Prosthetic Valve Endocarditis Due to *Veillonella dispar*: Successful Medical Treatment Following Penicillin Desensitization

Serious human disease due to *Veillonella* species is uncommon. We report the fifth case of well-documented infectious endocarditis in which *Veillonella* species was the sole pathogen. Optimal treatment, particularly for patients who are allergic to penicillin, is not well established.

A 56-year-old man presented to the hospital with a 2-week history of fatigue and a 1-week history of intermittent fever; he had undergone mechanical mitral and aortic valve replacements 3 years before presentation. He reported that he had collapsed 30 years before presentation and that this episode had been attributed to anaphylaxis following oral penicillin therapy.

After oral desensitization (skin testing was not performed before presentation) and a mobile vegetation on the mitral valve. Culture of three separate blood samples yielded *Veillonella dispers* that was susceptible to cefoxitin, chloramphenicol, metronidazole, and penicillin. The MIC of penicillin was 0.125 mg/L, and the MBC was 0.25 mg/L. Transesophageal echocardiography demonstrated a 10 x 16-mm sessile mass that was susceptible to cefoxitin, chloramphenicol, and vaginal flora in humans. *Veillonella* species are rarely identified as the sole pathogens in serious human infection and are usually considered “generally not important pathogens” [2].

We describe the fifth reported case of well-documented infectious endocarditis in which *Veillonella* species was the sole pathogen [3–6, table 1]. *Veillonella* species have also been isolated from injection drug users with polymicrobial endocarditis [7]. In one other case, *Veillonella parvula* was isolated from a lung abscess in a patient with echocardiographic vegetations, but blood cultures were negative [8].

All previously reported cases of endocarditis that were caused by *Veillonella* species alone were due to either *Veillonella alcalescens* or V. dispers. Two of four previously described patients had an infected prosthetic valve. Two reports described unusually indolent presentations of endocarditis.

Three of four previously described patients with endocarditis due to *Veillonella* species alone underwent valve replacement. The fourth was cured medically after ~18 months of therapy in 1946 [3]. Our patient was cured without surgical intervention.

Antibiotic regimens used to treat previously described patients were quite variable. There is limited experience with the use of metronidazole, either alone or in combination, in the treatment of endocarditis. Extensive experience with the use of penicillin in the treatment of endocarditis along with the documented susceptibility of the organism made it the drug of choice in our case in spite of the fact that our patient had a history of a serious reaction to penicillin. The success of medical therapy alone in our case suggests that penicillin should be considered the drug of choice in patients with endocarditis due to *Veillonella* species and reinforces the role of desensitization when penicillin is the preferred drug for the treatment of a serious infection.

Table 1. Summary of four previously reported cases of endocarditis due to *Veillonella* species alone.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sex/age (y)</th>
<th>Infected valve</th>
<th><em>Veillonella</em> species isolated</th>
<th>Site of specimen obtained for culture</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>[3]</td>
<td>M/35</td>
<td>Mitral native</td>
<td><em>V. alcalescens</em></td>
<td>Blood</td>
<td>Sulfamamide, penicillin, heparin, and para-aminobiphenyl tartrate; no surgery (18 mo total)</td>
</tr>
<tr>
<td>[4]</td>
<td>M/60</td>
<td>Aortic native</td>
<td><em>V. alcalescens</em></td>
<td>Valve*</td>
<td>Cephalin and gentamicin (2 w); oral penicillin V for 6 mo; surgery</td>
</tr>
<tr>
<td>[5]</td>
<td>F/57</td>
<td>Mitral prosthetic</td>
<td><em>V. dispers</em></td>
<td>Blood†</td>
<td>Ampicillin (2 w iv) and metronidazole (2 w oral); oral clindamycin; surgery</td>
</tr>
<tr>
<td>[6]</td>
<td>M/51</td>
<td>Mitral prosthetic</td>
<td><em>V. alcalescens</em></td>
<td>Blood†</td>
<td>Penicillin G (20–40 million units per d for 6 w iv); surgery</td>
</tr>
</tbody>
</table>

*Culture of blood specimens was negative.
†Culture of valve specimens was negative.

On physical examination he had a grade 1 systolic murmur, crisp mechanical heart sounds, and no peripheral findings of endocarditis. Culture of three separate blood samples yielded *Veillonella dispers* that was susceptible to cefoxitin, chloramphenicol, clindamycin, metronidazole, and penicillin. The MIC of penicillin was 0.125 mg/L, and the MBC was 0.25 mg/L. Transesophageal echocardiography demonstrated a 10 x 16-mm sessile mass and a mobile vegetation on the mitral valve.

After oral desensitization (skin testing was not performed because one of the recommended reagents was not available) [1], iv penicillin (18 million units per day) was administered for 6 weeks. The serum bactericidal level was 1:32 before penicillin was administered and 1:256 after the drug was administered. After treatment was completed, a transesophageal echocardiogram revealed complete resolution of the vegetations. A clinical examination was normal and repeated blood cultures were negative 6 months later.

*Veillonella* species are part of the normal oral, gastrointestinal, and vaginal flora in humans. *Veillonella* species are rarely identified as the sole pathogens in serious human infection and are usually considered “generally not important pathogens” [2].

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**Stan Houston, Dylan Taylor, and Robert Rennie**

*Divisions of Infectious Diseases and Cardiology, Department of Medicine, University of Alberta Hospitals, Edmonton, Alberta, Canada*
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 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 2. In 1994, a 71-year-old male presented with an extruded 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-

References


