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Mitral Stenosis Reversed by Medical Treatment for Heart Failure

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Abstract

It is reported that functional mitral stenosis frequently develops after ring annuloplasty for ischemic mitral regurgitation. The mechanism is a combination of annular size reduction by surgery and diastolic mitral valve tethering, restricting the anterior leaflet opening due to posteriorly displaced papillary muscles with left ventricular dilatation. We report the case of a 57-year-old man who had a history of successful mitral valve plasty for degenerative mitral regurgitation. Four years later he developed heart failure, severe hypertension, mild mitral regurgitation, and significant mitral stenosis, which were reversed by aggressive medical treatment for heart failure.

Functional mitral stenosis (MS) frequently develops after ring annuloplasty for ischemic mitral regurgitation (MR) [1]. The mechanism is a combination of annular size reduction by surgery and diastolic mitral valve tethering, restricting the anterior leaflet opening due to posteriorly displaced papillary muscles with left ventricular (LV) dilatation [2]. Functional MS has not been reported in patients with degenerative MR. However, functional MS could develop even in patients with a history of successful valve plasty and annular size reduction for degenerative MR if the LV were dilated by another disease. In addition, because functional MS is dynamic and proportional to LV dilatation [1, 2], treatment for heart failure (HF), acting to reduce the LV size, can potentially improve such functional MS.

A 52-year-old man had HF with severe MR due to prolapse of a degenerative mitral valve in 2008. He had grade 4/6 holosystolic murmur and a third heart sound in the apex. His blood pressure was 120/70 mm Hg, he underwent mitral valve plasty for degenerative MR, he had P3 prolapse with 2 chordal ruptures and he underwent triangular resection, edge-to-edge anastomosis, and ring annuloplasty with a Physio Ring 30 (Edwards Lifesciences, Irvine, CA) of just size. After the surgery, HF disappeared and postoperative echocardiography confirmed a normal sized LV with an end-diastolic volume index of 71 mL/m^2, normal LV ejection fraction of 0.70, no MR and preserved mitral valve opening with a mitral valve area

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(MVA) of 1.8 cm^2, and a mean pressure gradient of 4.4 mm Hg (Fig 1A). The MVA was obtained by a continuity equation (LV end-diastolic volume − LV end-systolic volume = mitral filling flow volume = MVA × velocity time integral of mitral filling flow by continuous wave Doppler echocardiography), which can be utilized even in the presence of MR. His mitral valve plasty was successfully performed without major concerns.

He developed HF again 4 years later. This time, he had severe hypertension of 191/104 mm Hg, diabetes mellitus, and grade 2 to 3/6 systolic as well as diastolic murmurs were audible at the apex. Echocardiography demonstrated moderately dilated LV with an end-diastolic volume index of 84 mL/m^2, reduced LV ejection fraction of 0.40, mild MR, and limited mitral valve opening, resulting in a significant MS with an MVA of 1.2 cm^2 and a mean pressure gradient of 8.4 mm Hg (Fig 1B). Coronary angiography revealed no significant lesions in the major branches. The patient was diagnosed as hypertensive LV dysfunction with mild MR and significant MS. Both systolic and diastolic mitral leaflet motions were restricted without any clear organic lesion. The etiology of the MR and MS was diagnosed as functional. As significant MS was associated with HF, surgical indication for MS was discussed. The possibility of MS to improve after medical treatment for HF was considered because functional MS is highly dynamic and can potentially respond to medical treatment [1, 2]. Consequently, surgery was not performed and aggressive medical treatment for HF and hypertension with vasodilators and diuretics was started. His HF symptom as well as cardiac murmurs disappeared with a reduction in blood pressure to 140/80 mm Hg. Repeated echocardiography 4 months later demonstrated a reduction in LV end-diastolic volume of 66 mL/m^2, disappearance of MR, improvement of the mitral valve opening to an MVA of 1.7 cm^2, and a mean pressure gradient of 4.6 mm Hg (Fig 1C).

Comment

In this patient there was no MS before the surgery by echocardiography. Direct observation at the time of surgery also confirmed no findings of rheumatic or other causes of MS. After the surgery, MVA was reduced but only modestly. Possible reasons for the modest reduction in MVA could be annular size reduction by the surgical ring implantation and mild restriction of leaflet motion by the leaflet resection and edge-to-edge anastomosis. However, the degree of MS early after surgery was not significant, with an MVA of 1.8 cm^2. Mitral regurgitation as well as HF symptoms disappeared after the surgery. Therefore, the surgery was successful.

However, this patient developed HF 4 years later. At this time, he had severe hypertension, moderate LV dilatation, LV dysfunction, mild MR, and significant MS. As the mitral valve demonstrated restricted closure and opening without any clear organic lesion, being consistent with systolic and diastolic tethering, a functional etiology of MR and MS was considered. There have been reports demonstrating development of functional MS following ring annuloplasty for ischemic MR with LV dilatation. This patient showed that functional MS could develop even in a patient with successful valve plasty for degenerative MR, if the LV is dilated by another disease.

Because functional MS is dynamic and proportional to LV dilatation [1, 2], the degree of functional MS can potentially vary considerably. At the time of second HF 4 years after valve plasty, MS was significant with an MVA of 1.2 cm^2. According to the guideline, the MS at that time could be considered indicative for surgery. After aggressive medical treatment for HF, the HF symptom disappeared and the MS was reversed to an MVA of 1.7 cm^2. At this point, insignificant MS can be considered as not indicative for surgery. Therefore, the surgical indications need to be carefully evaluated in patients with functional MS. In addition to the leaflet configurations, the serial evaluation strongly suggested that the MS in this patient was subvalvular and therefore dynamic, as opposed to annular MS with a fixed ring size.

This case report demonstrates that functional MS could develop even in a patient with a history of successful valve plasty for degenerative MR, if the LV is dilated by another disease. Functional MS can potentially be reversed by medical treatment and thus requires careful evaluation of the surgical indications.

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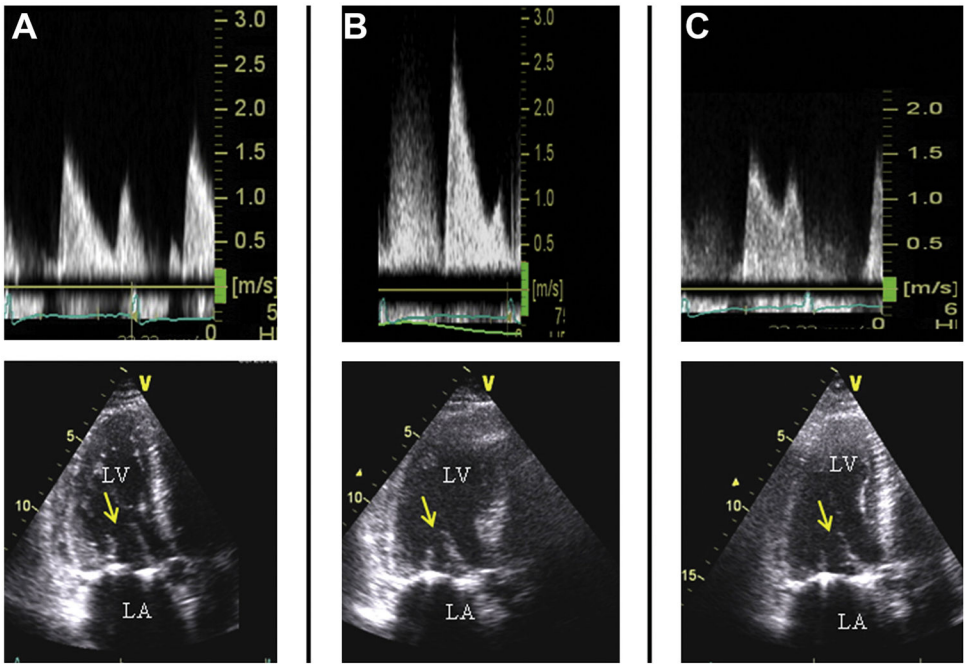

Fig. 1.

(A) Mitral filling flow velocity by continuous wave Doppler echocardiography and mitral valve full opening at early diastole by apical long axis view 2 weeks after valve plasty for degenerative mitral regurgitation. The transmitral pressure gradient is only modestly increased with a peak and mean gradient of 9.7 and 4.4 mm Hg, respectively. The mitral leaflet opening is preserved (yellow arrow) and the mitral valve area (MVA) by continuity equation was 1.8 cm^2. (B) At the time of the second heart failure that developed 4 years after surgery, the transmitral pressure gradient is significantly increased with a peak and mean gradient of 26.8 and 8.4 mm Hg, respectively. In association with left ventricular (LV) dilatation, the mitral leaflet opening is reduced (yellow arrow) with an MVA of 1.2 cm^2, indicating significant functional mitral stenosis. (C) Four months after aggressive medical treatment for heart failure, the transmitral pressure gradient is significantly improved with a peak and mean gradient of 9.3 and 4.6 mm Hg, respectively. With reverse LV remodeling, the mitral leaflet opening is improved (yellow arrow) with an MVA of 1.7 cm^2. (LA = left atrium.)