CORRESPONDENCE

Lymphocutaneous syndrome due to *Scedosporium apiospermum*

Scedosporium apiospermum is a ubiquitous fungus that can cause human infection by inhalation or after traumatic subcutaneous implantation. The clinical manifestations of infection by *S. apiospermum* are quite varied. The most common manifestation is mycetoma, a chronic granulomatous infection of the skin and subcutaneous tissue characterised by the triad of tumefaction, draining sinuses and grains [1]. The infection usually remains localised, and the course is indolent and chronic; eventually, however, it may disseminate by the lymphatic route, resembling sporotrichosis. Herein we report an unusual presentation of *S. apiospermum* soft tissue infection.

A 69-year-old woman was admitted because of a 3-week history of subcutaneous abscesses in the left upper extremity. She had a history of hypertension, rheumatoid arthritis, Sjögren's syndrome, chronic hepatitis C virus infection, and chronic renal failure; she had been receiving corticosteroids (prednisone, 10 mg daily) since 1995. She was admitted 1 month prior to current admission because of a 3-day history of fever, nausea and diarrhea. She was treated with parenteral hydration and antibiotics, with an improvement in her symptoms. Two days after discharge, she noted erythema and slight tenderness on the dorsum of her left hand, where a catheter had been inserted for parenteral administration of fluids. Over the following days, the dorsum of the hand became progressively swollen and erythematous, but painless, and suppurating nodules developed. She was treated empirically with trovafloxacin and cloxacillin, without improvement. While on therapy with cloxacillin, her clinical status deteriorated, with nausea, vomiting and diarrhea, and she was admitted to our hospital. Examination revealed a swollen area with dark-red erythema on the dorsum of her left hand, with bloody purulent discharge, and subcutaneous swelling extending to the elbow (Figure 1). Active and passive movements of the fingers and wrist were normal. Laboratory investigations showed hemoglobin 11.5 g/L, white cell count 21×10^9/L with 81% neutrophils, blood, urea, nitrogen (BUN) 145 mg/dL, serum creatinine 7.7 mg/dL, sodium 128 mEq/L, and potassium 3 mEq/L. Roentgenograms of the hand showed only soft tissue swelling. Surgical drainage was performed, and several pockets of thick, yellowish pus were widely debrided. Aerobic and anaerobic cultures of the pus were negative, but on Gram stain a filamentous fungus was disclosed. Culture on Saboraud's glucose agar medium grew *S. apiospermum*. The wounds were treated with daily povidone-iodine dressing changes and debridement, and itraconazole (200 mg daily) was started. On follow-up 10 weeks later, the swelling and ulceration had disappeared, with minimal residual erythema in the dorsum of the hand.

The lymphocutaneous syndrome (or nodular lymphangitis) may be produced by several fungal, bacterial, mycobacterial, parasitic and viral pathogens [2]. It is characterised by an initial lesion at the inoculation site, with secondary subcutaneous nodules and lymphangitis along the proximal lymphatic vessels draining the area. The most common causes of the lymphocutaneous syndrome are *Sporothrix schenckii*, *Nocardia brasiliensis*, *Mycobacterium marinum* and *Leishmania* spp. However, other microorganisms can rarely produce a sporotricoid-like soft tissue infection, including *S. apiospermum*.

Cutaneous infection by *S. apiospermum* is commonly expressed as a mycetoma. However, other lesions have been described in recent years, including cutaneous ulcers, subcutaneous abscesses, nodules, folliculitis-like eruptions, indurated erythema, and bullous necrotic purpura [3]. Whereas mycetoma is most common in healthy workers exposed to livestock and plants in tropical zones, most recently reported cases of *S. apiospermum* skin infection have been in immunocompromised hosts in developed countries. Infection due to *S. apiospermum* seems to be increasing. Miyamoto et al. [3] found 14 cases of skin infection by *S. apiospermum* reported between 1980 and 1997, with nine of these cases having been published between 1994 and 1997. The increasing occurrence of this infection is believed to be due to the enlarging immunosuppressed populations and the utilisation of invasive surgical procedures and prosthetic devices.

To our knowledge, only five cases of lymphocutaneous infection due to *S. apiospermum* have previously been reported [4–8]. Data from the case described here and the five previously reported cases are summarised in the Table 1. The analysis of these cases indicates that lymphocutaneous infection due to *S. apiospermum* affects patients with underlying immunosuppressive conditions. Patients usually report a history of penetrating trauma that results in implantation of the microorganism in the dermal tissue. Examination of the lesions showed swelling, erythema, ulcers and subcutaneous abscesses, with the characteristic spreading through lymphatic vessels of lymphangitis. In all cases, lesions localised at distal extremities.

Scedosporium spp. normally exist as saprophytic organisms and can be isolated from several sites worldwide, including soil, sewage, and polluted waters. Our patient acquired the infection in the hospital setting. It is plausible that the fungus contaminated her skin, and that it was introduced through a venipuncture. Contamination of the intravenous equipment seems unlikely, since no other cases have been detected, although this cannot be absolutely ruled out. *S. apiospermum* has been isolated from many sites, including cultivated indoor plants. Summerbell et al. [9] investigated the soils of five potted plants cultivated within a hospital for the presence of fungal pathogens and found that *S. apiospermum* was the most frequent opportunist.

Optimal treatment of *S. apiospermum* infections is not known; it has been advocated that treatment should include an antifungal agent along with surgical debridement when possible.

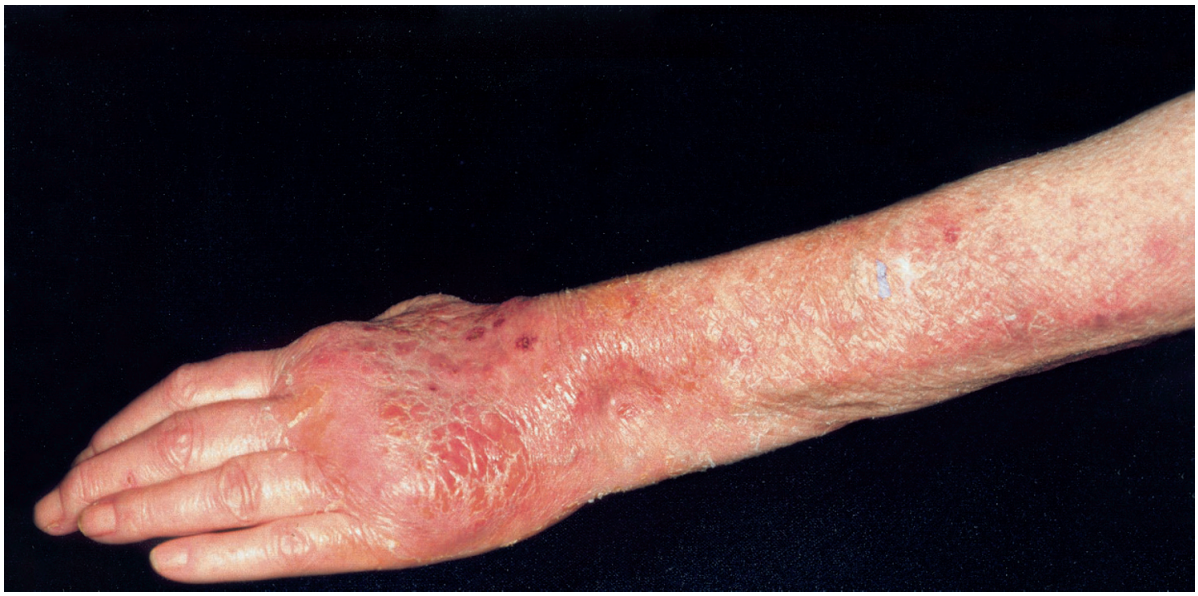

Figure 1 Skin lesion on the dorsum of the hand revealing a swollen area with dark-red erythema and subcutaneous swelling extending to the elbow.

Imidazolic agents have been advocated as the most active drugs against *S. apiospermum* [10]. Conversely, most strains of *Scedosporium* spp. are resistant to amphotericin B, which is considered the treatment of choice for serious fungal infections [10]. Besides itraconazole, both miconazole and ketoconazole have also been used to treat *S. apiospermum* infections [4,11]. In a recent study [12], voriconazole (UK-109,496), a new broad-spectrum triazole, showed greater in vitro activity against *S. apiospermum* than other antifungal agents. The combinations terbinafine–itraconazole [13] and amphotericin B–antifungal

Table 1 Lymphocutaneous infection due to *Scedosporium apiospermum*

Case (reference)	Age/sex	Underlying conditions	Localisation	Evolution	Previous trauma	Lesions	Treatment	Outcome
1 (4)	76/M	None	Upper extremity	1 year	No	Swelling, erythema, induration	Miconazole	Recurred (arthritis)
2 (5)	50/M	Corticosteroids, pemphigus vulgaris	Upper extremity	8 weeks	Yes	Ulcer, nodules	Sulfa drugs	Cured
3 (6)	73/M	Corticosteroids, RA	Lower extremity	16 weeks	No	Nodules, swelling	Itraconazole	Cured
4 (7)	63/M	Diabetes, alcohol abuse, AML, chemotherapy	Lower extremity	4 weeks	No	Swelling, nodules, ulcers	Itraconazole	Cured
5 (8)	67/M	Asthma, corticosteroids	Upper extremity	NR	NR	Erythema induration, purpura, ulcers, nodules	Fluconazole	Cured
6 (PR)	69/F	Diabetes, Sjögren, renal failure corticosteroids	Upper extremity	3 weeks	Yes	Erythema, nodules,	Itraconazole	Cured

AML, acute myeloblastic leukemia; NR, not reported; PR, present record; RA, rheumatoid arthritis.

azoles [14] have been shown to be synergistic against *Scedosporium* spp. Local therapy alone has been shown to be ineffective [5]. This particular antifungal susceptibility pattern makes prompt diagnosis of great importance, since immunocompromised hosts may present a rapidly deteriorating clinical course without appropriate therapy [15]. The optimal duration of therapy remains to be defined, although most authors have given antifungal drugs for a minimum of 3–4 weeks, associated with surgical debridement when indicated. Shorter courses have been associated with relapses [3].

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REFERENCES

1. Palestine RF, Rogers RS. Diagnosis and treatment of mycetoma. *J Am Acad Dermatol* 1982; 6: 107–11.
2. Smego RA, Castiglia M, Asperilla MO. Lymphocutaneous syndrome. *Medicine* 1999; 78: 38–63.
3. Miyamoto T, Sasaoka R, Kawaguchi M *et al*. *Scedosporium apiospermum* skin infection. A case report and review of the literature. *J Am Acad Dermatol* 1998; 39: 498–500.
4. Lutwick LI, Rytel MW, Peña-Yañez J, Galgiani JN, Stevens DA. Deep infections from *Petriellidium boydii* treated with miconazole. *JAMA* 1979; 241: 272–3.
5. Conti-Díaz LA. Micetomas y procesos premicetomatosos en el Uruguay. *Mycopathologia* 1980; 72: 59–64.
6. Cremer G, Bournerias I, Mhalla S *et al*. Scédosporiose cutanée non mycétomateuse chez un patient immunodéprimé. *J Mycol Med* 1994; 4: 111–14.
7. Török L, Simon G, Csornal A, Tápai M, Török I. *Scedosporium apiospermum* infection imitating lymphocutaneous sporotrichosis in a patient with myeloblastic-monocytic leukaemia. *Br J Dermatol* 1995; 133: 805–9.
8. Kusuhara M, Hachisuka H. Lymphocutaneous infection due to *Scedosporium apiospermum*. *Int J Dermatol* 1997; 36: 684–8.
9. Summerbell RC, Krajden S, Kane J. Potted plants in hospitals as reservoirs of pathogenic fungi. *Mycopathologia* 1989; 106: 13–22.
10. Hennequin C, Benailly N, Silly C *et al*. In vitro susceptibilities to amphotericin B, itraconazole, and miconazole of filamentous fungi isolated from patients with cystic fibrosis. *Antimicrob Agents Chemother* 1997; 41: 2064–6.
11. Ruxin TA, Steck WD, Helm TN, Bergfeld WF, Bolwell BJ. *Pseudallescheria boydii* in an immunocompromised host. *Arch Dermatol* 1996; 132: 382–4.
12. Cuenca-Estrella M, Ruiz-Díez B, Martínez-Suárez JV, Monzón A, Rodríguez-Tudela JL. Comparative in-vitro activity of voriconazole (UK-109,496) and six other antifungal agents against clinical isolates of *Scedosporium prolificans* and *Scedosporium apiospermum*. *J Antimicrob Chemother* 1999; 43: 149–51.
13. Meletiadis J, Mouton JW, Rodríguez-Tudela JL, Meis JFGM, Verweij PE. In vitro interaction of terbinafine with itraconazole against clinical isolates of *Scedosporium prolificans*. *Antimicrob Agents Chemother* 2000; 44: 470–2.
14. Walsh TJ, Peter J, McGough DA, Fothergill AW, Rinaldi MG, Pizzo PA. Activities of amphotericin B and antifungal azoles alone and in combination against *Pseudallescheria boydii*. *Antimicrob Agents Chemother* 1995; 39: 1361–4.
15. Berstein EF, Schuster MG, Stieritz DD, Heuman PC, Uitto J. Disseminated cutaneous *Pseudallescheria boydii*. *Br J Dermatol* 1995; 132: 456–60.

Pseudomonas mendocina **as a cause of chronic infective endocarditis in a patient with situs inversus**

Pseudomonas mendocina occurs in water and soil, like other pseudomonads [1,2], but is rarely recovered as a human pathogen. Its pathogenic role was first documented less than one decade ago as the infective agent causing mitral valve endocarditis in a 63-year-old man from Argentina [3]. We have recently isolated *P. mendocina* from three blood cultures from a woman with a tricuspid endocarditis [4], possibly lasting several years.

A 28-year-old woman with situs inversus was admitted to hospital in September 1999 because of abdominal pain, dyspnea, intermittent influenza like-symptoms, tricuspid stenosis and suspected endocarditis.

She had had three operations in 1979, 1982 and 1983 because of double-outlet right ventricle, ventricular septal defect (VSD) and pulmonary stenosis. An intraventricular baffle conducting blood from the left ventricle to the aorta was created. The VSD was closed by a Dacron patch, and the pulmonary cusps were resected.

In 1994, the patient was admitted to hospital because of 1 week of intermittent fever and suspected endocarditis. No signs of endocarditis could be found by echocardiogram at that time. However, she had an elevated hypersedimentation rate of 48 mm/h and elevated C-reactive protein of 900 mg/L. The antibody titers against staphylococci, streptococci or small Gram-negative rods, which often cause endocarditis, were all within the normal range.

After admission to hospital in September 1999, three sets of blood cultures were obtained and treatment with penicillin and gentamicin was begun because of suspected endocarditis. A transthoracic echocardiogram revealed significant tricuspid stenosis and fluttering vegetations on the systolic and the anterior tricuspid valves. After 48 h, Gram-negative bacilli were grown in all blood cultures and were identified as *P. mendocina*, based on biochemical reactions discussed previously [2,3,5]. The therapy was subsequently changed to ampicillin and gentamicin, due to