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VALVULAR HEART DISEASE

CASE REPORT: CLINICAL CASE

Unmasking Severe Systolic Anterior Motion With Left Ventricular Outflow Tract Obstruction Following Mitral Valve Repair

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ABSTRACT

A 38-year-old male with a history of myxomatous mitral valve disease post-repair presented with recurrent dyspnea during exertion. Initial evaluation showed mild systolic anterior motion and mitral regurgitation, but medical management was unsuccessful. The patient underwent reoperation; intraoperative transesophageal echocardiogram with provocation unmasked severe systolic anterior motion and torrential mitral regurgitation. (J Am Coll Cardiol Case Rep 2024;29:102258) © 2024 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/).

HISTORY OF PRESENTATION

A 38-year-old male presented for recurring dyspnea and chest discomfort on exertion, which had begun shortly following mitral valve repair surgery. He additionally reported palpitations and chest pain and lightheadedness on exertion and heart rate increase. Symptoms were particularly noticeable during activities such as firefighting (including moderate activity and light smoke inhalation), sports, and climbing stairs.

LEARNING OBJECTIVES

- To identify potential leaflet abnormalities in patients undergoing mitral valve repair using echocardiography.
- To emphasize the diagnostic role of transesophageal echocardiography with chemical provocation.
- To prevent postoperative complications by tailoring surgical technique.

PAST MEDICAL HISTORY

The patient had a history of myxomatous mitral valve disease for which he underwent mitral valve repair and patent foramen ovale closure 5 years prior, complicated by postsurgical pericarditis. Additional medical history was significant for obesity and obstructive sleep apnea on continuous positive airway pressure, as well as ulcerative colitis. Other surgical history included cholecystectomy.

DIFFERENTIAL DIAGNOSIS

Given the patient's history, possible causes included residual mitral regurgitation (MR), ischemic changes,

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ABBREVIATIONS AND ACRONYMS

LVOT = left ventricular outflow tract

MR = mitral regurgitation

SAM = systolic anterior motion

TEE = transesophageal echocardiography

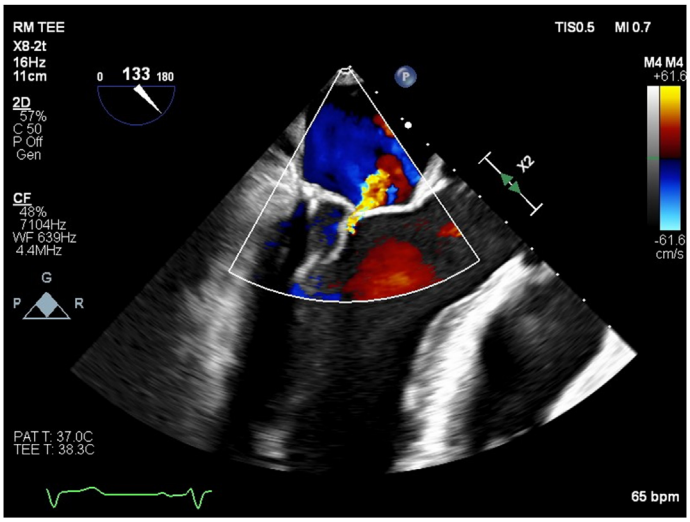
left ventricular (LV) dysfunction, LV outflow obstruction, or other postoperative complications.

INVESTIGATIONS

Initial evaluation included exercise right heart catheterization which reported an elevation in his wedge pressure to 29 mm Hg and a transesophageal echocardiogram (TEE) which showed mild MR with mitral stenosis. The patient was started on metoprolol without significant symptomatic improvement, and subsequently discontinued it. A trial of isosorbide mononitrate was also attempted to reduce elevation in intracardiac pressures with exercise, however, this did not result in symptom relief either.

Exercise and metabolic stress studies were performed. Electrocardiograms were all negative for ischemia and regional wall motion abnormalities. However, moderate chest pain and occasional premature ventricular contractions were noted during stress, without ST-segment changes. On stress echocardiography, the aortic root was dilated, with a maximal diameter of 4.2 cm. Mild concentric LV hypertrophy was observed, with an interventricular thickness of 1.2 cm and posterior wall thickness of 1.1 cm. The left atrium was also mildly dilated. Images showed evidence of mild to moderate MR with an eccentric, anteriorly directed jet. Regurgitant orifice area was 0.12 cm^2, peak and mean gradients were 9 mm Hg and 4 mm Hg, respectively, at 78 beats/min. Mild chordal systolic anterior motion (SAM) was also noted (Video 1), with an elongated anterior leaflet, but no significant left ventricular outflow tract (LVOT) obstruction was observed at rest. During stress, mitral valve (MV) peak and mean gradients were 23 mm Hg and 12 mm Hg, respectively, at 178 beats/min. However, the stress test was inconclusive, so repeat testing with chemical provocation was suggested. TEE confirmed the previous findings, and amyl nitrite administration showed an increase in MR but further evaluation was limited due to patient discomfort and nausea.

FIGURE 1 Intraoperative Transesophageal Echocardiogram at Rest



Intraoperative transesophageal echocardiogram showing mild mitral regurgitation and no systolic anterior motion at rest, with a relatively anterior coaptation point of the mitral valve leaflets.

MANAGEMENT

Given the lack of improvement with medical treatment, redo open heart surgery was scheduled. Intraoperative TEE showed mild-to-moderate MR due to prolapse and redundant leaflets (**Figure 1**, Video 2), with a relatively anterior coaptation point and an anteriorly directed MR jet. At rest under anesthesia, mild nonobstructive SAM was noted with normal LVOT gradient, along with residual prolapse of the P2-3 area, and normal pulmonary venous pattern. However, provocation with isoproterenol induced severe SAM with torrential MR, with peak LVOT gradient >40 mm Hg (**Figures 2 and 3**, Videos 3 and 4). The patient subsequently underwent mitral valve repair with triangular resection of prolapsing segment of P2, anterolateral papillary muscle reorientation with anterior leaflet plication, as well as valve sparing aortic root replacement.

DISCUSSION

SAM of the mitral valve anterior leaflet occurs in 4% to 8.4%[1] of patients after mitral valve repair. Management is often conservative with beta blockade, intravascular volume augmentation, and systemic vascular resistance increase; however, recurrent symptoms, in particular, with the presence of LVOT obstruction, may require reoperation.[2]

Ours is a case of a patient with symptomatic LVOT obstruction and MR due to SAM several years following mitral valve repair. Even though the patient's first repair was considered successful given the absence of residual MR, onset of symptoms following a short time interval after the surgery suggests that the choice of surgical technique subsequently proved to be suboptimal as it did not adequately take into consideration the risk of SAM. The particular feature of this case is the occult nature of obstruction and severe symptoms which were provoked by significant

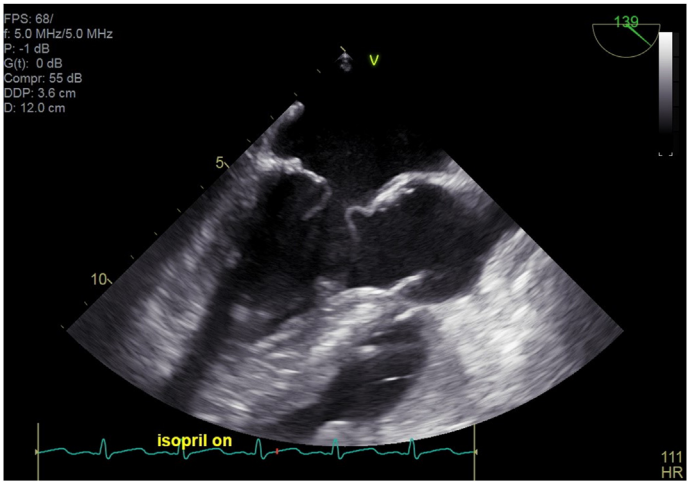
physical stress and eventually proven in the operating room under general anesthesia. One hypothesis to the lack of LVOT flow turbulence was decreased flow through the LVOT due to significant residual MR.

Factors contributing to SAM and LVOT obstruction include leaflet length and position of the coaptation point. A longer posterior leaflet with a lower anterior-to-posterior leaflet length ratio results in anterior displacement of the coaptation point which moves closer to the LVOT. This leaves more redundant leaflet slack close to the outflow tract which is susceptible to causing obstruction.[3,4] In our patient's case, the mechanism was SAM resulting in distortion of the anterior mitral valve leaflet, creating a wide coaptation gap, resulting in torrential MR (Figure 4). Other factors include a small LV cavity, septal hypertrophy, and hyperdynamic LV function, as well as bulging subaortic septum.[5] A smaller angle between the annular planes of the mitral and aortic valves may also contribute to dynamic displacement of the coaptation point.[6] Detailed analysis of the mitral apparatus, ideally with TEE, may help identify patients at risk and tailor surgical technique accordingly.

The patient had residual posterior leaflet prolapse; however, the associated MR was not severe enough to explain his symptoms. Preoperatively, the contribution of SAM to his symptoms remained unrecognized. Using TEE along with inducing tachycardia and hypotension revealed his severe SAM-related MR and helped us to create plan for surgical correction. Stress echocardiography and TEE with provocative maneuvers are both valuable diagnostic tools. Stress echo is often the first-line test for patients who can exercise and have an adequate acoustic window. TEE with provocative maneuvers such as amyl nitrate or isoproterenol offers superior imaging, particularly for more complex valve assessments, and is beneficial when transthoracic echocardiography is inconclusive or for patients unable to exercise.

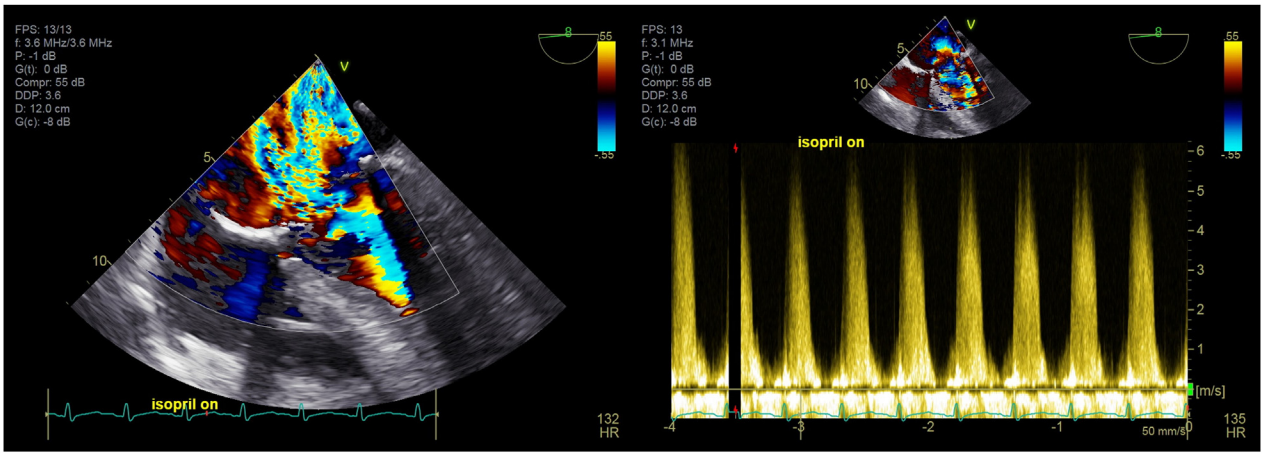
Suggested methods for both prevention and treatment are aimed at posterior migration of the mitral

FIGURE 2 Intraoperative Transesophageal Echocardiogram After Administration of Isoproterenol



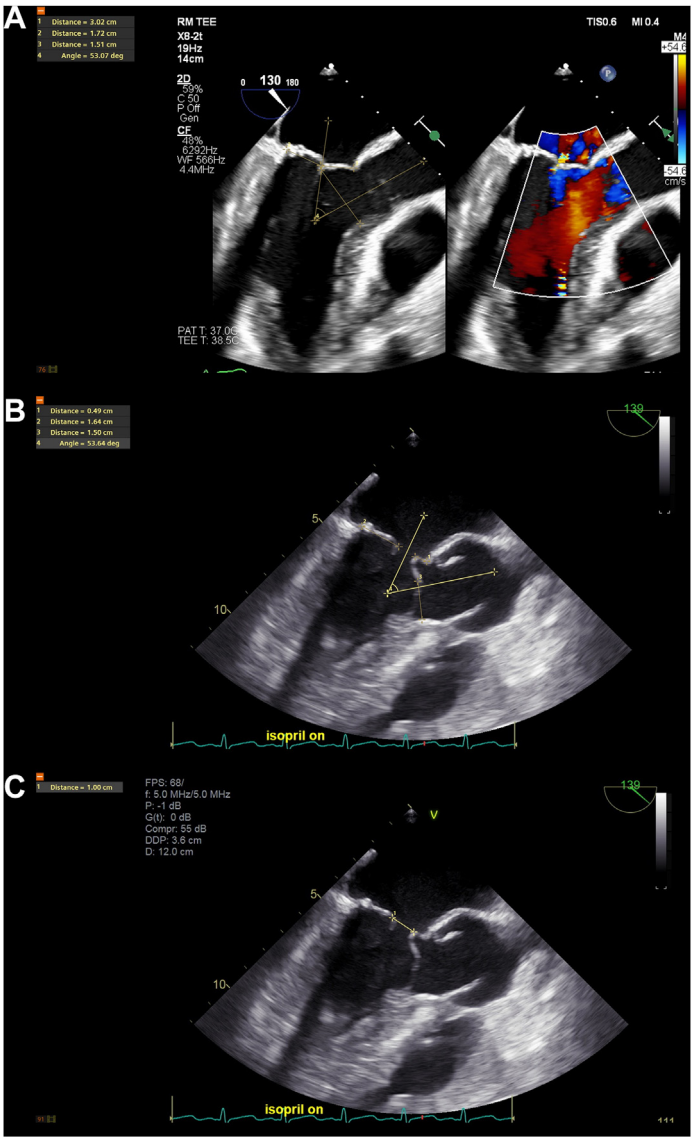
Intraoperative transesophageal echocardiogram after provocation showing a large mitral valve coaptation gap due to systolic anterior motion induced with isoproterenol.

FIGURE 3 Intraoperative Transesophageal Echocardiogram With Color Doppler After Administration of Isoproterenol



Intraoperative transesophageal echocardiogram with Doppler showing torrential mitral regurgitation following isoproterenol administration.

FIGURE 4 Intraoperative Transesophageal Echocardiogram Stills Showcasing Quantitative Measurements of MV Anatomy



Intraoperative transesophageal echocardiogram with quantitative measurements of MV components. (A) At rest: C-sept distance 3.02 cm; aorto-mitral angle 53.07 degrees; PMVL length 1.72 cm; and AMVL length 1.51 cm; coaptation gap 0.0 cm. (B, C) With isoproterenol: C-sept distance 1.50 cm; aorto-mitral angle 53.64°; PMVL length 1.64 cm; AMVL length 0.49 cm; and coaptation gap 1.0 cm. AMVL = anterior mitral valve leaflet; C-sept = coaptation point to septal distance (if <2.75 cm, increased risk of systolic anterior motion); MV = mitral valve; PMVL = posterior mitral valve leaflet.

leaflets coaptation point. These include the sliding posterior leaflet valvuloplasty, described by Jebara et al[7], in which the coaptation point is moved posteriorly. Another technique involves transposition of a secondary chordae tendineae into the anterior leaflet, when these are available, leading to tethering of the anterior leaflet and prevention of anterior movement.[8] Anterior leaflet plication was also suggested in patients with elongated anterior leaflets[9], and can be combined with papillary muscle reorientation[10] such as was done in our case.

FOLLOW-UP

The patient was seen at 1- and 4-months following surgery, at which he was asymptomatic and reported restored ability to perform daily activities. Follow-up echocardiogram 4 days later showed an ejection fraction of 54% ± 5% and trace MR with mild non-obstructive chordal SAM. LVOT gradient was 9 mm Hg with an increase to 11 mm Hg with amyl nitrite administration, and there was no change in SAM or MR. Estimated right pressures were within normal ranges. Repeat echocardiogram 4 months later showed similar findings consistent with symptomatic improvement.

CONCLUSIONS

This case highlights the importance of careful preoperative imaging and surgical planning in patients with mitral valve disease. Early identification of patients at risk for postoperative mitral leaflet abnormalities resulting in SAM and potentially symptomatic LVOT obstruction, preferably using TEE, should be encouraged for tailoring of surgical technique and prevention.

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KEY WORDS **left ventricular outflow tract obstruction, mitral regurgitation, mitral valve, systolic anterior motion, transesophageal echocardiography**

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