Pacemaker-Induced Endocarditis Due to Propionibacterium acnes

Propionibacterium acnes and other Propionibacterium species are branching, gram-positive, anaerobic bacilli that are part of the normal human microflora of the skin, conjunctiva, external ear, sebaceous follicles, and mouth and upper respiratory tract [1–5]. Although P. acnes is of low virulence, it has been identified as the etiologic agent in a variety of infections including CNS shunt infections, brain abscesses, endophthalmitis, neurosurgical wound infections, and, rarely, pulmonary infections [6–10]. Invasive disease usually involves a foreign body [5]. When identified in blood cultures, P. acnes is generally considered a skin contaminant. To our knowledge, we describe herein the first case of infectious endocarditis due to P. acnes associated with a pacemaker.

A 78-year-old man was admitted to Winthrop-University Hospital (Mineola, New York) for evaluation of syncope and intermittent fever with chills, night sweats, fatigue, and malaise of 6 months’ duration. The patient’s medical history included hypothyroidism, were identified as pseudomembranous colitis due to toxin producing clostridia. N Engl J Med 1994;330:257–61.

Admission laboratory studies included the following values: WBCs, 11.4/mm³; hemoglobin, 151 g/L; hematocrit, 45%; platelets, 50,000/mm³; and creatinine, 0.8 mg/dL. Results of a urinalysis were negative. A chest radiograph showed no infiltrate or effusion. An electrocardiogram showed ventricular-paced beats. A transesophageal echocardiogram (TEE) showed a normal left ventricular size; a mildly enlarged left atrium, right atrium, and right ventricle; mild aortic regurgitation, mitral regurgitation, and pulmonary regurgitation; and a large, 5-cm mass on the ventricular pacing lead.

A coronary angiogram showed two-vessel disease, and the patient underwent coronary artery bypass grafting with removal of a large right atrial mass that crossed through the tricuspid valve and surrounded one of the ventricular pacing leads (figure 1). The pacemaker leads and the pulse generator were removed and replaced with an epicardial lead and an abdominal pulse generator. Neither purulent discharge nor other signs of infection were noted at the old pulse generator site. Gram staining of the atrial thrombus revealed numerous gram-positive beaded branching rods, which were identified as P. acnes on culture.

After cardiac surgery, the patient was sent home with iv ampicillin/gentamicin to complete a 6-week course of therapy. He had an uneventful recovery.

We believe that the P. acnes isolated in this case was indeed the cause of infection for the following reasons. The organism was recovered in pure culture from surgically obtained tissue and was demonstrated by gram staining of the emulsion prepared from this tissue. Histological evaluation of the resected tissue, which revealed an organized, laminated clot fragment directly attached to the pacemaker lead, demonstrated large aggregates of pleomorphic

Figure 1. Pacemaker leads surrounded by organized, laminated clot containing Propionibacterium acnes, from a patient with endocarditis.

References

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Figure 1. Pacemaker leads surrounded by organized, laminated clot containing Propionibacterium acnes, from a patient with endocarditis.
gram-positive rods morphologically identified as *P. acnes*. The vegetative nature of this mass in association with the pacemaker lead confirmed the diagnosis of pacemaker-induced endocarditis.

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References


Isolated Cavitary Pulmonary *Mycobacterium avium* Complex Infection in a Patient with AIDS

*Mycobacterium avium* complex (MAC) infection in patients with AIDS typically causes a systemic, febrile, wasting illness [1]. We describe a case of isolated cavitary pulmonary MAC infection mimicking *Mycobacterium tuberculosis* infection in an individual with AIDS who was receiving therapy with protease inhibitors with a resultant low viral burden.

A 46-year-old man with a 10-year history of AIDS developed fever and fatigue and was noted to have a cavitary lesion in the right upper lobe on a chest roentgenogram. His medical history was remarkable for hypertension and moderate chronic obstructive pulmonary disease. The patient had a tobacco use history of 50 pack years and was still smoking two packs per day. Because of childhood exposure to tuberculosis and the chest roentgenography findings, he was treated presumptively for tuberculosis with an 8-week course of isoniazid, rifampin, pyrazinamide, and ethambutol. However, sputum cultures never yielded any mycobacteria, and antibiotic therapy was stopped. Ten months later, he was hospitalized because of a 2-week history of depression, dyspnea, scant hemoptysis, cough productive of green sputum, fever, and night sweats. At that time, the patient was receiving therapy with tizovudine, lamivudine, and ritonavir, his CD4 cell count was 302/mm$^3$, and the viral load was undetectable.

Physical examination revealed an afebrile, well-nourished man in no distress who had thrush, dry rales in all lung fields, and mild peripheral neuropathy. A chest roentgenogram and a chest CT scan showed bronchiectasis, peripheral streaking opacities, and a cavitary lesion in the right apex with smaller cavities in the lingula and the superior segment of the right lower lobe. Analysis of sputum revealed numerous acid-fast bacilli (AFB), and culture of sputum yielded *Streptococcus pneumoniae*. The patient was placed in respiratory isolation, and treatment for infections due to *M. tuberculosis* and *S. pneumoniae* was initiated. Analysis of four sputum samples showed AFB. However, after 1 month, DNA probe testing revealed that growth in all samples was only MAC. Blood cultures were all negative for MAC, indicating that the infection was limited to the pulmonary cavities.

The patient was released from isolation and discharged; medications at the time of discharge were clarithromycin, ethambutol, and rifabutin. All of his symptoms resolved. Results of follow-up analysis of sputum samples obtained 1, 3, and 5 months later were AFB-negative, and the cavities seen on the chest roentgenogram were markedly improved.

This case is unusual. MAC infection limited to pulmonary cavitary lesions has been described almost exclusively in HIV-negative individuals, typically elderly men with chronic obstructive pulmonary disease or other underlying lung disease. By contrast, in patients with AIDS, pulmonary MAC infection typically presents as a diffuse interstitial or reticulonodular infiltrate on a chest roentgenogram in the setting of a systemic, wasting, multiorgan infection when the CD4 cell count is <50/mm$^3$ [1]. Review of the literature revealed 10 cases of pulmonary cavitary lesions due to MAC in HIV-positive patients [2–5]. All but one patient had systemic MAC infection; this patient had a left lower lobe cavitary lesion on a chest roentgenogram with MAC-positive sputa and MAC-negative blood cultures [2]. Thus, our case is the second reported case of isolated cavitary MAC infection in a patient with AIDS.

We hypothesize that combination antiretroviral therapy including protease inhibitors with a resultant low viral burden was responsible for the atypical presentation of MAC infection in this case. The idea that potent antiretroviral therapy alters the natural history of opportunistic infections is also supported by a recent description of five HIV-positive patients receiving therapy with protease inhibitors who presented with MAC lymphadenitis without systemic MAC infection [6]. Further, it has been shown that protease inhibitors only partially restore the immune system in patients with HIV infection, making it plausible that opportunistic