DOI: 10.14260/jemds/2014/3575

ORIGINAL ARTICLE

A CLINICAL STUDY OF RHINOSPORIDIOSIS IN RURAL COASTAL POPULATION: OUR EXPERIENCE

Jarvis Raju Vadakkan[1], Ganeshbala A[2], Jalagandesh B[3]

HOW TO CITE THIS ARTICLE:
Jarvis Raju Vadakkan, Ganeshbala A, Jalagandesh B. "A Clinical Study of Rhinosporidiosis in Rural Coastal Population: Our Experience". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 51, October 09; Page: 11938-11942, DOI: 10.14260/jemds/2014/3575

ABSTRACT: INTRODUCTION: Rhinosporidiosis is a chronic infective disorder that is caused by Rhinosporidium seeberi. It usually presents with a soft polypoidal pedunculated or sessile mass arising from the nasal cavity. Nasal cavity and nasopharynx are the more common sites, followed by conjunctiva, maxillary sinuses and larynx. **AIM:** The aim of this study is to determine the age-sex distribution of rhinosporidiosis and evaluation of blood group that is commonly seen with rhinosporidiosis in a large group of study. **MATERIALS AND METHODS:** A cross sectional study of 100 patients was conducted in the outpatient department of ENT, Vinayaka mission medical college, Karaikal for a period of 2 years from August 2012- August 2014. During the study period, all cases that were diagnosed as rhinosporidiosis by histopathology were included in the study group. **RESULT:** Majority of patients in our study were young adults with a male preponderance. It showed an association with O blood group type. The common sites involved were the nasal cavity and nasopharynx followed by conjunctiva and larynx. **CONCLUSION:** Rhinosporidiosis is a disease with high degree of recurrence which requires careful assessment with panendoscopy, early detection and treatment. Careful follow up is very essential.

KEYWORDS: Rhinosporidiosis, Rhinosporidium seeberi, Dapsone.

INTRODUCTION: Rhinosporidiosis is a chronic granulomatous infective disorder that is caused by Rhinosporidium seeberi, whose taxonomy is still debated.[1] The first case was described by Guillermo Seeber from Buenos Aires in 1900. The causative organism was initially considered as a fungus, and As worth in 1923 pioneered in describing its life cycle establishing the nomenclature Rhinosporidium seeberi.[2]

The present state of research concerning the taxonomy of R. seeberi: whether it is a prokaryotic Cyanobacteria or a eukaryotic parasite is controversial. Recent studies found that a small subunit sequence of r DNA to be similar to that of members of the Dermocystidium genus: aquatic protistan fish parasites that belong to the DRIP clade. Though sporadic cases of rhinosporidiosis are reported from all over the world, almost more than 90% cases are reported from India, Sri Lanka and Pakistan.

Clinically, rhinosporidiosis presents as a polypoidal soft tissue mass, often pedunculated. Nasal cavity and nasopharynx are the commonest sites that are involved, accounting for more than 70% cases. Ocular lesions, particularly of the conjunctiva and lacrimal sac, account for 15% cases. Rare sites of involvement are lips, palate, uvula, maxillary antrum, epiglottis, larynx, trachea, bronchus, ear, scalp, vulva, penis, rectum and skin. Rarely, disseminated infections are also reported, involving limbs, trunks and viscera. Brain involvement may lead to fatality. Rarely, spontaneous regression of rhinosporidial nasal polyps has been documented.[3,4]

DOI: 10.14260/jemds/2014/3575

MATERIALS AND METHODS: The present study was conducted in the outpatient department of ENT, Vinayaka mission medical college, Karaikal for a period of 2 years from 2012- 2014. A total of 50 patients were included in our study. During the study period, all cases that were diagnosed as rhinosporidiosis by histopathology were included in the study group.

Detailed clinical data regarding age, sex and clinical presentations were collected. Routine blood examination with especially with ABO blood grouping was done in all cases of the study group. Histopathological samples were processed according to the standard recommendations. Apart from the routine hematoxylin and eosin staining (H and E), PAS and mucicarmine stains were also used. Final diagnosis was achieved by demonstrating thick-walled sporangia containing numerous endospores in a background of a fibrovascular stroma.

RESULTS:

AGE DISTRIBUTION:

Age group	No. of cases
Below 20 years	3(6%)
21-40 years	15(30%)
41-60 years	28(56%)
Above 60 years	4(8%)
Total	**50**

The most common age group affected was 20-40 years [Table 1]

Sex distribution

Sex distribution	No. of cases
Males	32(64%)
Females	18(36%)

Out of the 50 patients, 32 were males and 18 were females.

Site involved

Site	No. of cases
Nose and nasopharynx	45(90%)
Ocular	2(4%)
Larynx	1(2%)
Others (skin and hard palate)	2(4%)

DOI: 10.14260/jemds/2014/3575

Nasal cavity and nasopharynx were the most commonly affected sites (90%), followed by eye (4%). Other rare sites accounted for 3 (6%) cases.

PATIENT WITH LARYNGEAL RHINOSPORIDIOSIS WHO UNDERWENT TEMPORARY TRACHEOSTOMY (LEFT). THE SAME PATIENT WITH BILATERAL NASAL RHINOSPORIDIOSIS (RIGHT).

Most common presentation of the cases was presence of a mass in the nasal cavity, followed by nasal obstruction, epistaxis, and rhinorrhea and watering of the eyes. One patient presented with 42 episodes of recurrent rhinosporidiosis which was extensive from the nasal cavity to the nasopharynx. One patient presented with disseminated rhinosporidiosis involving the conjunctiva, larynx and the nasal cavity. ABO blood grouping was also done. Among them, blood group "O" was the most common (75%), followed by blood group "A"(20%).

DISCUSSION: R. seeberi is no longer considered as a classic fungus. Herr et al.[5] classified this organism as Mesomycetozoa which includes other fish and amphibian pathogens. Ground water is considered to be the natural habitant of R. seeberi. Human infection is presumed to occur due to contact of traumatized epithelium with contaminated water. Highest incidence of cases is reported among river-sand workers.[6] As our medical college is situated in a rural area, it caters to a large population of poor villagers and fishermen who are accustomed to take bath in ground water and are prone to mucosal injuries by sand or dust.

Male cases outnumbered female cases in our series (64% vs. 36%). Most of them belong to the age group of 20- 40 years of age. Nose and nasopharynx were involved in most (90%) of the cases, followed by eyes (2%). Rare sites of involvement in our series were lip, skin and larynx. Other workers reported similar experiences.[7] Rhinosporidiosis of nasal passage usually appears as a polypoid mass, granular, red in color with multiple yellowish pin head-sized spots representing underlying mature sporangia.

This gross appearance, though distinctive, is not diagnostic.[8] Rhinosporidial polyps are also reported from skin, subcutaneous tissue, conjunctiva and urethral meatus. Nasopharyngeal lesions

are often multilobed. Nasal obstruction is a prominent symptom in these cases. Epistaxis and rhinorrhea are the common manifestations of nasal and nasopharyngeal infestation.[7,9]

Watering of eyes, redness and itching are the prominent manifestations of ocular rhinosporidiosis. Our experience was similar. Uncommon features noted in our cases were warty growth in the lips, skin. Routine hematological investigations in our series did not reveal any significant abnormality. Majority of the cases presented with normal TLC. There was no significant rise of relative proportion of eosinophils. Similar experience has also been reported by other workers.[4,7-9] ABO blood group study showed that 75% of the cases were blood group O, followed by group A (20%).

Typical polypoid appearance of rhinosporidial lesions often helps in correct preoperative diagnosis. But atypical presentations may cause confusion with soft tissue tumors or papillomas. Aspiration cytology can be helpful in these cases.[10] Material can also be collected by scraping in case of superficially accessible lesions. Microscopically, demonstration of endospores of 5-10 μm and sporangium of 50-1000 μm in the cytological smears clinches the diagnosis. Background epithelioid granulomatous reaction can be evident, but eosinophils are rarely found.

Definitive diagnosis of rhinosporidiosis depends upon identification of the pathogen in its diverse stages on biopsied or resected tissues.[11] Histopathological sections show multiple sporangia in various stages of maturity, enclosed in a thin chitinous wall. The sporangia are 50-1000 μm in diameter, containing numerous endospores of diameter 5-10 μm. Overlying epithelium is usually hyperplastic and loose fibrovascular stroma infiltrated with lymphocytes, macrophages, plasma cells and even polymorphonuclear leucocytes. Rupture of sporangia can cause giant cell reaction.[11]

The mainstay of treatment is surgical excision of the lesion but recurrences are a challenge and the only drug useful in reducing recurrence is dapsone. Total excision of the polyp, preferably electrocautery of the base, is the recommended method of therapy. Recurrence may occur due to spillage of endospores in the surrounding mucosa during removal.[11] The only drug to have anti-rhinosporidal effect is dapsone, but it can only be used as an adjuvant to surgery. MTT assays have been used to determine the antirhinosporidial activity of drugs such as cycloserine, dapsone, trimethoprimsulphadiazine, ketoconazole, sodium stibogluconate and amphotericin B. As far as we know, ketoconazole has been used only once in nasal rhinosporidiosis along with cryotherapy and surgery with complete response.[11]

CONCLUSION: A proper history, clinical examination and combined treatment modality which involves surgical resection and adequate medical treatment with Dapsone still gives good result in treating this tumour like disease which tends to recur. Also this should be combined with regular follow up with panendoscopy to prevent and detect any possible recurrence in the aerodigestive tract.

REFERENCES:

1. Ahluwalia KB. New interpretations in rhinosporidiosis, enigmatic disease of the last nine decades. J Submicros Cytol Pathol. 1992; 24: 109–14.
2. Karunaratne WA. London: Athlone Press; 1964. Rhinosporidiosis in man; pp. 14–8.
3. Vukovic Z, Bobic-Radovanovic A, Latkovic Z, Radovanovic Z. An epidemiological investigation of the first outbreak of rhinosporidiosis in Europe. J Trop Med Hyg.1995; 98: 333–7.

4. Strickland GT. Hunter's Tropical Medicine. Philadelphia, PA: WB Saunders; 1984: 447.
5. Herr RA, Ajello L, Taylor JW, Arseculeratne SN, Mendoza L. Phylogenetic analysis of rhinosporidiumseeberi's18S small-subunit ribosomal DNA groups this pathogen among members of the protoctistan Mesomycetozoa clade. J Clin Microbiol. 1999; 37: 2750–4.
6. Karunaratne WAE. The pathology of rhinosporidiosis. J Path & Bact. 1936; 42: 193–202.
7. Sarker MM, Kibria AKMG, Haque MM. Disseminated subcutaneous rhinosporidiosis: a case report. TAJ. 2006; 19: 31–3.
8. Harissi-Dagher M, Robillard N, Corriveau C, Mabon M, Allaire GS. Histopathologically confirmed ocular rhinosporiodiosis in two Canadians. Can J Ophthalmol. 2006; 41: 226–9.
9. Kumari R, Laxmisha C, Thappa DM. Disseminated cutaneous rhinosporidiosis. Dermatol Online J. 2005; 11: 19.
10. Rippon JW. Medical Mycology, 2nd ed. Philadelphia, PA: WB Saunders; 1982: 325-333.
11. Ahluwalia KB. Causative agent of rhinosporidiosis. J Clin Microbiol. 2001; 39: 413-415.

AUTHORS:

1. Jarvis Raju Vadakkan
2. Ganeshbala A.
3. Jalagandesh B.

PARTICULARS OF CONTRIBUTORS:

1. Final year Post Graduate, Department of ENT, Vinayaka Mission Medical College and Hospital, Karaikal.
2. Assistant Professor, Department of ENT, Vinayaka Mission Medical College and Hospital, Karaikal.
3. 2nd Year Post Graduate, Department of ENT, Vinayaka Mission Medical College and Hospital, Karaikal.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Jarvis Raju Vadakkan,
Bethek Joel,
Vadakkan House,
Kumarapuram Post Office,
Pallikara, Ernakulam-683565,
Kerala.
Email: jarvisraju@gmail.com

Date of Submission: 22/09/2014.
Date of Peer Review: 23/09/2014.
Date of Acceptance: 30/09/2014.
Date of Publishing: 08/10/2014.