Prosthetic Valve Endocarditis Caused by Acinetobacter calcoaceticus subsp. lwoffi

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Acinetobacter spp. are uncommon etiologic agents of prosthetic valve endocarditis. Two patients with Acinetobacter calcoaceticus subsp. lwoffi prosthetic valve endocarditis are described. The patients were successfully treated with antibiotics (cefotaxime sodium and gentamicin sulfate); thus, we suggest medical treatment rather than early valve replacement in this particular type of infection.

Acinetobacter spp. are gram-negative, pleomorphic bacteria which may be found among the flora of the skin, oropharynx, and perineum and are divided into two subspecies, Acinetobacter calcoaceticus subsp. lwoffi (formerly Mima polymorpha) and A. calcoaceticus subsp. anitratus (formerly Herella vaginicola) (9). Nosocomial sources of infection of Acinetobacter spp. include intravenous catheters, respiratory equipment used in conjunction with endotracheal tubes or tracheotomy, and room air humidifiers (12). Previous surgery or antimicrobial therapy and residence in a intensive care unit increase susceptibility to Acinetobacter spp. infections (3). In spite of all the above, prosthetic valve endocarditis (PVE) caused by Acinetobacter spp. is very rare, and only sporadic cases have been reported (2, 14, 17). Two patients suffering from PVE caused by A. calcoaceticus subsp. lwoffi successfully treated with antibiotics (cefotaxime sodium and gentamicin sulfate) are reported.

Case reports. Patient 1. A 47-year-old man was admitted for evaluation of fever up to 38°C which developed 1 month before admission. Six weeks before admission the patient was involved in a road accident with multiple lacerations and was treated with oral ampicillin sodium. Fourteen years before he had undergone an aortic valve replacement (McGovern valve) for severe aortic stenosis (congenital bicuspid aortic valve). On physical examination his body weight was 69 kg, his temperature was 38°C, and he had a regular heart rate of 90 beats per min and blood pressure of 110/70. Few rales were heard over the lung bases. An early diastolic murmur grade 2/6 was heard at the lower left sternal border. There were no splenomegaly, purpura, petechiae, or signs of peripheral embolization. The erythrocyte sedimentation rate was 78 mm in the first hour, the hemoglobin concentration was 12.9 g/dl, and the leukocyte count was 5,800/μm³ with 70% neutrophils. Liver and renal function tests were normal. Chest roentgenogram showed diffuse interstitial congestion, left ventricular enlargement, and the prosthetic valve. An initial echocardiogram showed sinus rhythm with prolongation of the PR interval up to 0.26 s. Left bundle branch block appeared the next day and aroused suspicion of conduction system abscesses. A two-dimensional echocardiogram showed diminished contractions of the left and right ventricle compatible with severe cardiomyopathy. Thickening at the base of the anterior aspect of the prosthetic aortic valve was interpreted as vegetation. An M-mode echocardiogram showed fluttering of the mitral leaflets, as evidence of aortic insufficiency, and severe calcifications of the aortic valve. The distance between the mitral valve and the interventricular septum was more than 2 cm, indicating low left ventricular function. Three of three blood cultures yielded A. calcoaceticus subsp. lwoffi susceptible to cefotaxime, gentamicin, chloramphenicol, colistin, and co-trimoxazole. In light of the above-described clinical and laboratory findings, a diagnosis of PVE was made; in accord with susceptibility data, intravenous treatment with cefotaxime sodium (3 g/day) and gentamicin sulfate (240 mg/day), divided into three doses at 8-h intervals, was started. After 4 days, the patient’s body temperature returned to normal, and repeated blood cultures were negative. The treatment with cefotaxime was continued for 4 weeks, and the gentamicin treatment was continued for 10 days. Two weeks after the beginning of treatment the left bundle branch block disappeared. After 12 months the patient was well without fever. Two-dimensional echocardiography showed that the left ventricular function had improved significantly, and no vegetation was seen.

Patient 2. A 52-year-old patient was admitted for persistent fever up to 38°C which started 2 months after a second aortic valve replacement because of aortic insufficiency (Bjork-Shelly valve). Eight years before, he had undergone his first aortic valve replacement (Starr-Edwards valve) for severe aortic stenosis due to rheumatic fever. Two weeks before admission the patient underwent tooth extraction without antibiotic prophylaxis. On physical examination, the patient’s body weight was 71 kg, he had a temperature of 38°C, his blood pressure was 130/70, and his heart rate was 90 beats per min and regular. There were no signs of heart failure. An early diastolic murmur grade 2/6 was heard along the left sternal border. The liver was not enlarged. The spleen was palpated 2 cm below the left costal margin. There were no purpura, petechiae, or signs of peripheral embolization. The erythrocyte sedimentation rate was 74 mm in the first hour, the hemoglobin concentration was 11 g/dl, and the leukocyte count was 11,500/μm³ with a normal differential count. Liver and renal function tests were all normal. Chest X ray showed interstitial pulmonary congestion and left ventricular enlargement. An electrocardiogram showed normal sinus rhythm. An M-mode echocardiogram revealed decreased opening of the mitral valve, indicating poor left ventricular function. Two-dimensional echocardiography showed flutter of the mitral leaflets and thickening of the prosthetic aortic valve, interpreted as vegetation. Three of

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three blood cultures yielded *A. calcoaceticus* subsp. *lwoffi* organisms that were susceptible to cefotaxime, gentamicin, colistin, tetracycline, and co-trimoxazole. A diagnosis of PVE was made, and intravenous treatment with cefotaxime sodium (1 g three times a day) and gentamicin sulfate (80 mg three times a day) was started. From day 3 of treatment blood cultures became negative, and a week later the fever disappeared. Three weeks later, the spleen was no longer palpable. Cefotaxime was given for 4 weeks, and gentamicin was given for 10 days. On follow-up, 12 months later, there was no fever or signs of heart failure, no vegetations were seen on an echocardiogram, and the patient returned to work.

Blood cultures were performed with tryptic soy broth and thiglycolate broth (30-ml, model 6B BACTEC bottle; Johnston Laboratories, Inc., Towson, Md.). We used blood agar plates (tryptic soy agar plus 5% defibrinated sheep blood) and MacConkey agar plates (Hy Laboratories, Kiryat Weizman, Rehovot, Israel). Colonies on blood and MacConkey agars were 1 mm in diameter at 24 h, white, smooth, and nonhemolytic. They grew at different temperatures in the range of 25 to 40°C. Morphologically, gram-negative short and long coccobacilli were observed in pairs. The bacteria were nonmotile and showed the following characteristics: catalase positive, oxidase negative, positive growth on MacConkey agar, nitrate reduction negative, urease negative, citrate negative. The bacteria were tested for fermentation with 10% glucose, fructose, galactose, mannose, shannose, xylose, lactose, sucrose, melose, manniol, and lactose; no acid was produced from those carbohydrates. The above-described qualities indicated that the bacteria isolated in repeated blood cultures in the two patients were *A. calcoaceticus* subsp. *lwoffi* (10). In vitro susceptibility was tested by the method of Bauer et al. (1). Disks contained gentamicin (11 μg) and cefotaxime (30 μg). The antibacterial activity of the serum of both patients against the infective microorganism was determined by the serum bactericidal test (15) (Hy Laboratories). Serum bactericidal titers were determined 30 min before the first morning dose of cefotaxime and gentamicin for the trough level and 30 min after this dose for the peak level. The peak serum bactericidal titers in both patients ranged from 1:32 to 1:128, and trough levels ranged from 1:16 to 1:128.

**Discussion.** The incidence of PVE after cardiac valve replacement is reported between 1 and 9.4% and carries a high mortality rate ranging between 35 and 69% (5, 8, 14, 16). The organisms most frequently involved in PVE are *Staphylococcus* spp. and *Streptococcus* spp. (7, 8, 11, 14, 16). *Acinetobacter* spp. have only rarely been associated with PVE; only four cases have been reported in the medical literature (2, 13, 17). Two of these cases were caused by *A. calcoaceticus* subsp. *lwoffi* (2, 17).

The time interval between the valve replacement and the occurrence of *Acinetobacter* PVE in the four reported cases (2, 13, 17) and the present two patients (Table 1) shows that *Acinetobacter* PVE may occur both early and late after valve replacement.

Comparison of other features of the cases of *Acinetobacter* PVE recorded in the literature (Table 1) shows that there is no preference for a specific valve type or location in the patients with *Acinetobacter* PVE. Three patients had PVE in the prosthetic mitral valve, two patients had PVE in the prosthetic aortic valve, and one patient had PVE after double valve replacement, probably in both mitral and aortic prosthetic valves (Table 1).

<table>
<thead>
<tr>
<th>Reference</th>
<th><em>A. calcoaceticus</em> subspecies</th>
<th>Age of patient (yr)</th>
<th>Affected prosthetic valve(s)</th>
<th>Onset of PVE after valve replacement</th>
<th>Treatment</th>
<th>Valve replacement for PVE</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stein et al. (13), 1966</td>
<td><em>lwoffi</em></td>
<td>40</td>
<td>Mitral</td>
<td>3 days</td>
<td>Chloramphenicol, penicillin, and erythromycin</td>
<td>No</td>
<td>Survival</td>
</tr>
<tr>
<td>Stein et al. (13), 1966</td>
<td><em>lwoffi</em></td>
<td>38</td>
<td>Mitral</td>
<td>1 day</td>
<td>Chloramphenicol, methicillin, and penicillin</td>
<td>No</td>
<td>Survival</td>
</tr>
<tr>
<td>Yeh et al. (17), 1967</td>
<td><em>lwoffi</em></td>
<td>56</td>
<td>Mitral</td>
<td>3 yr</td>
<td>Chloramphenicol, methicillin, and colistin</td>
<td>No</td>
<td>Death</td>
</tr>
<tr>
<td>Block et al. (2), 1970</td>
<td><em>lwoffi</em></td>
<td>48</td>
<td>Mitral and aortic</td>
<td>31 mo</td>
<td>Penicillin, kanamycin, and ampicillin</td>
<td>No</td>
<td>Survival</td>
</tr>
<tr>
<td>Present study (patient 1)</td>
<td><em>lwoffi</em></td>
<td>47</td>
<td>Aortic</td>
<td>14 yr</td>
<td>Cefotaxime and gentamicin</td>
<td>No</td>
<td>Survival</td>
</tr>
<tr>
<td>Present study (patient 2)</td>
<td><em>lwoffi</em></td>
<td>52</td>
<td>Aortic</td>
<td>2 mo</td>
<td>Cefotaxime and gentamicin</td>
<td>No</td>
<td>Survival</td>
</tr>
</tbody>
</table>
rates range, according to different authors, between 35 and 69% (8, 11, 14, 16). Some authors (8, 14) suggest that in view of the high mortality rate of PVE, early replacement of the infected prosthesis should be considered in all patients with PVE, except those with streptococcal PVE. According to our two patients and the four cases reported previously (2, 13, 17), Acinetobacter PVE may have a better prognosis than other bacterial PVE. Intensive antibiotic treatment, rather than early valve replacement, is indicated, provided the functional class of heart failure is not advanced.

LITERATURE CITED