# RHINOSPORIDIOSIS IN WESTERN KENYA

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**Abstract:** Epidemiology and histopathology of rhinosporidiosis in Western Kenya are reported. During the period of six years, 1979 to 1984, we found 10 cases of rhinosporidiosis out of 18,969 surgical specimens in Western Kenya (Rift Valley, Nyanza and Western Provinces). The disease was mostly confined to young generations. Mostly affected site of the infection was the nostril, followed by the bulbar conjunctiva. Nyanza Province and the central area of Rift Valley Province were highly infected. All patients came from agricultural areas. Histologically the disease showed characteristic appearances. The various stages in the life cycle of fungal cells were found in the subepithelial connective tissues which was covered by papillomatous hyperplasia of the mucosal epithelium and accompanied with relatively scant inflammatory cell infiltration in spite of huge number of fungal cells. These findings suggest that rhinosporidiosis is one of the unique fungal diseases showing characteristic histological features. And possible source of the infection was discussed.

#### INTRODUCTION

Guillermo Seeber described the first case of rhinosporidiosis in Buenos Aires in 1900. The disease is a chronic granulomatous disease which is caused by one of the zygomycetes, *Rhinosporidium seeberi* (Satyanarayana, 1960).

The most common affected site of the infection is the mucous membrane of the nose and nasopharynx (Karunaratne, 1964). The very vascular, easy-bleeding, cauliflower-like and polypoid lesion protrudes frequently from the nose and occasionally causes respiratory disturbance. Cases of multiple lesions or visceral dissemination of rhinosporidiosis have been reported (Desmond, 1953; Agrawal *et al.*, 1959) but such cases are extremely rare and the disease is seldom fatal (Rajam *et al.*, 1955).

Histologically rhinosporidiosis is characterized by the presence of fungal cells of various stages in the life cycle in the subepithelial connective tissue of infected site.

The disease is endemic in Sri Lanka and India (Karunaratne, 1964). Reports of sporadic cases have been issued from Argentina, Brazil, Iran, the United States, South Africa, Central and East Africa and South Asia.

The mode of transmission remained still unclear, although water-borne infection is suspected (Rippon, 1982). It is, therefore, highly necessary to carry out an epidemiological survey of the disease more intensively.

In the present communication, we report the prevalence of rhinosporidiosis in Western Kenya and histological characteristics of the disease.

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### MATERIALS AND METHODS

Our study is based on the histological examination done on the surgical specimens which were brought to the two hospitals in Western Kenya, the Rift Valley Provincial General Hospital and Nyanza Provincial General Hospital.

During the period of six years, 1979 to 1984, a total of 18,969 surgical specimens were examined. When the specimens arrived the hospitals, the clinical data and general informations relevant to the disease were collected as completely as possible.

Histological examinations were performed by H.E., periodic acid Schiff (P.A.S.), reticulum, elastic van Gieson, methenamine silver and Azan Mallory stains.

#### RESULTS

I. Prevalence of the disease in Western Kenya

Out of 18,969 surgical specimens examined, 10 were diagnosed histologically as rhinosporidiosis. In Table 1, the age, sex, ethnic group and inhabitation of patients and site of infection

Case	Age	Sex	Site of lesion	Ethnic group	District	Province
1	6	М	Nostril	Luo	South Nyanza	Nyanza
2	. 6	М	Nostril	Luo	Kisumu	Nyanza
3	12	Μ	Nostril	Luo	Kisumu	Nyanza
4	18	Μ	Nostril	Kikuyu	Kisumu	Nyanza
5	13	F	Nostril	Luo	Kisumu	Nyanza
6	13	Μ	Nostril	Luhya	Nakuru	Rift Valley
7	3	Μ	Bulbar Conjunctiva	Kikuyu	Nakuru	Rift Valley
8	10	М	Nostril	Luo	South Nyanza	Nyanza
<b>9</b> <sup>i</sup>	A	F	Nostril	Luo	Nakuru	Rift Valley
10	?	М	Nostril	Kalenjin	Trans Nzoia	Rift Valley

A: Adult

Table 2Age distribution

Age	No. exam.	Rhinosporidiosis	
0-9	3,880	3	
10-19	1,552	5	
20-39	1,242	0	
40-	8,461	0	
Unknown	3,834	2	

	Table 3	Sex	distribution
Sex	No. e	xam.	Rhinosporidiosis

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Male	10,962	8	
Female	7,192	2	

Table	4	Ethnic	distribution

Ethnic group	No. exam.	Rhinosporidiosis	
Luo	4,682	6	
Kikuyu	1,785 3,420	2 1	
Kalenjin			
Luhya	3,012	1	
Kisii	1,124	0	
Maasai	247	0	

are described. Out of 10 cases, nine showed the lesion at the nostril. In Tables 2, 3 and 4, the age, sex and ethnic distribution of the disease are summarized. It is likely that the disease already occured before patients had been younger than 20 years old (P<0.05, test of independence by  $\chi^2$  distribution). Sex and ethnic incidence showed no significant differences. In Figure 1, geographical distribution of the disease is summarized. All patients were from

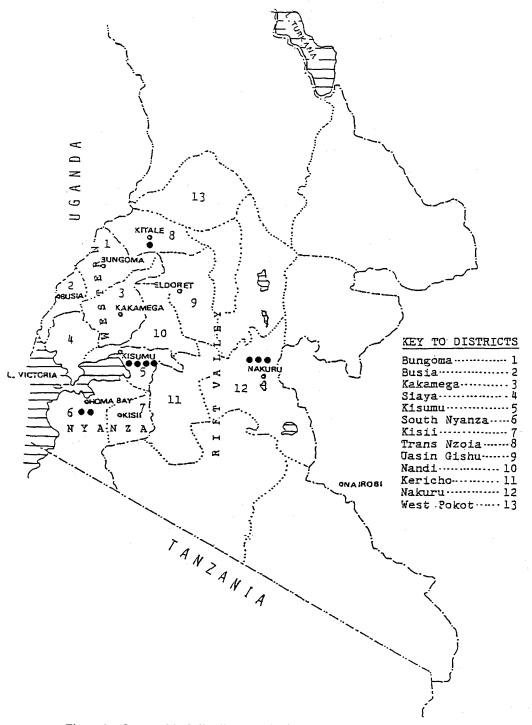


Figure 1 Geographical distribution of rhinosporidiosis in Western Kenya.

Nyanza Province and the central area of Rift Valley Province.

#### II. Histology

Histological examination revealed papillomatous hyperplasia of the mucosal epithelium. The mucosa was mostly covered by the stratified squamous epithelium and partially covered by the ciliated columnar epithelium, depending on the site of resection. There were various stages in the life cycle of fungal cells in the subepithelial connective tissue (Photo. 1). In some areas the stratified squamous epithelium was thickened with acanthosis and had a tendency to from down-growth and in other areas the epithelium was remakably thin, especially where there were projecting mature sporangia or ruptured sporangia, and destruction of the epithelium was observed where sporangia were bursting or discharging spores (Photo. 2). The subepithelial connective tissue was usually loose and edematous and there were many spores, trophocytes and sporangia of variable sizes in it. Mature sporangia showed up to  $300\,\mu$  in diameter (Photo. 3). In the connective tissue around fungal cells, there were slight inflammatory cell infiltration, including plasma cells and lymphocytes, and vascular proliferation and dilatation. Areas of small hemorrhage were common (Photo. 4). Some of mature sporangia showed rupture and were empty or collapsed after discharging spores. Giant cells of foreign-body type appeared occasionally in and around sporangia which had ruptured (Photo. 5). In some areas discharged spores were accompanied with small number of pus cells (Photo. 6). Except the presence of secondary infection, these cases of rhinosporidiosis did not show a tendency to be suppurative.



Photo. 1 Various stages in the life cycle of *Rhinosporidium seeberi* in the subepithelial connective tissue  $(H.E., \times 40)$ .

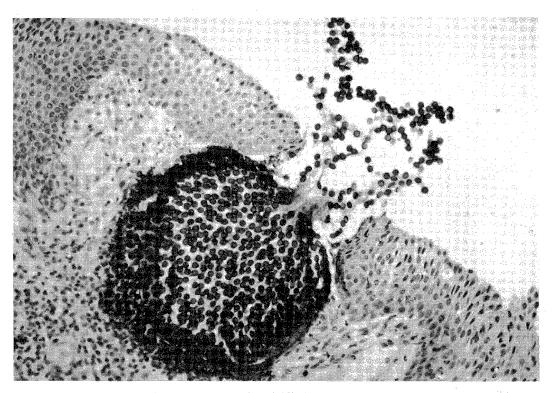


Photo. 2 A mature sporangium is discharging spores (H.E.,  $\times 100$ ).

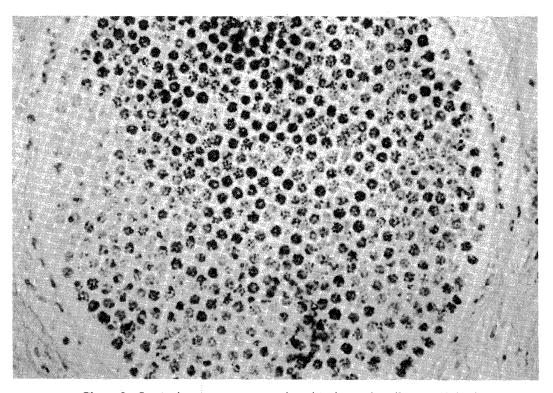


Photo. 3 Spores in a mature sporangium (methenamine silver,  $\times 400$ ).

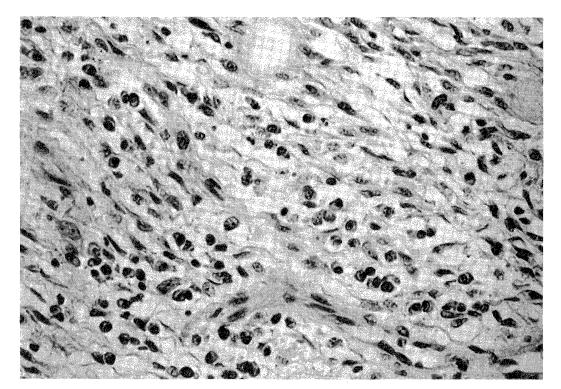


Photo. 4 Slight degree of inflammatory cell infiltration in the infected area (H.E., ×200).

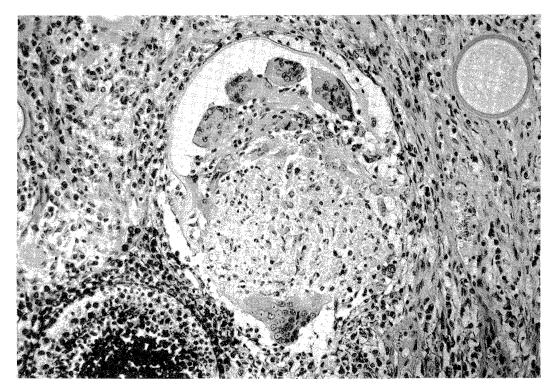


Photo. 5 Giant cell reaction in and around a ruptured sporangium (H.E.,  $\times 100).$ 

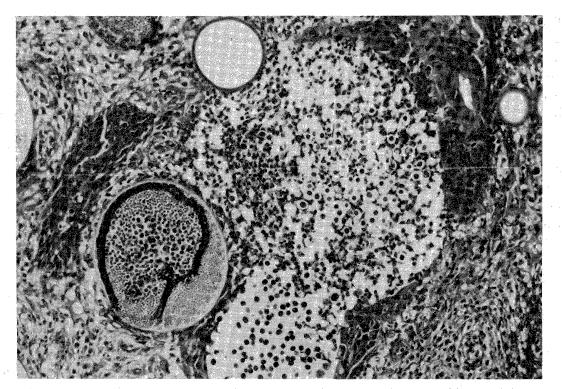


Photo. 6 Discharged spores and small amount of pus cells (H.E., ×100).

#### DISCUSSION

Rhinosporidiosis which is caused by *Rhinosporidium seeberi* is a chronic granulomatous disease and endemic in Sri Lanka and India. About 90 per cent of the reported cases in the world were from the both countries (Karunaratne, 1964). Although number of patients is few, the disease has been issued from many countries of the world and we found 10 cases of rhinosporidiosis in Western Kenya, during the period of six years, 1979 to 1984. Our results showed that the most common affected site of the infection is the nostril, followed by the bulbar conjunctiva. Karunaratne (1964) reported that the most common affected site is the mucous membrane of the nose and nasopharynx in Sri Lanka and India. However, it occasionally affects other sites; the trachea (Grewal and Rangam, 1959), larynx (Pillai, 1974), external ear, lips, conjunctiva, lacrymal sac, penis, vulva, vagina and urethra (Kutty and Unni, 1969).

We reported the relatively high incidence of the disease in young generations. The youngest is a three-year-old boy in Western Kenya. In Sri Lanka and India the disease is likely to come on in the age of 15-39 years old (Karunaratne, 1964). Reports from Sri Lanka and India show that males are more frequently infected than females. This is more evident in older age groups. Young females are affected as frequently as males (Karunaratne, 1964). Mohapatra (1971) reported that the high incidence in males is due to the possible increased exposure to the source of the infection. However, we could not find any significant differences on sex incidence. Cameron *et al.* (1973), also, could not find the difference of the disease incidence by sex in Kenya.

Present paper reported that in Nyanza Province and the central area of Rift Valley

Province the high incidence of the disease was observed. Nyanza Province is a tropical savannah. A mean annual rainfall in Nyanza Province is 750 to 1,250 mm. A mean annual temperature is 30 to 34°C. The central area of Rift Valley Province is a tropical highland and a mean annual rainfall is 1,000 to 1,500 mm and a mean annual temperature is 22 to 30°C (Vogel et al., 1974). We had examined over 1,000 surgical specimens obtained from the nomads who live in a desert or semi-desert area in Western Kenya but failed to find the disease. In Sri Lanka and India there is no significant difference of the incidence among the different races. However, the disease is likely to be found in the peasant or worker class who live in agricultural areas and drink water at water tanks, rivers and ponds (Karunaratne, 1964; Satyanarayana, 1960). In Western Kenya most of the patients were from agricultural areas and they used to take a bath and drink water on rivers, ponds and fresh-water lakes. These findings suggest that environmental factors play some important roles in the transmission of the disease and probably support the idea that water-borne transmission is one of the possible ways of infection (Cherian and Satyanarayana, 1949; Rajam et al., 1955). There is another possibility that the domestic animals such as cattles, mules and dogs are the possible sources of the infection (Myers et al., 1964; Stuart and O'Mally, 1975; Davidson and Nettles, 1977), but we could not find out any patient of the disease from the nomads in Western Kenya. Nyanza Province and the central area of Rift Valley Province are suitable investigation areas where research on mode of the transmission of the disease will be done.

Histologically rhinosporidiosis shows characteristic appearances. There are slight inflammatory cell infiltration around fungal cells and suppurative changes are rarely seen in spite of the presence of huge number of fungal cells. Such lesions are histologically very different from the changes of other fungal infections. These findings suggest that rhinosporidiosis is one of the unique fungal diseases showing characteristic histological features.

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ケニア西部におけるリノスポリディオシス

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リノスポリディオシスは Rhinosporidium seeberi (Seeber, 1900) によってひき起こされる慢性肉芽腫 性疾患である。本病原体はまだ分離培養されておらず、生活史は不明であるが、一部では藻菌類に属 すと言われている。易出血性、カリフラワー状の肉芽腫は主として鼻腔、咽頭に生じるほかに稀には 眼球結膜、陰茎、尿道などを侵し、臓器散布例の報告もある。スリランカやインドでは風土病的に比 較的頻繁に見られる疾患であり、他の熱帯、亜熱帯からも散発例が報告されているが、本邦からの報 告例はない。

我々は1979年から1984年に亙ってケニア西部,リフトバレー,ウェスタン,ニヤンザ州の外科生検 材料を病理組織学的に検索するとともに疫学的調査を行い,ケニア西部においてリノスポリディオシ スは高温で比較的湿潤なビクトリア湖沿岸,および湿潤な高原地帯であるリフトバレー州中央部に多 発し,その感染経路は水系であろうと言う結果を得た。

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