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Case Report

FATAL CELLULITIS CAUSED BY *APOPHYSOMYCES ELEGANS*

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Abstract

A case of cellulitis of the left lateral side of the face caused by the zygomycete *Apophysomyces elegans* in a healthy male following a road traffic accident is reported. The contaminated soil was the source of fungus. Broad aseptate fungal hyphae were seen in the necrosed tissues. Extensive tissue debridement and treatment with amphotericin B were not successful in controlling the rapid invasion of the tissues by the fungus. Patient developed angioinvasion, severe cellulitis and finally succumbed to the infection three weeks after admission.

Key words: *Apophysomyces elegans, cellulitis, zygomycetes*

Apophysomyces elegans is classified within the family mucorales of the class Zygomycetes.[1] It is a filamentous fungus occurring in soil and decaying vegetation as a common environmental contaminant. It is commonly found in tropical and subtropical regions.[2] The species was first isolated in 1979 from soil samples collected in a mango orchard in northern India.[3] The first human infection caused by this fungus was described in 1985 and since then quite a few cases have been reported in English literature. Most of the reported cases have come from regions with warm climates, such as the southern portions of the United States, Mexico, North Australia, and South India. We report a case for the rarity of the clinical picture.

Case Report

A 49-year-old male was brought to the emergency room with swelling and redness of the left side of the face extending to the left ear and neck, with signs of perichondritis and facial nerve palsy. He had deep cut injury over the left parietal area, laceration of the scalp, and left external ear due to a road traffic accident six days prior to this. The laceration had been sutured in another hospital.

There was no history of loss of consciousness, vomiting, or altered sensorium. Physical examination revealed the patient to be conscious, well oriented, afebrile, and normotensive. The patient also had bilateral acromioclavicular joint dislocation.

Cardiovascular system and respiratory system examination were normal. The patient was a known diabetic on irregular medication.

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Routine laboratory parameters at the time of admission were as follows: hemoglobin, 97 g/L; total count, 31.9 × 10^9/L; random blood sugar, 25.9 mmol/L; and urine positive for ketone bodies and sugar. Other parameters were within normal limits. Chest X-ray was normal. A diagnosis of diabetic ketoacidosis with cellulitis was arrived at.

Radical debridement of the left parietal, facial and neck area was done.

Tissue material was sent for mycological, bacteriological and histopathological examination. The tissue was minced and subjected to potassium hydroxide (KOH) examination. The KOH mount showed aseptate fungal elements (Fig. 1). After 72 hours of incubation, fungal culture showed white cottony growth with abundant aerial hyphae with no reverse pigment on Sabouraud dextrose agar (Hi-Media).

Lactophenol cotton-blue mount showed numerous sparsely septate ribbon like fungal hyphae with few rhizoids suggestive of zygomycetes but with no sporulation.

Sporulation was induced by water culture and incubated at 37°C. After seven days, microscopy showed sporangiophores having funnel-shaped apophyses and pyriform sporangia characteristic of *A. elegans* (Fig. 2).

Histological examination showed broad, sparsely septate, thin-walled hyphae with extensive areas of necrosis, cellulitis, and hemorrhage. No pyogenic organisms were isolated either from the wound or from the blood.

The patient was started on amphotericin B, following which he developed hypersensitive reaction to this drug and injection hydrocortisone had to be given.

There was an aggressive growth of the fungus grossly visible on the periphery of the debrided area. By the twentieth day, the patient's condition deteriorated and he developed signs of angioinvasion. CT scan of the brain showed right cerebral artery territory infarct and liver function tests showed

Figure 1: KOH mount of the tissue showing broad aseptate hyphae (×200)

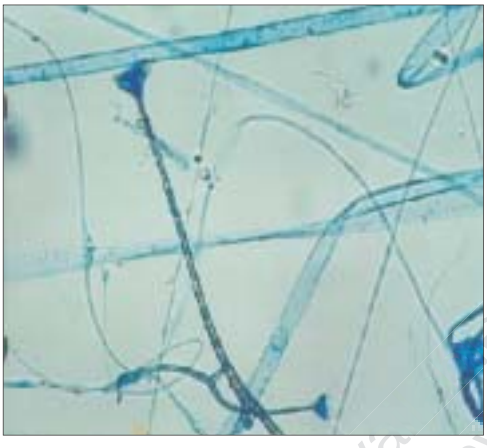

Figure 2: Lactophenol cotton-blue mount of *Apophysomyces elegans* showing sporangiophores having funnel-shaped apophyses and pyriform sporangia (×200)

hyperbilirubinemia. There was an increased total count with polymorphs as 90% and ESR was raised. The patient finally succumbed to the fungal infection.

Discussion

The zygomycoses are infections caused by fungi of the class *Zygomycetes,* comprising of the orders Mucorales and Entomophthorales. The Entomophthorales are rare causes of subcutaneous and mucocutaneous infections known as entomophthoromycosis, which largely affect immunocompetent hosts.

Fungi belonging to the order Mucorales are distributed into six families, all of which can cause cutaneous and deep infections. Species belonging to the family *Mucoraceae* are isolated more frequently from patients with mucormycosis than any other family. Among the *Mucoraceae, Rhizopus oryzae (rhizopus arrhizus)* is by far the most common cause of infection.[4]

Other less frequently isolated species of the *Mucoraceae* family that cause a similar spectrum of infections includes *Rhizopus microsporus* var *rhizopodiformis*, *Absidia corymbifera*, *A. elegans*, *Mucor* spp., and *Rhizomucor pusillus*.[4]

A. elegans is a rare cause of human Zygomycosis[4,5] and is usually acquired via traumatic implantations, insect bites, surgery and contamination of burn wounds. Invasive soft tissue infections develop on burns or wounds contaminated by soil.

A. elegans infections present most commonly as necrotizing fasciitis, osteomyelitis and angioinvasion.[5-7] Systemic and secondary renal and bladder infections[8,9] have also been reported. *A. elegans* tends to invade and grow within the vascular lumen like other species of the zygomycetes. Vascular invasion frequently causes thrombosis leading to ischaemic tissue necrosis.[10] Histopathological examination of most infected tissue with *A. elegans* has revealed hyphae in the thrombi. The rapid progress of necrosis, which usually occurs within days after inoculation, may be explained by the rapid growth within the vessels.

A hallmark of mucormycosis infections is the virtually uniform presence of extensive angioinvasion. This angioinvasion is associated with the ability of the organism to hematogenously disseminate from the original site of infection to other target organs. Hence, damage of and penetration through endothelial lining of blood vessels is likely to be a critical step in the pathogenesis of this organism. There was a delay in the antifungal treatment as the patient was brought to the hospital after cellulitis had spread extensively.

In vitro susceptibility data reported so far are very limited. Minimum inhibitory concentration (MIC) breakpoints for interpretation of *in vitro* susceptibility results have not been defined. Some of the isolates may yield relatively low amphotericin B MICs (in micrograms per milliliter). Amphotericin B appears as the sole antifungal drug, which may be active against *A. elegans* similar to the other members of the class Zygomycetes. Extensive surgical debridement and amphotericin B therapy may be efficacious *in vivo*.[9]

This case highlights the importance of fungal agents like *A. elegans* that can cause infection in previously healthy patients who suffer an injury to the cutaneous barrier. Infection with this Zygomycete should be considered when there is progressive necrosis of a wound in a previously healthy individual especially when there is an ineffective response to antibacterial chemotherapy.

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