

## Zygomycotic Necrotizing Fasciitis Caused by *Apophysomyces elegans*

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**A case of necrotizing fasciitis of the anterior abdominal wall caused by the zygomycete *Apophysomyces elegans* in a healthy male following inguinal herniorrhaphy is reported. The portal of entry of the fungus into the incised skin and subcutaneous tissues was probably through either contaminated surgical sutures or postoperative surgical dressings. Broad, aseptate fungal hyphae were seen in the necrosed tissues with an associated necrotizing vasculitis. Extensive tissue debridements and a low dose of amphotericin B were not successful in controlling the rapid invasion of the tissues by the fungus.**

*Apophysomyces elegans*, a member of the order *Mucorales* of the class *Zygomycetes*, is an infrequent causative agent of zygomycosis (8). The other genera of this class are well known in the etiology of zygomycosis in a compromised host (1). We present here a case of zygomycotic necrotizing fasciitis that developed postoperatively in a healthy young male.

**Case report.** A 27-year-old man presented on 13 June 1992 with a history of left inguinal herniorrhaphy performed at a private nursing home in Nalgonda in Andhra Pradesh, India. On postoperative day 9 he had developed edema and a collection of fluid at the site of the operation. The wound was reopened, and serosanguinous fluid, along with a few blood clots, was evacuated. The patient was referred to our institute on postoperative day 17 with signs and symptoms of septicemia and a spreading cellulitis of the lower abdominal wall. At the time of admission to our institute, he was noted to be dehydrated, with a pulse rate of 102/min. He had an anxious look and was restless. Edema and swelling of the lower abdomen and the left inguinal region extending to the scrotum were noticed. The left inguinal canal was found to be open and filled with necrotic material. With a clinical diagnosis of postoperative necrotizing fasciitis of the left inguinoscrotal region, an emergency debridement was carried out. Broad-spectrum antibiotic coverage consisting of cefotaxime, gentamicin, and metronidazole, active against both aerobic and anaerobic bacteria, was started. The excised tissue was submitted for histopathologic and microbiologic evaluation. Direct (saline and 20% KOH mounts) and lacto phenol cotton blue mounts of the necrotic material showed broad, aseptate, branching fungal elements resembling those of a zygomycete, with no evidence of any suppuration or bacterial infection. The material was inoculated onto routine bacteriological media and Sabouraud dextrose agar (SDA) with and without chloramphenicol and cycloheximide and incubated at both 25 and 37°C. All of the bacteriological cultures were sterile after 48 h, except for a profuse fungal growth on blood agar. On all of the SDA plates, there was a profusely growing, white, fluffy fungus with abundant aerial mycelia after 48 h of incubation. There was no evidence of sporulation. The correlation of the

fungus with the clinical signs and symptoms could not be established at this juncture, since the patient was otherwise a well-preserved young man with no evidence of immunosuppression or any other predisposing conditions such as diabetes mellitus or AIDS (the test for anti-human immunodeficiency virus antibodies was nonreactive).

Histopathology of the tissue, received later, showed many areas of necrosis and inflammation with randomly branched, broad nonseptate hyphae (Fig. 1). Necrotizing vasculitis involving arteries and veins was also seen. Morphological features were reported to be characteristic of zygomycosis.

In view of the histopathology and culture findings described above and considering the poor general condition of the patient, a low dose of amphotericin B (0.5 mg/kg) was added to the treatment regimen on postoperative day 23. Despite repeated and thorough debridements and amphotericin B therapy, the patient's general condition deteriorated

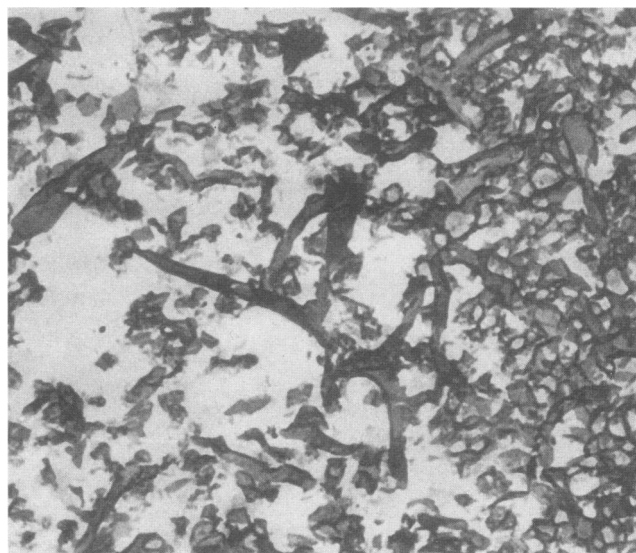


FIG. 1. Micrograph showing broad, nonseptate fungal filaments branching at right angles with a background of few lymphocytes. Stain, silver methenamine; magnification,  $\times 400$ .

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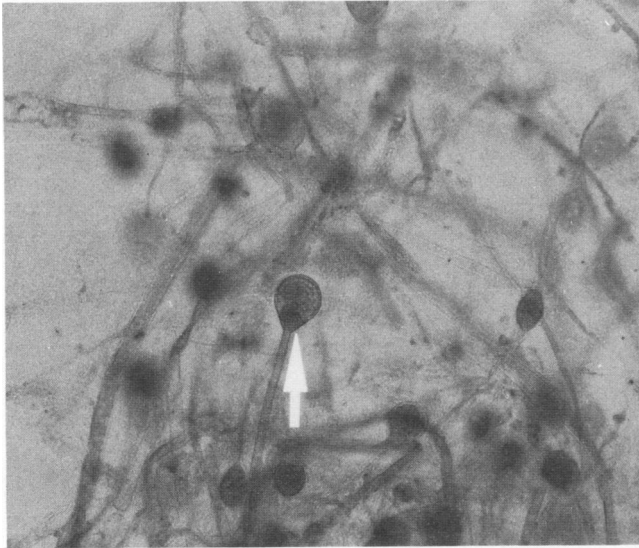


FIG. 2. Micrograph showing the sporangiophores, apophyses (arrow), and the sporangium of *A. elegans*. LPCB, lacto phenol cotton blue; magnification,  $\times 200$ .

further and the necrotic process was found to be spreading rapidly and extensively involving the anterior abdominal muscles, the left testis (which was excised), and the root of the penis deep to the femoral vessels up to the proximal half of the left thigh. The arterial blood gas analysis revealed metabolic acidosis. On postoperative day 28 (23 June) in the early morning hours, the patient developed cardiac and respiratory arrest and could not be revived.

The fungal isolate was sent to J. Vandepitte, Universitaire Ziekunhous, Leuven, Belgium. The fungus was identified as *A. elegans* by C. De Vroey, Head of the Laboratory for Mycology, Prince Leopold Institute of Tropical Medicine, Antwerp, Belgium.

**Mycological findings.** No sporulation was seen on slide cultures even after 7 days of incubation on SDA, corn meal dextrose agar, or potato dextrose agar. Stimulation of sporulation was attempted by the method of Ellis and Ajello (3). Abundant sporulation was seen after 5 days on 1% water agar by this procedure (Fig. 2). The microscopic features of the sporangiophores and the sporangia were characteristic of *A. elegans*, as was described previously (6).

*A. elegans* was first isolated from soil samples from a mango orchard in Northern India by Misra et al. in 1979 (6). They classified it under a new taxon, *Apophysomyces*, on the basis of its distinct and characteristic apophyses. Subsequently, several cases of the fungus as an etiologic agent in human zygomycosis were reported (2, 3, 5, 8, 10). More recently, a case of traumatic zygomycosis caused by *A. elegans* was reported in a healthy male (4). In all of these cases the portal of entry of the fungus, *A. elegans*, was

through traumatized skin. In the present case, the fungus had gained entry into the incised skin and subcutaneous tissue either through contaminated surgical sutures used during surgery or through the surgical wound dressings used postoperatively for the inguinal herniorrhaphy. The present patient was an otherwise healthy individual, with hematological parameters within normal limits (hemoglobin, 11.8 g%; total leukocyte count,  $13,500/\text{mm}^3$ ; differential count: polymorphs, 79%, leukocytes, 28%, eosinophils, 3%), except for the surgical stress that he underwent, as was seen in two cases by other investigators (4, 9).

*A. elegans* has certain morphological features similar to those of *Absidia* species, with regard to the sporangiophores arising typically internodally and not opposite to rhizoids, and to *Aspergillus* species with regard to the presence of hyphal foot cells. But the impressive and characteristic apophyses and the darkening and thickening of the sporangiophore wall below the apophyses distinctly differentiate fungi of the genus *Apophysomyces* from other, related fungi (6). No animal infections have been reported (7).

This case of zygomycotic necrotizing fasciitis is being reported because of the rarity of the etiologic agent and the rapidity with which the fungus led to the deterioration and death of an otherwise healthy young male. Necrotizing fasciitis usually occurs in an immunocompromised host. Hence, when it occurs in an immunocompetent host, the probability of a zygomycotic infection should also be considered and all efforts to culture and identify the etiologic fungus must be made to establish an early diagnosis.

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