# *Sporothrix schenckii* Fungemia without Disseminated Sporotrichosis

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**Fungemia is a rare complication of *Sporothrix schenckii* infection and has always been associated with disseminated sporotrichosis. We describe an immunocompetent patient with localized lymphocutaneous sporotrichosis from whose blood the fungus was isolated. A lysis-centrifugation blood culture system may have improved our ability to detect low-level *S. schenckii* fungemia.**

Infection with the dimorphic fungus *Sporothrix schenckii* usually presents as a chronic cutaneous-lymphatic infection. Although the organism has also been recovered from a variety of other sites, there have been only five previously reported cases of *S. schenckii* fungemia (2, 11, 17, 20, 34). In those patients, fungemia was associated with multifocal cutaneous infection and with osteoarticular and other visceral involvement. We report a case of *S. schenckii* fungemia in an immunocompetent man with only lymphocutaneous disease.

In April 1988, a healthy 27-year-old male tree nursery worker noticed a 0.5-cm papule on the lateral aspect of the upper right arm. The lesion progressively enlarged, ulcerated, and drained thick yellow fluid. In May 1988, an excisional skin biopsy showed well-developed tuberculoid granulomas. Special stains for acid-fast organisms, bacteria, and fungi were negative, but tissue was not cultured. Over the next month, the ulcer continued to drain, and satellite skin lesions developed. The patient failed to improve on oral tetracycline or ciprofloxacin. In June 1988, the wound was surgically debrided, but not cultured. The arm became more erythematous, swollen, and fluctuant, and the patient was admitted to the hospital 1 week after the debridement.

On admission the patient was afebrile and looked healthy. A 2- by 4-cm ulcerated lesion with exudation was present on the lateral aspect of the mid-right arm. Multiple, slightly smaller, indurated erythematous satellite lesions were also noted inferior and medial, and superior and lateral to the primary lesion. There was no lymphangitic streaking, adenopathy, or joint inflammation. The remainder of the physical examination was normal. The leukocyte count was 9,600/mm$^3$ (67% segmented neutrophils, 23% lymphocytes, 6% monocytes, 4% eosinophils); the erythrocyte sedimentation rate was 5 mm/h; and the electrolytes, blood urea nitrogen, creatinine, liver enzymes, and urinalysis were normal. The patient was not tested for human immunodeficiency virus antibody, but he was not a member of a group at high risk for human immunodeficiency virus infection. A roentgenogram of the right arm showed soft-tissue swelling but no subcutaneous gas or bony abnormalities. Computerized axial tomography of the right arm showed attenuation of the subcutaneous tissue but no abnormalities of the bones or joints. No bacteria were cultured from a needle aspirate of the lesions.

During the hospitalization, bacterial skin abscess and cellulitis were suspected, and a blood culture was obtained 5 days after the needle aspirate of the wound. After a povidone iodine skin scrub, 2.5 ml of blood from the left (uninfected) arm was collected into an Isolator 10 tube (E. I. Dupont and Co., Wilmington, Del.). After centrifugation, concentrated sediment from the Isolator tube was inoculated in equal portions onto two chocolate agar plates, one sheep blood agar plate, and one Sabouraud agar plate. After 4 days of incubation, more than 10 colonies of a white mold were observed growing on the Sabouraud agar plate. The white mold was identified as *S. schenckii* by standard methods (19).

Initially, intravenous amphotericin B was recommended, but the patient declined treatment. Three weeks after discharge, the right arm lesions had not improved, and right axillary lymphadenopathy developed. A two-phase Tc-99 bone scan was performed. It showed local soft-tissue radiotracer uptake with drainage to the axillary lymphatics, but there was no evidence of osteoarticular infection. A chest roentgenogram showed no abnormalities. Cultures of lesion drainage did not grow fungi or mycobacteria, and repeat blood cultures, which were collected in Bactec 6A and 7A bottles (Becton-Dickinson, Towson, Md.) and subcultured onto Sabouraud agar, were sterile.

One month after the patient was discharged from the hospital, treatment with oral saturated solution of potassium iodide (SSKI), at 120 drops per day in three divided doses, was initiated. After 1 month of treatment, there was a 50% reduction in the size of the skin lesions and a 60% reduction in axillary adenopathy. The patient was treated with SSKI for a total duration of 8 months, with complete resolution of the lymphocutaneous disease. There has been no evidence of cutaneous or systemic fungal infection 28 months after the cessation of treatment.

Extracutaneous sporotrichosis is rare and occurs mainly in immunocompromised patients. Cancer, chronic corticosteroid use, diabetes, sarcoidosis, and alcoholism are the most common conditions associated with infection (3, 18). Extracutaneous infection may be unifocal or multifocal. Unifocal disease may be osteoarticular, genitourinary, pulmonary, or ocular or it may affect the central nervous system

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TABLE 1. Clinical features in patients with *S. schenckii* fungemia

| Reference | Year | Age, (yr), gender[a] | Underlying illnesses | Sites of infection | No. of positive blood cultures | Time (days) to growth | Treatment | Outcome |
|---|---|---|---|---|---|---|---|---|
| 34 | 1908 | 50, M | Pulmonary congestion | Multiple skin nodules, muscle, periosteum | 1 | 6 | Unknown | Unknown |
| 11 | 1908 | 42, M | None | Multiple skin nodules, periosteum | 1 | 15 | Iodine preparation, >1 mo | Cure |
| 17 | 1909 | ">66," F | ?, active pulmonary tuberculosis | Multiple skin nodules | 1 | 6 | Iodine preparation, 8 wk | Cure |
| 20 | 1984 | 63, 63, F | Received systemic steroids after onset of sporotrichosis because of mistaken diagnosis of sarcoidosis | Multiple skin nodules; nasal septum; subglottic region; elbows, knee, vertebral body; lung | 1 | Not known | Amphotericin B, 2.9 g; ketoconazole, 19 mo | Cure |
| 2 | 1989 | 58, 58, M | Alcoholism, diabetes | Multiple skin nodules[b] | 3[c] | ≤3 | Amphotericin B, 2 g[d]; ketaconazole, 2 wk; itraconazole, >18 mo | Complete suppression on therapy |

[a] M, male; F, female.
[b] The patient was obtunded and hypotensive on presentation.
[c] Each culture also grew *Clostridium tertium*.
[d] Organism was resistant to amphotericin B in vitro.

(14, 18, 31, 35). In patients with disseminated disease, multiple sites are culture positive, including, in one report (18), the bone marrow, and there are often multiple infected skin nodules. Progression of disseminated infection is common, and infection may be fatal if it is left untreated. Although a case of pulmonary sporotrichosis successfully treated with oral SSKI has been reported (27) and several reports from the early French literature described apparent "cures" of disseminated disease with oral SSKI (35), iodides, generally, are not effective in the treatment of extracutaneous sporotrichosis. Intravenous amphotericin B has been recommended (3, 29).

Excluding the pulmonary form, which may occur secondary to inhalation of the organism, hematogenous spread is thought to be responsible for extracutaneous sporotrichosis. However, recovery of *S. schenckii* from the blood has been a rare occurrence. In our review of the literature, we found only five previously published cases of *S. schenckii* fungemia (2, 11, 17, 20, 34). All five patients had disseminated infection (Table 1). Heart blood from a sixth patient was positive on culture at the time of autopsy (18). Finally, a possible seventh case of fungemia was cited by Wilson and colleagues (35), but a review of the case report (33) did not find mention of fungemia. Culture of *S. schenckii* from the blood of patients with lymphocutaneous infections has not been reported previously.

Dimorphic fungi like *S. schenckii* are isolated from blood less frequently than yeasts are, but they are isolated more frequently than molds are (4, 5, 10, 13, 15, 21, 30). In patients with disseminated or endovascular infections caused by yeasts such as those of the genera *Cryptococcus* (6, 26), *Saccharomyces* (23), *Hansenula* (16), and *Malassezia* (7), fungemia is commonly documented. *Candida* fungemia is common in patients with endovascular infections and in disseminated infections in nonneutropenic patients, but it is unusual in disseminated infections in neutropenic hosts (9). In contrast, fungemia is rare in patients with disseminated or endovascular mold infections secondary to *Aspergillus* (8,

36) and *Mucorales* (32) spp. *Fusarium* species is the only mold for which fungemia commonly accompanies disseminated disease (28).

The dimorphic fungi of the genera *Histoplasma* and *Coccidiodes* have been isolated from blood in 50 to 71% of patients with acute disseminated infections (1, 12, 24). However, unlike patients with disseminated yeast infections, patients with acute disseminated *Histoplasma* or *Coccidiodes* infections are almost all severely immunocompromised. In contrast, some dimorphic fungi, such as *Blastomyces* species, very rarely cause fungemia (22). The ability to recover dimorphic fungi from blood has clearly been improved with the advent of lysis-centrifugation blood culture techniques (5, 13, 21, 22, 24). In one recent study, two patients with localized pulmonary histoplasmosis were found to have positive blood cultures by this methodology (25).

We believe that the possibility of disseminated disease was excluded in our patient by clinical and radiologic evaluation and by his complete response to oral potassium iodide, without relapse. Our patient probably had a transient fungemia associated with local disease. Our ability to detect fungemia was likely related to the sensitive blood culture method used.

We conclude that culture of *S. schenckii* from the blood is not necessarily indicative of systemic disease. We suggest that patients with *S. schenckii* fungemia be evaluated for the extent of their infection and that potassium iodide be considered as primary therapy if only lymphocutaneous disease is found and no immunodeficiency is apparent. Conversely, when *S. schenckii* infection appears to be confined to the skin and regional lymphatics, blood cultures are usually unnecessary.

## REFERENCES

1. **Ampel, N. M., K. J. Ryan, P. J. Carry, M. A. Wieden, and R. B. Schifman.** 1986. Fungemia due to *Coccidiodes immitis*. Medicine **65:**312–321.
2. **Baker, J. H., H. C. Goodpasture, H. R. Kuhns, and M. G. Rinaldi.** 1989. Fungemia caused by an amphotericin B-resistant isolate of *Sporothrix schenckii*. Arch. Pathol. Lab. Med. **113:**1279–1281.
3. **Bennett, J. E.** 1990. *Sporothrix schenckii*, p. 1972–1975. *In* G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett (ed.), Principles and practice of infectious diseases, 3rd ed. Churchill Livingstone Co., New York.
4. **Bille, J., L. Stockman, and G. D. Roberts.** 1982. Detection of yeasts and filamentous fungi in blood cultures during a 10-year period (1972 to 1981). J. Clin. Microbiol. **16:**968–970.
5. **Bille, J., L. Stockman, G. D. Roberts, C. D. Horstmeier, and D. M. Ilstrup.** 1983. Evaluation of a lysis-centrifugation system for recovery of yeasts and filamentous fungi from blood. J. Clin. Microbiol. **18:**469–471.
6. **Clark, R. A., D. Greer, W. Atkinson, G. T. Valainis, and N. Hyslop.** 1990. Spectrum of *Cryptococcus neoformans* infection in 68 patients infected with human immunodeficiency virus. Rev. Infect. Dis. **12:**768–777.
7. **Dankner, W. M., S. A. Spector, J. Fierer, and C. E. Davis.** 1987. Malassezia fungemia in neonates and adults: complication of hyperalimentation. Rev. Infect. Dis. **9:**743–753.
8. **Denning, D. W., and A. H. Williams.** 1987. Invasive pulmonary aspergillosis diagnosed by blood culture and successfully treated. Br. J. Dis. Chest **81:**300–304.
9. **Edwards, J. E., Jr.** 1990. Candida species, p. 1943–1958. *In* G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett (ed.), Principles and practice of infectious diseases, 3rd ed. Churchill Livingstone Co., New York.
10. **Eilard, T.** 1987. Isolation of fungi in blood cultures. Scand. J. Infect. Dis. **19:**145–156.
11. **Gaucher, Louste, Abrami, and Giroux.** 1908. Sporotrichose cutanee. Bull. Soc. Fr. Dermatol. Syphiligr. **19:**283–284.
12. **Goodwin, R. A., Jr., J. L. Shapiro, G. H. Thurman, S. S. Thurman, and R. M. Des Prez.** 1980. Disseminated histoplasmosis: clinical and pathological correlations. Medicine **59:**1–33.
13. **Guerra-Romero, L., R. S. Edson, F. R. Cockerill III, C. D. Horstmeier, and G. D. Roberts.** 1987. Comparison of DuPont Isolator and Roche Septi-Chek for detection of fungemia. J. Clin. Microbiol. **25:**1623–1625.
14. **Gullberg, R. M., A. Quintanilla, M. L. Levin, J. Williams, and J. P. Phair.** 1987. Sporotrichosis: recurrent cutaneous, articular, and central nervous system infection in a renal patient. Rev. Infect. Dis. **9:**369.
15. **Henry, N. K., C. A. McLimans, A. J. Wright, R. L. Thompson, W. R. Wilson, and A. Washington II.** 1983. Microbiological and clinical evaluation of the isolator lysis-centrifugation blood culture tube. J. Clin. Microbiol. **17:**864–869.
16. **Klein, A. S., G. T. Tortora, R. Malowitz, and W. H. Greene.** 1988. *Hansenula anomala*: a new fungal pathogen. Two case reports and a review of the literature. Arch. Intern. Med. **148:**1210–1213.
17. **Landouzy, L.** 1909. Sporotrichose hypodermique: gommeuse, ulcereuse, disseminee (sporotrichose de de Beurmann). Presse Med. **17:**785–789.
18. **Lynch, P. J., J. J. Vorhees, and E. R. Harrell.** 1970. Systemic sporotrichosis. Ann. Intern. Med. **73:**23–30.
19. **McGinnis, M. R.** 1985. Dermatiaceous fungi, p. 561–574. *In* E. H. Lennett, A. Balows, W. J. Hausler, Jr., and H. J. Shadomy (ed.), Manual of clinical microbiology, 4th ed. American Society for Microbiology, Washington, D.C.
20. **Morgan, M. A., F. R. Cockerill, D. A. Cortese, and G. D. Roberts.** 1984. Disseminated sporotrichosis with *Sporothrix schenckii* fungemia. Diagn. Microbiol. Infect. Dis. **2:**151–155.
21. **Murray, P. A.** 1991. Comparison of the lysis-centrifugation and agitated biphasic blood culture systems for detection of fungemia. J. Clin. Microbiol. **29:**96–98.
22. **Musial, C. E., W. R. Wilson, I. R. Sinkeldam, and G. D. Roberts.** 1987. Recovery of *Blastomyces dermatitidis* from blood of a patient with disseminated blastomycosis. J. Clin. Microbiol. **25:**1421–1423.
23. **Nielsen, H., J. Stenderup, and B. Bruun.** 1990. Fungemia with Saccharomycetaceae: report of four cases and review of the literature. Scand. J. Infect. Dis. **22:**581–584.
24. **Paya, C. V., G. D. Roberts, and F. R. Cockerill.** 1987. Laboratory methods for the diagnosis of disseminated histoplasmosis: clinical importance of the lysis-centrifugation blood culture technique. Mayo Clin. Proc. **62:**480–485.
25. **Paya, C. V., G. D. Roberts, and F. R. Cockerill III.** 1987. Transient fungemia in acute pulmonary histoplasmosis: detection by new blood-culturing techniques. J. Infect. Dis. **156:**313–315.
26. **Perfect, J. R., D. T. Durack, and H. A. Gallis.** 1983. Cryptococcemia. Medicine **62:**98–108.
27. **Pluss, J. L., and S. M. Opal.** 1986. Pulmonary sporotrichosis: review of treatment and outcome. Medicine **65:**143–153.
28. **Richardson, S. E., R. M. Bannatyne, R. C. Summerbell, J. Milliken, R. Gold, and S. S. Weitzman.** 1988. Disseminated fusarial infection in the immunocompromised host. Rev. Infect. Dis. **10:**1171–1181.
29. **Rippon, J. W.** 1988. Medical mycology: the pathogenic fungi and the pathogenic actinomycetes, p. 325–352. The W. B. Saunders Co., Philadelphia.
30. **Roberts, G. D., and J. A. Washington II.** 1975. Detection of fungi in blood cultures. J. Clin. Microbiol. **1:**309–310.
31. **Satterwhite, T. K., M. V. Kageler, R. H. Conklin, B. L. Portney, and H. L. DuPont.** 1978. Disseminated sporotrichosis. JAMA **240:**771–773.
32. **Sugar, A. M.** 1990. Agents of mucormycosis and related species, p. 1962–1972. *In* G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett (ed.), Principles and practice of infectious diseases, 3rd ed. Churchill Livingstone Co., New York.
33. **Von Patschkowski.** 1931. Beitrag zur Klinik der Sporotrichose. Muench. Med. Wochenschr. **81:**938–940.
34. **Widal, F., and A. Weill.** 1908. Sporotrichose gommeuse disseminees a noyaux tres confluents. Gommes dermiques pour la plupart; gommes hypodermiques et intra-musculaires; gomme sous-periostee tibiale. Presence du parasite dans le sang. Bull. Mem. Med. Hop. Paris **25:**944–947.
35. **Wilson, D. E., J. J. Mann, J. E. Bennett, and J. P. Utz.** 1967. Clinical features of extracutaneous sporotrichosis. Medicine **46:**265–279.
36. **Woods, G. L., R. P. Wood, and B. W. Shaw, Jr.** 1989. Aspergillus endocarditis in patients without prior cardiovascular surgery: report of a case in a liver transplant recipient and review. Rev. Infect. Dis. **11:**263–272.