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COMPARISON OF ARTIFICIAL NEOCHORDAE AND NATIVE CHORDAL TRANSFER IN THE REPAIR OF A FLAIL POSTERIOR MITRAL LEAFLET: AN EXPERIMENTAL STUDY

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Abstract

BACKGROUND—Surgical reconstruction of a flail posterior leaflet is a routine mitral valve repair, the techniques for which have evolved from leaflet resection to leaflet preservation. Artificial ePTFE neochordae are frequently used to stabilize the flail leaflet, and seldom translocation of the native secondary chordae of the valve to the leaflet free edge is used. In this study, we sought to investigate the efficacy of the two techniques to correct posterior leaflet prolapse and reduce mitral regurgitation, and quantify the acute post-repair leaflet kinematics.

METHODS—Adult porcine mitral valves ($N = 7$) were studied in a pulsatile left heart experimental model in which isolated P2 flail was mimicked by marginal chordal transection. Baseline conditions were established in each valve under normal conditions (control), and followed by induction of isolated P2 flail by transecting the two marginal chordae on the posterior leaflet free edge (disease). The flail posterior leaflet was reconstructed using artificial neochordae (repair 1) and then native chordal translocation (repair 2). Reduction in leaflet flail, changes in mitral regurgitation fraction, leaflet coaptation length, and posterior leaflet mobility were measured using B-mode echocardiography or color doppler.

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RESULTS—At baseline, all the valves were competent with no mitral regurgitation. After transection of the marginal chordae on the posterior leaflet, isolated P2 flail was evident with $13.7 \pm 13\%$ regurgitation. Reconstruction with artificial neochordae eliminated leaflet flail and reduced mitral regurgitation to $3.2 \pm 2.8\%$, and with chordal translocation leaflet flail was corrected and mitral regurgitation was measured at $2.3 \pm 2.6\%$. Using either repair techniques, leaflet coaptation and mobility of the repaired leaflets were adequate and comparable to the baseline measurements.

CONCLUSIONS—Comparable reduction leaflet flail and regurgitation, and restoration of physiological leaflet coaptation with the two techniques indicates that under acute conditions, use of artificial neochordae or native chordal translocations have similar benefits.

Keywords

Mitral valve repair; Posterior leaflet flail; Neochordae; Chordal transfer; Primary chordae; Secondary chordae; Mitral valve hemodynamics; Valve surgery; Cardiac surgery

INTRODUCTION

Isolated posterior leaflet flail is a common cause of mitral regurgitation (MR) and is amenable to surgical repair(1,2). Surgical techniques to repair this lesion have evolved from quadrangular resection(2), to limited triangular resection(3), and now to leaflet preserving artificial neochordal placement(4). The benefits of preserving the leaflet over resection are now appreciated, and there is increasing evidence of its benefits on long-term durability. Translocation of the native chordae is another leaflet preservation technique that was proposed for mitral repair, but has not gained as much clinical attention(5). It is based on the hypothesis that intact native chordae on a mitral leaflet can be re-located to a position on the same leaflet where there is a need for [additional] support. Efficacy of this technique in pediatric mitral valve repair, anterior mitral valve repair and in correcting functional mitral regurgitation in adults was already reported, though its application in repair of a flail posterior leaflet is unknown(5–7). This technique is simple and might serve as a surrogate to neochordoplasty to correct a flail leaflet, when applicable. Especially, in the setting of isolated posterior leaflet flail caused due to rupture of the marginal chordae, the secondary chordae are intact and may be re-located to the marginal edge of the same leaflet. In this study, we sought to investigate the efficacy of such chordal translocation in correcting posterior leaflet flail in a controlled experimental model, and compare with artificial neochordal replacement, the current gold standard for reconstruction of the posterior mitral leaflet.

MATERIAL AND METHODS

Specimen Selection

Porcine hearts were obtained from the local abattoir and transported to the laboratory on ice. Seven hearts (n=7) with an average mitral valve size of 28mm were used for this study. Each valve was sized using an Edwards Lifesciences® Physio II ring sizer, by measuring the anterior leaflet size and the distance between the trigone-to-trigone. Valves were extracted from these hearts and sutured on to a flexible silicone mitral annulus, whose planar area can be increased to a valve size of 40 and whose out-of-plane saddle shape can be adjusted from flat to 30% saddle. The valves were then mounted into a pulsatile left heart simulator for hemodynamic testing.

Pulsatile Left Heart Simulator

The pulsatile left heart simulator used in this study is a well-established setup that was previously used for similar mitral valve hemodynamic studies(8,9). The simulator is a computer controlled pressure-driven, compressible bladder system that generates the pulsatile flow and pressure conditions of the left heart as shown in Figure 1. The simulator has a left atrium, left ventricle and a silicone mitral annulus placed between the two chambers. The annulus plate has a D-shaped ring made of silicone that is flexible, allows increase in its planar area and can be changed in its non-planarity from a flat to a saddle shape. The mitral annulus was sutured onto the silicone ring with 3-0 silk sutures in a mattress fashion and papillary muscles were mounted on two positioning rods in the left ventricular chamber. Upon mounting the valve into the simulator, 0.9% saline solution (vol/vol) was used as the working media and the valve was studied under adult hemodynamic conditions of 70 beats/min at 120 mm Hg peak trans-mitral pressure and 5 L/min cardiac output. Trans-mitral pressure gradient was measured with a differential pressure transducer (DP9-40; Validyne Engineering, CA) and controlled via solenoid valves, and the flow rate was measured with an electromagnetic flow probe on the pulmonary vein from the lung reservoir to the left atrium (600; Breamer Inc, NC). The acquisition, signal conditioning, and analysis were performed with DAQCard 1200 (National Instruments, TX) and a custom data processing program (DAQ-ANAL 2.0).

Experimental Protocol

Each valve was studied under the experimental conditions depicted in Figure 2 -

Control/baseline—The annulus was first maintained at size 28 (the physiological size for this study) and then increased to 34mm to mimic the low range of annular dilatation seen in most patients with regurgitation from posterior leaflet flail(10). The papillary muscles were fixed in their normal positions; i.e. their tips perpendicular to the mitral annular plane and in the plane passing through leaflet coaptation. The valve was studied under pulsatile conditions, and hemodynamic and echocardiographic data were acquired.

P2 Leaflet Flail—Marginal chordae inserting into the free edge of the posterior leaflet central cusp (P2) were identified and isolated from rest of the chordae, and carefully transected at their insertion points into the leaflet using a 11-0 Scalpel blade. Leaflet flail was confirmed by filling the left ventricle passively with saline for each valve, and a photograph was taken to ensure that the % of the leaflet with flail remains similar to a reasonable extent. This model was previously used to compare the hemodynamic outcomes of leaflet resective and non-resective techniques(8). The valve was then studied under pulsatile conditions and necessary data were acquired.

Artificial Neochordae Placement—ePTFE sutures (CV-5, TH-22 sutures, GOR-E-TEX, AZ) were used for replacement of the transected chordae, similar to the clinical setting. Four neochordae loops were implanted along the free edge of the prolapsing P2 cusp, with two loops per papillary muscle. The loops were spaced out in a manner such that the entire flail leaflet edge was supported structurally. Once the chordae were inserted, the LV was filled passively with saline enabling mitral valve closure, and the chordae were then tied down on a pledget on the atrial side of the leaflet at their insertion sites. Each valve was then studied under pulsatile conditions and the measurements were repeated.

Chordal Translocation—Neochordae were carefully removed from the leaflets, and the intact secondary chordae on the P2 cusp were identified and carefully transected at their insertion zone into the leaflet using a scalpel. A double needle 5-0-prolene suture was carefully tailored into the tip of the secondary chorda, and the chord was moved few

millimeters (~3–5mm) radially towards the leaflet free edge and sutured into the leaflet, and reinforced with a Teflon pledget. The LV was filled with saline and valve closure was examined and the pulsatile flow measurements were repeated.

Measurement Methods and Endpoints

Regurgitant Fraction—Regurgitant fraction was measured as the ratio of the backward flow through the mitral valve to the total forward inflow through the mitral valve, both of them measured using the electromagnetic flow probe. Data were acquired over 15 consecutive cardiac cycles, averaged, and the area under the positive and negative part of the curve was measured. The regurgitant fraction was measured as the ratio of the backward flow to the total flow and expressed as a percentage fraction.

Leaflet Coaptation Length—2D M-mode echocardiography was performed on all valves, with the cardiac timing synchronized with the ultrasound system (Philips Medical Systems, Andover, MA). The images were exported into a custom image processing plugin that enabled conversion of the black and white images into negatives, which allowed better resolution of the images to measure the leaflet coaptation length. Coaptation length was defined as the distance from where the two leaflets meet to the end of the point where the two leaflets end or do not overlap anymore.

Posterior Leaflet Mobility—Using the same echocardiographic images, mobility of the repaired posterior leaflet was defined as the angle between the repaired leaflet and the mitral annulus at peak systole. It was hypothesized that if leaflet mobility was impaired after the repair, the measured systolic angle would be large as the leaflet cannot move from its diastolic to peak systolic position. The images were blinded, and were analyzed by persons trained in echocardiographic image analysis who had no knowledge of the purpose of the study.

Statistical Analysis

For each parameter, the data were checked for normality using the Anderson-Darling test in Minitab 15. Upon confirming normality, data were presented as mean $\pm$ standard deviation, and a paired t-test was used to compare the different groups.

RESULTS

Regurgitation Fraction

At baseline, none of the valves had mitral regurgitation and even after dilatation of the annulus to 34mm, trace mitral regurgitation was measured. Transection of the marginal chordae on the P2 cusp induced isolated P2 prolapse and moderate mitral regurgitation (13.7 ± 12.7). Placement of neochordae reduced regurgitation to trace levels (3.2 ± 2.8), corroborating the clinical data on its efficacy in restoring physiological valve function. Translocation of the anterior chordae tendineae to the marginal position had comparable hemodynamic outcomes, with a reduction in regurgitation to trace levels as well (2.34 ± 2.6) as shown in Figure 3A.

Leaflet Coaptation Length

At baseline, the mean coaptation length was 1.1 ± 0.4 cm at the A2 and P2 cusp overlap region as measured on 2D echocardiography. With P2 prolapse, complete loss of leaflet coaptation was measured along the central cusps. Neochordoplasty restored leaflet coaptation to physiological levels of 1.4 ± 0.4 cm, which was achieved even with chordal translocation where a coaptation of 1.25 ± 0.4 cm was measured as shown in Figure 3B.

Posterior Leaflet Mobility

Table 1 summarizes the posterior angle measured pre and post repair using neochordoplasty and chordal translocation. As expected, both the procedures restored physiological valve hemodynamics and coaptation without disturbing the native motion of the posterior leaflet.

COMMENT

Isolated posterior leaflet flail from acute chordal rupture or chordal elongation is the most frequent cause of mitral regurgitation observed clinically. Patients with this lesion are surgically treated, either with valve replacement or valve repair. In the current era of valve surgery, improved anatomical and functional knowledge of the native mitral valve has led to the adoption of Carpentier resective techniques(2) and Frater non-resective techniques(1) into the surgical armamentarium, both of which have shown acceptable outcomes. Neochordoplasty and chordal translocation are two such non-resective techniques that were developed several decades ago, with neochordoplasty gaining popular clinical use over chordal translocation(11,12). Though the benefits or failings of chordal translocation over neochordoplasty are not well understood, the use of native secondary chordae to repair leaflet flail has gained better traction. In this study, for the first time a quantitative comparison of chordal translocation with neochordoplasty in correcting posterior leaflet flail and repairing mitral regurgitation is presented.

Acute reduction of leaflet flail and mitral regurgitation was successful with chordal translocation, with hemodynamic valve function restored to baseline level and comparable with neochordoplasty. From the disease state, an 83% reduction in mitral regurgitation was achieved with chordal translocation, with trace mitral regurgitation left in some valves. It is reasonable to expect such reduction in mitral regurgitation, as the secondary chordae on the posterior leaflet are as extensible and mechanically strong as the posterior marginal chordae tendineae(13–16); thus acting as good replacements for the previously ruptured marginal chordae. Interestingly, loss of the two secondary chordae from their basal positions did not seem to impact the valve hemodynamics, unlike on the anterior leaflet, where removal of the secondary chordae alters the leaflet motion, valve function and chordal force distribution(9,9,17,18). In this study, however, measurement of the chordal forces before and after chordal translocation was not attempted, and warrants future study.

Physiological levels of leaflet coaptation after chordal translocation indicate that it is possible that the primary and secondary chordae may be functionally redundant on the posterior leaflet, unlike the anterior leaflet. This is a deductive speculation based on the fact that leaflet coaptation and mobility did not seem to change when the secondary chorda was transferred to the primary chordal position. Previous studies had demonstrated that the primary, secondary and tertiary chordae on the mitral anterior leaflet have specific role in controlling the leaflet coaptation, systolic shape and mobility. However, this study was unable to delineate any drastic changes in the posterior leaflet after chordal translocation, which might make the use of this technique attractive. Leaflet mobility after chordal translocation was unaltered as well, indicating that the secondary chordae are sufficiently extensible, and thus do not restrict the leaflet in systole. Structurally, the posterior leaflet is smaller than the anterior and the length of the primary and secondary chordae are similar in most valves. This structural uniqueness might be advantageous in a complex repair, where determining the correct length of the neochordae may be challenging. In addition to the procedural and functional benefits, comparable leaflet coaptation lengths and peak systolic shapes/curvatures indicate a similar stress distribution on the mitral valve leaflets after the two procedures. This deduction is based on a simplistic Laplace law, but direct leaflet strain measurements and finite element modeling may confirm these findings, both of which were not conducted in this study.

An important advantage of chordal translocation over neochordoplasty is the use of native secondary chordae tendineae that naturally emerge from the papillary muscle tip. These chordae consist of strong collagen core with surrounding elastin filaments, which allow for adequate extensibility and excellent strength, as much as an ePTFE suture loop. Secondly, the procedural complexity is significantly reduced as the chord is resected at the leaflet and can be easily relocated to a different part of the leaflet with a simple pledgeted knot, with minimal risk of dehiscence. Concerns regarding dehiscence of the suture through the chord may arise, but such potential failings can be overcome with the use of pledgets, as currently used with the neochordal technique. Thirdly, the ruptured marginal chordae and the translocated secondary chordae typically emerge from the same chordal stem and often have similar height, which reduces the risk of residual prolapse after chordal translocation. These benefits highlighted in this manuscript, are limited to those observed in this experimental model, but are supportive of gradual clinical adoption of this technique in appropriate patients.

Conclusions

In conclusion, this study demonstrates that secondary chordal translocation is an effective technique to correct posterior leaflet flail and associated mitral regurgitation. While neochordoplasty remains the procedure of choice, chordal translocation is an equally simple and effective technique that may be used when deemed appropriate by the surgeon, for equivalent results obtained with current techniques.

Limitations

Although this study demonstrates the efficacy of chordal translocation, it has some inherent limitations within the context of which the results must be interpreted. The experimental model is an idealized setting of P2 flail, and is not encompassing of the clinical spectrum of this lesion. Clinically, the size and extent of flail varies significantly between patients, and such variability will be challenging to mimic in this study. The valves used in this study are normal valves induced with acute flail, since it is not possible to get whole diseased flail valves from humans or animals for this study. In the actual clinical setting, the valves vary in the extent of their degeneration, and sometimes resection of the leaflet or complete valve replacement might be appropriate. While the model mimics the dynamic hemodynamics and motion of the valve, contraction of the papillary muscles is not reproduced. However, since papillary muscle contraction is considered to alter valve opening but not valve closure, the results of this study should hold. Notwithstanding these limitations, this study demonstrates the hemodynamic efficacy of chordal translocation, and provides the basis for its potential use for correction of this lesion.

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Figure 1.
Schematic of the in vitro pulsatile left heart simulator in which a native mitral valve was used to mimic isolated posterior leaflet flail and repair.

Figure 2.
Schematic of the experimental design for the study.

Figure 3.

(A) Changes in the regurgitation fraction in the setting of posterior leaflet flail and post repair using neochordae and chordal translocation; (B) Leaflet coaptation length at baseline and post-neochordoplasty and chordal translocation

Table 1

Posterior leaflet angle measured as an index of systolic leaflet mobility at baseline and post-repair in each of the tested mitral valves.

	Baseline	Neochordoplasty	Chordal Translocation
Posterior Leaflet Angle	29 ± 1.3	35.1 ± 2.4 ^{\$}	34 ± 3.2 ^{\$}

^{\$} implies statistically significant difference compared to baseline