Hypertrophic Cardiomyopathy – Changing Mitral Insufficiency, Non SAM Related

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Citation


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Abstract

Systolic anterior motion (SAM), as a cause of mitral insufficiency is a common condition in patients with hypertrophic cardiomyopathy (HCM). Much more rare are the cases of HCM with mitral insufficiency, in which the etiology of mitral insufficiency is not a SAM. Involving the subvalvular apparatus in the disease process, reduced left ventricular (LV) volumes potentiate each other, leading to a worsening of clinical symptoms. We present a patient with HCM, ischemic heart attacks and varying mitral insufficiency unprovoked by SAM, with pulmonary edema. At critical preoperative state we performed mitral valve replacement.

INTRODUCTION

Hypertrophic cardiomyopathy HCM can lead to worsening social status, disability, expected early death of patients. It occurs at a frequency of 2‰ in the population. Systolic anterior motion SAM as a cause of mitral insufficiency is a common condition in patients with HCM. Much more rare are the cases of HCM with mitral insufficiency, in which the etiology of mitral insufficiency is not a SAM. When the subvalvular apparatus is involved in the disease process, reduced left ventricular LV volumes potentiate each other, leading to a worsening of clinical symptoms. We describe a case of a patient with HCM, ischemic heart attacks and varying mitral insufficiency unprovoked by SAM. Early diagnosis and establishing the mechanism of mitral insufficiency are of principal importance for the surgical strategy and follow-up.

CASE REPORT

We present a 61 year old man with complaints of ischemic chest pain 2-3 times daily, unprovoked by physical effort. He had similar symptoms since 5 years. Recently he felt an exacerbation of symptoms in strength as well as duration of the attacks. He suffered from paroxysmal atrial fibrillation and arterial hypertension. His father and brother had a history of arterial hypertension. On admission the ECG revealed ST segment depression in I, aVL, V3-V6. Coronary arteriography showed no coronary artery disease, invasive left ventricular outflow tract (LVOT) gradient 36mmHg and mild mitral regurgitation. Echocardiography showed LV hypertrophy, end diastolic / end systolic volumes: 138/54ml, ejection fraction 60%. Interventricular septum and posterior wall thickness – 17/13mm, left atrial (LA) diameter – 46mm. Mild to moderate mitral insufficiency in condition of pulmonary edema. No SAM shown. Pulmonary hypertension of 110mmHg. During the hospital stay he deterioration with pulmonary edema and was transferred to then ICU and placed on mechanical ventilatory support. Transesophageal echo showed mild to severe mitral regurgitation.

The patient was scheduled for surgery and we performed mitral valve replacement with a mechanical valve prosthesis St. Jude Medical 31mm. Mitral insufficiency was IIIB type with papillary muscles hypertrophy and very shortened chords. Cross-clamp time was 69 minutes. The postoperative period was uneventful. The patient was extubated after 21 hours and postoperatively he was in stable condition and discharged on the 9th postoperative day from hospital. Echo at discharge showed normal function of the mitral valve prosthesis with mitral valve gradients 7.7/2.4mmHg. LVOT gradient – 10mmHg. Pulmonary artery pressure – 25mmHg. Ten months postoperatively the patient is in NYHA class I, without stenocardial symptoms or pulmonary disorder. 10th month Echo showed normal function of the mitral prosthesis.
with gradients – 6.6/2.5mmHg. No LVOT gradient. Interventricular septum / posterior wall thickness – 15/15mm, ejection fraction 61%, LA diameter – 36mm, LA surface – 16cm². Pulmonary artery pressure – 35mmHg.

COMMENT

The assessment of mitral regurgitation in HCM patients has some limitations due to the fact that the LV cavity is often small, even in the presence of severe mitral regurgitation [1]. Conventional quantitative and semiquantitative Doppler parameters are not validated in these patients. Qualitative measures of valve anatomy, continuous wave and colour Doppler, combined with left atrial size and estimation of pulmonary artery pressure are more helpful. In selected cases, transesophageal echo is suitable in defining the mechanism and severity of mitral valve regurgitation [1]. We recommend transesophageal echo for mitral valve assessment in any patient with LVOT obstruction.

In the case presented, septal ablation is not a method of choice, having in mind septal thickness and concomitant mitral regurgitation [1] and the critical preoperative state. Although septal ablation relieves left ventricular outflow tract gradients and improves functional status, the late results of this method of treatment are uncertain. Also the need for permanent pacing is higher with septal ablation compared with surgical myectomy [2], [3].

According to ESC guidelines for HCM, surgical myectomy is indicated in LVOT gradient ≥50mmHg in symptomatic patients [1]. SAM as a cause of mitral insufficiency is common in patients with HCM [4]. In this state, it is appropriate to perform septal myectomy and mitral valve surgery [5]. Mitral valve plasty in HCM is preferable to mitral valve replacement, especially in centers focused in this pathology [6]. Postoperative period in patients with mitral valve plasty is smoother than mitral replacement, with low level of hospital mortality (to 0% according to Hazelrigg et al.) [6].

The main tasks in HCM are to establish the severity of the LVOT obstruction, to establish the mechanism of mitral insufficiency, not to underestimate the degree of insufficiency.

References

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