

Disseminated cutaneous rhinosporidiosis: Varied morphological appearances on the skin

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ABSTRACT

Rhinosporidiosis is a chronic granulomatous disorder caused by *Rhinosporidium seeberi*. It frequently involves the nasopharynx and occasionally affects the skin. We report a case of 45-year-old man who had disseminated cutaneous rhinosporidiosis with cutaneous pseudohorn, its base arising from rhinosporidiosis. The case presented with multiple reddish lesions over the nose of six years duration. In the past year, he developed skin lesions over the left arm, forearm, back, and chest. On examination, polymorphic lesions of rhinosporidiosis in form of verrucous plaque, un ulcerated tumor, granulomatous growth, and furunculoid lesions were observed. Interestingly, there was a cutaneous horn over the chest which on histopathological examination showed hyperplastic epithelium with numerous globular cysts of varying shape, representing sporangia in different stages of development and transelimination. Computerized tomography scan of the chest showed bilateral opacities suggestive of lung involvement. On the basis of these clinical and histopathological findings, a diagnosis of nasal rhinosporidiosis with cutaneous and systemic dissemination was made.

Key words: Cutaneous horn, cutaneous rhinosporidiosis, disseminated

INTRODUCTION

Rhinosporidiosis, a chronic granulomatous disorder caused by *Rhinosporidium seeberi* is endemic in India and Sri Lanka but has also been reported from United States, South America, and Iran.^[1] Rhinosporidiosis frequently involves the nasopharynx (70%) presenting as a painless, friable, polypoidal growth, which may hang anteriorly into the nares or posteriorly into the pharynx.^[2] Cutaneous dissemination, although known, is quite rare.^[2] Systemic dissemination is also a possibility due to hematogenous spread of the spores.^[3] Cutaneous lesions in form of verrucous plaques,^[1-5] polypoidal growths,^[1] subcutaneous nodules,^[6] furunculoid lesions, ecthymatoid lesions, etc. is known, but very uncommon. Here, we report of a 45-year-old man with disseminated cutaneous rhinosporidiosis with systemic pulmonary involvement with a cutaneous horn secondary to rhinosporidiosis.

CASE REPORT

A 45-year-old man presented with a 6-year history of

multiple reddish lesions in the nose. Over the past one year, he had developed skin lesions on the left arm and forearm followed by similar lesions over the face, back, and chest. He first noticed a reddish, friable lesion in the right nostril associated with anosmia, nasal block, occasional hemorrhage, and crusting. Two years later, similar lesion evolved in the left nostril. He was a farmer by occupation and gave history of swimming in ponds in his village. The lesions had recurred after an earlier excision done by an otolaryngologist one year back.

On cutaneous examination, a solitary, oval reddish granulomatous growth (1 × 1 cm) with hemorrhagic crusting was seen on the nose medial to the medial canthus [Figure 1]. A hemispherical un ulcerated nodule (5 × 4 cm) was seen over the left forearm [Figure 2] along with a crusted verrucous plaque (4 × 4 cm) seen over the left arm [Figure 3]. Two small furunculoid lesions were seen over the chest [Figure 4] discharging pus. In addition, there was a single horny excrescence 8-mm long suggestive of a cutaneous horn

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Figure 1: Granulomatous growth near inner canthus



Figure 2: Subcutaneous lesion over the forearm



Figure 3: Verrucous plaque over the arm



Figure 4: Furuncle like lesions over the back



Figure 5: Cutaneous horn over the chest



Figure 6: Rhinosporidiosis nasal polyps with extension to the surrounding skin

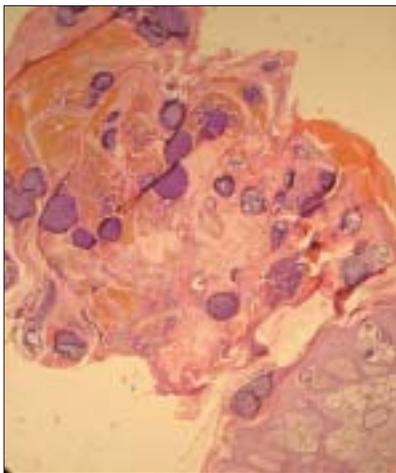


Figure 7: Histopathology section of cutaneous horn showing transelimination of sporangia in different stages of development from the skin to horn (H and E, x40)

overlying a papule on the chest [Figure 5]. On anterior rhinoscopy, reddish friable polyps studded with tiny white dots were seen in bilateral nasal cavities [Figure 6]. Oral cavity examination was normal. Bilateral immature cataracts were seen in the eyes. Systemic examination was normal.

Fine needle aspiration cytology from the lesion on giemsa stain showed lobular thick-walled sporangia. Histopathological examination of the skin biopsy specimen from the representative cutaneous lesion (arm) showed hyperplastic epithelium with numerous

globular cysts of varying shape, representing sporangia in different stages of development. The lesion appearing like cutaneous horn was also excised and sent for histopathological evaluation of the base which also turned out to be a rhinosporidiosis papule. Interestingly, the sporangia were seen extruding between the hyperplastic keratin layers of the horn along with areas of thrombosis [Figure 7]. On X-ray and CT scan, cystic lesions were also seen in the lungs. Ultrasound of the abdomen showed no focal lesions in liver or spleen. His serology for HIV infection by ELISA was negative. A final diagnosis of nasal rhinosporidiosis with cutaneous and systemic dissemination was made. All the cutaneous lesions were excised and he was then referred to an otolaryngologist for endoscopic removal of the nasal lesions. Following this, he was started on oral dapsone (100 mg/day).

DISCUSSION

Nasal rhinosporidiosis usually affects males (70–90%), and the incidence is greater in those aged between 20 and 40 years.^[7] Ocular infection is more prevalent in women, while nasal and nasopharyngeal infections preferentially affect males. The lesions are pink or purple-red friable polyps studded with minute white dots (strawberry like), which are sporangia containing the spores. Nasal obstruction and bleeding are the most common symptoms. The conjunctiva and lacrimal sac are involved in 15% of cases. Occasionally, rhinosporidiosis affects the lips, palate, uvula, maxillary antrum, epiglottis, larynx, trachea, bronchus, ear, scalp, vulva, vagina, penis, rectum, and the skin.^[7] In our case, nasal mucosa was the mucosal site involved along with extension to surrounding skin.

Cutaneous lesions in rhinosporidiosis are not very common and usually start as friable papillomas that become pedunculated. Cutaneous rhinosporidiosis may also present as warty papules and nodules with whitish spots, crusting, and bleeding on the surface. Three types of skin lesions can occur: (1) satellite lesions, in which skin adjacent to the nasal rhinosporidiosis is involved secondarily; (2) generalized cutaneous type with or without nasal involvement, occurring through hematogenous dissemination of the organism; and (3) primary cutaneous type associated with direct inoculation of organisms on to the skin.^[1] The various clinical differential diagnoses include warts, verrucous tuberculosis, and granuloma pyogenicum. Histological

differential diagnoses must include pyogenic granuloma, coccidioidomycosis, and myospherulosis, an iatrogenic condition related to application of nasal substances.^[7]

The diagnosis can easily be clinched by performing a giemsa-stained imprint smear or fine-needle aspiration cytology from the lesion.^[8]

Histopathology^[9] reveals enormous number of mycotic elements in the subepithelial connective tissue. These elements consist of sharply defined globular thick-walled cysts (sporangia), up to 0.5 mm in diameter, which contain numerous rounded endospores, 6–7 μ in diameter. Immature and collapsed sporangia are also present. It is not necessary to perform special staining because of the size of the agent. This disease must be differentiated from coccidioidomycosis whose different clinical presentation and smaller sporangia size (<60 μ in diameter) allow for an easy distinction. The cultivation of *R. seeberi* was performed by Levy *et al*, but was not confirmed by other researchers.^[7]

The taxonomy of the agent has always been controversial. Guillermo Seeber, who first described the disease in 1896, considered the sporangium of *R. seeberi* to be a sporozoan similar to coccidian. Though the agent was considered a fungus it was interpreted as a protozoan parasite, a cyanobacterium, and a carbohydrate waste product.^[7] Arseculeratne *et al*,^[10] had suggested that the phenomenon of ‘transepidermal elimination’ of sporangia of *R. seeberi* is rather the pathogen’s mechanism for endospore-dispersal than a nonspecific defense reaction of the host as previously thought,^[11] as seen on histology of ocular rhinosporidiosis. This theory is supported by our case as the histology showed sporangia in various stages of migration coming to the layers of epidermis coming to surface in the cutaneous horn. Juvenile, intermediate, and mature sporangia were noted in the subepithelial tissue and within the epithelium. These locations could suggest a progressive movement of the sporangia toward the epithelium from subepithelial sites. Herr *et al*,^[12] recently proposed that the organism should be considered in a new eukaryotic group of protists known as Mesomycetozoa. Fredericks *et al*,^[13] also agree with the concept of a novel clade of aquatic protistan parasites named Ichthyosporea, pointing to the similarities with other members of the DRIP clades (named for the organisms Dermocystidium, the Rosetta agent, Ichthyophonous, and Psorospermium) that infest

fish and amphibia. *Dermocystidium ranae* has also shown a similar process of 'transepidermal elimination', suggesting it is rather a device of the pathogens to expel and propagate their endospores. Ahluwalia *et al.*^[14] suggested, however, that the causative agent of rhinosporidiosis is the cyanobacterium *Microcystis aeruginosa*, isolated from clinical samples as well as from water samples in which patients had been bathing.

The life cycle of the parasite is complicated. The mature forms of the organism, known as sporangia, contain multiple sporangiospores. The trophocytes, the immature forms of *R. seeberi*, are smaller and thinner than sporangia and do not contain endospores. Sporangiospores are released at maturity and thereafter develop into trophocytes. It is possibly transmitted to humans by direct contact with spores through dust, through infected clothing or fingers, and through swimming in stagnant waters.^[1]

Surgical removal and electrodesiccation are the treatments of choice. Dapsone may arrest the maturation of sporangia and accelerate degenerative changes in them. The effete organisms are then removed by an accelerated granulomatous response.^[7]

Our case fits into the description for generalized cutaneous type with six different morphological types (satellite lesion adjacent to the nose, verrucous plaque, subcutaneous nodule, granulomatous growth, furunculoid lesion, and a cutaneous horn coexisting simultaneously). Involvement of lungs due to visceral dissemination has been reported but is very uncommon.^[15] Morphological variants of cutaneous rhinosporidiosis (recently called as dermosporidiosis) like verrucous plaque, subcutaneous nodules, granulomatous growth, and furunculoid and ecthymatoid lesions have been reported. There is no report of cutaneous horn-like lesion secondary

to rhinosporidiosis. Two other infections reported to have resulted in cutaneous horn include molluscum contagiosum and cutaneous leishmaniasis.

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