Valve Surgery in Acute Rheumatic Heart Disease*

One- to Four-Year Follow-up

M. Habbab, M.D.; and M. Al Zaibag, M.D.

Six patients with acute rheumatic carditis and intractable left ventricular failure, all in class 4 NYHA classification, underwent successful valve surgery combined with medical therapy. Two-dimensional echocardiography and Doppler studies showed all of them to have left ventricular dilatation, with good systolic function, together with severe mitral regurgitation; two patients also had severe aortic regurgitation. Over a mean follow-up period of two years, no mortality was recorded, and all six patients were in NYHA class 1-2. We conclude that valve replacement is not contraindicated in acute rheumatic carditis and may be preferable to repair. (Chest 1988; 94:830-33)

Acute rheumatic fever remains prevalent in Saudi Arabia and is an important cause of morbidity and mortality among the younger population. Rarely, it presents with congestive heart failure, due to severe cardiac valvular insufficiency, and refractory to medical therapy.

There is a firmly held general belief that active rheumatic carditis is a relative, if not an absolute contraindication to intracardiac surgery. However, a few reports have indicated that valve replacement in acute rheumatic fever may provide dramatic relief of symptoms in these patients.

The successful management of six cases of acute rheumatic carditis was reported, with the combination of medical therapy and surgical valve replacement or repair during the active phase of the disease, with a mean follow-up period of two years.

Patients and Methods

Between 1983 and 1986, acute rheumatic carditis was diagnosed in 49 patients in Riyadh Military Hospital. The diagnosis of acute rheumatic fever was based upon the modified Jones criteria and the presence of rheumatic carditis confirmed by the following factors: the appearance of a significant new murmur or change in the character of a previously present murmur; the development of a pericardial rub; a significant increase in the cardiac size; the macroscopic confirmation of active rheumatic carditis at operation; and histopathologic evidence of rheumatic carditis in the cardiac tissue specimen.

Medical therapy failed to control the severe left ventricular failure in six patients. The clinical characteristics of these patients are shown in Table 1.

All six patients had a raised erythrocyte sedimentation rate (ESR) ranging between 47 and 112, with a mean value of 79. The ASOT titer was also elevated, with a mean value of 386 units/ml, (range 250 to 600; normal<200). The electrocardiogram was abnormal in each patient: patient 2 was in fast atrial fibrillation; patients 1, 3, 4, 5, and 6 were in sinus tachycardia, and patients 4 and 5 also had prolongation of the PR interval. All patients had left atrial enlargement and left ventricular hypertrophy; patient 5 also had right ventricular hypertrophy. The chest x-ray film showed cardiomegaly with pulmonary congestion and left atrial enlargement in all patients.

Cross-Sectional Echocardiography and Doppler Study

On admission, during the illness and at six-month intervals during the postoperative follow-up period, 2-D echocardiogram, and continuous as well as pulsed Doppler studies were performed using a phased array system with 3.5-5 mHz transducers. All the standard views were obtained and the images recorded for subsequent analysis. Preoperative echocardiography and Doppler study results for all patients are listed in Table 1.

Table 1—Clinical Characteristics Before (B) and After (A) Surgery*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age/Sex</th>
<th>Previous Rheumatic Fever</th>
<th>SOB NYHA</th>
<th>Cardiac Lesion</th>
<th>Surgical Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10/M</td>
<td>+</td>
<td>4</td>
<td>2 Severe MR</td>
<td>MVR-B</td>
</tr>
<tr>
<td>2</td>
<td>22/F</td>
<td>+</td>
<td>4</td>
<td>2 Severe MR</td>
<td>MVR-B</td>
</tr>
<tr>
<td>3</td>
<td>13/M</td>
<td>+</td>
<td>4</td>
<td>1 Severe AR</td>
<td>AV Rep</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mild TR</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>15/M</td>
<td>+</td>
<td>4</td>
<td>1 Severe MR</td>
<td>MVR-Mech</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Severe TR</td>
<td>TV Rep</td>
</tr>
<tr>
<td>5</td>
<td>15/M</td>
<td>+</td>
<td>4</td>
<td>2 Severe MR</td>
<td>MV Rep†</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Severe TR</td>
<td>TV Rep</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mild AR</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>18/F</td>
<td>+</td>
<td>4</td>
<td>1 Severe MR</td>
<td>MVR-B</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Severe AR</td>
<td>AVR-B</td>
</tr>
</tbody>
</table>

*SOB is shortness of breath; NYHA, New York Heart Association; MR, mitral regurgitation; AR, aortic regurgitation; TR, tricuspid regurgitation; MVR-B, AVR-B, mitral and aortic valve replacement with bioprosthesis; MVR-Mech, mitral valve replacement with mechanical valve; MV Rep, TV Rep, AV Rep, mitral, tricuspid and aortic valve repair.

†MV Rep at second operation.

*From the Riyadh Cardiac Centre, Riyadh Military Hospital, Saudi Arabia.

Manuscript received October 16, 1987; revision accepted March 21.

Reprint requests: Dr S. Al Kasab, E225, Armed Forces Hospital, PO Box 7897, Riyadh, Saudi Arabia.
All six patients were treated with antifailure regimens of digoxin and furosemide (Lasix), and afterload reduction medication, in addition to antiinflammatory therapy of acetylsalicylic acid in a dose ranging from 100 to 150 mg/kg body weight. Ampicillin, 500 mg every eight hours, was given for seven days. Patients 3, 4, 5, and 6 also received prednisolone at a dose of 0.75 to 1 mg/kg body weight every 24 hours. Despite the above regimen, pulmonary edema remained uncontrolled in all six patients, and two patients required positive pressure ventilation; consequently, all patients underwent urgent surgical intervention. The surgical procedures are listed in Table 1.

Postoperatively, the antiinflammatory therapy was continued for four to six weeks, and all six patients received monthly penicillin prophylaxis for the prevention of recurrence of rheumatic fever.

Operative Technique

There were vascular and edematous adhesions around the heart. Myocardial preservation was achieved by cold potassium cardiopneumonic solution, in addition to topical cooling of the heart by continuous flow of cold saline solution.

The mitral valve anulus was dilated in all patients, and also the tricuspid valve anulus in patients 4 and 5. In patients 2 and 6, a ruptured chorda tendinea, previously seen on 2-D echocardiogram, was confirmed, as was the ruptured chordae in patient 5 which occurred after the original mitral valve repair.

In patients 1, 2, and 6, the mitral valve leaflets were thickened, with yellowish verrucous-fibrinous like vegetations on the surface. In patients 3, 4, and 5, the mitral valve leaflets looked thickened and slightly edematous, with mildly elongated chordae tendineae of the anterior mitral valve leaflet, and no evidence of stenosis. The aortic valve in patients 3 and 6 was thickened and retracted, with fibrinous vegetation on the leaflets in patient 6.

In patients 3 and 5, the mitral valve was repaired using a Duran ring, size 29 mm, without surgical intervention to the subvalvular apparatus. The aortic valve repair in patient 3 was performed by making a transverse incision approximately 4 mm above the commissure in the aortic root, and using commercially available bovine pericardium to extend the three leaflets. The extension was stitched to the patient's own commissure and leaflet, and suspended by stitching it to the aortic wall vertically along the commissural line.

RESULTS AND FOLLOW-UP

Postoperation

Patient 3 redeveloped a fever one week following mitral valve repair, with further deterioration in his clinical condition. However, this episode was controlled with medical therapy. Patient 5 remained febrile, and one week after the mitral valve repair, developed arthritis, a grade 4/6 midlate systolic murmur in the mitral area, radiating to the axilla, and progressed into acute left ventricular failure. The new murmur and failure were caused by mitral valve incompetence, due the rupture of a chorda tendinea (Fig 1) as a consequence of persistence of rheumatic activity. Subsequently, the patient underwent an urgent second operation for the replacement of the mitral valve with a mechanical prosthesis.

Patient 6 was readmitted, one month following surgery, with arthritis, a raised ESR, and an increased ASOT titer, all indicative of recurrence of rheumatic activity, though with no hemodynamic changes. This episode was controlled by modification of the medical therapy.

Follow-up Study

The mean follow-up period for the six patients was two years (one to four years). Clinically, patients 2, 4, and 6 are in NYHA class I with no demonstrable cardiac murmur. Patients 3 and 5 are in NYHA class II. Patient 3 has moderate mitral and aortic regurgitation; the mild aortic regurgitation of patient 5, which was detected on first admission, remains unchanged.

Four years after surgery, patient 1, (the first of the series) began to deteriorate and was readmitted in NYHA class III, with moderate mitral, moderate aortic, and moderately severe tricuspid regurgitation. After further medical therapy, he was discharged in NYHA class II. He has been scheduled for elective surgery.

The ECG in all six patients showed sinus rhythm, with no abnormality in patients 2, 4, and 6. Patients 1 and 3 showed left ventricular hypertrophy, and patient 5 had evidence of right ventricular hypertrophy.

The chest x-ray film showed normal findings in patients 2, 4, and 6 and showed mild cardiomegaly in patients 1, 3, and 5.

Two-Dimensional Echocardiography and Doppler

Two-D echocardiographic follow-up study results demonstrated good left ventricular function in all patients except patient 3, who had mild impairment of left ventricular function. The follow-up study results of the 2-dimensional echocardiography and Doppler studies of all patients are in Table 2.

There has been no mortality and no significant morbidity in this series.

DISCUSSION

Contrary to commonly held belief, our study clearly...
indicates that active rheumatic carditis is not a contra-
indication to valve replacement.

In this series, the principal cause of the patients' severe hemodynamic deterioration was valvular structural insufficiency. In turn, this led to severe intractable left ventricular failure, which was not controlled by intensive medical therapy; valvular surgery became mandatory as a lifesaving measure. These circumstances may differentiate this group of patients from those in whom the severe congestive heart failure is due primarily to poor left ventricular function, with minimal valvular insufficiency. In these cases, valvular surgery may be of no value.

The recurrence of rheumatic activity following valve replacement in acute rheumatic carditis has been reported by Papoun et al. In our series, despite ample antiinflammatory therapy postoperatively, rheumatic activity continued in the two patients with repaired valves and recurred in one whose valve was replaced. Despite the initial success, repaired valves seemed to be more prone than prosthetic valves to subsequent hemodynamic disturbances caused by further rheumatic activity. This contrast was well demonstrated by patient 5, (valve repaired) who suddenly developed acute left ventricular failure, whereas patient 6, (valve replaced), remained hemodynamically stable, despite the continued rheumatic activity.

The left ventricular function, valvular and subvalvular apparatus morphology, and hemodynamic abnormalities were easily assessed by 2-D echocardiography and Doppler study. This highlights the value of these noninvasive investigations, particularly when the general condition of the patients is poor, with severe congestive cardiac failure, allowing the stress of cardiac catheterization to be avoided.

Valve replacement has improved the prognosis of patients with chronic rheumatic valve disease, though the long-term problems of valve replacement, notably thromboembolism, anticoagulation, infection, hemolysis, and the durability of valves are very real in young patients. Despite these inherent difficulties, valve replacement to correct valvular insufficiency should not be deferred in a severely ill patient with acute rheumatic carditis and intractable left ventricular failure, particularly when myocardial function is preserved.

Valve replacement is a lifesaving method of treating patients with acute rheumatic carditis and intractable heart failure due to valvular insufficiency. Replacement may be preferable to valve repair in the acute phase of the disease.

ACKNOWLEDGMENT: The authors thank Dr. W. Sawyer for assistance with the manuscript.

REFERENCES
12 Gotsman MS, Van der Horst RL. Surgical management of severe mitral valve disease in childhood. Am Heart J 1975; 90:685-87