tial diagnosis, but the patient demonstrated no signs of infection and laboratory studies did not reveal a source of infection. Scattered cerebral infarction from arterial air emboli was unlikely because no evidence of ischemia was detected on a diffusion-weighted MRI.

The timing of cortical blindness in our patient, shortly after inadvertent arterial entry during attempted PICC line placement, and the focal leptomeningeal enhancement on MRI suggest that breakdown of the blood-brain barrier was iatrogenic. The precise mechanisms underlying this process remain unclear, but we postulate that transient hyperosmolarity and hypertension may have been involved in the pathogenesis of our patient’s symptoms.

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**Disseminated Fusarium Infection Presenting as Bilateral Endogenous Endophthalmitis in a Patient With Acute Myeloid Leukemia**

Fusarium is a fungal pathogen that may cause local, as well as potentially fatal, systemic infection. Fusarium species are the most common cause of keratomycosis in the southeastern United States, and exogenous endophthalmitis is well documented in this patient population. Endogenous Fusarium endophthalmitis, however, is a rare condition with only a few cases reported. Almost all cases involve disseminated infection in immunocompromised patients, either owing to leukemia or other severe systemic conditions. The visual prognosis is poor and disease-related mortality is high despite local and systemic antifungal treatment. We describe a patient with a history of acute myeloid leukemia who developed bilateral visual loss secondary to endogenous Fusarium endophthalmitis. The patient was subsequently diagnosed as having disseminated fusariosis and died 5 days after she was first examined.

**Report of a Case.** A 70-year-old white woman was referred for evaluation for a 3-day history of visual loss. The medical history was significant for myelodyplastic syndrome that converted to acute myeloid leukemia 4 months prior to the onset of her visual symptoms. The patient had been treated with a course of chemotherapy which was completed 19 days prior to her initial visit. During chemotherapy, she developed Klebsiella pneumoniae, which was treated with piperacillin-tazobactam, ciprofloxacin hydrochloride, and vancomycin hydrochloride. One day prior to the onset of visual symptoms, blood cultures grew vancomycin-resistant _Entero-coccus faecium_ and she was started on a regimen of linezolid.

On examination, visual acuity was hand motion in both eyes. At the bedside, the portable slitlamp examination demonstrated mild (1+) cells and flare in the aqueous of both eyes and a small fibrin clot on the surface of both crystalline lenses. The funduscopic examination revealed a hazy view due to significant vitritis in both eyes. The peripheral retina appeared white with nonperfused vessels. A pars plana vitreous aspirate was performed in both eyes. Samples were sent for gram stain, culture, and polymerase chain reaction studies. Vancomycin (1 mg/0.1 mL) and amikacin sulfate (400 µg/0.1 mL) were injected into the vitreous cavity of both eyes; the patient was allergic to cefazidime sodium. Two days after the vitreous tap, vitreous cultures from both eyes were positive for _Fusarium_ species. The blood cultures (drawn 1 day after the vitreous tap) became positive for organisms 3 days later, and biopsy results from a 2 × 2-cm skin lesion with white-yellow discharge performed 3 days after the vitreous tap were also positive for _Fusarium_ species post mortem. Two days after the vitreous tap, Amphotericin B (5 µg/0.1 mL) was injected into the vitreous cavity of both eyes and the patient was started on a regimen of intravenous amphotericin B (0.7 mg/kg per day) and oral fluconazole (400 mg/d). The following day, the patient died from multiorgan failure. Post mortem, the vitreous and blood fungal polymerase chain reaction assay results showed both _Fusarium_ species and _Candida glabrata_ (Figure).

**Comment.** The _Fusarium_ species are ubiquitous filamentous molds that are commonly found in soil and on plants. _Fusarium_ infection may occur as toxicosis following ingestion; however, _Fusarium_ infections are usually local and are associated with trauma (keratomycosis) or altered body surface (superficial burn-wound infections). In the setting of severe immunosuppression and neutropenia, _Fusarium_ species may cause potentially fatal dissemi-
molecular marker. Positive for fluid positive for positive for infection in a patient with AIDS. In a section of disseminated endophthalmitis has been reported as the initial manifestation of the systemic infection manifested itself; the initial way in which the systemic antifungal therapy may play a role in clinical improvement, it appears that neutrophilic recovery is the more important prognostic indicator in the eradication of any disseminated fusariosis.

Fusarium species have the propensity to invade and occlude intracellular vasculature leading to infarction and necrosis, and this may explain the ischemic appearance of the retina in our patient. In patients with disseminated infection, dermatologic lesions frequently demonstrate Fusarium organisms. In addition, the autopsy of a patient with disseminated Fusarium revealed diffuse involvement of the central nervous system. The isolation of Fusarium from our patient’s skin lesion and the rapid decline of her mental status are consistent with these findings.

To the best of our knowledge, this is the first report of bilateral endogenous Fusarium endophthalmitis as the initial manifestation of a disseminated infection in a patient with hematological malignancy. The early recognition and diagnosis of this disease by ophthalmologists and its early treatment in collaboration with other specialists may prevent its potentially fatal outcome.

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