STATE-OF-THE-ART CLINICAL ARTICLE

Sporotrichosis

Carol A. Kauffman

From the Division of Infectious Diseases, Ann Arbor Veterans Affairs Health System, University of Michigan Medical School, Ann Arbor, Michigan

Sporotrichosis is caused by *Sporothrix schenckii,* a dimorphic fungus that has worldwide distribution. The infection is usually localized to the skin and subcutaneous tissue. Osteoarticular and visceral involvement occurs mostly in patients who are immunocompromised and who have underlying alcoholism. Treatment for sporotrichosis is always required, even though the lesions are localized. Itraconazole has become the drug of choice for lymphocutaneous and osteoarticular forms of sporotrichosis. Pulmonary and disseminated sporotrichosis are more difficult to treat; depending on the host and the extent of disease, either amphotericin B or itraconazole should be used for these types of sporotrichosis.

Mycology

S. schenckii is one of several fungal pathogens that exhibit temperature dimorphism. In the environment or in the laboratory at temperatures ranging from 25°C to 30°C, *S. schenckii* grows as a mold. The organism grows readily on standard media, such as Sabouraud dextrose agar; within days to weeks, white to cream-colored colonies are seen, and these become brown to black and wrinkled over subsequent weeks. The conidia are either dark or hyaline and are arranged along the hyphae in a bouquet-like appearance.

In vivo at 37°C, *S. schenckii* exists as a yeast. In this form, the organism reproduces by budding and does not form conidia. Typically, the yeast forms of *S. schenckii* are 4–6 μm in diameter and are often cigar-shaped. In vitro at 37°C, *S. schenckii* assumes the yeast phase on supplemented media, such as brain-heart infusion or blood-cysteine-glucose agar. Some strains of *S. schenckii* grow best at temperatures no higher than 35°C. These thermointolerant strains are generally found only in fixed cutaneous lesions [1].

Publication of this State-of-the-Art Clinical Article has been made possible by an educational grant from Roche Laboratories.

Received 24 February 1999; revised 9 April 1999.

Financial support: National Institutes of Health (NIAID contract NO1-AI-65296).

Reprints or correspondence: Dr. Carol A. Kauffman, Department of Veterans Affairs Medical Center, 2215 Fuller Road, Ann Arbor, Michigan 48105 (ckauff@umich.edu).

Clinical Infectious Diseases 1999;29:231–7

1058–4838/99/2902–0001$03.00

S. schenckii var. *luriei* differs in both yeast and mold phases from typical *S. schenckii* and has been implicated in a few cases of typical sporotrichosis [2]. *Sporothrix cyanescens,* a saprophytic organism, has been documented as a cause of infection in at least one immunocompromised patient [3].

In addition to the usual fungal cell wall components, chitin, glucans, and mannans, *S. schenckii* contains a unique substance, L-rhamnose, that complexes with glycopeptides to form rhamnomannans that are not found in other fungal cell walls. Virulence factors of *S. schenckii* have not been entirely elucidated. The organism produces melanin, which has been found to be a virulence factor for other yeasts, and also extracellular proteinases, which may play a role in virulence [4, 5]. The ability to multiply at higher temperatures (37°C) is also probably a virulence factor for infection in many mammals. The thermointolerant strains that multiply poorly, if at all, above 35°C are unable to cause lymphatic or visceral involvement [1].

Immunology

The cellular response to *S. schenckii* infection is both neutrophilic and monocytic [6]. Antibody does not provide protection against infection. The fact that sporotrichosis is more severe and usually disseminated in nude mice and in patients with AIDS gives credence to the concept that T cell–mediated immunity is important in limiting the extent of infection with *S. schenckii* [7].

Epidemiology

S. schenckii is distributed throughout the world, but a preponderance of cases are reported from North and South America and Japan. The environmental niches in which the organism grows most luxuriantly include sphagnum moss, decaying vegetation, soil, and hay. Persons exposed to these environmental foci are at risk for developing infection. Thus, most infections occur in people with outdoor vocations or who participate in hobbies that take place out of doors. Specific activities that have been associated with sporotrichosis include rose gardening, topiary production, Christmas tree farming, hay baling, and masonry work [8–11]. Motor vehicle and other accidents that lead to inoculation of soil into skin and soft tissues also have been associated with sporotrichosis.

Several large outbreaks of sporotrichosis have been well-

described. One of the earliest and largest outbreaks occurred in Witwatersrand, South Africa, from 1941 to 1944 and involved almost 3,000 gold miners who developed sporotrichosis after the organism was inoculated by splinters of wood from contaminated timbers in the mines [12]. In the United States, the largest outbreak spread over 15 states and affected 84 patients, all of whom had received conifer seedlings that were packed in sphagnum moss that originated in Wisconsin and that contained *S. schenckii* [9].

For cases of pulmonary sporotrichosis, the mode of transmission is presumably inhalation of conidia aerosolized from soil or decaying vegetation. Discrete point sources have rarely been identified for cases of pulmonary sporotrichosis.

Another mode of transmission of *S. schenckii* is from bites or scratches of animals. The animals most commonly reported as causing zoonotic transmission of *S. schenckii* are armadillos and cats [13]. In the case of armadillos, the mammal is not infected but, while trying to escape capture, inflicts scratches and thereby inoculates *S. schenckii.* Cats develop serious and often fatal *S. schenckii* infection that is characterized by chronic ulcerative skin lesions (figure 1). Sporotrichosis is spread directly from the ulcers or from bites and scratches. In addition to armadillos and cats, insect stings, rodent and dog bites, and bird pecks have been associated with transmission of *S. schenckii* to humans.

Several cases of sporotrichosis have been described among laboratory workers who handled animals infected with or cultures of *S. schenckii* [14]. Generally, cutaneous inoculation has occurred, but ocular involvement subsequent to a splash of culture material into the eye has also been described.

Clinical Features

Sporotrichosis is primarily manifested as subacute to chronic cutaneous and subcutaneous infection. *S. schenckii* is a true pathogen in that normal healthy hosts are as much at risk for infection as are those who are immunosuppressed. However, the extent of disease varies with the state of immunity of the host. Disseminated visceral, osteoarticular, meningeal, and pulmonary sporotrichosis are most often seen in patients who have underlying risk factors. There is a strong association with alcoholism, but the reasons for this predisposition are not known. Other risk factors include diabetes mellitus, chronic obstructive pulmonary disease, and HIV infection.

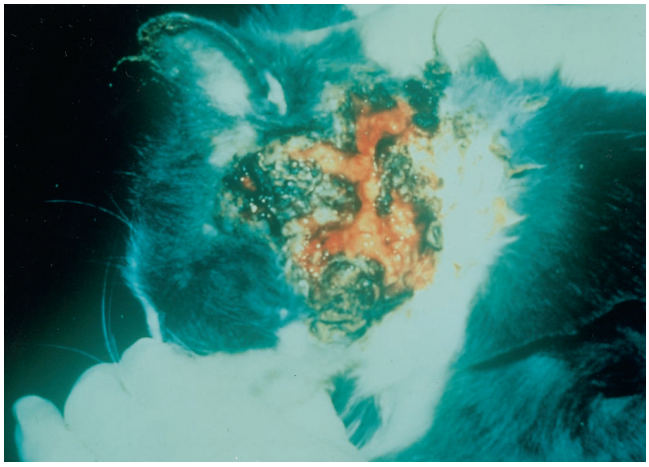

Figure 1. Ulcerated lesion on the side of the face of a cat with sporotrichosis (photo courtesy of Dr. Kurt Reed). From [13].

Cutaneous and lymphocutaneous sporotrichosis. This is the most common manifestation of infection with *S. schenckii.* Following inoculation of the conidia into the skin or subcutaneous tissue, a primary lesion develops within days to weeks at the site of inoculation. The lesion begins as a papule, slowly enlarges to become nodular, and often ulcerates. Pain is generally mild, the drainage is not grossly purulent, and systemic symptoms are not seen. Progression of the infection is characterized by nodular lesions that appear along the lymphatic distribution proximal to the initial lesion and lymphangitic streaking between the nodules [15] (figure 2). The characteristics of the nodules are similar to those of the primary lesion. The lymphocutaneous form of sporotrichosis must be differentiated from infections with nontuberculous mycobacteria, especially *Mycobacterium marinum;* nocardiosis, especially that due to *Nocardia brasiliensis;* tularemia; and leishmaniasis [16].

Some patients do not manifest lymphangitic spread but instead have fixed cutaneous lesions, which persist at the site of inoculation (figure 3). These lesions may be plaque-like or verrucous, and ulceration is uncommon. Although the lesions may remit transiently, they generally return and frequently persist for years until treated. Fixed cutaneous lesions are often caused by strains of *S. schenckii* that grow best at temperatures of $\leqslant$35°C. The differential diagnosis of this form of sporotrichosis includes mycobacterial infections, nocardiosis, chromoblastomycosis, blastomycosis, paracoccidioidomycosis, and leishmaniasis.

Pulmonary sporotrichosis. The manifestations of pulmonary infection with *S. schenckii* are similar to those of pulmonary tuberculosis. The typical patient is a middle-aged man with chronic obstructive pulmonary disease and alcoholism. Fever, night sweats, anorexia, weight loss, and fatigue are common. Cough productive of purulent sputum, hemoptysis, and dyspnea are the prominent chest complaints. The chest radiographic findings are similar to those of tuberculosis (figure 4). Cavities are frequently present, and they tend to occur in the upper lobes. The differential diagnosis includes other fungal infections (such as histoplasmosis, blastomycosis, and coccidioidomycosis), tuberculosis, and sarcoidosis. The course of untreated chronic cavitary pulmonary sporotrichosis is one of slow relentless progression.

Osteoarticular sporotrichosis. Osteoarticular infection may follow either cutaneous inoculation or hematogenous spread from the lungs. This form of sporotrichosis occurs at the

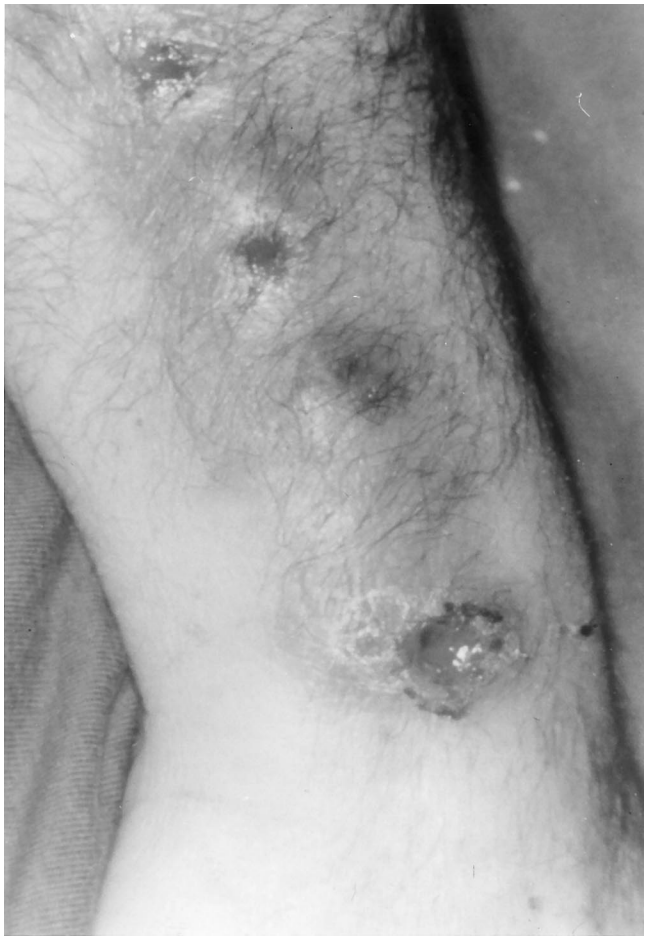

Figure 2. Lesions on the wrist of the 46-year-old owner of a nursery who, after working with sphagnum moss, developed nodular ulcerated lesions beginning on the right wrist and extending proximally. Culture of the drainage yielded *Sporothrix schenckii* (photo courtesy of Dr. Chatrchai Watanakunakorn). From [15].

site of an overlying cutaneous lesion, in bony structures distant from the initial cutaneous lesion, or as an isolated finding without obvious cutaneous or pulmonary involvement. Septic arthritis is more common than osteomyelitis. The knee is the joint most commonly infected, followed by the elbow, wrist, ankle, and joints of the hand. It is not unusual to have multiple joints involved. Bursitis and tenosynovitis, as well as nerve entrapment syndromes, also have been reported due to sporotrichosis.

In the absence of a cutaneous lesion, osteoarticular sporotrichosis is rarely considered as a diagnosis. Findings on examination of synovial fluid and histopathology are not specific, and the diagnosis is almost always made only when culture of synovial fluid or synovial tissue yields *S. schenckii.* Even with treatment, relapses are common, and the functional outcome for patients with osteoarticular sporotrichosis is often poor.

Disseminated and visceral sporotrichosis. Disseminated infection presents most often as disseminated lymphocutaneous lesions, but visceral dissemination has been reported in some instances. Patients with AIDS appear to be at risk for disseminated sporotrichosis, and in these patients, systemic symptoms are the rule, spread to the meninges commonly occurs, and the skin lesions are often atypical punched-out ulcerations with minimal inflammatory response (figure 5).

Sporotrichal meningitis may present as isolated chronic meningitis, similar to that seen in patients with histoplasmosis and coccidioidomycosis, or it may be one manifestation of disseminated sporotrichosis. Findings on examination of CSF are those of lymphocytic meningitis with an elevated protein level and a reduced glucose level. In addition to meningitis, discrete enhancing lesions on MRI and CT also have been reported for a few patients.

Diagnosis

The definitive diagnosis of sporotrichosis relies on isolating the organism from the site of infection. Material can be swabbed or aspirated from a cutaneous lesion, or the lesion can be biopsied. Sputum, synovial fluid, CSF, and, rarely, blood

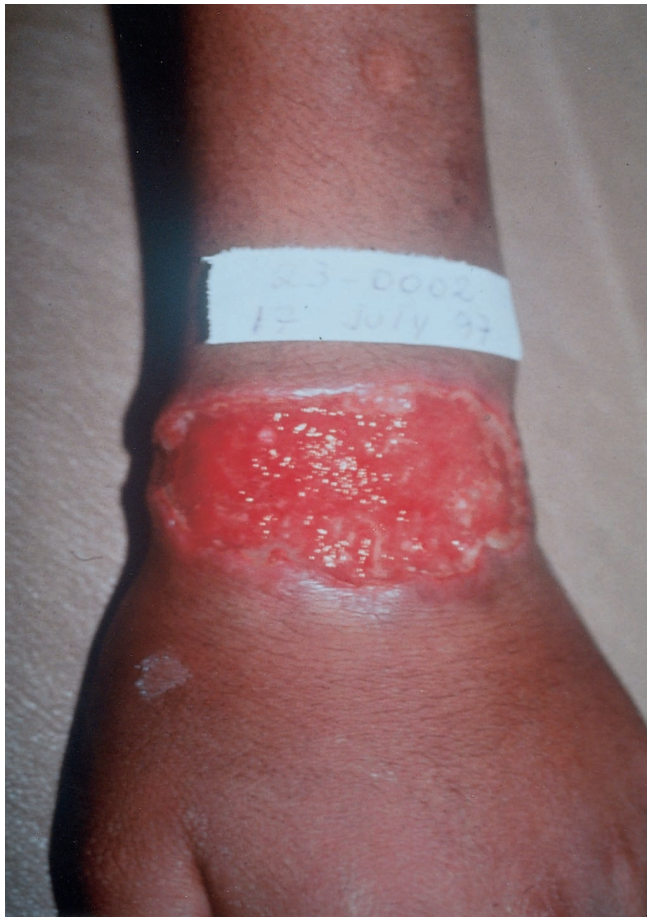

Figure 3. Fixed cutaneous lesion of sporotrichosis on the wrist of a patient from Peru (photo courtesy of Dr. Peter Pappas).

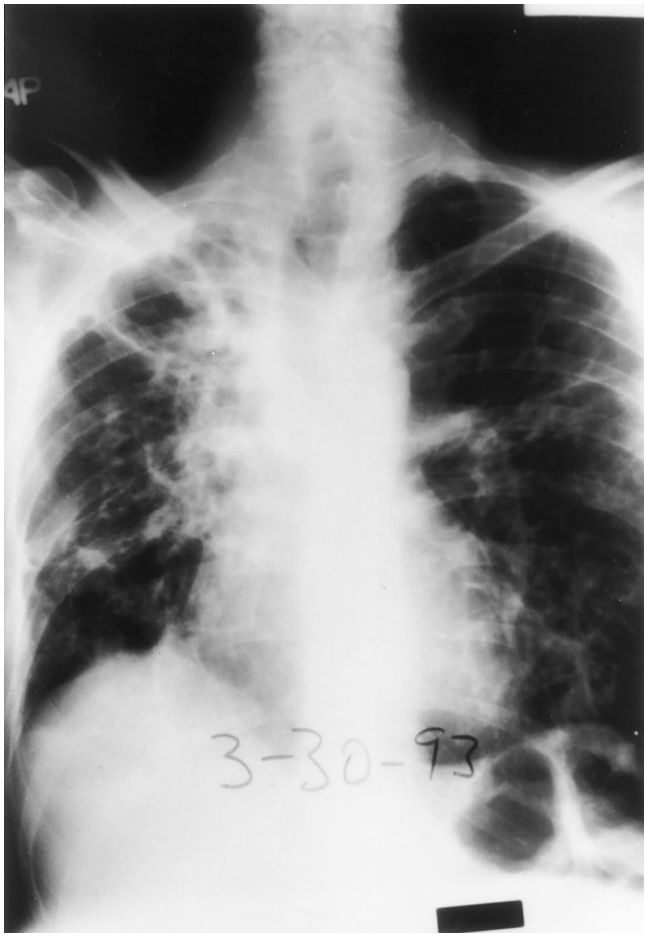

Figure 4. Chest radiograph of a 56-year-old heavy equipment operator who had diabetes mellitus and alcoholism and developed chronic cavity pulmonary sporotrichosis.

have been reported to yield *S. schenckii* when cultured. Generally, Sabouraud dextrose agar is used for primary isolation, and the culture is kept at room temperature. It is relatively easy to grow *S. schenckii* compared with some of the endemic fungi, such as *Histoplasma capsulatum.* Within days to weeks, hyphal growth occurs. The characteristic appearance of the conidia on the hyphae allows presumptive identification of the organism as *S. schenckii,* but conversion to the yeast form should be attempted to firmly establish the diagnosis of sporotrichosis.

Histopathologic examination of skin lesions usually shows mixed granulomatous and pyogenic inflammation. Multinucleated giant cells may be seen, and pseudoepitheliomatous hyperplasia may be present in skin lesions. Special stains, such as periodic acid–Schiff and methenamine silver, are required to see *S. schenckii* yeast forms in tissue. Classically, the yeasts are described as oval to cigar-shaped, and budding may be seen. Since the organisms are frequently present in small numbers, the biopsy often reveals no fungal elements.

The use of serology for the diagnosis of sporotrichosis has not progressed to the level noted with other fungal infections, such as histoplasmosis and coccidioidomycosis. Reference laboratories, such as those at the Centers for Disease Control and Prevention and various state public health laboratories, offer tube and latex agglutination assays. There have been no studies assessing the sensitivity and specificity of the available serological assays. Scott et al. [17] found that an EIA and a latex agglutination assay of CSF were useful for the diagnosis of sporotrichal meningitis. Although these assays were both sensitive and specific, they are not readily available.

Treatment

All forms of sporotrichosis require treatment with antifungal agents or other local measures. Other than rare cases of meningeal sporotrichosis and disseminated sporotrichosis in immunocompromised hosts, infection with *S. schenckii* is rarely life-threatening. Thus, urgent use of intravenous antifungal agents is rarely required. The preferred treatment, as well as the outcome, depends on the site of infection (table 1).

Lymphocutaneous and cutaneous sporotrichosis. The classic therapy for this form of sporotrichosis, which has been used since the beginning of the 20th century, is saturated solution of potassium iodide (SSKI). In spite of this extensive clinical experience, it still is not known why SSKI is effective for the treatment of sporotrichosis. The starting dosage is 5 drops in water or juice three times a day; this is increased by 5 drops per dose each week until the maximum dosage of 40–50 drops three times a day is reached. SSKI is generally given for 3–6 months. However, side effects (nausea, rash, fever, metallic taste, salivary gland swelling) are common, and it is more difficult to administer a solution than to administer capsules or tablets.

Itraconazole has become the drug of choice for lymphocutaneous and cutaneous sporotrichosis. The usual dosage is

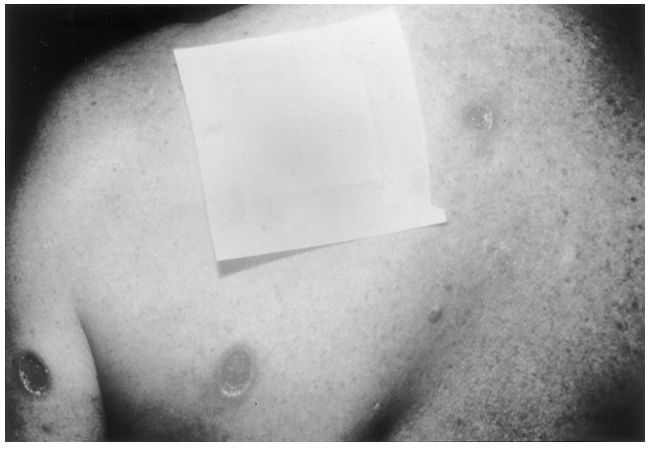

Figure 5. Punched-out ulcerated skin lesions due to *Sporothrix schenckii* in a patient with AIDS who had a CD4 cell count of 56 cells/μL. He also had sporotrichal meningitis (photo courtesy of Dr. Elliot Goldstein).

Table 1. Treatment modalities for various forms of sporotrichosis.

Treatment	Disease type	Advantages	Disadvantages
Local hyperthemia	Cutaneous, ? lymphocutaneous	Inexpensive, simple	Requires rigid compliance by patient; useful only for cutaneous, ? lymphocutaneous forms
Saturated solution of potassium iodide	Cutaneous, lymphocutaneous	Inexpensive	Frequent side effects; inconvenient liquid form
Itraconazole	Cutaneous, pulmonary, lymphocutaneous, osteoarticular	Few side effects; once or twice daily dosage	Absorption can be erratic; drug interactions common
Fluconazole	Cutaneous, lymphocutaneous, osteoarticular	Absorption good; few side effects; once daily dosage	Less efficacious than itraconazole (need large dosage)
Amphotericin B	Disseminated, pulmonary, meningeal	Required for serious visceral infection	Side effects common; iv formulation

100–200 mg daily. The drug should be given with food unless the oral suspension is used, in which case that formulation is given on an empty stomach. The success rate is close to 100% if treatment is continued for 3–6 months [18, 19].

Fluconazole is modestly effective for the treatment of sporotrichosis but has a lower success rate than that noted with itraconazole [20]. If a patient does not tolerate or has serious drug interactions with itraconazole, then fluconazole could be used. The fluconazole dosage should be at least 400 mg daily to ensure success.

Cutaneous sporotrichosis can sometimes be treated with local heat therapy. The basis for this form of treatment most likely relates to the observations that some strains of *S. schenckii,* often those isolated from fixed cutaneous lesions, exhibit growth inhibition at temperatures above 35°C, and isolates from cutaneous, but not visceral, lesions are killed at temperatures of 39°C [1]. Hot baths (~45°C), local applications of hot compresses, and hand-held heaters have been used [21]. In general, this form of treatment requires faithful application of heat for ~1 hour each day for several months. The best success rates, not surprisingly, are noted with cutaneous rather than lymphocutaneous lesions.

Pulmonary sporotrichosis. Pulmonary involvement with *S. schenckii* is chronic and progressive, responding poorly to antifungal therapy [22]. The greatest experience has been with amphotericin B; there is less experience with azoles. SSKI should not be used for pulmonary sporotrichosis. For amphotericin B, a total of 1–2 g should be given, and for itraconazole, 200 mg twice daily should be used. Response rates of ~30% have been noted with all antifungal regimens [19, 20, 22]. When feasible, surgical resection of involved lung tissue provides definitive treatment. Unfortunately, many of the patients who develop sporotrichosis have extensive chronic obstructive pulmonary disease and cannot tolerate a surgical procedure.

Osteoarticular sporotrichosis. This form of sporotrichosis tends to be indolent, and the response to treatment is similarly slow. Since patients rarely have systemic symptoms, amphotericin B is not used frequently. Itraconazole has become the drug of choice; the dosage is 200 mg twice daily. SSKI should not be used, and fluconazole (response rate of 38%) is not as effective as itraconazole (response rate of 86%) [19, 20, 23]. Although intraarticular injection of amphotericin B has been reported as beneficial [24], this use of amphotericin B is not recommended as routine therapy for sporotrichal arthritis.

Disseminated sporotrichosis. Disseminated disease, especially since it occurs mostly in immunocompromised patients, usually requires treatment with amphotericin B. However, after initial improvement, treatment usually can be changed to itraconazole, 200 mg twice daily with food. For patients who have both HIV infection and sporotrichosis, lifelong suppressive therapy with itraconazole is recommended [25, 26].

Patients with sporotrichal meningitis should be treated with amphotericin B. Perhaps itraconazole could play a role in long-term suppression after an initial response to amphotericin B. Even though its levels in CSF are very low or unmeasurable, itraconazole has more intrinsic activity against *S. schenckii* and probably is preferred to fluconazole, with its higher levels in CSF but lesser antifungal activity against *S. schenckii.* Too few patients have been described to establish the most effective treatment for this serious form of infection with *S. schenckii.*

References

1. Kwon-Chung KJ. Comparison of isolates of *Sporothrix schenckii* obtained from fixed cutaneous lesions with isolates from other types of lesions. J Infect Dis **1979**;139:424–31.
2. Padhye AA, Kaufman L, Durry E, et al. Fatal pulmonary sporotrichosis caused by *Sporothrix schenckii* var. *luriei* in India. J Clin Microbiol **1992**;30:2492–4.
3. Tambini R, Farina C, Fiocchi R, et al. Possible pathogenic role for *Sporothrix cyanescens* isolated from a lung lesion in a heart transplant patient. J Med Vet Mycol **1996**;34:195–8.
4. Dixon DM, Duncan RA, Hurd NJ. Use of a mouse model to evaluate clinical and environmental isolates of *Sporothrix* spp. from the largest U.S. epidemic of sporotrichosis. J Clin Microbiol **1992**;30:951–4.
5. Tsuboi R, Sanada T, Takamori K, Ogawa H. Isolation and characterization of *Sporothrix schenckii.* J Bacteriol **1987**;169:4104–9.
6. Cunningham KM, Bulmer GS, Rhoades ER. Phagocytosis and intracellular fate of *Sporothrix schenckii.* J Infect Dis **1979**;140:815–7.
7. Shiraishi A, Nakagagi K, Arai T. Role of cell-mediated immunity in the resistance to experimental sporotrichosis in mice. Mycopathologia **1992**;120:15–21.

8. Coles FB, Schuchat A, Hibbs JR, et al. A multistate outbreak of sporotrichosis associated with sphagnum moss. Am J Epidemiol **1992**;136: 475–87.
9. Dixon DM, Salkin IF, Duncan RA, et al. Isolation and characterization of *Sporothrix schenckii* from clinical and environmental sources associated with the largest U.S. epidemic of sporotrichosis. J Clin Microbiol **1991**;29:1106–13.
10. Hajjeh R, McDonnell S, Reef S, et al. Outbreak of sporotrichosis among tree nursery workers. J Infect Dis **1997**;176:499–504.
11. Dooley DP, Bostic PS, Beckius ML. Spook house sporotrichosis: a point source outbreak of sporotrichosis associated with hay bale props in a Halloween haunted house. Arch Intern Med **1997**;157:1885–7.
12. Lurie HI. Five unusual cases of sporotrichosis from South Africa showing lesions in muscles, bones, and viscera. Br J Surg **1963**;50:585–91.
13. Reed KD, Moore FM, Geiger GE, Stemper ME. Zoonotic transmission of sporotrichosis: case report and review. Clin Infect Dis **1993**;16:384–7.
14. Cooper CR, Dixon DM, Salkin IF. Laboratory-acquired sporotrichosis. J Med Vet Mycol **1992**;30:169–71.
15. Watanakunakorn C. Photo Quiz. Clin Infect Dis **1996**;22:765.
16. Kostman JR, DiNubile MJ. Nodular lymphangitis: a distinctive but often unrecognized syndrome. Ann Intern Med **1993**;118:883–8.
17. Scott EN, Kaufman L, Brown AC, Muchmore HG. Serologic studies in the diagnosis and management of meningitis due to *Sporothrix schenckii.* N Engl J Med **1987**;317:935–40.
18. Restrepo A, Robledo J, Gomez I, Tabares AM, Gutierrez R. Itraconazole therapy in lymphangitic and cutaneous sporotrichosis. Arch Dermatol **1986**;122:413–7.
19. Sharkey-Mathis PK, Kauffman CA, Graybill JR, et al. Treatment of sporotrichosis with itraconazole. Am J Med **1993**;95:279–85.
20. Kauffman CA, Pappas PG, McKinsey DS, et al. Treatment of lymphocutaneous and visceral sporotrichosis with fluconazole. Clin Infect Dis **1996**;22:46–50.
21. Hiruma M, Kawada A, Yoshida M, Kouya M. Hyperthermic treatment of sporotrichosis: experimental use of infrared and far infrared rays. Mycoses **1992**;35:293–9.
22. Pluss JL, Opal SM. Pulmonary sporotrichosis: review of treatment and outcome. Medicine (Baltimore) **1986**;65:143–53.
23. Winn RE, Anderson J, Piper J, Aronson NE, Pluss J. Systemic sporotrichosis treated with itraconazole. Clin Infect Dis **1993**;17:210–7.
24. Downs NH, Hinthorn DR, Mhatre VR, Liu C. Intra-articular amphotericin B treatment of *Sporothrix schenckii* arthritis. Arch Intern Med **1989**; 149:954–5.
25. Bolao F, Podzamczer D, Ventin M, Gudiol F. Efficacy of acute phase and maintenance therapy with itraconazole in an AIDS patient with sporotrichosis. Eur J Clin Microbiol Infect Dis **1994**;13:609–12.
26. Al-Tawfiq JA, Wools KK. Disseminated sporotrichosis and *Sporothrix schenckii* fungemia as the initial presentation of human immunodeficiency virus infection. Clin Infect Dis **1998**;26:1403–6.

Suggested Additional Reading

Kauffman CA. Old and new therapies for sporotrichosis. Clin Infect Dis **1995**;21:981–5.

Kwon-Chung KJ, Bennett JE. Sporotrichosis. In: Medical mycology. Philadelphia: Lea & Febiger, **1992**:707–29.

The "Conflict-of-Interest Policy" of the Office of Continuing Medical Education, UCLA School of Medicine, requires that faculty participating in a CME activity disclose to the audience any relationship with a pharmaceutical or equipment company which might pose a potential, apparent, or real conflict of interest with regard to their contribution to the program. The author reports no conflict of interest.

OFFICE OF CONTINUING MEDICAL EDUCATION
UCLA SCHOOL OF MEDICINE

This test affords you the opportunity to assess your knowledge and understanding of the material presented in the preceding clinical article, ″Sporotrichosis″ by Carol A. Kauffman, and to earn continuing medical education (CME) credit.

CME Sponsor

The Office of Continuing Medical Education, UCLA School of Medicine, is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Accreditation

The Office of Continuing Medical Education, UCLA School of Medicine, designates this educational activity for up to 1 hour in category I credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

To earn credit, read the State-of-the-Art Clinical Article carefully and answer the following questions. Mark your answers, only one answer per question, by circling the correct responses on the answer card (usually found toward the front of the issue), and mail this card after affixing first-class postage. To earn credit, a minimum score of 80% must be obtained.

Certificates of CME credit will be awarded on a per-volume (biannual) basis. Each answer card must be submitted within 3 months of the date of the issue.

---

1. Which activity has been associated with outbreaks of sporotrichosis?
   A. Baling hay
   B. Gold mining
   C. Topiary production
   D. Christmas tree farming
   E. All of the above
2. Which animal is most likely to be associated with transmission of sporotrichosis?
   A. Dogs
   B. Ferrets
   C. Cats
   D. Chickens
   E. Box turtles
3. The manifestations of pulmonary sporotrichosis are similar to those of:
   A. tuberculosis.
   B. lymphangitic spread of carcinoma.
   C. pneumococcal pneumonia.
   D. *Pneumocystis carinii* pneumonia.
   E. aspergillosis.
4. Patients with AIDS are most likely to have which type of sporotrichosis?
   A. Fixed cutaneous
   B. Disseminated
   C. Osteoarticular
   D. Lymphocutaneous
   E. All of the above
5. A patient consults you because of a nodular rash that began on the hand and has spread proximally along the forearm. Some of the nodules have ulcerated. In addition to sporotrichosis, which other infections can lead to this clinical picture?
   A. *Nocardia brasiliensis* infection
   B. *Mycobacterium marinum* infection
   C. Tularemia
   D. Leishmaniasis
   E. All of the above
6. The following is a characteristic of strains of *Sporothrix schenckii* isolated from fixed cutaneous lesions:
   A. They do not grow in vitro
   B. Temperatures of >37°C are essential for their growth
   C. Special media other than Sabouraud agar are required
   D. They grow best at temperatures of <35°C
   E. All have been identified as *S. schenckii* var. *luriei*
7. The diagnosis of sporotrichosis is made most often by which of the following?
   A. Serology
   B. Skin testing
   C. Culture
   D. Histopathology
   E. PCR of involved tissues
8. Local hyperthermia has proved to be useful for which type of sporotrichosis?
   A. Osteoarticular
   B. Pulmonary
   C. Meningeal
   D. Disseminated
   E. Fixed cutaneous
9. Which of the following is the usual therapy for disseminated visceral sporotrichosis?
   A. Itraconazole
   B. Ketoconazole
   C. Fluconazole
   D. Amphotericin B
   E. Flucytosine
10. Which of the following is the treatment of choice for lymphocutaneous sporotrichosis?
    A. Fluconazole
    B. Ketoconazole
    C. Amphotericin B
    D. Itraconazole
    E. Griseofulvin