Soft tissue infection of the scalp complicated by mucormycosis in an immunocompromised patient: a case report

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Mucormycosis is a rare but life-threatening angioinvasive fungal infection classically presenting in patients with uncontrolled diabetes and ketoacidosis, which results in rapidly progressive soft tissue infection and necrosis. Mucormycosis is commonly associated with rhino-orbital-cerebral infections; however, primary cutaneous mucormycosis, which involves the skin, also occurs. Diagnosis of the infection implies identifying the organism with histopathology and culture confirmation. Treatment consists of radical surgical debridement and antifungal therapy. The disease prognosis remains poor despite these interventions. This case aims to highlight this rare condition and discuss some aspects of managing mucormycosis and its outcomes. The authors present a case of an adult female with uncontrolled diabetes and advanced human immunodeficiency virus (HIV) admitted to intensive care in diabetic ketoacidosis (DKA) after surgical debridement of a necrotising scalp infection. Her condition was further complicated by mucormycosis in the wound area. Despite interventions, the necrosis spread rapidly, and care was ultimately withdrawn.

Keywords: soft tissue infection, mucormycosis, immunocompromised patient

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Introduction

Mucormycosis is a rare but life-threatening angioinvasive fungal infection. It is opportunistic in nature, mainly affecting immunocompromised individuals, such as those with uncontrolled diabetes or ketoacidosis.[1] It has various clinical syndromes, with rhino-orbital-cerebral (affecting the sinuses, eyes, and brain) infection being the most common.[3] However, primary cutaneous mucormycosis is also described, typically as a complication of trauma or wounds.[1]

Diagnosis of mucormycosis requires a high index of suspicion, guided by clinical presentation consistent with the affected system, followed by identifying the organism through histopathology, with culture confirmation to verify the findings.[4] Treatment involves prompt, aggressive surgical debridement followed by intravenous antifungal therapy.[5]

The authors present a case involving an adult female with uncontrolled diabetes and advanced human immunodeficiency virus (HIV). She was admitted to intensive care in diabetic ketoacidosis (DKA) following surgical debridement for a necrotising soft tissue infection of the scalp. Her condition was further complicated by wound mucormycosis. Despite interventions, necrosis spread rapidly, and care was ultimately withdrawn.

This case aims to highlight the significance of mucormycosis, a rare but severe fungal infection, and discuss the critical aspects of its management and associated outcomes. Understanding this condition is vital for early recognition and intervention, which can significantly impact patient prognosis.

Case report

A 40-year-old female with known uncontrolled type 2 diabetes mellitus (HbA1C 17.3% on admission) and advanced HIV (CD4 191 on admission) was brought to the emergency centre (EC) with confusion and a worsening scalp infection. The patient was admitted a week prior with a scalp abscess, which was booked for formal debridement in the theatre. Computed tomography (CT) showed a large subgaleal abscess with surrounding cellulitis (Figure 1). However, she absconded before the formal debridement.

On re-presentation eight days later, the patient was clinically dehydrated, tachypnoeic, tachycardic, with an altered mental state. Examination of the scalp revealed diffuse scalp swelling, characterised by necrotic, ulcerative skin changes and foul-smelling drainage (Figure 2), raising a high suspicion of a necrotising soft tissue infection. Laboratory investigations indicated that she was in DKA, with incalculably high blood glucose. Formal blood tests showed a white cell count (WCC) of 27×10^9/L and C-reactive protein (CRP) of 705 mg/L, indicating an acute infective state. Her previous surgical specimen had grown *Klebsiella pneumoniae*, for which she was started on a culture-directed antibiotic with a carbapenem (ertapenem).

An appropriate DKA protocol was initiated with antibiotic therapy in the EC, and the patient was taken to the theatre. Intraoperatively, findings were in keeping with necrotising soft tissue infection. In line with this diagnosis, the scalp was thoroughly debrided of all non-viable tissue, continuing until healthy, bleeding edges were reached (Figure 3). Tissue samples were sent for histology, microscopy, culture and sensitivity

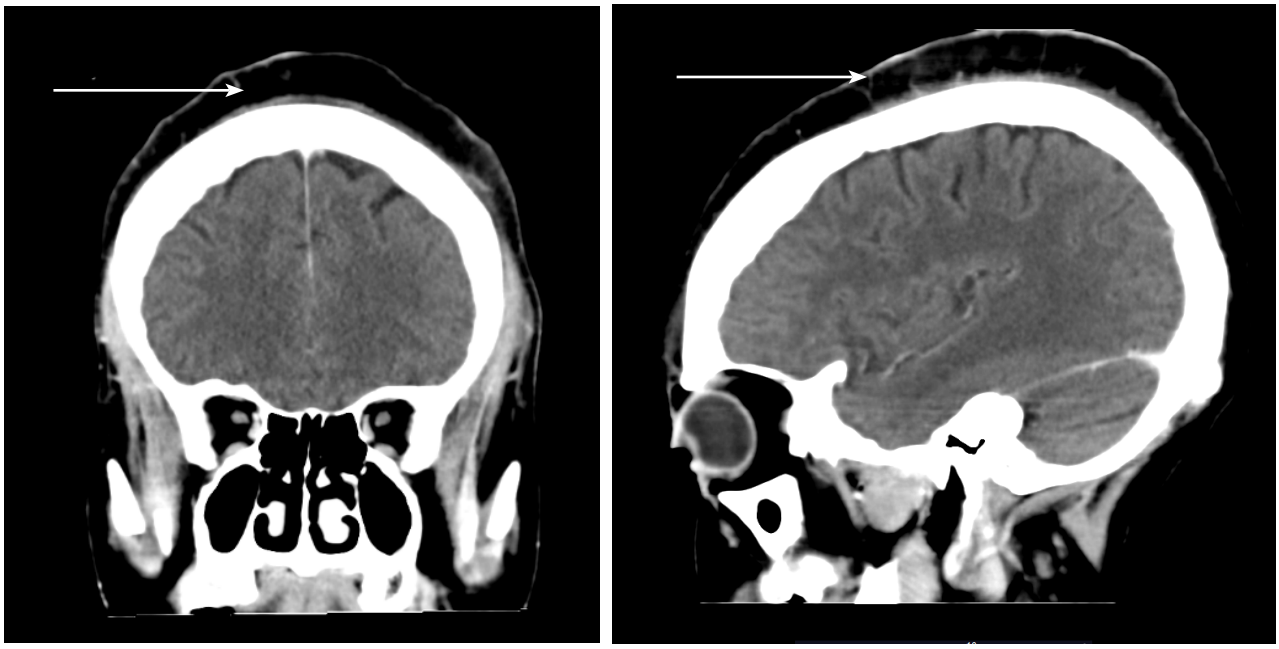

Figure 1: CT image of the scalp demonstrating a large subgaleal abscess with surrounding cellulitis

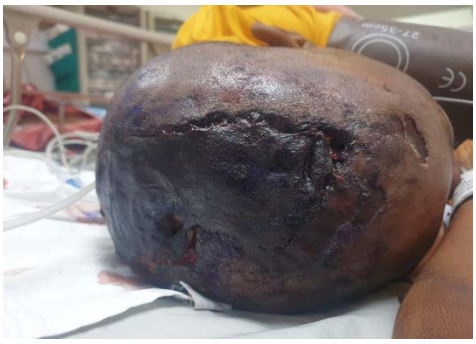

Figure 2: Initial wound of the scalp on re-presentation with diffuse scalp swelling, necrotic and ulcerative skin changes, and foul-smelling drainage, suspicious for necrotising fasciitis

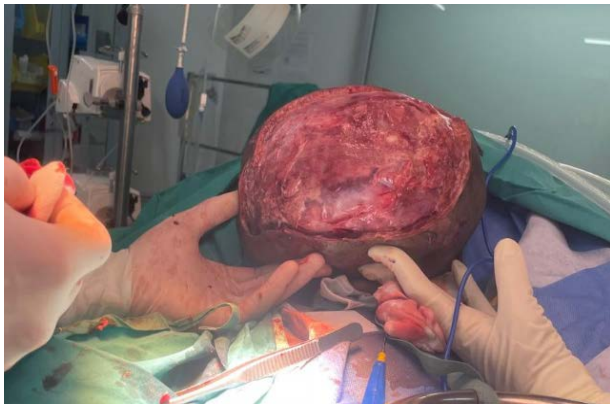

Figure 3: Post-debridement of necrotising fasciitis, with healthy bleeding edges (day 0)

(MC&S). The histology eventually confirmed necrotising fasciitis, while *Klebsiella pneumoniae* was re-cultured from the specimens.

The wound was dressed with chlorhexidine 0.5% tulle (Bactigras®, Smith & Nephew, UK) and a topical antiseptic (Acriflavine®, Kyron Laboratories, South Africa), and the patient was taken to the intensive care unit (ICU) for ventilatory and hemodynamic support. In the ICU, meropenem was initiated instead of ertapenem, and clindamycin was added to inhibit toxin production. Additionally, a sputum sample tested positive for rifampicin-sensitive tuberculosis using GeneXpert. Subsequently, during the patient's stay, her DKA resolved, and her inotropic support was stopped. Her wound was reviewed and dressed with Bactrigras® daily.

Two days after debridement, the wound appeared dry with clean edges, showing no signs of pus or slough, indicating that the disease had not progressed. By this stage, her Glasgow Coma Scale (GCS) score improved to approximately 5T/10T off sedation, indicating a partial recovery of consciousness. However, she still required ongoing ventilatory support due to persistent respiratory compromise.

On day four post-surgery, the anterior edges of the wound exhibited a black, necrotic appearance (Figure 4). In response, the surgeons prescribed daily dressings with collagenase ointment (Iruxol® Mono, Smith & Nephew, UK) for chemical debridement and instructed monitoring for any further spread. By day seven post-debridement, the necrotic tissue extended progressively, reaching the orbital rim bilaterally (Figure 5). At this stage, primary cutaneous mucormycosis of the scalp was suspected. A repeat tissue biopsy was performed for urgent histological analysis, and antifungal therapy with amphotericin B was initiated. The patient's condition subsequently deteriorated, requiring the re-initiation of inotropic support.

The patient's GCS score and overall condition continued to deteriorate, with worsening organ dysfunction despite ongoing treatment. Intracranial spread of the mucormycosis was suspected as her GCS score dropped to 2T/10T, accompanied by the loss of brainstem reflexes. A multidisciplinary team decided against further surgical intervention and opted for palliative care. The patient succumbed to her illness on day eight post-debridement. Histological analysis later confirmed the presence of mucormycosis (Figure 6).

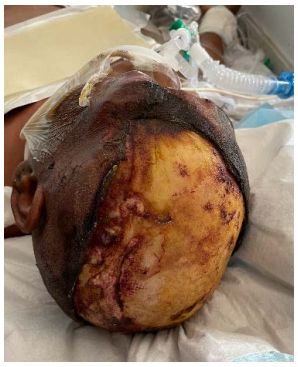

Figure 4: Necrotic edge started to develop on the anterior aspect of the wound (day four post-debridement)

Figure 5: Mucormycosis clinically suspected with necrosis extending to the bilateral orbital rim (day seven post-debridement)

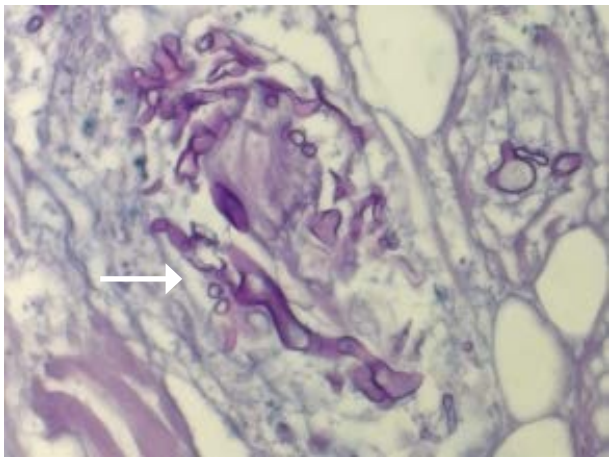

Figure 6: Histological sample from skin punch biopsy

The histology report described an extensive necrotising skin and soft tissue infection. A large amount of mucor fungal elements (broad branching hyphae) were present in the necrotic skin, subcutaneous soft tissue, and blood vessel walls, as well as perineurally.

Discussion

Mucormycosis is caused by a fungus of the *Zygomycetes* class in the order *Mucorales*.[1] This fungus is found widely in the environment and rarely affects immunocompetent individuals.[1] Invasive mucormycosis usually affects immunocompromised individuals, predisposing them to infection, most commonly patients with uncontrolled diabetes, HIV, glucocorticoid treatment, and trauma.[1] The mortality rate associated with the invasive form of mucormycosis is reported to be as high as 96% in cases of disseminated disease.[2]

The fungus causes extensive angioinvasion, leading to vessel thrombosis, followed by tissue infarction and necrosis.[2] Mucormycosis rapidly progresses to invade adjacent structures and underlying tissues.[2] The infection manifests in several clinical forms, with signs and symptoms varying based on the affected organ system.[3]

Diagnosis of mucormycosis is confirmed through histopathology and culture; however, these methods often yield negative results even in cases of disseminated disease.[4] Histopathology reveals a characteristic appearance of *Mucorales*, with broad, irregularly branched, and non-septate hyphae.[4] Given the rapid progression of this disease, clinicians should prioritise a clinical diagnosis to avoid delaying treatment while awaiting culture results. Early intervention is critical for improving patient outcomes.

In this case, the patient had a cutaneous form of mucormycosis following an initial soft tissue infection. Uncomplicated skin and soft tissue infections are typically diagnosed clinically. Purulent infections necessitate incision and drainage, while non-purulent infections require empiric antibiotic therapy.[4,5]

The mainstay of treatment for mucormycosis includes early diagnosis and urgent and aggressive surgical debridement as soon as the infection is suspected to limit its spread.[7] Serial debridement may be necessary if necrosis continues to advance.[7] Prompt intravenous antifungal therapy and optimisation of underlying conditions are also crucial. In the case reported, the patient absconded after the initial diagnosis of an uncomplicated abscess, which probably contributed to the progression of the disease in an immunocompromised patient.

The first-line treatment for mucormycosis is amphotericin B, with the lipid formulation being preferred due to its reduced nephrotoxicity. Initial dosing typically involves a once-daily intravenous dose of 5 mg/kg, which may be increased to 10 mg/kg if necessary.[6] Treatment with amphotericin B should continue until the patient shows significant clinical improvement, after which step-down therapy can be considered. Posaconazole is the drug of choice for second-line or step-down therapy.[6]

This case study highlights the critical importance of early diagnosis and intervention in managing mucormycosis. The patient developed necrotic tissue by day four post-debridement, yet the diagnosis of mucormycosis was only suspected by day seven after the infection had already spread significantly. Unfortunately, the patient succumbed to the disease the following day.

Upon thorough evaluation of her disseminated disease in the context of multiple comorbidities, the decision was made to transition to palliative care. This underscores the necessity for clinicians to discuss the prognosis and make informed decisions about palliation before resorting to futile interventions.

Conclusion

The clinical case illustrates that delays in diagnosis can lead to fatal outcomes and emphasises that early recognition and prompt intervention are crucial in managing suspected mucormycosis infections. Frequent clinical reviews and maintaining a high index of suspicion for this infection are essential components of effective management and can ultimately be lifesaving.

Conflict of interest
The authors declare no conflict of interest.

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Ethical approval
Ethical approval was obtained from the Robert Mangaliso Sobukwe Hospital Department of Health from the institution's medical director. Informed patient permission for the case report has been fulfilled.

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