

MANAGING THE MITRAL VALVE IN HCM SURGERY: TIPS AND TRICKS

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Abstract

Mitral regurgitation (MR) in hypertrophic cardiomyopathy (HCM) patients is mainly due to systolic anterior motion (SAM) of the mitral valve (MV). However, other mechanisms contributing to mitral regurgitation may coexist as a result of further structural abnormalities.

SAM might occur because of the increased septal thickness alone or due to simultaneous MV or subvalvular apparatus anomalies, such as mitral leaflet elongation, papillary muscle body anomalies, accessory papillary muscles or additional papillary muscle heads. Additionally, anomalous mitral chordae or the recently described mitral-aortic discontinuity (leading to a longer anterior mitral leaflet (AML)) can contribute to abnormal physiology. A closed aortomitral angle may also contribute.

During intraoperative echocardiographic assessment, it is important to thoroughly evaluate the MV and the regurgitant jet to understand the mechanism(s) that cause MR in HCM patients. Although myectomy alone is frequently enough to correct SAM, concomitant MV procedures may be needed, especially when the septum is thin (<16-18 mm) and/or there is intrinsic MV disease. Detection of concomitant regurgitation mechanisms beyond SAM can eventually be identified preoperatively, either by direct structural detection (valve prolapse), by pharmacological palliation of SAM with vasopressors and negative inotropic agents or suspected by identification of anteriorly and centrally directed regurgitant mitral jets.

Surgical techniques that can be employed to contribute to SAM elimination include plication/extension/retention plasty of the AML, resection/release/reorientation of papillary muscles, division of anomalous chordae, edge-to-edge repair, or, at times, prosthetic MV replacement. If there is structural MV disease concomitant to HCM, appropriately tailored techniques to address the MV may be used.

Transoesophageal echocardiography at the end of the procedure should demonstrate elimination of SAM, resolution of LVOT obstruction, and appropriate coaptation of the MV leaflets and nearly resolution of MR. Provocation with inotropes can be used to ensure no latent obstruction persists.

Keywords: Hypertrophic Cardiomyopathy, systolic anterior motion, mitral regurgitation, septal myectomy

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is characterized by increased left ventricular wall thickness (hypertrophy), which can lead to dynamic left ventricular outflow tract (LVOT) obstruction, diastolic dysfunction, myocardial ischemia, arrhythmias, restriction and mitral regurgitation. Abnormalities of the mitral valve and papillary muscles are common in HCM patients and often contribute to systolic anterior motion (SAM) and LVOT obstruction. While myectomy typically corrects outflow tract obstruction and SAM, thereby resolving mitral regurgitation, additional mitral valve intervention may be necessary when significant anomalies in the mitral valve or subvalvular apparatus are present, particularly in patients with a thinner septum.

MITRAL REGURGITATION IN HCM

Mitral regurgitation (MR) in HCM can occur due to multiple mechanisms. Systolic anterior motion (SAM) is the main mechanism for MR in HCM (Figure 2) due to three primary reasons: the redirection of the left ventricle outflow stream by a mid-septal bulge, the high drag coefficient of the mitral valve, and specific anatomical features in HCM patients. These features include anterior displacement of the mitral valve coaptation point due to papillary muscle abnormalities, elongated chordae tendineae, elongated anterior mitral leaflets (AML), and abnormal connections between the papillary muscles and the anterior left ventricular wall. These factors lead to poor leaflet coaptation, causing an eccentric MR jet directed posteriorly. SAM and

obstruction can worsen due to a reduced aorto-septal angle, decreased intravascular volume, hypercontractility, or low afterload.¹⁻⁴

Among dedicated hypertrophic cardiomyopathy centres, mitral valve intervention alongside extended myectomy varies.³ A retrospective review of 1559 consecutive operations for left ventricular outflow tract obstruction in the Cleveland Clinic included 33% of myectomies with mitral valve or subvalvular apparatus intervention and 2% isolated mitral valve interventions without myectomy. The mitral valve was replaced due to left ventricular outflow tract (LVOT) obstruction in around 8% of cases undergoing mitral valve interventions. Patients with mitral interventions had a thinner septum and less myocardium removed compared to those without.⁵ The Mayo Clinic reported only 9% of concomitant mitral valve procedures in a total of 2004 cases of myectomy due to HCM. In more than 75% of cases, the valve was repaired.⁶ In a large study of STS database (n=2382), mitral valve surgery was needed in 34% of cases and repair was possible in only 62% of patients.⁷

MITRAL VALVE/SUBVALVULAR APPARATUS ANOMALIES IN HCM

Mitral valve and papillary muscle abnormalities occur in about 10-20% of HCM patients. These anomalies alongside the basal interventricular septum hypertrophy contribute to SAM, result in LVOT obstruction, loss of mitral leaflet coaptation, and MR. In some patients, dynamic LVOT obstruction occurs despite minimal left ventricular hypertrophy, with MV apparatus abnormalities playing a major role.^{8,9}

Common features include abnormally large or elongated mitral leaflets (particularly the anterior leaflet), anterior displacement of the anterolateral papillary muscle and its abnormal muscular connections with the anterolateral wall of the left ventricle. Other findings include shortened, elongated or thickened papillary muscles, anomalous attachments of the anterolateral papillary muscle to the middle portion of the anterior mitral leaflet or ventricular walls, and fibrotic, retracted secondary chordae with abnormal attachments to the anterior leaflet. These abnormalities lift and tent the MV anteriorly, leading to SAM and MR.^{3,10}

Mitral leaflet elongation

Mitral leaflet elongation is one of the most commonly found anomalies, specially elongation of the AML.¹¹ Mitral leaflets are longer in patients with obstructive HCM than with nonobstructive HCM.¹² An elongated AML (> 16 mm/m² or >30 mm) may contribute to SAM and obstruction of the LVOT, and thus surgical correction may be needed.²

Papillary muscles anomalies

Papillary muscles (PM) anomalies are multiple and might coexist. The most common abnormalities of the papillary muscles in HCM include anterior displacement of

the anterolateral papillary muscle (ALPM) with respect to the interventricular septum (IVS), abnormal attachments of the ALPM to the septum or anterolateral wall, direct insertion of the ALPM into the anterior leaflet without chordae (Figure 3), PM thickening (>1.1 cm in short axis view), and multi-headed or accessory PM. These variations can cause midcavitary obstruction and predispose to SAM by resulting in an anterior position of the mitral valve coaptation plane, thereby increasing the overlap between the left ventricular outflow stream and the mitral valve. An anteriorly positioned head of the ALPM or its chordae can also tent the mitral leaflets anteriorly, reducing posterior traction and restraint on the mitral leaflets, thereby contributing to SAM.^{1, 8, 12-14}

Anomalous chordae

Anomalous secondary chordae may attach to the middle of the anterior leaflet, lifting and tenting it anteriorly, predisposing to SAM and mitral regurgitation (Figures 4 and 5).¹⁵

These chordae can also prevent proper coaptation of the mitral valve leaflets, leading to MR. Three-dimensional echocardiography is crucial for identifying the exact location of these chords. Visualizing the mitral valve from the left ventricle (LV) side helps differentiate abnormal connections. Surgical release of the mitral leaflet from these secondary chords can decrease SAM, improve coaptation, and reduce MR.¹²

A study by Ferrazzi et al. involved 39 symptomatic patients with severe LVOT obstruction but moderate septal hypertrophy (≤19 mm). They underwent chordal cutting with a shallow septal myectomy. Compared to a control group that only had a shallow myectomy, the chordal cutting group showed a significant post-operative increase in the AML-annulus ratio and a decrease in the mitral valve tenting area, indicating better leaflet coaptation and reduced displacement. All patients in the chordal cutting group showed clinical and echocardiographic improvement.¹⁶

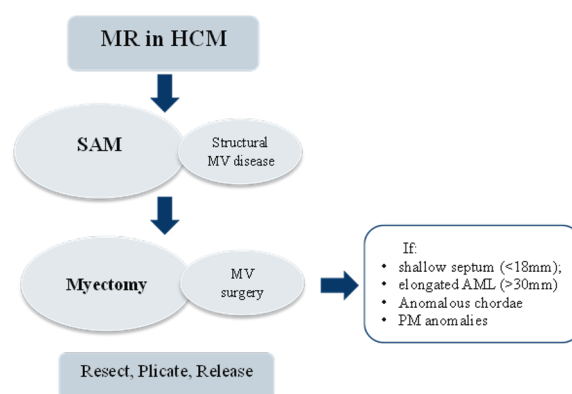
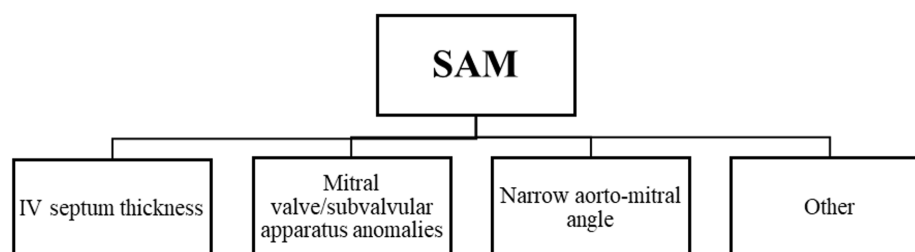


Figure 1

Managing the mitral valve in HCM surgery – a summary of main mechanisms and management. AML: anterior mitral leaflet; HCM: hypertrophic cardiomyopathy; MR: mitral regurgitation; MV: mitral valve; PM: papillary muscles; SAM: systolic anterior motion.


Figure 2

Mechanisms for SAM (systolic anterior motion) in HCM (hypertrophic obstructive cardiomyopathy).
IV: interventricular.

Other

In the recently described entity - mitral-aortic discontinuity - there is a muscle band interposed between the AML and aortic valve which works as a structural elongation of the leaflet. This was mainly found in young patients undergoing surgery for HCM.¹⁷

In some cases, calcification of the mitral leaflets or annulus can occur in patients with SAM and mitral-septal contact. A sign that calcification or fibrosis is causing MR is when the regurgitant jet is directed anteriorly or mid-direction, whereas MR caused solely by SAM usually results in a posteriorly and laterally directed jet. The goal of modern myectomy surgery should be to preserve the patient's native mitral valve to avoid the complications of mitral valve replacement. However, this may not be possible when calcification or fibrosis makes successful repair unlikely.¹²

TOE PRE-CPB

Intraoperative two- and three-dimensional transoesophageal echocardiography (TOE) is essential for patients with HCM. TOE is used to measure septal thickness, mitral leaflet length, and assess the subvalvular apparatus. Three-dimensional TOE from the LV side is particularly useful for visualizing the mitral valve and identifying segmental mitral valve disease. Interactive communication with the surgeon before and after bypass is crucial. TOE assessments help determine if MR is due to SAM or structural defects. In SAM-related MR there is systolic anterior displacement of AML into the LVOT and the jet is typically directed posteriorly and laterally, whereas an anteriorly directed regurgitant jet indicates structural defects such as calcification, posterior mitral leaflet flail, thickening, or a cleft.¹⁸

During the postinduction examination, patients often experience decreased preload and afterload, predisposing them to SAM.¹ To ensure MR is only due to SAM, loading conditions can be pharmacologically manipulated to try to abolish SAM. If MR still persists after SAM suppression, another mechanism beyond LVOT obstruction should be sought. The mitral valve should be carefully examined, particularly the length of the AML, the presence of

anomalous chordae and anomalies of the PM – namely their thickness, position and insertion. Intraoperative TOE also guides septal resection by measuring the maximal thickness at the point of SAM-septal contact.¹⁹

Preoperative planning for HCM should also routinely include cardiac magnetic resonance (CMR) imaging, which excels in assessing the subvalvular apparatus, including the morphology and location of papillary muscles. CMR provides detailed views of ventricular septal hypertrophy, mitral valve structure, and subvalvular morphology, including papillary muscles, accessory muscle bundles, and chordal connections.^{10, 19}

SURGERY: HOW TO MANAGE MITRAL VALVE ANOMALIES

Myectomy is the primary intervention for patients with HCM and is generally sufficient to improve MR in most cases. However, concomitant mitral valve procedures may be necessary, particularly when SAM occurs without significant septal hypertrophy (≤ 18 mm), as mitral valve abnormalities become the primary contributors to obstruction. In these cases, mitral repair is essential, given that the myectomy may be limited to a few millimetres. Historically, mitral valve replacement was advised for these patients, but current strategies emphasize on preserving and repairing the native valve.^{3,4}

Surgeons often address mitral leaflet and PM/chordal abnormalities concurrently with the myectomy when these issues are identified during preoperative assessment and intraoperative inspection. Addressing mitral valve pathology alongside the myectomy may be beneficial due to the inherent difficulty in precisely controlling the depth of myectomy. Correcting SAM contributors on both sides of the outflow tract helps ensure a successful surgical outcome. Although some abnormalities in the mitral valve leaflet may have minimal functional impact when an adequate myectomy is performed, it is still crucial to consider additional procedures for PM abnormalities that contribute to LV outflow tract obstruction.^{6,8}

Various surgical approaches have been developed

to correct mitral valve dysfunctions in the context of HCM. These techniques include vertical or horizontal plication of the anterior leaflet, extension of the anterior leaflet with a stiff pericardial patch (sometimes combined with posterior leaflet height reduction), the RPR repair (which involves Resection of the septum, Plication of the anterior leaflet, and Release of abnormal PM attachments), reorientation or realignment of the PMs, and transaortic secondary chordae cutting.³ The myectomy procedure primarily involves resecting an extended portion of the septum. Plication addresses the redundancy of the anterior leaflet,

especially when it is excessively long, while release targets the separation of any abnormal PM attachments to the lateral LV wall.²⁰

The choice of surgical technique is influenced by the thickness of the interventricular (IV) septum and the length of the AML. When the septum is thin and the AML is elongated, the surgical focus is primarily on plicating the leaflet and releasing any anomalous PM or chordal attachments, with only a shallow myectomy. In cases where the septum is thick and the AML is elongated, myectomy remains the central procedure, with plication and release performed only if obstruction or MR persist after the extended myectomy. When the AML is of normal length, only resection and release are usually sufficient.

Procedures such as anterior leaflet plication and resection of anomalous PMs or thickened secondary chordae are essential for repositioning the mitral leaflets away from the ejection flow stream, while preserving all attachments to the leading edge of the anterior leaflet to avoid iatrogenic mitral valve incompetence.^{8, 21} All such procedures have to be individualized on the basis of each patient overall cardiac architecture and severity of physiopathology.

Myectomy

Initially, the limited resection of the subaortic septal bulge was performed using Morrow's trough myectomy technique. However, due to its unpredictable outcomes and risks, such as inadequate relief of obstruction or ventricular septal defects, Messmer introduced the extended myectomy. In this technique, the septal bulge is resected to the base of the papillary muscles, ensuring a more even distribution of septal thickness while avoiding ventricular septal defects



Figure 3

Direct insertion of a papillary muscle into the anterior leaflet without chordae.

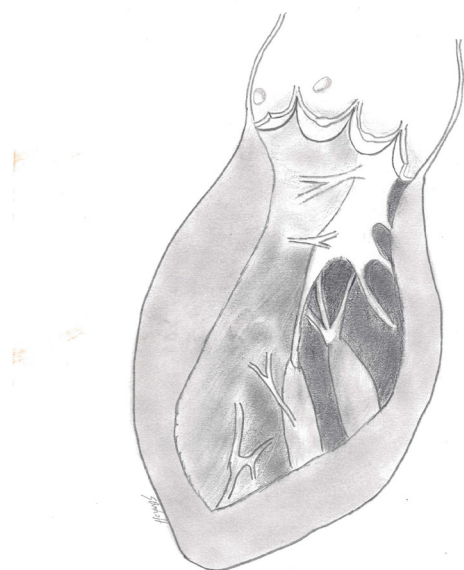


Figure 4

Anomalous secondary chordae attaching the anterior mitral leaflet to the septum.

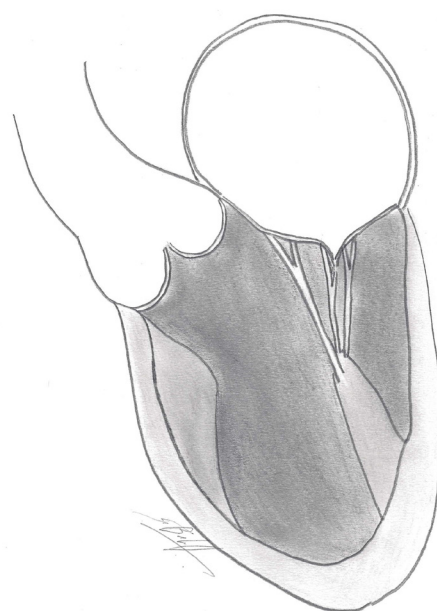
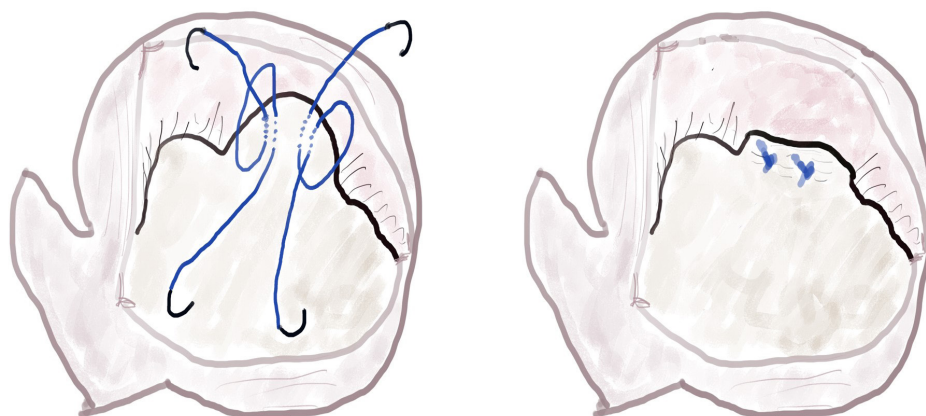


Figure 5

Anomalous secondary chordae attaching to the middle of the anterior mitral leaflet, causing tenting.


Figure 6

Free edge plication of the anterior mitral leaflet.

and aortic regurgitation by sparing 3-5 mm below the aortic valve. This approach minimizes drag on the mitral leaflets by directing flow away from the mitral valve.^{2,8}

Consequently, the extended myectomy, which separates the inflow and outflow portions of the left ventricle more effectively, is now the preferred operation in HCM surgical centres.

Plication/partial excision/extension of anterior mitral leaflet

Plication is recommended for patients at increased risk of suboptimal hemodynamic outcomes due to residual SAM linked to increased mobility, size, or length of the AML. The anterior leaflet's mobility is evaluated by retracting the chordae tendineae with a hook, often identifying a small fibrotic area that contacts the ventricular septum.²

The vertical plication technique, proposed by McIntosh et al., involves plication of the AML along its long axis to reduce billowing but risks altering the coaptation line, potentially resulting in central MR.

On the other hand, the mostly described horizontal plication technique, part of the resection–plication–release (RPR) procedure described by Swistel and coworkers, places 5-0 fine vertical mattress stitches along the short axis of the AML, below the mitro-aortic curtain. Sutures are typically placed horizontally across the leaflet, just superior to the chordal attachments, with the amount of plication (2 to 6 mm) determined by preoperative echocardiography. It is feasible through the aortotomy, is simple to do and easily reversible.^{11, 12}

Considering the anterior leaflet's behaviour, horizontal plication is often preferred over vertical plication, as it shortens the leaflet's excursion into the outflow tract and stiffens its midportion, reducing its tendency to billow out. This technique has the advantage

of leaving the coaptation zone untouched while changing the coaptation point, bringing it closer to the free edge of the leaflets. This minimizes the risk of reactive thickening and fibrosis that could cause central insufficiency later in life. Although vertical plication has shown success in some reports, it often negatively impacts the leaflet's coaptation zone, leading to central regurgitation.^{11, 12, 20}

Another option when excess of tissue, that does not participate into the coaptation surface, is present in the A2 mitral region is a free-edge plication (Figure 6), which involves placing a U-suture that plicates the excess tissue at the A2 free edge. This technique is reversible by removing the sutures if MR is detected post-plication.^{5, 13} Alternatively, most recently, Swistel²² has proposed excision of this free edge segment of A2 – residual leaflet – usually associated with slack chordae that do not support the mitral apparatus.

Another technique, the anterior mitral leaflet extension, involves a vertical incision of the AML and suturing an oval autologous pericardium to reduce the surface exposed to the LVOT. The reduction (2 to 5 mm) is evaluated preoperatively and confirmed intraoperatively. This stiffens the central portion of the AML and extends the width but not the length of the leaflet. This procedure is more time consuming, usually done through a left atriotomy and is technically more challenging, although it can be adjusted if MR is detected post-procedurally.¹¹⁻¹³

The choice of technique depends on the specific anatomical and functional characteristics of the AML, as well as the results from preoperative echocardiography and intraoperative assessments.

Resection/release/reorientation of papillary muscles

Anomalous PM can cause LVOT obstruction and SAM of the mitral valve. These muscles may obstruct the LVOT by impacting the septum during systole, leading to

midventricular obstruction, or by drawing the mitral leaflets into the ejection flow stream, thereby causing SAM. These abnormalities are often identifiable on TTE and even more precisely on three-dimensional TOE, particularly when viewing the mitral valve en face from the left ventricle.^{11, 18}

The management of anomalous PMs depends on their insertion point on the mitral valve leaflet. When these muscles are inserted near the free edge of the leaflet, resection is generally avoided to preserve mitral valve competence. Instead, the septal resection is extended, and the muscles are thinned along their longitudinal axis to reduce obstruction. However, if the anomalous PMs are inserted into the body of the anterior mitral leaflet, they can be safely excised without compromising valve function. When these muscles do not provide support to the leaflet, complete resection is also an option.

In cases where the anomalous PMs are large and directly obstruct flow but cannot be excised due to the risk of causing a flail leaflet, a longitudinal resection to thin the muscle, combined with extended myectomy, can be performed. If the muscle is particularly thick, it can be shaved down to its base to reduce its obstructive impact.^{11, 18, 23}

Extended myectomy, as described by Messmer and Schoendube, involves not only resecting the septum but also extending the resection laterally into the free wall above the base of the anterolateral papillary muscle (the shape of an inverted funnel), and thinning hypertrophied muscle heads. During this procedure, muscular connections between the papillary muscle head and the left ventricular free wall are divided and excised. This allows the anterior papillary muscle to move posteriorly, pulling the anterior mitral leaflet out of the ejection stream, which helps realign the mitral annulus and aortic valve into a more parallel orientation, relieving SAM. Severing the abnormal connections binding the papillary muscles to the ventricular wall also allows the mitral valve to assume a more posterior position away from the LVOT and its associated drag forces.^{12, 18}

Surgical reorientation techniques further help manage LVOT obstruction. One approach uses pledgeted mattress sutures, which are placed posterior to the most posterior papillary muscle head and passed through the anterior one. These sutures are then tied to fix the mobile papillary muscle towards the posterior left ventricle, reducing its mobility and preventing it from being drawn into the LVOT. Another technique involves identifying the abnormal insertion of the papillary muscle and mobilizing it posteriorly towards the posterior papillary muscle group, using sutures to realign the anterolateral papillary muscle group away from the LVOT. The sutures should be passed through the fibrous portion of the papillary muscle to avoid ischemia. The number of sutures (1–2) varies with the goal of realigning the anterolateral papillary muscle group towards the posterior medial group.¹³

Surgeons from the Mayo Clinic have shown that accessory papillary muscle heads and anomalous chordae that do not support the leading edge of the valve leaflet

can be removed. If resection risks causing a flail leaflet, only the anterior component is excised, leaving the posterior part attached. This approach allows the papillary muscles to move to a more posterior position in the left ventricle without compromising their function in mitral valve closure. When complete excision is not feasible, the muscle can be thinned with longitudinal resection to its base in combination with extended myectomy, ensuring that the mitral valve is adequately supported while reducing the risk of obstruction.¹²

Overall, the complete mobilization and strategic reorientation of papillary muscles are essential for the relief of SAM and LVOT obstruction. Careful surgical planning is required to avoid compromising the blood supply to these muscles while ensuring that the mitral valve assumes a more anatomically favourable position within the left ventricle.

Resection/release of anomalous chordae

Anomalous secondary chordae inserting into the aortic side of the AML, especially in patients with a thin septum, tether it and displace the MV apparatus toward the IVS. Cutting these moves the leaflet coaptation point more posteriorly, restores the mobility of the entire MV leaflet and decreases MV tenting. It also prevents the anterior displacement of the MV leaflets during systole, thereby reducing the risk of LVOT obstruction. However, it is essential to preserve all the attachments to the free edge of the AML to avoid iatrogenic mitral valve incompetence.¹³

Other (edge-to-edge, mitral valve replacement)

Edge-to-edge

In some situations, mitral valve abnormalities in the context of HCM are treated using the "edge-to-edge" (E2E) or Alfieri technique. Shah and coworkers reported on a small series of 24 patients with HCM associated with SAM due to an elongated AML and at least moderate mitral regurgitation. They observed positive clinical and echocardiographic outcomes with this approach. However, this technique can increase the risk of mitral stenosis, especially in patients with a small annulus. Therefore, the E2E technique should probably be reserved as a bailout option in selected cases to avoid mitral valve replacement.¹¹

The E2E technique involves stitching together the middle portion of the free edges of the mitral leaflets to create a double-orifice mitral valve. It can be performed through the aortotomy by placing two 4-0 polyester figure-of-eight sutures between A2 and P2, which are the junctions between the anteromedial and posterolateral papillary muscle chords. In the setting of HCM, this technique allows the surgeon to posteriorly fix the coaptation point of the mitral valve leaflets, preventing dragging by anchoring the posterior leaflet to hold the anterior leaflet away from the LVOT. This prevents SAM and LVOT obstruction.¹³

This technique also addresses organic mitral abnormalities such as leaflet prolapse or flail. The E2E approach

is advantageous because it is an easy, quick, and reproducible procedure that can be performed either trans-atrial or trans-aortic with shorter cardiopulmonary bypass (CPB) and aortic cross-clamp times compared to more complex techniques. It can be used electively or during a second pump run as a rescue procedure to eliminate residual gradients or persistent SAM-related mitral regurgitation after an isolated myectomy. Care must be taken to limit the width of the E2E suture to avoid stenosis, considering the relatively small area of the mitral valve in HCM.³

Mitral Valve Replacement

Mitral valve replacement should be a last resort when the MV cannot be repaired due to severe intrinsic valve abnormalities or calcification that do not allow a successful repair.^{2, 3, 11} It is still unclear whether mitral replacement is a better choice in patients without SAM after successful relief of obstruction when MR grade is only moderate.

TOE POST-CPB

TOE should show elimination of SAM, reduction/elimination of MR, resolution of LVOT obstruction, and appropriate coaptation of the mitral valve leaflets considering the baseline anatomy of each patient. The subvalvular apparatus is evaluated to confirm the successful release of the mitral valve leaflets. Visualization of resected papillary muscles, chordae tendineae, and papillary muscle heads, which previously retracted the anterior leaflet, should no longer be observed. The coaptation should be more posterior if papillary muscles were released. It is essential to exclude the removal of chordae integral to mitral valve coaptation or excessive papillary muscle release, as this could lead to new or worsened MR due to prolapse or flail leaflets during post-myectomy TOE.¹⁸

The peak gradient through the LVOT is reassessed and compared with preoperative values. If a significant gradient is present, further intervention may be warranted based on the location and aetiology. After the initial post-bypass assessment, in selected patients, dobutamine may be administered at 10 mg/kg/min to increase heart rate and contractility. When there is no chronotropic response, patients can be paced at 100 to 110 beats/min. Nitroglycerine may also be administered to reduce the systolic pressure below 100 mm Hg.⁵ A repeat assessment focusing on SAM, changes in MR, and any increase in peak gradient is performed. This ensures the patient can withstand exercise post-discharge and has no latent obstruction. If residual obstruction or moderate to severe MR are present at rest or after dobutamine, reinstituting cardiopulmonary bypass and additional surgical repair may be necessary.¹⁸ It is important to recognize that this inotropic challenge should be balanced against the risks of extending septectomy or further manipulating the mitral valve. Thus, this should be reserved for patients at evident risk of latent intraventricular obstruction. Induction of a premature ventricular contraction and post extra systolic beat gradient is a softer provocative manoeuvre that fits the majority of patients.²⁴

CONCLUSION

MR in HCM is mainly due to SAM, which occurs mostly because of the septal hypertrophy, but may also be associated with mitral valve anomalies. Mitral valve intervention may be needed in addition to myectomy in 10-30% of cases, especially when the septum is thin, the AML elongated or there are anomalous chordae or PM. Studies have shown that treating these patients in HCM dedicated centres leads to a higher surgical success without the need for MV replacement.⁶

Conflict of interest

The authors have no conflicts of interest to declare.

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