Abstract

Introduction: Isolated aortic valve repair (AVR) has been gaining increasing interest in recent times. Results of isolated aortic valve repair have been reported to be variable. Various techniques have been utilized. We analyzed our experience with isolated valve repair using autologous pericardial patch plasty and compared the results to an age-matched collective with aortic valve repair without the use of additional material.

Methods: Between January 1997 and June 2005, pericardial patch plasty of the aortic valve was performed in 42 patients (PATCH). During the same period, 42 patients after AVR without the use of additional material were age matched (NO-PATCH). Mean age in both groups was 52 years with a majority of male patients (PATCH ratio, 3.7:1; NO-PATCH ratio, 5:1). Valve anatomy was similar in both groups. All patients were followed by echocardiography for a cumulative follow-up of 2341 patient months (mean 28 ± 23 months).

Results: No patient died in the hospital in neither group. The average systolic gradient was 5.9 ± 2.2 mmHg in PATCH and 4.8 ± 2.1 mmHg in NO-PATCH; p = 0.17. Freedom from aortic regurgitation ≥ I° was 87.8% in PATCH and 95.0% in NO-PATCH after 5 years (p = 0.21). Freedom from reoperation was 97.6% in PATCH and 97.4% in NO-PATCH (p = 0.96).

Conclusions: Aortic regurgitation can be treated effectively by aortic valve repair using pericardial patch plasty. The functional results are satisfactory. With the application of this technique also more complex pathologies of the aortic valve can be addressed adequately thus extending the concept of valve preservation in patients with aortic regurgitation.

Keywords: Valvular disease; Aortic valve; Valve repair

1. Introduction

Aortic valve repair has evolved as a surgical alternative for aortic valve regurgitation in the past two decades. With increased experience, various pathomechanisms have been understood and addressed surgically. Aortic root dilatation in the presence of normal cusps as the most common pathology could be corrected by valve preserving replacement of the proximal aorta. Good results have been reported with different modifications [1,2]. An isolated cusp prolapse either with or without root dilatation could be corrected with good results normal and sufficient cusp tissue provided [3,4]. Thus, the regurgitant aortic valve can be corrected in the majority of cases by restoration of normal root dimensions and/or normalization of cusp configuration. In more complex pathologies, such as retraction, calcification or destruction of the cusps aortic valve repair remains challenging and demands additional surgical tools.

Aortic valve regurgitation due to defects of the cusp tissue represents a higher level of complexity in both pathology and repair strategy. Structural changes of one or more of the cusps may have significant implications on the geometry and function of the aortic valve as a whole. The recognition of the exact lesion and the selection of the adequate surgical tools are mandatory for a successful repair. As in most other valve repair procedures, the understanding of the pathomechanism depends mostly on experience. The proper addressing of the specific problem demands a high surgical flexibility, therefore not always allowing for reproducibility.

Attempts to replace cusp tissue with biological material have already been made since the late 1960s. Facia lata, dura mater, and bovine pericardium have been utilized in small number of patients but the results have not been favorable in the majority of cases [5–7]. Autologous pericardium is easily available for immediate aortic valve repair in every patient. Thus, it appears to be an ideal material to correct structural defects of the aortic valve cusps. There has been only limited experience with the utilization of autologous pericardium for aortic valve repair in the past [8]. It appears that in the presence of considerable structural defects of the cusps most surgeons prefer to replace the valve.

We have used autologous pericardium for aortic valve repair procedures systematically in the past 9 years. We have
retrospectively analyzed our experience with pericardial patch repair of the aortic valve and compared the results to those of cusp repair without the addition of material.

2. Patients and methods

Between January 1997 and June 2005, a total of 428 patients underwent reconstructive surgery for pure aortic regurgitation with or without concomitant cardiac or aortic disease. Of these, 171 patients were treated by aortic valve repair without concomitant replacement, such as root remodeling or valve reimplantation. In 42 patients aortic valve repair was performed utilizing an autologous pericardial patch thus representing the study group (PATCH). Forty-two additional patients after aortic valve repair without the use of additional tissue were matched for age, comorbidity, and valve anatomy from the overall collective and served as control (NO-PATCH).

2.1. Operative technique

All patients were operated upon using median sternotomy and cannulation of ascending aorta and right atrium. In presence of acute aortic dissection, the femoral or right axillary artery was selected for arterial cannulation. After cross-clamping and transverse aortotomy, cardioplegic arrest was induced by infusion of cold blood cardioplegia directly to the coronary ostia.

The aortic root was then assessed systematically. The degree of root dilatation was determined measuring both sinotubular (ST) and aorto-ventricular (AV) diameters. If ST diameter was below 33 mm and AV diameter below 30 mm, valve reconstruction without root replacement was considered feasible. In tricuspid valves, cusp prolapse was assessed using a 7-0 Prolene suture (Ethicon, Hamburg, Germany) passed through the nodules of Arantii. The suture was then placed under gentle traction in several directions, thus facilitating the estimation of the relative length of all free cusp margins. Cusp prolapse was defined as an excessive length of one or more free cusp margin (more than 3 mm).

In the presence of prolapse, the cusp margin was shortened by plication of the central portions of the free cusp margin (5-0 or 6-0 Prolene, Ethicon). Single sutures were placed until identical length of all cusp margins was achieved.

In the presence of structural defects, autologous pericardium was used for cusp tissue replacement and/or augmentation. A patch of pericardium of adequate size was taken and fixed on a piece of carton with hemoclips. The pericardium was tanned in a 0.2% glutaraldehyde solution for 1—2 min and then rinsed in physiologic saline solution.

The dimension of the pericardial patch was determined according to the size and shape of the structural defect of the cusp. In most occasions, the patch was intentionally left too large and was stepwise tailored to fit. Any remaining excessive patch tissue was plicated with the suture (6-0 Prolene). The continuous sutures were anchored on both sides in the cusp tissue. If replacement of the free cusp margin with pericardium was necessary, the free edge was reinforced using a 6-0 PTFE running suture taking special care not to shorten the neo-cusp margin (n = 7).

In tricuspid aortic valves congenital fenestrations with prolapse were corrected with implantation of an autologous pericardial patch (n = 15), shortening the free margin with the patch dimensions (Fig. 1). Additional cusp plication was performed as necessary. Perforations as a correlate of healed endocarditis were primarily closed with autologous pericardium (n = 7). In the presence of cusp retraction the valve tissue was augmented at its free margin by a strip of autologous pericardial pericardium (n = 6).

In bicuspid valves similar procedures were applied. A median raphe with pliable tissue quality was not resected, but direct plication of the fused cusp was performed at its free margin. Triangular resection of the median raphe with re-approximation of the two rudimentary cusps was only undertaken if direct suture adaptation was difficult (fibrosis, calcification, extreme redundancy of cusp tissue). In the presence of extreme calcifications, the resected cusp tissue was replaced with an autologous pericardial patch; n = 8; Fig. 2). Unicuspid valves were corrected by augmenting cusp tissue creating a bicuspid valve. The fused commissure(s) were separated and the resulting cusp defects augmented by implantation of pericardial patches (n = 6; Fig. 3).
Calcifications of the cusp base as a concomitant pathology were present in two patients with tricuspid and three patients with bicuspid valve anatomy. In these instances, the cusp was decalcified to achieve better mobility. In two cases, a tissue defect after decalcification had to be closed by autologous pericardium. Severe calcification of the aortic annulus was not encountered.

The root was plicated beneath the commissures in most procedures (PATCH, \( n = 24 \); NO-PATCH, \( n = 31 \)) to stabilize repair in both bicuspid and tricuspid anatomies [9]. This was omitted in the presence of small root dimensions. In all procedures, aortic valve configuration and cusp coaptation was initially assessed filling the aortic root with saline.

Postoperatively patients received normal thrombosis prophylaxis with low-molecular heparin subcutaneously. In the presence of vascular prosthesis, patients were treated with low-dose acetyl salicylic acid (100 mg per day). Anticoagulation therapy with coumadin derivates was only used in the presence of persistent atrial fibrillation.

2.2. Echocardiography

After weaning from cardiopulmonary bypass, reconstructed aortic valve was assessed by transesophageal echocardiography (HDI 3000, ATL Technologies, Hagen, Germany; Sequioa Ultrasound System, Acuson GmbH, Nürnberg, Germany) under stable conditions and maintenance of a diastolic blood pressure of 70 mmHg. The degree of valve incompetence was analyzed in a semiquantitative fashion by intensity and slope of the regurgitation signal (continuous wave Doppler) and the width of the regurgitation jet in relation to the diameter of the left ventricular outflow tract (color Doppler) [10]. Systolic flow gradients across the valve were measured by continuous wave Doppler. For postoperative surveillance, transthoracic echocardiography was performed at discharge and at 3, 6, 9, and 12 months and every 12 months thereafter.

All echocardiographies were performed by the same experienced echocardiographers (H.F.L., D.A., F.L.) and documented on videotape.
All data were recorded retrospectively and mean values ± standard deviations were calculated. Actuarial Kaplan–Meier analysis of freedom from significant aortic regurgitation and freedom from reoperation were calculated. Data were statistically analyzed by contingency table and χ²-test or analysis of variance (ANOVA) when applicable (SigmaStat 2.0, SPSS Inc., Chicago, IL, USA). Kaplan–Meier curves were compared by Mantel–Haenszel test (Prism 3.0, GraphPad Software Inc., San Diego, CA, USA). p-values of less than 0.05 were considered statistically significant.

3. Results

Patients in both groups were of similar age (PATCH, 51.8 ± 19.1; NO-PATCH, 52.1 ± 18.3; \( p = 0.94 \)). Male-to-female ratio was comparable in both groups (PATCH, 3.7:1; NO-PATCH, 5:1, \( p = 0.78 \)). The presence of bicuspid valve anatomy was comparable in both groups (PATCH, 16/42; NO-PATCH, 20/42; \( p = 0.51 \)).

The average degree of preoperative aortic regurgitation was higher in the patients undergoing aortic valve repair with a pericardial patch (3.4 ± 0.8 vs NO-PATCH, 2.8 ± 0.9; \( p = 0.04 \)). In both groups, the most common comorbidity was coronary artery disease and additional pathology of the mitral valve (Table 1). The mean times of cardiopulmonary bypass were similar in both groups (PATCH, 80.2 ± 25.0; NO-PATCH, 71.9 ± 23.2; \( p = 0.12 \)). The time of aortic cross-clamp was significantly longer after cusp repair with a pericardial patch (56.4 ± 18.0 vs NO-PATCH, 48.4 ± 6.5; \( p = 0.008 \)).

No patient died in the perioperative period in either group, and no patient had to be returned to the operation theatre for postoperative bleeding. After patch repair, one patient suffered a stroke periopeatively (2.4%). In the control group, one patient suffered from aspiration pneumonia (2.4%). There was no occurrence of AV-block in patients of neither group.

Early postoperative echocardiographic assessment of the degree of aortic regurgitation showed comparable results in both groups (PATCH, 0.8 ± 0.9; NO-PATCH, 0.7 ± 0.6; \( p = 0.65 \)). Peak systolic gradients across the valve were 5.9 ± 2.2 mmHg after patch repair and 4.8 ± 2.1 mmHg after aortic valve repair without patch use (\( p = 0.17 \)).

Follow-up ranged from 3 to 103 months with mean follow-up of 28 months (cumulative follow-up 195 patient years). In the PATCH group, all patients were alive. In the NO-PATCH group, two patients died during follow-up. One patient died of unknown cause 14 months postoperatively, the other patient died 21 months postoperatively due to ventricular arrhythmia. Thromboembolism or anticoagulation-associated bleeding was not observed in any patient of both groups. One patient had to undergo reoperation with valve replacement for acute endocarditis 3 months after pericardial patch repair of the aortic valve in another center. The transeosophageal echocardiography showed healed endocarditic destruction with an annular abscess. Peak systolic gradients did not change during follow-up. Freedom from significant aortic regurgitation (≥II°) at 5 years was 87.8% after patch repair and 94.9% after no-patch repair REMO (\( p = 0.21 \); Fig. 4). The mechanisms of recurrent aortic regurgitation following patch repair were a recurrent mild cusps prolapse due to a rupture of a plication suture in two cases, the presence of two commissural jets in one patient, and the finding of a central regurgitation jet in one patient, which has decreased over time. In the NO-PATCH group, two patients were again found to have moderate aortic regurgitation due to recurrent prolapse after decalcification of the cusps. Reoperation-free survival was 97.6% after patch repair and 97.4% after aortic valve repair without the use of pericardium (\( p = 0.96 \); Fig. 5).

4. Discussion

The concept of aortic valve preserving operations in patients with aortic valve regurgitation has gained wider acceptance in recent years. The systematic approach to the individual pathology has led to a better understanding of the problem. In an increasing number of patients the native valve has been preserved thus avoiding the necessity of a valve prosthesis and its associated comorbidity [11].
A variable degree of root dilatation has been identified in more than 50% of contemporary patients with aortic regurgitation [12, 13]. Several modifications of valve preservation have been introduced and brought into clinical routine with good long-term results [1, 2]. Cusp prolapse with or without root dilatation has been identified as a common pathomechanism responsible for aortic valve regurgitation [14]. In our total experience we have found it to occur in more than 70% of regurgitant aortic valves. Different repair techniques have been increasingly applied [15–18]. The overall results have been favorable, although the necessity for cusp repair has been reported as a risk factor for repair failure [19].

Thus, in aortic regurgitation not infrequently a more complex pathology is encountered intraoperatively. In addition, structural defects can be present such as congenital fenestrations, postendocarditic perforations, posterothematic retractions of the cusps, or other congenital anomalies. These defects demand the application of additional surgical tools, such as cusp tissue replacement.

The results of cusp tissue substitution with various materials (fascia lata, dura mater, bovine pericardium) have not been uniformly favorable in the past [5–7]. In retrospect it remains unclear whether these results were a consequence of the material used or the technique applied. In the past two decades only few groups systematically investigated and utilized autologous pericardium for partial or total cusp replacement [17, 20, 21]. The advantage of autologous pericardium is its easy availability. Pretreatment with glutaraldehyde prevents secondary shrinkage and possibly also calcifications. Little is known about the long-term fate of pericardium implanted in aortic valve position [21, 22].

Our systematic application of pericardial patches in more complex pathology shows good functional results, which are almost identical to prolapse repair without the implantation of material. The degree of aortic regurgitation is significantly reduced. There was no operative mortality and only a limited comorbidity of the procedure. Myocardial ischemic times were not longer than the ones of other cardiac procedures. The repaired valves showed a good stability as judged by a freedom from reoperation of close to 98% and a freedom from significant aortic regurgitation of close to 88% after 5 years. The systolic pressure gradients remained stable over time. We did not see relevant calcifications yet. The incidence of endocarditis was also low with a follow-up of up to 7 years.

In addition to good results with pericardial as material, our results also indicated that aortic valve morphology more complex than simple prolapse is amenable to repair with good results.

While repair does place the patient at risk for early reoperation also, valve replacement is associated with some risk of reoperation in the first years after implant (e.g., prosthetic valve endocarditis). The risk of reoperation is not increased by previous repair, and indeed we have found replacement after repair technically easier than re-replacement of failing prosthesis.

Repair, however, requires standardized and systematic approach [23]. The major challenge to surgical judgment is that in addition to root dilatation and prolapse structural defects have to be corrected. Despite possible concerns, we have been able to reproducibly achieve good aortic valve geometry and function.

Because of the obvious limitation of this small series of a single center, more experience with larger patients numbers and a true long-term follow-up are required to provide more reliable information.

Due to our experience, however, the majority of regurgitant aortic valves due to defects of cusp tissue can be repaired successfully using autologous pericardium to substitute cusp tissue. The results are similar to those of less complex aortic valve repair procedures and comparable to more established operations such as valve-preserving aortic replacement [1, 2] or the pulmonary autograft [24, 25]. We thus feel that the augmentation or partial replacement of diseased aortic cusps with autologous pericardium is safe and feasible. As this tool extends the possibilities of aortic valve repair, we advocate its consideration as a surgical alternative to valve replacement also in the presence of more complex valve pathologies.

References


Appendix A. Conference discussion

Dr M. Song (Seoul, Korea): I am afraid there are several points I can’t agree with you. The aortic leaflet is a very sensitive structure, which is able to shrink and expand during the cardiac cycle. But the pericardium which was used for leaflet correction can’t adjust the dimensional change of the aortic root especially at the sinotubular junction during the cardiac cycle. So unless you restrict the motion of the sinotubular junction together with leaflet correction with pericardium, the progression or recurrence of aortic regurgitation is inevitable. I always place an internal ring at the sinotubular junction to prevent damage to the corrected leaflet by restricting the motion of sinotubular junction when I perform aortic leaflet repair with pericardium. With this method, I haven’t experienced any significant progression of aortic regurgitation in my recent 47 cases.

Dr Lausberg: I am not sure I understand the question right. As this is a clinical study, we did not do any mechanical evaluation in the lab. For stabilization, we do place subcommissural plication sutures for stabilization as introduced first by Cabrol in the majority of patients, especially in bicuspid anatomy. The average mean systolic valve gradients have been between 5 and 6 mmHg, and they stay stable through the follow-up.

Dr R. Dion (Leiden, The Netherlands): I have two short questions. First, are you treating the fresh autologous pericardium with glutaraldehyde, or don’t you treat it first? And how frequently are you using the reinforcement of the free edge of the aortic cusp with Gore-Tex: do you use it, and in which circumstances?

Dr Lausberg: To answer your first question, we did not use untreated ‘fresh’ pericardium. The harvested pericardial patch was fixed in a 0.2% glutaraldehyde solution for 1–2 min on the table. Regarding the second question, we have performed reinforcement of the free margin of the neo-cusps, especially when rheumatic cusp retraction was corrected by decalcification and, if necessary, limited stenotic component, for example, in the presence of calcifications of the cusp base, which can be corrected by decalcification and, if necessary, pericardial patch replacement. But in patients with a relevant stenosis as indicated by a gradient of more than 10–15 mmHg, the probability of successful repair is lower.