Post-mitral valve repair systolic anterior motion produced by non-obstructive septal bulge

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Abstract

Objective: Systolic anterior motion (SAM) may rarely occur after mitral valve reconstruction due to different anatomic factors. Several techniques have been described to reduce the incidence of post-repair SAM, e.g. leaflet sliding plasty. However, SAM can still occur after these special procedures. We reviewed data of patients developing SAM with significant mitral regurgitation due to non-obstructive septal bulge.

Methods: During a 2-year period mitral valve repair was performed in 358 patients. Five of 358 (1.4%) patients with a mean age of 52 ± 10.5 years developed post-repair SAM with severe mitral insufficiency due to non-obstructive septal bulge. Data of these patients were analyzed retrospectively and controlled after a mean follow-up of 18 ± 2.7 months.

Results: Preoperative echocardiography showed end-diastolic septum diameter of 7, 10, 10, 11 and 15 mm. The ratio between end-diastolic septum diameter and free wall diameter was 1 in four patients and 1.25 in one patient. There was no left ventricular outflow tract obstruction (LVOT). Intraoperative data revealed large myxomatous anterior (four patients) and posterior (three patients) leaflets. Quadrangular resection of posterior leaflet was carried out in four patients and sliding plasty in one patient. Cause for post-repair mitral regurgitation was a non-obstructive septal bulge. During a second pump run septal bulge was resected. Mean aortic cross-clamp time and cardiopulmonary bypass time for this procedure was 15 ± 1.4 and 28 ± 3.1 min, respectively. Mitral regurgitation disappeared in all patients immediately after this procedure. The grade of mitral regurgitation at follow-up was 0–1 in all patients. One patient had subaortic gradient of 36 mmHg.

Conclusions: If mitral regurgitation occurs after primary successful mitral repair, septum bulge should always be considered as the primary cause for SAM even there is no preoperative gradient in LVOT. Before performing time-consuming corrective operations to relieve SAM, a septum resection should be carried out during a short second pump run.

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1. Introduction

Systolic anterior motion (SAM) with left ventricular outflow tract obstruction and mitral valve regurgitation may occur after mitral valve reconstruction [1]. The major anatomic factors incriminated in the genesis of post-repair SAM include degenerative mitral valve insufficiency with excess leaflet tissue, nondilated left ventricular cavity, narrow mitro-aortic angle and anterior displaced mitral coaptation line [2,3]. Others found that the relatively greater contribution of the posterior leaflet to the coaptation of the mitral valve prior to repair is responsible for SAM after mitral valve reconstruction [4]. Frequency of SAM after mitral valve repair varies between 1 and 16% [2,3,5–7]. Several techniques are believed to reduce the incidence of SAM including triangular anterior leaflet resection [6] and sliding plasty of the posterior leaflet [2,8]. However, SAM can still occur after these special procedures [1,5,9–11]. Several surgical options have been proposed in these cases such as the ‘Pomeroy procedure’ [12], transaortic resection of redundant leaflet tissue [13], valve replacement [14,15] and complete or partial removal of the annuloplasty ring.
Some authors think that this problem can be resolved by medical treatment only [5,6,16]. In general, asymmetric septal hypertrophy (ASH) also contributes to the development of SAM [17]. Thus, one should suggest that surgical resection of hypertrophied septum could probably eliminate post-repair mitral insufficiency, even in cases without subvalvular gradients. Indeed, Perier et al. was able to relieve SAM by left ventricular myectomy after mitral valve reconstruction [8].

This study was undertaken to analyze the patients developing SAM with mitral regurgitation after primary successful mitral valve repair who were treated by resection of a non-obstructive septal bulge during a second pump run.

2. Patients and methods

Data of all patients developing SAM after mitral valve reconstruction were evaluated retrospectively. Between March 2001 and April 2003, a total of 358 patients had mitral valve repair. Of these, five patients (1.4%) with a mean age of 52 ± 10.5 years developed SAM with mitral insufficiency after primary successful reconstruction. Mean ejection fraction was 64 ± 4.5% (56–68%) and mean left ventricular end-diastolic diameter indexed to the body area was 31 ± 3.4 mm/m² (27–36). Intraoperative echocardiography revealed bulged septum without subaortic gradient which was believed to be the cause of SAM.

2.1. Surgical technique

2.1.1. Mitral valve reconstruction

After induction of anesthesia a transesophageal echocardiography (TEE) probe is positioned routinely in all patients undergoing mitral valve repair. The heart is exposed through a median sternotomy. After full heparinization the aorta and both venae cavae are cannulated. The patient is put on normothermic cardiopulmonary bypass (CPB). The aorta is cross-clamped and ante- and retrograde cardioplegia (Buckberg or Calafiore solution) is administered. Access to the mitral valve is performed either via left or right atrium depending on whether the tricuspid valve needs concomitant reconstruction. The mitral valve is examined and reconstruction is performed mostly in accordance to the Carpentier technique [18]. Additionally, the annulus is reshaped with a flexible partial posterior ring (Annuloflex, Sulzer Carbomedics Inc, TX, USA; SJM Tailor, St Jude Medical, MN, USA). The intertrigonal distance is measured and in cases of larger than normal leaflets the implanted ring is oversized by one size. After reconstruction the patient is rewarmed and weaned from CPB. The surgical result is controlled by TEE. Mean cross-clamp time and CPB time for mitral valve repair was 49 ± 2.9 and 73 ± 9.5 min, respectively.

2.1.2. Septal myectomy (Morrow)

Post-repair SAM with recurrent mitral regurgitation is diagnosed by TEE. If asymmetric septal hypertrophy is diagnosed a second pump run is initiated even there is no subaortic gradient (Fig. 1a,b). The aorta is cross-clamped and cardioplegia is infused again. Through an oblique aortotomy the hypertrophied ventricular septum is resected according to the technique described by Morrow [19]. Rewarming is initiated, aortotomy closed and CPB discontinued. Again, the surgical result is controlled by TEE (Fig. 2a,b). Cross-clamp time and CPB time for transaortic myectomy was 15 ± 4.4 and 28 ± 3.1 min, respectively.

Fig. 1. (a) Intraoperative transesophageal echocardiography showing anterior leaflet (AL) attached to the septal bulge (SB) causing anterior displacement of AL towards left ventricular outflow tract (LVOT). (b) Schematic representation of (a) showing anterior leaflet (AL) attaching to the septal bulge (SB) due to anterior displacement. This mechanism causes a gap between AL and posterior leaflet (PL) producing mitral regurgitation.
2.2. Follow-up

Clinical examination and transthoracic echocardiography was performed in all patients who developed post-repair SAM treated by septal myectomy after a mean follow-up of 18 ± 2.7 months.

3. Results

Preoperative echocardiographic data of the five patients with post-repair SAM are depicted in Table 1. Septum diameter was within normal range in all patients as was the ratio between septum diameter and free left ventricular wall thickness. There was no special remark outlining septal hypertrophy in the echocardiography report. Furthermore, no gradient in left ventricular outflow tract was measured.

Intraoperative findings are shown in Table 2. In four patients there was a prolapse of the posterior leaflet (Carpentier class type 2) and in one patient there was a restricted posterior leaflet (type 3). In four patients tissue quality was myxomatous and both anterior and posterior leaflets were larger than normal. Due to this excessive tissue it was necessary to partially resect posterior leaflet in four patients. A sliding leaflet plasty was performed in one patient.

After the initial mitral valve reconstruction the patients were weaned from CPB without catecholamines and sufficient ventricular filling as shown on TEE. Mean arterial pressure was 65 ± 4.9 mmHg and mean PAP 29 ± 1.7 mmHg after the first pump run. After resection of septal bulge mean pressure was 77 ± 3.7 mmHg and mean PAP 25 ± 2.5 mmHg. Catecholamines were not administered also after the second pump run.

Data of post-pump echocardiography are shown in Table 3. A subaortic bulging of the septum was observed in all patients. During systole anterior leaflet came into contact with the bulged septum causing mitral regurgitation (Fig. 1b). Mitral regurgitation was eccentric in two cases and central in three cases. In one case regurgitation was holosystolic and in four cases it occurred during the second part of systole. Echocardiographic data from follow-up examinations are depicted in Table 4. Minimal central mitral regurgitation was observed in three patients. In one patient there was a subaortic peak gradient of 36 mm. In the same patient septum thickness and free left ventricular wall thickness increased by 8 and 7 mm, respectively, compared to preoperative values. Preoperative and postoperative values were comparable in the four remaining patients.

![Image](https://example.com/figure2a.png)

**Fig. 2.** (a) Intraoperative transesophageal echocardiography showing anterior leaflet (AL) which does not attach to the septal bulge (SB) any longer since this has been resected. There is normal coaptation of AL and posterior leaflet (PL). (b) Schematic representation of (a). The septal bulge (SB) has been resected and there is normal coaptation of anterior (AL) and posterior (PL) leaflet. Left ventricular outflow tract (LVOT) is widely open after myectomy.

### Table 1

Preoperative echocardiography in patients with post-repair SAM

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>SD (mm)</th>
<th>FWT (mm)</th>
<th>Ratio SD/FWT</th>
<th>MVI grade</th>
<th>LVOT pre (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15</td>
<td>15</td>
<td>1</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
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<td>1</td>
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<td>5</td>
<td>10</td>
<td>8</td>
<td>1.25</td>
<td>4</td>
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</tbody>
</table>

SD, end-diastolic diameter of septum; FWT, free wall thickness; MVI, grade of mitral valve insufficiency (1–4); LVOT pre, gradient in left ventricular outflow tract.
4. Discussion

In our series post-repair SAM with significant mitral valve insufficiency occurred in 1.4%. According to the literature the incidence of postoperative SAM with mitral regurgitation varies between 1 and 16% [2,3,5–7]. Several causes for this phenomenon have been recognized. Freeman et al. found that a hyperdynamic state caused by postcardiopulmonary bypass hypovolemia in combination with catecholamine infusion is responsible for SAM after mitral valve repair in most patients [20]. However, ventricular filling was sufficient in our patients, as demonstrated by intraoperative echocardiography, and catecholamines were not administered. Nevertheless, our patients developed severe mitral regurgitation due to SAM. Other causes of post-repair mitral valve insufficiency are residual prolapse, persistent leaflet restriction or excessive anterior leaflet plication [20]. Furthermore, Jебара et al. found that a predictor for postoperative SAM was a discrepancy between ventricular cavity and the amount of leaflet tissue [2]. As shown in Table 2, four of our patients had posterior leaflet prolapse and tissue quality was myxomatous in all but one patient. Indeed, in all patients there was excessive leaflet tissue. Residual prolapse or persistent leaflet restriction was not found intraoperatively. Maslow et al. compared patients who developed post-operative SAM with patients who did not. He found that patients with post-repair SAM had larger anterior and posterior leaflet lengths, providing increased leaflet tissue available for SAM [4]. Furthermore, he found that patients who developed post-repair SAM showed a smaller distance between septum and leaflet coaptation point. In our patients intraoperative echocardiography revealed a discrete septum bulge producing an attachment of the anterior leaflet to the septum (Fig. 1a,b).

Coworkers of Carpentier found that the incidence of post-repair SAM with LVOTO was reduced by combining leaflet resection with a sliding plasty [2,8]. In one of our five patients a classical sliding plasty was performed but this patient also developed SAM. This observation was made by others as well [10].

There are several options to treat postoperative SAM. Discontinuation of catecholamines and volume loading are the initial steps for obvious reasons [21]. Grossi et al. found that SAM after Carpentier mitral reconstruction should be managed medically in the majority of patients [5]. We were reluctant in believing that SAM would disappear in our patients by medical treatment only.

Several authors observed SAM after the Carpentier procedure [9,11,20]. These authors suggested replacing the valve during a second pump run which is, in our opinion, too radical a procedure. Others think that a rigid ring itself may produce LVOTO [22]. Indeed, there are case reports describing relief of SAM after removal of the annuloplasty ring [3,9]. It is well known that small rigid rings may increase the risk of SAM since they may displace the anterior leaflet coaptation line. This is one reason why we use flexible and only partial rings in order to minimize the risk of SAM. Ring diameter varied between 28 and 36 mm.

### Table 2

Intraoperative data

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>CFC</th>
<th>Chorda rupture</th>
<th>TQAL</th>
<th>TQPL</th>
<th>Surgical technique</th>
<th>RD (mm)</th>
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<td>1</td>
<td>Type 2 P₂₋₁</td>
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<td>Prolapse</td>
<td>QR + MA</td>
<td>33</td>
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<td>2</td>
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<td>Yes</td>
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<td>Prolapse</td>
<td>QR + CABG + MA</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>Type 3</td>
<td>No</td>
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<td>PMS + CT + MA</td>
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<td>4</td>
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<td>QR + SP + MA</td>
<td>30</td>
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<tr>
<td>5</td>
<td>Type 2 P₂</td>
<td>Yes</td>
<td>Myxomatous, thickened</td>
<td>Myxomatous, thickened</td>
<td>QR + MA</td>
<td>36</td>
</tr>
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</table>

CFC, Carpentier functional class; TQAL, tissue quality of anterior leaflet; TQPL, tissue quality posterior leaflet; RD, ring diameter; QR, quadrangular resection; MA, mitral annuloplasty; CABG, coronary artery bypass grafting; PMS, papillary muscle splitting; CT, commissurotomy; SP, sliding plasty.

### Table 3

Post-pump echocardiography

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>SB</th>
<th>MVI 1</th>
<th>MVI 2</th>
<th>LVOT post (mmHg)</th>
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<td>1</td>
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<td>Yes</td>
<td>2–3</td>
<td>0</td>
<td>0</td>
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<tr>
<td>3</td>
<td>Yes</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>3–4</td>
<td>0–1</td>
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<tr>
<td>5</td>
<td>Yes</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

LVOT post, gradient after mitral valve repair and myectomy; MVI 1, mitral insufficiency grade after first pump; MVI 2, mitral insufficiency grade after second pump; SB, subaortic septal bulging before myectomy.

### Table 4

Echocardiographic data at follow up

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>SD (mm)</th>
<th>FWT (nm)</th>
<th>Ratio SD/FWD</th>
<th>MVI grade</th>
<th>LVOT (mmHg)</th>
<th>PV (cm/s)</th>
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<tr>
<td>1</td>
<td>13</td>
<td>14</td>
<td>0.93</td>
<td>0–1</td>
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<td>3</td>
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<td>10</td>
<td>1.1</td>
<td>0–1</td>
<td>No</td>
<td>85</td>
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<td>4</td>
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<td>11</td>
<td>1</td>
<td>0–1</td>
<td>No</td>
<td>90</td>
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<tr>
<td>5</td>
<td>11</td>
<td>11</td>
<td>1</td>
<td>0–1</td>
<td>No</td>
<td>NA</td>
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</tbody>
</table>

LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; SD, septum diameter; FWT, free wall thickness; MVI, grade of mitral valve insufficiency (1–4); LVOT, left ventricular outflow tract; PV, peak velocity; NA, not available due to bad echo quality.
as shown in Table 2, and we do not believe that SAM was a result of using a ring. There is even a case report describing SAM after mitral valve repair without a ring [15].

Whenever SAM occurs after successful mitral valve repair one should evaluate the shape of the septum. If there is a bulge reducing the distance between the leaflet coaptation point and the septum, a second pump run should be initiated even if the anterior and posterior leaflets are larger than normal. This operation can be performed very quickly with clamp times of around 15 min. If removal of the bulge is not successful, other more time-consuming techniques such as leaflet plication, triangular anterior leaflet resection or the Pomeroy procedure should be performed [6,12,20]. Mitral valve replacement should not be necessary in any case.

References