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CASE REPORT

Acral necrosis by *Stenotrophomonas maltophilia*

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ABSTRACT

Background *Stenotrophomonas maltophilia* (SM) has been considered a nosocomial pathogen. Nevertheless, community acquired infection may occur more frequently than usually recognized.

Case We describe distal necrosis of the fingers by SM in a farmer, contracted in the community and successfully treated with a combination of cotrimoxazole and ciprofloxacin. The patient was diagnosed with chronic lymphocytic leukaemia 6 months later.

Conclusions This unusual presentation shows that infection with SM should be included in the differential diagnosis of the skin and soft tissue infection, even in apparently healthy patients.

Key words: necrosis, skin and soft tissue infection, *Stenotrophomonas maltophilia*

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Introduction

Stenotrophomonas maltophilia (SM) is a Gram-negative bacillus, formerly known as *Pseudomonas* or *Xanthomonas maltophilia*.[1] It is implicated as an opportunistic pathogen in hospitalized, neutropenic patients or those on broad-spectrum antibiotics.[2,3] Rare cases of cutaneous infections in farmers have been reported, as it is a microorganism isolated from soil and agricultural plants.[4]

To our knowledge, this is one of the first cases to describe distal necrosis of the fingers due to community acquired SM infection by an apparently healthy patient.

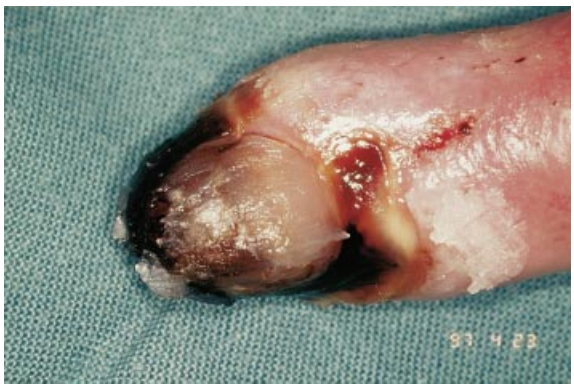

fig. 1 Necrosis of fifth finger extremity.

Case report

A 78-year-old Caucasian male farmer, living in a rural area, with a cigarette consumption of 40/day and an alcohol intake of about 130 g/day. His past medical history was unrevealing. He sought medical assistance due to incapacitating pain and progressive distal necrosis of the fourth and fifth fingers for 18 months. There was no history of Raynaud's syndrome. The patient had been treated with several antibiotics, with doubtful compliance, and without success.

On examination, there were erythematous and erosive lesions of the fingers and adherent black crusts on the distal extremities of the fourth and fifth fingers, which were removed with difficulty, leaving ulcers filled with a purulent exudate (figs 1 and 2). The remainder of the physical examination, together with a specialist cardiac and vascular evaluation, did not show any significant abnormalities. Laboratory examination showed a macrocytic anaemia due to folic acid deficiency (Hb, 11 g/dl; folate, 3 mg/ml; B_{12}, 656 pg/ml), and a white cell count of 11 000 leucocytes/mL (27% neutrophils, 63% lymphocytes). Liver and renal function tests, HIV serology, immunological screen (autoantibodies, cryoglobulins, antiphospholipid and anticardiolipid antibodies) were within normal range. Chest X-ray was normal and X-ray of the hands ruled out osteomyelitis. Histopathology showed epithelial hyperplasia and a dense homogeneous collagenization on the upper dermis. A

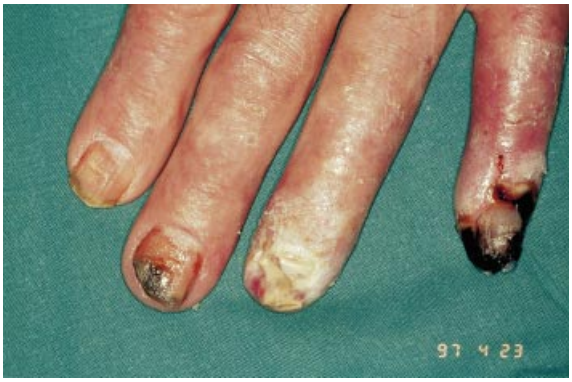

fig. 2 Purulent exudate after removing the nail of the fourth finger.

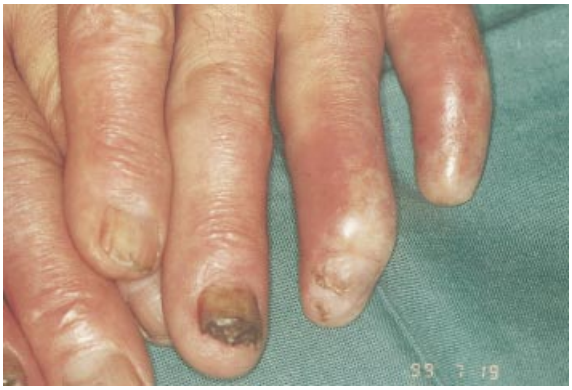

fig. 3 Resolution resulted in fourth nail dystrophy and anonychia of the fifth.

culture of the purulent exudate, in blood, chocolate and MacConkey agar, grew an abundant monoculture, identified by Vitek and API20NE as SM. The isolates were sensitive to cotrimoxazole [trimethoprim/sulphamethoxazole (TM/SMZ)] and doxycycline, with intermediate susceptibility to ciprofloxacin.

Antibiotic therapy, with oral TM/SMZ 160/800 mg twice daily, for 4 weeks, resulted in progressive improvement of the lesions. On recommencing farm work without gloves, there was a clinical deterioration. A combination of oral TM/SMZ 160/800 mg and ciprofloxacin 750 mg, twice daily was then instituted. There was clinical improvement despite the discontinuance by the patient after 2 weeks (fig. 3). Six months later, the patient was diagnosed with stage A chronic lymphocytic leukaemia, with a persistent mild lymphocytosis in peripheral blood.

Comments

SM is a Gram-negative bacillus, the sole member of the *Stenotrophomonas* genus. Its routes of acquisition are not completely known. It has been isolated from several environments where it occupies ecological niches, such as water, soil, and especially grasses, wheat and other cereals.[1,4] It is also detected in food sources, including frozen fish, milk and eggs. In hospitals, it has been isolated from blood-sampling tubes, sphygmomanometers, disinfectant solutions, oxygen humidifier water reservoirs, dialysis machines and hands of health-care personnel.[1]

It is an important opportunistic nosocomial pathogen, associated with a significant mortality rate, especially in patients who are neutropenic, debilitated, immunocompromised, or on broad-spectrum antibiotics.[2,3,5] There are references suggesting the existence of an altered microenvironment in neoplastic tissue favourable for the multiplication of SM. Consequently, in the presence of SM infection, malignancy should be ruled out.[6]

SM is responsible for a wide spectrum of clinical manifestations, namely endocarditis, respiratory, urinary, gastrointestinal infections and bacteraemia, frequently without a known portal of entry.

The most frequently described skin and soft tissue manifestations due to SM infection are subcutaneous nodules[2,7,8] and primary or metastatic cellulitis, which may manifest as ecthyma gangrenosum.[1,5] The latter is probably connected to the production of protease and elastase enzymes by the bacteria[9] and seen in malignant disease, especially in haematological patients. SM is frequently isolated from surgical wounds (e.g. tracheostomies, vascular catheter sites) or traumatic wounds such as injuries caused by corn-harvesting machines.[4] Other manifestations include umbilical cellulitis,[10] prepatellar bursitis[11] infections of burn wounds,[12] cat scratches and human bite wounds.

Treatment in these situations is complicated by the emergence of mutations and resistant strains to numerous broad-spectrum antibiotics.[1,3,13,14] Moreover, *in vitro* susceptibility data frequently do not correlate with a successful clinical outcome.[1] Cotrimoxazole with or without ticarcillin-clavulanate is the current treatment of choice.[2] Other agents considered in documented studies are minocycline, doxycycline and quinolones. New generation quinolones (trovafloxacin, clinafloxacin, moxifloxacin, sparfloxacin) have an enhanced *in vitro* activity against SM and could become an alternative if clinical studies confirm these results.[15] Some authors refer to the synergistic effect of a combination of antimicrobial agents.[1,16]

Despite the fact that SM has been considered to be a nosocomially acquired pathogen, community acquired infection with this bacterium may occur more frequently than previously recognized.[1] The present case is an example: an elderly patient in a rural setting, in contact with products that are the natural habitat of the pathogen and with no evidence of the usual associated risk factors. Some degree of immune deficiency due to his heavy alcoholic and smoking habits may be postulated.[17] Subsequently, a chronic lymphocytic leukaemia was diagnosed – reinforcing the opinion, that with the presence of SM infection in the absence of predisposing factors, an associated malignancy should be ruled out. On the other hand, this case is even more unusual because of the clinical presentation and excellent therapeutic result.

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