Pre-operative systolic anterior motion of the mitral valve in a patient undergoing mitral valve repair

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Abstract

A patient with myxomatous mitral valve prolapse underwent mitral valve repair due to severe symptomatic mitral regurgitation. Preoperative echocardiography demonstrated systolic anterior motion of the mitral valve. This finding disappeared once spontaneous chordal rupture occurred, resulting in a flail posterior mitral leaflet. As the patient was considered at high risk of developing post-repair SAM, he was operated on using surgical techniques aimed at lowering the risk of this complication. Despite this, post-repair SAM did develop and could only be eliminated by a surgical edge-to-edge (Alfieri) repair.

Keywords

Cardiac surgery, echocardiography, mitral valve, valve repair

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Introduction

Systolic anterior motion (SAM) of the mitral valve (MV) leaflets towards the interventricular septum may lead to dynamic left ventricular outflow tract (LVOT) obstruction associated, at times, with profound haemodynamic compromise. Although most commonly associated with hypertrophic cardiomyopathy, it can also occur in other settings, including after surgical repair of the MV.¹ We report a unique case of a patient with myxomatous MV disease and MV prolapse who underwent MV repair due to severe symptomatic mitral regurgitation (MR). Interestingly, preoperative echocardiography demonstrated MV SAM. This finding disappeared once spontaneous chordal rupture occurred, resulting in a flail posterior MV leaflet. As the patient was considered at high risk of developing post-MV repair SAM, he was operated on using surgical techniques aimed at lowering the risk of this complication. Despite this, post-repair SAM did develop and could only be eliminated by a surgical edge-to-edge (Alfieri) repair. A brief review of the literature regarding the prediction, surgical preventive measures, and treatment of post-operative SAM is presented.

Case presentation

A 61-year-old male patient was referred for transthoracic echocardiography due to an incidental finding of a systolic

heart murmur during a routine physical examination. His past medical history was unremarkable. Notably, there was no history of hypertension or family history of cardiomyopathy. Echocardiography revealed mild enlargement of the left ventricle (LV) (end-diastolic diameter 58 mm), mildly thickened LV walls (end-diastolic interventricular septum thickness 14 mm; posterior wall thickness 12 mm), and a normal LV ejection fraction (~70%). Myxomatous MV disease was evident, with severe prolapse of all three posterior leaflet scallops and severe mitral regurgitation (MR), with an eccentric anteriorly directed MR jet. The non-prolapsing anterior MV leaflet was elongated and exhibited mild anterior leaflet SAM (Figure 1 and Supplementary Video Clip 1, available online), without dynamic LVOT obstruction at

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Figure 1. Apical 4-chamber view (left-sided cardiac chambers at the left side of the picture; mid-systolic frame) demonstrating posterior mitral valve leaflet prolapse (arrow) and anterior leaflet systolic anterior motion (asterisk).

rest or during a Valsalva manoeuvre. Mild myxomatous disease of the tricuspid valve was also apparent, resulting in mild-to-moderate tricuspid regurgitation. The estimated pulmonary artery systolic pressure was normal (32 mmHg). The left atrium was mildly dilated and right ventricular systolic function appeared normal. As the patient was asymptomatic and had preserved LV function, he was advised to continue routine follow-up at the heart valves clinic at our institution. The complex MV anatomy, which lowered the chances of successful valve repair, also played a role in our decision to pursue a conservative therapeutic approach.

Two years later the patient started to suffer from dyspnoea on exertion (New York Heart Association class II symptoms). A repeat echocardiographic study revealed rupture of multiple chords resulting in a large flail segment of the posterior MV leaflet [flail middle (P2) scallop] causing, in turn, severe MR (Figure 2 and Supplementary Video Clips 2–3). Interestingly, once the posterior leaflet became flail, SAM of the anterior leaflet was no longer evident. Compared to the previous examination, LV size increased (end-diastolic diameter 62 mm) without worsening of LV function, the left atrium became severely dilated, tricuspid regurgitation worsened (moderate tricuspid regurgitation was evident), and pulmonary artery systolic pressure rose steeply to 68 mmHg. Due to the appearance of symptoms and the abovementioned echocardiographic findings, the patient was referred to surgery with an intention to perform MV repair.

On the basis of the sequence of echocardiographic findings described above, post-operative SAM was anticipated

and the surgical technique was modified in an attempt to prevent its occurrence. MV repair included: (a) quadrangular resection of most of the large middle posterior (P2) flail scallop; (b) suturing of the posterior leaflet in order to decrease in its height and move the coaptation point of the leaflets posteriorly; (c) implantation of two artificial chords to the non-resected posterior leaflet segments (on both sides of the resected segment); and (d) sliding annuloplasty with implantation of a large 38-mm open annuloplasty ring. However, despite the undertaking of these preventive measures, an intra-operative, post-repair transoesophageal echocardiographic examination revealed severe MV SAM, which caused profound LVOT obstruction and haemodynamic compromise (Figure 3 and Supplementary Video Clip 4). Conservative attempts to relieve this disturbance failed and surgical repair was necessary. An Alfieri technique was performed, with the two MV leaflets sutured at their middle aspect, resulting in a double orifice MV and abolishment of SAM with its associated LVOT obstruction.

Since the operation (2 years ago), the patient remained completely asymptomatic, without echocardiographic signs of signs of MV dysfunction (mitral stenosis, MR, or SAM) (Figure 4 and Supplementary Video Clip 5). LV size and function, as well as pulmonary artery pressure, returned to normal.

Discussion

SAM is not an uncommon complication of MV repair surgery, occurring in approximately 5–10% of patients.² This relatively high incidence of SAM can be partially explained by haemodynamic changes that occur during this operation, such as increased contractility (with or without positive inotropic medications) and/or reduced preload or afterload, that are associated with an increased propensity for the development of SAM. However, different studies have suggested that certain intrinsic properties of the MV itself and its orientation relative to the interventricular septum are associated with an increased risk for the development of SAM.³

Maslow et al.³ defined the preoperative parameters associated with an increased risk of developing post-MV repair SAM. These include: (a) a lower ratio of the anterior to posterior MV leaflets length; and (b) a smaller distance between the coaptation point of the MV leaflets and the interventricular septum. Another feature in SAM-prone patients is MV leaflet elongation, a common finding in patients with myxomatous MV disease. These features result in a larger portion of the anterior leaflet residing inside the left ventricle, adjacent to the LVOT. Assessing the presence of the features associated with SAM necessitates performance of measurements on a MV in which the leaflets coapt. As many patients suffering from myxomatous MV present with flail leaflets and, hence, have

Figure 2. (a) Apical 4-chamber view (left-sided cardiac chambers at the left side of the picture) demonstrating chordal rupture with a flail posterior mitral valve leaflet (arrow) and disappearance of systolic anterior motion (asterisk). (b) Transoesophageal echocardiography with colour flow imaging from the mid-oesophageal long-axis view demonstrating the flail segment (arrow) and severe mitral regurgitation. (c) 3-Dimensional transoesophageal echocardiography demonstrating a large flail P2 segment (asterisk) and ruptured chordae (arrow).

Figure 3. (a) Intra-operative post-cardiopulmonary bypass transoesophageal echocardiography (4-chamber mid-oesophageal view) showing severe systolic anterior motion (arrow) postmitral valve repair.

impaired leaflet coaptation, obtaining measurements to assess the risk of SAM in these patients may not be feasible. Although some have suggested such measurements can be obtained even in this group of patients, by calculating where the coaptation point would have occurred and then extrapolating the parameters listed above,³ this can result in inaccurate measurements and a wrong assessment of the risk for developing this complication.

Once patients are identified as being at increased risk of developing post-repair SAM, they can be operated on by the use of surgical techniques addressed to lower this risk. These techniques use different approaches to achieve the common goal of creating a post-repair MV with features associated with a lower risk of developing SAM, i.e. a valve with a coaptation point located more posteriorly and with a minimal part of the anterior leaflet beyond the coaptation point. Surgical techniques that have been shown to obtain such a post-repair MV include the use of large

Figure 4. Post-operative transthoracic echocardiography. (a) Apical long-axis view demonstrating the abnormal mitral valve morphology following the Alfieri valve repair, with prominent doming of the two surgically connected valve leaflets. (b) Parasternal basal short-axis view demonstrating a double-orifice mitral valve (asterisks) and the connection between the 2 mitral valve leaflets (arrow). (c) Continuous-wave Doppler across the mitral valve, demonstrating the absence of a significant trans-mitral pressure gradient following the Alfieri valve repair.

annuloplasty rings (or not using them at all) and/or resection or shortening of either the posterior or anterior MV leaflets.⁴⁻⁸ An alternative technique that has been recently described by Sternik and Zehr⁹ consists of transferring of secondary chordae of the posterior leaflet, which would otherwise have been resected during mid-posterior leaflet (P2) resection, to the middle of the underside aspect of the mid-anterior leaflet. This would keep the redundant tip of the anterior leaflet away from the LVOT and prevent SAM.

Nevertheless, even with the use of appropriate surgical techniques, post-repair SAM can develop, sometimes resulting in severe haemodynamic compromise. If this occurs, conservative measures are first undertaken with the aim of reversing the haemodynamic parameters that contribute to the development and worsening of SAM. Intravenous administration of fluids, cessation of positive inotropic agents, and therapy with beta-blockers are all aimed at reducing SAM and are, at times, sufficient to reverse this complication. If these measures do not succeed

in eliminating SAM, a surgical repair is contemplated.¹⁰ One possibility for such a repair is the use of the Alfieri edge-to-edge technique in which the free edges of anterior and posterior leaflets are sutured together.¹¹ This prevents the anterior movement of the anterior leaflet during systole and, thus, prevents the development of SAM. Studies have shown that the use of this technique can resolve post-repair SAM with good mid-term follow-up results.¹² Moreover, as this technique prevents the development of SAM, it has been used in advance to prevent the appearance of post-repair SAM in patients undergoing MV repair who were identified as being at increased risk for the development of this complication by a preoperative echocardiographic examination.¹²

We believe that the case presented highlights some important aspects of post-MV repair SAM. First, it presents a relatively uncommon combination of myxomatous MV disease and pre-repair SAM. In the case presented, the mild thickening of the LV walls in the absence of systemic

hypertension suggests that our patient may have also had an underlying hypertrophic cardiomyopathy, a disease which is commonly associated with MV SAM.

Second, it illustrates the main limitation of current methods used to assess the risk of SAM, i.e. the need to perform various measurements on a valve with leaflets that coapt. The patient described here was regarded as being at high risk for developing post-repair SAM only due to the finding of SAM on his first echocardiographic examination, performed before chordal rupture had occurred. If he had presented to us first only when symptoms occurred (following the development of a flail posterior leaflet), no SAM would have been recognized, since the development of a flail posterior leaflet eliminated it. In this scenario, assessment of the risk of SAM would have not been feasible since the valve leaflets did not coapt.

Third, it exemplifies the fact that, in some patients, post-repair SAM develops even after the use of surgical techniques aimed at preventing its development. In the case presented here, despite the use of a large annuloplasty ring and shortening of the posterior leaflet, post-repair SAM developed.

Finally, it represents yet another fine example of an elegant Alfieri edge-to-edge technique for the surgical treatment of post-MV repair SAM. This is a technically easy-to-perform technique that allows rapid elimination of post-repair SAM, with good long-term durability and no associated interference with the normal function of the MV. Interestingly, percutaneous edge-to-edge repair is currently being used for the treatment of MR.¹³ Perhaps in the future, LVOT obstruction caused by SAM can be treated percutaneously as well.

Conflict of interest

The authors declare that there are no conflicts of interest.

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