

Partial Left Ventriculectomy for End-stage Cardiomyopathy: Report of a Case

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Cardiac transplantation is an established treatment for end-stage heart failure, but its use is very limited. Partial left ventriculectomy has been reported as an alternative treatment for end-stage dilated cardiomyopathy. However, it has been well recognized that emergency partial left ventriculectomy for intractable decompensation is associated with poor survival. We report a case of a 68-year-old man with a left ventricular end-diastolic diameter of 108 mm, who underwent emergency extended partial left ventriculectomy, without papillary muscle resection, and mitral valve replacement with chordae preservation to deal with ongoing cardiogenic shock caused by end-stage dilated cardiomyopathy. The patient's cardiac status and general condition improved after the operation, and he survived the crisis. This operation should be considered as an alternative strategy for patients with septal motion and very large left ventricle. Thus, we report a successful extended partial left ventriculectomy and mitral valve replacement for end-stage dilated cardiomyopathy with very large left ventricular end-diastolic diameter. (Ann Thorac Cardiovasc Surg 2004; 10: 307–10)

Key words: cardiomyoplasty, mitral valve repair, mitral valve replacement

Case Report

A 68-year-old man first developed congestive heart failure in 1998. Several medical treatments were administered, but, by 2002, medical treatment was no longer effective at controlling the heart failure (hypotension, renal dysfunction) (Fig. 1). With inotropic support (dopamine 2 $\mu\text{g}/\text{kg}/\text{min}$, dobutamine 2 $\mu\text{g}/\text{kg}/\text{min}$, and milrinone 0.5 $\mu\text{g}/\text{kg}/\text{min}$), the patient was categorized, preoperatively, as class IV according to the New York Heart Association (NYHA) classification. On preoperative echocardiogram, left ventricular end-diastolic diameter (LVEDd) was excessively dilated (108 mm), and only the septal portion of the left ventricle was slightly kinetic with an ejection

fraction (EF) of 13%, fractional shortening (%FS) of 6%, and moderate mitral regurgitation (MR) (Fig. 2). However, the mitral annulus was not dilated (26 mm), and the MR jet was eccentric to the atrial posterior wall without anterior leaflet prolapse.

The patient was anesthetized in the orthopnea position with the support of intra-aortic balloon pumping (IABP). Surgery was performed via a median sternotomy. Hypothermic (28°C) cardiopulmonary bypass was instituted and ventriculectomy was performed under ventricular fibrillation. We regarded the myocardium which was akinetic on preoperative echocardiography and on intraoperative inspection under the decompressed situation as damaged. First, the anterolateral ventricular wall myocardium was resected between the left anterior descending artery and the anterior papillary muscle, starting at the apex of the heart, and then proceeding superiorly to approach the vicinity of the circumflex artery (Fig. 3). Secondly, the inferior myocardial wall was resected between the posterior descending artery and the posterior papillary muscle, followed by resection of the myocar-

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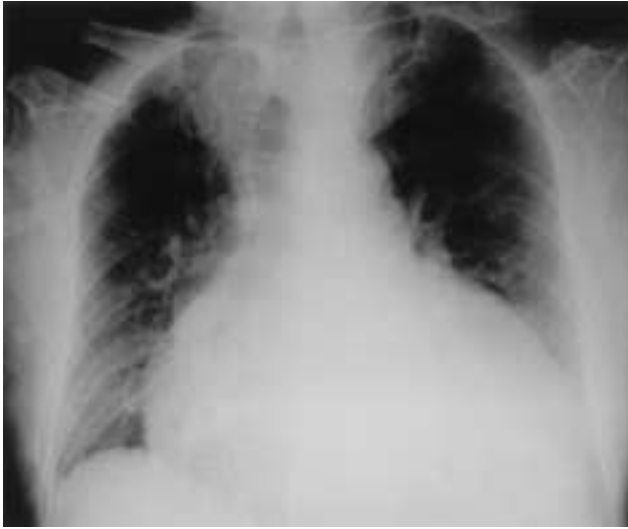


Fig. 1. Chest X-ray showing an extremely enlarged heart (80% cardiothoracic ratio).



Fig. 3. The interior cavity seen via the anterolateral wall incision, parallel to the left anterior descending artery. Long and short arrows indicate the posterior and anterior papillary muscles, respectively. The left ventricle was extremely dilated, and the left ventricular wall was thin.

dium between the anterior and posterior papillary muscles. A total of 159 g of the myocardium was resected (Fig. 4). After closure of the left ventricle with strips of felt, a right-sided left atriotomy was performed under cardioplegic arrest. Since an initially attempted edge-to-edge mitral valve repair was not effective in attaining perfect control of the MR, mitral valve replacement with preservation of the chordae was performed. The patient was weaned from cardiopulmonary bypass smoothly with the support of IABP and inotropes. The histology of the resected muscle showed massive interstitial fibrosis,

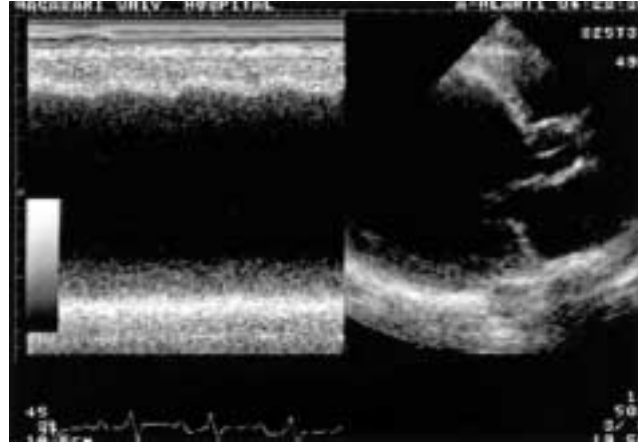


Fig. 2. Echocardiogram showing that the LVEDd was 108 mm, EF=13%, and %FS=6%.

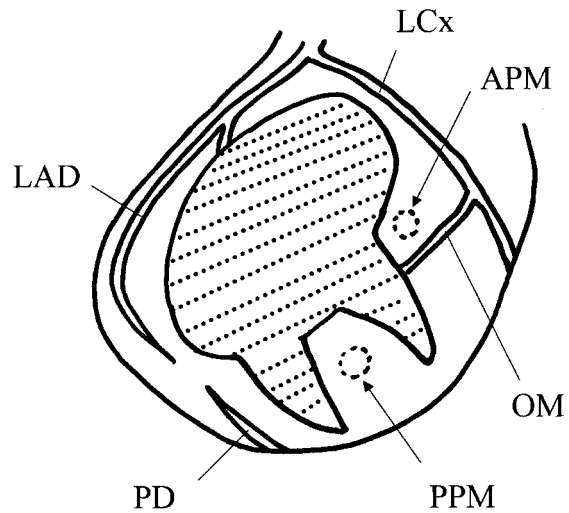


Fig. 4. Schematic illustration of the resection. The shaded area was resected.

LAD; Left anterior descending artery, LCx; Left circumflex artery, OM; Obtuse marginal branch, PD; Posterior descending artery, APM; Anterior papillary muscle, PPM; Posterior papillary muscle

swelling of nucleuses, and variation in size of the myocytes (Fig. 5).

The postoperative course was uneventful. IABP was discontinued at 10 days, and inotropic support was subsequently tapered off, 17 days after the operation. Three months after the operation, his NYHA class improved from class IV to class II, and his peak oxygen consumption was 12.5 ml/kg/min. The cardiothoracic ratio decreased from 80% to 55% (Fig. 6). EF and %FS increased

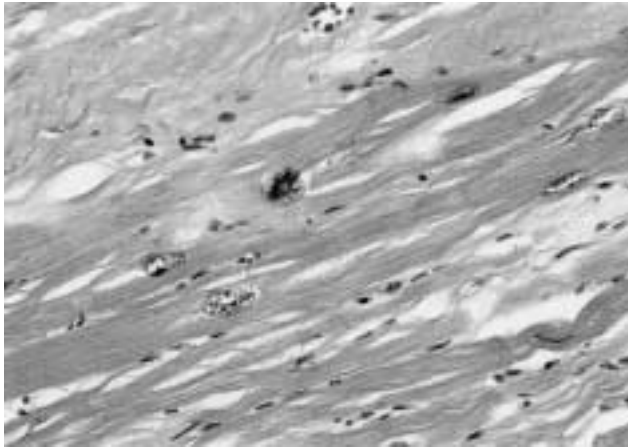


Fig. 5. Histology of the resected muscle showed that massive interstitial fibrosis, swelling of nucleuses, and variation in size of the myocytes. (Hematoxylin-eosin stain, original magnification $\times 250$)

from 13% (with inotropic support) to 25% (without inotropic support), and from 6% to 12%, respectively. LVEDd decreased from 108 mm to 71 mm (Fig. 7). Twelve months later, no remarkable changes have occurred in the patient's NYHA class, peak oxygen consumption (12.3 ml/kg/min), and LVEDd (73 mm).

Discussion

The aim of partial left ventriculectomy (PLV) is to resect the damaged lesions on the left ventricular (LV) wall, and thus decrease the LV diameter to a more optimal size.¹⁾ According to Laplace's law, following PLV, wall stress is reduced and myocardial oxygen consumption decreases, while LV contractility increases.²⁾ PLV is accepted as an alternative option for the treatment of patients with end-stage dilated cardiomyopathy. However, emergency operation for the ongoing cardiogenic shock is recognized to carry extremely high risk.^{3,4)} Suma et al. reported that 6 of 7 patients undergoing emergency PLV due to cardiogenic shock died while still in the hospital.¹⁾ Excessive resection improves systolic function while decreasing diastolic compliance, resulting in reduced LV function.⁵⁾ However, the optimal postoperative LV size and the proper degree of LV resection have not been determined. The goal of PLV was to return the LVEDd to near normal. The LVEDd after resection was predicted by a simple formula: for every 3.14 cm of LV muscle circumference resected, the LVEDd was decreased by 1 cm. Since resection between the papillary muscles was insufficient

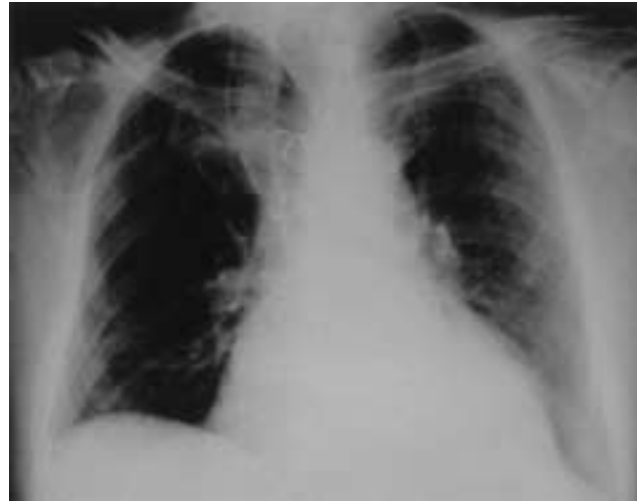


Fig. 6. Postoperative chest X-ray showing a 55% cardiothoracic ratio.



Fig. 7. Postoperative echocardiogram showing the decrease of the left ventricular dimension and improvement of left ventricular wall motion (LVEDd 71 mm, EF=25%).

for such an enlarged LV, we judged that we needed to resect more LV muscle to return the LVEDd to near normal. Previously, McCarthy et al. transected one or both papillary muscles to resect more of the LV wall, and then translocated the resected heads of the papillary muscles to preserve the mitral apparatus.⁴⁾ However we did not resect the papillary muscles from the LV wall, since we thought such resection of the papillary muscles could damage LV contractility, even if translocated.

To deal with the MR, we first attempted to control the MR by ringless edge-to-edge mitral valve repair,⁶⁾ but we could not achieve perfect control of the MR. We avoided ring annuloplasty due to the fact that the smaller preop-

erative mitral valve area carries a higher risk of post-repair mitral stenosis. We abandoned the mitral valve repair, to avoid post-operative LV dilatation caused by volume overload from the residual MR.⁷⁾ We believe that the failure of the mitral valve repair was a result of the large structural change occurring in the mitral apparatus following a large distortion of the LV. McCarthy et al. reported one patient who had an unusual eccentric jet and residual MR after attempted edge-to-edge repair who finally underwent mitral valve replacement.⁴⁾ The possibility exists that eccentric MR cannot be controlled perfectly by edge-to-edge mitral valve repair.

In conclusion, this extended PLV was effective in rescuing a patient who was at high risk of mortality and decreased the LVEDd. Thus, this extended PLV can be considered an alternative strategy for end-stage dilated cardiomyopathy patients with septal wall viability and enlarged LV.

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