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CASE REPORT: CLINICAL CASE

SAM and Severe Mitral Regurgitation Post-Acute Type A Aortic Dissection Surgery Treated With MitraClip

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ABSTRACT

Post-operative systolic anterior motion associated with mitral regurgitation can be a challenging combination. We present the case of a 64-year-old male patient managed by MitraClip (Abbott Laboratories, Abbott Park, Illinois) implantation for systolic anterior motion and severe mitral regurgitation in the early post-operative period after aortic dissection surgery. This is the first description of MitraClip use post-aortic dissection. (Level of Difficulty: Intermediate.) (J Am Coll Cardiol Case Rep 2020;2:1582-6) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

lthough mitral valve (MV) systolic anterior motion (SAM) was initially described in patients with hypertrophic obstructive

LEARNING OBJECTIVES

- Precise and prompt recognition of this potentially lethal association is vital to provide adequate clinical and interventional management.
- MitraClip can be an effective and lifesaving alternative to manage SAM associated with severe MR in patients with high surgical risk or a contraindication to conventional cardiac surgery.

cardiomyopathy, it can also be present in patients with complex dynamic left ventricular anatomy. This "dynamic SAM" can potentially occur after cardiac surgery and significantly affect perioperative management and the patient's prognosis (1).

The MitraClip system (Abbott Laboratories, Abbott Park, Illinois) has been used to manage new onset SAM after MV repair (2) or aortic valve (AV) interventions (3). Clinical hemodynamic outcomes suggest that it could be a feasible and safe alternative in these settings (4).

We describe a rare case of SAM associated with severe mitral regurgitation (MR) complicating the early post-operative period after treatment of type A

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acute aortic dissection (AAD), which was successfully managed with a MitraClip intervention.

HISTORY OF PRESENTATION

A 64-year-old man was admitted to the emergency department with a sudden onset of retrosternal discomfort radiating to the back and superior abdomen associated with weakness in the right arm. On emergency department arrival, vital signs were as follows: noninvasive blood pressure 134/100 mm Hg, heart rate 86 beats/min, oxygen saturation level 98%, Glasgow Coma Scale score 15. The right lower limb was colder than the left, and he had no palpable pulse.

PAST MEDICAL HISTORY

His previous medical history included hypertension, ascending aorta ectasia (46-mm diameter, 2-mm progression in the last 2 years), persistent atrial fibrillation treated with oral anticoagulant therapy (rivaroxaban 20 mg/day); active smoking (70 packyears), and alcohol abuse. A previous transthoracic echocardiogram (performed 10 months earlier) showed mild to moderate AV regurgitation, mild MR, and posterior leaflet prolapse.

INVESTIGATIONS

The chest radiograph showed mediastinal widening, the electrocardiogram was normal, and chest

computed tomography revealed a Stanford type A AAD involving the entire ascending, descending, and abdominal aorta, with no pericardial or pleural effusion.

A transesophageal echocardiogram (TEE) showed a tricuspid AV with moderate regurgitation, moderate MR, and nonsignificant SAM (Figures 1A and 1B, Video 1). Left ventricular ejection fraction was 60%.

MANAGEMENT

Emergency aortic surgery was indicated. Cardiopulmonary bypass was established through the right subclavian artery and right atrium. Under antegrade cerebral perfusion and moderate hypothermia, the ascending aorta was inspected. An AV resuspension, with ascending aorta and hemiarch replacement, was performed using a 28-mm Dacron (Abbott Vascular, Santa Clara, California) graft and a Teflon (Chemours, Wilmington, Delaware) felt sandwich technique. Cardiopulmonary bypass and aortic cross-clamping times were 216 and 117 min, respectively. Because of a significant coagulation disturbance and excessive bleeding, a strategy of delayed sternal closure was adopted. At the end of the procedure, the SAM was classified as moderate (5) (no left ventricular outflow tract [LVOT] obstruction, moderate MR) and easily reversible (reversible with intravascular volume expansion).

ABBREVIATIONS AND ACRONYMS



echocardiogram







Images show (A) severe mitral regurgitation with an eccentric regurgitant jet directed anteriorly to the atrial roof, combined with (B) systolic anterior motion. Abbreviations as in Figure 1.

During the first post-operative days, hemodynamic stabilization was not obtained, and high doses of inotropic and vasopressor agents were required. TEE was repeated and revealed severe MR, an anteriorly directed eccentric jet, P_1 to P_2 leaflet prolapse, and severe and persistent SAM (no LVOT obstruction, severe MR, not reversible with intravascular volume expansion and increase in the afterload) (Figures 2A and 2B, Videos 2 and 3).

The case was discussed among colleagues, and the patient was judged to be at too high risk for immediate surgical reintervention. Hence a percutaneous MV approach was planned.

On the seventh post-operative day, MitraClip implantation was performed using 2 XTR clips. Under TEE guidance, the first clip was oriented to the A₂-P₂ segment. Grasping was achieved using X-plane visualization, and the TEE confirmed a broad tissue bridge between A₂ and P₂. The second clip was implanted in a slightly more lateral position. By the end of the procedure, there was a mild central residual MR, 2 mm Hg mean gradient, and no residual SAM (Figures 3A and 3B, Videos 4A and 4B). Because of the patient's baseline hemodynamic condition and right ventricular dysfunction, an 8-mm Amplatzer atrial septal defect closure device (Abbott Laboratories) was used to occlude the iatrogenic atrial septal defect. Hemodynamic parameters, such as invasive blood pressure (before, 94/52 mm Hg; after, 117/55 mm Hg) and mean left atrial pressure (before, 20 mm Hg; after, 10 mm Hg) improved immediately.

Following MitraClip implantation, the patient showed clinical improvement. He changed from a status of complete hemodynamic instability, under ventilatory and inotropic support, to a stable condition. He was weaned from mechanical ventilation and inotropic support, and his chest was definitively closed 2 days after the MitraClip was implanted.

DISCUSSION

SAM of the MV, combined with significant MR, with or without significant LVOT obstruction, can be a lifethreatening condition requiring precise and prompt diagnosis and management, especially if it complicates the early post-operative setting.

Following aortic stenosis interventions, SAM is an often unrecognized cause of hemodynamic instability (3). One of the proposed pathophysiological explanations is that, by relieving the LVOT obstruction imposed by the aortic stenosis, the LVOT pressure drops, thereby reducing the LVOT cross-sectional area resulting from high-velocity flow (Bernoulli theorem). This "low-pressure zone" would affect the MV apparatus directly by "pulling" the anterior leaflet toward the LVOT (3). After aortic regurgitation interventions, although possible, SAM is rarer once the dilated left ventricle tends to widen during systole, thus preventing anterior leaflet-LVOT apposition (6).

We report the case of a rare and potentially lethal combination of SAM and severe MR in the early postoperative phase of a surgical procedure for AAD. Post-





operative hypovolemia and hypotension, combined with reduced systemic vascular resistance and persistent atrial fibrillation, may have led to left ventricular underfilling. This condition, aggravated by the presence of a hyperdynamic state resulting from inotropic agent and excessive sympathetic activation, has been identified as a potential SAMaggravating mechanism (6,7).

Regarding the MR mechanism, it is noteworthy that, although MR mediated by SAM is traditionally characterized by a posteriorly directed jet on Doppler echocardiography, our patient had a predominantly anteriorly directed eccentric jet, which suggests the presence of an intrinsic MV disease. This statement is strengthened by the observation that, in patients with SAM and central or anterior MR jets, a significantly elongated posterior leaflet is usually present (8). A posterior leaflet basal portion bulging beyond the anterior leaflet may reduce the amount of posterior leaflet area effectively available to follow and coapt with the anterior leaflet toward the septum, thus explaining why MR is usually more significant when prolapse is present (9). Nonetheless, a more careful analysis of the available echocardiographic images could suggest the presence of chordal SAM associated with MV prolapse and increased left ventricular contractility, rather than true SAM involving the anterior leaflet body.

When MR worsens following aorta surgery, investigators have proposed that because of the proximity between the MV and the AV, surgical intervention in the latter can damage the former or alter the normal dynamics of the aortomitral curtain (10). In our patient, the AV resuspension technique could have displaced the MV leaflets towards the aorta, thereby reducing the coaptation length and increasing the posterior leaflet prolapse. This surgically provoked MV anterior translocation may have aggravated the pre-existing MR and impaired clinical recovery.

In the present case, transcatheter MV repair was considered the best option because of the patient's hemodynamic instability and absolute refractoriness to medical management, both of which created a condition of "too high risk" for open cardiac surgery reintervention. Despite the successful outcome obtained in this patient, it is important to keep in mind that conservative medical therapy is usually enough to stabilize patients with post-operative SAM. A 2-step approach, consisting of intravascular volume expansion simultaneously with any inotropic drug discontinuation (first step), followed by maneuvers to increase the afterload simultaneously and in the short term with a bolus administration of esmolol (1 mg/kg) (second step, applied when the first step fails to elicit a response), has been proposed. Following these steps, invasive management is indicated when the SAM does not disappear after conservative management (persistent SAM) (5).

This case shows that, in selected patients, Mitra-Clip use can be a feasible alternative to address refractory MR and SAM, with excellent safety profile and hemodynamic outcomes.

FOLLOW-UP

After chest closure, the patient had no major complications related to the MitraClip procedure. At 1and 3-month follow-up, he had no cardiovascular symptoms. The 1-month echocardiographic image revealed mild AV regurgitation, mild MR, and 3 mm Hg mean MV gradient.

CONCLUSIONS

To the best of our knowledge, this is the first description of MitraClip implantation in

the immediate post-operative period in a patient with type A AAD surgery performed to address SAM and severe MR. In this setting, less invasive percutaneous management can be considered as a first-line option instead of higher-risk redo surgery.

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KEY WORDS aorta, dissection, mitral valve, post-operative

APPENDIX For supplemental videos, please see the online version of this paper.