

## Case Report

# Gangrenous Cutaneous Mucormycosis of the Anterior Chest Wall

Abdullateef A Dawood, Nayef S Al-Ahmad, Atef M Hussainy  
Department of Surgery, Al-Sabah Hospital, Kuwait

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**ABSTRACT**

Mucormycosis is a rare but serious fungal infection. We report a case of progressive gangrenous cutaneous mucormycosis of the anterior chest wall.

KEY-WORDS: fungal infection, zycomycosis

**INTRODUCTION**

Mucormycosis is the general name of infections caused by fungi of the family *Mucoraceae*, order *Mucorales*, class *Zygomycetes*. Infections by *Zygomycetes* are globally referred to as zygomycosis or phycomycosis. The *Mucoraceae* family, responsible specifically for mucormycosis, includes the genera *Rhizopus*, *Mucor*, *Rhizomucor*, *Absidia*, *Cunninghamella* and *Saksena*. The most common clinical syndromes associated with mucormycosis are rhino-cerebral and pulmonary infections, although gastrointestinal, disseminated and other forms of infections are occasionally encountered<sup>[1]</sup>. While cutaneous mucormycosis in association with local trauma has been classically described<sup>[2-4]</sup>, its frequency has been increasing in recent years mostly in immunocompromised patients<sup>[5]</sup>. Mucormycosis is rarely seen in Kuwait, and its incidence is unknown. It is not on the list of infectious diseases notifiable by the Ministry of Health.

**CASE REPORT**

A 60-year old Kuwaiti diabetic male patient was admitted as an emergency case with a ten-day history of a progressive anterior chest wall inflammatory lesion. When the lesion first appeared, the patient treated it with traditional medicine, using alum ("shaba" in Arabic), a topical astringent composed mostly of aluminum potassium sulfate, with a styptic effect (provoking the contraction of tissues and/or blood vessels). The lesion flared up following the application of the alum and subsequent medical treatment by unspecified antibiotics had no effects. The patient is retired and has no medical problems except for diabetes mellitus and no history of contact with contaminated blood. The patient lives in average Kuwaiti accommodations.

Upon examination, the patient's general status was satisfactory and unremarkable except for a temperature of 38.1 °C. Local examination showed an extensive cellulitis of most of the anterior chest wall with central patches of gangrene, marked tenderness, and possible underlying collection. The initial investigations showed marked leucocytosis, Hb 128 gm/l, platelets 152,000/l. Chest X-rays were normal. Parenteral antibiotics were started with Ceftriaxone 2 gm IV/12 hourly and Metronidazole 500mg IV/8 hourly. Surgical incision of the lesion was done draining a small amount of pus that was sent for bacteriology. Initial examinations reported no bacterial growth.

Forty-eight hours later, the gangrenous area had become more defined and well demarcated, surrounded by an area of redness and edema, and reaching to the root of the neck, both nipples, and the lower chest (Fig. 1). Leucocytosis kept rising. A wide surgical excision was performed on that day and all dead tissues were sent for histopathology. Histopathology reported extensive coagulative necrosis with focal viable areas, neutrophilic abscess in the sub-cutaneous fat with a patchy chronic inflammation consisting of lymphocytes, plasma cells and histiocytes. Scattered throughout the necrotic area were hyphae of infiltrating mucormycosis (Fig. 2). This report confirmed a diagnosis of "necrotic mucormycosis". Systemic and local amphotericin B were started in combination with imipenem/cilastatin sodium 500 mg six hourly I.V.

Despite the start of an anti-mycotic treatment, the patient's status kept deteriorating. In the following 48 hours, the skin edges and part of the pectoralis muscle had become gangrenous, and a new subcutaneous lesion had appeared in the right

Address correspondence to:

Dr. A. Dawood, P.O. Box: 4073, Mishrif - 40188, Kuwait. Tel: (965) 539-3540, Fax: (965) 539-3541, E-mail: Bufahad86@hotmail.com



Fig. 1: Gangrenous anterior chest wall

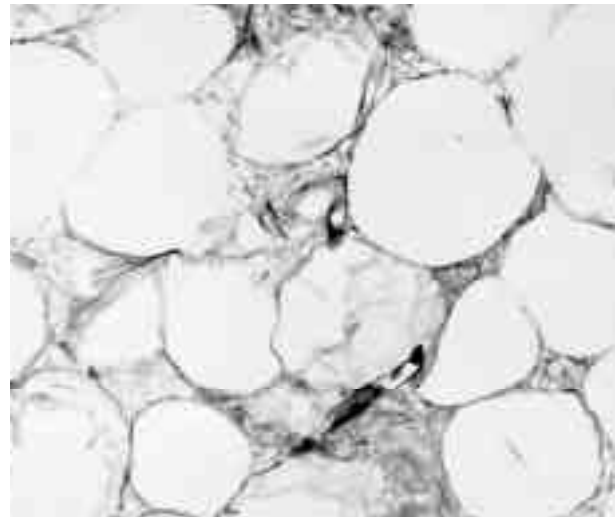


Fig. 2: Aseptate fungal hyphae of mucor with right angle branching (Grocott stain)



Fig. 3: New lesion at right hypochondrium

hypochondrium (Fig. 3). Surgical excision was performed again for both chest and abdominal lesions. The patient, fully sedated, was moved to the intensive care ward and remained on a ventilator with muscle relaxants. Daily local debridement and local amphotericin B soakings were performed. Notwithstanding all this care, a new lesion appeared on the left hypochondrium region. The patient developed acute renal failure prompting a change in therapy from amphotericin B to liposomal amphotericin B, a form known to be less nephrotoxic<sup>[5]</sup>. The patient ultimately developed multi-organ failure 22 days after admission and died.

## DISCUSSION

The fungi responsible for mucormycosis are found throughout nature. Humans have a natural resistance to those fungi despite their ubiquitous presence in nature. The fungi isolated from this patient grew at a temperature of 30-37 °C and

was eventually identified as *Absidia corymbifera*. The microscopic examination revealed a coenocytic mycelium with sporocystophores intensively branched in grapes or corymbs, and piriform sporocysts with ovoid columellae showing large, conical apophyses.

Mucormycosis has been described extensively in patients with diabetes, hematological malignancies, HIV and organ transplants<sup>[5-7]</sup>. In trauma patients, the initial soil contamination of the wound leads to fungal proliferation and invasion of subcutaneous tissues, muscles and fascias. Then, because of their peculiar affinity for blood vessels, mucorellae invade vascular structures and cause tissue necrosis.

Rapid diagnosis of cutaneous mucormycosis is best obtained by biopsy of the tissue and microscopic examination. There are two forms of cutaneous mucormycotic infections: superficial and gangrenous. Both appear to require violation of the skin for the induction of the disease process<sup>[8]</sup>. This patient's use of alum to "burn" his initial boil created an area of necrotic tissues where spores of the fungus proliferated and spread to subcutaneous vessels already compromised by diabetes, leading to a chain of events that could not be controlled.

Superficial infections have a gradual onset and slowly progressing symptoms, thus the deep tissues or vascular invasion is not present on histological examination, and systemic complaints such as pain and fever may be lacking. Often the skin lesions appear as pustules with rare ulceration or eschar formation<sup>[9]</sup>. These superficial infections are usually indolent, and local debridement without parenteral antifungal therapy is sufficient treatment, with simultaneous management of the patient's underlying medical condition.

Gangrenous mucormycosis is a disease of rapid onset and clinical manifestations. Erythematous macules usually occur within 48-72 hrs after the initial

insulting event. These maculae quickly coalesce forming ulcers and eventually eschars. The ulcers are painful and continue to expand, causing induration and erythema in underlying cutaneous structures<sup>[10]</sup>. The disease is self-propagating, the vascular invasion and subsequent tissue necrosis allow for acidic tissue conditions conducive to the organism's growth. The fulminating course of the gangrenous form requires urgent action.

The successful treatment of serious mycoses such as mucormycosis infections depends on the following steps:

1. Lesions should be recognized early and suspected as mucormycosis infection.
2. Diagnosis should be rapidly done based on a tissue biopsy to differentiate mucormycosis infection from other *Aspergillus* infections. The culture of exudates or discharges alone is not reliable for diagnosis.
3. Immunosuppressive drugs should be discontinued if they had been started earlier.
4. Parenteral antifungal therapy must be started as soon as possible.
5. Surgical debridement should be aggressively performed.

Aggressive surgical therapy remains the cornerstone of treatment and a negative surgical margin appears necessary to ensure the likelihood of success in the treatment of these patients. Systemic antimycotic therapy is indicated in all cases of gangrenous cutaneous mucormycosis. The length of this therapy is controversial and often determined by the patient's clinical picture and tolerance to the side effects of these antifungal agents. The wet-to-dry dressings of amphotericin B impregnated bandages are an empiric, time-honored therapy, which may aid in the management.

In conclusion, primary cutaneous mucormycosis is a rare clinical entity, which can be fatal. This case illustrates some of the difficulties related to the

diagnosis and treatment of mucormycosis. The diagnosis is often made quite late, especially in areas where fungal infections are relatively rare. Once the infection spreads, it is often impossible to control it even with massive doses of anti-fungal agents. Aggressive surgical debridement is the cornerstone of treatment, though it failed to save the life of this particular patient.

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