *Rhinosporidium*, the causative agent, frequently causes granulomatous inflammation of the upper respiratory mucosa. As a result of its physical characteristics being similar to those of fungi and protozoa, it is frequently thought to be a fungus (Tiwari *et al*., 2015). It is endemic to India and affects both people and domestic animals. The condition typically affects the mucous membrane of the nose and nasopharynx, though there have also been cases where it has affected the larynx, genitourinary system, eye, ear, trachea, and bronchi (Babu *et al*., 2012).

In contrast to cattle, the millet-like eruption and nasal mucosa congestion in buffalo indicate a more specific host. Nasal epithelium growth is shown as granulomas in chronic infections. This

granuloma is a cylindric, ulcerated, and pedunculated growth that blocks one nostril's anterior entrance (Kumar *et a*l., 2016). A polypoid, soft mass of the nose (the most common site), eye, conjunctiva, or urethra is how the disease manifests clinically. Another distinguishing characteristic is osteolytic bone infiltration (Nambiar *et al*., 2017). Many farm, domestic, and wild animal species, such as cattle, buffaloes, dogs, cats, goats, horses, mules, and several kinds of ducks, swans, and waterfowl, are susceptible to them (Vilela *et al.,* 2012).

Pathophysiology, life cycle and transmission

Rhinosporidium seeberi infects the epithelium (transepithelial infection), which is most frequently detected in nasal locations (Lupi *et al*., 2005). Although the exact processes of transmission are unknown, the pathogen is thought to enter through the traumatized epithelium. For instance, *Rhinosporidiosis* in river workers is brought on by sand abrasions and contact with groundwater, which is thought to be a reservoir for *R. seeberi*. The mucous membranes of the nose and nasopharynx are the area's most frequently affected, followed by the oropharynx, trachea, bronchi, ear, eye, and genitourinary system. Through the lymphatic system and blood, it can also spread to other locations.

Many sporangiospores or endospores are present in the sporangia, the mature form of the microbe. As endospores come into touch with tissues, they are released and activated, and they continue to invade the area persistently before maturing into trophocytes. Animal urine and feces also contain the fungus' spores, which can harm swimmers in contaminated ponds. The mammal host develops cellular and immunological responses (De Silva et al., 2001).

Microscopic examination

Macroscopically, the growth was cylinder-shaped, reddish, and mucus-coated. *Rhinosporidium seeberi* appears as huge, spherical formations that range in size from 50 to 100 mm and appear as yellowish, pinhead-sized patches on the polyp. These microscopic structures feature a heavily eosinophilic wall that encloses smaller, spherical structures made of amorphous eosinophilic material. The size variation of these structures corresponds to different phases in the development of the organism. (Duarte and others, 2015).

The stains Grocott-methenamine Gomori's silver, periodic acid-Schiff, and mucicarmine are used to reveal the organism's microscopic characteristics (Arseculeratne *et al*., 2005; Guarnerand Brandt, 2011). Low magnification microscopic analysis of H&E-stained sections revealed numerous small (immature) to big (mature) sporangia and highly inflammatory connective tissue in the stroma.

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Clinical manifestations

In animals, distinctive clinical symptoms included dyspnoea, rhinitis, mucus production, and snoring. Mucous membranes that have been deeply and specifically infected with rhinosporidiosis. It typically manifests clinically as a polypoid, soft tissue mass in the nose, throat, eye, or ear, though it can also develop in the oropharynx, larynx, trachea, bronchi, or genitourinary tract (Latchumikanthan *et al*., 2014, Duarte *et al.*, 2015).

Clinically, the disease may present in one of four forms: disseminated, nasal, ocular, or cutaneous. Cutaneous lesions can emerge as papules or nodules under the skin that resemble warts (Putthia *et al*., 2018). The primary symptom of the condition is the development of a polypoidal, reddish, friable, pedunculated, hyperplastic soft tissue mass in the nasal region, which normally progresses slowly and chronically (Shastry *et al.*, 2018).

Diagnosis and treatment

Polyps has been described for diagnosis, pathognomonic histopathology of excised lesions (Singh *et al*., 2017). The best way to treat this condition is with a full surgical excision (Justice *et al.*, 2013). To stop recurrence, applying cautery or cryopexy to the base of the lesion that was removed may be a useful adjunct. Amphotericin B (by local injection) has been recommended as an adjunctive therapy.

Recently, multidrug techniques in the treatment of disseminated disease utilizing cycloserine, dapsone, and ketoconazole have been proposed due to the incidence of refractory cases (Bhat, 2014). Although clinical and radiographic detection of granulomas might be useful, the histology of granulomas is the definitive diagnostic tool.

Conclusion

Nowadays, *Rhinosporidiosis* is regarded as an emerging infectious illness. *Rhinosporidium seeb eri*, a pathogen that has been recognized for more than a century, is the culprit. It contributes to the onset of a chronic, slow-progressing granulomatous disease and typically displays benign polypoid behavior, primar ily involving the nose, nasopharynx, and/or eyes.

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