Polymicrobial Brain Abscess in a Patient Infected with Human Immunodeficiency Virus

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Although intracranial mass lesions that occur as a result of infection have commonly been reported in patients infected with human immunodeficiency virus, polymicrobial pyogenic brain abscess has not been described in this setting. We report the first case of a patient with a polymicrobial brain abscess involving *Streptococcus bovis*, *Fusobacterium necrophorum*, *Peptostreptococcus*, and group G *Streptococcus*, and we review the relevant world literature.

Polymicrobial brain abscesses are rarely described in HIV-infected patients, and when they do occur, the implicated pathogens are not the pyogenic organisms found in the healthy host. Rather, mycobacteria, fungi, protozoa, and *Listeria* species have all been reported as causative pathogens [1–6]. To our knowledge, we report the first case of a patient with both HIV infection and polymicrobial pyogenic brain abscess, and we review the world literature.

Case Report

A 41-year-old homosexual male, a dog groomer with a history of alcohol abuse and a remote history of iv drug use, presented to Hahneman University Hospital in February 1993. HIV infection had originally been diagnosed in 1986. In 1991, his CD4 cell count was ~250/mm³, and zidovudine therapy was started. However, he was not compliant with this medication and discontinued antiretroviral therapy within several months.

He remained asymptomatic until February 1993, when he presented to the hospital with a 4–5-day history of progressive weakness of the right upper extremity and several episodes of “right arm stiffening with head turning to the right.” Loss of consciousness was noted during two of these episodes along with severe left-sided headache. The patient denied a history of opportunistic infections, fever, chills, weight loss, nausea, vomiting, diarrhea, headaches, visual changes, sinusitis, or otitis media.

On physical examination, the patient was afebrile and had normal vital signs. He was awake and alert and exhibited normal mentation. His speech was slightly slurred, and he had mild right facial asymmetry. Extraocular movements were intact; his pupils were equal in size and reactive, and the fundi were normal. He had hypesthesia, decreased tone, and paresis in his right arm. He had no meningeal signs. Findings of the remainder of the examination were unremarkable.

Laboratory studies performed on admission revealed a WBC count of 3,800/mm³ with a normal differential. Findings on a chest roentgenogram were unremarkable. A contrast-enhanced CT of the head revealed a multiloculated ring-enhancing lesion in the frontoparietal lobe associated with white matter vasogenic edema and local mass effect (figure 1a). Therapy with phenytoin and empirical therapy with pyrimethamine and sulfadiazine for possible toxoplasmosis were begun.

A lumbar puncture performed by physicians from the patient’s primary service yielded the following results: glucose, 56 mg/dL; protein, 42 mg/dL; WBCs, 6/μL (40% neutrophils and 60% lymphocytes); and RBCs, 10/μL. Tests for CSF cryptococcal antigen were negative, as was a VDRL as well as smears for bacteria and acid-fast bacilli. After ~5 days of treatment with pyrimethamine and sulfadiazine, his CD4 cell count was reported to be 336/μL (28% of total lymphocytes), and titers of antibody to *Toxoplasma* were negative.

He subsequently underwent a stereotactic brain biopsy. Twenty mL of yellowish-green, foul-smelling material were aspirated, a finding that was believed to be consistent with pyogenic brain abscess. The patient’s antimicrobial regimen was changed to ceftriaxone, vancomycin, and metronidazole pending further microbiologic data. Final cultures of the aspi-
rate yielded *Streptococcus bovis*, *Fusobacterium necrophorum*, *Peptostreptococcus* species, and group C *Streptococcus*. After receiving ceftriaxone and metronidazole therapy for about 1 week, the patient developed a severe diffuse maculopapular eruption. This eruption was believed to be related to the ceftriaxone, and his antibiotic regimen was changed to chloramphenicol (1,000 mg iv q6h).

An extensive work-up was initiated to identify the primary source of the abscess, and an active search for *S. bovis* was made. The patient reported that he had been sexually abstinent for 4 years. A dental examination revealed a grossly decayed tooth, but the patient refused to have it further evaluated. Findings on a chest roentgenogram and on plain films of the sinuses did not reveal any abnormalities. An echocardiogram revealed a myxomatous mitral valve without vegetations. A barium enema was negative for colonic lesions.

After receiving a total of 8 weeks of antibiotic therapy, the patient developed moderate leukopenia, and chloramphenicol therapy was discontinued. He was intolerant to multiple antibiotics and consequently was unable to receive any additional antimicrobial therapy. He experienced near-total recovery of all neurological function with only minimal impairment of fine motor tasks and returned to full-time work as a dog groomer. Follow-up CT and MRI scans demonstrated progressive resolution of the lesion (figure 1b). At a 3 1/2-year follow-up visit, the patient was well and his neurological function was stable.

**Discussion**

Intracranial mass lesions are commonly described in patients with AIDS and most often occur as a result of infection. In large series of cases that have been reported, *Toxoplasma* is the organism most frequently isolated from stereotactic brain biopsy specimens from patients with AIDS; mycobacteria, *Nocardia* species, *Listeria* species, and fungal pathogens have also been isolated from biopsy specimens from these patients [4–6]. The only prior report of a polymicrobial brain abscess in an AIDS patient involved a patient with cerebral toxoplasmosis and concurrent infection that was presumed to be due to *Candida* species and coagulase-negative staphylococci [7]. We were not able to find any case reports of polymicrobial brain abscesses caused solely by bacterial pyogenic pathogens in patients with AIDS.

As is the case for ~20% of patients with brain abscesses, the primary source of infection in our patient’s case remained cryptogenic despite a full investigation [8]. Given his preliminary abnormal dental evaluation, it is possible that our patient’s brain abscess was related to an odontogenic focus. This question remains unanswered since further dental work-up was not permitted.

*S. bovis* is an extremely rare cause of brain abscess; only three cases of *S. bovis* brain abscess have been described in the literature to date, none of which were polymicrobial. Two

![Figure 1](image-url)
of these patients, including one with endocarditis, had colonic lesions similar to those described in association with *S. bovis* infective endocarditis [9, 10]. The third patient did not have any gastrointestinal pathology; his frontal brain abscess was presumed to be related to hematogenous spread from a pulmonary source [11]. Similarly, our patient had neither endocarditis nor colonic pathology. Consequently, patients with *S. bovis* brain abscess do not necessarily need to have either infective endocarditis or associated colonic pathology.

The only two cases of *S. bovis* infection in association with HIV infection that have been reported are our patient’s case and a recent case of *S. bovis* bacteremia and meningitis in an HIV-infected individual with strongyloidiasis and hyperinfection syndrome [12]. To our knowledge, our case is the first report of *S. bovis* brain abscess in an HIV-infected patient. It is unclear whether this patient’s HIV infection specifically predisposed him to the development of this abscess. Of note, our patient did not report any job-related factors or sexual practices that might have exposed him to colonic flora.

There are no specific guidelines for the treatment of “routine” polymicrobial brain abscesses in HIV-infected individuals. In particular, it is unknown whether these patients may require more prolonged treatment. Our patient’s therapy was discontinued at 8 weeks, primarily because he had developed multiple antibiotic allergies and intolerances that precluded more prolonged treatment. Nevertheless, on the basis of findings during 3½ years of follow-up, this duration of therapy seems to have been adequate in his case.

Our case illustrates that polymicrobial pyogenic brain abscesses can occur in HIV-infected individuals. On the basis of the findings in our case and given that cerebral toxoplasmosis would be an unusual occurrence in HIV-infected individuals whose CD4 cell counts exceed 200/µL, we believe that early brain biopsy should be considered for HIV-infected patients who present with unexplained space-occupying cerebral lesions.

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**References**


