Brief Reports

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person-years in period B ( P = .03; RR = 1.93; 95% CI = 1.02–3.67). Changes in incidence over time (expressed as episodes per 100 person-years) are shown in figure 1.

There was no statistically significant difference in risk factors for community- and hospital-acquired bacteremia between the two study periods. Intravenous drug abuse and neutropenia were significantly ( P < .05) associated with the development of community-acquired bacteremia, whereas presence of central venous catheters and prolonged hospitalization equally increased the risk of nosocomial bacteremia in both the study periods.

The results of our study show a statistically significant decrease in the incidence of both community- and hospital-acquired bacteremia in 1997, 1 year after HAART was established as the standard treatment for HIV-infected patients in our facility. One could hypothesize that these results are partially the consequence of the well-known immune restoration induced by HAART [1]. In fact, as expected, for our patients receiving HAART, the median CD4+ cell count increased significantly and the plasma HIV RNA level decreased (data not shown). However, in addition to immune restoration, there are other cofactors that might be implicated in the decrease in incidence of bacteremia. First, the decrease in duration of hospitalization recently [3] reported for patients who undergo HAART, also observed in our study, is clearly associated with a reduction in the incidence of nosocomial bacteremia. Secondly, although HAART causes a decrease in the incidence of several AIDS-related illnesses, the therapy indirectly produces a reduction in drug-induced neutropenia and in central venous catheters usage, both of which are well-known risk factors for bacteremia [4].

In conclusion, our study is the first to demonstrate reduction in the incidence of bacteremia (from 11.8 episodes to 6.3 per 100 person-years) among HIV-infected individuals during the last year, when extensive use of antiretroviral combination therapy with protease inhibitors became the standard therapy for HIV infection. Such a reduction in incidence of bacteremia after only 1 year of HAART could have a substantial impact on future patient morbidity and health care costs.

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References


Native Valve Endocarditis Due to a Nocardia-like Organism

Infections due to Nocardia species have been recognized in humans for more than a century, since Eppinger described the first case in 1890 [1]. Nocardial infections have a predilection for the lungs, skin and soft tissues, and the CNS [2]. Endocarditis due to Nocardia species is rare, having been reported previously in only six patients, all in the setting of prosthetic valve placements [3–7]. To our knowledge, based on a review of the English-language literature, we describe the first case of nocardial endocarditis associated with a native mitral valve.

A 46-year-old Hispanic woman was admitted to Pomona Valley Hospital Medical Center (Pomona, CA) on 11 August 1994 for evaluation of pain and swelling of her left second toe for 1 week and a fever of 3 days’ duration. She was unaware of the occurrence of any injury or insect bite to the affected toe. She denied any history of headache, sore throat, cough, shortness of breath, chest pain, nausea, vomiting, diarrhea, painful or frequent urination, skin rashes, or joint swellings, and there was no history of intravenous drug use, recent dental work, or prior heart disease. A physical examination revealed a temperature of 38.3°C; a 3/6 holosystolic murmur radiating to the left axilla; and a tender left second toe with subcutaneous, erythematous nodules. Laboratory evaluation revealed the following values: WBC count, 8.7 × 10^9/L (67% polymorphonuclear leukocytes); hemoglobin level, 10.8 g/dL; and platelet count, 375 × 10^9/L. Electrocardiography was normal and a chest radiograph revealed left lower lobe atelectasis. Results of a urinalysis revealed trace proteinuria, 15 RBCs per high power field, and 6 WBCs per high power field. Transthoracic echocardiography revealed a thickened anterior mitral-valve leaflet with a vegetation, mitral valve prolapse, and moderate-to-severe regurgitation. Multiple sets of cultures of blood obtained on admission (three), on 13 August (two), on 14 August (two), on 15 August (one), and on 16 August (three) yielded no bacteria. Antibiotic therapy with intravenous penicillin, nafcillin, and gentamicin was instituted empirically for treatment of suspected endocarditis on 16 August.

On 19 August, the patient developed severe left ventricular failure which necessitated intubation. Transesophageal echocardiography confirmed a markedly flailed anterior mitral valve leaflet with a vegetation. Emergency mitral valve replacement surgery was performed on 20 August. Histopathologic evaluation of the mitral valve specimen revealed vegetative endocarditis; acute and chronic inflammation; and dense collections of filamentous, beaded organisms within the vegetations. The organisms were variably gram positive—positive by Gomori’s methenamine silver staining, neg-
Table 1. Reported cases of endocarditis due to *Nocardia* species.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Reference</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>Site of involvement</th>
<th>Antibiotic therapy</th>
<th>Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [5] 1973</td>
<td></td>
<td></td>
<td>34/F</td>
<td></td>
<td>Prosthetic mitral valve</td>
<td>None</td>
<td>Died before specific therapy</td>
<td>Right groin abscess after surgery, cultures yielded <em>Nocardia asteroides</em> and may have been the source; infection 4 mo after valve replacement; no pulmonary lesion noted</td>
</tr>
<tr>
<td>2 [4] 1979</td>
<td></td>
<td></td>
<td>64/F</td>
<td></td>
<td>Prosthetic aortic valve</td>
<td>Ampicillin and gentamicin</td>
<td>Died before specific therapy</td>
<td>Endocarditis due to <em>N. asteroides</em> 6 mo after valve replacement; no pulmonary involvement</td>
</tr>
<tr>
<td>3 [6] 1985</td>
<td></td>
<td></td>
<td>53/M</td>
<td></td>
<td>Aortic root aneurysm after aortic valve replacement</td>
<td>Streptomycin, tetracycline, vancomycin, sulfonamides</td>
<td>Died</td>
<td>Infection due to <em>N. asteroides</em> 5 mo after valve replacement; no pulmonary involvement</td>
</tr>
<tr>
<td>4 [3] 1987</td>
<td></td>
<td></td>
<td>61/M</td>
<td></td>
<td>Prosthetic aortic valve</td>
<td>Imipenem/cilastatin for 3 w, then TMP-SMZ</td>
<td>Recovered</td>
<td>Onset of infection due to <em>N. asteroides</em>, biovar B (<em>Nocardia farcinica</em>) only a few days after surgery; no pulmonary involvement</td>
</tr>
<tr>
<td>5 [7] 1988</td>
<td></td>
<td></td>
<td>61/M</td>
<td></td>
<td>Aortic root aneurysm after aortic valve replacement</td>
<td>Amikacin plus imipenem/cilastatin, then amikacin plus cefotiam</td>
<td>Recovered</td>
<td>Infection due to <em>N. asteroides</em> 3 w after valve replacement; no pulmonary involvement</td>
</tr>
<tr>
<td>6 [7] 1988</td>
<td></td>
<td></td>
<td>65/M</td>
<td></td>
<td>Aortic root aneurysm after aortic valve replacement</td>
<td>None</td>
<td>Died before specific therapy</td>
<td><em>N. asteroides</em> sternotomy infection after surgery; no pulmonary involvement</td>
</tr>
<tr>
<td>7 [PR] 1990</td>
<td></td>
<td>46/F</td>
<td></td>
<td></td>
<td>Native mitral valve</td>
<td>TMP-SMZ plus imipenem/cilastatin for 1 mo, then TMP-SMZ for 9 mo</td>
<td>Recovered</td>
<td>Recurrent soft-tissue abscesses; organisms resembling <em>Nocardia</em> species on histology of valve specimen but cultures negative, possibly due to prior antibiotic therapy</td>
</tr>
</tbody>
</table>

**NOTE.** PR = present report; TMP-SMZ = trimethoprim-sulfamethoxazole.

Active by Ziehl-Neelsen staining; but staining with use of Fite’s method revealed acid fast organisms. These results were characteristic of endocarditis due to *Nocardia* species. Cultures of the mitral valve specimen were negative (including mycobacteria and fungi). There was no evidence of HIV infection, underlying immunosuppressive illness, or CNS nocardiosis. Postoperatively the patient was treated with a combination of trimethoprim-sulfamethoxazole (TMP-SMZ) and imipenem/cilastatin for 1 month, followed by TMP-SMZ alone for 9 months. No evidence of recurrent infection has been noted for >2 years after discontinuation of antibiotic therapy.

A review of the patient’s previous medical records revealed evidence of recurrent skin infections, beginning in November 1992, when she first developed a painful swelling of her left ankle. The left ankle condition did not respond to empiric antibiotic therapy, including that with dicloxacillin. The patient was hospitalized in February 1993 for a draining sinus and persistent cellulitis of her left ankle. Evaluation for osteomyelitis, tuberculosis, coccidioidomycosis, and HIV was negative. Gram staining of the ankle drainage specimen revealed branching, beaded, gram-positive bacilli, suggestive of *Nocardia* species. Cultures of the ankle fluid specimen were negative for pyogenic organisms, mycobacteria, and fungi. Histopathologic evaluation of a left ankle biopsy specimen revealed nonspecific inflammation. The patient’s condition responded to a 3-week course of combination TMP-SMZ and ciprofloxacin therapy. In December 1993 she was evaluated for recurring abscesses of her left elbow and right leg with underlying osteomyelitis, and no specific pathogen was identified. The abscessed regions were debrided and, later, skin grafting was undertaken. She received empiric therapy with oxofloxacin, doxycycline, and rifampin for treatment of possible brucellosis.

Although the organism was not recovered from cultures of the mitral valve specimen, the histopathologic findings were typical of infection due to *Nocardia* species. Moreover, the clinical picture of recurrent skin and soft-tissue infections and the detection of *Nocardia* species on previous gram staining of a left ankle abscess specimen were consistent with disseminated nocardiosis. Although previous antibiotic therapy did not effect a cure for our patient, it is conceivable that the therapy impaired the recovery of organisms on culture. Given the presence of recurrent soft-tissue abscesses and the absence of pulmonary lesions in our patient, it is likely that the endocardial infection originated from the skin sites. In addition, the existence of a flail mitral valve may have predisposed her to the valvular infection.

Other than for one report of endocarditis due to *Nocardia farcinica* [3], all cases of nocardial endocarditis reported in the literature have been due to *Nocardia asteroides*, a species that is generally susceptible in vitro to antimicrobials such as sulfadiazine, minocycline, cefotaxime, ceftriaxone, ampicillin/sulbactam, imipenem/cilastatin, amikacin, and quinolones [8]. Our patient initially received combination TMP-SMZ and imipenem/cilastatin therapy for 1 month, given that this combination has shown in vitro synergy for 80% of all *N. asteroides* strains [9]. There is a dismal prognosis associated with endocarditis due to *Nocardia* species; four of six
patients reported with this condition died of this disease (table 1). Valve replacement is necessitated in cases where nocardial endocarditis develops on a prosthetic valve [7]. It is curious that none of the patients with nocardial endocarditis, including the one we described, had any pulmonary or CNS involvement. Nocardial endocarditis should be considered when infection develops in the setting of prosthetic valve placement or recurrent skin and soft-tissue infections due to these organisms, especially if cultures of blood are negative for bacterial pathogens.

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References


