Whipple's disease. Awareness of unusual manifestations of clinical relapse, along with application of PCR, will permit more accurate diagnosis and administration of more effective antibiotic treatment.

A. Frésard, C. Guglielminotti, P. Berthelot, A. Ros, F. Farizon, C. Dauga, H. Roussel, and F. Lucht
Service des Maladies Infectieuses et Tropicales, Laboratoire de Bactériologie et Virologie, Centre d'Orthopédie et de Traumatologie, and Service de Médecine Interne, Centre Hospitalier et Universitaire de Saint-Etienne, Saint-Etienne; and Unités des Entérobactéries, Institut Pasteur, Paris, France

References
2. Fleming JL, Wiesner RH, Shorter RG. Whipple's disease: clinical, biochem-
ical, and histopathologic features and assessment of treatment in 29 pa-
293–301.
4. Sambrook J, Fritsch EF, Maniatis T. Molecular cloning: a laboratory man-
ual. 2nd ed. Cold Spring Harbor, New York: Cold Spring Harbor Labo-
ratory, 1989.
1343–6.

Corynebacterium striatum: An Undescribed Agent of Pacemaker-Related Endocarditis

Infection complicates implantation of permanent pacemakers in 1%-7% of cases [1]. Pocket and wire infections predominate (90% of cases), while endocarditis is rare (<10% of cases) [1]. We report a case of pacemaker-related endocarditis in a patient who had relapsing Corynebacterium striatum bacteremia.

A 73-year-old man who underwent pacemaker implantation 6 years previously was admitted to the hospital for evaluation of fever. Because of decubitus, the battery was replaced in 1991 and again in February 1994; the electrode was cut and the distal part was left in place. In July the patient was admitted to the hospital because of fever and a draining sinus tract that initially appeared in the supravacular fossa. Cultures of blood and material from the draining sinus tract yielded C. striatum, but transthoracic echocardiographic findings were normal. Treatment with vancomycin was started, but he continued to be febrile. One month later, the battery was again removed, and the electrode was cut. After a 4-week course of vancomycin therapy, he was discharged and instructed to take oral co-trimoxazole and rifampin for another 4 weeks.

Two weeks later, the patient's fever recurred, and he was readmitted. No inflammatory signs were seen. Chest films showed the remaining portion of the old electrode in the right atrium, and blood cultures again yielded C. striatum. A transesophageal echocardiogram revealed vegetations on the tricuspid valve. The patient was treated for 4 more weeks, and 6 months later, he remains well.

Infection secondary to implantation of permanent pacemakers occurs infrequently—usually during the first 6 months after surgery [1]. We have been able to identify only 103 cases, although the incidence seems to be increasing [1–3]. Staphylococcus aureus is the predominant causative agent (50% of cases), followed by Staphylococcus epidermidis (25%) and gram-negative rods (25%) [1, 2]. Ours is the first known case of pacemaker-related endocarditis due to a Corynebacterium species. The two species most commonly involved in other kinds of endocarditis are C. pseudodiphtheriticum (previously known as C. hofmannii) [6] and C. jeikeium (group JK) [4, 5]. One case of native valve endocarditis due to C. striatum has recently been described [5]. Septicemia, wound infections, and reoperations are the main risk factors for endocardi-
tis due to Corynebacterium species [2]. Murmurs are infrequently present [2], and echocardiography often fails to demonstrate vege-
tations [6].

Parenteral antibiotics, followed by removal of the pacemaker system, with replacement on the other side of the chest, is the recommended therapy [2]. Removal of the electrode may be difficult and necessitate cardiomyectomy. In patients for whom surgery is contraindicated, another attempt at therapy with parenteral antibi-
otics, followed by oral treatment (for at least 6 months), may be an alternative [3, 7]. Vancomycin or penicillin with an aminoglyco-
side should be effective against most strains of Corynebacterium [5, 8]; however, therapy should be guided by susceptibility testing, as this report (as well as others) illustrates [5].

The patient we have described is unusual for several reasons: endocarditis related to implantation of a long-term endocardial pacemaker is rare; infection of a permanent pacemaker with Corynebacterium species has not been previously reported; the infected electrode wire had to be withdrawn by transjugular traction; and a vegetation was discovered 1 week after the wire was removed.

Maria Melero-Bascones, Patricia Muñoz, Marta Rodríguez-Crétienx, and Emilio Bouza
Division of Microbiology and Infectious Diseases, Hospital Universitario "Gregorio Marañón," Madrid, Spain

References
299–305.
Granulomatous Pneumocystis carinii Myositis Presenting as an Intramuscular Mass

Pneumocystis carinii pneumonia (PCP) is the most common opportunistic infection in patients with AIDS, whereas extrapulmonary P. carinii infection remains a relatively unusual occurrence [1-4]. When extrapulmonary infection does occur, the organisms are most commonly seen in the liver, lymph nodes, spleen, and bone marrow; less frequently, the pleurae, kidneys, adrenal glands, gastrointestinal tract, thymus, heart, pancreas, thyroid, eye, middle ear, brain, pituitary, parathyroid glands, and skin are involved [1-7]. We report what we believe to be the first case in which P. carinii infection presented in an intramuscular site in a patient with AIDS.

A 30-year-old homosexual man presented with a history of HIV infection and severe molluscum contagiosum; his CD4 lymphocyte count was 49/mm³. Treatment with zidovudine (500 mg/d) and aerosolized pentamidine (as prophylaxis for PCP pneumonia) were initiated. One year later, he reported loss of vision, photophobia, and eye irritation. The results of ophthalmoscopic examination were compatible with cytomegalovirus (CMV) retinitis. He was treated with ganciclovir, which was subsequently replaced with foscamet. One month later, he developed muscle spasms, pain, and swelling in his right thigh. A biopsy of the thigh muscle was performed, and he was treated with trimethoprim-sulfamethoxazole; the leg pain and swelling resolved. During the next year, he had recurrent episodes of Pseudomonas aeruginosa infection and CMV retinitis, but findings on his chest roentgenogram remained normal, and his sputum was negative for P. carinii throughout his life. Five months later, he died of disseminated Mycobacterium avium complex infection. Permission for a postmortem examination was denied.

Histopathologic examination of the biopsy specimen revealed scattered, residual skeletal muscle fibers (some of which showed regenerative changes) separated by a foamy, eosinophilic exudate (figure 1). A granulation tissue response was present, in association with mild, perivascular granulomatous inflammation. Gomori’s methenamine silver stain demonstrated P. carinii cysts (figure 1, inset). Fite stain, periodic acid–Schiff stain, and Giemsa stain were negative for organisms other than P. carinii, and the immunoperoxidase reaction for CMV was negative as well.

Although originally seen as a pulmonary pathogen, P. carinii is being recovered with increasing frequency from extrapulmonary sites in patients with AIDS [1-7]. In many of these cases, there has been no concomitant pulmonary involvement [1-4, 6]. The findings on chest roentgenograms and sputum examinations were normal for our patient, and no further evidence of P. carinii infection developed during the last 1.5 years of his life; these circumstances militate against additional sites of involvement in our patient.

Extrapulmonary P. carinii infection presents histopathologically as a foamy, eosinophilic exudate, occasionally with a granulomatous component, with cysts identified in appropriate stains; this pathology is similar to that of PCP pneumonia [1, 2, 4-8]. In our case this pathology was present, in a previously unreported site. In light of this finding, P. carinii myositis should be considered

Figure 1. A foamy, eosinophilic exudate in the thigh muscle of a patient with Pneumocystis carinii myositis and HIV infection separates proliferating capillaries with surrounding granulomatous inflammation. Rare residual skeletal muscle fibers (arrowheads), including some with reactive changes, are present (stain, hematoxylin-eosin). Inset: P. carinii cysts (arrowheads) are seen within the exudate (stain, Gomori’s methenamine silver; bar in figure = 75 μm; bar in inset = 25 μm).