Outcomes of surgical intervention for isolated active mitral valve endocarditis

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Objective: Although several studies have examined the outcomes of mitral valve repair for infective endocarditis, no studies have documented the long-term outcomes of surgical intervention for active endocarditis confined to the mitral valve.

Methods: One hundred four patients underwent surgical intervention for active infective endocarditis confined to the mitral valve over a 27-year period (mean age, 50 ± 18 years; 52% female). The infected valve was native in 81 patients, previously repaired 6 patients, and prosthetic in 17 patients. *Staphylococcus aureus* was the most commonly isolated (32%) source of infection. Twenty-eight (27%) patients had annular abscesses. Surgical intervention consisted of valve repair or replacement for limited infection and radical resection, annular patch reconstruction, and valve replacement for annular abscess. Mean follow-up was 5.6 ± 4.4 years (range, 0–20 years) and was complete.

Results: There were 9 (8.7%) in-hospital deaths and 28 (27%) late deaths. Overall survival at 5, 7, and 10 years was 73% ± 5%, 68% ± 5%, and 58% ± 6%, respectively. At 7 years, freedom from recurrent endocarditis was 89% ± 4% and freedom from reoperation was 94% ± 3%. Event-free survival at 7 and 10 years was 60% ± 6% and 46% ± 7%, respectively, and was significantly higher in patients with native endocarditis versus those with nonnative endocarditis (ie, prosthetic or previously repaired; 7 years: 63% ± 7% vs 50% ± 12%, $P < .005$). Preoperative shock, *S aureus* infection, and bioprosthesis insertion were independent predictors of death from all causes. The patients in the bioprosthesis group were older (57 ± 20 years vs 44 ± 15 years in the mechanical group and 46 ± 12 years in the repair group, $P = .003$).

Conclusions: Surgical intervention for isolated active mitral valve endocarditis remains difficult, with high morbidity and mortality in the long term. Event-free survival is worse in those who have nonnative mitral valve endocarditis.

ACD

Infective endocarditis continues to carry high mortality and morbidity. No significant improvement has been seen in the past 2 decades.[1] Early diagnosis followed by appropriate antibiotic therapy forms the cornerstone of treatment.[2] Where medical management fails or complications of endocarditis ensue, timely surgical intervention is crucial for patient survival. Surgical intervention for endocarditis can be technically challenging because of the extensive tissue destruction and abscess formation that can arise, and reconstruction after radical debridement might be required.

Previously, we reported that in patients undergoing surgical intervention for endocarditis, mitral valve infection was seen in approximately two thirds of cases, half as isolated mitral involvement and half in conjunction with aortic involvement as double-valve endocarditis.[3] Although several studies have looked at outcomes after surgical intervention for infective endocarditis as a whole,[4-6] studies focusing on mitral valve endocarditis have mainly examined the possible role of mitral valve repair.[7-9] They have also been compounded by including patients with other infected or diseased valves who required concomitant operations. There have been no studies that have documented the long-term outcome of surgical intervention in patients who have active infective endocarditis that is confined to the mitral valve only.

MATERIALS AND METHODS

A review of the Toronto General Hospital cardiac surgery database revealed 104 patients who had undergone surgical intervention for active isolated mitral valve endocarditis from 1978 to 2004. Where necessary, relevant information was also derived by reviewing hospital medical records. Approval was obtained from the ethics review board of the hospital. Only patients with definite endocarditis were included, as evidenced by appropriate combinations of clinical features, operative findings, histopathology, and microbiology. Indications for surgical intervention were 1 or more of the following: shock (n = 15 [14%]), congestive cardiac failure (n = 60 [58%]), cerebral embolism (n = 29 [28%]), and perivalvular abscess (n = 28 [27%]). Table 1 shows the clinical characteristics of the patients. Our series included 2 intravenous drug abusers. There were no specific contraindications to surgical intervention, and each patient with endocarditis referred for surgical intervention was considered on a case-by-case basis. Surgical intervention was not offered only when it was deemed that it would not provide a substantial benefit in prognosis and quality of life.

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Abbreviations and Acronyms
CI = confidence interval
HR = hazard ratio

Infecting Organisms

Data for the infecting organism were available for 96 patients. *Staphylococcus aureus* was the most commonly encountered and present in 33 (32%) patients; *Streptococcus viridans* was present in 23 (22%) patients, *Staphylococcus epidermidis* was present in 9 (9%) patients, *Enterococcus faecalis* was present in 1 (1%) patient, other *Streptococcus* species were present in 11 (11%) patients, other bacteria were present in 11 (11%) patients, and culture-negative endocarditis was present in 8 (8%) patients.

Operative Procedures

Our principles of surgical intervention for infective endocarditis consisted of uncompromising and thorough excision of all infected material and tissues, cutting back until healthy tissues were encountered. This was followed by appropriate reconstruction. Where infection was limited to the leaflets of the native valve or to the prosthetic valve, valve repair or simple replacement was performed. Valve repair was our preferred option, provided adequate valve tissue was left after complete excision of infected material. Where infection had extended to or beyond the annulus, as seen with abscess formation, surgical intervention consisted of radical resection of all infected tissue, followed by reconstruction as necessary with valve replacement, as has been previously described in detail.[10-12] The choice of prosthesis was determined by means of informed consent of the patient in combination with the operating surgeon's judgment. Defects in the mitral annulus, ventricular muscle, or left atrial wall resulting from radical resection were patched with autologous or bovine pericardium.

Pre-existing mitral valve pathology was noted in 53 patients: myxomatous (n = 33 [33%]), rheumatic (n = 8 [8%]), prosthetic dysfunction (n = 10 [10%]), and ischemic (n = 2 [2%]). Operative data are summarized in Table 2. One patient very early in this series had prosthetic mitral valve endocarditis with a perivalvular leak 4 months after undergoing mitral valve replacement. Endocarditis was not apparent at the time of surgical intervention, and successful repair of the leak was undertaken. Patients in this series had no endocarditis involvement of any other valve and did not require any additional valve operations. Average cardiopulmonary bypass time was 93 ± 38 minutes, with a crossclamp time of 69 ± 30 minutes.

All patients received intravenous antibiotics courses as per microbiology advice, usually continuing for 4 to 6 weeks after the operation. Once fully ambulatory and fit for discharge, patients had an in-dwelling intravenous catheter sited and were discharged home. A home care team of community nurses would visit the patient daily to administer the antibiotics as prescribed.

Follow-up

All patients were reviewed annually by the referring cardiologist, and data were collected prospectively. Our dedicated research personnel verified all cardiac events and maintained a comprehensive database. The mean follow-up was 5.6 ± 4.35 years (range, 0–20 years) and was complete.

Statistics

Continuous variables were reported as mean values ± standard deviation. All data analyses were performed with SAS 9.1 Software (SAS Institute, Cary, NC). Fisher's exact tests or χ^2 tests were used to evaluate categorical variables univariately and were reported as frequencies. The Student *t* test was used to analyze continuous variables that had normal distribution, and the Wilcoxon rank test was used for variables that had nonparametric distribution. The Kaplan–Meier method was used to calculate estimates for long-term survival, freedom from reoperation, and event-free survival. Event-free survival was defined as the freedom from thromboembolism (including stroke), valve thrombosis, hemolysis, structural valve dysfunction, perivalvular leak, major bleeding event, endocarditis, reoperation, myocardial infarction, and death in hospital survivors. All preoperative variables with a univariate *P* value of less than .25 or those judged to be clinically important were submitted to the multivariable model for Cox regression analysis to determine the independent predictors of operative and late deaths.

TABLE 1. Clinical characteristics

Characteristic	No.	Percentage
Age (y)	50 ± 18	
Male sex	50	48
Comorbid conditions		
Diabetes mellitus	13	13
Hypertension	15	15
Chronic obstructive lung disease	2	2
Renal failure	7	7
Recent myocardial infarction (<30 d)	3	3
Recent stroke or transient ischemic attack (<30 d)	29	28
Peripheral vascular disease	7	7
New York Heart Association class		
I	6	6
II	2	2
III	10	10
IV	85	83
Preoperative rhythm		
Sinus rhythm	84	81
Atrial fibrillation	20	19
Cardiogenic or septic shock	15	14
Congestive cardiac failure	60	58
Endocarditis		
Native, never operated	81	78
Previous repair	6	6
Prosthetic	17	16
Previous cardiac surgery	26	25
CABG	3	3
Aortic valve replacement	2	2
Mitral valve repair	6	6
Mitral valve replacement	17	16
TV repair	2	2
Left ventricular ejection fraction		
≥40%	83	89
<40%	10	11
Timing of operation		
Elective/same hospitalization	45	51
Urgent/emergency	43	49

CABG, Coronary artery bypass grafting; *TV*, tricuspid valve.

RESULTS

There were 9 (8.7%) in-hospital deaths. Operative mortalities are summarized for the various subgroups in Table 3. Although cause of death was usually multifactorial, the main causes were multiorgan failure (n = 4), stroke (n = 1), septicemia (n = 1), respiratory failure (n = 1), cardiac arrest (n = 1), and retroperitoneal hemorrhage (n = 1). Of the