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Case Report

Saksenaea vasiformis: a multifaceted approach to a complex case

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ABSTRACT

Saksenaea vasiformis is a rare, opportunistic Mucorales fungus capable of causing rapidly progressive cutaneous and subcutaneous infections, including necrotising fasciitis. Early diagnosis is challenging due to its poor sporulation on routine media and often subtle initial clinical features. We describe a case of a soft-tissue infection in a co-morbid 75-year-old man from rural Australia with limited healthcare engagement. Despite broad-spectrum antibacterial therapy, operative intervention and negative-pressure wound-therapy, the wound continued to deteriorate. Eventually, sequencing of the internal transcribed spacer region confirmed *S. vasiformis*. The patient was commenced on liposomal amphotericin B followed by prolonged oral itraconazole due to underlying renal impairment and geographic barriers to ongoing inpatient care. This case underscores the need for early suspicion of atypical fungal pathogens in necrotic soft-tissue infections unresponsive to antibacterial therapy and highlights the importance of prompt surgical management, infectious diseases input, specialised fungal diagnostics and tailored antifungal treatment.

Keywords: *Saksenaea vasiformis* infection, Mucormycosis, Surgical debridement, Wound infection

INTRODUCTION

Saksenaea vasiformis is a species of fungus belonging to the order Mucorales, within the subphylum Mucoromycotina. It was first identified in 1953 as a pathogen within the facial wounds of a young man.1 In more recent years, based on molecular, morphological and physiological characteristics, *S. vasiformis* has been revealed as a complex of species that includes *S. oblongispora, S. erythrospora, S. loutrophoriformis, S. trapezispora,* and *S. dorisiae*, with all but S. dorisiae implicated in mammalian infection.[2] It is a rare cause of the aggressive, invasive spectrum of disease caused by mucormycoses which can present with rhino-cerebral, pulmonary, cutaneous and subcutaneous infections. Identified risk factors for Saksenaea infections include poorly controlled diabetes mellitus, cancer, use of immunosuppressants like corticosteroids, renal insufficiency and burn wounds with approximately 80% of the reported cases of *Saksenaea vasiformis* infections being reported in immunocompetent hosts.[3,4]

CASE REPORT

We present a case of a 75-year-old male patient who initially presented to a regional centre before being transferred to our tertiary hospital. He was from a rural community in South Australia with a background of hypertension, chronic kidney disease (baseline creatinine 140-160), and atrial fibrillation. He was an active smoker of marijuana six cigarettes daily, occasional smoker of methamphetamine, and consumed six standard drinks within 1 week. His reported regular medications were apixaban, perindopril, and pantoprazole, however his healthcare engagement was poor and medication compliance was variable.

He presented to his local health service with a 4-day history of an evolving lesion to the right medial thigh with progressive surrounding cellulitis without any reported trauma or obvious evidence of bites. He did note that he had similar lesions in his arm thought secondary to insect bites 4 weeks prior which resolved spontaneously. After discussion with our centre's general surgical team, he was transferred for urgent surgical review.

On review at our centre, he was haemodynamically stable but had intermittent febrile episodes. Initial examination showed a 3×3cm lesion on the right medial thigh that was firm, tender and surrounded with indurated, erythematous tissue. There was a central break in the lesion without discharge. The lesion was superior to the knee but there was normal knee range of movement, no obvious knee effusion or features of joint involvement.

Right knee radiograph demonstrated no acute fracture or joint effusion but there was subcutaneous oedema surrounding the lesion. Bloods taken at time of arrival demonstrated as follows: haemoglobin level of 139, white cell count 15×109/l, C–reactive protein of 72, serum creatinine 165, albumin 33. A superficial wound swab showed no polymorphs or organisms on microscopy and only skin flora was reported on final cultures.

He was continued on piperacillin/tazobactam after receiving a single dose at the regional hospital, and vancomycin was commenced as he was colonised with methicillin-resistant *Staphylococcus aureus* (MRSA). He was listed for an operative incision and drainage; however, this was delayed by 48 hours to allow for the apixaban washout period. There was minimal clinical response to the antibiotic regimen preceding the operative intervention.

Intraoperatively, necrotic tissue spanning 8×8×4 cm had extended down to the muscular fascia. This was excised and debrided until healthy fat and skin was on view (Figure 1), followed by wound packing of the opened cavity with alginate calcium sodium dressing to aid haemostasis. Excised tissue was sent for microscopy and bacterial culture. Given the necrotic tissue findings intra-operatively, antibiotic coverage was broadened post-operatively to cover for necrotising fasciitis with meropenem, clindamycin, and vancomycin. Initial microscopy was reported as 1-2+ polymorphs with no bacteria seen. Bacterial cultures grew *Staphylococcus epidermidis*. Antibacterial therapy was rationalized to vancomycin and flucloxacillin after two days. At day 4 post first debridement, scant growth of a fungus was seen on chocolate and anaerobic plain agar plates and referred to the mycology laboratory for identification. The significance of the unidentified organism was unclear and as the patient was clinical improving, empiric antifungal therapy was not initiated at this point.

The patient received daily wound packing before being transitioned to vacuum assisted closure (VAC) therapy after six days. However, after initial improvement his wound became increasingly necrotic, with worsening surrounding cellulitis and he returned for additional operative debridement to healthy granulation tissue eight days after the initial debridement (Figure 2). Specimens sent from the second debridement were also negative on direct fungal microscopy, but a fungus grew on agar plates after two days.

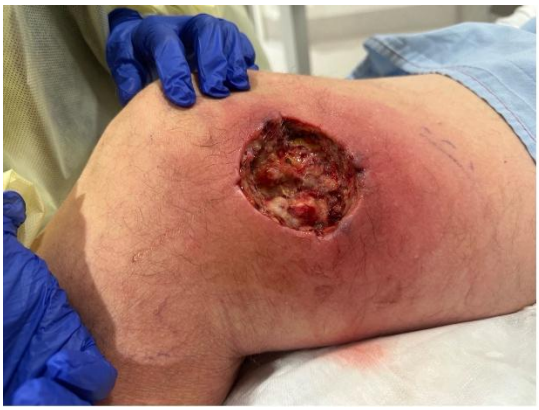

Figure 1: Right medial thigh wound post first wound debridement.

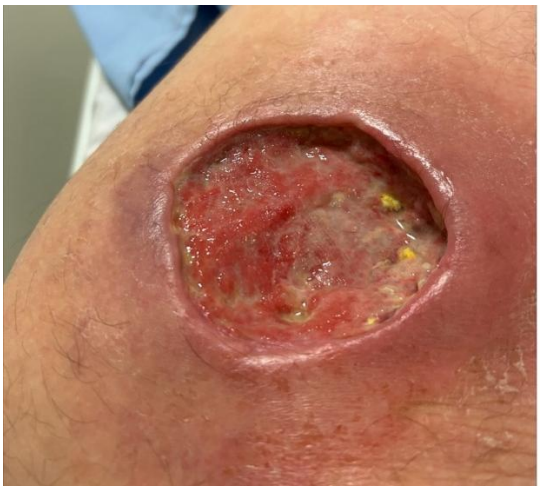

Figure 2: Right medial thigh wound post second debridement prior to re-application of VAC dressing.

The fungal hyphae from culture were wide, ribbon-like, pauciseptate with irregular branching, resembling a fungus within the Mucorales group. The infectious diseases (ID) team was consulted and intravenous liposomal amphotericin B was commenced. Flucloxacillin and vancomycin was ceased after a two-week course. The fungal organism was identified as *S. vasiformis* by sequencing of the internal transcribed spacer (ITS) ribosomal DNA region.

Given the patient's chronic renal impairment and to minimise nephrotoxicity, liposomal amphotericin B was administered by extended daily infusion over 16 hours with pre- and post- hydration.

Amphotericin B as ceased on day 14 post first-debridement and the patient was commenced on oral itraconazole as step-down oral therapy with 3 months of treatment planned.

Outcome and follow up

In preparation for discharge, the patient's usual anticoagulation regimen of apixaban was changed to enoxaparin due to drug-drug interaction with itraconazole. Additionally, a VAC dressing was reapplied to the wound cavity. He was discharged with daily country home nurses for enoxaparin administration, VAC dressing management and ongoing remote ID outpatient review. These reviews were conducted by video link with assistance of the Royal Flying Doctor nursing service. Figure 3 illustrates wound progress on oral therapy. Follow up 4 months post-treatment showed resolution of the infection (Figure 4).

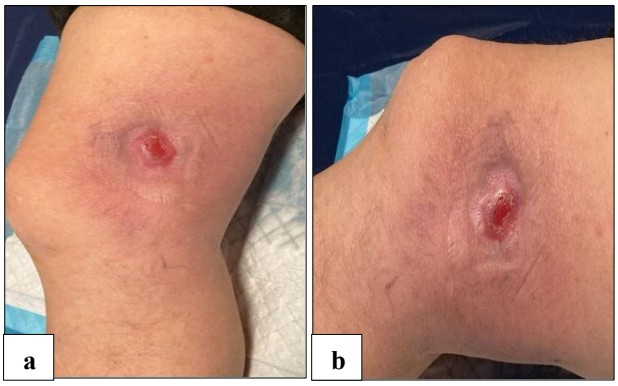

Figure 3 (a and b): Right medial thigh wound 8-weeks initial debridement.

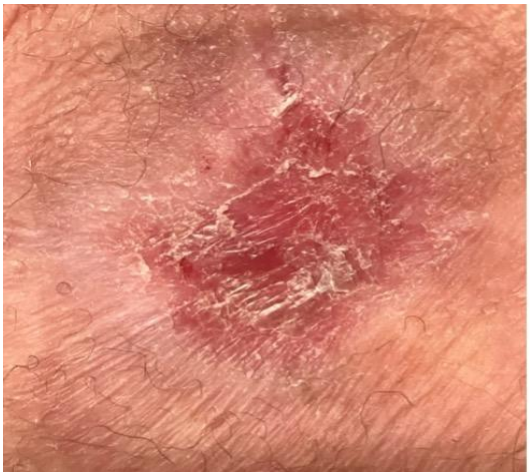

Figure 4: Right medial thigh wound 4 months post initial debridement.

DISCUSSION

Like other fungi of the Mucorales, *Saksenaea spp.* are found worldwide and associated with soil and organic material (wood, fruit, excrements). Primary cutaneous infection usually occurs through implantation due to trauma, surgery or burn wounds. It can be limited to local invasion but may also disseminate to deeper sites through secondary vascular invasion and haematogenous spread, resulting in cases of necrotising fasciitis and osteomyelitis. Cutaneous mucormycoses have been reported after natural disasters including volcanic eruptions and tsunamis, as well as following scorpion stings, magpie pecks and vehicle related trauma.[5-7] The clinical course of mucormycosis can be rapid, aggressive and often fatal.[8] Mortality remains high, with a case series in India of 65 cases demonstrating 37.5% mortality.[9] Disease can rapidly progress towards life threatening complications including necrotizing fasciitis.

Localised treatment

In ulcerated necrotic tissue, early and adequate surgical intervention for wound debridement in conjunction with appropriate antimicrobial coverage is the gold standard. In cases of *S. vasiformis* infection, morphological identification is challenging due to non-sporulation of this genus without interventions requiring time and specific expertise.

Molecular-based identification methods are typically faster. Instances of cellulitis that fail either to improve with typical antibacterial therapy or to grow causative bacteria should arouse clinical suspicion of organisms requiring specific culture conditions such as mycobacteria and fungi, which require specific microscopy and cultures to be ordered on surgical samples.[10]

Negative-pressure wound therapy via VAC dressing aims to remove excess fluid and increase circulation to the healing wound by stimulating revascularisation.[11] VAC therapy has become gold standard in treating complex wounds, however given the progression of erythema and exudate of the wound in this case despite the VAC dressing, there was some retrospective concern the dressing may have contributed to microbiological proliferation.[11,12] However, this remains controversial in the literature and prompts further in-vivo studies to evaluate the impact on fungal growth with these dressings.[13] Additionally, VAC dressing changes are usually performed at two-to-three-day intervals, which may delay detection of wound deterioration in the early period, particularly if an organism is not yet identified.

There is a paucity of high-quality evidence relating to the use and efficacy of instillation of topical antimicrobial agents with VAC dressings, particularly with respect to fungal infections. A case report on a 53-year-old man with a mucormycosis infection of the Achilles tendon demonstrated good recovery from the use of the adjunct instillation wound VAC with topical amphotericin B.[14]

The use of topical agents alone would not be recommended in the management of even localized *S. vasiformis* due to its propensity for invasive infection.

Systemic treatment

There is limited evidence to guide systemic antifungal treatment options of *S. vasiformis*. In general, polyene therapy with amphotericin B is the first line treatment for all Mucorales fungi. Due to the poorly-sporulating nature of this genus, in vitro susceptibility testing of antifungal drugs is rarely possible. In published studies, amphotericin B, posaconazole and itraconazole have had good activity against *Saksenaea spp.*, and as with other Mucorales fungi, voriconazole, fluconazole and the echinocandin class have no activity.[15,17] The primary limitation of amphotericin B remains its nephrotoxicity; lipid formulations currently in routine use, are significantly less nephrotoxic, providing safety at higher doses. Pre-and post-dose hydration to improve renal perfusion can also be protective against nephrotoxicity. In our case, the patient was changed to the supra-bioavailable formulation of itraconazole 100 mg daily given its favourable pharmacological profile, especially in the context of the patient's chronic kidney disease which meant long-term amphotericin B would be unsuitable. In addition, the patient lived in a rural town approximately 600 km away from the nearest tertiary centre, which meant an easily locally available oral medication was preferable. In reviewing the available literature, this is one of only a few cases where itraconazole has been used for the successful treatment of proven *S. vasiformis* infection.

Mycology considerations

Surgical tissue should be cultured intact, or with gentle teasing apart of pieces, to avoid damage to pauciseptate hyphae. Infections with *S. vasiformis* (Figure 5) can be challenging to diagnose as they do not sporulate on standard fungal media and instead require specialized culture techniques.[22] Sporulation may be induced with growth on nutrient-depleted media such as "tap water agar" or Czapek Dox Agar after 7-14 days.

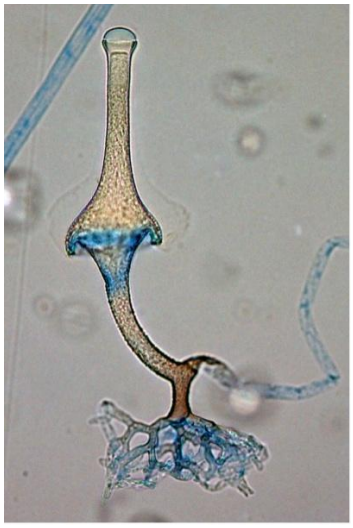

Figure 5: *Saksenaea vasiformis* sporangiophore (65-100 µm long) with vase-shaped sporangium and distinctive pigmented rhizoids.

The identification of a vase-shaped sporangium (hence the *vasiformis* epithet) with pigmented rhizoids is pathognomonic for *S. vasiformis* complex.[23] However molecular identifications is preferable for any non-sporulating Mucorales fungus, typically by sequencing of the ITS ribosomal DNA region. In cases where cultures remain negative but evidence for fungal infection is demonstrated by direct microscopy, panfungal PCR can be attempted, with diagnostic sensitivity improving with the use of fresh tissue rather than formalin fixed tissue.[29]

CONCLUSION

This case highlights the importance of early clinical suspicion for atypical fungal pathogens such as the *Saksenaea* species in necrotic cutaneous infections unresponsive to standard therapy. Timely surgical intervention, multidisciplinary management, and tailored antifungal therapy are critical for optimal outcomes, particularly in patients with comorbidities and limited healthcare access.

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