Pacemaker Infection Caused by Staphylococcus schleiferi, a Member of the Human Preaxillary Flora: Four Case Reports

Infection of the pacemaker pocket, the endocardial lead, or both, occurs in 1%-7% of patients with permanent pacemakers [1] and may present as a simple pocket infection, sepsis, or endocarditis. The pacemaker pocket itself is the most common site of entry, but the pacing wire and adjacent traumatized endothelium often become infected later; in such cases, the prognosis is poor [1]. Approximately 75% of pacemaker infections are caused either by coagulase-negative staphylococci (the lesions usually appear late) or by Staphylococcus aureus (the lesions typically appear within a few weeks after implantation of a pacemaker) [2].

Staphylococcus schleiferi is a coagulase-negative staphylococcus that has rarely been associated with human infections; its relation with humans is poorly characterized [3-5]. To our knowledge, this organism has not been described as a cause of pacemaker infections. We report four cases of infection by S. schleiferi that spread from the skin and caused extrusion of the patients’ pacemakers.

Case 1. In 1993, a 62-year-old male diabetic presented with an extruded pulse generator 12 months after implantation of a pacemaker. S. schleiferi was isolated from the removed generator (the leads were retained). The patient was treated with oral pristinamycin for 2 weeks. One month later he became febrile, and S. schleiferi was isolated from a blood culture. He was given intravenous teicoplanin for 2 weeks and oral pristinamycin for 5 weeks. Three months later, the fever reappeared (temperature, 39°C), and five blood cultures yielded S. schleiferi. The entire pacing system was removed, and S. schleiferi was again isolated in culture. A new generator with an epicardial electrode was inserted in the lower left abdominal wall, and the patient was cured after a 4-week course of treatment with teicoplanin and ciprofloxacin.

Case 2. In 1994, a 71-year-old male presented with a pacemaker-pocket infection, an extruded generator, and cutaneous inflammation but no fever 10 months after implantation of a pacemaker. S. schleiferi was isolated from the wound, the pulse generator, and the leads on removal of the pacemaker. Three blood cultures were negative. The patient was treated with pristinamycin for 10 days. A new pacemaker was inserted on the contralateral side 5 months later, and he was cured. Before the insertion of the new generator, S. schleiferi was isolated from the skin around the pacemaker pocket.

Case 3. In 1994, an 83-year-old male presented with an extruded pulse generator and cutaneous inflammation 7 months after insertion of a pacemaker. Blood cultures were negative. S. schleiferi was isolated from the wound and the pulse generator when it was changed. The patient was given oxacillin for 8 days. Two months later, he was readmitted to the hospital because of repeated extrusion of the generator, which was removed. S. schleiferi was isolated again from the generator. A new pacing system was inserted on the same side, and oral ampicillin was given for 8 days. There were no further recurrences of the infection.

Case 4. In 1994, a 77-year-old male presented with an extruded pacemaker 6 weeks after surgery. Physical examination revealed pus around the generator, but the patient was not febrile, and blood cultures remained negative. S. schleiferi was isolated from the pus as well as the generator and leads following their removal. Ten days later a new pacing system was inserted. The patient was cured after treatment with oxacillin and ofloxacin for 15 days.

In the four cases described above, the only pathogen isolated from the removed pacemakers and leads was S. schleiferi, which was identified with use of the ID 32 STAPH gallery (bioMérieux, Marcy l’Etoile, France). These severe infections occurred in patients at two different hospitals, between 6 weeks and 12 months after surgery, and caused cutaneous inflammation and extrusion of the pulse generators.

Infection appears to start in the pacemaker pocket but may extend down the pacemaker wire; bacteremia subsequently developed in one of these cases. Although S. schleiferi is usually susceptible to all antistaphylococcal agents including penicillin G [3], various antibiotics were given to the patients, and in each case, cure was achieved only by removal of the entire pacing system. Attempts to retain the infected leads contributed to the development of bacteremia in case 1.

We cultured prospective samples of skin (before preaxillary incisions were made) from the manually created subcutaneous pockets and from the pulse generators in 104 patients; S. schleiferi was isolated from five of these patients, one of whom developed bacteremia 4 months later without pacemaker extrusion or pocket inflammation. Findings on transesophageal echograms were normal for all five pa-
Sterile, Caseous Mitral Valve "Abscess" Mimicking Infective Endocarditis

Dyspnea is a common complaint of patients with valvular heart disease. It is uncommon, however, for echocardiography to reveal a valvular mass in these patients. The differential diagnosis usually includes infective endocarditis and neoplasms. We describe a patient who presented with shortness of breath and was found to have a noninfectious, nonneoplastic lesion of the mitral valve that has been termed "sterile, caseous mitral valve abscess."

A 65-year-old female presented with shortness of breath that had worsened over several months. She denied chest pain, orthopnea, paroxysmal nocturnal dyspnea, or fever, and she was not taking any medications.

Physical examination revealed a respiratory rate of 16 and a temperature of 37°C. The patient’s lungs were clear to auscultation, and a grade 2/6 systolic ejection murmur was heard best at the apex. There was no hepatosplenomegaly, pedal edema, or peripheral emboli. On admission to the hospital, her WBC count was 3,900/mm³. The chest roentgenogram did not show infiltrates or effusions, and the cardiac silhouette was normal.

A transesophageal echocardiogram revealed a 2.0 cm × 3.0 cm mass attached to the ventricular side of the posterior leaflet of the mitral valve (figure 1) and severe mitral regurgitation. The mass was excised, and a prosthetic mitral valve was implanted. At surgery, a nodular, exophytic mass adherent to the posterior portion of the mitral valve was removed. Sectioning revealed a pasty, white material that filled the center of the mass. Cultures for bacteria, fungi, and acid-fast bacilli did not yield any infectious organisms, and histochemical stains were also negative. Histological examination showed basophilic, amorphous material with scattered calcifications. No granulomas were present.

A sterile, caseous mitral annular abscess was diagnosed. The patient did not receive any treatment with antibacterial agents, and she remains asymptomatic 6 months after surgery.

Sterile, caseous mitral annular abscess is an uncommon disease that may present with symptoms and echocardiographic findings similar to those of infective endocarditis. In 1983, Kronzon et al. [1] described three patients who presented with dyspnea and were found to have intramyocardial masses involving the mitral valve annulus; when these masses were excised, they had the appearance and consistency of glazier’s putty. Infectious and neoplastic etiologies were excluded in each of these cases, and biochemical analyses demonstrated that the acellular material consisted of fatty acids, cholesterol, and calcium.

While the clinical, radiological, and histopathological features in our case are similar to those described by Kronzon et al., our case differs in that the lesion was entirely extramyocardial; the lesion appeared to arise from the mitral valve itself, thereby mimicking infective endocarditis on echocardiographic studies. To our knowledge, our case represents only the fourth reported instance of antemortem diagnosis of this unusual entity.

In a postmortem study of 258 patients with mitral annular calcifications, Pomerance and Davies [2] described similar cases of "caseous abscesses" in 3% of their patients. Mitral ring calcification is a relatively common age-related finding that generally occurs in individuals >70 years of age [3]. The association between these two entities and their similar histological appearances suggest a common pathogenetic mechanism that may represent a pathological acceleration of the normal aging process.

Whether other factors such as underlying metabolism and hemodynamic or cardiovascular abnormalities are involved in influencing the size and clinical significance of these mitral valve lesions is unknown. We question whether the terminology used to describe the lesions is correct, since an abscess represents a localized collection of pus (leukocytes), and the entity described in this report is free of inflammatory cells. Perhaps...