

Papillary Muscle Resection as a Treatment for Midventricular Obstruction

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We have experienced a surgically treated case of midventricular obstruction. A simple mitral valve replacement was effective to relieve the obstruction and to increase the cavity of the left ventricular outflow tract without a myectomy through the aortic valve. (Ann Thorac Cardiovasc Surg 2002; 8: 109–11)

Key words: hypertrophic cardiomyopathy, midventricular obstruction

Introduction

We surgically treated a case of hypertrophic cardiomyopathy in which midventricular obstruction was evident. Resection of the hypertrophic papillary muscle played an important role in relieving the obstruction. A comparison of the preoperative and postoperative left ventriculography (LVG) and transesophageal echocardiography (TEE) showed not only a relief of the obstruction but also an increase in the cavity of the left ventricular outflow tract (LVOT).

A 77-year-old female presented with recurrent angina on effort. She had demonstrated systolic murmur for more than 10 years. Transthoracic echocardiography showed mild mitral valve regurgitation due to posterior leaflet prolapse, hypertrophic septal muscle, systolic anterior movement of the mitral cusp and the pressure gradient at the level of the obstruction was 130 mmHg on cardiac echo. The thickness of the septal muscle was 20 mm in length and posterior wall thickness was 18 mm in length. LVG showed the obstruction was located at the level of the middle septal muscle. The pigtail catheter could not be inserted through the obstruction. The septal thickness and the hypertrophic papillary muscles caused the obstruction (Fig. 1a). Mitral valve replacement using a low profile mechanical valve with papillary muscle resection

was thought to be effective to relieve the obstruction, because the obstruction was caused mainly by the hypertrophic papillary muscle and progressing mitral valve regurgitation. Intraoperative TEE also demonstrated mild mitral valve regurgitation, systolic anterior movement of the anterior leaflet and the midventricular obstruction (Fig. 2a). At surgery, the movement of the anterior leaflet was restricted due to the short chorda with large muscle bars. The subaortic area was not narrow. Mitral valve replacement with resection of the papillary muscles was performed. Intraoperative TEE demonstrated widening of the LVOT without pressure gradient (Fig. 2b). Histologic examination of the muscle showed findings of hypertrophic cardiomyopathy including hypertrophy of the myocytes, some areas of interstitial fibrosis, and prominent perivascular fibrosis. Postoperative transthoracic echocardiography showed that LVOT was widened, that the pressure gradient was 23 mmHg and that the septal thickness was reduced to 14 mm in length. Postoperative LVG showed the shortening of the papillary muscle caused a widening of the LVOT. A pigtail catheter was easily inserted into the apex. Pressure gradient could not be detected from the apex to the left ventricular outflow tract (Fig. 1b). She was discharged without any event and has been well since.

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Comment

Midventricular obstruction is frequently caused by the anomalous insertion of a papillary muscle directly into the anterior mitral leaflet without interposition of chordae tendineae.¹⁾ Though the hypertrophic papillary muscle was

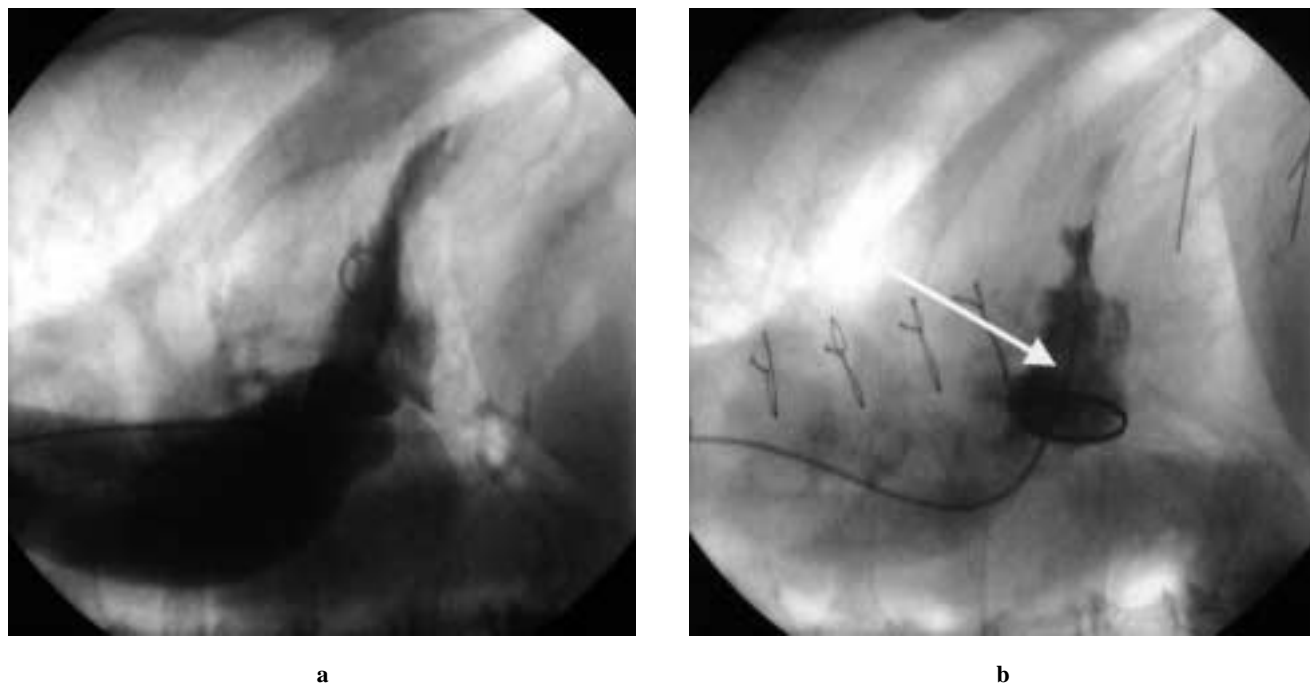


Fig. 1. a: Preoperative left ventriculography. The septal thickness and the hypertrophic papillary muscles caused the obstruction. The pigtail catheter could not be inserted through the obstruction point.
b: Postoperative left ventriculography showed the shortening of the papillary muscle caused a widening of the left ventricular outflow tract (LVOT) (arrow). A pigtail catheter was easily inserted into the apex. Pressure gradient could not be detected from the apex to the LVOT.

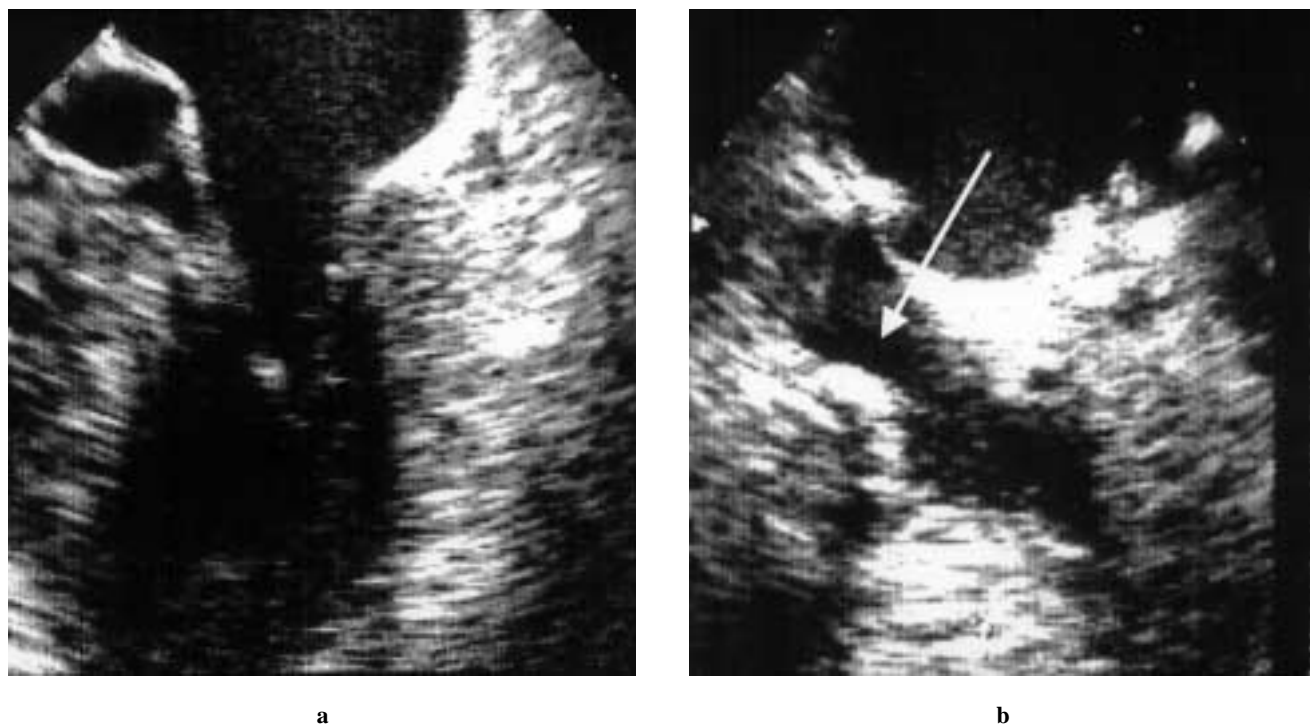


Fig. 2. a: Transesophageal echocardiography (TEE) demonstrated systolic anterior movement of the anterior leaflet and left ventricular outflow obstruction.
b: Postoperative TEE demonstrated widening of the LVOT (arrow) without pressure gradient.

not directly connected to the anterior leaflet in our case, the mechanism of the obstruction was the same as direct papillary muscle insertion into the mitral valve, because midcavity muscular obstruction was recognized between the papillary muscle and anterior septal muscle.

Regarding surgical treatment, Maron and associates reported a novel extended myectomy including the hypertrophic septal and papillary muscle to relieve the obstruction.²⁾ The standard septal myectomy/myotomy may not be sufficient to relieve the midventricular obstruction because of the lack of volume reduction for the hypertrophic papillary muscle. However, for those patients with mitral valve pathology like our case, this novel technique is not proposed. Mitral valve replacement has previously been advocated in hypertrophic cardiomyopathy with mitral valve disease.³⁻⁵⁾ Obstruction due to systolic anterior movement of the anterior leaflet in the subaortic area will be excluded. In the treatment of midventricular obstruction, MVR with papillary muscle resection relieves the obstruction between the papillary muscle and septal muscle and may increase the subaortic area. In this case, TEE demonstrated that the LVOT was widely dilated immediately after surgery. The relief of the obstruction and the widening of the LVOT were evident in the postoperative LVG. Thus, we believe that the MVR is theoretically a reasonable method for hypertrophic cardiomyopathy with mitral pathology.

References

1. Klues HG, Roberts WC, Maron BJ. Anomalous insertion of papillary muscle directly into anterior mitral leaflet in hypertrophic cardiomyopathy: significance in producing left ventricular outflow obstruction. *Circulation* 1991; **84**: 1188-97.
2. Maron BJ, Nishimura RA, Danielson GK. Pitfalls in clinical recognition and a novel operative approach for hypertrophic cardiomyopathy with severe outflow obstruction due to anomalous papillary muscle. *Circulation* 1998; **98**: 2505-8.
3. Heric B, Lytle BW, Miller DP, et al. Surgical management of hypertrophic obstructive cardiomyopathy. Early and late results. *J Thorac Cardiovasc Surg* 1995; **110**: 195-206.
4. McIntosh CL, Greenberg GJ, Maron BJ, et al. Clinical and hemodynamic results after mitral valve replacement in patients with obstructive hypertrophic cardiomyopathy. *Ann Thorac Surg* 1989; **47**: 236-46.
5. Walker WS, Reid KG, Cameron EW, Walbaum PR, Kitchin AH. Comparison of ventricular septal surgery and mitral valve replacement for hypertrophic obstructive cardiomyopathy. *Ann Thorac Surg* 1989; **48**: 528-34.