Case Report

Disseminated sporotrichosis in a patient with a past history of lepromatous leprosy: a case report

Su-Ming Wong* & Jyh Jong Tang†
*Dermatology Unit, Department of Medicine, University Malaya, Kuala Lumpur, and the †Department of Dermatology, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

Disseminated sporotrichosis is uncommon and usually occurs in patients who are immunodeficient. Here we describe a male patient who was otherwise in good physical condition, who presented with disseminated sporotrichosis. The only significant event in his past medical history was lepromatous leprosy which had been treated 42 years earlier.

Keywords disseminated sporotrichosis, leprosy

Introduction

Sporothrix schenckii is widely known as the etiologic agent of sporotrichosis. Recent gene sequencing has shown that other Sporothrix species (now referred to as the S. schenckii complex) may also cause infection in humans. These include S. albicans, S. brasiliensis and S. globosa [1]. Disseminated sporotrichosis is rare and usually occurs in the context of a deficient cell-mediated immunity. It often results from haematogenic dissemination of the pathogen from the initial site of transmission, whether cutaneous or respiratory [2]. The majority of disseminated cases of sporotrichosis involve internal organs, although cases of disseminated cutaneous infections without systemic involvement have also been reported [3]. Here, we describe a case of a man who presented with disseminated sporotrichosis who had a previous history of lepromatous leprosy.

Case report

A 61-year-old owner of a flower nursery who had a previous history of lepromatous leprosy, presented with generalized crusted skin ulcers of three-month duration. The lesions initially started as nodules on the face which then spread to trunk and limbs. Subsequently, these lesions formed abscesses which ruptured and became crusted ulcers. He had associated pain, fever, anorexia and loss of weight. There was no history of preceding trauma or animal bites. His only significant medical history was that of lepromatous leprosy in 1968 which had been treated. Prior to this presentation, he had been well and there were no signs and symptoms to suggest a recurrence of leprosy. On examination, he was afebrile and there was no jaundice or lymphadenopathy. There were generalized skin ulcerations covered by necrotic crust and slough with punched out margins (Fig. 1A–C). His hands and feet were deformed as a result of nerve damage because of his leprosy. Systemic examination was normal. Our differential diagnosis initially included a relapse of leprosy with ulcerative erythema nodosum leprosum, disseminated fungal infection, disseminated herpes infection and cutaneous melioidosis. A skin biopsy revealed a granulomatous reaction with numerous budding yeast-like cells in the dermis (Fig. 2). Slit skin smears were negative for acid fast bacilli. Sporothrix schenckii was isolated from portions of the skin biopsy inoculated onto Sabouraud glucose agar and incubated at 25°C (Fig. 3A). Dimorphism was proven by the finding of the mycelia form of the pathogen in cultures incubated at 28°C (Fig. 3B) and yeast forms at 37°C. Screening for HIV, hepatitis B, hepatitis C and syphilis was negative. In addition, screening for underlying primary immunodeficiency such as total T-cell, B-cell,
immunoglobulin, CD4, natural killer (NK) cell and phagocytic function tests using flow cytometric analysis were all normal except for a slightly reduced CD8 count. Herpes immunofluorescence and melioidosis serology were also negative.

He was diagnosed as having disseminated cutaneous sporotrichosis with no internal organ involvement. Intravenous (IV) amphotericin B (0.5 mg/kg/day) was started and his skin lesions slowly dried up. After two weeks of treatment, amphotericin B was changed to oral itraconazole at 200 mg twice a day and his skin lesions continued to improve. The patient subsequently developed a bacterial nosocomial sepsis. Blood cultures grew *Acinetobacter*, *Klebsiella* and Methicillin-resistant *Staphylococcus aureus* (MRSA). He was started on multiple courses of intravenous broad-spectrum antibiotics, but his condition deteriorated and he eventually succumbed.

**Discussion**

Disseminated sporotrichosis is almost always associated with an immunodeficient or debilitated state of the patient caused by alcoholism, diabetes, sarcoidosis, chronic obstructive airway disease, tuberculosis, organ transplantation, malignancy, use of immunosuppressive agents or acquired immunodeficiency syndrome (AIDS) [1,4,5]. The majority of disseminated cases have been reported in HIV patients [6–8].

Clinically, disseminated sporotrichosis in AIDS patients is highly variable and includes such symptoms as ulceration,
Disseminated sporotrichosis in a patient with lepromatous leprosy

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after one month of antifungal therapy although he subsequently died due to nosocomial sepsis.

Acknowledgements

We would like to thank the doctors and all other staff involved in the care of this patient at the Department of Dermatology, Hospital Kuala Lumpur.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References