

Necrotizing Soft Tissue Infection of the Glans Penis due to Atypical *Candida* Species Complicated with Fournier's Gangrene

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Sir,

Necrotizing soft tissue infections are characterized by tissue necrosis, ranging from superficial dermal involvement to deeper involvement of the fascia and muscle layers. Between 500 and 1500 cases are reported annually in the USA, and the mortality ranges from 24–34%. Most infections are polybacterial and are usually caused by a mix of aerobes and anaerobes (1).

In contrast, necrotizing soft tissue infections due to fungi are very rare (2–4). We describe here a case of superficial necrosis of the glans penis due to *Candida glabrata* and *C. tropicalis* complicated with conventional Fournier's gangrene of the scrotum in an immunosuppressed patient.

CASE REPORT

A 59-year-old man was referred to our department with a painful lesion on the glans penis. His medical history was complex and included a renal transplantation in 2004, type 1 diabetes mellitus and arterial hypertension. Glycaemic control was inadequate, with an HbA1c level of 8.6%, but graft function was stable on immunosuppressive therapy with azathioprine, cyclosporine and prednisolone. Examination revealed a pale-yellow, tender, non-infiltrated patch affecting part of the glans penis. Venereal disease was ruled out and bacterial and fungal swabs from the lesion were negative. A punch biopsy was obtained from the glans penis, and histological examination showed superficial tissue necrosis with no signs of secondary inflammation, calcified blood vessels or fungal infection. Direct immunofluorescence was also negative. The absence of inflammation was probably due to the immunosuppressive therapy.

Despite topical treatment with clobetasol propionate and mupirocin the lesion enlarged over a period of 6 weeks to cover the entire glans penis as a white necrotic patch (Fig. 1). Furthermore, the situation was complicated by necrosis of the scrotal and perineal skin, typical of Fournier's gangrene (Fig. 2). A further punch biopsy from the glans penis, obtained under aseptic conditions, was cultured and grew *C. glabrata* and *C. tropicalis*, but not bacteria. The patient was immediately treated by resection of the necrotic scrotal skin and the left testis. Culture of the scrotal tissue was positive for *Escherichia coli*, *Enterococcus faecalis*, mixed anaerobes, *C. glabrata* and *C. tropicalis*. Both yeasts were sensitive to amphotericin B, which, however, was considered



Fig. 1. Pale-yellow patch covering the glans penis, superficial necrosis.

contraindicated because of the risk of nephrotoxicity. The isolated *C. glabrata* strain showed azole resistance, but was sensitive to caspofungin. Therefore, intravenous caspofungin (50 mg once daily) was administered as well as tazobactam (Tazocin®), ciprofloxacin (Ciproxin®) and metronidazole with clinical improvement of the penile lesion within 5 days (Fig. 3). The scrotal and perineal areas were surgically revised several times and the patient made a full recovery.



Fig. 2. Necrosis of the scrotal and perineal skin, Fournier's gangrene.



Fig. 3. Clinical improvement after 5 days of treatment with intravenous caspofungin.

DISCUSSION

To our knowledge, this is the first report of initial superficial penile necrosis due to *C. glabrata* and *C. tropicalis* complicated with conventional Fournier's gangrene of the scrotum. In general, necrotizing soft tissue infections caused by these fungi are rare (2–4); they are usually caused by bacteria.

These infections are not clearly defined, but they can be regarded as soft tissue infections associated with tissue necrosis. All soft tissue compartments, from the superficial dermal layers to deep fascia and muscle, can be affected (5). Intravenous drug abuse, alcoholism, chronic renal insufficiency, peripheral vascular disease, heart disease, trauma, diabetes mellitus, immunosuppression and obesity appear to be associated with an increased susceptibility (5, 6).

Diagnosis is often difficult as there are no distinct clinical features and because the epidermis may appear relatively normal despite extensive involvement of deeper tissue layers. Diagnostic delay may, in turn, lead to delayed surgical debridement, resulting in higher mortality. Therefore, the key to an initial diagnosis

seems to be the recognition of a necrotic component as this will identify patients requiring surgical debridement (5, 6). Our patient initially presented with an atypical superficial necrosis on part of the glans penis with no apparent cause and there was no indication for surgical debridement before the onset of scrotal involvement when fulminant Fournier's gangrene was apparent. The responsible organism was not identified despite thorough work-up until tissue was cultured. We therefore recommend that a fungal aetiology is considered early in the case of any necrotizing soft tissue infection in the presence of immunosuppression. Interestingly, yeasts other than *C. albicans* are more often found to be the cause of invasive fungal infections in immunocompromised patients and a relatively large number of these non-albicans *Candida* species are resistant to azole therapy, whereas resistance to caspofungin is virtually non-existent (7). Therefore, caspofungin should be considered over traditional azole therapy if empirical anti-fungal therapy is initiated.

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