Cutaneous Infection with *Scedosporium apiospermum* in a Patient Treated with Corticosteroids

Sir,

*Scedosporium* is a widely distributed fungus that is common in the environment. Human infection with this fungus is facilitated by immunosuppression caused by certain diseases or their treatment. We report here a case of cutaneous scedosporiosis occurring in a man treated with corticosteroids for systemic sarcoidosis.

CASE REPORT

A 64-year-old man was referred with a 3-month history of 2 contiguous subcutaneous, non-inflammatory painless nodules on the back of his right elbow (Fig. 1). His health was good on admission. The skin in this area was thin, fragile and atrophic, with numerous ecchymotic lesions. Cutaneous biopsy of 1 nodule showed a granulomatous dermal infiltrate with giant cells. Periodic Acid-Schiff staining revealed intra- and extra-cellular spores and septate hyphae. Oral ketoconazole was prescribed for 3 weeks. Six months later, cutaneous examination showed 4 new non-inflammatory nodules on the right elbow, adjacent to the biopsy scar. Further biopsy showed a similar pattern to the first, and culture on Sabouraud's agar revealed colonies of *Scedosporium apiospermum*. Serum tests for scedosporiosis were negative. The patient was retired, and undertook some gardening, but the mould was not found in the soil or vegetation in the patient’s garden. The patient was treated with oral itraconazole (400 mg twice daily) for 1 month. Six months later, the remaining nodules appeared atrophic and no other lesion had appeared.

The patient had a previous history of systemic sarcoidosis with initial hypercalcaemia, granulomatous nephropathy, hepatitis and cytopenia that had been treated with prednisone for 18 months. The initial dose of 1 mg/kg/day was progressively tapered, without relapse during the first year, but then the dose was increased again because of recurrence of the disease. The dose was 0.2 mg/kg/day at the first occurrence of cutaneous nodules and 0.5 mg/kg/day at the second. Humoral immunity was preserved, with normal immunoglobulin levels (IgA 2.36 g/l, normal 1.06 – 3.39 g/l; IgG 8.20 g/l, normal 6.50 – 12.10 g/l; IgM 2.32 g/l, normal 0.52 – 1.48 g/l). The lymphocyte count was 1 336 g/l, with normal CD4 and CD8 counts (908/mm³ and 415/ mm³, respectively). Chest X-ray and computed tomodensitometry of the chest were normal. HIV-testing was negative.

DISCUSSION

Cutaneous nodules are well known in systemic sarcoidosis, and they are usually due to granulomatous lesions of the skin. The first biopsy did not show any sarcoidal lesion, but did reveal fungal infection of the skin. Diagnosis of cutaneous infection with *Scedosporium apiospermum* was made on the basis of the histology examination, which revealed the presence of hyphae deep within the dermis, and because *Scedosporium apiospermum* was isolated from a skin biopsy culture.

*Scedosporium apiospermum* (the anamorphic asexual form of *Pseudallescheria boydii*) is a widely-distributed mould that can be found in the soil, manure and decaying vegetation. The portal of entry can be the lungs, sinuses, or a site of trauma to the skin. This fungus is a classical cause of mycetoma in tropical countries. Penetration can occasionally lead to localized infection (bones, joints, skin, eyes), or can cause severe generalized infection in immunosuppressed patients (leukaemia, corticotherapy (1), chronic granulomatous diseases (2), AIDS). Cutaneous infection gives rise to sporotrichoid nodules with possible ulceration (3).

Our patient had cutaneous fragility due to long-term corticotherapy, and often injured himself while gardening. He may have inoculated himself with *Scedosporium apiospermum* during this activity, infection being facilitated by the immunosuppression due both to sarcoidosis (4) and its treatment. However, no signs of disseminated fungal infection were found, and in particular there was no respiratory involvement.

Itraconazole was chosen because it has been reported to be successful in curing infection with *Scedosporium apiospermum* (5) in some cases. Other antifungal agents (ammphotericin B, ketoconazole, fluconazole) are often ineffective, except for miconazole (6).

REFERENCES

Disseminated Punctate Intraepidermal Haemorrhage: A Widespread Counterpart of Black Heel

Sir,

The appearance of petechiae in a patient is always a distressing event. However, there are banal causes of purpura, such as the one we report here.

CASE REPORT

An 80-year-old man was presented with decubitus ulcers. He had paraplegia secondary to severe osteoarthrosis of the lumbar spine and usually stayed in bed barefoot. He had no signs of other diseases, in particular vascular or cardiac disease. On physical examination he was found to have numerous, well-demarcated petechiae disseminated on the dorsal and plantar aspects of both feet. Petechiae were also present on the toe webs. They were not palpable and, considering the petechiae’s sharp margins and “superficial” appearance, we performed a light curettage, which detached them (Fig. 1). Biopsy of one of the lesions showed the presence of a subcorneal mass of eosinophilic amorphous material, close to acrosyringiums. This material stained blue-green with Patent blue V, favouring a haematic origin (1). Haemosiderophages were present, scattered on the papillary dermis. A haemogram was normal. When asked about previous trauma, the patient could only recall a difficult transport from his home, with several sudden movements of his legs. The lesions disappeared spontaneously after 1 week.

DISCUSSION

The most frequent form of intraepidermal haemorrhage is “black heel”, well known as a differential diagnosis of pigmented lesions on the feet. Other forms of intraepidermal haemorrhage have been described, such as lesions similar to black heel, but located in other areas of the foot (2), or grouped palmar petechiae (3 – 5). The name “post-traumatic punctate haemorrhage” has been proposed as a unifying term (5). Other related lesions are subungual splinter haematomas, black subungual dots in patients with chronic radiodermitis, and posttraumatic haemorrhage under circumscribed hyperkeratosis (2). We have not found any descriptions of punctate disseminated lesions on the feet as seen in our patient.

The pathogenesis of previously mentioned lesions has been considered to be traumatic, although other causes are possible, as described for subungual splinter haematomas (such as infectious endocarditis, antiphospholipid antibody syndrome, or arterial catheterization). After a haemorrhage in the papillary dermis, blood is eliminated transepidermically, through the least resistant periductal areas (2). The fact that our patient could not walk might be a predisposing factor for his lesions, as the skin of his feet might be thinner than usual. These characteristics lesions could be mistaken for purpura associated with severe illness. The simple method of trimming the surface of the lesions can make the diagnosis clear and might avoid unnecessary work-up for the patient.

REFERENCES


Accepted March 31, 1999.

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