Re-repair of the mitral valve as a primary strategy for early and late failures of mitral valve repair†

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Abstract

OBJECTIVES: With the expanding uptake of mitral valve repair as the primary therapy for mitral valve regurgitation, an increasing cohort of patients are presenting with failures following valve repair. These patients have traditionally been treated by mitral valve replacement. We have adopted an aggressive strategy of valve re-repair for failures of mitral valve repair and present our mid-term results.

METHODS: Fifty-three consecutive adults underwent reoperation by a single surgical team for failed non-rheumatic mitral valve repair. Primary valve repair had been done for degenerative (n = 38), congenital (n = 6), infective (n = 3), functional (n = 1) or unknown (n = 5) mitral disease. The reoperative mitral procedure occurred at a median interval of 3 (interquartile range 0.9–6.5) years from the primary mitral valve repair. Valve re-repair was attempted if the anterior leaflet was sufficiently pliable, and lesions causing recurrence were identifiable and deemed treatable. Standard repair techniques were employed in re-repair procedures.

RESULTS: Valve analysis showed that the mode of failure was progression of original disease in 19 (36%), technical failure in 20 (38%) and new disease in 14 (26%) patients. Valve re-repair was successfully accomplished in 45 (85%) patients. Re-repair was most frequent when the prior aetiology was degenerative (34 of 38, 90%) as opposed to non-degenerative (11 of 15, 73%). There were no hospital deaths. Four-year patient survival was 97%. Freedoms from moderate mitral regurgitation were 100, 95, 88 and 80% at discharge and at 1, 3, and 4 years, respectively. There were no reoperations in the follow-up period.

CONCLUSIONS: Re-repair of the mitral valve is feasible in most of the cases of failed mitral valve repair of non-rheumatic aetiology and has acceptable mid-term outcomes. The relatively high prevalence of technical failures as the mechanism of failure of the primary mitral valve repair suggests the need for ongoing surgical education and continuing development and refinement of repair techniques.

Keywords: Mitral valve repair • Mitral valve re-repair • Recurrent mitral regurgitation

INTRODUCTION

Mitrail valve repair has an inherent failure rate. A linearized failure rate of 1–4% per year, for example, is reported after degenerative valve repairs [1–3]. Additionally, imperfect repairs predispose to early and late failures [4]. With the increasing uptake of mitral valve repair in the last decade [5], more patients are presenting to surgeons with failures of mitral valve repair that can occur due to technical failures, progression of native disease or new pathology [4]. The default therapy for most of the patients with a failed mitral valve repair is a mitral valve replacement. We have adopted an aggressive approach of valve re-repair as our therapy of choice for failed valve repairs and report our outcomes.

MATERIALS AND METHODS

Study design

We retrospectively analysed 53 consecutive patients with non-rheumatic disease who underwent repeat mitral valve surgery for failed mitral valve repair by a single surgical team (Authors ACA and DHA) between June 2003 and July 2011. All patients had undergone only one prior mitral valve repair. The research protocol was approved by the Mount Sinai School of Medicine Institutional Review Board and was compliant with Health Insurance Portability and Accountability Act regulations and the ethical guidelines of the 1975 Declaration of Helsinki. The approval included a waiver of informed consent and a request to access the data of decedents.

Definition of original aetiology and mode of failure

We defined the aetiology of the original mitral valve disease by reviewing operative and echocardiographic reports. The mode of
failure was classified as the progression of original disease, technical failure or new disease, according to our previously published criteria [4] based on findings at reoperation.

**Surgical techniques**

All operations were performed via reoperative median sternotomy. We employed cardiopulmonary bypass with mild hypothermia and antegrade and retrograde cold blood cardioplegia for myocardial protection. We accessed the mitral valve via a left atriotomy in the interatrial groove. In all cases, the valve procedure commenced with a thorough analysis of the mode of failure and the lesion(s) causing regurgitation. If the mode of failure was felt to be either uncorrectable, or highly likely to recur, then the valve was replaced; typically this would include valves with extensive pannus or fibrosis and valves with a non-pliable or destroyed anterior leaflet. For all other valves, we undertook a trial period of re-repair. At any time in the re-repair process, if it became apparent that re-repair would be unsuccessful, then we proceeded with mitral valve replacement.

Valve re-repair was undertaken using a ‘lesion-based’ approach. Failures due to dehiscence, improper sizing or the choice of annuloplasty ring type were treated with corrective annuloplasty. Prolapse was treated with conventional resection or neochordal or chordal transposition techniques. Posterior leaflet tethering, leaflet retraction, suture disruption and excessive tension on suture lines were treated with tissue augmentation with the glutaraldehyde-fixed autologous pericardium. Systolic anterior motion was treated by tissue resection, sometimes in combination with leaflet dissection. Additionally, any untreated primary lesions, such as excess tissue, calcification, retraction or prolapse, were treated as appropriate. The tricuspid valve was systematically evaluated in all patients with a very low threshold for tricuspid annuloplasty, the indication being tricuspid annular dilatation or mild or greater tricuspid regurgitation (tricuspid valve repair was performed in 38 of the 53 cases). Atrial arrhythmias were concurrently treated with cryoablation.

**Endpoints and follow-up**

The primary endpoint of the study was patient survival. The second endpoint was freedom from recurrent mitral regurgitation (MR), moderate or greater as defined by the guidelines of the American Society of Echocardiography [6]. Follow-up survival information for documented US patients was obtained by matching each patient’s Social Security number with the web-based Social Security death index (http://ssdi.rootsweb.ancestry.com/). It was completed in 100% of the patients, and the median follow-up for survival was 41.0 (interquartile range [IQR] 26.7–58.1) months in the re-repair group and 31.7 (IQR 21.3–58.4) months in the replacement group. Follow-up echocardiogram reports were obtained in the re-repair group from the referring cardiologists. The median echocardiographic follow-up was 15.4 (IQR 6.2–41.5) months. All patients had at least one postoperative echocardiogram (86% had at least one echocardiogram after hospital discharge).

**Statistical analysis**

Continuous variables were expressed as means ± 1 standard deviations if normally distributed and medians and IQRs if not normally distributed. Categorical variables were presented as proportions and absolute numbers. Differences between groups were detected using the χ² test or Fisher’s exact test for categorical variables and unpaired t-test or the Mann–Whitney U-test for continuous variables, as appropriate. Event rates ± 1 standard error were estimated using the Kaplan–Meier method. Logistic regression analysis was used to test the univariate and multivariate predictors of re-repair feasibility. For multivariate analyses, variables with a P-value of ≤0.25 on univariate analysis were included into a final model. Results are presented as odds ratios (ORs) with corresponding 95% confidence intervals (95% CIs). A P-value of <0.05 was considered to be statistically significant. All P-values were the results of two-tailed tests. The statistical analysis was performed using IBM SPSS Statistics for Windows, version 19.0 (SPSS, Inc., IBM Corporation, Armonk, NY, USA).

**RESULTS**

**Original aetiology and prior repair**

The clinical characteristics of the study population are depicted in Table 1. The primary mitral valve repair had been done for degenerative (n = 38), congenital (n = 6), infective (n = 3), cardiomyopathy (n = 1) or indeterminate (insufficient information available from the prior procedure to allow classification) (n = 5) mitral disease. The initial surgery was done in our centre in 14 (24%) cases and in outside hospitals in 39 (76%). The reoperative mitral procedure occurred at a median interval of 3 (IQR 0.9–6.5) years from the primary mitral valve repair.

**Mode of failure and re-repair feasibility**

We classified the mode of failure as the progression of original disease in 19 (36%) patients, technical failure in 20 (38%) and new disease in 14 (26%). Characteristics of re-repair and replacement patients are summarized in Table 1. A variety of techniques were used for re-repair (Table 2). Forty of 45 patients had an annuloplasty ring placed at the time of primary mitral valve repair. Annuloplasty type was complete rigid or semi-rigid (n = 20), rigid partial band (n = 7), flexible complete ring (n = 5), flexible band (n = 1), pericardium or suture (n = 3) and unclear (n = 4). In 36 of the 40 patients with an annuloplasty ring, the first annuloplasty ring was removed and, in 4 patients, the previous ring was kept. The ring type used for re-repair was a rigid or semi-rigid complete ring in all cases. The median ring size was 28.

Mitrval valve re-repair was successfully accomplished in 45 (85%) patients. Re-repair was feasible in 90% (34 of 38) where the prior aetiology was degenerative as opposed to 73% (11 of 15) where the aetiology was non-degenerative. Where the mode of failure was technical, 7 of 20 (35%) of the operations had been performed via a thoracotomy approach, compared with the non-technical failures, where 2 of 33 (6%) were performed via thoracotomy (P = 0.02). On multivariate analysis, degenerative aetiology was the only identified factor that was independently associated with higher re-repair feasibility (adjusted OR = 11.0, 95% CI 1.23–98.5, P = 0.032). Frequency of re-repair was higher if the mode of failure was technical (18 of 20, 90%) or there was progression of native disease (17/19, 89%) compared with new disease (10/14, 71%), but this difference was not statistically significant (P = 0.26). A summary of the patients who
underwent valve replacement, including the reasons the valve could not be re-repaired, is summarized in Table 3.

**Operative outcomes**

There were no in-hospital deaths in either group. Two patients experienced perioperative stroke, and 1 had a deep sternal wound infection. In the re-repair group, predischarge echocardiography revealed an MR grade as none or trace in 38, mild in 6 and mild-to-moderate in 1.

**Survival and re-repair durability**

In patients who underwent re-repair, there were 3 late deaths during the follow-up. The first was a 90-year old female who died 6 years after valve repair of natural causes, the second was a 73-year old who died of lung cancer 3 years after re-repair and the last was a 62-year old male who died suddenly 5 years after re-repair—his echocardiogram 3 months prior to his death showed no MR and preserved ventricular function. The 4- and 5-year survivals were therefore 97 ± 3 and 90 ± 7%, respectively (Fig. 1). Freedoms from moderate MR were 100, 95 ± 4, 88 ± 7 and 80 ± 10% at discharge and at 1, 3 and 4 years, respectively (Fig. 2). Table 4 details of the patients who had recurrent MR—the MR was no worse than moderate on the most recent echocardiogram in any of the patients. There were no reoperations in the study period. There was no early or late mortality detected during the follow-up for the 8 patients who underwent replacement.

**DISCUSSION**

Our series demonstrates that, with a systematic approach emphasizing valve re-repair as a primary strategy, valve preservation is possible in most of the patients with a failed mitral valve repair. Patient survival with re-repair was very good, and re-repairs remained durable to the mid-term. Additionally, our analysis of modes of failure after mitral valve repairs provides clues on how the durability of mitral valve repair may be improved.
Why do mitral valve repairs fail?

The progression of native disease was the mechanism of failure in about a third of our patients. In degenerative disease, this is related to the prolapse of new segments due to new chordal elongation or rupture. This provides scope for future study as to whether prophylactic measures, such as placement of artificial chords on non-prolapsing segments with thinned chordae, can prevent the development of new prolapse in degenerative mitral valve repair.

Technical failure is a dominant cause of failures of mitral repair and constituted 37% of our cases. Other series have found technical failures to account for 42–50% of reoperations after mitral valve repair [7, 8], which causes us to wonder whether half of repair failures are potentially preventable. Shin et al. [8] reviewed echocardiograms on patients presenting for re-repair and found that 32% had 2+ or greater MR on their immediate follow-up echocardiogram. Residual MR has been long recognized as a significant predictor of subsequent failure of mitral valve repair and need for reoperation [9]. Focused surgical training, and directing of complex cases to surgeons skilled in advanced valve repair techniques [10], could potentially reduce technical failures. While the increased uptake of mitral valve repair in recent years [5] represents a significant advance in surgical therapy for MR, such expansion should not be at the expense of the durability of the repair. Many of the technical ‘errors’ we observed were relatively simple, and thus potentially avoidable, such as inappropriate ring sizing, over-aggressive leaflet resection and uncorrected primary lesions. Our observation of a higher prevalence of technical failure in repairs done via thoracotomy is a reminder to surgeons not to

Table 3: Replacement cases with reasons re-repair were not feasible

<table>
<thead>
<tr>
<th>Case</th>
<th>Age at re-repair (years)</th>
<th>Original aetiology</th>
<th>Interval from initial repair (years)</th>
<th>Mode of failure in initial repair</th>
<th>Reason for replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>62</td>
<td>Endocarditis</td>
<td>0.1</td>
<td>Progression of native disease</td>
<td>Severe recurrent endocarditis</td>
</tr>
<tr>
<td>2</td>
<td>49</td>
<td>Congenital</td>
<td>2</td>
<td>Progression of native disease</td>
<td>Dense fibrous reaction onto the leaflet, scarring of the sub-valvular apparatus.</td>
</tr>
<tr>
<td>3</td>
<td>51</td>
<td>Congenital</td>
<td>0.5</td>
<td>Technical</td>
<td>Complex congenital anomaly, horseshoe single papillary muscle with thick bulky heads, frozen anterior commissure, 2 mm height of posterior leaflet</td>
</tr>
<tr>
<td>4</td>
<td>72</td>
<td>Primary SAM</td>
<td>0.02</td>
<td>Technical</td>
<td>Partial patch disruption, not enough integrity of tissue remained</td>
</tr>
<tr>
<td>5</td>
<td>71</td>
<td>Degenerative</td>
<td>1.5</td>
<td>New disease</td>
<td>Severe pannus ingrowth and inflammatory reaction</td>
</tr>
<tr>
<td>6</td>
<td>54</td>
<td>Degenerative</td>
<td>1.1</td>
<td>New disease</td>
<td>Severe pannus ingrowth, dime sized orifice</td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>Degenerative</td>
<td>13</td>
<td>New disease</td>
<td>Scarring and shortening of the posterior leaflet</td>
</tr>
<tr>
<td>8</td>
<td>62</td>
<td>Degenerative</td>
<td>1</td>
<td>New disease</td>
<td>Significant vegetations and perforations of the anterior leaflet</td>
</tr>
</tbody>
</table>

SAM: systolic anterior motion.
Re-repair feasibility and technique

Mitral valve re-repair in the setting of recurrent MR has been reported by a few groups with re-repair rates in non-rheumatic aetiology ranging from 21 to 50% [7, 14–16]. In our study, we achieved a substantially higher repair rate. Notably, we were able to re-repair 90% of failed degenerative repairs compared with 36–48% in previous series [7, 14–16]. Our study differs from others, as ours was not a retrospective analysis of prior practice, but rather we have presented the results of a prospective and systematic strategy to re-repair all cases. We believe that it is this systematic commitment to re-repair that drives our very high re-repair rate. To achieve re-repair, we feel that it is critical to adhere to basic principles of mitral valve repair as outlined by Carpentier et al. [17], as one would in a primary repair.

The first step of re-repair is to do a thorough valve analysis and to identify the lesions causing regurgitation. If lesions are deemed treatable, the ring is typically removed and repair undertaken as we would in a primary case using conventional techniques. Where possible, we prefer to excise all old suture lines and existing artificial chordae. Occasionally, if the lesion is fairly simple—such as a new chord rupture without leaflet scarring—we have performed repair without the removal of the existing annuloplasty ring. An important key to achieving a durable re-repair is to avoid repeating the technical errors that caused the first repair to fail. If there were any uncorrected lesions at the first repair, leaving them inadequately corrected at re-repair will predispose to failure of the second repair. We therefore only persist with re-repair if we can confidently identify the lesion(s) causing failure and can reasonably expect that any identified lesion(s) will be adequately treated after re-repair. When there is extensive scarring and pannus ingrowth we replace such valves, as we are concerned that such reaction could also cause failure of a re-repaired valve.

Survival and durability of mitral valve re-repair

In previous studies, mitral valve re-repair was associated with superior survival, compared with valve replacement [7, 14]. In our study, the number of patients who underwent replacement was not large enough to be compared with the re-repair group, but the re-repair group had an excellent mid-term survival and durability. Despite having a very low threshold for re-repair, our mid-term results remain comparable with those of previous studies [7, 14–16]. Future study is, however, required to see how these re-repairs will fare in the longer term.

Table 4: Details of failed re-repair cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Age at re-repair</th>
<th>Original aetiology</th>
<th>Mode of failure in initial repair</th>
<th>Re-repair technique</th>
<th>Interval to failure (years)</th>
<th>Severity of MR in the last echo (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>Degenerative</td>
<td>Technical (SAM)</td>
<td>P2 shortening, Gore-Tex to A2, new annuloplasty</td>
<td>0.5</td>
<td>Moderate, 0.5 years</td>
</tr>
<tr>
<td>2</td>
<td>81</td>
<td>Degenerative</td>
<td>Technical</td>
<td>Posterior annulus decalcification, patch augmentation, new annuloplasty</td>
<td>1</td>
<td>Moderate, 1 years</td>
</tr>
<tr>
<td>3</td>
<td>47</td>
<td>Degenerative</td>
<td>Progression of native disease</td>
<td>Gore-Tex to P3, new annuloplasty</td>
<td>2.6</td>
<td>Moderate, 4.8 years</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>Degenerative + healed endocarditis</td>
<td>Technical</td>
<td>Commissuroplasty, gap closure between P1, 2 and 2, 3</td>
<td>4.6</td>
<td>Moderate, 4.8 years</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>Congenital</td>
<td>Progression of native disease</td>
<td>Patch augmentation, annuloplasty</td>
<td>3.5</td>
<td>Moderate, 3.5 years</td>
</tr>
</tbody>
</table>

MR: mitral regurgitation; SAM: systolic anterior motion.

There are several factors that may limit the robustness or generalizability of our study results. First, our institution is a high-volume mitral valve reference centre with a dedicated valve repair team. Secondly, our repair rates for primary valves are much higher than is seen in most of the centres [1], and this may partly explain the high repair rates in the re-repair setting. Thirdly, because of our interest in re-repair, there may be a referral bias such that patients coming to us for re-repair may not be typical of those seen in other centres. Fourthly, the sample size is relatively small, with a limited long-term follow-up. Finally, patients in this study had their initial mitral valve repair at different institutions with a variable level of expertise, such that our observations on failures of repair may not necessarily be representative of all valve centres. Regardless, our systematic approach to re-repair and the rigorous prospective data collection in our study are unique and allowed.
for detailed analysis of repair failure and re-repair—we believe that this strength outweighs the limitations.

Conclusion

Re-repair of the mitral valve is feasible in most cases of non-rheumatic disease, particularly in failed degenerative repair where up to 90% of valves are re-repairable. Mid-term outcomes are very good with valve re-repair. The relatively high prevalence of technical failures as the mechanism of failure of the initial mitral valve repair is a reminder that there remains a need for continuing development, refinement and outcome analysis of surgical practice and techniques in mitral valve repair.

Conflict of interest: David H. Adams receives inventor royalties from Edwards Lifesciences and Medtronic.

REFERENCES


APPENDIX. CONFERENCE DISCUSSION

Dr T. Mesana (Ottawa, ON, Canada): This is an important study from a very successful, expert mitral valve repair centre, Dr Adams’ group, whom everybody here knows.

You presented 53 patients who underwent reoperation after failed non-rheumatic mitral valve repair, a majority of which were mitral valve prolapse. You described a very significant rate of repair of 90% in the subset of degenerative valves, which is much higher than the previously reported re-repair rate in the literature, so your results are really remarkable. You also described very good long-term results, at least in terms of durability of your re-repair, because you had 80 to 90% at three and five years. So very, very impressive results.

Importantly, also in only 14 out of the 53 patients was the initial surgery performed in your own centre. The remainder, over two-thirds of the patients, were from other centres, which I think is also a mark of the excellence of your centre.

So I have one comment and a couple of questions that I hope you will not find too hard. The first comment is that you should not present non-degenerative data which creates some pollution. I think two-thirds of your data, or even more, were on degenerative mitral valve prolapse initially repaired, and there were 15 other patients. In five of these 15 you didn’t even know the aetiology, and there were two endocarditis and a few congenital. You should just focus on the re-repair of the degenerative mitral valve prolapse, for which you have enough material.

An important piece of information from your presentation today concerns a significant number of patients who had so-called mini-mitral somewhere else. My question is, since many centres are doing more mini-mitral without the required level of expertise in repair, do you expect to see more and more of these patients coming to your centre? And a supplementary question to that one is, in these patients where you had re-repaired through a sternotomy and who had a previous right thoracotomy of some sort, do you think that the first time you would have done a mini-thoracotomy or you would have done a sternotomy?

Dr Castillo: One thing that I would like to highlight is that it is not that uncommon in our centre to have patients referred from other centres. In terms of approach, we favour a small midline incision usually between 8 and 10 cm. In the reoperative setting, we have also favoured a sternotomy approach regardless of the first incisional approach, although we limit the dissection of the left heart. I am not sure we can attribute the need for re-repair based on the choice of access approach, but it is true that a right thoracotomy approach does require additional experience, and may be associated with inadequate repair in less experienced hands.

Dr Mesana: The majority of the time, the reasons for re-repair is a technical failure at the first attempt, but you have not described any failure of neo-Gore-tex, which does exist. In particular in patients who are operated in the first place with neo-Gore-Tex and have very large ventricles, they start to downsize their ventricles with no MR and then the Gore-Tex becomes a little bit too long and then they have some degree of MR. So my question is, why have you not seen neo-Gore-Tex disease? Is it because you don’t believe it happens or because around your centre there are not so many surgeons doing a pure Gore-Tex technique?

Dr Castillo: Well, in our centre, the Gore-Tex technique is not really used as frequently as it can be used in Europe. We most commonly combine resection with neochordal techniques, using Gore-Tex only as a support to either anterior leaflet prolapse or as a support for posterior leaflet resection, which I will show in our next talk.

In terms of modes of failure, approximately one-third were technical failure, one-third progression of the disease, what we call neochordal ruptures, and the remaining third a mixture of new lesions.

And regarding the last point, we had a failure four months after surgery because of PTFE chordal rupture that we just sent in for publication. PTFE chords actually rupture. And we changed: we used to use S-0 for the loop-in-loop and now we are moving towards using only 3-0 because of these problems.

Dr W. Saber (Cairo, Egypt): Can you tell us about your surgical technique to have this very nice view of the mitral valve and the subvalvular structures in redo cases regarding the type of dissection or left atrial incision?

Dr Castillo: We use the same approach for every single patient. Even if it is a re-repair case, we proceed with median sternotomy, and go through Söndergaard’s groove.
Dr Saber: And you free the heart completely during dissection?
Dr Castillo: Yes.
Dr Saber: Totally, posteriorly, laterally?
Dr Castillo: As much as possible.
Dr R. Varghese (New York, NY, USA): If I could just answer, I was able to make it out of New York and thank Javier for presenting on our behalf.
Just to answer some of those questions, we don’t dissect the heart out off the lateral wall in the reop. The majority of patients are cannulated prior to opening the sternum. They have a CAT scan preoperatively. If we are concerned at all about the aorta or right ventricle being close to the sternum we cannulate. Almost 90% of the patients will have axillary cannulation prior to opening the sternum.
And with respect to the question of Dr. Mesana regarding chordal failure, we have seen some incidence of chordal failure. As Javier mentioned, we had one loop-in-loop technique where the Gore-Tex neo-chord ruptured. In the majority of cases that we have seen with chordal failure, the chordal length is inappropriate in that they are either too long or too short and result in restriction.