Original Article

Validation of a decision-making strategy for systolic anterior motion following mitral valve repair

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ABSTRACT

Low cardiac output syndrome and hypotension are dreadful consequences of systolic anterior motion (SAM) after a mitral valve (MV) repair. The management of SAM in the operating room remains controversial. We validate a recently suggested two-step management method and classification of this complication. This was a teaching hospital-based observational study. We validated a novel two-step conservative management method, consisting in intravascular volume expansion and discontinuation of inotropic drugs (step 1), and increasing the afterload by ascending aorta manual compression while administering esmolol e.v. (step 2). We also validate a novel classification of SAM: easy-to-revert (responding to step 1), difficult-to-revert (responding to step 2), or persistent. Fifty patients had an easy-to-revert while 26 had a difficult-to-revert SAM; 4 patients had a persistent condition (promptly diagnosed through our decisional algorithm) and underwent an immediate second pump run to repeat the mitral repair surgery. We confirmed that SAM after a repair of a degenerative MV is common and validated a simple two-step conservative management method that allows to clearly identify those few patients who require immediate surgical revision.

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INTRODUCTION

Systolic anterior motion (SAM) after a mitral valve (MV) repair can be limited to minor chordal protrusion without left ventricular outflow tract (LVOT) obstruction or be severe with LVOT obstruction. Clinical presentations vary from unaltered hemodynamic to life-threatening low cardiac output syndrome and hypotension. A simple two-step conservative management (expanding intravascular volume and discontinuing any inotropic drug as a first step and, most importantly, increasing the afterload through manual compression of the ascending aorta while administering an intravenous bolus of esmolol as a second step) method resolved the SAM in most patients in a recent case series[1] and allowed to identify those few patients who needed surgical revision. The main problem that surgeons and anesthesiologists face in the theatre is how to differentiate between transient (hemodynamic) and permanent (anatomic) SAM. While "hemodynamic SAM" can be solved by reducing the tachycardia, the "anatomic SAM" needs surgical repair. Unfortunately, in the theatre there is no time to wait for a spontaneous recover of the hemodynamic status and decisions should be taken promptly (within seconds or minutes).

We present the perioperative management of 574 consecutive patients undergoing MV repair for degenerative mitral regurgitation and the medical or surgical resolution of their SAM validating the efficacy of the recently suggested[1] two-step management method and classification of this complication that allows a uniform management of SAM and abolish the need for delayed reoperation (revision or valve replacement).

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MATERIALS AND METHODS

After the ethical committee approval and patients' written consent, we collected data of 574 consecutive patients (mean age 55 ± 15 years, 49% females) who underwent a MV repair for degenerative disease (myxomatous valve, leaflet prolapse, or flail) at our hospital over a 2-year period. Patients with functional MV regurgitation, rheumatic heart disease, or hypertrophic obstructive cardiomyopathy were not included. A great variety of anatomical lesions and clinical conditions were present, with surgical indications being always adherent to the current guidelines.[1,2] A wide spectrum of left ventricle end-diastolic diameter (mean ± standard deviation, 57 ± 9 mm), left atrium size (49 ± 6 mm), ejection fraction (56 ± 10%), and New York Heart Association (NYHA) class was included in this study. The majority of the patients (59%) were classified as NYHA class I or II, whereas 35% were in NYHA class III. Atrial fibrillation was present in a minority of patients (20%). Patients who submitted to tricuspid annuloplasty and concomitant Coronary artery bypass graft (CABG) were enrolled. All patients had severe MR according to a preoperative quantitative echocardiographic examination.[1,2]

The operation was carried out either through midline sternotomy or with a minimally invasive approach using cardiopulmonary bypass (CPB), mild hypothermia, and cardioplegic arrest of the heart by four surgeons.

The valve was approached through the left atrium. Techniques of MV repair were applied according to the anatomical lesions responsible for MR. For posterior leaflet prolapse, a quadrangular resection was used. When the height of the posterior leaflet was greater than 15 mm, a sliding plasty was invariably associated.[3] In anterior or bi-leaflet prolapse, the edge-to-edge (EE) technique, as described by our group,[4,5] has been used. Occasionally, artificial chordae were used alone or in combination with the above-mentioned techniques. The type of mitral repair which was carried out is given in Table 1.

A prosthetic ring was implanted in all cases. The appropriate ring size was chosen on the basis of the intertrigonal distance and the area of anterior leaflet, according to the well-established techniques of mitral repair in degenerative mitral disease.[6,7]

In all patients, intraoperative transesophageal echocardiography (TEE) was performed in the operative room immediately after the interruption of CPB by a trained anesthesiologist. SAM was defined as any portion of mitral valve leaflet or chordal structure protruding into the LVOT.[8] Significant colour Doppler aliasing in the LVOT was considered consistent with turbulent flow and obstruction. Velocities through the LVOT were measured with continuous wave Doppler. Concomitant MR was recorded. The degree of SAM was determined on the basis of the gradient across the LVOT and the severity of MR as follows: the degree of SAM was considered mild when LVOT obstruction was absent and mitral regurgitation was absent or negligible; moderate when LVOT maximum pressure gradient (ΔP max) ranged between 20 and 50 mmHg; and severe when LVOT ΔP max was >50 mmHg or greater.[9]

When some degree of SAM was diagnosed after CPB interruption, a well-defined management protocol was applied, including two consecutive steps (step 2 was applied if SAM was not solved by step 1) with the great vessels still cannulated and before heparin reversal in case a return to CPB became necessary.

Table 1: Types of surgical technique for MV repair performed on 574 patients with myxomatous disease, leaflet prolapse, or flail

Types of surgical techniques for MV repair	Number of patients	Number (percentage) of systolic anterior motion	Classification of systolic anterior motion: easy/difficult/persistent	Reoperation
QR*	135	23 (17.0)	17/5/1	1
QR + sliding†	267	39 (14.6)	22/16/1	1
Central E to E‡	84	10 (11.9)	5/4/1	1
Paracommissural E to E§	49	5 (10.2)	3/1/1	1
AC‖	24	2 (8.3)	2/0/0	0
Other procedures	15	1 (6.7)	1	0
Total	574	80 (13.9)	50/26/4	4

*QR: Quadrangular resection of the posterior mitral valve leaflet without the sliding technique, †QR + sliding: Quadrangular resection of the posterior mitral valve leaflet with the sliding technique, ‡Central E to E: Central edge-to-edge technique, §Paracommissural E to E: Paracommissural edge-to-edge technique, ‖AC: Anterior leaflet procedures with artificial chordae, Figures in parenthesis are in percentage

Step 1 was represented by established procedures as intravascular volume expansion and simultaneously discontinuing any inotropic drug.

Step 2 was represented by a maneuver intended to acutely increase the afterload (partial digital occlusion of the ascending aorta) and simultaneous administration of a bolus of esmolol, 1 mg/kg.[10]

The effect was invariably immediate and could be observed by echocardiography. The heart rate dropped significantly while arterial blood pressure significantly rose.

Volume expansion was gradually performed over a period of few minutes allowing for the adaptation of and monitoring pulmonary artery pressure.

The discontinuation of inotropic drugs was performed in those few patients who inappropriately started them because of SAM-induced hypotension before performing postoperative TEE examination.

The compression, after the inspection and palpation of the ascending aorta, acutely increased the afterload and it was maintained for a 30-s period. Simultaneously, esmolol was given at the dose of 1 mg/kg.

The goal of this conservative management in the operating room was to abolish SAM (absence of the LVOT obstruction and MR).

SAM was classified according to its reversibility as easy-to-revert, difficult-to-revert, and persistent.

SAM was defined as "easy-to-revert" when it disappeared only after intravascular volume expansion and/or discontinuination of any inotropic drug. SAM was defined as "difficult-to-revert" when it disappeared after increasing the afterload and administering a bolus of esmolol (1 mg/kg). It was defined as "persistent" if did not disappear after conservative management.

If there was little or no improvement (SAM remaining at least moderate: ΔP max in the LVOT ranging between 20 and 50 mmHg and/or mitral regurgitation graded mild-moderate) after conservative management, reoperation was immediately performed since the great vessels were still cannulated and heparin reversal was not yet performed.

RESULTS

SAM after MV repair occurred in 80 out of 574 patients (13.9%; Table 1). Overall, the incidence of SAM was not statistically different according to surgical techniques of MV repair ($P = 0.07$).

Four patients with persistent SAM not responding to conservative management required the revision of repair or valve replacement. Surgical reinterventions were performed immediately with a second pump run and a successful mitral repair in all patients. These patients had no SAM after reintervention.

Twenty-six patients had a difficult-to-revert SAM which required the administration of a bolus of esmolol, 1 mg/kg, and an afterload increase by means of manual compression of the ascending aorta.

Fifty patients had an easy-to-revert SAM which disappeared with the discontinuation of inotropic drugs and intravascular volume expansion without the need for an afterload increase or beta-blockers.

No patient had SAM detected at the echocardiographic control performed by a cardiologist before discharge from the hospital.

DISCUSSION

After validating this simple management and diagnostic protocol, we think that the management of SAM after a MV repair for degenerative disease (myxomatous valve, leaflet prolapse, or flail) could be now performed in a standardized manner.[1]

SAM after a MV repair for degenerative disease (myxomatous valve, leaflet prolapse, or flail) is common: 13.9% in this case series, 11% at Cleveland Clinic,[2] and 9.8% at San Raffaele Hospital.[1] In most cases, a two-step conservative management method (expanding intravascular volume and discontinuing any inotropic drug as a first step and, most importantly, increasing the afterload through manual compression of the ascending aorta while administering an intravenous bolus of esmolol as a second step) resolved the SAM.

The novelty and the importance of this case series that validated the previously published one[1] is that while in the first experience we had delayed surgical operation in two out the five patients who were operated, in this

validation case series we decided to immediately treat all patients with persistent SAM without increasing the risk of organ damage due to subtle low cardiac output syndrome and organ damage in the case of delayed surgical operation.

Though SAM is a well-known complication after MV repair, there were not clear guidelines for its treatment, and different attitudes in management have been advocated, ranging from prolonged medical treatment[9,11] to immediate surgical reintervention.[12-14]

This two-step protocol aims at classifying SAM on the basis of severity and reversibility, and has implications for further decision making regarding treatment. The acute increase in the afterload, obtained through manual compression of the ascending aorta, is a very useful maneuver which was described once before.[1] The alternative, afterload increase via vasoconstrictors, could require time, may be unsuccessful, and may cause drug-related side effects.

Our study, focusing on patients undergoing a MV repair who are at the highest risk for postoperative SAM (myxomatous disease, leaflet prolapse, or flail), can only be compared to Brown *et al.*'s[9] and to Crescenzi *et al.*'s[1] studies.

Brown *et al.*[9] reported an incidence of SAM of 11% (with respect to our 13.9%). A retrospective chart review of 1589 patients undergoing MV repair at Mayo Clinic over a 10-year period identified 174 cases of early intraoperative SAM who were conservatively managed, and 4 cases (2.3% of patients with SAM compared to our 0.8%) requiring revision of repair or valve replacement: 2 of their patients underwent immediate operation while the other 2 were operated upon within the first 4 postoperative weeks for SAM-related complications, 1 for pulmonary edema and the other one for SAM coupled with a LVOT obstruction gradient of 100 mmHg and hemolytic anemia. Notably, 62 of 174 (36%) of their patients had SAM at hospital discharge. This discordant result could be partially due to the fact that we aimed at obtaining complete reversibility of SAM before hospital discharge,[15] and in part to the different surgical techniques (quadrangular resection of the posterior MV leaflet with sliding plasty, and central —EE technique) we performed in many patients.

Crescenzi *et al.*[1] presented data of 608 consecutive patients who underwent a MV repair for degenerative MV disease and described, for the first time, the two-step conservative management method, consisting in intravascular volume expansion and discontinuation of the inotropic drug (step 1), and increasing the afterload by ascending aorta manual compression while administering beta-blockers (step 2), introducing the novel classification of SAM: easy-to-revert (responding to step 1), difficult-to-revert (responding to step 2), or persistent. The overall incidence of SAM was 9.8% (60/608): 40 patients had an easy-to-revert while 15 had a difficult-to-revert SAM; 5 patients had a persistent condition and underwent surgery within 48 h. Surgical reinterventions were performed either immediately (three patients with a poor hemodynamic status) or within 48 h (two patients with a progressive deterioration of clinical conditions).

The slightly higher incidence of mild SAM (8.7% vs. 6.6%) in our recent experience when compared to our previous experience[1] could be explained by an higher attention to the diagnosis of SAM in the study period.

The slightly higher incidence of difficult-to-revert SAM (4.5% vs. 2.5%) in our recent experience when compared to our previous experience[1] could be easily explained by an earlier use of the second step in the daily clinical practice: while administering i.v. fluids and interrupting inotropic drugs, we often started the bolus administration of esmolol and the manual compression of the aorta. The results were good with an immediate diagnosis of four cases of persistent SAM that immediately had a second pump run.

SAM complicating a MV repair was first described in 1977,[16] and may cause serious postoperative complications.

There have been numerous studies and hypotheses to explain the SAM of the MV causing left ventricular outflow obstruction and transient MR after a MV repair procedure. Most experts believe that high blood flow velocities in the LVOT produce a Venturi effect that in part may lead to the anterior motion of the MV leaflets toward the septum. This creates incomplete coaptation of the MV leaflets with resultant MR, which is most commonly eccentric and posteriorly directed. However, it is unlikely that the Venturi effect is the sole cause of the anterior motion of the MV.

The long-term implications of medical therapy for SAM are unknown. It is likely that patients with

postoperative SAM subsequently have recurring episodes of catecholamine surges as well as intermittent hypovolemia. During these episodes, these patients have demonstrated the anatomic architecture to develop SAM and significant MR. Repeated episodes of this may be the underlying pathology that results in a worse outcome in these patients as recently suggested by a recent observational study.[17] This study was the first to suggest that patients with less than moderate MR due to SAM in the immediate postoperative period seem to do well in the long term while patients with semiquantitatively assessed moderate-to-severe MR and SAM have an increased incidence of recurrent, significant MR.

This was not the case with our case series since we aggressively treated all SAM and all mitral residual regurgitation cases.

Other groups recently suggested a completely different strategy to diagnose the severity of postoperative SAM. A provocative test was performed with the great vessels still cannulated and with the goal of determining if SAM would be tolerated should the patient become hypovolemic, tachycardic, and vasodilated postoperatively.[18] For 15 min, ventricular pacing at 120 beats/min was instituted, and nitroglycerin, 200 μg/min, and dopamine, 7 μg/kg/min, were administered. The authors wanted to know how well the patient would tolerate SAM under aggravating conditions. The patient presented a "grayzone" situation in which hemodynamics were favorable after CPB, but the presence of SAM was clear. The authors strongly recommend that such patients receive chronic beta-blocker therapy and be advised to remain well-hydrated and to report deteriorating exercise tolerance to their cardiologist immediately.

Other authors[19] suggested that since SAM can occur later in the postoperative period, an isoproterenol challenge may be useful to unmask this situation.

We think that endogenous perioperative cathecolaminergic surge together and hypovolemia caused by vasodilatation from most anesthetic drugs together with hypotension commonly seen immediately after CPB represent a natural stress test.

In our opinion, esmolol is the most suitable drug to reduce bradycardia in this setting. Nonetheless, we acknowledge that other authors have suggested to use other drugs such as the class Ia, antiarrhythmic drug cibenzoline[20] or the ultra-short-acting and beta 1-selective adrenoceptor antagonist landiolol.[21]

Limitations of the study

A competent mitral valve without SAM is clearly the goal and this strategy that provides the surgical team a quick and simple maneuver with good short-term results. Even if no patient had SAM at hospital discharge, further follow-up is necessary to confirm the durability of this new technique.

CONCLUSION

SAM after mitral repair responded to a well-defined standardized and validated management protocol consisting of a two-step conservative maneuver that permitted to identify patients needing immediate surgical revision.

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