Pacemaker Lead Infection and Vertebral Osteomyelitis Presumed Due to *Cardiobacterium hominis*

*Cardiobacterium hominis* is a member of the HACEK (*Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikenella*, and *Kingella*) group of fastidious, gram-negative, aerobic bacilli, which are rare causes of endocarditis. The presence of *C. hominis* in the blood is virtually diagnostic of endocarditis, and extracardiac infections caused by *C. hominis* have been reported only twice [1, 2]. We describe a patient with two unique features of *C. hominis* bacteremia: pacemaker lead infection and osteomyelitis.

A 76-year-old woman was admitted to the MetroWest Medical Center (Framingham, MA) because of fever and lower back pain. These symptoms began >2 months earlier and had led to an evaluation at another hospital. Findings on a bone scan and CT of the lumbar spine were abnormal, but were interpreted as consistent with degenerative changes. Cultures of blood obtained on admission were negative, and the fever subsided. One week later, the fever recurred and a CT scan of the lumbar spine revealed narrowing of the joint space between L4 and L5 with erosion of the superior end-plate of L5 (figure 1). Two blood cultures were negative. A biopsy of the lumbar spine was obtained under CT guidance, and histopathologic evaluation was nondiagnostic; cultures of the biopsy specimen were negative. A chest radiograph revealed an infiltrate. Intraavenous cefuroxime was administered, and the patient became afebrile.

One month later, the back pain worsened and the fever recurred. Blood was drawn for culture and 10 days later the laboratory reported that the culture yielded gram-negative rods. Additional blood was obtained for cultures, and the patient was admitted to the hospital for further evaluation. At the time of the admission, the patient was complaining of back pain that radiated to the lower abdomen and both thighs, and she gave a history of a recent 22-pound weight loss. The medical history was remarkable for diabetes mellitus, coronary artery disease, and placement of a dual-chamber cardiac pacemaker for bradycardia 13 years earlier. The physical examination revealed a non-ill-appearing, afebrile woman (temperature, 98.4°F). There was no erythema or fluctuance over the site of the pacemaker generator box. A grade 2/5, late systolic murmur was present at the left sternal border. There was mild point tenderness over L4 and L5.

Laboratory evaluation revealed the following values: WBC count, 9,500/mm³; hemoglobin level, 8.2 g/dL; and a normal platelet count. The erythrocyte sedimentation rate was 89 mm/hour. The blood culture isolate was identified as *C. hominis*, and additional cultures of blood yielded the same organism. A transesophageal echocardiogram revealed a large, multilobular vegetation attached to the atrial pacing wire, but no valvular vegetations were seen. Subsequently, the entire pacemaker system was removed surgically, and the patient was treated with iv ceftriaxone for 6 weeks.

![Figure 1. CT scan of the lumbar spine of a 76-year-old woman with pacemaker lead infection and vertebral osteomyelitis (presumed due to *Cardiobacterium hominis*) showing destruction of the fifth lumbar vertebra.](https://academic.oup.com/cid/article-abstract/27/4/890/428710)

Vertebral osteomyelitis should always be suspected when patients present with fever, back pain, and vertebral tenderness. Imaging studies are important in attaining the diagnosis [3]; in our patient, the CT scan was virtually diagnostic of spine infection and diskitis. Although the culture of the bone biopsy specimen was negative, the sensitivity of a biopsy specimen evaluation and the diagnosis of vertebral osteomyelitis may be as low as 25% [4]. Because *C. hominis* is fastidious, it is not surprising that the culture was negative. The delay in making the diagnosis in our case is not unusual in patients with infections due to *C. hominis* [1]. Factors that contribute to the diagnostic difficulties associated with this condition include the slow growth of *C. hominis*, inadequate duration of culture incubation, and prior antibiotic therapy.

To our knowledge, we describe the first case of pacemaker lead infection caused by *C. hominis*. Permanent pacemaker infection is unusual (incidence, 0.2% to 3%) and most commonly caused by staphylococci [5]. Diagnosis may be difficult and is determined on the basis of the clinical signs of infection, the presence of positive blood cultures, and echocardiography. The diagnostic value of esophageal echocardiography is superior to that of transesophageal studies [6]. Cure usually requires surgical removal of the entire pacing system.

This case illustrates the need for clinicians to suspect fastidious organisms and to alert microbiology laboratory personnel when cultures are negative for patients with evidence of systemic infection.

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References

An Appendiceal Abscess Due to Mycobacterium kansasii in a Child with AIDS

Appendicitis caused by nontuberculous mycobacteria among patients with HIV/AIDS is rare and is usually associated with disseminated mycobacterial infection [1, 2]. Mycobacterium kansasii causes pulmonary and disseminated disease in patients with AIDS [3–6]; however, appendicitis due to an infection with this organism has not been reported. We describe the first case of appendicitis and appendiceal abscess due to M. kansasii in a patient with AIDS.

A 12-year-old boy was diagnosed with AIDS in August 1993, when he presented for evaluation of failure to thrive, and oropharyngeal and esophageal candidiasis. At that time, his CD4⁺ cell count was 10/mm³. Therapy with zidovudine, trimethoprim-sulfamethoxazole, and flucloxacillin was initiated. In September 1993, he presented for evaluation of a 1-day history of abdominal pain and fever. He did not have diarrhea, vomiting, or any signs or symptoms related to his respiratory tract, urinary tract, or neurological or musculoskeletal systems. Physical examination revealed an axillary temperature of 38°C and diffuse abdominal tenderness with maximum severity at McBurney’s point; bowel sounds were absent. The remainder of the physical examination findings were unremarkable. Laboratory studies revealed the following values: WBCs, 13.1 × 10⁹/L (90% polymorphonuclear cells, 7% lymphocytes, and 3% monocytes); hemoglobin, 106 g/L; platelets, 269 × 10⁹/L; and erythrocyte sedimentation rate, 75 mm/h. The coagulation profile, blood urea nitrogen level, serum creatinine level, serum electrolyte levels, liver enzyme levels, and bilirubin level were normal.

A chest radiograph showed normal findings. An abdominal radiograph showed absence of air in the right iliac fossa with multiple fluid levels and an obscured right psoas margin. Ultrasonography revealed a well-localated, bilobed, fluid collection in the right iliac fossa and an echogenic material within the collection consistent with an abscess. The differential diagnosis included appendiceal abscess, intestinal perforation, and intestinal obstruction due to an AIDS-related pathology (e.g., lymphoma, Kaposi’s sarcoma, mycobacterial infection, cytomegalovirus infection, toxoplasma adenitis).

Therapy with ampicillin, amikacin, and metronidazole was initiated, and the patient underwent a laparotomy. Intraoperative findings included a turbid, yellow peritoneal fluid; a 5-cm × 6-cm mass surrounding the appendix and involving the wall of the cecum; and a 4-cm × 5-cm mass just medial to the ascending colon in the mesentery of the small bowel that consisted of matted lymph nodes. Operative procedures included biopsies of the masses and aspiration of purulent fluid from the periappendicular mass. Histological examination of the biopsy specimens showed caseating granulomatous inflammation and a large number of acid-fast bacilli. No malignant cells or inclusion bodies were seen. Gram staining and Grocott-Gomori methenamine–silver nitrate staining did not show any organisms.

Empirical therapy withisoniazid, rifampicin, ethambutol, and clarithromycin was initiated. Cultures of the biopsy specimens and the abscess aspirate yielded a mycobacterium that was identified as M. kansasii [7]. Cultures of blood, gastric aspirate, stool, urine, and peritoneal fluid did not yield mycobacteria or other pathogens. The patient’s condition improved markedly. Therapy with ampicillin, amikacin, and metronidazole was discontinued after 1 week. CT scans of the abdomen, after 3 and 6 months, showed regression of the abdominal masses and complete resolution, respectively. After 12 months, therapy with ethambutol and isoniazid was discontinued, and that with rifampicin and clarithromycin was maintained. In October 1996, the patient developed pneumococcal meningitis and septicemia and he died within 24 hours after presentation to the hospital. An autopsy was not performed.

Acute iliac fossa pain in patients with HIV/AIDS is usually caused by suppurative inflammation of the appendix; however, specific AIDS-related opportunistic infections and neoplasms such as Kaposi’s sarcoma, non-Hodgkin’s lymphoma, cytomegalovirus, and mycobacterial infections have been implicated in a minority of cases [1, 2, 8, 9]. There is no single symptom or sign that distinguishes bacterial appendicitis from other AIDS-related abdominal syndromes. Consequently, a correct diagnosis is often delayed, resulting in a 40% perforation rate at the time of presentation [8]. Appendicitis caused by atypical mycobacteria is rare and is usually associated with disseminated mycobacterial disease [1, 2].

Disseminated M. kansasii infections usually occur late in the course of HIV infection, when the CD4⁺ cell count is low (mean, ±62/mm³; median, ±24/mm³) and immunosuppression is advanced [3–6]. Infections due to M. kansasii commonly manifest as chronic pulmonary disease with diffuse interstitial and nodular infiltrates and endobronchitis, but extrapulmonary sites may be involved in ±50% of cases [3–6], including lymph nodes, soft tissues, bone marrow, liver, CSF, sinuses, mouth, brain, bone, and small bowel. To our knowledge, on the basis of a MEDLINE search of the English-language literature (1966–1997), we described the first case of M. kansasii causing appendicitis and a periappendicular abscess. Unlike patients with appendicitis due to Mycobacterium avium complex [1, 2], the patient we described did not have disseminated M. kansasii, as evidenced by the lack of signs and symptoms that are usually associated with disseminated...