Valve Replacement in Patients with Active Infective Endocarditis

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SUMMARY Eleven of 138 patients with infective endocarditis (IE) who underwent cardiac valve replacement for IE during a 12½-year period had active IE. Eight of the 11 (all with aortic IE) had positive blood cultures within 48 hours preoperatively; six of the eight had positive Gram stains and cultures of the excised cardiac tissue. All 11 patients had Class IV cardiac functional disability (New York Heart Association classification) at the time of surgery. Staphylococci (three patients with Staphylococcus aureus and one with S. epidermidis) were the most frequent isolates. Three patients died; two of these three deaths occurred in patients who had a sudden onset preoperatively of severe aortic regurgitation and heart failure. In one patient (S. epidermidis infection) prosthetic valve endocarditis developed. Cardiac valve replacement may be performed successfully in patients with active IE even when blood cultures are positive in the immediate perioperative period. The hemodynamic status of patients with IE should be the determining factor in the timing of cardiac valve replacement, rather than the activity of the infection or the length of preoperative antimicrobial therapy. A radical surgical procedure may be necessary in patients with myocardial or aortic abscesses in whom conventional aortic valve replacement is not possible.

Materials and Methods

Our criteria for IE have been described.26 Our criteria for active IE were patients who were still receiving antimicrobial therapy for IE together with at least one of the following: 1) blood cultures positive within 48 hours preoperatively, or 2) fever and new embolic phenomena within 48 hours preoperatively.

Criteria for the functional classification of congestive heart failure were according to the New York Heart Association.26 Mortality was defined as death occurring within two months of cardiac valve replacement. Follow-up ranged from 16 months to 14 years.

Of the 138 patients with IE who underwent cardiac valve replacement during this period, 11 (8%) had active IE. Ten of these 11 patients had received at least one course of antimicrobial therapy elsewhere for IE before admission to Mayo Clinic hospitals. Urgent cardiac valve replacement was necessary in these 11 patients before antimicrobial therapy could be completed, because of severe, progressive Class IV cardiac disability that was unresponsive to medical therapy.

The microbiologic cause of IE in these 11 patients and in the remaining 127 patients and the respective class of heart failure present at surgery are shown in table 1. Staphylococci (three patients with S. aureus and one with S. epidermidis) were the most frequent isolates from patients with active IE. Eight of the 11 patients (all with aortic IE) had positive blood cultures within 48 hours preoperatively. Six of these eight had positive Gram stains and cultures of the excised cardiac tissue. Of the three patients (all with mitral IE) with perioperative negative blood cultures, all had received long-term antimicrobial therapy elsewhere. Use of the antimicrobials was discontinued on admission to the Mayo Clinic, and the patients had relapses clinically. Cultures of the excised valve tissue in these three patients were negative, but in all three patients gram-positive cocci were noted on staining of the vegetations.

The average duration of preoperative antimicrobial therapy at the Mayo Clinic is shown in table 2. All patients with Class II and III cardiac disability had
TABLE 1. Microorganisms Involved and NY Heart Association Functional Classification in 138 Patients With IE

<table>
<thead>
<tr>
<th>Microorganisms</th>
<th>Class II</th>
<th>Class III</th>
<th>Inactive</th>
<th>Active</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viridans streptococci</td>
<td>34</td>
<td>4</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>Group D streptococci</td>
<td>16</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>S. epidermidis</td>
<td>7</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>S. aureus</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Other organisms</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Negative blood cultures</td>
<td>22</td>
<td>3</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>88</td>
<td>15</td>
<td>24</td>
<td>11</td>
</tr>
</tbody>
</table>

completed antimicrobial therapy preoperatively. Six patients with Class IV disability and inactive IE were still receiving antimicrobial therapy at the time of surgery. The average duration of preoperative antimicrobial therapy was longer in Class IV patients with inactive IE than in those with active IE; however, the number of patients is too small to permit accurate statistical evaluation. In patients with active IE, the average duration of therapy was the same in survivors and in those who died. Postoperatively, all patients with active IE and those with inactive IE who had not completed antimicrobial therapy preoperatively received at least four weeks of parenteral antimicrobial therapy. None of the patients received oral antimicrobial agents after the completion of parenteral therapy.

Results

The mortality was higher among patients with Class IV disability and active IE (three of 11 patients, 27%) than among patients with Class IV status and inactive IE (three of 24 patients, 13%), but the number of patients is too small to reach statistical significance in mortality between the two groups of patients. The higher mortality among patients with active IE may be related to the higher frequency of occurrence of sudden onset of severe aortic insufficiency and myocardial abscesses in this group compared with patients with inactive IE. Two of the three deaths among patients with active IE occurred in patients who had the sudden onset preoperatively of severe aortic regurgitation and congestive heart failure (table 3). One of these two patients (with S. aureus infection) had aortic wall and myocardial abscesses and died seven days postoperatively from complications of heart failure caused by valve dehiscence. Postmortem cultures were negative. The other patient (with Pseudomonas cepacia infection) with sudden-onset severe aortic regurgitation and congestive heart failure died on the day of operation from severe heart failure and cardiac arrest. The third patient (with S. epidermidis IE) who died had multiple myocardial abscesses and experienced early prosthetic valve endocarditis. The infected prosthetic valve was excised, and a second Starr-Edwards valve was implanted in the ascending aorta. The patient died approximately four months later of cardiomyopathy and heart failure. The cardiomyopathy was thought to be caused by the previous aortic incompetence and resultant heart failure. At postmortem examination no evidence of active infection was noted, and postmortem cultures were negative. Among patients with Class IV disability and inactive IE, three patients had sudden onset of severe aortic insufficiency. One of these three died postoperatively of cardiac dysrhythmia and circulatory collapse. The two survivors underwent surgery one and two days after the onset of acute heart failure. The patient who died underwent surgery five days after sudden onset of severe heart failure, and this delay before surgery may have contributed to death postoperatively. None of the patients with functional Class IV status and inactive IE had myocardial abscess.

The single case of prosthetic valve endocarditis occurred in the patient with S. epidermidis IE. Two additional patients required replacement of aortic prostheses because of subsequent prosthetic valve dehiscence. In a patient with viridans streptococcal IE, an aortic regurgitant murmur was noted on the 17th postoperative day; the murmur did not progress in severity. Class II cardiac disability was present and was treated medically. The aortic prosthesis was replaced three months after the initial operation. At surgery, partial dehiscence of the valve was present. Gram staining and culture of the prosthetic valve were negative. Four years after reoperation, the patient had Class I functional status. In the other patient (S. aureus infection) who required reoperation, an aortic regurgitant murmur developed six weeks postoperatively. The aortic prosthesis was replaced at this time. Partial valve dehiscence was noted at surgery; Gram staining and culture of the valve were negative. Class I status was present at follow-up two years after reoperation. The patient died at home three and one-half years later, presumably from a dysrhythmia. Among patients with inactive IE, three of 24 patients (12.5%) with Class IV, one of 15 (7%) with Class III, and three of 88 (3%) patients with Class II disability required subsequent replacement of cardiac valve prostheses because of valve dehiscence.

TABLE 2. Mean Duration of Preoperative Antimicrobial Therapy and Mortality by New York Heart Association Functional Classification

<table>
<thead>
<tr>
<th>NYHA Classification</th>
<th>Mean duration of treatment, days (no. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lived</td>
</tr>
<tr>
<td>II</td>
<td>24 (81)</td>
</tr>
<tr>
<td>III</td>
<td>26 (14)</td>
</tr>
<tr>
<td>IV</td>
<td></td>
</tr>
<tr>
<td>Inactive IE</td>
<td>33 (21)</td>
</tr>
<tr>
<td>Active IE</td>
<td>5 (8)</td>
</tr>
</tbody>
</table>
Table 3. Patients With Active Infective Endocarditis

<table>
<thead>
<tr>
<th>Patient no., sex, and age (yr)</th>
<th>Valve</th>
<th>Organism</th>
<th>PVE*</th>
<th>Postop valve dehiscence, re-replacement</th>
<th>Outcome</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, F, 57</td>
<td>Aortic</td>
<td>Viridans strep</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Follow-up 3 yr, Class I functional status</td>
</tr>
<tr>
<td>2, M, 15</td>
<td>Aortic</td>
<td>S. epidermidis</td>
<td>Yes</td>
<td>Yes</td>
<td>Died†</td>
<td>Myocardial and aortic annulus abscesses, 5 days postop—valve dehiscence. Reoperation 14 days postop. Aortic prosthesis, ascending aorta; saph vei anast with R cor art. and L ant. desc cor art. to aorta, superior to prost valve. Patient died 4 mo after second operation from severe failure due to cardiomyopathy. Postmortem—no infection, cultures negative</td>
</tr>
<tr>
<td>3, M, 59</td>
<td>Aortic</td>
<td>P. cepacia</td>
<td>No</td>
<td>No</td>
<td>Died</td>
<td>Sudden onset preoperatively of severe aortic regurgitation. Patient died immediately postop. Postmortem—cultures positive</td>
</tr>
<tr>
<td>4, M, 17</td>
<td>Aortic</td>
<td>S. aureus</td>
<td>No</td>
<td>Yes</td>
<td>Lived</td>
<td>Postoperative Class II disability. Valve dehiscence occurred 6 wk postop. Re-replacement of aortic prosthesis. Follow-up 2 yr later—Class II disability. Died elsewhere 3½ yr after surgery, presumably from dysrhythmia</td>
</tr>
<tr>
<td>5, M, 72</td>
<td>Aortic</td>
<td>Viridans strep</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Follow-up 1 yr after surgery, Class II disability</td>
</tr>
<tr>
<td>6, F, 25</td>
<td>Aortic</td>
<td>Viridans strep</td>
<td>No</td>
<td>Yes</td>
<td>Lived</td>
<td>Valve dehiscence with aortic regurgitant murmur 17 days postop. Stable Class II disability. Valve replaced 3 mo after surgery. Follow-up 4 yr, no symptoms</td>
</tr>
<tr>
<td>7, M, 34</td>
<td>Aortic</td>
<td>S. aureus</td>
<td>No</td>
<td>No</td>
<td>Died</td>
<td>Sudden onset preoperatively of severe aortic regurgitation. Abscesses of aortic annulus. Died 7 days postop from cardiac arrest. Postmortem—valve dehiscence, cultures negative</td>
</tr>
<tr>
<td>8, M, 33</td>
<td>Aortic</td>
<td>S. aureus</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Follow-up 2 yr, no symptoms</td>
</tr>
<tr>
<td>9, F, 44</td>
<td>Mitral</td>
<td>Gram-pos cocci</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Ruptured chordae at surgery. Follow-up 2 yr, no symptoms</td>
</tr>
<tr>
<td>10, F, 16</td>
<td>Mitral</td>
<td>Gram-pos cocci</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Ruptured chordae at surgery. Brain abscess drained 6 mo postop. Cultures negative. Follow-up 3 yr, no symptoms</td>
</tr>
<tr>
<td>11, F, 34</td>
<td>Mitral</td>
<td>Gram-pos cocci</td>
<td>No</td>
<td>No</td>
<td>Lived</td>
<td>Ruptured chordae at surgery. Follow-up 3 yr, no symptoms</td>
</tr>
</tbody>
</table>

*PVE = prosthetic valve endocarditis.
†Died 4 months after second operation.

Discussion

From our data and those of others, it is clear that cardiac valve replacement can be accomplished successfully in patients who have active endocarditis and severe heart failure. Few data are available, however, on cardiac valve replacement in patients with positive blood cultures in the perioperative period. Our experience and that of others suggest that the risk of valve dehiscence and prosthetic valve endocarditis in these patients may be higher, especially if myocardial or aortic abscesses are present, than in patients who have inactive IE. However, in patients with severe heart failure complicating IE, procrastination in cardiac valve replacement in an attempt to stabilize heart failure by medical therapy and to complete a course of antimicrobial therapy preoperatively usually results in death from cardiac failure.

The risk of prosthetic valve endocarditis can be minimized by meticulous surgical debridement of friable, necrotic material. However, in patients with myocardial or aortic wall abscesses, infection may be so extensive that conventional aortic valve replacement may not be feasible technically. In both of our patients who had abscesses, valve dehiscence developed in the early postoperative period; one had prosthetic valve endocarditis. In these patients, a radical surgical approach may offer the only possibility of stabilizing, at least temporarily, cardiac hemodynamics, and it may allow additional time for antimicrobial therapy and for healing of cardiac and aortic tissue.

One surgical approach to these patients was reported in detail earlier. Briefly, the infected natural or prosthetic valve is excised and the area is debrided. A new aortic prosthesis is then implanted in the ascending aorta. The coronary ostia are closed. Coronary artery perfusion is reestablished by a segment of autogenous saphenous vein anastomosed end-to-side to the right coronary artery and end-to-side to the aorta distal to the prosthesis. Another segment of vein is used to construct a "Y" extension from the right cor-
Coronary artery graft to the left anterior descending coronary artery.

Since this initial report, we have had experience with two additional patients who had similar operations. In these two patients, minimal cardiac symptoms are present at five and 12 months postoperatively. Experience with this surgical procedure is limited, and radical surgery of this type should be reserved for those patients with myocardial or aortic wall abscesses in whom conventional aortic valve replacement is not possible. The long-term hemodynamic consequences of this surgical procedure are not known. Of critical importance in these patients is whether the vein grafts will continue to provide adequate coronary artery perfusion. So far, neither of the surviving patients has had angina (nor did the patient who died four months postoperatively). In these patients, if necessary, the aortic prosthesis could later be reimplemented in the usual location after sufficient time has elapsed to permit healing of the infected aortic annulus. More experience is needed for fuller evaluation of this surgical approach.

From our data we conclude that 1) cardiac valve replacement may be performed successfully in patients with active IE even when blood cultures are positive in the immediate perioperative period; 2) the hemodynamic status of patients with IE should be the determining factor in the timing of cardiac valve replacement, rather than the activity of the infection or the length of preoperative antimicrobial therapy; and 3) a radical surgical procedure may be necessary in patients with myocardial or aortic root abscesses in whom conventional aortic valve replacement is not possible.

References
Valve replacement in patients with active infective endocarditis.
W R Wilson, G K Danielson, E R Giuliani, J A Washington, 2nd, P M Jaumin and J E Geraci

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