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Mitral Annular Disjunction: A Roadmap for the Surgeon

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Graphical abstract

Mitral Annular Disjunction – Surgical considerations

Summary

Pseudo-MAD is related to:

- mitral valve prolapse and severe MR
- myocardial fibrosis
- ventricular arrhythmias

Surgical treatment of MAD includes:

- Ring annuloplasty using deep stitches at the posterior annulus
- Leaflet resuspension to avoid SAM

Dedicated rhythm control protocols are indispensable



Legend: MAD: Mitral annular disjunction, MR: mitral regurgitation, SAM: systolic anterior motion

Abstract

Objectives: Mitral annular disjunction (MAD) is a structural abnormality of the mitral annulus fibrosus, associated with myxomatous leaflet degeneration, mitral valve prolapse (MVP), and ventricular arrhythmias. The combination of annular dilatation and abnormal annular motion increases mechanical stress on the mitral leaflets, triggering the degenerative process.

Methods: This review summarizes the major pathophysiologic, diagnostic and therapeutic measures for the treatment of patients with MAD and an indication for mitral surgery.

Results: The diagnosis is primarily based on non-invasive imaging techniques. Echocardiography is the first choice due to its ability to assess real-time mitral valve function. Cardiac computed tomography and magnetic resonance imaging provide more detailed information on the extent of MAD and the presence of calcifications. Indications for surgical mitral valve treatment are based on current recommendations. In cases with MAD and moderate mitral regurgitation, early intervention may be advocated in the presence of arrhythmogenic MVP. Long-term outcomes after treatment are assessed through multimodal imaging and electrocardiogram monitoring. A ring annuloplasty is an important cornerstone of treatment. Stabilization of the mitral annulus abolishes functional prolapse and increases the antiarrhythmic effect of mitral surgery. However, postoperative arrhythmic burden may persist in some cases, requiring continuous monitoring and sometimes an additional device therapy.

Conclusions: MAD represents a complex anatomical and functional entity associated with diagnostic challenges and rhythm abnormalities. Although the current indications for surgical treatment follow the recommendations for treatment of primary mitral regurgitation, early treatment may be important especially in patients with arrhythmias.

Keywords: mitral regurgitation; mitral repair; valve surgery; ventricular arrhythmias.

INTRODUCTION

The mitral annulus can be defined as the hinge line between the mitral leaflets and the atrioventricular junction. While the anterior annulus is anatomically defined by the fibrous tissue between the left and right fibrous trigones, the attachment of the posterior mitral leaflet to the hinge varies significantly.¹ The anterior annulus is commonly characterized by thick and well organized fibrous structures, producing a cord-like segment of ring. However, in the posterior part, the hinge of the leaflet maybe atrially displaced and is not always connected to the atrioventricular junction. The posterior annulus can develop as a cord-like fibrous tissue, as a thin curtain-like structure, or can be covered by adipose tissue (**Figure 1A and B**).² The laxity of the mitral annulus and the suspension of the leaflets to the atrioventricular areas are likely key factors for progressive annular dilatation and the characteristic pathological annular motion.

Mitral annulus disjunction (MAD) is a structural abnormality of the annulus fibrosus defined as a distinct separation between the insertion of the posterior mitral valve (MV) leaflet at the level of the left atrium and the ventricular myocardium.³ In addition, the annulus expands and flattens during systole and the left ventricular wall moves outwards in relation to the annulus. This motion increases the mechanical stress on the leaflets and the tendinous chords and is presumed to trigger the degenerative process of the MV. In some cases, an extensive MAD will result in generalized excess of MV tissue, mucoid degeneration and bileaflet prolapse, all of which are typical characteristics of Barlow's disease (**Figure 1C**). On the contrary a restricted MAD will be responsible for a focal posterior leaflet prolapse with limited area of mucoid degeneration. It is important to understand that some degree of MAD exists in normal MV.

True MAD refers to an anatomical disjunction between the insertion site of the mitral leaflet at the atrial endocardium and the ventricular myocardium, present in both diastole and systole, and not associated with severe MR. It may involve the anterior and the posterior annulus and has been identified in up to

96% of the normal population.⁴ Importantly, extension of MAD into the paracommissural areas is considered as a normal variant without clinical implications. Pseudo-MAD refers to an apparent separation of the atrioventricular junction that occurs only during systole, caused by a billowing leaflet and the systolic curling of the ventricular myocardium. The latter results in a relative prolapse of the posterior or both mitral leaflets and is associated with MVP.⁵

Over time, mechanical stress on the leaflet and the subvalvular apparatus triggers a proliferative cascade which is characterized by compensatory growth and thickening of MV leaflets and the chordae tendinae. In case of extensive MAD, when annular dilation progresses beyond the compensatory capacity of leaflet growth, coaptation is lost, leading to central MV regurgitation.⁶ At this stage, there is still no true anatomical prolapse but rather a "functional prolapse" which results from a combination of bileaflet curling and systolic annulus dilatation.⁷ With time, excess tension and mechanical stress on the chordae tendinae, especially of the posterior leaflet, may cause their elongation or rupture, producing a subsequent anatomical prolapse or flail.⁸ The mechanical stress imposed on the subvalvular apparatus and the mitral annulus may lead to focal fibrosis of the papillary muscles and/or the ventricular myocardium (**Figure 2**). This is considered a potential contributor to ventricular arrhythmias connected to the arrhythmogenic MV syndrome.⁹ In clinical practice, MAD is associated with myxomatous leaflet degeneration, MV prolapse (MVP), and ventricular arrhythmias.

DIAGNOSTIC CRITERIA OF MAD

The diagnosis of MAD is mainly based on non-invasive imaging (**Table 1**), which, unlike the mere surgical inspection, can provide valuable functional information. Echocardiography remains the first-line imaging modality due to its wide accessibility and ability to assess real-time MV function. Cardiac computed tomography (CT) and magnetic resonance imaging (CMR) play

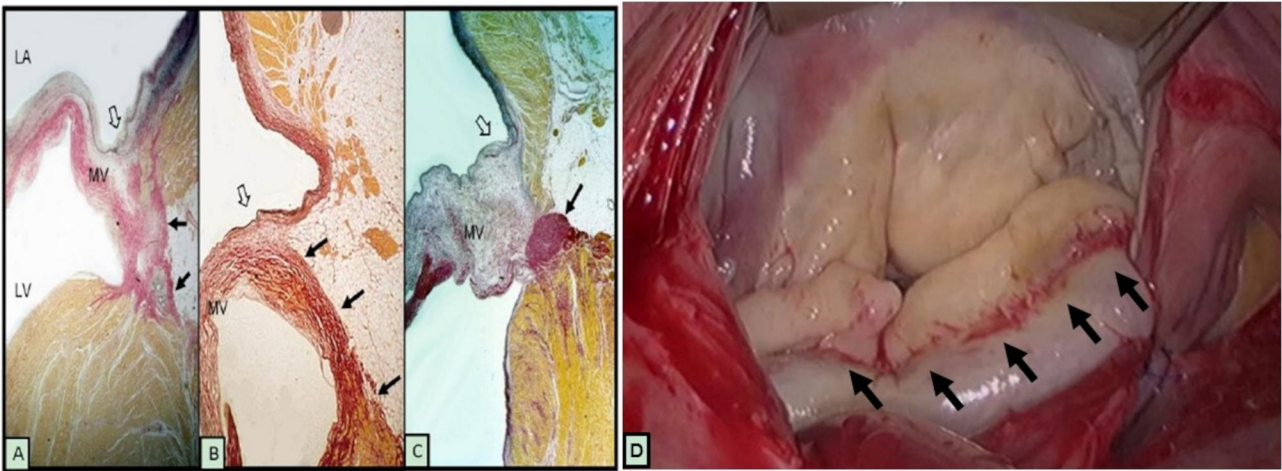


Figure 1. Typical characteristics of mitral annular disjunction. Posterior leaflet attachment to mitral annulus (open arrows), attachment of the posterior leaflet to fibrous tissue of the atrioventricular junction (black arrows) in a normal valve (A and B). Myxomatous posterior mitral leaflet hinged to the atrial wall and a cord-like annulus (Arrow). Reproduced with permission from Wunderlich et al.² Typical characteristics of mitral annular disjunction with excessive valve tissue, mucoid degeneration affecting the posterior leaflet (C). Note the signs of fibrosis (black arrows) detected at the area of maximal annular mobility (D). LA, left atrium; LV, left ventricle; MV, mitral valve.

Table 1. Comparison of Imaging Modalities for the Diagnosis of MAD			
Modality	Criteria	Strengths	Limitations
TTE/TOE	MAD best visualized in parasternal or mid-oesophageal views and both in systole and diastole to exclude pseudo-MAD; Minimum systolic separation minimum >2 mm.	Widely available, real-time imaging/functional evaluation	Operator dependence, limited acoustic windows
CT	MAD defined as systolic separation >3 mm on gated images, thin-slice reconstruction of LV long-axis view	High spatial resolution, detailed anatomic characterization	Ionizing radiation, contrast exposure
CMR	MAD defined as systolic separation >2 mm on long axis views; dynamic cine evaluation to exclude pseudo-MAD	Tissue characterization, no radiation, superior dynamic assessment	Higher cost, longer acquisition time, limited availability and expertise

complementary roles, offering enhanced spatial resolution and detailed anatomical information.

Echocardiography

MAD can be identified by transthoracic echocardiography (TTE) as an atrial insertion of the posterior MV leaflet. It is mainly visualized in systole from a 2-dimensional parasternal long-axis view with the highest frame rate possible (Figure 3A and 3B); its length is measured as the maximal distance between the left atrial wall-mitral annular plane and the bulging LV myocardium.⁹ Measurements > 2 mm are considered diagnostic, although thresholds of 3–5 mm may improve specificity. Mid-oesophageal long-axis view from trans-oesophageal echocardiography are used to provide the same measurement in borderline cases or suboptimal TTE quality (Figure 3). However, MAD is rarely focal and its circumferential extension along the posterior MV annulus should be also explored using 2-, 3-, and 4-chamber apical views. With this purpose, 3D echocardiography can also be performed to identify the presence of multi-segmental MAD¹⁰ (Figure 3C). Although MAD is measured in systole, frame-by-frame dynamic examination of the images including diastole, should be performed, as a true MAD is present throughout the entire

cardiac cycle. On the contrary, pseudo-MAD is identified only during systole, and since there is no true displacement of leaflet insertion, it can be observed only in the presence of MV prolapse.⁵ It is important to note that in presence of MAD, MV annulus level is in principle redefined at the level of leaflet insertion⁹ (Figure 3D). Importantly, other MV annulus abnormalities accompany MAD and should be systematically described. Of particular importance is the identification of “curling”, as a systolic outward and apical motion of the LV basal myocardium, which results in a mid-late systolic abnormal annular expansion worsening mitral regurgitation (MR).¹⁰ In some cases, also focal annular calcifications are observed. Notably in case of repeated examinations, the evaluation of MAD and related annulus abnormalities should be reported over time and might be useful to evaluate progression and relation to clinical outcome.⁶

Cardiac computed tomography

Cardiac CT allows for structural and anatomic characterization in MAD. However, the requirement for ionizing radiation and contrast media currently limits its use. It provides exceptional spatial resolution, facilitating precise delineation of the MV annular anatomy and adjacent structures, as well as the presence

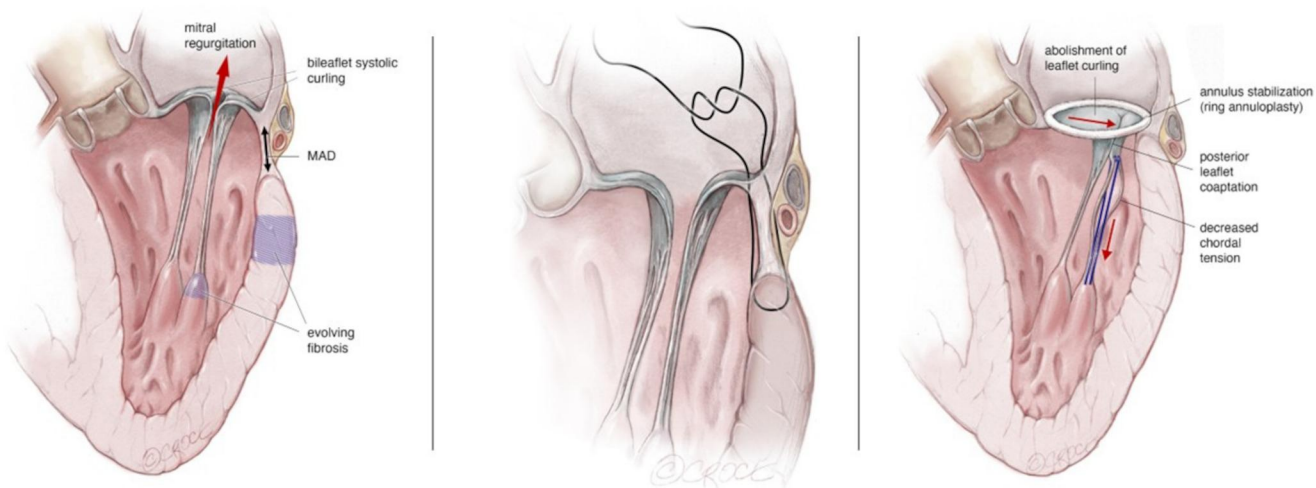


Figure 2. Surgical treatment of MAD. Evolving myocardial fibrosis due to mechanical stress (left). Use of deep annular stitches to stabilize the annulus (middle). Use of artificial chordae to reduce the mechanical stress on the leaflet.

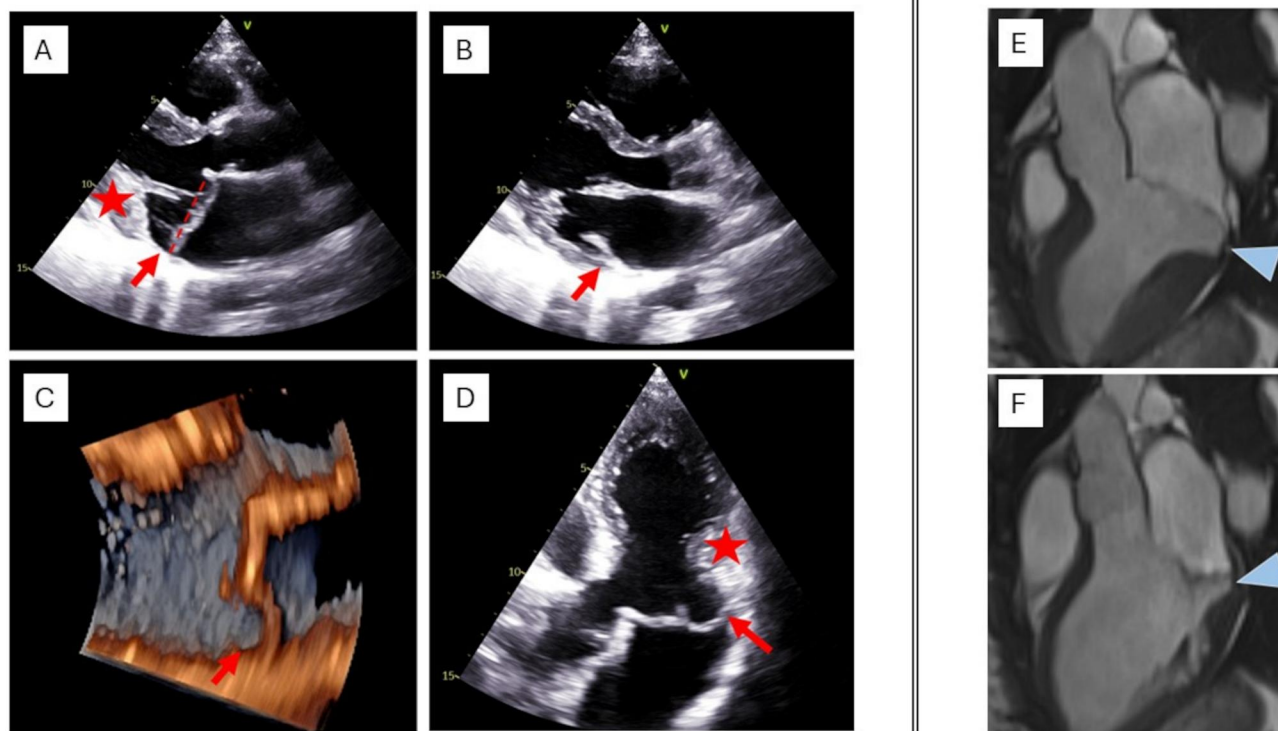


Figure 3. Mitral annular disjunction (red arrows) identified in systole (A) but not clearly visualized in diastole (B). The star indicates a posterolateral, basal myocardial hypertrophy. The dotted line indicates the MV annulus level. Another example of MAD (red arrows) visualized by 3D TTE (C) and confirmed in an apical 3-chamber view (D). Assessment of MAD by CMR (E, F). Cine three-chamber views of a patient with MAD of the P2-segment (arrowheads) measuring 11 mm in systole, demonstrating how MAD can be better assessed at end-systole (E) as compared to end-diastole (F).

of calcifications; The technique allows for quantification and localization of calcified tissue, and differentiation between fibrosis and real calcifications if echocardiography is inconclusive. Thin-slice reconstructions (≤ 0.5 mm) and multiplanar reformations are critical for accurate measurement. Retrospective ECG-gated CT acquisitions capture the dynamic motion of the mitral annulus, although the assessment of MAD in diastole (and therefore the distinction between “true” and “pseudo-MAD”) remains challenging.

Cardiac magnetic resonance imaging

CMR is well-suited to assess the presence and extension of MAD thanks to the good spatial resolution and the optimal contrast between myocardium and blood pool. This technique would also provide important information on chamber volume quantification. It also enables comprehensive myocardial tissue characterization, including detection of late gadolinium enhancement (LGE), and

increased values of myocardial T1 relaxation times and extracellular volume.¹¹ However, due to the limited availability and to the lack of expertise, this imaging modality is typically underused.

By CMR, MAD is ideally assessed using balanced steady-state free precession cine sequences acquired by using breath-hold and retrospective ECG-triggered sequences, a temporal resolution <45 ms between phases and with flip angles of 50°–80°.^{12,13} Longitudinal MAD length is best measured at end-systole in long-axis views (**Figure 2E**), while to determine the circumferential MAD extent, respective basal short-axis views shall be used.¹⁴ Up to now, there is no uniform CMR threshold definition of MAD, with some authors reporting any extent of disjunction, disjunction >1 mm, >2 mm¹⁵ or even >5 mm¹⁶ (**Figure 2F**).

INDICATIONS AND TIMING OF SURGERY

Indications for MV treatment are based on the current recommendations for the treatment of MV disease.¹⁷ According to them, the presence of MAD without severe MR even in the context of ventricular arrhythmias is not considered to be an important indications modifier. Nevertheless, based on the natural history of the disease, the majority of the patients with moderate MR and severe MAD are exposed to increased tension forces at the level of the primary chordae and the mitral annulus leading to elongation and rupture. In addition, long-lasting moderate MR may worsen during exercise leading to subsequent dilatation of the mitral annulus. Therefore, in most of the patients with severe MAD, moderate MR will deteriorate to severe earlier than in other types of chronic MR.⁶ However, the duration of the transition phase may vary significantly. To illustrate the different phases of the disease and the indication for surgical treatment we present 3 different scenarios in patients with MAD and MR.

Scenario #1: Patient with MAD and severe MR

A 34-year-old woman was referred for severe MR with MAD in the context of bi-leaflet prolapse. The first diagnosis of MVP occurred 3 years prior, during a pre-pregnancy cardiological screening, and had gradually progressed to severe MR accompanied by New York Heart Association class II dyspnoic symptoms at the time of referral. Transoesophageal echocardiography showed a Barlow's disease, with bi-leaflet prolapse and pseudo-MAD (13 mm) (**Figure 4**). MR was severe and characterized by multiple meso-telosystolic jets, with the largest regurgitant jet located centrally (A2-P2) and at the anterolateral commissure (A1-P1). A 24-h Holter ECG revealed few premature ventricular beats (posterior papillary muscle morphology) and excluded atrial fibrillation or other ventricular arrhythmias. In this case, the indication for surgery is based on the treatment of symptomatic severe primary MR due to bi-leaflet prolapse in a Barlow valve with MAD to improve symptoms and prognosis. At this stage, it remains unknown whether the treatment of mitral disease affects premature ventricular extrasystoles.

Scenario #2: Patient with MAD, mitral annular calcifications, severe MR, arrhythmogenic MVP and out of hospital cardiac arrest

A 77-year-old male patient was admitted to the emergency department after out of hospital cardiac arrest. After initial

resuscitation and defibrillation of a ventricular fibrillation with an automatic external defibrillation, spontaneous circulation was restored. TTE revealed pseudo-MAD (17 mm) with severe primary MR on the grounds of primary chordae rupture and posterior leaflet flail due to Barlow's disease. CMR revealed focal late gadolinium enhancement in the posterior papillary muscle verifying focal fibrotic spots, while CT demonstrated areas of posterior mitral annulus calcifications (MAC). Myocardial fibrosis has been recognized as a trigger of ventricular arrhythmias. The patient was scheduled for urgent MV repair with annular debridement. In this case, the indication of urgent treatment is based on the treatment of the mitral pathology along with the possibility to reduce potential triggering of life-threatening arrhythmias by mechanical stress related to MAD. Apart from that, implantable cardiac defibrillator (ICD) implantation was indicated as secondary prophylaxis to address the residual risk of ventricular arrhythmias due to detected myocardial fibrosis (**Figure S1**).

Scenario #3: Patient with MAD and moderate to severe MR

A 43-year-old male patient with moderate MR and Barlow's disease has been followed in an outpatient clinic on a yearly basis for the last 4 years. During the last follow-up, the patient mentioned increased symptoms of exhaustion and palpitations. The echocardiographic examination revealed a progression from moderate to severe MR on the grounds of a functional bi-leaflet prolapse. There were signs of left ventricular dilatation (LVESD = 45 mm) and systolic left ventricular dysfunction (EF = 43%). Two ancestors in patient's family had a history of sudden cardiac death and ventricular arrhythmias without connection to a known mitral disease (aunt died at the age of 55, mother survived an episode of ventricular fibrillation at the age of 50). MV surgery was indicated for the treatment of progressive (from moderate to severe) MR to improve symptoms and prognosis due to left ventricular dysfunction and dilatation. Genetic assessment excluded channelopathies or other known hereditary forms of syndromes associated with ventricular arrhythmias (**Figure 5**).

The aforementioned scenarios underscore the importance of identifying MR related to MAD at an early stage and before the development of anatomical and functional complications. These complications may range from life-threatening arrhythmias to severe annular calcifications, both of which significantly affect the success of mitral repair and patient's prognosis. To date, indications for intervention are limited to patients with severe MR. However, close echocardiographical and clinical monitoring are essential for early detection of disease progression, which is crucial for identifying potential candidates for early surgery, even before the onset of symptoms.

REPAIR TECHNIQUES IN MAD: RATIONALE AND PATIENT SELECTION

Patients with severe primary MR and MAD may be referred to surgery at different stages of the disease. Understanding the pathophysiological mechanisms involved in the development of MR in patients with MAD is of paramount importance for achieving a successful and durable surgical result. Pre-operative

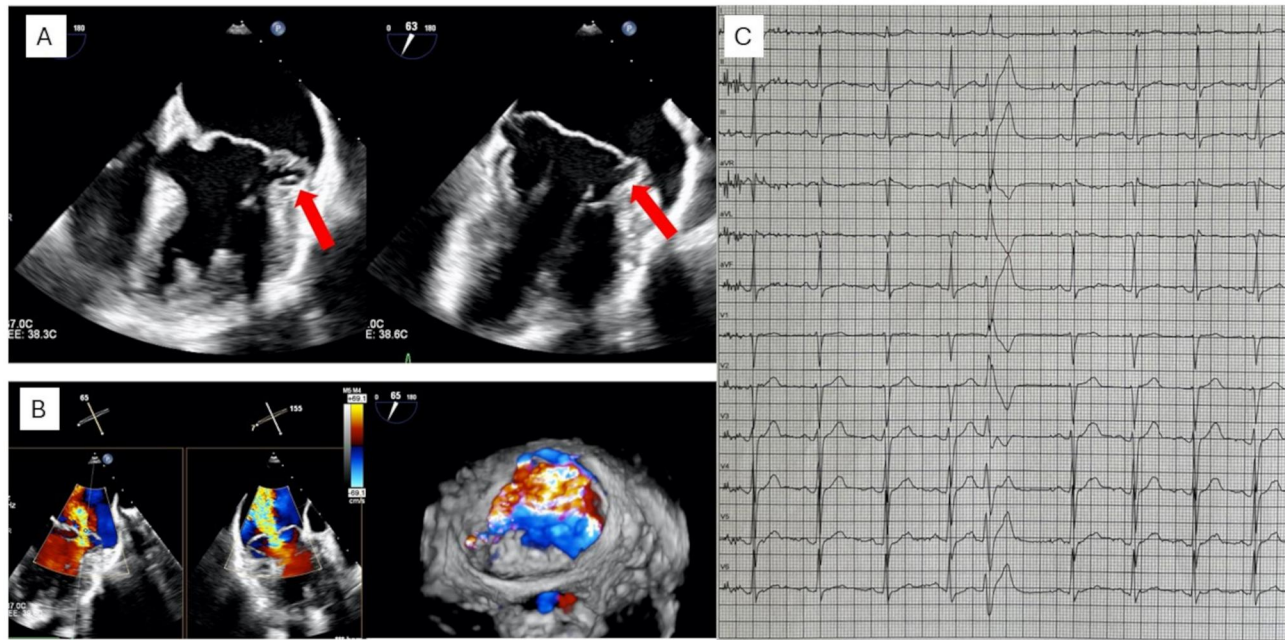


Figure 4. A Patient with MAD and severe MR. Pre-operative TOE showing a severely myxomatous mitral valve (A), with bi-leaflet prolapse and pseudo-MAD (arrow) as well as jets location (B). ECG showing normal sinus rhythm with a premature ventricular contraction originating from posterior papillary muscle (C).

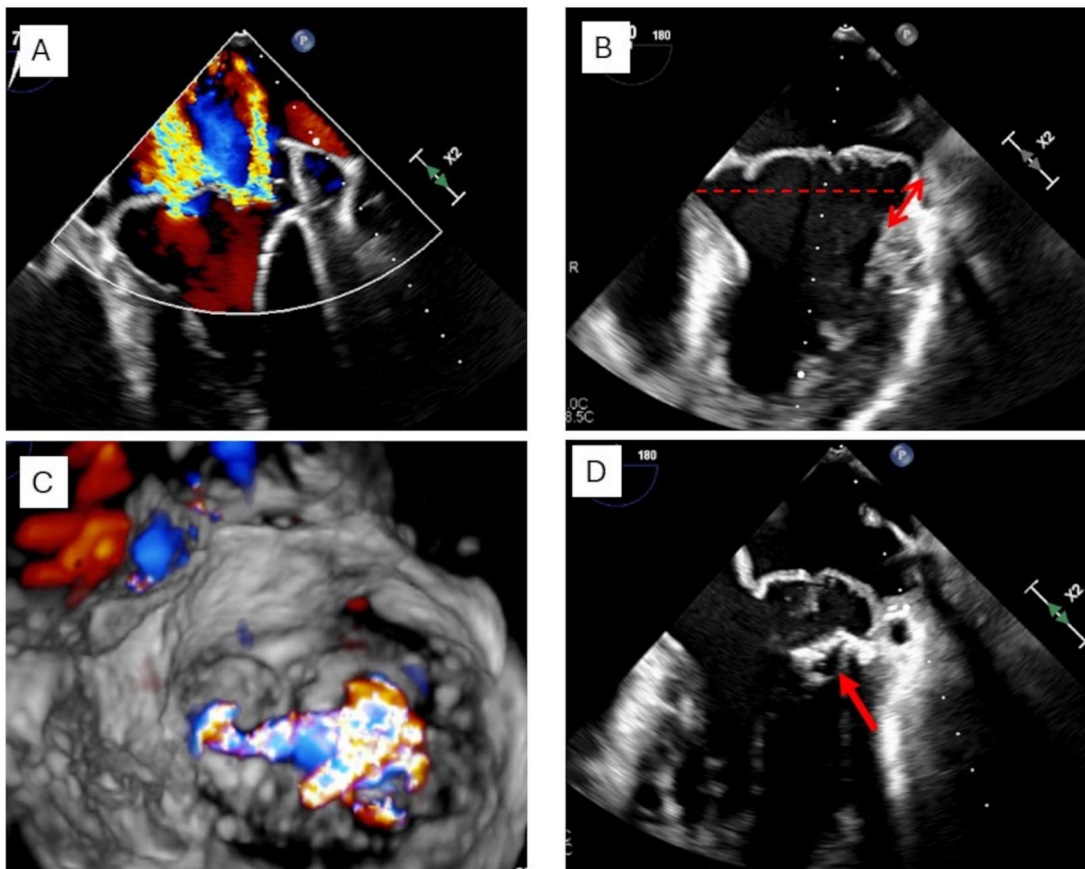


Figure 5. Patient with MAD and Symptomatic Moderate to Severe MR and History of Palpitations. Presence of 2 central jets related to a myxomatous degeneration (A), bi-leaflet prolapse (dotted line) and mitral annular disjunction (arrow) (B) with a wide loss of coaptation at the end-systole (C). Severe annular and subannular calcification (arrow) at the lateral commissure and the posterior annulus corresponding to P1 and lateral P2 segment (D).

echocardiography and intraoperative surgical valve analysis are crucial in guiding surgical repair. Specific lesions not revealed during echocardiography (ie deep indentations between scallops, leaflet perforations, leaflet prolapse masked by prolapse of opposite leaflet segments and focal infiltration/calcification of the leaflet and the annulus,) should be systematically assessed.

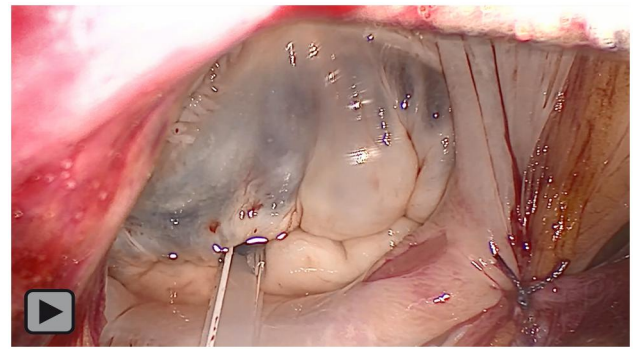
MAD is commonly presented as annular fibrosis (**Figure 1D**) and in combination with Barlow's disease. Determining the extent and location of annular fibrosis or calcification, myxomatous degeneration and the presence of jet lesions will guide the surgeon in addressing the affected part of the valve. Subsegmental analysis of leaflet pliability and mobility in relation to a non-prolapsing reference point, along with assessment of excess leaflet tissue in height and width is crucial for creating an adequate surface of coaptation and preventing of systolic anterior motion (SAM). Functional prolapse is a condition closely associated to MAD and can be recognized by the almost complete abolishment of MR at water test prior to the beginning of any repair measures (**Video 1**). Finally, the anatomy of the subvalvular apparatus is highly variable and may present with distinct lesions (chordae elongation or rupture) that need to be addressed during MV repair.

ANNULOPLASTY-ONLY IN PATIENTS WITH FUNCTIONAL PROLAPSE

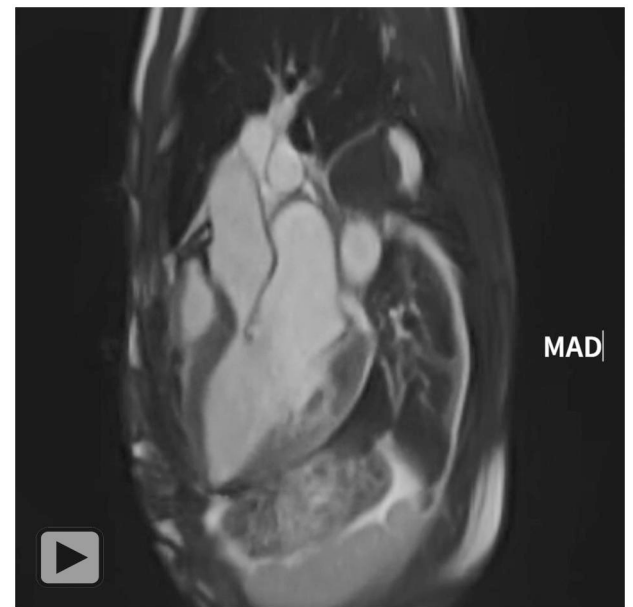
MAD is associated with a pathological leaflet motion caused by excessive movement of the posterior annulus. Therefore, remodelling and stabilizing the mitral annulus during MV repair is therefore essential for the treatment of MAD, regardless of the leaflet repair technique used. In cases presented with a central regurgitation jet, indicating the absence of additional leaflet pathology, annuloplasty alone can correct the functional prolapse by eliminating abnormal and excessive annular motion and addressing potential annular dilation (**Video 2**). This is achieved by reducing the septo-lateral annular diameter and creating a broad surface of leaflet-to-leaflet coaptation, as in most cases of chronic MR. The use of deep annuloplasty stitches particularly along the posterior annulus, aims to engage the base of the left ventricular myocardium and reduce the atrioventricular gap, which is characteristic for MAD (**Figure 2**). This technique stabilizes the mitral annulus in an early systolic frame. At an early disease stage, annuloplasty alone may restore MV competence. Increased recognition and understanding of the concept of functional prolapse have simplified the repair strategy in patients with MAD and Barlow's phenotype. Consequently, in these patients, anterior leaflet billowing usually does not require direct intervention, as annuloplasty resolves the functional component of leaflet curling.¹⁸ However, the coaptation line may shift anteriorly after repair, predisposing to SAM.¹⁹ In such cases, additional posterior leaflet repair techniques may be required to reduce effective height.

ANNULOPLASTY WITH POSTERIOR LEAFLET REPAIR IN PATIENTS WITH FUNCTIONAL PROLAPSE

In patients presented at early stage with functional prolapse and MAD, the risk of SAM should be carefully evaluated. In the subgroup of patients with high risk of SAM, it is crucial to reduce the



Video 1. Water-Test Demonstrating a Competent Mitral Valve Before Repair in a Patient With Functional Prolapse and MAD. This can be used as a proof of the presence of functional prolapse.



Video 2. Abolishment of Abnormal Annular Movement and Restoration of Leaflet Coaptation by Ring Annuloplasty.

height of the posterior leaflet, moving the closure line posteriorly, away from the Left Ventricular Outflow Tract (LVOT) and towards the inflow portion of the LV. To accomplish this goal and depending on the amount of excess tissue in width and height, the surgeon can use resectional techniques (i.e. quadrangular resection and leaflet sliding technique, butterfly technique,²⁰ resection of the base of the posterior leaflet, foldoplasty²¹) or techniques respecting the posterior leaflet. When choosing to respect, either verticalization of the posterior leaflet, by implanting shortening neochords,²² or by using the so-called snail technique²³ can be used (**Figure 2**). Both resectional and non-resectional techniques are equally efficient in obtaining excellent early long-term outcomes in terms of repair rate and repair durability.^{24,25}

ANNULOPLASTY WITH POSTERIOR LEAFLET REPAIR IN PATIENTS WITH ANATOMICAL POSTERIOR LEAFLET PROLAPSE OR FLAIL

In patients with MAD-associated mitral disease at a more advanced stage, true prolapse of posterior leaflet, due to chordal

elongation or rupture may develop. Both conditions will result in an eccentric jet on echocardiography, characteristic of an asymmetric Barlow valve with severe MR. In addition to the obligatory annular remodelling,²⁶ all above-mentioned surgical techniques can be applied to treat MVP. In recent years, a gradual shift in paradigm of posterior leaflet repair is observed²²; due to the easy application of neochords in minimally invasive procedures, surgeons tend to use more artificial chordae rather than resection techniques. Many studies have demonstrated that repair rates and durability are comparable for both techniques.^{25,27,28}

ANNULOPLASTY WITH LEAFLET REPAIR IN PATIENTS WITH ANATOMICAL ANTERIOR LEAFLET PROLAPSE OR FLAIL

The need to address the anterior leaflet in patients with bi-leaflet curling and functional prolapse has been abandoned overtime if no true elongated or ruptured chordae are found in valve analysis.¹⁸ Obviously, a true prolapse of the anterior leaflet should always be corrected. While in posterior leaflet prolapse, resection is a commonly applied repair technique, it is infrequently performed at the anterior leaflet. Chordal replacement, either using artificial or autologous chords (chordal transfer) are the most common repair strategies.²⁹ Chordal replacement to address anterior leaflet prolapse may be more prone to early and mid-term repair failure when compared to correction of the posterior leaflet prolapse.^{30,31} This may be due to the forces applying on the anterior leaflet and the narrow margin between restriction and residual prolapse.

One of the difficulties of correcting anterior leaflet prolapse using artificial chords is the adjustment of the length of the chords. Most surgeons use the anterior annulus as a reference. Using pre-measured loops referenced on the length of adjacent autologous chords is a well reproducible alternative. When using this technique, it is paramount to secure several loops at the prolapsing part of the anterior leaflet to evenly distribute forces. Finally, the choice of annuloplasty device may be crucial in those patients. The use of a complete ring rather than an open band, allows for anterior annulus stabilization, which is needed to obtain sufficient leaflet coaptation.³²

ANNULOPLASTY WITH EDGE-TO-EDGE REPAIR

The edge-to-edge technique, has been initially introduced for the treatment of primary MR and represents an important tool for patients with MAD and Barlow phenotype, especially at the presence of complex pathologies (ie, bi-leaflet, anterior leaflet or commissural prolapse). The combination of edge-to-edge repair and ring annuloplasty leads to complete abolishment of MAD, durable treatment of leaflet prolapse and abolishment of SAM. Additionally, it serves as a bailout option in cases of suboptimal immediate result of conventional mitral repair techniques.³³ It must be emphasized that any form of edge-to-edge repair, whether surgical or transcatheter, performed without the addition of annuloplasty fundamentally fails to address the primary mechanism of mitral disease, which lies in MAD and annular hypermobility. The absence of annuloplasty inevitably leads to persistent mechanical stress on the primary chordae, promoting

their progressive elongation and eventual rupture, and thereby predisposing to recurrent or residual MR.

A successful application of the technique hinges on adhering to key principles, such as limiting the suture to the zone of the primary regurgitant jet (central or commissural), employing a horizontal mattress suture followed by a running suture, and respecting leaflet symmetry to avoid distortion (**Video 3**). Of note, the distance of the sutures from the free edge is adjusted based on leaflet redundancy, ensuring an effective and balanced repair.

Despite its broad applicability, contraindications to the edge-to-edge technique include those scenarios with high probability of developing mitral stenosis such as MAD with leaflet or severe annular calcifications. Moreover, it is not recommended in cases of non-localized regurgitant jet and multiple mitral lesions involving non-facing segments of the anterior and posterior leaflets. Finally, it should be avoided whenever annular dilatation cannot be corrected by a concomitant annuloplasty, as the absence of annuloplasty is likely associated with early failure of the repair.³⁴ Indeed, both complete rings and posterior bands, in the context of Barlow disease, have shown similar outcomes in terms of effectiveness and durability.^{35,36}

SELECTION OF ANNULOPLASTY RING

Annuloplasty remains one of the mainstays of MV repair. In contrast to conventional cases of primary MR, the principal objective of annuloplasty in patients with MAD is the correction of annular hypermobility, whereas the reduction of annular dilatation and the increase of leaflet coaptation serve as secondary goals. Stabilizing the posterior annulus constitutes a critical component of surgical management of MAD, as it prevents systolic curling of the posterior leaflet and subsequent residual prolapse. Therefore, the use of deep annuloplasty stitches engaging the ventricular myocardium is of paramount importance for the effective elimination of MAD. While the ring implantation remains standard practice, the choice of ring type and configuration is still a matter of ongoing debate.

Carpentier first described the importance of a “remodelling annuloplasty” with a rigid ring that mimics the mitral annulus in systole. Rigid rings are crucial for the treatment of annular dilatation, which is a consequence of chronic Primary Mitral Regurgitation (PMR). However, increased understanding of the pathophysiology of PMR enabled repair with complete semi-rigid rings, open rings or bands, which yield comparable results



Video 3. Edge-to-Edge Repair Technique and Annuloplasty to Treat a Patients with Bi-Leaflet Prolapse.

in terms of survival, freedom from reoperation and freedom from recurrent MR up to 14 years after surgery.^{35,37-39} Theoretically, open rings or bands in addition to posterior annulus stabilization, might have some technical and pathophysiological advantages. Compared with complete rings, posterior annuloplasty devices do not require suturing the anterior annulus, therefore resulting in a shorter, easier (especially when visibility of the anterior annulus is limited) and safer (lower risk of aortic valve injury) procedure. Moreover, bands are technically forgiving (lower risk of valve distortion) and, through repositioning of commissural stitches, they confer the possibility of readjustment (**Figure S2**).

From a pathophysiological standpoint, the implantation of a posterior annuloplasty device helps in preserving annular dynamics in diastole and systole, leaving larger diastolic opening and decreasing the risk of SAM. During systole, both the anterior leaflet and the anterior annulus move posteriorly and apically to allow expansion of the left ventricular outflow tract and the aortic root.⁴⁰ As shown with 3D cine phase-contrast flow CMR semi-rigid complete rings restrict aortic annulus motion, disturbing aortic outflow patterns, while partial flexible or open rings and bands do not.⁴¹ Moreover, posterior partial annuloplasty has been associated with lower postoperative gradients when compared with complete rings,^{26,42} and this difference is even more evident during exercise.⁴³

Although annuloplasty has been shown to abolish MAD,^{10,44} a proper comparison between the different types of rings in terms of efficacy in MAD elimination has not been performed yet. The area of maximal disjunction is located at the infero-lateral part of the posterior annulus (corresponding to P1-P2 leaflet segment), therefore making a posterior annuloplasty device potentially enough to ensure MAD elimination after mitral repair. Anyhow, careful attention is required in this context when placing the ring stitches, as rare cases of post-operative MAD persistence have been reported.¹⁰

SPECIAL CONDITIONS

Syndromic MVP

Syndromic MVP has been associated with several genetic conditions with high genotypic and phenotypic heterogeneity (**Table S1**). The biomedical pathways associated with it include altered migration of primordial cell populations, increased TGF- β signalling, impaired synthesis and remodelling of extracellular matrix (ECM) components, as well as alterations in proteoglycan production. Most of them result in an increased proliferation of valve interstitial cells as well as migration of inflammatory cells into the leaflet tissue.⁴⁵ Furthermore, secretion of ECM proteins, accumulation of low stiffness collagen fibres and calcium deposition contribute to further activation of inflammatory pathways. This results in increased leaflet thickness and development of excessive tissue.⁴⁶ In contrast to non-syndromic MVP, syndromic conditions are characterized by increased fibrosis and calcifications which further alter valve stiffness and geometry. Progressing tissue degeneration leads to increased mechanical stress on valvular and subvalvular structures, altered proteoglycan production and apoptosis which activates a vicious cycle leading to chordae rupture. The presence of mechanical stress causes accelerated progression of MR with an annual increase in regurgitant volume of 18.4 ml per year.⁴⁶

MR represents, after aortic aneurysms, the second most common cardiovascular pathology of patients with Marfan syndrome. MVP, MAD and excessive valve tissue indicative of Barlow's valve are typically found in Marfan patients with MV involvement. In the largest series to date, MV repair was associated with improved survival as compared to replacement. However, one-third of the patients had at least moderate recurrent MR at a median follow-up of 16 years, while only 2 patients underwent reoperation.⁴⁷

Mitral annular calcifications

MAC is a degenerative process of the fibrous portion of the atrioventricular junction found in 8%–15% of the general population and when present, is associated with significant MV disease. The calcification pattern starts at the posterior annulus and extends anteriorly towards the fibrotic trigones.⁴⁸ This degenerative process is accelerated by mechanical stress induced by increased filling pressures (ie arterial hypertension, atrial fibrillation) or by annulus deformation (ie hypertrophic cardiomyopathy or MAD).⁴⁹

From a pathophysiological view, the mitral annulus is exposed to significant mechanical stress which activates inflammation and subsequent fibrosis. The presence of myofibroblastic differentiation of valvular interstitial cells as well as infiltration and proliferation of inflammatory cells support the hypothesis of calcification through mechanotransduction.⁵⁰ The hypermobile annular tissue gradually becomes fibrotic, whereas fibrosis may extend from the annulus to the LV myocardium and/or the papillary muscles.⁵¹ Calcification is the result of alterations in interactions between valve interstitial cells and ECM, which trigger further calcification, tissue degeneration, and chordal rupture, thus exacerbating MR. Patients may be referred to surgery or MV intervention at different stages of MAC (**Figure 6**).

Surgical techniques may vary according to the extension of MAC and may include standard types of repair without the need to remove calcium in patients with limited subannular calcifications. In more advanced cases, detachment of the posterior leaflet from the annulus followed by annular debridement with annulus reinforcement and reattachment of the posterior leaflet can be considered (ie young patients with extensive MAC). Annuloplasty sutures are more atrially placed in these cases to avoid rupture of the calcium bar. It is important to mention that the placement of an annuloplasty ring does not target annular hypermobility in cases of MAC but rather to re-establish an adequate coaptation area and avoid residual MR. Older patients usually require surgical or transcatheter MV replacement depending on clinical and anatomical conditions and the risk of atrioventricular dehiscence. Surgical MV replacement can be performed with annular debridement and patch reconstruction prior to prosthesis implantation or using a transcatheter heart valve with no or minimal decalcification.

Systolic anterior motion

Systolic anterior motion (SAM) refers to a mechanical obstruction of the left ventricular outflow when dragging forces during systole push the anterior mitral leaflet towards the interventricular septum. Patients with excessive leaflet tissue referred for treatment of primary mitral disease are at risk of SAM, especially if conditions such as hypermobile anterior leaflet, hypertrophy

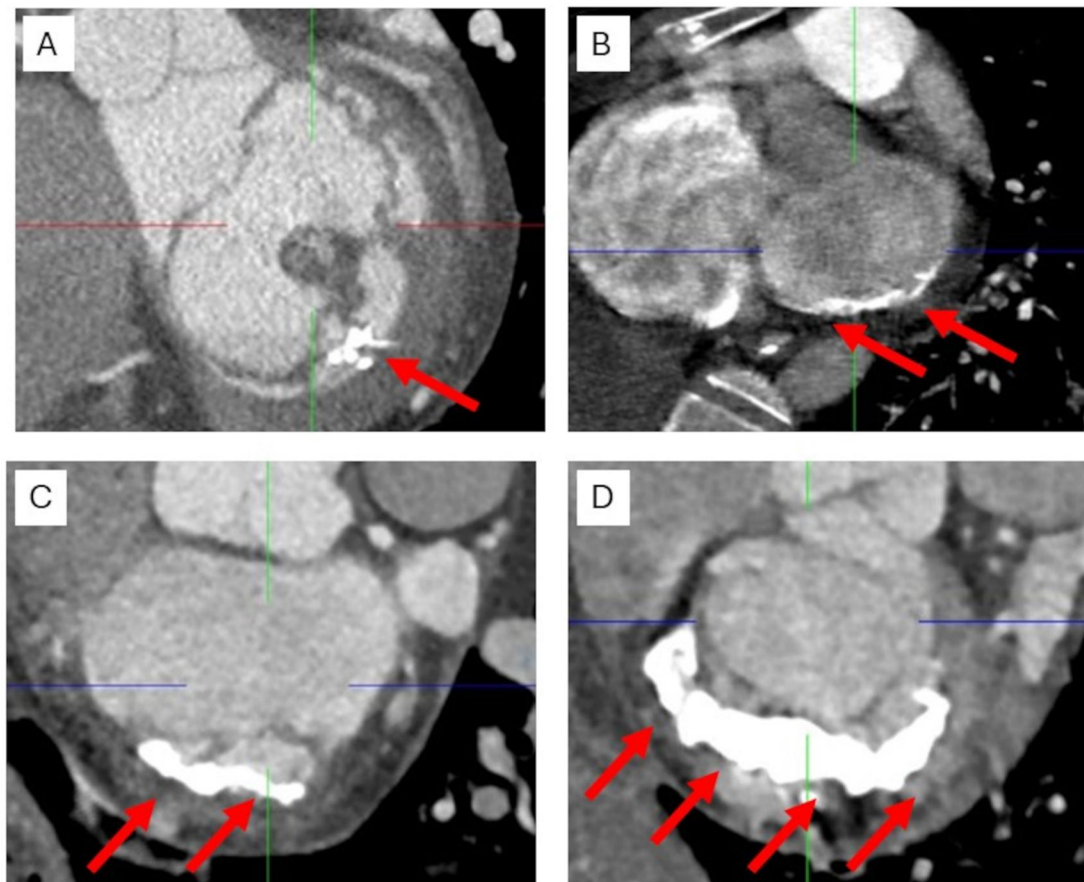


Figure 6. CT Scan from 4 Different Patients Presenting with Mitral Annular Calcification (Arrows) at Different Stages. Spotty calcification affecting a small part of the posterior annulus (A). Thin calcification affecting only the hinge of the posterior leaflet corresponding to P1 and P2 segment (B). Moderate calcification involving part of the posterior annulus and the base of the posterior leaflet (C). Heavy (Shoe-Horse-Like) calcification of the posterior annulus affecting part of the posterior leaflet (D).

of the interventricular septum, aortomitral angle $<120^\circ$, C-sept distance <25 mm, tall posterior leaflet and use of smaller annuloplasty rings are present.⁵² Patients with MAD referred for surgical treatment commonly present with a Barlow's phenotype with excessive leaflet tissue and bi-leaflet curling and SAM can occur after repair if leaflet coaptation is anteriorly misplaced. Treatment of SAM can be achieved by improving ventricular filling, treating tachycardia and abandon inotropic stimulation. In patients with severe SAM accompanied by severe residual MR despite conservative measures, surgical re-repair is indicated. This can be achieved by the use of significantly larger annuloplasty rings, by ventricularization of the excessive posterior leaflet or by surgical edge-to-edge techniques.⁵³

Assessment of MAD treatment—long-term results

The assessment of long-term results after MAD treatment is based on multimodality imaging and electrocardiography (ECG) monitoring.

ECG-monitoring

Incompletely treated MAD after mitral annuloplasty may be related to persistent ventricular arrhythmia.¹⁰ Furthermore, even after successful MVR with accurate annuloplasty stabilization of MAD, worsening of arrhythmias might still happen in around

20% of patients after surgery.⁴⁴ According to the European Heart Rhythm Association (EHRA) expert consensus statement, a standard 24-h Holter monitoring and a close follow-up are warranted in patients with MVP or complex ventricular ectopy, respectively.⁹ Additionally, due to the possibility of disease progression, periodic Holter may be advised as part of the routine follow-up. Developing specific risk stratification protocols is necessary for patients not responsive to the antiarrhythmic effect of MV surgery. Meticulous electrophysiologic evaluation is of utmost importance in these patients. Reasonable medical management options include beta blockers and amiodarone.

According to the EHRA expert consensus, an implantable loop recorder (ILR) may be advised in patients with MVP and unexplained syncope in whom non-invasive ECG monitoring remains inconclusive.⁹ ILR may also be advised to patients with arrhythmic MVP with negative findings on CMR and high-risk features (Table 2).

Primary prevention with an ICD is recommended in patients with arrhythmic MVP, left ventricular ejection fraction $<35\%$, and symptomatic heart failure despite ≥ 3 months of optimal medical therapy.⁹ An ICD may also be considered in patients with a history of unexplained syncope and sustained or non-sustained ventricular tachycardia likely arising from the mitral apparatus or in patients with one high-risk feature and 2 or more phenotypic risk features. Patients with arrhythmic MVP

Table 2. Risk Features of Malignant Arrhythmias and Sudden Cardiac Death

High risk features	Phenotypic risk features
Sustained ventricular tachycardia	T-wave inversion in the inferior leads
Non-sustained ventricular tachycardia	Repetitive documented polymorphic premature ventricular contractions
Unexplained syncope	Mitral annular disjunction
	Redundant mitral leaflets
	Enlarged left atrium
	Ejection fraction $\leq 50\%$

and a documented history of ventricular fibrillation or haemodynamically not tolerated ventricular tachycardia should receive an ICD as a secondary prevention strategy.

Echocardiography

Immediately after MV repair, MAD should no longer be detected on intraoperative transoesophageal echocardiography. Procedures performed at a more advanced stage of MAD, that is involving the anterior leaflet or both leaflets are associated with a higher risk of recurrent MR when compared to an isolated posterior leaflet prolapse.^{54,55} Therefore, baseline echocardiographic assessment following MV repair is essential to detect residual MR.^{54,56} The recurrence of significant MR is more common in the first year following repair.⁵⁴ This allows an increase of the follow-up time interval if the postoperative result remains stable in the long term.⁵⁷ Long-term follow-up should take into consideration serial assessment of function and dimension of the left ventricle.⁵⁴ Early repair failure was found to have a different mechanism compared to late failure, is more likely to relate to a technical failure and is more amenable to redo repair.⁵⁸ Late recurrent MR is typically related to the progression of the degenerative MV disease rather than failure of the repair⁵⁹ (Figure S3).

IMPACT OF SURGERY ON ARRHYTHMIAS

The genesis of ventricular arrhythmias (VA) in patients with MAD is a multifactorial process. Several potential contributors are implicated, including severe MR,⁶⁰ myocardial fibrosis,^{61–64} prolapse-induced myocardial stretch.^{65,66} MV surgery, by stabilizing the annulus, eliminating prolapse, and correcting MR, is theoretically expected to reduce electrical instability and VA. However, current evidence on its efficacy remains controversial.

Older case reports and series described the potentially beneficial role of MV surgery in alleviating symptoms and reducing VA burden in patients with MVP and severe arrhythmias but without significant MR.⁶⁷ A renewed interest in the topic has recently fueled a surge of new investigations on the antiarrhythmic effects of surgery in patients with concomitant significant MR, but the results have been inconsistent. While some series reported a post-surgical reduction in VA burden, many others suggested that mitral surgery may not uniformly affect it.^{68,69} Undoubtedly, the presence of MAD is associated with a 3-fold increased long-term risk for ventricular arrhythmias as compared to non-MAD patients. This risk remains similar regardless of MV repair or replacement.⁷⁰

As a matter of fact, the proportion of arrhythmogenic MVP patients free from recurrent VA at follow-up ranges between 55%⁴⁴ and 75.9%,⁷¹ and different characteristics have been associated with persistent post-surgical VA. Those include age at intervention, complexity of pre-operative VA⁷¹ and the absence of posterior MAD, thus suggesting the presence of additional electrical triggers, which may not be addressed by mitral surgery.^{44,72} Moreover, no repair technique has yet demonstrated superior antiarrhythmic effects over the others.^{44,71} A potential explanation for the unpredictability of surgery's impact on VA relies in MVP pathophysiology itself. While an early referral seems imperative to ensure antiarrhythmic efficacy, this premise clashes with the reality that MR usually develops late, after years of prolapse and stress on the left ventricular myocardium.⁷¹ Some groups have proposed adding concomitant cryoablation of the papillary muscles during MV surgery.^{73,74} It remains though unclear to which extent the induction of additional fibrotic lesions will decrease or increase the existing arrhythmic risk. Multimodality imaging, using late gadolinium enhancement CMR⁷⁵ or hybrid PET/CMR,⁷⁶ allows identifying areas of myocardial fibrosis which can be addressed during surgery.

It is worth noting that some reports^{44,77} have described the onset of a significant post-operative arrhythmic burden after successful surgery in a subset of patients without history of arrhythmia. This finding indicates the possibility of a myocardial excitability progression during MVP subjects' natural history. A proarrhythmic effect of surgery itself cannot be excluded, and arrhythmogenic mechanisms—such as the creation of new substrate through post-surgical scarring—are postulated but have not been confirmed. A description of the available evidence on the impact of mitral surgery on VA is summarized in Table S2. At least 4 prospective trials (NCT05562804, NCT06255457, NCT05051033, and R01HL166720) are ongoing and will soon provide more insights on the topic.

GAPS IN EVIDENCE

Despite increasing recognition of mitral annular disjunction (MAD) as a clinically relevant entity several important gaps in evidence persist. First, the lack of standardized diagnostic criteria across imaging modalities leads to inconsistent identification and limits comparability between studies. Second, the natural history of MAD remains poorly defined. Longitudinal data are lacking to determine which patients with moderate MR and MAD are at greatest risk of progression to severe disease or arrhythmic complications. Consequently, the timing of surgical intervention in these patients remains uncertain. Current guidelines largely reflect the presence of severe MR and do not incorporate MAD-specific biomechanics or arrhythmic risk. Furthermore, although MV repair can anatomically correct MAD, its influence on ventricular arrhythmia burden is inconsistently reported. The pathophysiological mechanisms linking MAD and arrhythmogenesis also remain incompletely understood, with ongoing debate as to whether MAD constitutes a true arrhythmic substrate or reflects broader myocardial vulnerability due to local fibrosis. In addition, the role of MAC in the context of MAD is insufficiently studied. The causative relationship between annular hypermobility and calcification remains speculative, and surgical outcomes in patients with coexisting MAC are under-reported. Finally, there are no validated risk stratification tools for the postoperative assessment of

arrhythmic risk or repair durability, and the genetic and paediatric aspects of MAD remain largely unexplored. These gaps limit tailored decision-making and underscore the need for prospective, multimodal research.

SUPPLEMENTARY MATERIAL

[Supplementary material](#) is available at *EJCTS* online.

CONFLICTS OF INTEREST

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DATA AVAILABILITY

The data underlying this article are available in the article and in its [online supplementary material](#).

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