Mitral Valve Replacement After 14 Years Of Closed Mitral Commissurotomy

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Citation

Abstract
Mitral stenosis is the most frequently encountered valvular pathology and may require surgical intervention when the lesion is severe in rheumatic etiology. Closed mitral commissurotomy (CMC) was the first effective intervention in valvular heart disease. CMC provides excellent long-term hemodynamic and clinical improvement in appropriately selected patients.

In this study we're presenting a successful mitral valve replacement in reoperation of a case who had CMC operation 14 years ago.

We think also, when symptomatic deterioration occurs late after CMC, MVR restores clinical and hemodynamic improvement in many patients

INTRODUCTION
Although it's incidence is decreasing in western countries, rheumatic mitral disease is still frequent in developing countries. Mitral stenosis is the most frequently encountered valvular pathology and may require surgical intervention when the lesion is severe in rheumatic etiology. The choice of operative technique is also very important. In patients with eligible mitral valve pathologies, closed mitral commissurotomy (CMC) may be an option before 1990(1). CMC was a successful treatment but open mitral commissurotomy took its place after 1990s.

CASE PRESENTATION
Our case was 36 years old woman and was followed for rheumatic mitral stenosis (MS) diagnosis for 22 years. CMC was performed 14 years ago in our patient by our same surgeons. She was admitted to our Cardiology Outpatient Clinic for dyspnea and increasing fatigue for last 3 years. She was in New York Heart Association (NYHA) functional class III at presentation. She was evaluated by clinical, hematological, electrocardiographical and echocardiographical examination preoperatively. She had a sinus rhythm in electrocardiography. Transthoracic echocardiography (TTE) showed a very fibrotic mitral valve. The mitral orifice area was 1.4cm² (Figure 1).

Diameter of left atrium and left ventricle were normal. And her pulmonary arterial pressure (PAP) was 45mmHg. Peak gradient was 29mmHg and mean was 19mmHg (Figure 2).
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**Figure 2**

Mitral valve leaflets were thick, calcific and their opening was extremely limited (Figure 3).

**Figure 3**

Left ventricle EF was 60%. Other valves' functions included minimal tricuspid regurgitation. Her cardiac coronary arteriography (CAG) and cardiac catheterization were performed. It was confirmed that EF:40%. Coronary arteries were normal. RV, PAP, PCWP, LV and aorta pressures were 70/0/10mmHg, 70/30mmHg, 20mmHg, 130/0/15mmHg and 130/70mmHg respectively.

She went under operation. She was operated under endotracheal general anesthesia and in supine position. Following a median sternotomy, pericard was opened longitudinally. Constricting layers of epicardium were separated if possible. After heparinization, extra-corporeal circulation is established between the venae cavae and the ascending aorta. A cross clamp was placed on aorta and by antegrade intermittent isothermic blood cardioplegy from aortic root, cardiac arrest was established. Hypothermia was moderate (28°C). A vent was placed via the right superior pulmonary vein. The mitral valve is approached via a standard right atriotomy for transseptal approach (Figure 4).

**Figure 4**

The entire valvular apparatus was carefully examined in order to assess the feasibility of reconstructive surgery and to plan the operative technique. The valvular apparatus was then mobilized as an entire unit with a nerve hook in order to assess tissue flexibility and to identify leaflet restriction. The anterior leaflet was thickened and fibrotic and cords were shortened. The posterior leaflet was completely thickened. The cords of the posterior leaflets were also thickened and shortened. There was a fusion defect from anterolateral commissure and posteromedial commissure. There was a minimal space only from central part. For these reasons, mitral valve reconstruction was impossible (Figure 5).
The native valve was resected. Mitral valve replacement (27 no bileaflet mechanical valve) was performed standardly. Valve competence and closure were excellent (Figure 6).

Iatrogenic atrial septal defect and right atriotomy closed. She was no required inotropic support during weaning from cardiopulmonary bypass and early postoperative period. The volume of blood transfused was one unit. The quantity of mediastinal drainage was 450 cc. She was extubated after an intubation duration 8 hours and stayed in the intensive care for 2 days. The hospital stay was 8 days. The functional capacity of our patient improved dramatically and she was in NYHA functional class I. Postoperatively at the discharge day and after 3 months an echocardiographic investigation was revealed no dysfunction for the replaced mitral valve.

DISCUSSION

Etiology plays a very important role in correction difficulties and in terms of late results affected by the progression of the disease. Mitral stenosis is the most frequently encountered valvular pathology in rheumatic etiology.

Closed mitral comissurotomy (CMC) was the first effective intervention in valvular heart disease. The possibility of opening a stenotic mitral valve by digital splitting of the fused commissures was apparently suggested by Samways as early as 1898. Recently, CMC has been reexplored due to concepts of less invasive valvular surgery. In patients with stenotic mitral valves with pliable and uncalcified leaflets and no thrombus in left atrium, CMC is a safe and cost-effective procedure as stated previously. Avoidance from cardiopulmonary bypass and short duration of operation is a very important advantage of the CMC. Reoperation is performed generally 5–20 years after first CMC operation. Mitral restenosis is the most frequent cause of reoperation. Our patient's cardiac functions were optimal protected with the aid of CMC.

In the study of Toumbouras et al., seven hundred and fifty-four patients who underwent CMC between 1958 and 1993 (71% female, 29% male; mean age 39 years) for acquired mitral stenosis were reviewed postoperatively. Particular attention was given to those patients who later required MVR. The total follow up experience was 9,607.9 years. Eighty-two patients (11%) needed a repeat CMC and 30% of these patients had subsequent MVR. In all, 146 patients (19.3%) required MVR a mean of 17.0 years after commissurotomy (range one to 35 years). The indications for MVR were restenosis (59%), residual stenosis with or without mild mitral regurgitation (30%), and moderate to severe regurgitation (11%). Among survivors, 88% improved at least one functional class after MVR and the majority was free of congestive heart failure. It is concluded that CMC provides excellent long term clinical improvement in appropriately selected patients. The mean time interval of 17 years between CMC and late MVR reveals the efficacy of CMC to achieve satisfactory long term results.

In the study of Salerno et al., a 25-year experience with 139 patients undergoing closed mitral comissurotomy is reviewed. The primary indication for CMC was mitral stenosis, but 24 patients also had other less important valvular defects. Postoperative complications occurred in 3%, and operative mortality was 2.0%. Follow-up revealed restenosis in 6% of the patients, mitral regurgitation in 14%, etc.
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complications in 7%, and late deaths in 3%. Reoperation, required in 32% (44 of 139 patients), included a second closed mitral commissurotomy (21 patients), open mitral commissurotomy (4), mitral valve replacement (MVR) (18), and MVR after a second closed mitral commissurotomy (2). Improvement in NYHA classification was found in 82% of these patients. Operative mortality was 9.5% for patients having a second closed mitral commissurotomy and 20% for those having MVR.

Ates et al. evaluate long-term survival and valve-related complications as well as prognostic factors for mid- and long-term outcome after closed mitral commissurotomy, covering a follow-up period of 14 years. 36 patients underwent closed mitral commissurotomy. After closed mitral commissurotomy, the mitral valve areas of these patients were increased substantially, from 0.9 to 2.11 cm. No further operation after initial closed mitral commissurotomy was required in 86% of the patients and NYHA functional classification was improved in 94% . Postoperative complications and operative mortality were not seen. No early mortality occurred in closed mitral commissurotomy patients. Reoperation was essential for 5 patients following closed mitral commissurotomy; 2 procedures were open mitral commissurotomies and 3 were mitral valve replacements. For their opinion the mitral valve area was significantly increased and the mean mitral valve gradient was reduced in patients after closed mitral commissurotomy.

However Suzuki et al. evaluated and compared the results of CMC, OMC and MVR in their study. In the CMC group the survival rate and the event free rate were lower, and the rate of reoperation was higher than in the other two groups.

We think also that Closed mitral commissurotomy is a safe alternative to open mitral commissurotomy and balloon mitral commissurotomy in selected patients. And when symptomatic deterioration occurs late after CMC, MVR restores clinical and hemodynamic improvement in many patients.

Mitral valve repair in rheumatic disease is technically more difficult, and there is little information on the long-term stability of this technique. Rheumatic lesions have been more difficult to treat conservatively and have a higher rate of repeat operations for early and late valve dysfunction, which oscillates between 10% and 27%.

CORRESPONDENCE TO

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