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RESEARCH ARTICLE

OUR EXPERIENCE IN AN ENDEMIC RHINOSPORIDIOSIS DISTRICT:

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ABSTRACT

Rhinosporidiosis and its causative pathogen *Rhinosporidium seeberi* have been known for over a hundred years. Yet unresolved enigmas in rhinosporidiosis include the mode of infection, mechanisms of spread, mechanisms of immunity, and some aspects of histopathology. This article discuss about the usual presentation of rhinosporidiosis and how effectively it was managed in endemic district.

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INTRODUCTION

Rhinosporidiosis a tropical disease of ENT has always been a subject of controversies and changing scenarios. Being a country which has endemic areas ,this article has been to share our experience of Ent surgeons of peripheral teaching institute in a place named Thiruvarur of Tamilnadu, the southern tip of India.

Rhinosporidiosis

Theories

Theories of mode of spread

- Demellow's theory of direct transmission
- Autoinoculation theory of Karunarathnae (responsible for satellite lesions)
- Haematogenous spread -to distant sites
- Lymphatic spread -causing lymphadenitis (rarity)

Demellow's theory of direct transmission -
This theory propounded by Demellow had its acceptance for quite some time. He postulated that infection always occurred as a result of direct transmission of the organism.

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When nasal mucosa comes into contact with infected material while bathing in common ponds, infection found its way into the nasal mucosa. Karunarathnae accounted for satellite lesions in skin and conjunctival mucosa as a result of auto inoculation. Rhinosporidiosis affecting distant sites could be accounted for only through haematogenous spread. Karunarathnae also postulated that Rhinosporidium existed in a dimorphic state. It existed as a saprophyte in soil and water and it took a yeast form when it reached inside the tissues. This dimorphic capability helped it to survive hostile environments for a long period of time. Life cycle (recent): Since rhinosporidium seeberi has defied all efforts to culture it, any detail regarding its life cycle will have to be taken with a pinch of salt. This life cycle has been postulated by studying the various forms of rhinosporidium seen in infected tissue. Trophozoite / Juvenile sporangium -It is 6 -100 microns in diameter, unilamellar, stains positive with PAS, it has a single large nucleus, (6micron stage), or multiple nuclei (100 microns stage), lipid granules are present. Intermediate sporangium -100 -150 microns in diameter. It has a bilamellar wall, outer chitinous and inner cellulose. It contains mucin. There is no organized nucleus, lipid globules are seen. Immature spores are seen within the cytoplasm. There are no mature spores. Mature sporangium -100 -400 microns in diameter, with a thin bilamellar cell wall. Inside the cytoplasm immature and mature spores are seen. They are found embedded in a mucoid matrix. Electron dense bodies are seen in the cytoplasm. The bilamellar cell wall has one weak spot known as the operculum.

Maturation of spores occur in both centrifugal and centripetal fashion. This spot does not have chitinous lining, but is lined only by a cellulose wall. The mature spores find their way out through this operculum on rupture. The mature spores on rupture are surrounded by mucoid matrix giving it a comet appearance. Mature spores give rise to electron dense bodies which are the ultimate infective unit.

Endemicity: Of all the reported cases 95 % were from India and Srilanka. An all India survey conducted in 1957 revealed that this disease is unknown in states of Jammu & Kashmir, Himachal Pradesh, Punjab, Haryana, and North Eastern states of India. In the state of TamilNadu 4 endemic areas have been identified in the survey, (Madurai, Ramnad, Rajapalayam, and Sivaganga). The common denominator in these areas is the habit of people taking bath in common pondsReasons for endemicity of Rhinosporidiosis has to be explained why this disease is endemic in certain parts of South India and in the dry zone of Srilanka. If stagnant water could be the reason then the chemical and physical characteristics of the water needs to be defined. In addition other aquatic organisms may also be playing an important synergistic reaction. This aspect need to be elucidated. Text book of microbiology is replete with examples of such synergism i.e. lactobacillus with trichomonas, and Wolbachia with filarial nematodes. Host factors responsible for endemicity: Eventhough quite a large number of people living in the endemic areas take bath in common ponds only a few develop the disease. This indicates a predisposing, though obscure factors in the host. Blood group studies indicate that rhinosporidiosis is common in patients with group O (70%), the next high incidence was in group AB. Jain reported that blood group distribution is too variable to draw any conclusion. Larger series must be studied for any meaningful analysis. HLA typing also must be studied. The possibility of non-specific immune reactivity especially macrophages in protecting the individual from Rhinosporidium seeberi must be considered.

Our experience: We as ENT surgeons of regional institute had been attending to rhinosporidiosis on all theatre days atleast 2 patients per week surprisingly. The new institute had rhinosporidiosis excision and cautery as our regular surgical procedure.Thus started our journey into the rhinosporidiosis case. We follow endoscopic excision and base cauterization and post-operative dapsone as the modality of treatment. From our experience we did not have a single recurrence of the surgical patients for a follow up of 5 years from 2011 to 2015.
But the interesting cases were three in number out of about 240 patients treated.The first one was young lady about 19 years who had been operated 14 years before for rhinosporidiosis. And when she came to our opd we saw an atypical whitish mass in the nasopharynx. Considering her age and the appearance we thought it could be Hodgkin's lymphoma. Anyway planned for an endoscopic excision biopsy. We found it difficult to maneuver the mass through the nasal cavity as it was fibrotic and firm unlike rhinosporidiosis.so we delivered it orally by using a Boyle daves mouth gag. Surprisingly HPE reported it as rhinosporidiosis. The next case was an old man about 55years with a mass in the nasal cavity and nasopharynx hanging into the oropharynx. The appearance was that of rhinosporidiosis. Here while excision we found that the mass was adherent to soft palate and uvula.so it was clamped and cauterized and then removed. The last one was a rhinosporidiosis of the lacrimal sac. With the ophthalmologist help it was excised and sent for HPE.

Conclusion

This has been to sensitize and share fellow ENT surgeons about the variations in the presentations of rhinosporidiosis. as in places other than endemic areas incidence is rare. Hence the diagnosis can be missed because they may present atypically. The treatment role should be played wisely since the recurrence rate is also high especially in endemic area.

REFERENCES

Arseculeratne SN. 2000. An update on rhinosporidiosis and Rhinosporidium seeberi. 2nd SAARC ENT Congress, Kathmandu, Nepal; guest lecture.

Arseculeratne SN., Atapattu DN., Rajapakse RPVJ., Balasooriya P., Fernando R., Wijewardena T. 1999. The humoral immune response in human rhinosporidiosis. *Proc KandySoc Med.,*21: 9.

Arseculeratne, SN, Panabokke RG, Atapattu DN. 2002. Lymphadenitis, trans-epidermal elimination and unusual histopathology in human rhinosporidiosis. *Mycopathologia,* 52:57-69.

Ashworth JH. On Rhinosporidium seeberi (Wernicke, 1903) with special reference to its sporulation and affinities. Trans Roy Soc, Edin 1923; 53:301-342.

Atapattu DN., Arseculeratne SN., Rajapakse RPVJ., Perera NAND., Eriyagama NB. 1999. Purification of the endospores and sporangia of Rhinosporidium seeberi on Percoll columns. *Mycopathologia,*145:113-119.

Atapattu DN., Arseculeratne SN., Rajapakse RPVJ., Perera NAND., Razak MAA. 2000. Is human IgG bound by endospores and sporangia of Rhinosporidium seeberi? - A possible mechanism of immune evasion. *Proc Kandy Soc Med.,*22:34.

Beattie JM. 1906. Rhinosporidium kinealyi: a sporozoon of the nasal mucous membrane. *J Path Bact.,*11:270-275.

Chau KY., Pretorius JM., Stewart AW. 2000. Myospherulosis in Renal Cell Carcinoma. *Arch Path Lab Med.,*124:1476-1479.

Chitravel V., Sundararaj T. 1982. Detection of circulating antigen in patients with rhinosporidiosis. *Sabouraudia. ,*20:185-191.

Chitravel V., Sundararaj V., Subramanian S., Kumaresan M., Kunjithapadam S. 1981. Cell mediated immune response in human cases of rhinosporidiosis. *Sabouraudia,*19:135-142.

Daszak P., Berger L., Cunningham A., Hyatt AD., Green DE., Speare R. 1999. Emerging infectious diseases and amphibian population declines. *Emerging Infectious Diseases.* 5(6):735-748.

De Mello MT. 1954. Rhinosporidiosis. In Medical Mycology. RDGPh Simons Ed. (Elsevier Publ.Co., Amsterdam):368-383.

De Silva NR., HuegelHeino, Atapattu DN., Arseculeratne SN., Kumarasiri R., Gunawardena S., Balasooriya P., Fernando R. 2001. Cell-mediated immune responses in human rhinosporidiosis. *Mycopathologia,*152:59-68.

Herr RA., Ajello L., Taylor JW., Arseculeratne SN., Mendoza L. 1999. Phylogenetic analysis of Rhinosporidium seeberi's 18S Small-subunit Ribosomal DNA groups this pathogen among members of the ProtoctistanMesomycetozoa clade. *J clinmicrobiol,*37(9):2750-2754.

Herr RA., Mendoza L., Arseculeratne SN., Ajello L. 1999. Immunolocalization of an endogenous antigenic material of Rhinosporidium seeberi expressed only during mature

sporangial development. *FEMS Immunol Med Microbiol.,* 23:205-212.

Hutt MSR., Fernandes BJ., Templeton AC. 1971. Myospherulosis (Subcutaneous Spherulocystic Disease). Trans Roy Soc Trop Med Hyg., 65:182-188.

JayasekeraSharmini, Arseculeratne SN., Atapattu DN., Kumarasiri R., Tilakaratne WM. 2001. Cell-mediated immune responses (CMIR) to Rhinosporidium seeberi in mice. *Mycopathologia,*152:69-79.

Job A., Venkateswaran S., Mathan M., Krishnaswami H., Raman R. 1993. Medical therapy of rhinosporidiosis with dapsone. *J Laryngol Otol.,* 107:809-812.

Karunaratne WAE. 1934. The pathology of rhinosporidiosis. *J Path Bact.,*XLII:193-202.

Karunaratne WAE. 1964. Rhinosporidiosis in Man: (The Athlone Press, London).

McClatchie S., Bremner AD. 1969. Unusual subcutaneous swellings in African patients. *East Afr Med J.,*46:625-633

MinchinEA., Fantham HB. 1905. Rhinosporidium kinealyin.g., n.sp., a new sporozoon from the mucous membrane of the septum nasi of man. *Quart J Microscop Sci.,*49:521-532.

Mosmann TR., Sad S. 1996. The expanding universe of T-cell subsets: Th1, Th2 and more. Immunology today, 17(3):138-146.

Nair KK. 1979. Clinical trial of diaminodiphenylsulphone (DDS) in nasal and nasopharyngeal rhinosporidiosis Laryngoscope, 89:291-295.

Rajam RV, Viswanathan GC. Rhinosporidiosis: A study with a report of a fatal case with systemic dissemination. Ind J Surg 1955;17:269-298.

Reddy D., Lakshminarayana CS. 1962. Investigation into transmission, growth and serology in rhinosporidiosis. *Indian J Med Res.,*50:363-370

Thianprasit M., Thagernpol K. 1989. Rhinosporidiosis. *Curr Top Med Mycol.,*3:64-85.

Tirumurti TS. 1914. Rhinosporidium kinealyi. The Practitioner 93:704-719.

Vanbreuseghem R. 1958. Mycoses of man and animals, (Sir Isaac Pitman & Sons, London).

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