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Sporotrichosis

R. Morris-Jones

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University of Zurich

✉ R. Morris-Jones, St John's Institute of Dermatology, St Thomas' Hospital, London, SE1 7EH, UK. E-mail: themojos@cwcom.net

Abstract

Summary Sporotrichosis is a cutaneous fungal infection with a global distribution. The disease has several clinical forms, primarily cutaneous with associated lymphadenopathy. However, dissemination to osteoarticular structures and viscera may occur, both in healthy and immunosuppressed individuals; such disseminated forms usually follow spread after inhalation of fungal spores. Cutaneous infection is usually associated with trauma during the course of outdoor work, and treatment is required for the majority of patients. Potassium iodide (the treatment of choice in endemic areas) is an effective and inexpensive therapy; however, its adverse effects and complicated dosage regimen often weigh against its use in developed countries, where itraconazole is the antimycotic of choice.

Introduction

Benjamin Schenck reported the first case of sporotrichosis in 1896 when he was a medical student in the USA. He isolated the causative organism and sent the fungal culture to Erwin Smith a mycologist who concluded that it belonged to the genus *Sporotrichum*.¹ In 1900 Hekton and Perkins were the first to name this pathogenic fungus *Sporothrix schenckii*.² Subsequently *S. schenckii* has been identified worldwide and in recent years most reported cases have been from Central and South America (Mexico, Colombia, Brazil and Peru) where infection rates in rural areas may approach 1 case per 1000 of the population.³

Epidemiology

Ecological factors that promote the growth and viability of *S. schenckii* include the presence of

organic material such as sphagnum moss, decaying vegetation, soil, wood and hay.⁴⁻⁶ Farmers, gardeners and forestry workers are at increased risk of infection. Zoonotic transmission has been reported, including bites or scratches from rodents, parrots, cats, dogs, horses and armadillos.⁷ The disease occurs most commonly in a sporadic fashion but large outbreaks have been reported usually in association with exposure to wood or soil. A major outbreak of sporotrichosis occurred in the gold mines of Witwatersrand, South Africa in the early 1940s, when 3000 cases were identified. These infections occurred in miners who brushed against the timbers used as structural supports. The large number of patients in an isolated setting allowed for an extensive study of the ecology and distribution of the organism.⁸ *S. schenckii* most commonly enters the body through traumatic implantation, leading to cutaneous or subcutaneous infections. However only 10–62% of patients recall any history of trauma.⁹ Patients with sporotrichosis are usually healthy adults less than 30 years of age, but 15% of cases are children under the age of 10 years.¹⁰ Regional variation in the age and sex distribution of cases is usually attributable to different exposure conditions.

Clinical manifestations

Most patients with sporotrichosis have localized disease in the skin and subcutaneous tissues. It is believed that transmission occurs mainly through inoculation, but inhalation resulting in pulmonary or disseminated disease may also rarely develop.

Lymphocutaneous disease

Lymphocutaneous sporotrichosis is the most common form of the disease. Exposed body sites such as the face and limbs are most frequently affected.³ A study in Japan found that the face accounted for 92% of all lesions in children, with the upper extremity being the most common site in adolescent and adult patients.¹¹ The first symptoms appear approximately 3 weeks after the infection is acquired, but this may be delayed up to 6 months. Early lesions consist of small painless erythematous papules, which enlarge over days or weeks. Although the lesions may remain single the usual course is subsequent discrete nodular lesions spreading progressively to more proximal sites. This is the so-called 'sporotrichoid' pattern of spread ([Fig. 1](#)). Draining lymphadenitis and lymphadenopathy is characteristic.





Figure 1

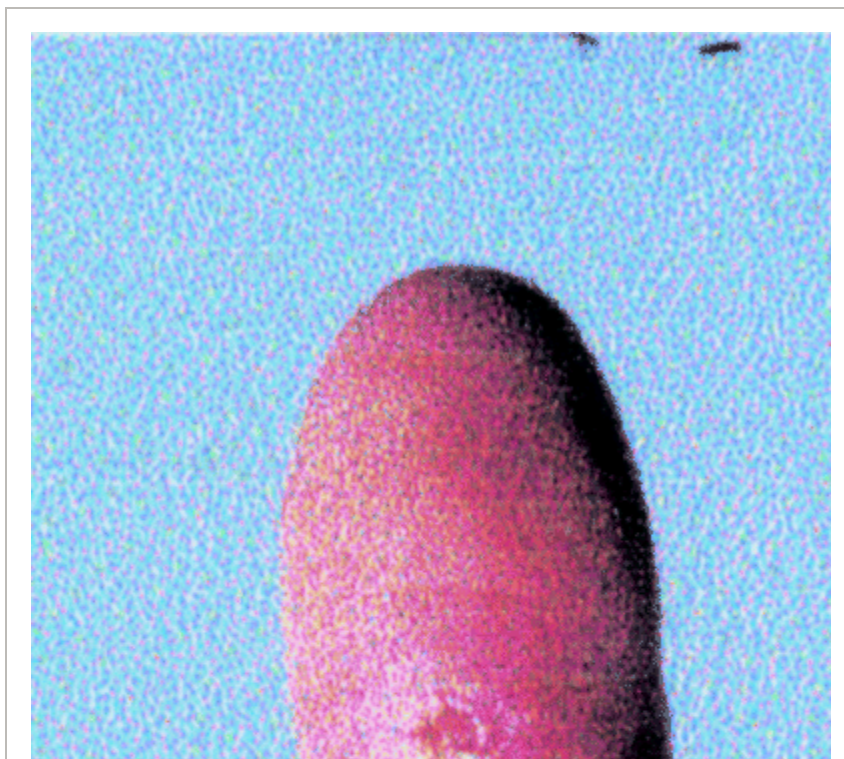
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Sporotrichoid spread of cutaneous sporotrichosis. (Courtesy of F. Montero-Gei, University of Costa Rica)

The secondary lesions produced along lymphatic channels follow the same indolent course as the primary lesion, and consist of papules, nodules or plaques that are erythematous with either a smooth or verrucous surface. Some lesions can become violaceous and then ulcerate in the centre. Small quantities of serous fluid or pus may be discharged from the lesions. Usually the nodules heal with scarring, which is rarely spontaneous, and the majority of patients require treatment.

Fixed cutaneous

Fixed cutaneous sporotrichosis occurs less commonly, and is characterized by localized lesions without lymphatic involvement ([Fig. 2](#)). Sites commonly affected are the face, neck, trunk or legs. These lesions are, once more, usually nodules, which can ulcerate, and occasionally small satellite lesions appear nearby.



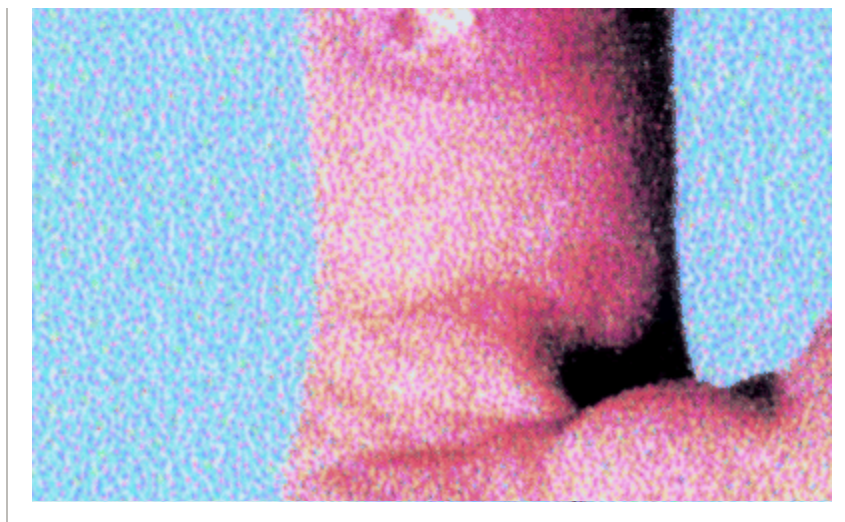


Figure 2

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Laboratory acquired cutaneous sporotrichosis. (Courtesy of R. J. May)

Disseminated cutaneous

Disseminated cutaneous sporotrichosis is rarely reported. The clinical picture is one of multiple lesions scattered over several body sites and may herald the onset of osteoarticular sporotrichosis. Haematogenous spread is thought to underlie cutaneous dissemination.

Extracuticular sporotrichosis

Extracuticular sporotrichosis occurs by haematogenous spread, occasionally directly from the primary inoculation site or regional lymph nodes¹² but more usually following spread from a pulmonary focus. Dissemination of the disease can affect large joints (knee, elbow, ankle or wrist), when patients complain of a gradual onset of stiffness and pain. Slow progression to osteomyelitis usually results.

Sporotrichosis can invade the central nervous system and the clinical picture is then one of multiple brain abscesses or chronic meningitis. Pulmonary disease is rare. Patients usually present with a cough, low-grade fever and weight loss, and massive haemoptysis, which can be fatal.¹³ Eighty-five per cent have a lesion in an upper lobe of the lung, and X-ray appearances may mimic tuberculosis. Systemic spread can be difficult to treat, and may be associated with substantial morbidity and mortality. Recently extra-cutaneous sporotrichosis has been reported as an emerging mycosis in HIV-seropositive individuals.^{14, 15}

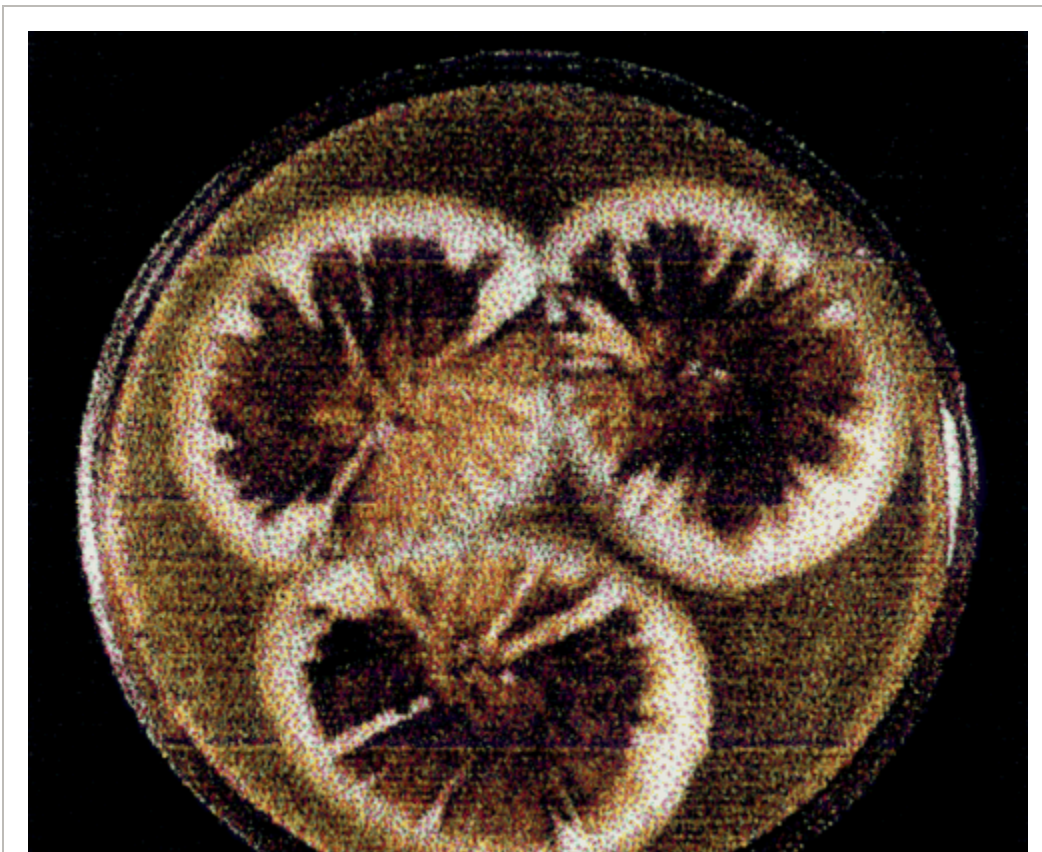
Differential diagnosis

The differential diagnosis of the cutaneous lesions includes *Nocardia* infections,

chromoblastomycosis, syphilis, *Mycobacterium marinum* or *M. kansasii*, *M. chelonae* or the 'tuberculosis verrucosa cutis' lesion of *M. tuberculosis*, granuloma annulare, foreign body granuloma, and chronic staphylococcal ecthyma. Lesions on the face can be mistaken for paracoccidioiodomycosis, blastomycosis, leishmaniasis, an insect or spider bite or even cat-scratch disease. Ulcerating necrotic lesions can mimic pyoderma gangrenosum.

Laboratory diagnosis

The gold standard for a definitive diagnosis is fungal culture. Specimens are usually obtained by skin biopsy from cutaneous lesions. *Sporothrix* can be grown on routine mycological plates (such as Sabouraud's dextrose agar, or Mycosel) at 25 °C. Growth is usually seen after 3–5 days, but cultures should be held for 4–5 weeks. Initially the colonies are cream-coloured, but after a few weeks they become brown or black (Fig. 3). Demonstration of dimorphism helps to confirm the identity of *S. schenckii*, and yeasts are produced by incubation of cultures at 37 °C in blood–glucose–cysteine agar, or brain–heart infusion broth. Direct examination of pus or biopsy material is usually unhelpful, because of the paucity of fungal cells; however, the yeast form can be demonstrated using specific fungal stains (i.e. periodic acid–Schiff and Gomori methenamine-silver). Histological features of sporotrichosis include: epidermal hyperplasia, hyperkeratosis, intraepidermal abscesses and mixed granulomas with asteroid bodies. The *sporothrix* asteroid body (Fig. 4) is found in 40% of cases where the organisms can be found; it is extracellular and consists of a central yeast with radiating eosinophilic spicules.¹⁶



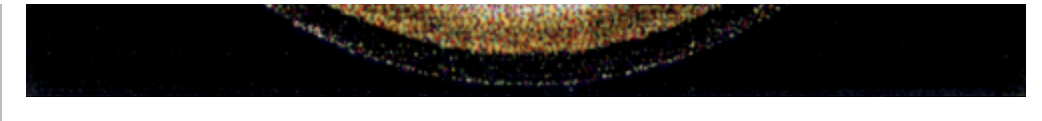


Figure 3

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Colonies of *Sporothrix schenckii* isolated at 26 °C. (Courtesy of R. J. Hay)

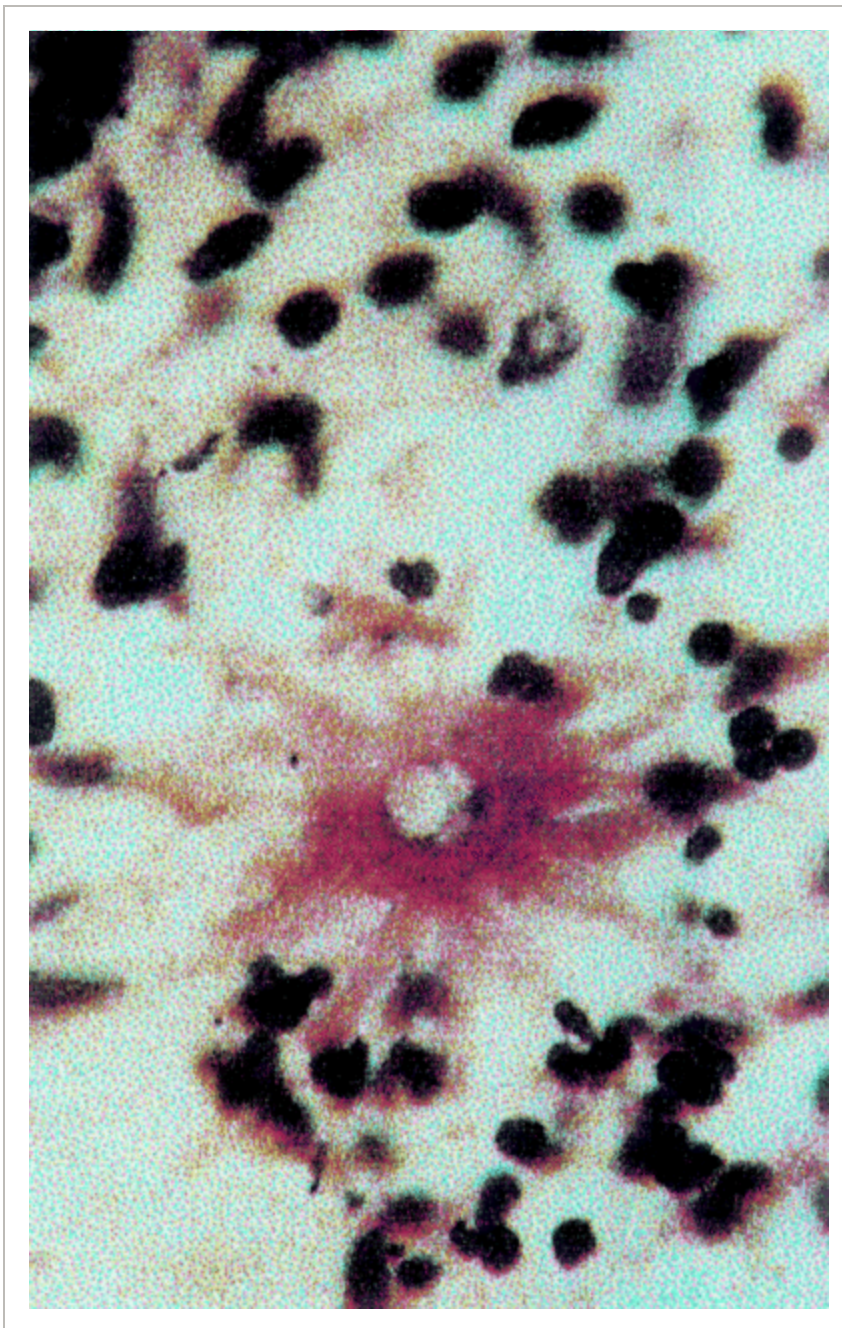


Figure 4

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Sporothrix asteroid body. (Courtesy of the Department of Medical Mycology, St John's)

Institute of Dermatology, St Thomas' Hospital. London UK)

The sporotrichin skin test detects a delayed-type hypersensitivity reaction, and can be a useful diagnostic tool. It is usually positive in >90% of proven cases of sporotrichosis, but it may indicate previous infection.¹⁷ A skin-test survey was conducted using sporotrichin and peptidoglycan antigens in northern India, where delayed-type hypersensitivity positivity rates were approximately 30% in villages where sporotrichosis had been reported, and 6% in 'control' areas with no clinical cases.¹⁸

Immunohistochemical and ELISA serodiagnostic techniques are not yet available in most clinical laboratories. Tube agglutination and latex particle agglutination tests can be used to detect antibodies. Serology is usually only helpful in diagnosing the rarer extracutaneous forms of sporotrichosis. However, Loureiro *et al.* have isolated an antigenic fraction of the cell wall of *S. schenckii* that binds concanavalin A. When this was tested on the sera of 35 patients with known cutaneous sporotrichosis there was 100% reactivity. This may represent a useful diagnostic tool for the future.¹⁹

Treatment

A saturated solution of potassium iodide has been used to treat cutaneous sporotrichosis since the early 1900s, and it is still the mainstay of treatment in most endemic areas. It is effective and inexpensive; however, the dosage regimen is complicated, and common side-effects include metallic taste, salivary gland enlargement and rash. Treatment is usually initiated with 5 drops three times daily and is increased as tolerated to 40–50 drops three times daily (equivalent to 250 mg to 1 g three times daily). Treatment should be continued for at least 4 weeks after apparent clinical 'cure'. Some patients are allergic to iodides, and some may respond only slowly, it is then that thermotherapy can be a useful alternative or adjunct therapy (this has sometimes been curative).²⁰ Itraconazole 100–200 mg daily is the treatment of choice for cutaneous sporotrichosis, in situations where cost does not preclude its use. For fixed cutaneous or lymphocutaneous disease treatment should continue for 3–6 months, and for osteoarticular disease at least 12 months. Meningeal and disseminated forms require amphotericin B, and in immunocompromised patients such as those with HIV infection, life-long suppression thereafter will then be required with itraconazole.

Terbinafine 250 mg twice daily is an effective treatment for cutaneous disease, with a rapid response and high cure rates being reported by a number of authors.^{21, 22} Extension of treatment beyond mycological cure may be unnecessary due to its fungicidal activity, but this has yet to be established.

Fluconazole 400 mg daily cured 71% of 14 patients with lymphocutaneous disease, but a

smaller percentage of extracutaneous cases, and is therefore considered only moderately effective in the treatment of sporotrichosis.²³

What's new?

Despite its global importance there is a paucity of information about the pathogenesis of *S. schenckii*, although a recent study in Mexico has demonstrated that *Sporothrix* produces melanin.²⁴ Melanization of human fungal pathogens is now seen as being of increasing importance in terms of virulence. Establishing the importance of melanization in *S. schenckii* will provide insight into some of its biological roles, and may provide a potential target for therapeutic intervention. Herbicides that target melanin biosynthesis are already commercially available for agricultural use, such as tricyclazole, which is used to control the 'rice-blast' fungus *Magnaporthe grisea*.²⁵ Therefore, by specifically targeting the biosynthetic pathway of fungal melanin we may produce a powerful new tool in the battle against human fungal pathogens.

Acknowledgements

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