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Valve: Research

Management of Mild or Moderate Calcific Aortic Valve Disease in Patients Undergoing Operation for Subaortic Obstruction

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ABSTRACT

BACKGROUND This study sought to evaluate short- and long-term outcomes of aortic valve (AV) intervention by decalcification or replacement for the treatment of mild to moderate calcific AV stenosis (AS) in patients undergoing transaortic septal myectomy for subaortic left ventricular outflow tract (LVOT) obstruction.

METHODS Between 2000 and 2023, study investigators identified 137 consecutive patients undergoing myectomy with or without membranectomy for mild to moderate calcific AS. Of these patients, 93 underwent surgical intervention on the AV and formed the primary cohort; 44 patients who did not undergo AV intervention were used as a control group. Subaortic LVOT obstruction was the primary indication for surgery.

RESULTS The median age of patients with AV intervention was 71.3 years (range, 66.5-76.9 years), and 50.5% of the patients were female. Forty-four patients (47.3%) underwent AV decalcification, whereas 49 (52.7%) had AV replacement, with no significant differences in clinical features. Valvular cusp calcification was less extensive in the decalcification group ($P < .001$). Early postoperatively, AV gradients improved in both groups (each $P < .001$) to similar extents. No patients in the decalcification group experienced severe AV regurgitation. Rates of AV reintervention were similar between groups ($P = .84$) and were lower than in the control group. Long-term survival was comparable between both groups and matched patients who underwent isolated myectomy without AV disease.

CONCLUSIONS Mild to moderate AS may complicate operative treatment of subaortic LVOT obstruction in adults. When surgical management is guided by visual determination of the extent of cusp calcification, AV decalcification yields good early hemodynamic results and late survival and reoperation rates similar to AV replacement.

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Calcific aortic valve (AV) disease resulting in AV stenosis (AS) is the most common valvular disorder in the United States, and it occurs in approximately 3 million individuals aged more than 75 years.[1] A global epidemiologic study reported an estimated 12.6 million cases of calcific AV disease in 2017.[2] Guideline-based class I indications for AS intervention include symptomatic severe AS, asymptomatic severe AS with left ventricular ejection fraction <50%, and asymptomatic severe AS

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in patients undergoing cardiac surgery for other indications.[3,4] For patients with mild or moderate AS who are undergoing cardiac surgery for other indications, there is limited evidence (American College of Cardiology/American Heart Association [ACC/AHA] class 2b indication and European Society of Cardiology/European Association for Cardio-Thoracic Surgery class 2a indication) for the need for valve intervention,[3,4] and the dilemma is whether to proceed with valve replacement, which may be premature, or leave the valve while anticipating slow progression of stenosis.

Transaortic septal myectomy (SM) for relief of dynamic subaortic obstruction is used increasingly in older patients with obstructive hypertrophic cardiomyopathy (oHCM). In our practice, 29.1% of patients undergoing SM are aged 65 years or older,[5] and concomitant calcific AV disease is not uncommon in this older cohort. Further, surgical decision making regarding associated mild or moderate valvular AS at the time of myectomy differs from that in other cardiac procedures because the surgeon may visually assess the valve and has easy access for intervention.

Our management strategy for associated calcific AV disease with resulting mild or moderate AS encountered during operation for SM has been based on direct visual assessment of the extent of cusp calcification. The aim of this study was to evaluate short-term and long-term outcomes of AV replacement or AV decalcification for the treatment of mild or moderate calcific AS in patients undergoing transaortic SM for subaortic left ventricular outflow tract (LVOT) obstruction.

MATERIAL AND METHODS

STUDY DESIGN. The Mayo Clinic Institutional Review Board (IRB) in Rochester, Minnesota approved the study (IRB #23-010210, October 23, 2023), and all patients authorized the use of their clinical data for research. Between 2000 and 2023, 2662 patients underwent transaortic SM with or without membranectomy. From this cohort, we included 137 patients who had mild or moderate calcific AS on preoperative echocardiographic examination or intraoperative visual assessment. From that group, 93 patients had surgical intervention on the AV and constituted the primary cohort in our study. The remaining 44 patients had mild AS on preoperative echocardiography but did not undergo intervention because of minimal or absent calcification on the basis of intraoperative evaluation, and they were used as a control group in our secondary analysis. Excluded from the study were patients with AV gradients >40 mm Hg, patients who underwent concomitant procedures on the ascending aorta, and patients in whom aortic regurgitation (AR) was the primary AV disease. Our inclusion and exclusion criteria are summarized in a flow diagram (Supplemental Figure 1). The annual trends of surgically treated patients are illustrated in Supplemental Figure 2. More than 80% of the procedures were performed by 2 surgeons.

DATA COLLECTION AND STUDY GROUP. Relevant demographic characteristics, comorbidities, echocardiographic data, operative data, immediate postoperative outcomes, and long-term outcomes were collected from a prospectively maintained institutional cardiovascular surgery database. Variables were defined according to the criteria outlined by The Society of Thoracic Surgeons Adult Cardiac Surgery Database. The diagnosis of oHCM was assigned by experienced cardiologists and cardiac surgeons. Comprehensive resting transthoracic echocardiography (TTE) was performed in all patients before SM and before hospital discharge, and studies were performed in accordance with the guidelines of the American Society of Echocardiography. The degree of AS was quantified by the mean aortic transvalvular gradient. In 11 patients, it was not possible to distinguish between subaortic and valvular velocity measurements to calculate gradients, so the severity of valve stenosis (mild or moderate) was assessed from AV area measurements (valve planimetry). Details pertaining to AV morphology and degree of calcification were manually obtained from operative reports and from preoperative TTE and transesophageal echocardiography reports.

OPERATIVE METHOD FOR AORTIC VALVE DECALCIFICATION. For all patients, the AV was inspected after initial aortotomy, with subsequent steps contingent on the condition of the valve cusps. In instances where the cusps were extensively calcified, in particular when calcification involved all 3 cusps, the cusps were excised, and the annulus was debrided as necessary for planned AV replacement. The myectomy was then performed, and the prosthesis was implanted in the standard fashion.

Conversely, if the calcified cusps exhibited sufficient flexibility, especially if calcification predominantly involved only 1 cusp, a decision was made to repair with decalcification. First, the

SM was performed. If a stiffened noncoronary cusp impeded exposure of the subaortic septum, it was retracted by a stay suture passed through the nodule of Arantius,[6] thus allowing the myectomy to proceed. On completion of the myectomy, decalcification of the cusps was performed manually using a pituitary rongeur (Supplemental Figure 3). Deposits of calcium were gently removed from the outflow surface, with special care taken not to damage the structural integrity. An operative Video illustrating the procedure of aortic cusp decalcification is provided in the Supplemental Material.

OUTCOMES OF INTEREST. The end points of interest in this study were change in AV mean gradient, AR at the time of follow-up TTE, need for AV reintervention, and long-term all-cause mortality. AV reintervention was defined as the need for AV replacement as a result of severe AV restenosis or AR in patients who previously underwent a decalcification procedure or prosthetic valve dysfunction (thrombosis or regurgitation) in those who had initial AV replacement.

STATISTICAL ANALYSIS. Categorical variables are presented as frequencies and percentages, and continuous variables are presented as medians with interquartile ranges (IQRs). Given the small sample size, we assessed baseline and operative differences between AV replacement and decalcification groups by using standardized differences and statistical tests. Standardized differences were computed as differences in proportions or means divided by standard errors. Standardized differences >0.2, 0.5, and 0.8 indicated small, moderate, and large baseline imbalances, respectively. Statistical differences across groups were compared using χ^2 and Fisher exact tests for categorical variables and the Mann-Whitney *U* test for continuous variables. The Wilcoxon signed rank test and the McNemar test for paired proportions were used to assess changes in AV gradient and AV regurgitation, respectively.

The Kaplan-Meier method was used to assess long-term survival rates. To assess the safety of adding an AV intervention on long-term survival, we used propensity matching, pairing the patients in our cohort (91 patients) with an equal number of patients from the general HCM cohort who underwent isolated transaortic SM. Adjustments were made for baseline differences, including sex, body mass index, diabetes, and the need for concomitant coronary artery bypass grafting (CABG). Additional details, including the matching process and covariates, are presented in the Supplemental Material (Supplemental Figure 4). The need for AV reintervention among the AV replacement and decalcification groups was examined through survival with competing risk analysis, considering death as the competing risk. As a secondary analysis, we examined the importance of AV intervention in delaying progression to severe stenosis by comparing the rate of AV reintervention in our cohort with that of patients with mild AS and minimal cusp calcification who did not undergo an AV procedure at the time of SM. The baseline characteristics of the control group are presented in the Supplemental Material. Analysis was conducted using R software version 4.1.3 (R Foundation), and some of the graphs were designed in GraphPad Prism version 10.

RESULTS

BASELINE CHARACTERISTICS AND ECHOCARDIOGRAPHIC VARIABLES. Baseline characteristics are presented for the primary cohort consisting of patients undergoing SM and concomitant AV procedures. The characteristics for the control group who did not have any AV intervention are provided in Supplemental Table 1. The median age of patients in the primary cohort was 71.3 years (IQR, 66.5-76.9 years), and 47 patients were female (50.5%). Most patients (n = 87; 94%) had a diagnosis of oHCM, and 8 patients (8.6%) underwent excision of a subaortic membrane in addition to SM. Sixty-four (83.1%) patients were in New York Heart Association functional class III to IV (Table 1).

Transaortic myectomy was performed in all patients. Forty-four patients (47.3%) underwent concomitant AV decalcification, whereas 49 (52.7%) had AV replacement. Patients in the AV decalcification and replacement groups were similar regarding all clinical features, with no statistically significant ($P > .05$) differences in any baseline characteristics. On the basis of standardized difference values, imbalances among surgical groups were absent for all variables (SD ≤0.2) except for previous sternotomy, diabetes, and oHCM diagnosis, where imbalances are judged to be small or moderate (0.2 <SD <0.5).

Regarding echocardiographic variables, the preoperative resting LVOT gradient was similar in the AV decalcification vs replacement group (67.5 [IQR, 30-92]; 62.5[IQR, 33.2-88]; $P = .94$). The transvalvular mean AV gradient was also comparable in the decalcification group (22.5 mm Hg [IQR, 15.8-28 mm Hg]) and the replacement group

TABLE 1 Baseline and Echocardiographic Characteristics of Patients Undergoing Septal Myectomy and Concomitant Aortic Valve Decalcification or Replacement

Variables	Aortic Surgery Type		Total Cohort	*P* Value	Standardized Difference
	Replacement	Decalcification			
Female, n (%)	25 (51)	22 (50)	47 (50.5)	.922	0.02
Age, y (IQR)	72.1 (66.7–77.4)	71.1 (66.2–76.5)	71.3 (66.5–76.9)	.712	0.14
Race, n (%)					
White	46 (93.9)	38 (92.7)	84 (93.3)	1	0.04
Other	3 (6.1)	6 (7.3)	9 (6.7)		
Body mass index, kg/m^2 (IQR)	29 (26–34.2)	29.8 (27.6–33.4)	29.6 (26.1–33.5)	.564	0.015
Diabetes, n (%)	9 (18.4)	12 (27.3)	21 (22.6)	.305	0.21
Creatinine, mg/dL (IQR)	1 (0.8–1.2)	1 (0.9–1.2)	1 (0.88–1.2)	.613	0.035
Endocarditis, n (%)	1 (2)	1 (2.3)	2 (2.2)	1	0.073
Cerebrovascular disease, n (%)	4 (8.2)	4 (9.1)	8 (8.6)	1	0.032
Hypertension, n (%)	41 (83.7)	37 (84.1)	78 (83.9)	.956	0.011
Current smoking, n (%)	3 (7)	2 (5)	5 (6)	1	0.084
NYHA functional class III or IV, n (%)	37 (84.1)	27 (81.8)	64 (83.1)	.792	0.061
Myocardial infarction, n (%)	6 (12.2)	4 (9.1)	10 (10.8)	.744	0.1
Recent AF, n (%)	8 (16.7)	5 (11.4)	13 (14.1)	.466	0.15
Chronic lung disease stage II to III, n (%)	7 (14.6)	4 (9.3)	11 (11.9)	.438	0.16
Previous sternotomy, n (%)	2 (4.1)	0 (0)	2 (2.2)	.496	0.29
Presence of implantable cardiac device, n (%)	4 (8.2)	2 (4.5)	6 (6.5)	.68	0.15
Use of beta-blockers, n (%)	32 (65.3)	31 (70.5)	63 (67.7)	.596	0.1
Diagnosis of oHCM, n (%)	49 (98)	41 (93.2)	89 (95.7)	.341	0.23
Transvalvular AV mean gradient, mm Hg (IQR)	25 (20–29)	22.5 (15.75–28)	24 (18.5–28)	.139	0.28
Aortic stenosis, n (%)					
Mild	12 (25)	18 (40)	30 (32.3)		
Moderate	37 (75)	26 (60)	63 (67.7)	.3	0.11
Resting LVOT gradient, mm Hg (IQR)	62.5 (33.2–88)	67.5 (30–92)	62.5 (31.75–88)	.939	0.06
Mild or moderate AV regurgitation, n (%)	21 (44.7)	18 (40.9)	39 (42.9)	.71	0.07

Values are n (%) or median (interquartile range (IQR)). Echocardiographic variables were obtained by transthoracic echocardiography. AF, atrial fibrillation; AV, aortic valve; LVOT, left ventricular outflow tract; NYHA, New York Heart Association; oHCM, obstructive hypertrophic cardiomyopathy.

(25 mm Hg [IQR, 20-29 mm Hg]) ($P = .139$ and SD = 0.28). The incidence of mild AS was 40% in the decalcification group and 25% in the replacement group ($P = .11$; SD = 0.3). There was no significant difference in the transvalvular AV gradient among patients with 1 calcified cusp (23 mm Hg [IQR, 16-28 mm Hg]), 2 calcified cusps (22 mm Hg [IQR, 15-25 mm Hg]), or 3 calcified cusps (25 mm Hg [IQR, 20-29 mm Hg]) (overall $P = .214$; pairwise P values = .288 and .751). No patients had severe AV regurgitation at the time of surgery. Further echocardiographic data are provided in Supplemental Table 2.

OPERATIVE DETAILS. Transaortic SM for relief of subaortic obstruction was the primary indication for surgery in all patients. Valvular disease was less extensive in the decalcification group, in which 32 patients (72.7%) had calcification in only 1 cusp, whereas in the AV replacement group, 33 patients (68.8%) had calcification in 3 cusps ($P < .001$) (Figure 1). In the AV replacement group, 38 patients (77.6%) received a bioprosthesis, and the remainder received a mechanical valve prosthesis. Thirteen patients (14%) had concomitant mitral valve procedures, and 24 (25.8%) had concomitant CABG; this was not significantly different across the 2 surgical groups ($P = 0.198$; SD = 0.27; and $P = .889$; SD = 0.034, respectively) (Table 2).

POSTOPERATIVE SHORT-TERM AND LONG-TERM OUTCOMES. Postoperative atrial fibrillation occurred less frequently in the AV decalcification group (31.8%) compared with the AV replacement group (55.1%; $P = .024$; SD = 0.5). Two patients (2.2%) had postoperative neurologic events, 8 (8.6%) required prolonged ventilation, and 36 (38.7%) needed postoperative blood products, but no significant differences were observed in these outcomes when comparing the 2 surgical groups. Two patients (2.2%) died during their hospital stay, and the causes of death are provided in Supplemental Table 3.

SM relieved subaortic obstruction in both surgical groups, and the median LVOT gradient

before hospital discharge was 0 mm Hg (0-0). The AV mean gradient before hospital discharge was similar in both groups (13 [10-17.7] and 12.5 [9.7-16.2]; $P = .48$; SD = 0.12). Figure 2 illustrates the reduction in AV gradient from surgery to hospital discharge, which was significant in both groups (−10.5 [−16 to −3.75] and −7 [−12 to −3.5]; $P < .001$) and comparable in extent ($P = .263$; SD = 0.22).

During a median follow-up period of 4.1 years (IQR, 1.4-7.9), 31 (33.3%) patients died. The 1-, 5-, and 10-year survival estimates for patients undergoing AV intervention (decalcification or replacement) with transaortic SM were 94% (95% CI, 88-99), 77% (95% CI, 67-88), and 50% (95% CI, 36-67), respectively (Figure 3A). The 1-year, 5-year, and 10-year survival estimates for a matched cohort of the general HCM population undergoing isolated transaortic SM without any AV intervention were 99 % (95% CI, 97-100), 87% (80-96), and 60% (47-77) (Figure 3A). There was no statistically significant difference in long-term survival between the 2 groups ($P = .31$).

When categorized into AV decalcification and replacement groups, 1-year survival was 92.3% (95% CI, 84.3-100) in the decalcification group and 95.6% (95% CI, 89.7-100) in the replacement group. Similarly, 5-year survival was 81.1% (95% CI, 67.9-96.7) and 70.7% (95% CI, 57.4-87.1), respectively. This is further illustrated in Figure 3B ($P = .17$).

Overall, 9 of the 91 hospital survivors (9.9%) underwent late AV replacement (or re-replacement), 6 in the decalcification group (all for valvular stenosis), and 3 in the AV replacement group (prosthetic valve degeneration or paravalvular leak). The 5-year cumulative incidence of valve replacement (or re-replacement) was 5.1% (95% CI, 0.76-34.26) in the decalcification group and 3.1% (95% CI, 0.46-21.68) in the replacement group. As seen in Figure 4A, the cumulative incidence function curves show no difference between the groups in the need for future AV reintervention ($P = .84$).

In our secondary analysis comparing the need for future AV intervention between patients who had concomitant AV surgery (replacement or decalcification) and a control group consisting of patients with mild AS and minimal cusp calcification but no AV intervention (Figure 4B), the cumulative incidence of future AV replacement was greater in the control group (5-year incidence of 11% [95% CI, 3-40]) compared with that of our cohort (5-year cumulative incidence of 4% [95% CI, 1-17]) ($P = .028$).

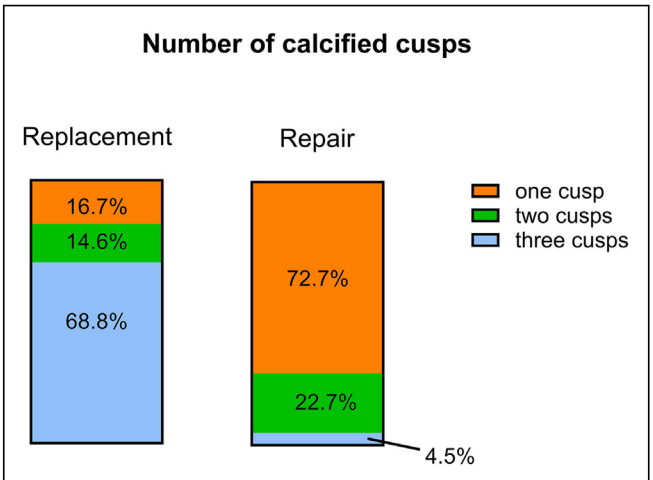

FIGURE 1 Number of calcified cusps in patients undergoing septal myectomy and concomitant aortic valve decalcification or replacement on the basis of intraoperative inspection.

In the AV decalcification group, 25 patients (56.8%) had long-term echocardiographic follow-up at a median of 3.9 years (IQR, 2.2-8.9 years); none experienced severe AR. Additionally, there was no significant change in the degree of AV regurgitation between the preoperative visit and the last postoperative follow-up visit (Figure 5) ($P = .55$).

COMMENT

Calcific AS is the most common acquired valvular heart disease in high-income countries, and its prevalence increases with age, by affecting 2% to 4% of individuals aged more than 65 years.[7] Many older patients with subaortic obstruction secondary to oHCM or other disease may have associated mild, moderate, or severe calcific AS.[8,9] The ACC/AHA guideline lists a class IIB indication for AV replacement in patients with moderate AS who are undergoing cardiac operations for other indications.[3]

The intraoperative decision in the management of patients with mild to moderate AS balances the risk of progression of valvular stenosis (and future intervention) with the unnecessary exposure to potential hazards of a prosthesis implanted prematurely. Multiple investigations have documented accelerated progression of AS in patients with calcific AV disease and baseline mild or moderate AS.[1,10,11] Amanullah and colleagues[12] reported that 62% of patients with moderate AS progressed to severe stenosis over a median follow-up of 2.5 years. More recently, Ye and

TABLE 2 Operative Details and Postoperative Outcomes of Patients Undergoing Septal Myectomy and Concomitant Aortic Valve Decalcification or Aortic Valve Replacement

Variables	Replacement	Decalcification	Total Cohort	*P* Value	Standardized Difference
Bicuspid valves, n (%)	3 (6.1)	2 (4.5)	5 (5.4)	1	0.071
Number of decalcified cusps, n (%)					
1	…	33 (68.8)	…	…	…
2	…	7 (14.6)	…	…	…
3	…	8 (16.7)	…	…	…
Type of artificial valve, n (%)					
Bioprosthesis	38 (77.6)	…	72.7	…	…
Mechanical prosthesis	11 (22.4)	…	…	…	…
Concomitant MV procedure, n (%)	9 (18.4)	4 (9.1)	13 (14)	.198	0.27
Concomitant CABG, n (%)	13 (26.5)	11 (25)	24 (25.8)	.866	0.034
Cross-clamp time, min (IQR)	59 (46–80.5)	28 (23–44.75)	46 (28–63.5)	<.001	1.36
Perfusion time, min (IQR)	70 (57–95)	40.5 (30.25–54.5)	57 (38.5–78.5)	<.001	1.16
Postoperative AF, n (%)	27 (55.1)	14 (31.8)	41 (44.1)	.024	0.49
Postoperative stroke, n (%)	2 (4.1)	1 (2.3)	3 (3.2)	1	0.1
Need for a pacemaker, n (%)	4 (8.2)	3 (6.8)	7 (7.5)	1	0.053
Prolonged ventilation, n (%)	6 (12.2)	2 (4.5)	8 (8.6)	.273	0.27
Need for postoperative blood transfusion, n (%)	22 (44.9)	14 (31.8)	36 (38.7)	.196	0.25
Renal failure, n (%)	2 (4.1)	2 (4.5)	4 (4.3)	1	0.019
Total ICU stay, h (IQR)	26.8 (20.7–67.5)	24.9 (21.2–43.1)	25.2 (21–52)	.327	0.37
Total hospital stay, d (IQR)	7 (5–9)	6 (5–7)	6 (5–8)	.09	0.29
Hospital death, n (%)	1 (2)	1 (2)	2 (2.2)	1	0
AV gradient before hospital discharge, mm Hg (IQR)	13.5 (10–17.7)	12.5 (9.7–16.2)	13 (10–17)	.486	0.12
Need for future AV replacement, n (%)	3 (6.1)	6 (13.6)	9 (9.7)	.299	0.23

Values are n (%) or median (interquartile range (IQR)). AF, atrial fibrillation; AV, aortic valve; CABG, coronary artery bypass grafting; ICU, intensive care unit; MV, mitral valve.

colleagues[13] demonstrated that in patients with nonsevere AS, the extent of valve calcification was a robust prognostic factor for long-term survival.

Studies of the progression of AS, however, are based on Doppler echocardiographic assessment of initial severity, and this assessment may be modified by direct intraoperative inspection of the valve cusps. Specifically, if direct inspection of the calcified AV cusps suggests less severe stenosis than that estimated by preoperative Doppler echocardiography, the options are leaving the valve as is or proceeding with decalcification to improve cusp mobility and perhaps delaying progression to severe stenosis. In the present study, we found that the need for future AV replacement was significantly greater in the control group, where calcification was not addressed during myectomy, compared with our cohort, where either AV decalcification or replacement was performed. Although the severity of AS and degree of calcification at baseline were more significant in the treatment group compared with the control group, surgical intervention at the time of myectomy appears to delay progress to severe AS requiring future AV replacement.

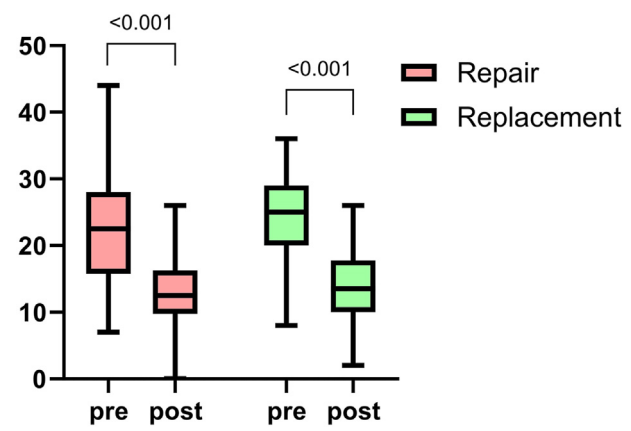

FIGURE 2 Change in aortic valve gradient before (pre) and after (post) surgical intervention in patients undergoing septal myectomy and concomitant aortic valve decalcification or replacement.

Mechanical AV decalcification with valvuloplasty was the first surgical therapy of calcific AS before prostheses became available.[14,15] Indeed, in the 1980s, there was a resurgence in interest in this technique with the introduction of ultrasonic devices for AV decalcification. However, multiple

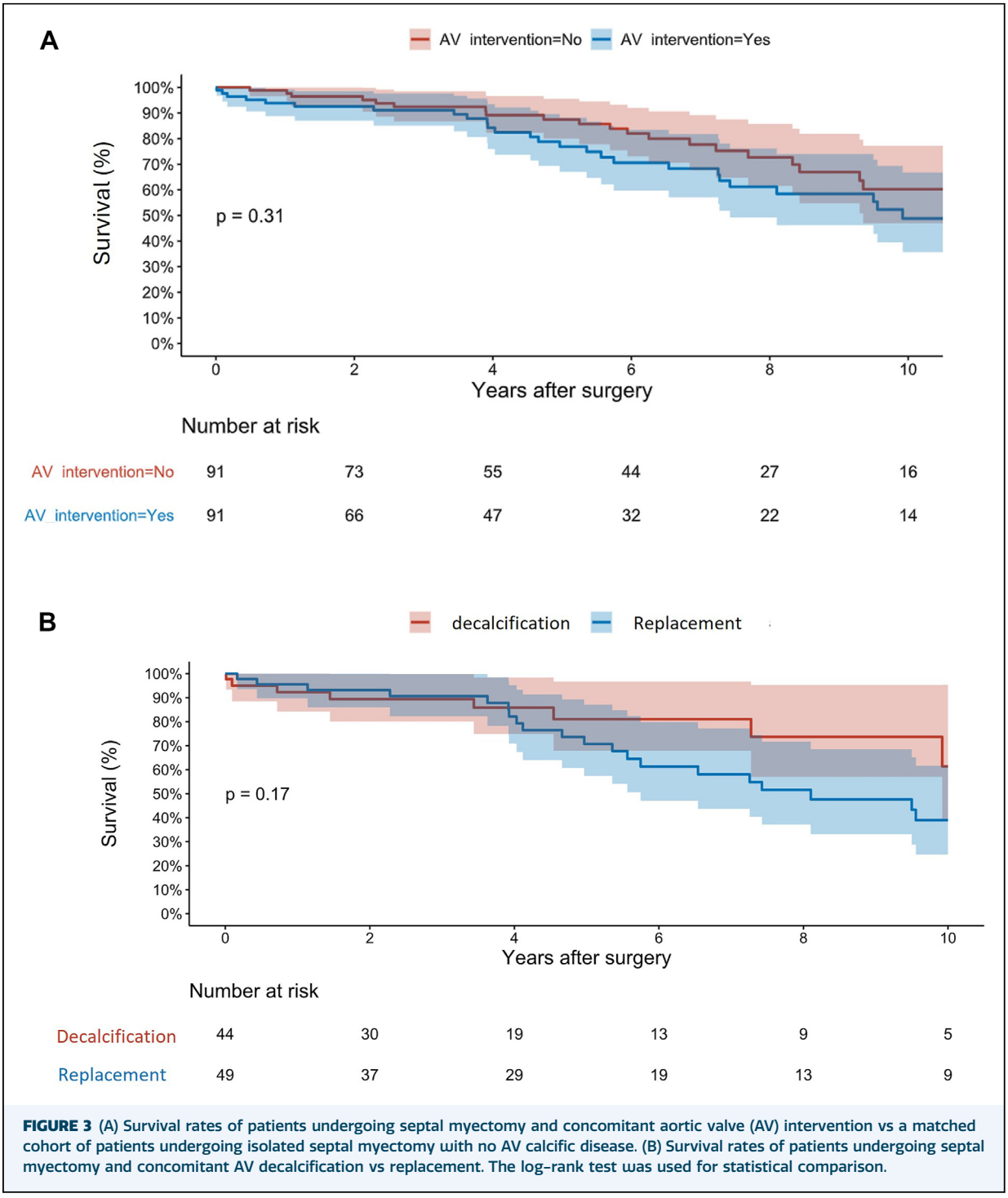

FIGURE 3 (A) Survival rates of patients undergoing septal myectomy and concomitant aortic valve (AV) intervention vs a matched cohort of patients undergoing isolated septal myectomy with no AV calcific disease. (B) Survival rates of patients undergoing septal myectomy and concomitant AV decalcification vs replacement. The log-rank test was used for statistical comparison.

studies reported an increased incidence of postoperative midterm valve insufficiency resulting from cusp scarring and retraction.[16-18] Freeman and colleagues[19] found that among 61 patients undergoing ultrasonic AV decalcification, hemodynamically severe AV regurgitation developed in 26% within the first postoperative year. Importantly, most of these studies assessed AV decalcification only in patients with severe AS with extensive calcification involving all cusps, and the technique was performed using ultrasonic devices rather than being done manually. In a study by Touati and colleagues,[20] assessing AV decalcification in patients with asymptomatic moderate AS who were undergoing CABG, manual debridement of the AV was

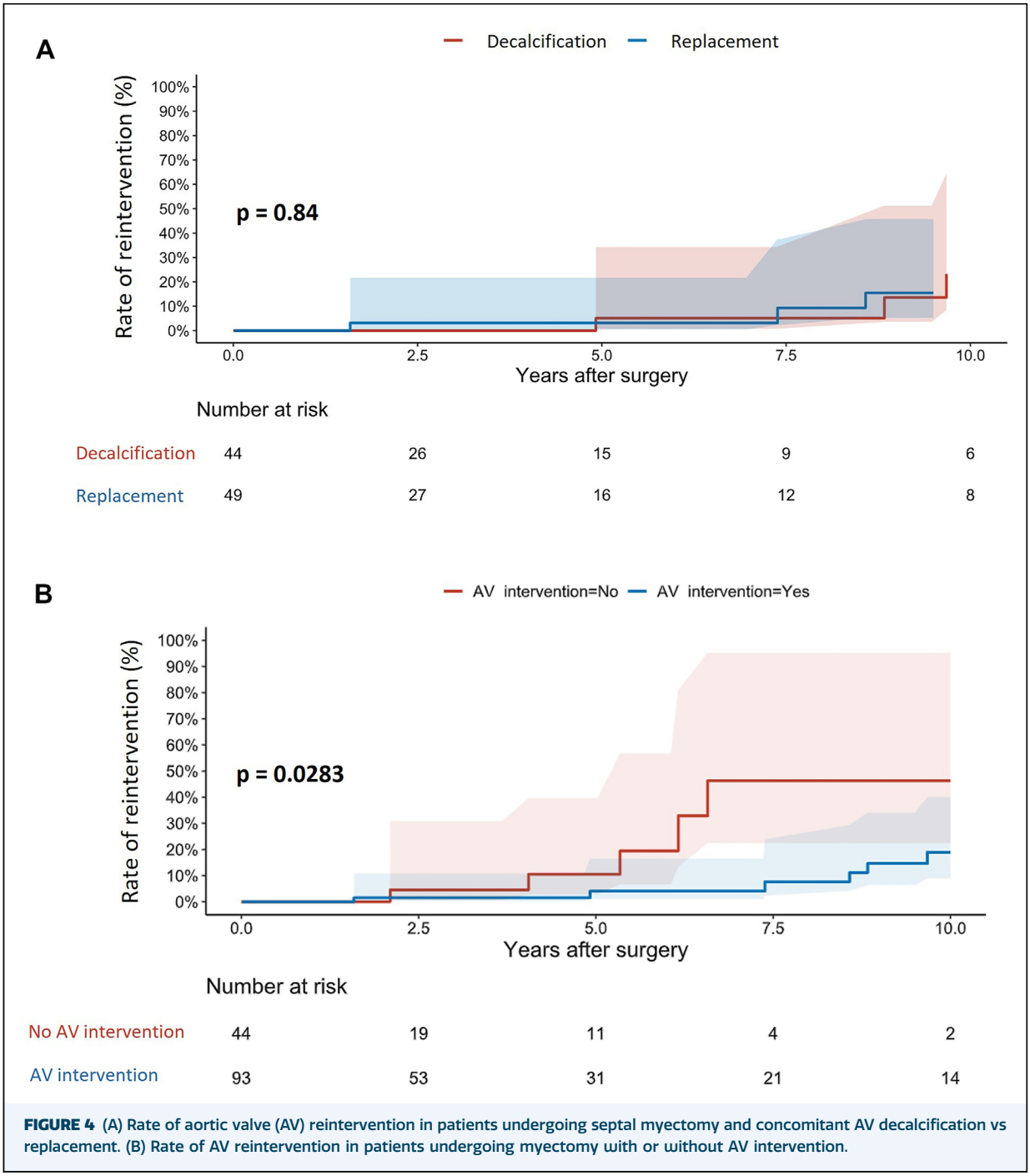

FIGURE 4 (A) Rate of aortic valve (AV) reintervention in patients undergoing septal myectomy and concomitant AV decalcification vs replacement. (B) Rate of AV reintervention in patients undergoing myectomy with or without AV intervention.

associated with acceptable medium- and long-term hemodynamic outcomes (AV restenosis or insufficiency), especially in cases of senile calcification with no or only moderate degrees of regurgitation at baseline. Another study, by Baeza and colleagues,[21] demonstrated that combining manual debridement with limited ultrasonic aspiration as an adjunct significantly reduced the aortic gradient, with no incidence of aortic insufficiency observed 3 years postoperatively. In the present study, manual AV decalcification was performed when calcification affected only 1 or 2 cusps and none of the patients experienced severe AV regurgitation during follow-up.

Our results suggest that when the decision to perform AV decalcification or replacement is based on intraoperative inspection of the degree of AV cusp calcification, both interventions can effectively normalize AV hemodynamics. In the short term, both the extent of AV gradient relief and the

AV gradients before hospital discharge were similar in the 2 surgical groups, and this finding is consistent with those of multiple previous studies.[16,19] In the long term, the rates of AV reintervention were not significantly different, as suggested by the cumulative incidence curves. Six patients in the decalcification group required future AV replacement because of progressive restenosis, with the first AV replacement performed 5 years after decalcification, and the median duration between decalcification and future AV reintervention was 10.2 years (IQR, 7.9-11.5 years). These findings underscore the durability of this procedure when performed in appropriate conditions.

Previous investigations of decalcification techniques have been constrained by short postoperative follow-up periods, thereby compromising the assessment of longer-term survival.[19] In the current study spanning a median follow-up duration of 6.2 years (95% CI, 4.0-8.3), patients undergoing AV decalcification had similar survival rates compared with patients with AV replacement. The overall survival of both surgical groups was comparable to that of a slightly younger cohort of patients with oHCM and concomitant moderate or severe AS who underwent surgical myectomy, as reported by Desai and colleagues.[8] In that study, 5-year survival was 83% compared with 74% (IQR, 64.5%-86%) in the present investigation.[8] However, patients in the current study appeared to be at higher risk of late mortality compared with the overall SM population at our clinic, where the 5-year survival rate is 95.7% (IQR, 94.75%-96.65%).[5] Patients in the present study were older as compared with the typical age of patients who undergo SM, and they had higher prevalences of diabetes and hypertension. Indeed, when we matched the patients in our cohort with those in the overall SM population on the basis of age, sex, body mass index, and the need for concomitant CABG (as a surrogate for ischemic disease), we found no significant difference in survival between the 2 groups. This finding suggests that the poorer midterm survival observed in this cohort is driven primarily by the higher prevalence of comorbidities rather than by the additional AV intervention.

STUDY LIMITATIONS. The study's findings are from a single tertiary center. Multivariable modeling was not possible because of the small sample size, but statistical and clinical assessments suggest that both surgical groups were comparable at baseline, thereby reducing the potential for confounding in outcome analysis. Additionally, no optimal control group consisting of patients with mild or moderate AS and significant calcification on intraoperative assessment who did not undergo AV interventions was available, thus limiting comparison. When encountering mild to moderate calcific AS, surgeons at our clinic systematically opted for valve decalcification or replacement after carefully considering the risk of stenosis progression against the potential drawbacks of unnecessary early surgery.

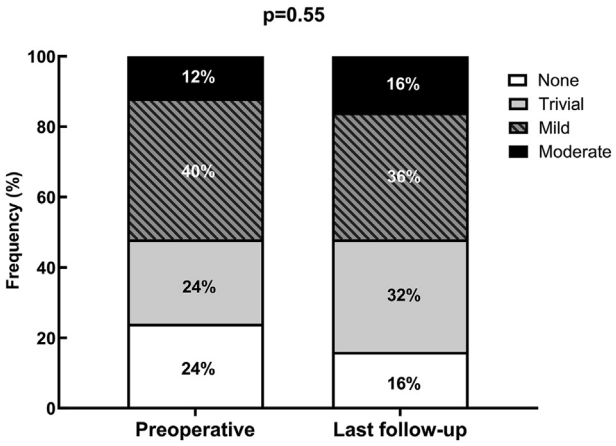

FIGURE 5 Paired proportions of degree of aortic valve (AV) regurgitation preoperatively and at the last echocardiographic follow-up for 25 patients undergoing septal myectomy and concomitant AV decalcification. McNemar test was used for statistical comparison.

CONCLUSION. Mild or moderate calcific AS is prevalent in older adult patients and may confound operation for subaortic LVOT obstruction in patients with oHCM or a subaortic membrane. Surgical management should balance the risk of progression of untreated AS (and the need for future intervention) with the unnecessary exposure to the potential hazards of a prematurely implanted prosthesis. Direct inspection of the AV as an added assessment of the severity of valve stenosis can direct surgical treatment with decalcification for more limited disease and AV replacement for patients with more extensive calcification, especially calcification involving all 3 cusps. Outcomes of this strategy have yielded satisfactory outcomes over a 23-year experience on the basis of on the results of the present study.

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The authors have no conflicts of interest to disclose.

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