Aerococcus viridans is an infrequent human pathogen and few cases of infective endocarditis have been reported. A case involving a 69-year-old man with colon cancer and hemicolecotomy 14 years previously, without recurrence, is reported. A diagnosis of native mitral valve endocarditis was established on the basis of clinical presentation, characteristic echocardiographic findings and pathological specimen examination after urgent valve replacement. A viridans endocarditis appears to be particularly virulent, requiring a surgical approach in four of 10 cases reported and death in one of nine. Given the aggressive nature of A viridans endocarditis and the variable time to diagnosis (a few days to seven months), prompt recognition of symptoms and echocardiography, in addition to blood cultures, should be performed when symptoms persist.

Key Words: A viridans; Endocarditis; Mitral valve

Aerococcus viridans is a microaerophilic, Gram-positive, catalase-negative coccus with a strong tendency toward tetrad formation. It has growth characteristics similar to that of streptococci and enterococci. Aerococci are environmental isolates ubiquitously found in the air of housing premises (hospitals, schoolrooms, factories, offices), dust, raw vegetables, animals and animal products, as well as human skin (1). Three Aerococcus species have been implicated as rare pathogens in humans. Aerococcus urinae causes endocarditis (2-11), septicaemia, urinary tract infections/uresepsis, pyelonephritis, spondylodiscitis, spontaneous bacterial peritonitis, peritonitis, lymphadenitis and soft tissue infections (12-25). Aerococcus sanguinicola causes bacteremia (26), endocarditis (26), urosepsis (26) and cholecystitis (26). A viridans causes urinary tract infections (27,28), bacteremia, endocarditis (Table 1), para-aortic abscess, meningitis, spondylodiscitis and septic arthritis (29-44). Risk factors for A viridans systemic infections (bacteremia/endocarditis) have not yet been fully elucidated; however, granulocytopenia, oral mucositis, prolonged hospitalization, previous antibiotic therapy, invasive procedures and implantation of foreign bodies have been associated with severe infections with A viridans (36). Previous valvular abnormalities, such as rheumatic valve disease, have been described as predisposing conditions for infective endocarditis (38). Reports in which no obvious immunosuppressive factor could be found are rare. This was the case in our patient.

Studies from clinical cases and specimens demonstrated that strains are generally susceptible to β-lactam antimicrobials (eg, penicillin, ampicillin, amoxicillin-clavulanic acid) and vancomycin (40-44). On the other hand, resistance was found to quinolones, tetracycline, clindamycin and streptomycin (40-44); penicillin resistance was reported in two instances to levels >250 µg/mL (44). Resistance to gentamicin has also been reported occasionally for human clinical strains (39,41).

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otherwise normal on echocardiography. Two different sets of blood cultures grew *A. viridans*, with sensitivities to ceftriaxone (0.25 µg/mL) and penicillin (0.03 µg/mL).

Surgery was performed six days after admission because of general deterioration, evidence of moderate to severe mitral regurgitation and fenestration of the anterior mitral valve leaflet. Blood-based cardio-pulmonary bypass was administered for myocardial protection. Through a left atriotomy, direct visualization of the mitral valve revealed large vegetations on the anterior and posterior leaflets with marked anatomical destruction of the valve (Figure 1A). Closer examination showed marked distortion of the valve apparatus, with a fenestration of the anterior leaflet (Figure 1B and 1C). The annulus was preserved. Mitral valve replacement using a bioprosthetic valve was performed. Care was taken to preserve the papillary-annular continuity to maintain ventricular function by saving as much of the mitral subvalvular apparatus as possible. Both the surgery and the postoperative course were uneventful. On postoperative day 7, he was discharged home without complications. A six-week treatment of penicillin was provided via pump through an indwelling intravenous line. Repeat postoperative blood cultures were negative for any growth.

Gross pathological examination (Figure 1) of the mitral valve revealed a firm thrombus, 2.0 cm in its greatest dimension, attached.

**Table 1**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, years/sex</th>
<th>Clinical presentation</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Complications</th>
<th>Resistant</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>49/M</td>
<td>Subacute (7 months): Fever; polymyalgia; mild AR murmur</td>
<td>14×15 mm AV veg on TEE</td>
<td>Ampicillin/amikacin</td>
<td>AV thickening/mild AR, Medical Rx. No relapse at 18 months</td>
<td>Quinolones/aminoglycosides</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>62/M</td>
<td>Subacute (1 month): Fever; weight loss; dyspnea/MR murmur; Janeway lesion; splenomegaly</td>
<td>13×12 mm veg on anterior mitral leaflet</td>
<td>Ceftriaxone/amikacin</td>
<td>Splenectomy for splenic abscesses. MVR. No relapse at 12 months</td>
<td>Quinolones</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>40/M</td>
<td>Subacute (3 weeks): Fever/fatigue; arthralgias; Osler nodes</td>
<td>14×11 mm veg at aortic cusp on TEE</td>
<td>Penicillin G/gentamicin</td>
<td>Medical Rx. No relapse at 38 months</td>
<td>Penicillin/amikillin/ceftaxine/gentamicin</td>
<td>40</td>
</tr>
<tr>
<td>4</td>
<td>45/F</td>
<td>Acute (3 days): Fever; dyspnea; ataxia; Janeway lesion; MR murmur; CHF</td>
<td>7×6 mm veg on anterior leaflet and posterior leaflet 5×3 mm</td>
<td>Penicillin G/gentamicin</td>
<td>MVR</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>10/M</td>
<td>Subacute (1 month): Fever; dyspnea; arthralgia; hematuria; clubbing; MR murmur; CHF</td>
<td>Veg on mitral leaflet</td>
<td>Norfloxacin/amikacin</td>
<td>Compensated MR. Medical Rx.</td>
<td>Penicillin/amikillin/ceftaxine/gentamicin</td>
<td>41</td>
</tr>
<tr>
<td>6</td>
<td>28/M</td>
<td>Subacute (6 months): Fever; rheumatic complaints; hematuria; AR murmur</td>
<td>Flail aortic leaflet with veg</td>
<td>Penicillin G/gentamicin</td>
<td>AoVR. No relapse at 6 months</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>44/M</td>
<td>Acute (few days): Fever; hematuria; splenomegaly; AR murmur</td>
<td>Not reported</td>
<td>Penicillin/streptomycin</td>
<td>CHF 6 weeks post discharge. Sudden death</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>54/M</td>
<td>Subacute (4 months): Fever; back pain; systolic murmur</td>
<td>Billowing leaflet</td>
<td>Penicillin</td>
<td>Medical Rx. No relapse at 10 weeks</td>
<td>Sulfonamide</td>
<td>43</td>
</tr>
<tr>
<td>9</td>
<td>58/M</td>
<td>Acute (4 days): Fever; pyuria; hematuria; altered LOC; diastolic murmur</td>
<td>10 mm veg on noncoronary cusp of AV on TEE</td>
<td>Cefotaxime/vancocycin</td>
<td>Medical Rx. Well at 3 months outpatient follow-up</td>
<td>Chloramphenicol/clindamycin/erythromycin/gentamicin/trimethoprim-sulfamethoxazole</td>
<td>39</td>
</tr>
<tr>
<td>10</td>
<td>44/F</td>
<td>Subacute (2 weeks): Fever; AF; systolic murmur at AV area</td>
<td>Rheumatic AV and MV; moderate MS; oscillating mass 11×10 mm, MG 35 mmHg across AV on TEE</td>
<td>Ampicillin/sublactam/gentamicin</td>
<td>Enlarging veg (21×10 mm) causing AV obstruction s/p 3 weeks of oral antibiotics requiring AoVR/MVR</td>
<td>38</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 1** A *Intraoperative view of the mitral valve through a left atriotomy*. Vegetations (arrows are seen on the anterior and posterior valve leaflets). B Resected specimen. The anterior valve leaflet is perforated (arrow). Large vegetations are seen on the anterior valve leaflet and the Pi segment of the posterior leaflet. Note the intense inflammation and fibrotic reaction contributing to the distortion of the valve apparatus. C Ventricular aspect of the resected mitral valve showing the perforation of the anterior leaflet (arrow).
to the ventricular aspect of a somewhat thickened valve. The thrombotic lesion appeared to have involved the valve, creating full-thickness valvular perforations measuring 1.0 cm and 0.7 cm in its greatest dimension (Figures 1B and 1C). Histological assessment of the valve revealed a prominent fibrinous exudate with neutrophils and tissue destruction, as well as areas of organization and fibrosis (Figures 2A to 2D). Furthermore, microscopy revealed a prominent population of Gram-positive cocci in clusters (Figure 2E), consistent with the A. viridans found in the patient’s blood. Necrotic debris and fibrotic destruction of the valve are shown in Figure 2F.

**DISCUSSION**

We identified 10 previously reported cases of A. viridans endocarditis (Table 1). In all reported cases, vegetation were identified on the mitral or aortic valves. One case resulted in congestive heart failure and death after discharge. In three cases, the isolated strain of A. viridans was resistant to penicillin or quinolones. The laboratory findings were often nonspecific, with elevation in markers of inflammation (C-reactive protein, erythrocyte sedimentation rate and a mild leukocytosis). Blood cultures and echocardiography provided the diagnosis in all instances reported.

As in the present case, the symptoms of endocarditis are often nonspecific. From the onset of symptoms to first medical contact, a delay of at least five weeks was reported. This delay likely contributed to the destruction and perforation of the mitral valve leaflet.

**CONCLUSION**

A viridans endocarditis appears to be particularly virulent, despite its often long latency period (subacute 73% [eight of 11 cases] and acute 23% [three of 11 cases]) and comparatively lower mortality rates (9% [one of 11 cases]) as opposed to left-sided, native valve Staphylococcus aureus infective endocarditis (30% to 40%). It nonetheless required a surgical approach in 45% (five of 11) of the cases reported. Given the aggressive nature of A. viridans endocarditis and the variable time to diagnosis (a few days to seven months), prompt recognition of symptoms, blood cultures and echocardiography, including transoesophageal echocardiography, should be performed when blood cultures are positive and suspicion index is high.

**REFERENCES**

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