



Acute cutaneous zygomycosis of the scalp: A case report and literature review



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Summary Cutaneous zygomycosis is the third most common form of zygomycosis. However, scalp involvement is rare for this disease. In this study, we present a case of acute zygomycosis in a diabetic patient who was effectively treated with local debridement, amphotericin B lipid complex and posaconazole.

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Introduction

The incidence of invasive zygomycosis has been increasing over the past two decades [1–4]; this is most likely secondary to the increasing prevalence of diabetes mellitus and high risk immunocompromised patients [4]. Cutaneous zygomycosis is the third most common form of zygomycosis (19%) [5], and it can be superficial, locally invasive or disseminated primarily from the skin to other non-contiguous organs or, rarely, from other organs to the skin [5,6]. Upper and lower extremities are common sites of involvement, although any area of the skin can be affected by zygomycosis [4]. However, scalp involvement is rare [4,7–10].

In this study, we present a case of scalp mucormycosis linked to use of a local herbal treatment.

Case report

A 76-year-old Saudi male with a history of poorly controlled diabetes, hypertension and blindness presented with a 5-day history of a black scalp wound associated with localized pain and pruritus. He had previously applied an herbal medication to a small trauma-induced laceration on the scalp caused by a falling object at home. He had no history of fever, headache or change in level of consciousness and no new neurological symptoms.

On examination, he was afebrile with stable vital signs. Multiple erythematous indurated

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Figure 1 Day 0 at presentation.

plaques with overlying pustules were present on the scalp. Several well-demarcated ulcers with overlying black eschars, with the largest measuring 6 cm × 4 cm and the smallest measuring 0.5 cm × 0.5 cm, were also presented (Fig. 1). The initial wound culture grew both gram-positive and gram-negative bacteria. His blood culture was negative for bacteria.

His white cell count was 17.7×10^9 per liter (L), with 79% neutrophils, 17 g/dL hemoglobin and 345×10^9 /L platelets. BUN was 5.6 mmol/L, creatinine was 61 μ mol/L, CO₂ was 19 mmol/L and RBS was 16.8 mmol/L. The results of his liver function tests were normal. His last HbA1c was 13%.

His initial antibiotic treatment was 1 g IV cefepime Q12H. The AFB smear was negative. He did not improve clinically after 5 days (see Fig. 2). He had increased erythema and induration over his scalp wound but, otherwise, remained afebrile. A head CT did not show any signs of bone invasion or intracranial extension. Tissue culture and histopathology results confirmed a diagnosis of mucormycosis (*Rhizopus* species) (Figs. 2 and 3). He was treated with surgical debridement and 5 mg/kg IV lipid complex amphotericin B lipid complex daily for 1 month, followed by posaconazole for 3 months. The patient's lesions completely resolved.

Discussion

Zygomycosis was first described as a cause of human disease in 1885, and the first cutaneous case

was reported in 1929 [10]. Cutaneous zygomycosis is the third most common form of zygomycosis (11–19%) following rhinocerebral (39%) and pulmonary disease (24%) [4–6]. Unlike other presentations of mucormycosis, 40–50% of patients with cutaneous zygomycosis are immunocompetent [4,5,11]. Trauma is the most common predisposing factor for cutaneous zygomycosis, especially when associated with soiling.

Risk factors for cutaneous zygomycosis include uncontrolled diabetes, diabetic ketoacidosis, burns, chronic renal failure solid organ transplants, hematological malignancies, neutropenia, steroid use, prolonged use of voriconazole and deferoxamine and low birth weight [4,7,11–14].

Cutaneous zygomycosis can present as a localized, deep or disseminated disease [4]. Almost half of patients with cutaneous zygomycosis have their disease confined to cutaneous and subcutaneous tissues, while the disease may extend to deeper tissues, such as bones, tendons and muscles in 24% of cases. Disseminated disease is defined as hematogenous spread of zygomycetes from the skin to other noncontiguous organs (20%) or from other organs to the skin (3%) [4,5]. Fungal elements are isolated from multiple noncontiguous sites [5]. Blood cultures are rarely positive.

Direct inoculation of the skin after penetrating trauma, surgery, burns, motor vehicle accidents, falls and insect bites is the usual port of entry for this disease [6]. Contact with soils or vegetation containing zygomycetes increases the chance of acquiring the disease [15]. In our patient, it was not possible to determine if the primary trauma to the scalp or a secondary infection caused by the use of a contaminated herbal treatment caused the patient's disease. Contamination of such herbal products has been previously documented [16,17].

Cutaneous zygomycosis can also be hospital-acquired [18]. Risk factors in the hospital include contaminated venous access, types of adhesives used, occlusive dressings, burn wounds and post-operative wounds. Sites of cutaneous zygomycosis formation described in the literature include intramuscular injection, insulin injection and catheter insertion sites [19–22].

The onset of cutaneous zygomycosis can be gradual and slowly progressing or aggressive and fulminant [4,23], and its clinical presentation is determined by the immunity of the host, the virulence of the fungi and the timing of diagnosis and intervention. The skin lesion appears red and indurated and then progresses to a black eschar and large ulcers. It can progress to necrotizing fasciitis and extend to deeper tissues if left untreated [24,25]. In cases of disseminated

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