Endophthalmitis and Lumbar Diskitis Due to *Acremonium falciforme* in a Splenectomized Patient

Bone infections caused by *Acremonium* species are uncommon, and, to our knowledge, there have been no reports of sequential acremonium bone infections at two distant sites. We describe a patient who had previously undergone splenectomy who spontaneously developed acremonium endophthalmitis followed by diskitis of the lumber spine.

A 62-year-old man underwent a right carotid endarterectomy on 25 January 1993 because of symptoms of stenosis; in 1991 he had undergone nephrectomy and splenectomy for renal cell carcinoma. He was rehospitalized on 4 February with a 1-day history of pain and decreased vision in the right eye, which were unresponsive to treatment with eye drops containing cortisone and atropine. There was no history of direct trauma to the eye or of intravenous drug abuse.

Examination revealed that the pupil of the right eye was dilated and nonreactive, the cornea was mildly edematous, and the anterior chamber was deep with 4+ cells, preventing visualization of the fundus. Specimens from the aqueous and vitreous chambers were obtained for culture, and gentamicin and vancomycin were injected into both the vitreous and the subconjunctival space. A three-port trans–pars plana vitrectomy was performed to obtain a vitreous specimen and to partially remove debris. Vancomycin and gentamicin were again injected into the vitreous, and gentamicin was given intravenously from 5 February to 8 February 1993.

The vitreous cultures yielded an unidentified fungus, and on 7 February treatment with intravenous and intraocular amphotericin B was begun. On 11 February, the fungus was identified as an *Acremonium* species. Because the infection appeared to be unresponsive to therapy, the involved eye was removed. On 13 February, amphotericin B therapy was discontinued when the patient developed renal dysfunction after receiving a total dose of 121 mg. He was discharged on 16 February. Two cultures of anterior chamber fluid were both negative, but cultures of specimens from the posterior chamber of the eye were positive for *Acremonium falciforme*. Pathological examination of the enucleated eye revealed fungal elements in the rear of the posterior chamber and in the anterior portion of the vitreous. Blood cultures were negative.

One month later, the patient noted the gradual onset of intermittent midline lower back pain. Because of this pain, he was rehospitalized 4 months later; a bone scan revealed an abnormality in the L4–L5 interspace. End-plate erosion and diskitis at L4–L5 were identified 4 months later; a bone scan revealed an abnormality in the L4–L5 interspace. End-plate erosion and diskitis at L4–L5 were identified 4 months later; a bone scan revealed an abnormality in the L4–L5 interspace. End-plate erosion and diskitis at L4–L5 were identified on roentgenograms and an MRI.

On 15 July 1993 yellow fluid devoid of WBCs was found during L4–L5 microdiscectomy. No organisms were seen on gram staining. Cultures of the fluid and surgical samples yielded *A. falciforme*. The erythrocyte sedimentation rate (Westergren method) was 85 mm/h.

The patient was treated with oral fluconazole and amphotericin B (total dose, 336 mg) from 21 July until 17 August, when both drugs were discontinued because of the results of antifungal susceptibility tests; the tests indicated that the isolate was resistant to amphotericin B and fluconazole but susceptible to ketoconazole, miconazole, itraconazole, and saperconazole.

He was given oral itraconazole (200 mg twice daily) for 45 weeks (until 30 June 1994). His back pain slowly abated, and by early March 1994 the erythrocyte sedimentation rate had normalized. In November 1994, another MRI showed no evidence of diskitis. The patient remains well 18 months after treatment with antifungal agents was discontinued.

*A. falciforme* causes ~80% of acremonium infections in humans [1]. Our report is unique in that the acremonium infection involved two distant sites in the patient's body, the eye and the disk space. The only preceding trauma was the right carotid endarterectomy performed 9 days before his symptoms began. Since no other cases of acremonium infection have been reported locally, we cannot suggest an environmental source [2].

Although *Acremonium* species often infect the eye, infection of bone, other than that secondary to mycetoma in the tropics, is rare in the United States [1, 3]; only one patient with acremonium osteomyelitis of the calvarium [4] and two patients with septic arthritis of the knee [5, 6] have been described.

Oral itraconazole was successfully used to treat two patients with *A. falciforme* infection; one had mycetoma of the face [7], and the other was an infant with severe combined immunodeficiency disease [8].

The appropriate therapy for acremonium infections is unknown. The results of treatment of acremonium endophthalmitis have generally been poor [9]. Many *Acremonium* isolates are resistant to amphotericin B, ketoconazole, and itraconazole [10]. However, ketoconazole, was successfully used to treat mycetomas in Madras, India [3]. Itraconazole may prove useful for treating acremonium infections if in vitro susceptibility is proven.

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References

Successful Treatment of *Stomatococcus mucilaginosus* Meningitis with Intravenous Vancomycin and Intravenous Ceftriaxone

*Stomatococcus mucilaginosus* is a gram-positive bacterium normally found in the oropharynx and upper respiratory tract [1–3]. *S. mucilaginosus* has been reported to cause bacteremia, intravascular catheter infections, endocarditis, peritonitis, respiratory infections, and intracranial infections [3]. Five detailed reports of *S. mucilaginosus* meningitis in immunocompromised hosts have been published [2–6]. The one patient who survived longer than the period of acute treatment received intrathecal vancomycin and intravenous antibiotics; thus, the recommendation was made that intrathecal vancomycin be administered to patients with meningitis due to *S. mucilaginosus*. We describe a case of *S. mucilaginosus* meningitis in a child with leukemia in whom infection resolved without the use of intrathecal vancomycin therapy.

A 5-year-old child with relapsed acute lymphoblastic leukemia developed chemotherapy-induced neutropenia; three of five blood cultures yielded *S. mucilaginosus*, and four of five blood cultures yielded *Streptococcus mitis*. CSF obtained by lumbar puncture and a transthoracic echocardiogram did not show any abnormalities. The patient received a 7-day course of vancomycin and a 17-day course of cefazidime through a Hickman catheter before his parents requested that chemotherapy and antibiotic therapy be discontinued.

The patient’s neutropenia resolved; he became afebrile and remained clinically stable until 6 weeks later when he awoke with a stiff neck. He had no fever, chills, nausea, sore throat, or chest pain. Findings on physical examination were unremarkable. His peripheral WBC count was 20,300/mm³ with 76% polymorphonuclear leukocytes, 4% lymphocytes, 4% monocytes, and 7% blast cells. Laboratory studies of the CSF revealed the following values: WBC count, 1,130/mm³ with 88% polymorphonuclear leukocytes and 10% lymphocytes; RBC count, 10/mm³; glucose, 43 mg/dL; and protein, 124 mg/dL. Gram-stained smears of CSF revealed sheets of polymorphonuclear leukocytes with occasional intracellular gram-positive cocci. Findings on a chest radiograph and a head CT scan were normal.

Treatment with intravenous ceftriaxone (500 mg b.i.d.) and ampicillin was discontinued when cultures of CSF yielded only *S. mucilaginosus*. The electrophoretic patterns determined by use of the restriction enzyme HindIII indicated that the blood and CSF isolates were identical (data not shown).

Over the next 24 hours, the patient’s condition markedly improved, and he became afebrile and alert. Cultures of both blood samples drawn before antibiotic therapy was started were sterile, and the pleocytosis had almost completely resolved. *S. mucilaginosus* is reportedly susceptible to vancomycin, ampicillin, cefotaxime, imipenem, and rifampin [7] and not reliably susceptible to penicillin, clindamycin, or oxacillin [1, 5–8]. Intrathecal vancomycin, when administered as an adjunct to intravenous antibiotics, eradicated *S. mucilaginosus* from the CSF in two cases [2, 4]; however, one of the patients subsequently had a seizure and died 2 weeks later [4]. Because the other patient survived, it was recommended that intrathecal vancomycin be given to patients with meningitis due to *S. mucilaginosus*. In our patient’s case, *S. mucilaginosus* was successfully eradicated from the CSF after treatment with intravenous vancomycin and intravenous ceftriaxone was given for 21 days.

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