Mitral Valve Replacement With or Without Subvalvular Apparatus Preservation

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Abstract

Background: Mitral Valve Replacement (MVR) for mitral valve disease continues to be associated with long-term morbidity and mortality. Despite improvements in myocardial protection and prosthetic valves, the rate of morbidity has not decreased significantly over the years. The most common cause of death following MVR is cardiac failure.

Preservation of the subvalvular apparatus maintains Left Ventricle (LV) function and thus improves survival. Repair is not always feasible or successful, particularly with rheumatic valve disease in young patients and severely disorganized valves. A claim against preservation of the Anterior Mitral Leaflet (AML) was that only undersized valve prosthesis could be implanted. Another argument against preservation of the anterior leaflet is that it might cause obstruction of the Left Ventricular Outflow Obstruction (LVOT).

Objective: The objective of this study was to assess the immediate and short-term (6 months) changes in LV performance after MVR with preservation of chordae tendinea of AML.

Methods: This study was done in department of Cardiothoracic Surgery at Kasr Al-Aini Faculty of Medicine after approval of the local ethical committee from 2011 to 2013. Sixty patients with severe mitral disease were included in the study for operative and short term postoperative results to evaluate the impact of SVP during surgery on the operative and postoperative outcome.

Results: The sixty patients were divided into two groups where 30 of them underwent preservation of AML, these patients had better LV function in the early and the short term postoperative period.

Conclusion: Results of this study concluded that SVP of AML leads to better postoperative outcome. We recommend its application on a greater scale of cases of MVR.

Key Words: MVR – AML – Preservation – Subvalvular apparatus.

Introduction

THE optimum management of mitral valve insufficiency is valve repair [1]. Often valve replacement is necessary, however, as repair is impossible because of anatomical or aetiological considerations [2,3].

The first Mitral Valve Replacement (MVR) involved implantation of a starr-edwards prosthetic valve following complete excision of the mitral leaflets, chordae tendinae and the heads of the papillary muscles [4]. Initial experience with MVR was complicated by an increased incidence of low cardiac output syndrome and associated morbidity and mortality. Subsequently, several strategies were implemented to improve postoperative outcomes, including Sub-Valvular Apparatus Preservation (SVP) [5].

The concept of SVP is more than 40 years old [6]. Despite the publication of several studies since the late-1970s suggesting that left ventricular function and mortality were improved following SVP, particularly in patients with mitral regurgitation, it is sometimes not undertaken [7]. Whilst technical considerations may limit adoption of SVP [8], surgical strategies to overcome these technical pitfalls have been discussed in the literature in some depth [6]. Arguably uncertainty about the long-term impact of SVP on patient-focused outcomes such as free of event survival and quality of life may be a factor.

The main cause of death after MVR is myocardial failure [9]. Several animal and human echocardiographic physiological studies have shown better maintenance of left ventricular function following SVP [6]. It is suggested that this is because papillary muscles are important to left ventricular contraction as they draw the mitral ring toward the apex, causing shortening of the long axis and sphericity of the chamber, thereby contributing to better ejection of blood [10]. Despite existing evidence suggesting that SVP reduces morbidity and mor-
tality [7] the sub-valvular apparatus is not always preserved often because it is not possible to preserve them. It is argued that the preserved sub-valvular apparatus prevent an adequately sized prosthetic valve from being used, and cause left ventricular outflow tract obstruction, by interfering with prosthetic valve function [11]. Whilst techniques to eliminate outflow tract obstruction following sub-valvular apparatus preservation have been described [12], it is reported that some of the preservation techniques cause alteration of the left ventricular geometry, causing rupture of the papillary muscles, systemic embolization and dehiscence of the mitral leaflets from their transposed positions as well as increasing ischemic time [11]. Finally SVP is often not technically possible because of active endocarditis, anatomical or pathophysiological considerations [8].

Patients and Methods

In this study, sixty patients who underwent MVR at the Cardiothoracic Surgery Department, Kasr El-Aini Hospital, Cairo University, in the period between May 2010 and February 2013.

All patients were preoperatively evaluated by History taking, clinical examination and had laboratory, ECG, radiological and echocardiography.

Surgical procedure:

All patients under general anesthesia underwent median sternotomy, routine aortobicaval cannulation, went on bypass and aorta cross clamped, myocardial protection done using hypothermia to 20c, local ice slush, intermittent cold blood cardioplegia, left atriotomy then patients were divided into two groups.

Group A: Thirty patients who underwent MVR without preservation of the chordae tendineae of the anterior mitral leaflet and only preserving the Posterior Mitral Leaflet (PML).

Group B: Thirty patients who underwent MVR with complete or partial preservation of the chordae tendineae of the AML + PML.

Informed consent was obtained from all patients before inclusion in the study.

In both groups MVR done using interrupted 2/0 ethibond stitch, closure of atriotomy, deairing, aorta clamped, offbypass, proper hemostasis and closure of sternotomy. Echocardiography done postoperatively in all patient.

Results

Postoperative echocardiography was done before discharge around the 7th postoperative day and 6 months postoperatively to assess the function of the prosthesis; it was normal in all cases. The mean gradient over the mitral prosthesis was 3.734 ±0.3075mmHg in Group A and 3.931 ±0.291 in Group B. The LV end diastolic diameter (LVEDD), LV End Systolic Diameter (LVESD), Ejection Fraction (EF), and Pulmonary Artery Systolic Pressure (PASP) were measured and compared with the preoperative values.

In Group B, both early and 6 months postoperative echo showed higher EF than in Group A and this is statistically significant (p-value=0.001 and 0.00013 respectively).

In Group A, the mean LVEDD decreased from 6.08±1.3 preoperatively to 6.04±1.08 at 7th day postoperatively, but the difference was not statistically significant, p=0.42. The mean LVEDD continued to decrease significantly to be 5.89±1.12, 6 months postoperatively with a p-value of 0.064 which is statistically not significant. The mean LVESD showed mild reduction from 4.19±1.1 preoperatively to 4.02±0.96 in the early postoperative period. The reduction was also mild after 6 months postoperatively as the mean LVESD was nonsignificantly reduced to 3.87±0.63 (p=0.31).

In Group B, the mean LVEDD decreased from 5.97±1.24 preoperatively to 5.79±1.13 at 7th day postoperatively, but the difference was not statistically significant, p=0.08. The mean LVEDD continued to decrease significantly to be 5.48±1.9, 6 months postoperatively with a p-value of 0.042 which is statistically significant. The mean LVESD showed mild reduction from 4.34±0.83 preoperatively to 4.11±0.91 in the early postoperative period. The reduction was evident after 6 months postoperatively as the mean LVESD was significantly reduced to 3.68±0.96 (p=0.05).

Table (1): Preop. and postop. LVEDD.

<table>
<thead>
<tr>
<th></th>
<th>7 days postoperative Mean S.D</th>
<th>6 months postoperative Mean S.D</th>
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<tbody>
<tr>
<td>Group A</td>
<td>6.08±1.03</td>
<td>5.89±1.12</td>
</tr>
<tr>
<td>Group B</td>
<td>5.97±1.24</td>
<td>5.48±1.9</td>
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<tr>
<td>p-value</td>
<td>0.22</td>
<td>0.042</td>
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Significance Non significant Non significant Significant
Table (2): Preop. and postop. LVESD.

<table>
<thead>
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<th></th>
<th>Preoperative</th>
<th>7 days postoperative</th>
<th>6 months postoperative</th>
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<tbody>
<tr>
<td>Group A</td>
<td>4.19±1.1</td>
<td>4.02±0.96</td>
<td>3.89±0.63</td>
</tr>
<tr>
<td>Group B</td>
<td>4.34±0.83</td>
<td>4.11±0.91</td>
<td>3.68±0.96</td>
</tr>
<tr>
<td>ρ-value</td>
<td>0.31</td>
<td>0.05</td>
<td></td>
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<tr>
<td>Significance</td>
<td>Non significant</td>
<td>Non significant</td>
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Table (3): Pre-and post-operative ejection fraction in the echocardiography.

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<th>Preoperative</th>
<th>7 days postoperative</th>
<th>6 months postoperative</th>
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<tr>
<td>Group A</td>
<td>61.8±7.9</td>
<td>43.6±5.9</td>
<td>58.4±6.1</td>
</tr>
<tr>
<td>Group B</td>
<td>60.4±5.9</td>
<td>48.1±4.2</td>
<td>64.5±5.2</td>
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<tr>
<td>ρ-value</td>
<td>0.442</td>
<td>0.0167</td>
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<tr>
<td>Significance</td>
<td>Non significant</td>
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Discussion

Rushmer and his colleagues [13] demonstrated that the papillary muscles play an important role in the left ventricular contraction. Referring to Rushmer’s results, Lillehei and his colleagues [6] showed that preservation of the chordae of the posterior mitral leaflet resulted in a reduction of mortality from 37 to 14% in patients with MVR.

Most investigators have noted a significant reduction of the LVEF in patients with mitral valve replacement after MVR without preservation of the chordae tendineae. The decrease in the LVEF after conventional MVR was caused by several factors such as decreased preload, increased afterload, or impaired contractile function. After MVR for mitral regurgitation, preload is decreased by removal of the regurgitant volume and afterload is decreased by disappearance of the low impedance ejection root into the left atrium. The main factor responsible for decreased ejection performance is thought to be interruption of ventricular valvular interaction with the change in loading condition.

Lillehei’s technique was reintroduced by David and coworkers [6] and by Hetzer and his colleagues [15]. They reported on beneficial effects of the chordal preservation in a larger series of patients regarding the clinical status of the patients with respect to the need for catecholamines and other clinical parameters.

Miki and his colleagues [14] published a new technique that allowed not only preservation of the posterior, but also of the anterior mitral leaflet.

Many surgeons continue to retain only the posterior leaflet with chordae tendineae because
of concerns over greater technical complexity, longer operating time, potential interference with mechanical leaflet motion, need to undervise the mitral prosthesis, and the possibility of creating Left Ventricular Outflow Tract Obstruction (LVO-TO) [12].

In our study the mean age was 31.53 ±6.68 and 31.47±5.60 years. The mean age in our study is lower than other studies because the rheumatic affection is evident in this age group in Egypt. Natsuaki and associates [16] reported a mean age of 55±10 in their study. Okita and his colleagues [12] reported mean age of 52.6 years, in spite that the valve disease in their study was solely of rheumatic origin. Chowdhury and his colleagues [17] reported a mean age of 35.6 ±19.0; their study was done in India and the valve pathology was rheumatic mitral disease.

Females contributed to 58% of the patients in our study, which shows that female affection is more. In the study of Okita and his colleagues [17] females contributed by 76%.

In Egypt, many patients with mitral stenosis represent to us in very late stages with heavily calcified leaflets and severe pulmonary hypertension (PASP above 90mmHg). These patients were excluded from our study. Zakai SB [18] and colleagues in 2010 found an interesting finding was that of PA pressures and LA size between the groups. It was significantly improved in the preservation groups as compared to the resection group, while in our study there weren't statistically significant difference in the improvement of the pulmonary artery pressure.

Chowdhury and his colleagues [5] had a higher preoperative incidence of AF; 62.8% in the non-chordal group, 62% in the posterior chordal-preservation group and 72% in the total preservation group. Postoperatively the incidence of AF was 40%, 46%, 42.4% with no statistically significant difference (p =0.61). They concluded that that chordopapillary preservation techniques did not affect the outcome of postoperative atrial fibrillation.

Mitral valve pathology in our study (Group B) was 15 patients (50%) had isolated Mitral Regurgitation (MR), 8 patients (27%) had isolated Mitral Stenosis (MS), and 7 patients (23%) had Double Mitral lesion (DM). Lesion most of the preservation studies were conducted on patients with mitral regurgitation, few investigations have been conducted in patients with mitral stenosis in which the stiff subvalvular complex was preserved or replaced with artificial chordae. Okita and his colleagues had conducted a study only on patients with pure mitral stenosis.

In our study, the condition of the subvalvular apparatus determined the technique used for preservation of annulopapillary continuity. When the annulus was not severely thickened, which was the case in 6 patients (20%), we used the technique in which a colleagues [21] semi-elliptical-shaped piece of tissue is excised from anterior leaflet. When the anterior leaflet was severely thickened and calcified, in 24 patients (80%), it was divided into 2 to 5 chordal segments depending on the size of the valvular leaflet. Each segment then is trimmed into chordal buttons and reattached to the annulus in an anatomic fashion. In patients of Group A, all of the native chordal structures of the anterior mitral leaflet were resected because the subvalvular apparatus was markedly diseased, there was fusion of the chordae tendineae, foreshortening of the chordal apparatus, or papillary muscle thickening, so, posterior mitral leaflet preservation only was done.

Kayagioglu and his colleagues [20] reported that the LVEDD and LVESD decrease in the preservation group and increased in the conventional group postoperatively but the changes were statistically insignificant. EF decrease slightly postoperative in patients with preserved chordae, however it decreased significantly in patients with conventional MVR.

Gaiotto and his colleagues [19] reported a reduction in the LVEDD (p=0.038) and LVESD (p=0.008) in patients with end stage cardiomyopathy who underwent MVR with preservation of the chordae tendineae.

Chowdhury and his colleagues [8] reported that the Left Ventricular End-Systolic Volume (LVESV) decreased slightly from the preoperative level in the total excision group in the immediate postoperative period. Although there was gradual improvement at 1 to 4 years of follow-up, the improvement was not statistically significant. The remaining groups (the posterior preservation and the total preservation) demonstrated statistically significant reduction in LVESV in the immediate as well as the late postoperative period. The total chordal group demonstrated greater fractional change as compared to the posterior chordal and the non chordal group.

Fractional shortening was not universally used by most authors. Muthialu and his reported that
the FS was 34% preoperatively vs. 26% postoperatively in the conventional group, and 31% vs. 29% in the preservation group, \( p=0.06 \).

Conclusions:

We concluded that total chordal preservation is possible in the majority of patients undergoing MVR for rheumatic heart disease, as this study showed significant improvement in the LV function in the patients in which the AML was preserved. In the minority of patients in whom the native chordae couldn’t be preserved; preservation of the annulopapillary continuity can be achieved by replacement of the chordae with artificial chordae. These techniques improve left ventricular ejection fraction and result in reduction in both left ventricular systolic and diastolic diameters. These techniques can be safely performed without effect on the choice of the prosthetic size or fear of left ventricular outflow tract obstruction or interference with prosthetic leaflets motion.

References


المتاحة: الصمام الميترالي هو الصمام الموجود ما بين البطين الايسر والأدنى الايسر للقلب. ويتعرض الصمام الميترالي للمرض في كثير من الأحيان، منها بسبب الأمراض الروماتيزمية وهو السبب الأكثر انتشارًا في مصر.

إن العلاج الأمثل لحالات ضيق أو ارتجاع الصمام الميترال هو إصلاح الصمام لاعتبارات كثيرة ولكن في كثير من الأحيان وخاصة في حالات الحبي الروماتيزمية يكون أصلاح الصمام غير ممكن وبالتالي يكون تغيير الصمام هو الحل الأمثل.

منهجية البحث: تم عمل هذه الدراسة بمستشفى جامعة القاهرة في الفترة من 2011 وحتى 2013 وقد شملت 60 مريض أجريت لهم جراحة استبدال الصمام الميترالي وتم تقسيمهم لمجموعتين: الأولى 20 مريض تم تغيير الصمام مع الحفاظ على جهاز ما تحت الصمام والثانية تم إجراء الجراحة مع إزالة جهاز ما تحت الصمام وتم متابعة المرضى بعد الجراحة لتقييم كفاءة الجزء الأساسي من عضلة القلب بواسطة الموجات الصوتية.

النتائج: أظهرت النتائج أن المجموعة الأولى كانت كفاءة عضلة القلب أفضل بعد الجراحة وأن معدل فشل الجزء الأساسي من عضلة القلب كان أقل في هذه المجموعة التي تم الأمر فيها على جهاز مصطفى الصمام.

الخلاصة: أنه يفضل على الأطباء على جهاز ما تحت الصمام في حالات تغيير الصمام الميترالي كلما كان ذلك ممكناً.