Cardiology in the Young (2014), **24**, 1104–1107
doi:10.1017/S1047951114002017

Original Article

A three-step technique for repair of rheumatic disease of the mitral valve*

Matthew P. Thomas, Vinay Badhwar

Center for Mitral Valve Disease, Heart and Vascular Institute, University of Pittsburgh, Pittsburgh, Pennsylvania, United States of America

Abstract: This manuscript presents a technically straightforward technique to allow for mitral valve repair in the patient with rheumatic mitral stenosis. This non-resection technique allows for the correction of both mitral stenosis and regurgitation without requiring complex subvalvar procedures and eliminates the concerns for postoperative systolic anterior motion. The authors feel this three-part technique of bi-commissural release, anterior leaflet augmentation, and oversized annuloplasty may allow for a more reproducible approach to repair of the rheumatic mitral valve.

Keywords: Rheumatic heart disease; mitral stenosis; mitral repair; surgical techniques

Received: 14 September 2014; Accepted: 14 September 2014

Despite its reduced prevalence in developed countries, the global burden of rheumatic heart disease represents a significant cause of morbidity and mortality. Rheumatic heart disease is responsible for ~332,000 deaths annually, with an estimated 15.6 million people currently affected.[1,2] Acute rheumatic fever most commonly affects children between 6 and 15 years of age, with < 20% of initial episodes occurring in the adult population.[3] Rheumatic fever is caused by antecedent incompletely/untreated treated group A streptococcal pharyngeal infection. Antibody cross-reactivity secondary to molecular mimicry is thought to be the causative factor in the wide-spread inflammation occurring typically 2–3 weeks after acute infection. This may result in neurologic, integumentary, orthopaedic, and cardiovascular pathology.

Specific to the cardiovascular system, a pancarditis is typically observed. Endocardial inflammation with subsequent fibrosis affecting mainly the left-sided heart valves is seen in about 50% of patients with acute rheumatic stenosis.[4] T-cells, activated by binding with anti-streptococcal M-protein antigens, invade the valvar endothelium and release tumour necrosis factor and interleukins.[5] Classic valvar changes are observed, including leaflet thickening, commissural fusion, premature calcification, and chordal thickening and restriction. After a latent period that may range from months to decades, valve destruction reaches clinical significance, but may be observed earlier in the course of the disease. The mitral valve is most commonly affected. A history of rheumatic fever can be elicited from 60% of patients presenting with isolated mitral stenosis, with a higher percentage observed in those presenting with juvenile stenosis. The most common findings of rheumatic mitral disease include leaflet thickening and fibrosis, commissural and subvalvar fusion, followed by late calcific degeneration.

Given the relatively young age of those presenting for surgical correction of rheumatic mitral valve disease, coupled with the current limitations of valve replacement, a durable mitral valve repair may provide an attractive surgical option. Traditionally, valve repairs for rheumatic mitral stenosis were directed at simple commissurotomy. Although balloon mitral

*Presented at All Children's Hospital Johns Hopkins Medicine 14th International Symposium on Congenital Heart Disease, Saint Petersburg, Florida, 15–18 February 2014, Special Focus: Diseases of the Cardiac Valves from the Fetus to the Adult, Co-Sponsor: The American Association for Thoracic Surgery (AATS).

Correspondence to: V. Badhwar, MD, Center for Mitral Valve Disease, Heart and Vascular Institute, University of Pittsburgh, Presbyterian Hospital, 200 Lothrop Street, Suite C700, Pittsburgh, PA 15213, United States of America. Tel: +412 648 6314; Fax: +412 692 2184; E-mail: badhwarv@upmc.edu

valuloplasty offers a transcather option for commissurotomy for those with favourable anatomy, some patients with advanced mitral valve disease require more complete repairs consisting not only of commissurotomy but more complex procedures involving intricate choral adjustments and leaflet procedures. Secondary to the surgical complexities involved, many surgeons opt for valve replacement over repair in this population. Valve replacements in the young are clearly not ideal as they guarantee the necessity of future reoperation as well as the obvious prosthetic liabilities that accompany this option. Valve repair in the juvenile patient with significant rheumatic mitral disease offers the potential for a warfarin-free solution to their mitral stenosis and/or regurgitation.

We present a technically straightforward technique to allow for mitral valve repair in the patient with rheumatic mitral stenosis. Our non-resection technique allows for the correction of both mitral stenosis and regurgitation without requiring complex subvalvar procedures and eliminates the concerns for postoperative systolic anterior motion. We feel this three-part technique[6] of bi-commissural release, anterior leaflet augmentation, and oversized annuloplasty may allow for a more reproducible approach to rheumatic mitral valve repair.

Operative technique

The procedure is most often performed through standard median sternotomy. A generous pericardial patch is harvested upon entry and prepared in 0.6% glutaraldehyde bath for 8–10 minutes, followed by two subsequent 6-minute rinses in 0.9% saline solution. Standard aortic and bicaval cannulation is utilised. Antegrade, and optional retrograde, cardioplegia is used for cardiac protection. After cardioplegic arrest, the mitral valve is exposed through the

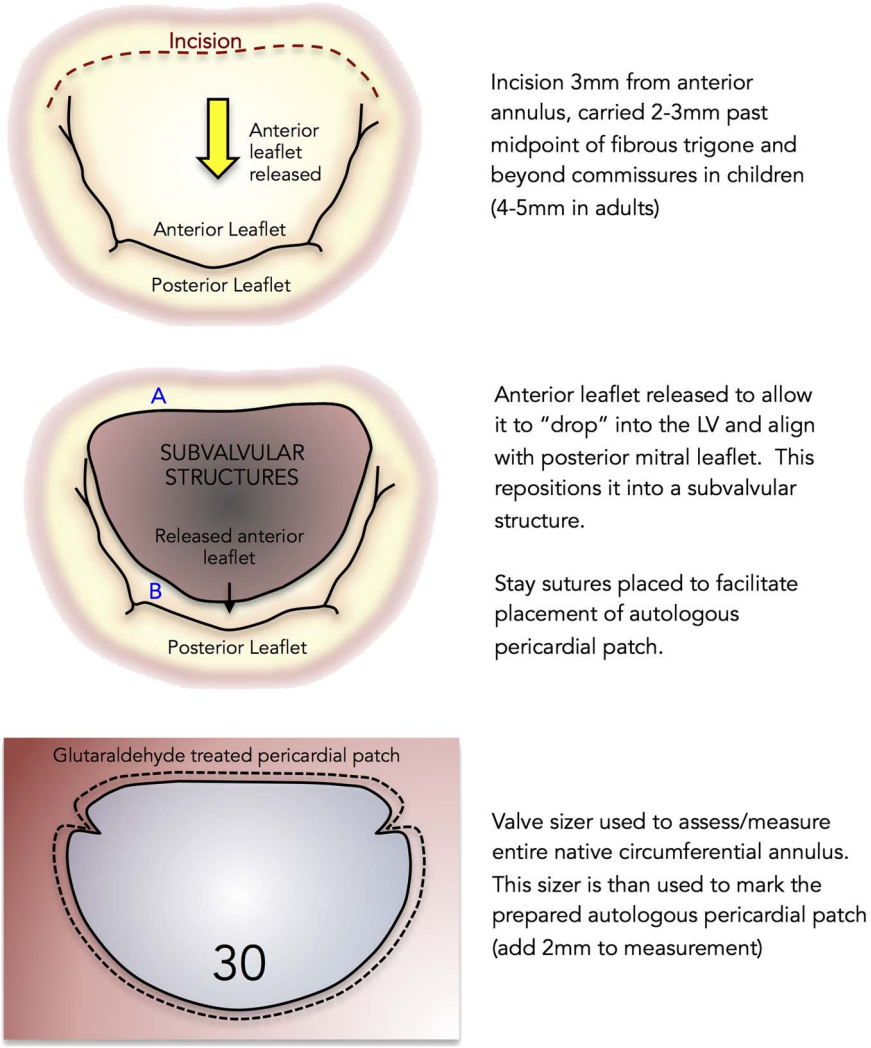

Figure 1.

https://doi.org/10.1017/S1047951114002017 Published online by Cambridge University Press

interatrial groove. It is of paramount importance to assure complete visualisation of the entire anterior and posterior leaflets. After thorough valve analysis, bi-commissural release is performed. The commissures are incised ~3 mm from the mitral annulus with the incision progressing towards the mitral orifice and this extends through the mid portion of the papillary muscle. The anterior leaflet is then detached from the annulus and allowed to drop into the left ventricle to form the new zone of coaptation. This is performed by first incising the anterior leaflet 2–3mm from the hinge point. The incision is initiated at the midpoint of the leaflet and progresses laterally 2–3mm beyond the midpoint of both fibrous trigones (Fig 1). When completed, the anterior leaflet tissues should "drop" into the left ventrical, with the atrial portion of the leaflet tissue now participating in the coaptation zone. In addition, the anterior leaflet should completely lie along the posterior leaflet across the entire line of coaptation. An annular sizer is utilised to determine the entire circumference of the mitral orifice. Subsequently, this functions as a template for the creation of the autologous pericardial patch. When cutting the patch to size, 2 mm is added to the measured dimensions circumferentially to the selected sizer. The patch is sewn to the cut edges of the anterior leaflet using 4-0 or 5-0 monofilament polypropylene suture. The procedure is completed with an oversized annuloplasty ring. A complete annuloplasty ring should be avoided so as to not disrupt the anterior suture line. We use a semi-rigid band upsized 1- or 2-sizes (Fig 2).

Results

From July, 2011 to July, 2014, we have successfully applied this technique in 35 patients. There were no operative or 30-day mortalities in our series. Immediate postoperative echocardiographic valve analysis confirmed correction of mitral stenosis and regurgitation. All patients had mitral gradients < 5 mmHg, and demonstrated none-to-trivial mitral regurgitation. One patient in our series required reoperation for severe, asymptomatic recurrent mitral regurgitation. In this case of a 48-year-old dialysis-dependent woman with calcific disease, reoperation confirmed an intact anterior augmentation with a prolapsing P3 scallop as the causative aetiology and she was managed with prosthetic mitral valve replacement. We continue to follow-up our patients in our multidisciplinary mitral valve clinic. Clinical follow-up is 86% (30/35) complete. Complete follow-up is obtained, but the remaining achieved though annual reports from other countries where the cases were performed. Thus far, there have been no late deaths or reoperations. Median follow-up is

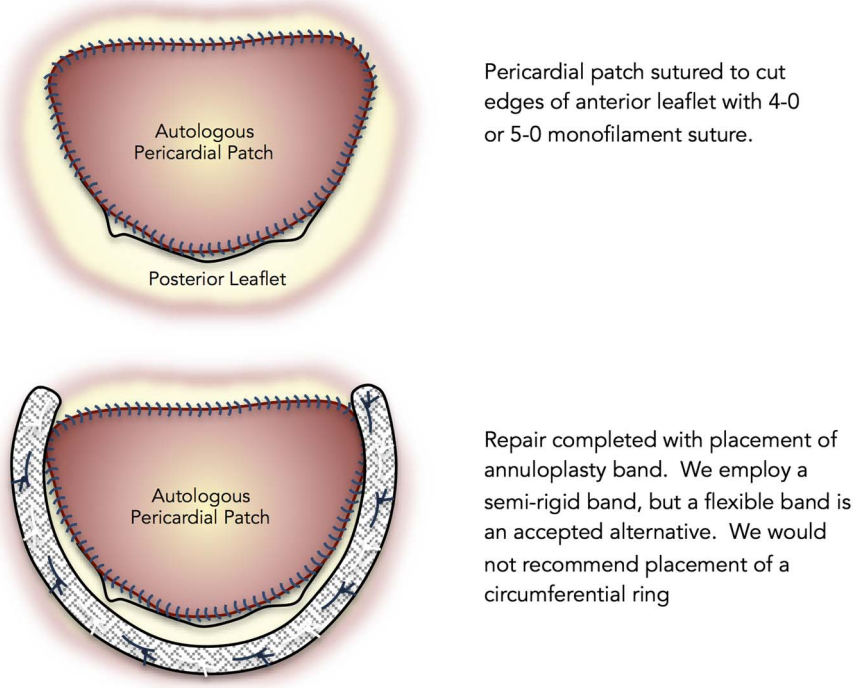

Figure 2.

https://doi.org/10.1017/S1047951114002017 Published online by Cambridge University Press

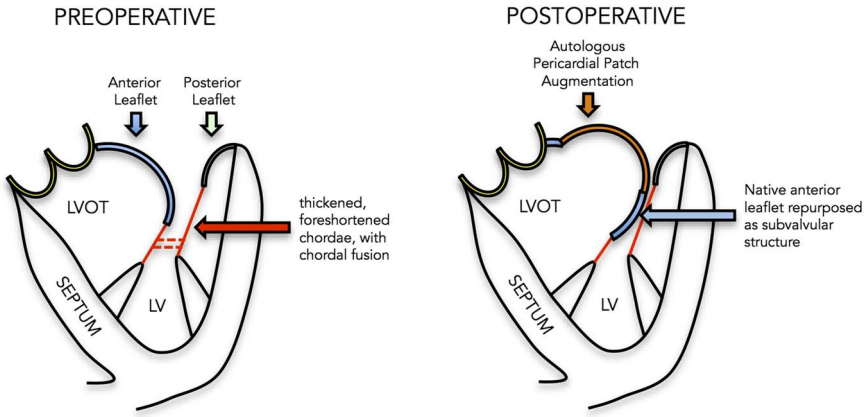

Figure 3.

2 years, with echocardiographic follow-up demonstrating no significant recurrent stenosis or regurgitation.

Discussion

Rheumatic mitral repair must address the five pathoanatomic changes present in this disease: commissural fusion, subvalvar restriction, leaflet immobility, reduced coaptation depth, and overall reduced mitral orifice area. We feel that the above technique addresses all of these components with relative technical simplicity. Commissural fusion is corrected with bi-commissural release. Subvalvar restriction, leaflet immobility, and reduced coaptation depth are all addressed by repurposing the native anterior leaflet as a subvalvar structure. This negates the need for complex neo-chordal replacement procedures, as the height of the released native anterior leaflet that has been augmented often compensates for pre-existing chordal foreshortening. A large mitral orifice area is assured with bilateral commissurotomies and the addition of autologous pericardium for leaflet augmentation. In addition, the coaptation zone either remains in its normal anatomic position or is displaced slightly towards the posterior leaflet. This, coupled with the sail-like effect obtained by leaflet augmentation, effectively eliminates any concerns for postoperative systolic anterior motion (Fig 3). This repair technique provides a clear benefit for young children with symptomatic rheumatic mitral stenosis or regurgitation to avoid the liabilities of a prosthesis or warfarin. Ongoing follow-up is critical to confirm long-term durability.

References

1. Karthikeyan G, Zuhlke L, Engel M, et al. Rationale and design of a global rheumatic heart disease registry: the REMEDY study. Am Heart J 2012; 163: 535–540. e1.
2. World Heart Federation. Rheumatic heart disease fact sheet. World Heart Federation, 2012. Retrieved August 15, 2014, from http://www.world-heart-federation.org/press/fact-sheets/rheumatic-heart-disease.
3. Kumar V, Abbas AK, Fausto N, Mitchell RN. Robbins Basic Pathology, 8th edn. Saunders Elsevier, Philadelphia, Pennsylvania, United States of America, 2007: 403–406; ISBN: 978-1-4160-2973-1.
4. Caldas AM, Terreri MT, Moises VA, et al. What is the true frequency of carditis in acute rheumatic fever? A prospective clinical and Doppler blind study of 56 children with up to 60 months of follow-up evaluation. Pediatr Cardiol 2008; 29: 1048–1053.
5. Guilherme L, Cury P, Demarchi LM, et al. Rheumatic heart disease: proinflammatory cytokines play a role in the progression and maintenance of valvular lesions. Am J Pathol 2004; 165: 1583–1591.
6. Chan PG, Hayanga AJ, Badhwar V. Repair of rheumatic mitral stenosis with anterior leaflet augmentation, bi-commissural release and oversized annuloplasty: a durable and simplified approach. Multimed Man Cardiothorac Surg 2014; 2014:mmt020. doi: 10.1093/mmcts/mmt020. PMID: 24435097.