individual contact lens cases that had been contaminated. Disseminated infection is reported most often in patients who have a hematologic malignancy, are neutropenic, have received a stem cell or solid organ transplant, or have a severe burn.

Clinical Manifestations In immunocompetent persons, *Fusarium* species cause localized infections of various organs. These organisms commonly cause fungal keratitis, which can extend into the anterior chamber of the eye; cause loss of vision; and require corneal transplantation. Onychomycosis due to *Fusarium* species, while basically an annoyance in immunocompetent patients, is a source of subsequent hematogenous dissemination and should be aggressively sought and treated in neutropenic patients. In profoundly immunocompromised patients, fusariosis is angioinvasive, and clinical manifestations mimic those of aspergillosis. Pulmonary infection is characterized by multiple nodular lesions. Sinus infection is likely to lead to invasion of adjacent structures. Disseminated fusariosis occurs primarily in neutropenic patients with hematologic malignancies and in allogeneic stem cell transplant recipients, especially those with graft-versus-host disease. Disseminated fusariosis differs from disseminated aspergillosis in that skin lesions are extremely common with fusariosis; the lesions are nodular or necrotic, are usually painful, and appear over time in different locations (Fig. 243-2).

Diagnosis The diagnostic approach usually includes both documentation of the growth of *Fusarium* species from involved tissue and demonstration of invasion by histopathologic techniques that show septate hyphae in tissues. The organism is difficult to differentiate from *Aspergillus* species in tissues; thus, identification with culture is imperative. An extremely helpful diagnostic clue is growth in blood cultures, which are positive in as many as 50% of patients with disseminated fusariosis. There are no serologic assays for *Fusarium*. PCR techniques have proved useful but are available only through fungal reference laboratories.

Treatment and Prognosis *Fusarium* species are resistant to many antifungal agents. A lipid formulation of AmB (at least 5 mg/kg daily), voriconazole (200–400 mg twice daily), or posaconazole (300 mg daily) is recommended. Many physicians use both a lipid formulation of AmB and either voriconazole or posaconazole because susceptibility information is not available when therapy must be initiated. Serum drug levels should be monitored with either azole to ensure that absorption is adequate and with voriconazole to avoid toxicity. Mortality rates for disseminated fusariosis have been as high as 85%. With the improved antifungal therapy now available, mortality rates have fallen to ~50%. However, if neutropenia persists, the mortality rate approaches 100%.

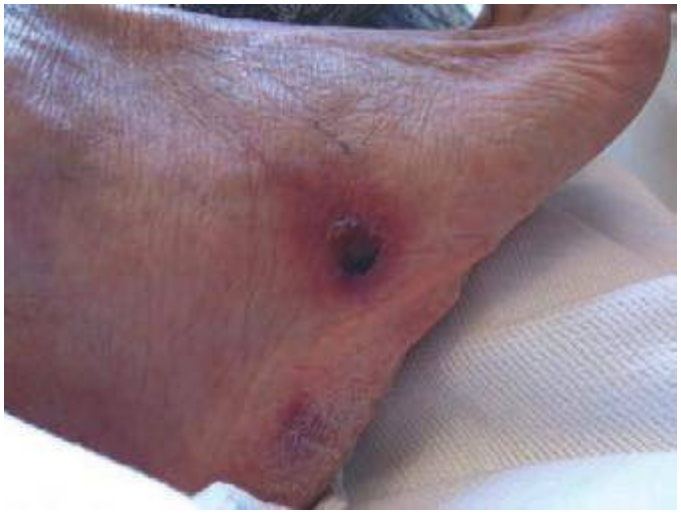

FIGURE 243-2 **Painful necrotic foot lesion** that developed over a week in a woman who had acute leukemia and who had been neutropenic for 2 months. *Fusarium* species were grown from a punch biopsy. *(Courtesy of Dr. Nessrine Ktaich.)*

SCEDOSPORIOSIS

Etiologic Agent The genus *Scedosporium* includes several pathogens. The major causes of human infections are *Scedosporium apiospermum*, which in its sexual state is termed *Pseudallescheria boydii*, and *S. prolificans*. The *S. apiospermum* complex encompasses several species but will be referred to here simply as *S. apiospermum*.

Epidemiology and Pathogenesis *S. apiospermum* is found worldwide in temperate climates in tidal flats, swamps, ponds, manure, and soil. This organism is known as a cause of pneumonia, disseminated infection, and brain abscess in near-drowning victims. *S. prolificans* is also found in soil but is more geographically restricted. Infection occurs predominantly through inhalation of conidia, but direct inoculation through the skin or into the eye also can occur.

Clinical Manifestations Among immunocompetent persons, *Scedosporium* species are a prominent cause of eumycetoma. Keratitis as a result of accidental corneal inoculation is a sight-threatening infection. In patients who have hematologic malignancies (especially acute leukemia with neutropenia), recipients of solid organ or stem cell transplants, and patients receiving glucocorticoids, *Scedosporium* species are angioinvasive, causing pneumonia and widespread dissemination with abscesses. Pulmonary infection mimics aspergillosis; nodules, cavities, and lobar infiltrates are common. Disseminated infection involves the skin, heart, brain, and many other organs. Skin lesions are not as common or as painful as those of fusariosis.

Diagnosis Diagnosis depends on the growth of *Scedosporium* species from involved tissue and the demonstration of invasion by histopathologic techniques that show septate hyphae in tissues. Culture evidence is essential because *Scedosporium* species are difficult to differentiate from *Aspergillus* in tissues. Demonstration of tissue invasion is essential because these ubiquitous environmental molds can be mere contaminants or colonizers. *S. prolificans* can grow in blood cultures, but *S. apiospermum* usually does not. There are no serologic assays for *Scedosporium*. PCR techniques have proved useful but are available only through fungal reference laboratories.

Treatment and Prognosis *Scedosporium* species are resistant to AmB, echinocandins, and some azoles. Voriconazole is the agent of choice for *S. apiospermum*, and posaconazole also has been used for this infection. *S. prolificans* is resistant in vitro to almost every available antifungal agent; the addition of agents such as terbinafine to a voriconazole regimen has been attempted because in vitro data suggest possible synergy against some strains of *S. prolificans*. Mortality rates for invasive *S. apiospermum* infection are ~50%, but those for invasive *S. prolificans* infection remain as high as 85–100%.

TRICHOSPORONOSIS

Etiologic Agent The genus *Trichosporon* contains many species, some of which cause localized infection of hair and nails. The major pathogen responsible for invasive infection is *Trichosporon asahii*. *Trichosporon* species grow as yeast-like colonies in vitro; in vivo, however, hyphae, pseudohyphae, and arthroconidia can also be seen.

Epidemiology and Pathogenesis These yeasts are commonly found in soil, sewage, and water and in rare instances can colonize human skin and the human gastrointestinal tract. Most infections follow fungal inhalation or entry via central venous catheters. Systemic infection occurs almost exclusively in immunocompromised hosts, including those who have hematologic malignancies, are neutropenic, have received a solid organ transplant, or are receiving glucocorticoids.

Clinical Manifestations Disseminated trichosporonosis resembles invasive candidiasis, and fungemia is often the initial manifestation of infection. Pneumonia, skin lesions, and sepsis syndrome are common. The skin lesions begin as papules or nodules surrounded by erythema and progress to central necrosis. A chronic form of infection mimics hepatosplenic candidiasis (chronic disseminated candidiasis).