Keynote Lecture Series

Tips and tricks in addressing mitral annular calcification in mitral valve surgery

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Mitral annular calcification (MAC) is a heterogeneous condition that can complicate mitral valve (MV) repair and replacement operations. Surgery in the context of MAC varies considerably. Relatively simple repairs or replacements can be performed in several patients without the need to remove the calcium bar. However, extensive annular debridement, reconstruction of the atrioventricular junction, reconstruction of the intervalvular fibrous body, and mitral and aortic valve replacement may be necessary in some instances. Outcomes of these patients are directly related to the avoidance of technical problems amid a variety of potential anatomic challenges and pitfalls. Careful preoperative assessment, patient selection, detailed preoperative planning, and intraoperative judgment are required to optimize the chance of a successful outcome. Here we describe our approach to the MV patients with MAC, including preoperative planning, intraoperative technical tips and tricks, as well as a discussion of outcomes and remaining questions in this challenging population. This review only includes patients with MAC and MV dysfunction. It excludes those with associated aortic valve disease. The article contains a lecture on MV surgery in patients with MAC. We also highlight emerging experimental approaches, including hybrid and percutaneous techniques.

Keywords: Mitral annular calcification (MAC); mitral annulus reconstruction; mitral valve replacement (MVR); mitral valve repair; commando operation

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Introduction

Mitral annular calcification (MAC) is a chronic, progressive, degenerative disorder involving the fibrous attachment of the mitral valve (MV) (1-4). While this process can be clinically silent, it may be associated with significant valvular dysfunction and presents an added technical challenge and operative risk to MV valve surgery. MAC is a heterogeneous disease concerning pathoanatomy, is caused by multiple pathophysiologic processes, and as a result, variations in surgical approaches are needed. Nevertheless, the incidence of some degree of MAC may be increasing, as it was present in 18% of MV replacement (MVR) operations reported in the Society of Thoracic Surgeons (STS) Database, and was associated with nearly 6% operative mortality [odds ratio (OR): 1.24 compared to the absence of MAC] (5). Furthermore, the presence of MAC is independently associated with adverse patient prognosis, particularly among patients with progressive MAC (1,2). Unfortunately, the medical management of MAC patients with valvular dysfunction is poor, with a 50% mortality rate within 3 years (6). Therefore, surgical intervention is required when technically feasible and patient factors allow. In our experience, the chance of a successful outcome in this challenging population depends on careful patient selection, a detailed examination of preoperative investigations, and achieving technical success despite several potential anatomic challenges and pitfalls.

In this keynote article, we describe our approach to MV surgery in the context of MAC, including management of the damaged mitral annulus, MV repair strategies for patients with adequate leaflets, and technical pearls for MVR. We also mention the management of MAC when the fibrous skeleton and aortic valve are calcified, as well

as in late redo MVR. We provide a detailed description and video of our technique for MV surgery and annular reconstruction in patients with severe MAC. Finally, we discuss operative outcomes of MAC operations, emerging experimental approaches, and areas of ongoing research.

Incidence and pathology of MAC

MAC is identified in approximately 3% of the general population, with the highest proportion found in individuals over 70 years of age, females, and patients with other cardiovascular risk factors (3,4). Among over 13,000 patients undergoing echocardiography at a single center, 14% were identified to have MAC, and of these, 2.2% had hemodynamically significant mitral stenosis (MS) while 11.9% had mitral regurgitation (MR) (6). In patients under the age of 50 with MAC, over 50% have chronic kidney disease, 25% have hemodynamically significant MR, and 5% MS (7).

The worst prognosis is among patients with progressive MAC, and the degree of progression is associated with the degree of MAC severity (2). Hemodynamic progression of the associated valve lesion is less common than structural progression of MAC (2). Therefore, MAC can progress quite extensively before a patient develops symptoms or presents to the surgical clinic for consideration of intervention. Similarly, the presence of MAC without valvular dysfunction or symptoms does not require intervention. Those with mixed valve disease tend to be most symptomatic, and those with MS have the most severe calcification (2,8). Nevertheless, even patients with mild MAC on echocardiography develop MV dysfunction 15% of the time at a median follow-up of 4.7 [interquartile range (IQR), 2.7–6.9] years (9).

The pathophysiology of MAC is multifactorial. Dystrophic calcification occurs when endothelial or endocardial damage creates an environment that promotes the deposition of calcium salts. This process can be triggered by age-related degeneration, rheumatic disease, previous MVR, or by radiotherapy, most commonly in late survivors of Hodgkin's lymphoma. Such triggers likely produce a positive feedback loop in which calcium increases mechanical stress on the endocardium and further potentiates calcium deposition. There is an established link with atherosclerosis and a growing understanding of the role of inflammation in the pathogenesis of MAC (10,11). Radiotherapy-induced MAC is an essential and growing subgroup of patients, mainly because they are often young. The phenotype of these patients can be highly challenging to manage—heavy calcification typically affects the entire base of the heart and frequently the aortic valve, aortic sinuses, which sometimes causes coronary artery ostial stenoses. The degree of calcification is dose-dependent, with a 10% risk of valve complications 30 years post-treatment among patients receiving >30 Gy (12). Moderate or severe valvular dysfunction has been reported in as high as 17% of patients with high-dose mediastinal radiation for Hodgkin's lymphoma at a median follow-up of 20 years (13). Another cause of MAC in young patients is the presence of rare genetic metabolic conditions such as Hurler syndrome (mucopolysaccharidosis) (14).

Most cases of MAC involve the posterior annulus, with the majority being confined to the medial commissure (11,15). However, in 15% of cases, the calcium extends towards the lateral trigone, 10.5% occurs from trigone to trigone, and 1.5% of patients have circumferential calcification. MAC is usually confined to the annulus and leaflets. Involvement of the ventricular muscle occurs in 12% of patients, and 4.5% of individuals have calcified papillary muscles (16). Such involvement of the subvalvular apparatus presents significant technical complexity and risk of complications. Therefore, this group should be identified and approached with caution.

In addition to valvular dysfunction, the presence of MAC may be associated with conduction abnormalities, atrial fibrillation, left ventricular hypertrophy, and left atrial dilation, which present additional challenges during the operative and perioperative management of these patients. The presence of MAC also increases the risk of infective endocarditis, and infected MAC represents a particularly challenging subgroup of patients. Ultimately, the indication to proceed with surgery is based on the presence of hemodynamically significant valvular dysfunction and the patient's symptoms (17). We show a case of infected MAC in our accompanying lecture.

Surgical indications for surgery in patients with MAC

The presence of MAC itself does not constitute an indication for surgery. Instead, patients with indications for MV surgery based on valvular dysfunction and symptoms should be discussed in a heart team for decision-making regarding surgery (Class I) (17). The heart team discussion aims to assess operative risks that are not captured by the STS risk classification, using multimodal imaging for

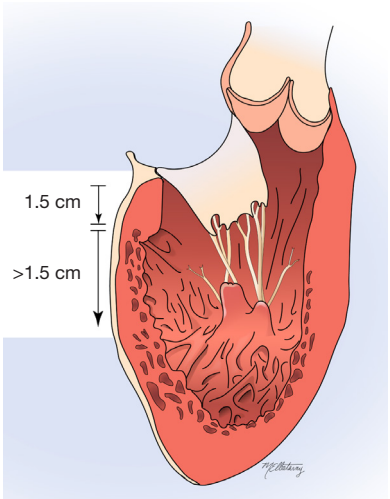

Figure 1 Sketch of the left ventricle showing the compact and trabeculated muscle at the base of the ventricle. Extension of calcium deep into the myocardium beyond the transition zone from compact to trabeculated muscle (typically 15 mm) is associated with an increased risk of patch dehiscence, false aneurysm, rupture of the posterior wall, and/or rupture of the atrioventricular junction.

guidance. Those with symptomatic moderate or severe MR and symptomatic moderate or severe MS should be considered for intervention when clinical factors are not prohibitive for surgery (17). For asymptomatic patients or those with only mild symptoms and severe MAC, consensus recommendations are to carefully weigh the risks of intervention with watchful waiting and goal-directed medical therapy (Class IIb) (17).

Our approach is to wait for the presence of symptoms before offering surgery to patients with severe MAC and challenging anatomy. By severe MAC, we mean a calcium bar that extends into the ventricular muscle beyond 10 to 15 mm and involves most of the compact myocardium in the base of the heart (*Figure 1*). Calcium bars that affect only the endocardium, such as those extending into the papillary muscle through calcified chordae tendineae, can be safely resected but often require an endocardial patch and MVR.

We believe special consideration should be given to patients with MR due to leaflet prolapse and MAC. We offer surgery to asymptomatic patients if a comprehensive preoperative assessment indicates that MV repair is highly feasible. This is mainly dependent on the extent of MAC and it should not involve the ventricular muscle. If we believe that MVR may be needed, we delay surgery until symptoms appear or the ventricular function begins to deteriorate.

When MAC and MV dysfunction occurs with concomitant moderate or worse aortic stenosis (AS), the intervalvular fibrous body is almost inevitably involved, and the patient will require a more aggressive commando operation (18). It is essential to note that in these cases, posterior annular patching is often required, not only complicating the procedure but also increasing the operative risk (18). Thus, this subset of patients constitutes the highest-risk group among patients who need the commando procedure. Another difficult group of patients is those with symptomatic prosthetic valve dysfunction and MAC, often with multiple previous MVRs. In our experience, patients who require redo MVR after two or more decades from the index operation frequently have a calcified annulus and proximal ventricular wall, and redo MVR may be complex because of a lack of fibrous tissue to hold the new prosthetic valve.

MAC can occur in patients with AS in the absence of MV dysfunction. Among patients undergoing transcatheter aortic valve implantation (TAVI), 20% also have concomitant MAC. These should be managed with TAVI alone when the MV function is reasonable (19). Furthermore, in patients undergoing open surgery for other cardiac indications with incidental MAC, the MAC itself need not be addressed (19).

Finally, we agree with the consensus guidelines that patients with severe frailty or comorbidity should be managed without open surgery (17). Specifically, recommendations are to consider transcatheter edge-to-edge repair to palliate inoperable patients with severe MR or transcatheter MV replacement (TMVR) with severe MR and MS (17).

Preoperative assessment of patients with MAC

Surgical intervention for MAC can range from relatively simple conservative approaches to very extensive reconstructions, particularly when the intervalvular fibrous body and aortic valve are also calcified. Thus, careful assessment of multimodal preoperative imaging is crucial for characterizing this heterogeneous disease, predicting the feasibility of intervention, and determining the extent of the surgical procedure that may be required (18,19).

Patients with mitral annulus disjunction (MAD) and MAC may present with ventricular dysrhythmias, and a Holter monitor test should be performed to quantify the burden of premature ventricular contractions, particularly in

patients with a history of frequent palpitations. Conduction defects should also be identified preoperatively to evaluate the risk of a permanent pacemaker postoperatively when the medial fibrous trigone is calcified. Left atrial dilation is a key variable on echocardiographic assessment to predict adequacy of exposure for complex repairs and the need for concomitant atrial reduction. It has been our experience that severe atrial dilation with paper-thin walls, such as in those with longstanding calcific rheumatic MS, renders the concomitant Maze procedure much less effective in eliminating atrial fibrillation. In these patients, the atrial wall becomes significantly thinned and acts as a non-contractile passive conduit. Patients with rheumatic heart disease may have a calcified atrial wall, further complicating the operation.

A complete echocardiographic assessment should include evaluation of other valves. Patients with moderate or worse aortic valve dysfunction or the presence of severe calcification of the entire fibrous skeleton may require the much more extensive commando operation. Paradoxically, in our experience, resection of the calcium bar and reconstruction of the mitral annulus through the aortic root and dome of the left atrium is often simpler than through the left atrium, particularly in patients with a small left atrium. Finally, in patients requiring combined aortic and MV replacement with a narrow left ventricular outflow tract, the commando procedure facilitates the implantation of heart valves of adequate size (18).

Coronary angiography and computed tomography (CT) scan are part of the preoperative assessment of MAC patients. The spatial resolution of CT is more precise for quantifying the extent of annular calcification, the depth of extension into the left ventricle, and the mitral annular dimensions (17). These features are critical for predicting the extent of decalcification required, the feasibility of implanting an adequate-sized valve, and the risks of radical decalcification, particularly when calcification extends deep into the ventricle and/or the papillary muscles. In our experience, the extension of calcium beyond 10 to 15 mm into the ventricle, where muscle typically becomes trabeculated, is associated with an increased risk of patch dehiscence, false aneurysm, rupture of the posterior wall, and/or rupture of the atrioventricular junction (*Figure 1*). These patients, frequently elderly females with MV dysfunction due to fibroelastic deficiency or multiple previous MVRs, should be approached with caution. Calcium scoring with Agatston units can provide a quantitative measure of the overall severity of MAC (17). Coronary angiography will not only demonstrate the presence of severe calcification, but it is also essential for determining the presence of coronary artery disease and coronary artery dominance when the calcium block is resected and the atrioventricular junction is reconstructed. Coronary disease may be present even in young patients who had radiotherapy (1,3,4). Depending on the extent of surgical intervention required, concomitant coronary artery bypass may increase operative risk excessively (18).

Thorough patient clinical assessment is required in addition to imaging investigations. MAC patients frequently have significant medical comorbidities, including advanced age, peripheral vascular disease, renal dysfunction, previous radiation, and frailty. While preoperative risk calculators can help predict the risk of surgical intervention, it is essential to note that much of the risk in these patients is derived from factors not captured by risk scores. Thus, the STS risk scoring and others will underestimate the operative risk incurred by the need for concomitant annular reconstruction, the presence of dense radiation-induced adhesions, and the anatomic risks of paravalvular leak, atrioventricular groove dissociation, left ventricular rupture, and severe bleeding from technical problems. Nevertheless, it is critical to identify patients with excessive frailty and comorbidity who are not operative candidates; these may be more optimally managed with transcatheter approaches or medical management. However, the risk of death with medical management alone is as high as 50% in 3 years (8). We have counselled some patients with pervasive disease and medical comorbidity that the odds would be more favorable than surgical intervention. Indeed, we recently turned down a patient with severe annular and ventricular calcification for conventional surgery who was subsequently treated with hybrid transcatheter intervention at another center and died soon after surgery (*Figure 2*).

Upon review of clinical and anatomic factors, we take the following approach to addressing MAC surgically in appropriately selected candidates. Those with adequate quality leaflets (typically myxomatous or fibroelastic deficiency) are planned for MV repair. When MAC is limited to the posterior mitral annulus, it is either left *in situ* or resected with leaflet reattachment to the posterior compact myocardium, or a patch is used to reconstruct the atrioventricular junction, and the posterior leaflet is sutured to the patch. Patients with fibrotic leaflets, rheumatic disease, or MS are managed with MVR. Assuming that the MAC does not preclude the implantation of an adequately sized valve for the patient's body surface area, it is left *in situ*,

Figure 2 Preoperative CT scan image of a patient with extensive MAC involving the intervalvular fibrous body and the ventricular muscle. The patient was declined for conventional surgery. A few months later, he had open surgery with transcatheter valve deployment in the mitral position. He died soon after surgery. CT, computed tomography; MAC, mitral annular calcification.

particularly in elderly or comorbid patients who require expeditious operations, as long as a bioprosthetic heart valve can be safely secured. MAC, which encroaches excessively on the MV orifice, is excised totally *en bloc* before MVR. Concomitant annular reconstruction with a pericardial patch to cover the atrioventricular junction is required. Use of leaflet tissue or the atrial wall to patch the defect can be performed, but it is not as secure as a pericardial patch; therefore, it is not recommended, particularly when the annular defect is significant.

MV repair in patients with MR due to leaflet prolapse with MAC

In our experience, most patients with MR due to leaflet prolapse and MAC limited to the posterior mitral annulus can have MV repair. Patients with MR due to advanced myxomatous degeneration and MAC frequently have associated MAD. In these patients, the calcium bar as well as the insertion of the enlarged posterior leaflet is atrialized, and the MAC does not invade the myocardium. We described our approach to MV repair in patients with MAD years ago (20). The entire posterior leaflet is detached from its atrial insertion, trimmed to a height of 15–18 mm, the false commissures are closed, all secondary chordae tendineae are resected, and the posterior leaflet is sutured directly to compact myocardium with a running 4-0 polypropylene suture. The annuloplasty sutures are placed through the compact myocardium before reattaching the posterior leaflet to the endocardium of the ventricle. The leaflet prolapse is corrected using Carpentier's classic techniques or with multiple interdependent loops of 5-0 expanded polytetrafluoroethylene sutures. Over the past three decades, we have exclusively used a posterior flexible band for annuloplasty (21). A video illustrating such a case is included in the lecture.

The MAC often invades the myocardium in patients with MR due to leaflet prolapse due to fibroelastic deficiency. The posterior leaflet is usually small, and one has to be careful when detaching it from the calcium bar. We prefer to resect the calcium bar by sharp dissection of the ventricular muscle from the block of calcium. We have tried ultrasonic debridement of the calcium bar but prefer sharp dissection. These patients often require reconstruction of the atrioventricular junction with a strip of fresh autologous pericardium or commercially available glutaraldehyde-fixed bovine pericardium. The patch should cover the entire area of resection of the MAC from the myocardium. It is sutured with a continuous 3-0 polypropylene suture from normal annulus to normal annulus, covering the defect left by the resection of the calcium bar. The suture bites should be approximately 5 mm apart and 7 mm from the free margin of the patch, and 8 mm deep in the myocardium. The circumflex artery is not damaged as long as sutures do not reach the epicardium. We prefer to pass all sutures through both the patch and the myocardium before pulling on each loop to ensure the suture line is hemostatic, and then tie the knots on each end of the patch and normal annulus. The patch is also sutured to the endocardium of the left atrium, 1 cm away from its junction to the atrioventricular junction to avoid injury to the circumflex artery. Next, multiple 2-0 polyester sutures are passed through the patch at the level of the proximal part of the ventricle. The posterior leaflet is sutured to the patch with a continuous 4-0 polypropylene suture. The prolapse is corrected by creating neochords with expanded polytetrafluoroethylene sutures. We avoid resection of any leaflet tissue in patients with fibroelastic deficiency because the leaflets are usually small. A flexible posterior band is used for annuloplasty. A video illustrating such a case is included in the lecture. Occasionally, we extend the height of the posterior leaflet with a separate patch of pericardium. It is essential to have the patch slightly larger than the defect in the atrioventricular junction. In a report describing our experience with MV repair for MR due to leaflet prolapse, only 2% of patients

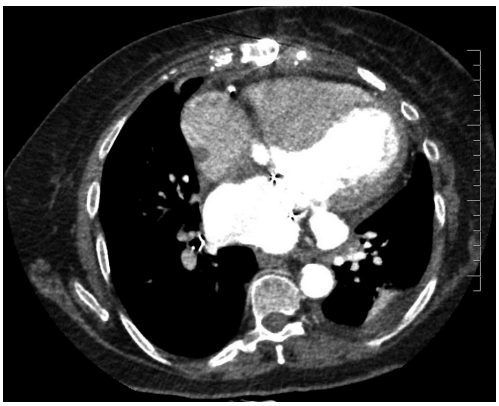

Figure 3 A CT scan image of a ruptured ventricle after patch reconstruction. A false aneurysm of the left ventricle after reconstruction of the mitral annulus and mitral valve re-replacement for MAC. CT, computed tomography; MAC, mitral annular calcification.

undergoing repair had extensive MAC that required annular reconstruction with a patch, but twice as many had resection of the MAC without annular patching (21).

MVR in patients with MAC

MVR is necessary in patients with MS as well as in those with MR and calcified leaflets or calcium that extends into the intervalvular fibrous body, or cases of infected MAC. Whenever possible, the prosthetic MV should be secured in the posterior leaflet and intervalvular fibrous body after excising the anterior leaflet. If the block of calcium is large and partially occludes the MV orifice, the calcium bar is resected *en bloc*, and the annulus reconstructed with glutaraldehyde bovine pericardium, as described above for MV repair. In cases of infected MAC, we use fresh autologous pericardium to repair the atrioventricular junction. A video illustrating such a case is included in the lecture. We usually resuspended both papillary muscles into the sewing ring of the prosthetic with a 4-0 expanded polytetrafluoroethylene suture in each papillary muscle. We believe this reduces the mechanical stress on the suture line between the patch and the posterior ventricular wall and the prosthetic valve in the patch.

Redo MVR with MAC

Reoperations on the MV two or more decades after the index MVR or MV repair with a rigid mitral annuloplasty ring may be difficult because the annulus may become calcified and involve the ventricular wall, and after explanation of the valve, there is no fibrous tissue to secure a new prosthetic MV. Securing a prosthetic MV in the ventricular muscle is associated with a risk of atrioventricular rupture and prosthetic valve dehiscence. Thus, we believe that these operations are safer when the mitral annulus is reconstructed with a patch before implanting a prosthetic MV (22). When the calcium extends into the trabeculated muscle, patching is not without complications. We recently operated on an 83-year-old woman with intractable heart failure due to severe MR caused by dehiscence of a mechanical valve 26 years after the initial MVR, and the calcium extended into the trabeculated muscle. Despite all care and multiple interrupted sutures, she developed a false aneurysm in the atrioventricular junction as illustrated in *Figure 3*. We are monitoring her with periodical CT scans. She may need a high-risk reintervention, either transcatheter or surgical, in the future.

Another scenario is that after multiple MV replacements, there may not be adequate distance between the insertion of the aortic cusps in the intervalvular fibrous body and enough tissue to place the sutures to secure the MV prosthesis superiorly. A dental mirror is helpful in these patients to assess whether there is sufficient intervalvular fibrous tissue to secure the MV prosthesis. If there is not, a patch should be sutured to the intervalvular fibrous body to extend its height, and the prosthetic valve is sutured to this patch. The patch does not need to be more than 10 to 12 mm in height and extend from the lateral to the medial fibrous trigones. Occasionally, we open the ascending aorta to make sure the sutures securing the patch do not damage or distort the aortic cusps.

Experimental surgical, hybrid, and non-surgical approaches

In patients with extreme cases of calcification and/or significant clinical comorbidities, various emerging strategies have been proposed. Qamar *et al.* describe a small case series of patients in whom extra-anatomic bypass of the MV was performed using a valved conduit secured from the left atrium to left ventricle (23). These patients presented with hypertrophic cardiomyopathy requiring myectomy, severe MS, and severe MAC extending deep into the ventricle. There were no in-hospital deaths, and a predischarge CT scan confirmed the patency of the conduit in all patients; however, long-term follow-up is not

available.

Another experimental technique involves a hybrid approach of open surgical implantation of a Sapien valve (Edwards Lifesciences, Irvine, CA, USA) in the mitral position (24,25). Techniques to overcome the rates of paravalvular leak include valve oversizing, aggressive balloon post-dilation, and securing a felt skirt to the prosthesis before implantation. Nevertheless, paravalvular leakage occurred in 22% of patients in this multicentre series, and 6/22 (27%) died in the first year of follow-up.

For selected patients who are not candidates for conventional surgery, percutaneous approaches are available as an alternative. A multicenter registry reports a series of 64 patients undergoing TMVR primarily for MS and advanced functional classes. Technical success was achieved in 72% of patients; however, 13% had procedural complications, including left ventricular outflow tract obstruction (LVOTO), valve embolization, valve perforation, and 30% died in the hospital (26). Overall, percutaneous interventions therefore remain a high-risk endeavor with LVOTO being a significant challenge. To mitigate the anterior mitral leaflet from causing LVOTO, there have been experimental attempts to perform laceration of the anterior mitral leaflet with or without implantation of the prosthesis within the lacerated anterior mitral leaflet (27-29). Early survival in the LAMPOON trial investigating TMVR with anterior mitral leaflet laceration was 87% therefore, these approaches remain associated with high procedural risk (27). Guerrero and colleagues published a position statement with guidance for risk stratification and suitability considerations for various transcatheter approaches in MAC patients (30). Components of risk assessment include anatomic, medical, and cardiac factors, and validation remains in progress (30).

Discussion

MAC is a highly heterogeneous condition that adds considerable technical complexity and operative risk to MV surgery, which traditional risk calculators often fail to account for. There is no consensus regarding the optimal approach for these complex patients, and as a result, several technical strategies have been described in various centers. Here, we describe our approach to MV surgery in MAC patients, considering patient factors and providing technical pearls for repair, replacement, and annulus management.

In general, the optimal management of MAC remains an area in need of further study, and the literature remains sparse regarding long-term outcomes. A meta-analysis comparing any MV intervention among patients with MAC *vs.* those without demonstrated increased risk of bleeding, paravalvular leak, need for permanent pacemaker, and reduced midterm survival [incident rate ratio (IRR): 1.32; 95% CI: 1.05–1.67; P=0.02] (31). The most extensive surgical series is available from the STS database with nearly 10,000 patients undergoing MVR with any degree of MAC. The operative mortality was higher in the MAC *vs.* no MAC groups (5.8% *vs.* 4.4%; P<0.001); however, long-term data are not available. Granular detail regarding the extent of MAC and operative strategy are also lacking (5). A single-center study from a high-volume institution has reported 1% mortality in MVR for MAC with a primarily conservative approach, which was comparable to replacement in non-MAC indications (22). Still, the presence of MAC was associated with increased risk of death over the long term compared to non-MAC counterparts, and fewer than 50% of MAC patients were alive 10 years after MVR.

MV repair for MAC is associated with low mortality in experienced centers (21,32-34). Early valve failures are more common in patients with MAC, and operative mortalities are often driven by technical factors associated with annular decalcification and reconstruction (32). In a small series where a more aggressive approach to annular debridement was taken with either repair or replacement, operative mortality was slightly high (5%). However, right ventricular and hepatic failure were causative factors (35). A conservative calcium-respecting approach may be reasonable, if feasible, to limit technical complexity and operative risk and is supported by the recent American Association for Thoracic Surgery (AATS) expert guidance document (17).

In both myxomatous and fibroelastic deficiency MVs, the calcium bar most commonly affects P2, followed by P1, then P3 (15). One distinction is that Barlow's valves the calcium bar infrequently invades the myocardium, and it can be easily removed without damaging the muscle. Thus, using the technique we described in 2008 for patients with MAD, one can detach most of the posterior leaflet from its atrialized position, remove the calcium bar, and suture the posterior leaflet in compact myocardium without the need for annular reconstruction (20). By comparison, the leaflets are small in patients with fibroelastic deficiency, making repair more difficult because the annulus often has to be reconstructed with pericardium before the leaflet can be sutured back in its place. Some surgeons have described partial annuloplasty in small valves with MAC to facilitate

improved leaflet coaptation without producing stenosis, thereby avoiding resection of the calcium (36). We have employed this strategy in several patients with a calcified lateral commissure and chords that extend into the anterior papillary muscle.

While conservative approaches may limit technical complexity and operative risk, reoperations appear to be primarily performed on patients in whom decalcification was not performed during the index operation (32). Intuitively, decalcification could be correlated with durability of repair as progression of calcium is likely a driver of disease progression and need for intervention, although this remains unproven. Nevertheless, optimization of long-term durability must be counterbalanced with immediate operative risk. In pervasive MAC, particularly that extending into the medial trigone, the risk of postoperative heart block is significant. Fortunately, a permanent pacemaker does not appear to impair long-term survival after MVR (37).

When complete reconstruction of the fibrous skeleton of the heart is necessary, the operation becomes a more serious undertaking with greater operative risks, particularly if the calcium bar has to be removed from the posterior mitral annulus. It should be performed only in high-volume centers (38,39). We recently described our operative technique for reconstruction of the fibrous skeleton of the heart (18). The indication for this operation was dystrophic calcification of the base of the heart in 34% of our patients (62 patients) (38).

It is essential to emphasize that, regardless of the need for annular decalcification, MV repair in MAC patients is often a complex and lengthy operation, as multiple lesions must be addressed in the leaflets, annulus, chords, and papillary muscles. For this reason, we continue to prefer a sternotomy approach, which provides excellent exposure and limits cross-clamp time in these often highly comorbid patients. At our center, this approach has facilitated MV repair with very low operative mortality despite high case complexity (21). However, in high-volume minimally invasive centers, complex repairs in the context of MAC have also been described using a robotic approach (33,40). Others still consider MAC to be a relative contraindication to robotic access and advocate for a more selective approach only in simpler cases (34). Saran and colleagues report a primarily calcium-respecting approach in nearly 2,000 open MVR patients, even in cases of circumferential calcification (22). They implement adjunctive strategies such as placing annular stitches from the right atrial side of the interatrial septum to minimize the risk of heart block and from the aortic side to mitigate paravalvular leak (22).

It is important to recall that the presence of MAC is independently associated with worse prognosis; therefore, careful patient selection for more aggressive surgical techniques is required, as non-surgical factors in these individuals may limit long-term survival. We continue to believe that frail, elderly, highly comorbid patients with modest life expectancy are best managed with an expeditious operation and short cross-clamp time. Studies comparing repair and replacement strategies as well as calcium-respecting vs resecting strategies, particularly over the long term, remain limited and an area of ongoing study. Ongoing research in optimal management of MAC should focus on examining intervention strategies tailored to patient anatomic and clinical factors with a high degree of granularity and long-term follow-up.

In summary, MAC is a heterogeneous condition complicating MV surgery. Tailoring the surgical approach to patient anatomic and clinical factors is required. A detailed preoperative assessment is paramount to gain an understanding of the risk of intervention, optimal timing, and to ensure that no surprises are encountered in the operating room. In some patients with limited calcification, repair or replacement can be achieved with a relatively simple operation; however, extreme calcification requiring radical debridement and patching is a major enterprise. A substantial proportion of early excessive mortality or failure of reconstructive strategies in MAC occurs as a direct result of technical problems or inappropriate patient selection. Hybrid and percutaneous techniques are emerging as alternative approaches for patients with prohibitive anatomic or clinical factors; however, the risks of these interventions are also high. A heart team discussion of complex cases and detailed planning can maximize the chances of technical and clinical success, as well as a reasonable outcome, in these patients.

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