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CASE REPORT

Propionibacterium acnes brain abscess in a patient with HIV-1 infection

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Introduction

Propionibacterium acnes is a rod-shaped, gram-positive anaerobe that commonly colonizes skin crypts. Although an established, albeit rare, causative organism of intracerebral abscess, it most commonly occurs in the setting of prior surgery or trauma. There have been rare reports of *P. acnes* infections in the setting of HIV, but none of intracerebral abscess (El Karoui et al. 2007; Kronish et al. 1996; Cambanis et al. 2006). We describe the case of an HIV-positive patient on stable combination antiretroviral therapy (cART) who was diagnosed with *P. acnes* brain abscess in the absence of established risk factors.

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Case

A 41-year-old right-handed man presented to the emergency room after a witnessed focal motor seizure involving twitching of the left face progressed to generalization for 2–3 min. He had no history of surgery or trauma. He had been diagnosed with HIV-1 6 years prior and had been taking cART since. Current CD4 count was 448 cells/μL, and plasma viral load was undetectable; nadir CD4 count was 150 cells/μL several years prior. He had been taking emtricitabine/tenofovir/efavirenz for nearly 2 years. He also had a history of pulmonary tuberculosis 6 years prior, for which he had been treated with antibiotics for 1 year. He had no history of intravenous drug use. Examination immediately after the seizure was notable for upper motor neuron pattern facial weakness on the left. General examination revealed normal dentition and no skin wounds or genital lesions. Gadolinium-enhanced MRI of the brain showed a 1.2-cm lobulated, rim-enhancing lesion with a smooth border and internal restricted diffusion in the right precentral gyrus (Fig. 1a, b). Single-voxel magnetic resonance spectroscopy showed normal choline, creatine, and *N*-acetyl cysteine peaks but a prominent lipid/lactate peak at 1.2 ppm. These imaging findings were all suggestive of abscess. Cerebrospinal fluid (CSF) analysis showed normal glucose and total protein, six white blood cells (94 % lymphocytes), and three red blood

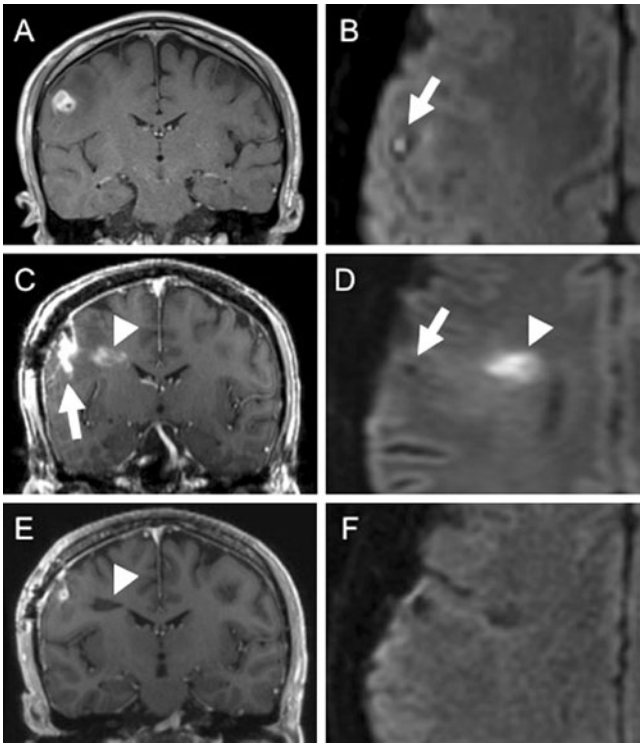

Fig. 1 Sequential magnetic resonance imaging of the brain in a patient with *P. acnes* intraparenchymal abscess. **a** Coronal T1 MRI with gadolinium at initial presentation shows a 1.2-cm multilobulated rim-enhancing lesion in the right frontal lobe with surrounding edema. **b** Axial diffusion weighted imaging at the same time point as **a** shows reduced diffusivity (hyperintensity) within the center of the lesion (*arrow*). **c** Coronal T1 MRI with gadolinium following surgical resection of the mass shows post-surgical changes within the biopsy cavity (*arrow*) and a new rim-enhancing lesion medial to the biopsy site (*arrowhead*). **d** Axial diffusion weighted imaging at the same time point as **c** shows no reduced diffusivity within the biopsy cavity (*arrow*) and reduced diffusivity within the center of the new lesion (*arrowhead*). **e** Coronal T1 MRI with gadolinium following antibiotic therapy shows interval resolution of enhancement (*arrowhead*). **f** Axial diffusion weighted imaging at the same time point as **e** shows complete resolution of abnormal reduced diffusivity

cells; gram stain and culture, acid-fast bacillus stain and culture, cryptococcal antigen, and fungal culture from the CSF were all negative. Biopsy of the lesion revealed reactive gliosis and a chronic mixed inflammatory infiltrate, consisting of lymphocytes, plasma cells, and macrophages (Fig. 2). Evaluation for systemic infection, including transthoracic echocardiogram; computed tomography of the chest, abdomen, and pelvis; urine culture; and aerobic and anaerobic blood cultures were all negative.

One week later, anaerobic culture from the biopsy grew *P. acnes*. The biopsy sample was also sent to the Centers for Disease Control for 16S ribosomal RNA sequencing, which also returned positive for *P. acnes*. The patient's exam at that time was notable for new left shoulder abduction weakness. Repeat imaging showed a new 1.9-cm lobulated, rim-enhancing lesion in the white matter subjacent to but

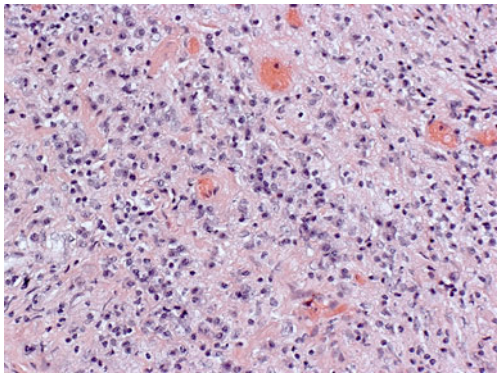

Fig. 2 A photomicrograph of hematoxylin and eosin staining shows mononuclear inflammatory infiltrates in the brain biopsy specimen; 16S ribosomal RNA sequencing of the lesion was positive for *P. acnes*

discontinuous with the prior surgical site (Fig. 1c, d). Treatment with intravenous penicillin was initiated. Four weeks later, the patient's left shoulder abduction weakness had resolved, and repeat MRI showed resolution of the diffusion restriction and new rim-enhancing lesion seen on the prior image and some residual enhancement around the biopsied lesion (Fig. 1e, f).

Discussion

Brain abscesses caused by *P. acnes* are rare and usually occur in the setting of prior trauma or surgical manipulation (Barazi et al. 2003; Chung et al. 2011; Kim et al. 2009; Kranick et al. 2009; Ramos et al. 1995; Richards et al. 1989; Senneville et al. 1997), suggesting direct seeding of the intracerebral compartment by this fastidious, anaerobic skin commensal. Dental abscess has also been a reported predisposing factor (Ingham et al. 1978) for mixed cerebral abscess containing *P. acnes*, suggesting concomitant bacteremia by organisms with sufficient virulence to overwhelm the blood–brain barrier. This patient had no known history of mechanical disruption of the blood–brain barrier or overt risk for transient, mixed bacteremia preceding the onset of his symptoms. While he did undergo a surgical procedure prior to the appearance of the second lesion, this was discontiguous with the surgical site. Although *P. acnes* can be a contaminant, it is unlikely in this patient given the positive sequencing result for *P. acnes*, the lack of growth of any other organisms, and the clinical and radiologic improvement with penicillin therapy.

In the setting of HIV infection, persistent chronic inflammation and shed viral proteins affect the integrity of the blood–brain barrier, even with ongoing cART (Li et al. 2009; Strazza et al. 2011). Hence, in this HIV-infected patient without a history of trauma or surgery, this raises the possibility that *P. acnes* may have gained access to the

brain parenchyma as a complication of HIV infection itself. As this patient had a CD4 count of 448 cells/μL, immune compromise seems unlikely to have played a role. The appearance of a second lesion subjacent to the original abscess site may relate to the surgical procedure or to predilection of the site to infection, suggesting focality to the blood–brain barrier breakdown.

In conclusion, this is the first report of *P. acnes* intracerebral abscess in the setting of HIV infection. The portal of entry into the central nervous system for this microorganism is unknown in this patient, but in the face of HIV co-infection, disruption of the blood–brain barrier mediated by viral proteins and/or chronic immune activation may have been a predisposing factor.

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