Infections Due to Dematiaceous Fungi in Organ Transplant Recipients: Case Report and Review

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Dematiaceous fungi are being increasingly recognized as pathogens in organ transplant recipients. We describe a case of invasive esophagitis due to Cladophialophora bantiana in a small bowel transplant recipient and review a total of 34 cases of infections due to dematiaceous fungi in organ transplant recipients. The median time to the onset of fungal infection after transplantation was 22 months. Clinically, two distinct patterns of infections were observed: 79% of the patients had skin and/or soft-tissue infections or joint infections (predominantly due to Exophiala species), and 21% had systemic invasive infections (predominantly brain abscesses due to Ochroconis gallopavum [Dactyliaria gallopava, Dactyliaria constricta var. gallopava]). The overall mortality rate among the patients with skin and/or soft-tissue infections or joint infections and the patients with systemic invasive disease was 7% and 57%, respectively; two of five patients with brain abscesses were cured with antifungal therapy. Recognition of infections due to dematiaceous fungi is important since these infections, unlike invasive aspergillosis, may be more amenable to therapy.

Dematiaceous fungi (dark-pigmented fungi) are characterized by the presence of melanin or melanin-like pigments in their vegetative cell walls, conidia, or both. These saprophytic fungi are widely distributed in the environment, particularly in soil, wood, and other plant matter [1, 2]. Infections caused by dematiaceous fungi include mycetoma, chromoblastomycosis, and phaeohyphomycosis.

Mycetoma is a tumorous growth of the skin that is notable for the formation of granules in the tissue [1]. Chromoblastomycosis is a superficial or subcutaneous skin infection characterized by the presence of thick-walled muriform cells with intersecting cross-walls (sclerotic bodies) [1]. Phaeohyphomycosis comprises a heterogeneous group of infections ranging from superficial, cutaneous, or subcutaneous infections to disseminated invasive disease caused by yeastlike cells or hyphae of a variety of dematiaceous fungi [1, 2].

Dematiaceous fungi are being increasingly recognized as human pathogens, particularly in immunocompromised hosts (including transplant recipients) [1–5]. However, most cases of dematiaceous fungus infections after organ transplantation have been reported individually. Consequently, the clinical spectrum, response to therapy, and outcome of such infections in transplant recipients are difficult to discern.

As our awareness of the pathogenic potential of these opportunistic fungi increases and as the microbiological techniques for their detection or identification improve, it is likely that these fungi will assume an increasingly important role as pathogens in organ transplant recipients. We describe a case of phaeohyphomycosis in a small bowel transplant recipient and review the literature on infections due to dematiaceous fungi in organ transplant recipients.

Methods

Cases of dematiaceous fungus infections in solid organ transplant recipients that were reported in the literature were identified through a MEDLINE search. Keywords used in the search included phaeohyphomycosis, chromoblastomycosis, Cladosporium, Cladophialophora, Dactyliaria, Phialophora, Exserohilum, Scopulariopsis, Alternaria, Curvularia, Bipolaris, and Exophiala. Reference lists of the identified articles and meeting abstracts were used to find additional cases. Thirty-three cases were identified [3–28]; these cases and the present case are reviewed. A Portuguese report that lacked the description of the case in the English-language abstract was excluded.

Case Report

A 36-year-old man underwent small bowel and right hemicolectomy allograft transplantation in July 1993 for treatment of desmoid tumor of the bowel (Gardner’s syndrome). Late-onset chronic rejection of the graft ensued in July 1995, and total parenteral nutrition was reinstituted. Results of liver function tests that were performed over the following months were consistent with cholestatic hepatitis, and liver biopsy revealed cholestasis considered to be secondary to parenteral hyperalimentation.

In October 1995, upper endoscopy was performed for evaluation of dysphagia and hematemesis; this procedure revealed a 2 × 2-cm deep ulcer in the distal esophagus. Histopathologic
examination of an ulcer specimen demonstrated septic fungal hyphae with invasive disease in the mucosa and submucosa. Culture of an ulcer specimen yielded pure growth of Cladophialophora bantiana (Xylohypha bantiana, Cladosporium trichoides). The identification of the fungus was confirmed by the fungal testing laboratory at the University of Texas Health Science Center at San Antonio. There was no clinical evidence of fungal infection elsewhere, and chest radiographs were unremarkable.

Itraconazole therapy (200 mg orally three times a day) was initiated. In November 1995, the small bowel allotraft was removed because of chronic rejection. Repeated endoscopy revealed the persistent ulcer. Therapy was switched to amphotericin B (1 mg/[kg \cdot d]), which was changed 7 days later to amphotericin B colloidal dispersion (Amphocil, Sequus Pharmaceuticals, Menlo Park, CA; 6 mg/[kg \cdot d]).

Seven weeks after the initial diagnosis, repeated endoscopy showed improvement in his condition but not complete resolution of the ulcer; histopathologic examination no longer demonstrated fungal hyphae. However, the patient died 1 week later of progressive liver failure, acute respiratory distress syndrome, and multiorgan failure. Permission for an autopsy was not granted, although bronchoalveolar lavage fluid obtained before his death was negative for fungi.

Literature Review

Of 34 organ transplant recipients with dematiaceous fungus infections [3–28] (including the patient described here), 50% (17) were renal transplant recipients, 23.5% (8) were liver transplant recipients, 23.5% (8) were heart transplant recipients, and 3% (1) were small bowel transplant recipients. Seventy-nine percent (22) of the 28 patients for whom sex was reported were male. The median age of these patients was 44 years (range, 9–68 years). The median time to the onset of fungal infection after transplantation was 22 months (range, 55 days to 6 years); the onset of fungal infection was assessed from the time of initial transplantation. Six percent (two) of the 34 cases occurred after retransplantation [5–21]. Only 21% of the cases occurred within 6 months of transplantation.

Clinical features. Clinically, two main types of infections due to dematiaceous fungi were observed in organ transplant recipients: of the 34 patients, 76% (26) had skin and/or soft-tissue infections, and 21% (seven) had systemic invasive infections. Three percent (one) of the 34 patients had septic arthritis [19]. Systemic infections occurred in 63% (5) of the 8 liver transplant recipients [1, 5, 21–23], 13% (1) of the 8 heart transplant recipients [4], and none of the 17 renal transplant patients; the other systemic infection occurred in the small bowel transplant recipient (present case).

Skin lesions due to dematiaceous fungi presented as papules, plaques, pustules, nodules, or nonhealing ulcers [3, 6–20, 24–27]; all lesions were proven by biopsy. Skin lesions occurred a median of 2 years (range, 2 months to 6 years) after transplantation. All skin lesions were present on the extremities: 58% on the upper extremities and 42% on the lower extremities. Lesions were usually painless and indolent or subacute in onset (noted a median of 3 months [range, 2 weeks to 9 months] before presentation). Eight percent (two) of the 26 patients with skin and/or soft-tissue lesions due to dematiaceous fungi presented with erythematous lesions on the extremities that mimicked bacterial cellulitis; these lesions were treated initially as bacterial cellulitis [11, 25]. Eight percent (two) of the 26 patients had bursitis (patellar [25] and olecranon [26]), and 4% (one) had synovitis (wrist) in addition to the skin lesions overlying the bursa [13]. In all three of these cases, cultures of aspirates or surgical drainage fluid from the bursa or synovium yielded dematiaceous fungi [13, 25, 26].

Systemic invasive infections due to dematiaceous fungi occurred in 21% (7) of the 34 patients, including 5 patients with brain abscesses [1, 5, 21–23], 1 with pneumonia [4], and 1 with esophagitis (present case). Brain abscesses occurred as early as 44 days (after third transplantation) and as late as 2 years (median, 3 months) after transplantation (table 1). All five cases of brain abscesses occurred in liver transplant recipients.

At the time of presentation, one to three ring-enhancing lesions were demonstrated in these five cases; in four cases, the lesions were located in the frontoparietal lobe, and in one case, they were located in the temporoparietal lobe. Skin lesions preceded the brain abscess in only one of the five cases [21]. One of the five patients with brain abscesses had concomitant pulmonary involvement (lung nodules) [22]. Isolated pulmonary involvement (cavitary pneumonia) occurring 3 months after transplantation was the presenting feature in a heart transplant recipient; skin lesions were not detected in this case [4].

Fungal species. Of 16 cases of skin lesions, 32% (5) were due to Exophiala jeaneselmi: 19% (3), to Alternaria species; 13% (2), to Exophiala spinifera: 6% (1), to Phialophora parasitica: 6% (1), to Fonsecia pedrosoi: 6% (1), to Exophiala castellani (Exophiala mansonii): 6% (1), to Exserohilum rostratum: 6% (1), to Exophiala pisciphila: and 6% (1), to Bipolaris hawaiensis (table 2). In an additional seven cases of skin lesions (five renal and two heart transplant patients), the diagnosis was based on the histopathologic demonstration of dark-pigmented fungi in the lesions; however, the identity of the fungus was not reported.

All three cases of bursitis and/or synovitis were due to Exophiala species (E. jeaneselmi, two; E. castellani, one). The one case of septic arthritis was due to P. parasitica [19]. Of the five cases of brain abscesses, 60% (3) were due to Ochroconis gallopavum (Dactylaria gallopava, Dactylaria constricta var. gallopava); 20% (1), to C. bantiana, and 20% (1), to Scopulariopsis brumptii. Pneumonia in the heart transplant patient was due to D. constricta [4], and esophagitis in the present case was due to C. bantiana.

Immunosuppression and risk factors. Of the 26 patients with dematiaceous fungus infections for whom immunosuppressive regimens were reported, 42% (11) received primary immu-
**Table 1.** Summary of data on cases of systemic invasive infection due to dematiaceous fungi in organ transplant recipients.

<table>
<thead>
<tr>
<th>Reference, type of organ transplant</th>
<th>Retransplantation</th>
<th>Time of onset of fungal infection after transplantation</th>
<th>Fungus</th>
<th>Site(s) of infection</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[5], liver</td>
<td>Yes</td>
<td>44 d*</td>
<td>Cladosporium bantiana</td>
<td>Brain</td>
<td>Amphotericin B</td>
<td>Died</td>
</tr>
<tr>
<td>[21], liver</td>
<td>Yes</td>
<td>3 mo</td>
<td>Scopulariopsis brunnea</td>
<td>Brain, skin</td>
<td>Amphotericin B</td>
<td>Died</td>
</tr>
<tr>
<td>[22], liver</td>
<td>No</td>
<td>21 mo</td>
<td>Ochroconis gallopavum</td>
<td>Brain, lungs</td>
<td>Amphotericin B colloidal dispersion† plus fluconazole</td>
<td>Survived</td>
</tr>
<tr>
<td>[1], liver</td>
<td>No</td>
<td>24 mo</td>
<td>O. gallopavum</td>
<td>Brain</td>
<td>Amphotericin B</td>
<td>Died</td>
</tr>
<tr>
<td>[23], liver</td>
<td>No</td>
<td>55 d</td>
<td>O. gallopavum</td>
<td>Brain</td>
<td>Amphotericin colloidal dispersion plus itraconazole and fluconazole</td>
<td>Survived</td>
</tr>
<tr>
<td>[4], heart</td>
<td>No</td>
<td>3 mo</td>
<td>Dactylaria constricta</td>
<td>Lungs</td>
<td>Amphotericin B</td>
<td>Survived</td>
</tr>
<tr>
<td>[PR], small bowel</td>
<td>No</td>
<td>30 mo</td>
<td>C. bantiana</td>
<td>Esophagus</td>
<td>Itraconazole followed by amphotericin B colloidal dispersion</td>
<td>Died</td>
</tr>
</tbody>
</table>

**NOTE.** PR = present report.
* Infection occurred 44 days after third transplantation and 8 months after initial transplantation.
† Amphocil (Sequus Pharmaceuticals, Menlo Park, CA).

tosuppressive treatment with cyclosporine; 38% (10), azathioprine; 15% (4), tacrolimus; and 4% (1), cyclophosphamide. Thirty-eight percent (10) of the 26 patients had rejection episodes preceding the fungal infection [1, 3–5, 8, 11, 20, 21, 26]. Twenty-three percent (six) of the 26 patients had received OKT3 monoclonal antibodies or antilymphocyte globulin before the onset of fungal infection [3–5, 11, 25]; however, none of the patients had received these agents within 3 months of the fungal infection.

History of environmental exposure to soil or plant matter or trauma with contaminated material (wood, thorn, or splinters) was reported in 48% (13) of 27 cases. Fifty-three percent (seven) of these 13 patients with environmental exposure were avid gardeners, florists, farmers, or landscapers, and their occupations were believed to have placed them at risk for the acquisition of these fungal infections.

**Outcome.** The overall mortality rate among the 34 organ transplant patients with dematiaceous fungus infections was 18% (six); 57% (four) of the seven patients with systemic invasive disease died compared with 7% (two) of the 27 patients with cutaneous and/or soft-tissue infections or joint infections. Death was directly attributable to fungal infection in three of the four patients with systemic invasive infections who died [1, 5, 21], whereas death was unrelated to fungal infection in two of the two patients with cutaneous and soft-tissue infections who died [12, 14].

**Response to therapy in patients with skin and/or soft-tissue infections.** Thirteen of the 26 patients with skin and/or soft-tissue infections underwent surgical excision with or without antifungal therapy. Thirty-eight percent (five) of the 13 patients were cured with surgical excision alone [6, 11, 16–18]. An additional 31% (four) of the 13 patients received antifungal therapy following the excision, which cured their lesions; antifungal therapy included ketoconazole for 1 to 2 years for 2 patients [10, 11], amphotericin B (total dose, 1 g) plus fluconazole for 8 weeks for 1 [26], and amphotericin B (total dose, 450 mg) for 1 [3].

In three of the 13 cases, no improvement was observed with antifungal therapy (amphotericin B, ketoconazole, and dimethyl sulfoxide, 1 patient [27]; ketoconazole, 1 [24]; and fluconazole, 1 [17]) until the excision was done; one of these patients relapsed after the excision, but subsequently the patient's condition improved during a 6-month course of itraconazole therapy [24]. One of the 13 patients continued to have recurrent lesions at a 1-year follow-up visit despite excision and therapy with amphotericin B (for 2 months) followed by ketoconazole [8].

In seven cases, the skin lesions healed with antifungal therapy alone without surgical excision of the lesion. Three of these patients received itraconazole therapy [25]; one patient did not respond to fluconazole therapy, but his lesions cleared rapidly with a 3-month course of itraconazole therapy. This patient relapsed 6 months after itraconazole therapy was discontinued, but he responded again upon the reinitiation of itraconazole therapy. Of the seven patients, 1 was cured with a 6-month course of fluconazole therapy [7]; 1, with a 4-week course of amphotericin B therapy [20]; and 1, with ketoconazole therapy (administered for an unspecified period) [15]. In one case, no improvement was observed with fluconazole...
therapy (50 mg daily for 2 months); this patient was eventually cured with a 6-week course of amphotericin B therapy and a 6-month course of flucytosine therapy [7]. No response was observed with topical nystatin therapy in one case [14].

Response to therapy in patients with systemic invasive infections. Three of the five patients with brain abscesses (due to C. bantiana, S. brumptii, and O. gallopavum) died of their infections [1, 5, 21]. The patient infected with C. bantiana, who had received amphotericin B therapy (dose unspecified), died 8 days after the diagnosis [5]. The other two patients died 48 days [21] and 2 months [1] after the diagnosis; these patients had received cumulative doses of amphotericin B of 2.4 g and 2.5 g, respectively. Two of the five patients with brain abscesses (due to O. gallopavum in both cases) survived [22, 23]; both patients had received therapy with amphotericin B (later substituted with amphotericin B colloidal dispersion) and flucytosine. The heart transplant recipient was cured with a 4-week course of amphotericin B therapy (cumulative dose, 811 mg) without surgical treatment [4].

Discussion

Sixty-five percent of the dematiaceous fungus infections following organ transplantation in this review occurred in the 1990s, thus suggesting that such fungi are emerging pathogens in transplant recipients. This increased rate parallels the rising incidence of such infections in other immunocompromised patients [1, 2] and is largely due to improved laboratory identification of these fungi and an increasing population of immunocompromised patients at risk for opportunistic infections.

Clinical presentation of dematiaceous fungus infections in organ transplant recipients was distinct from that of the more commonly observed fungal infections after transplantation (e.g., candidiasis and aspergillosis). The latter infections usually occur early (within 3 months of transplantation) and present predominantly as systemic invasive infections [29].

Our review showed that infections due to dematiaceous fungi occurred late (~2 years after transplantation) and presented most frequently as skin and/or soft-tissue infections. The fact that virtually all such lesions occurred on the extremities suggests that traumatic inoculation of the fungus was the likely mode of acquisition. However, patients may not recall inoculation or trauma in all cases; environmental or accidental trauma preceded such lesions in 48% of the cases in this review. The lesions were usually painless, had indolent courses, and smoldered for weeks to months before being brought to the attention of a physician. An unusual presentation was cellulitis of the extremities that was clinically indistinguishable from bacterial cellulitis. We have previously described such a presentation due to cryptococcosis in transplant recipients [30].

Skin and soft-tissue dematiaceous fungus infections were predominantly due to Exophiala organisms (E. jeaneselmei being the most frequently implicated species). Although systemic diseases due to Exophiala species (e.g., endophthalmitis, lung abscess, esophagitis, and peritonitis) have been reported, cutaneous and subcutaneous infections are also predominant clinical manifestations of infections due to Exophiala species in the nontransplant setting [6]. Systemic invasive diseases due to Exophiala species were not observed in organ transplant recipients in this review.

Dematiaceous fungi belonging to the genera Dactyliaria, Ochroconis, and Scolecosbasidium are noteworthy for their neurotrophic potential and their predilection to cause brain abscesses [28, 31, 32]. Of the five cases of brain abscesses due to dematiaceous fungi in this review, three were due to O. gallopavum. Hematogenous dissemination from a presumed pulmonary source is believed to be the mode of acquisition of brain infection; however, chest radiography did not reveal pulmonary infiltrates in the patient with brain abscess due to C. bantiana [5], and pulmonary infiltrates were not detected in 26 culture-documented cases of CNS lesions due to C. bantiana in nontransplant settings [28]. Pulmonary infiltrates were, however, detected in two of three cases of brain abscess due to O. gallopavum [1, 22] in this review.

Although the number of cases was small, the peculiar predilection of the dematiaceous fungus lesions for frontoparietal lobes in this review suggests that contiguous infection from the sinuses could also be a possible source of CNS infections.

Table 2. Dematiaceous fungi associated with infections in organ transplant recipients.

<table>
<thead>
<tr>
<th>Infection (no. of cases), fungal pathogen</th>
<th>Percent (no. of cases/total no. of cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin lesions* (16)</td>
<td></td>
</tr>
<tr>
<td>Exophiala species</td>
<td>57 (9/16)</td>
</tr>
<tr>
<td>E. jeaneselmei</td>
<td>32 (5/16)</td>
</tr>
<tr>
<td>E. spinifera</td>
<td>13 (2/16)</td>
</tr>
<tr>
<td>E. castellani</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>E. pisciphila</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>Alternaria species</td>
<td>19 (3/16)</td>
</tr>
<tr>
<td>Phialophora parasitica</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>Fonsecaea pedrosoi</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>Bipolaris hawaiensis</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>Exserohilum rostratum</td>
<td>6 (1/16)</td>
</tr>
<tr>
<td>Synovitis and/or bursitis (3)</td>
<td></td>
</tr>
<tr>
<td>Exophiala species</td>
<td>100 (3/3)</td>
</tr>
<tr>
<td>E. jeaneselmei</td>
<td>67 (2/3)</td>
</tr>
<tr>
<td>E. castellani</td>
<td>33 (3/3)</td>
</tr>
<tr>
<td>Septic arthritis (1)</td>
<td></td>
</tr>
<tr>
<td>P. parasitica</td>
<td>100 (1/1)</td>
</tr>
<tr>
<td>Systemic invasive infections (7)</td>
<td></td>
</tr>
<tr>
<td>Brain abscess (5)</td>
<td></td>
</tr>
<tr>
<td>Ochroconis gallopavum</td>
<td>60 (3/5)</td>
</tr>
<tr>
<td>Scopulariopsis brumptii</td>
<td>20 (1/5)</td>
</tr>
<tr>
<td>Cladophialophora bantiana</td>
<td>20 (1/5)</td>
</tr>
<tr>
<td>Pneumonia (1)</td>
<td></td>
</tr>
<tr>
<td>Dactyliaria constricta</td>
<td>100 (1/1)</td>
</tr>
<tr>
<td>Esophagitis (1)</td>
<td></td>
</tr>
<tr>
<td>C. bantiana</td>
<td>100 (1/1)</td>
</tr>
</tbody>
</table>

* Fungal isolates were not identified in seven cases.
Skin lesions may not accompany CNS infections; only one of seven patients with systemic invasive infections had skin lesions.

Our patient’s infection (esophageal ulcer due to C. bantiana) was diagnosed on the basis of histopathologic detection of tissue invasion and pure growth in culture. At the time of detection of esophagitis, invasive disease elsewhere was neither suspected nor diagnosed. Four cases of isolated esophagitis due to phaeohyphomycosis [33, 34], including one in a patient with AIDS [33], have been previously described in nontransplant patients; all of these cases were due to Exophiala species.

The optimal treatment for dematiaceous fungi infections is controversial, although complete surgical excision of the lesion (if feasible) is critical in the management of cutaneous lesions. Several patients whose cases were reviewed here were cured with surgery alone or were unsuccessfully treated until excision was performed. Although a variety of antifungal agents have been used in combination with surgical intervention, the efficacy of specific agents in this setting is difficult to discern. In up to 20% of the patients treated surgically, recurrence may occur [8, 18, 24]; relapse is usually at the site of the earlier lesion. Variable results have been reported following treatment of skin lesions with amphotericin B, flucytosine, or ketoconazole alone. Successful treatment with flucytosine (without surgery) of a renal transplant recipient who had a cutaneous lesion has been reported [7].

Dematiaceous fungi are generally highly susceptible to itraconazole, and successful treatment with itraconazole has been observed clinically for transplant [24, 25] and nontransplant patients [24]. Although a controlled treatment trial has never been performed, excision of cutaneous or subcutaneous lesions with or without a 3- to 6-month course of itraconazole is reasonable therapy. The precise dose and duration of itraconazole have not been delineated. However, it should be realized that relapse or recurrence following the conclusion of itraconazole therapy or, rarely, the progression of cutaneous disease during itraconazole therapy [24] may be observed.

Surgical resection is also recommended as the standard treatment for CNS lesions due to phaeohyphomycosis [28]. Complete neurosurgical resection or drainage of the lesion was the most important therapeutic intervention determining survival in a report of 26 cases of CNS lesions due to C. bantiana in nontransplant patients [28]; systemic antifungal therapy did not influence survival. Experimental C. bantiana phaeohyphomycosis was highly responsive to flucytosine therapy in one report [35].

In a murine model of systemic infection with this fungus, the mortality rate was reduced by 50% following treatment with flucytosine [35]. Although the optimal antifungal therapy for CNS phaeohyphomycosis has not been determined, combination therapy with amphotericin B and flucytosine is prudent and reasonable for CNS lesions, particularly for poorly resectable or unresectable lesions. Newer amphotericin B preparations have fewer nephrotoxic effects and are likely to be as efficacious as amphotericin B; in this report, two patients with brain abscesses due to O. gallopavum were cured with amphotericin B colloidal dispersion and flucytosine [22, 23]. Amphotericin B alone proved efficacious as treatment for cavitary pneumonia due to phaeohyphomycosis in a heart transplant recipient [4].

Our patient was treated initially with itraconazole, but his treatment was later switched to amphotericin B because of concerns of inadequate absorption since his small bowel allograft was removed due to chronic rejection. Although his esophageal ulcer was persistent (but smaller after 8 weeks of therapy), histopathologic examination no longer demonstrated the fungus.

In summary, dematiaceous fungi are being increasingly reported as pathogens and will likely assume an important role as opportunistic pathogens in organ transplant recipients. Two distinct patterns of infections due to dematiaceous fungi were observed in organ transplant recipients: skin and soft-tissue infections predominantly due to Exophiala species and, less commonly, systemic invasive disease (predominantly brain abscesses due to O. gallopavum). Although the precise role of surgery and optimal antifungal therapy in the management of these infections remains to be defined, their recognition is important since these infections may be amenable to treatment and are more responsive to therapy than is aspergillosis in organ transplant recipients.

References


