

# Aortic Valve Replacement Combined with Endoventricular Circulatory Patch Plasty (Dor Operation) in a Patient with Aortic Valve Stenosis and Severe Ischemic Cardiomyopathy

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**A 58-year-old woman with ischemic cardiomyopathy and aortic valve stenosis, underwent aortic valve replacement and simultaneous endoventricular circulatory patch plasty (Dor operation). She underwent coronary artery bypass grafting for severe triple vessel disease 10 years ago. Recently she started to show severe congestive heart failure. Aortic valve stenosis with a pressure gradient of 85-mmHg was also found. Coronary bypasses were all patent, but the left ventricle (LV) was severely dilated (LVDd/Ds=71/61 mm) and the ischemic cardiomyopathy was considered as the cause. She successfully underwent aortic valve replacement and endoventricular circulatory patch plasty. The initial postoperative course was complicated with intractable ventricular arrhythmia, but the subsequent course was smooth and the patient was discharged with improved symptoms (New York Heart Association, NYHA Class II). Postoperative catheterization showed decreased left ventricular volume and improved contractility. This case implies the role of LV remodeling procedure in the ischemic cardiomyopathy combined with aortic valve lesion. (Ann Thorac Cardiovasc Surg 2001; 7: 170–4)**

**Key words:** ischemic cardiomyopathy, endoventricular circular patch plasty, aortic valve stenosis, aortic valve replacement

## Introduction

It has been well recognized that left ventricular aneurysmectomy leads to significant improvements in the symptoms of congestive heart failure.<sup>1)</sup> This concept has been expanded from the dyskinetic (aneurysmal) to the akinetic ventricle, which previously was not indicated for ventricular reconstruction.<sup>2)</sup> Since 1984, Dor and co-workers have developed the use of an endoventricular circular patch, which is sutured over the contractile area

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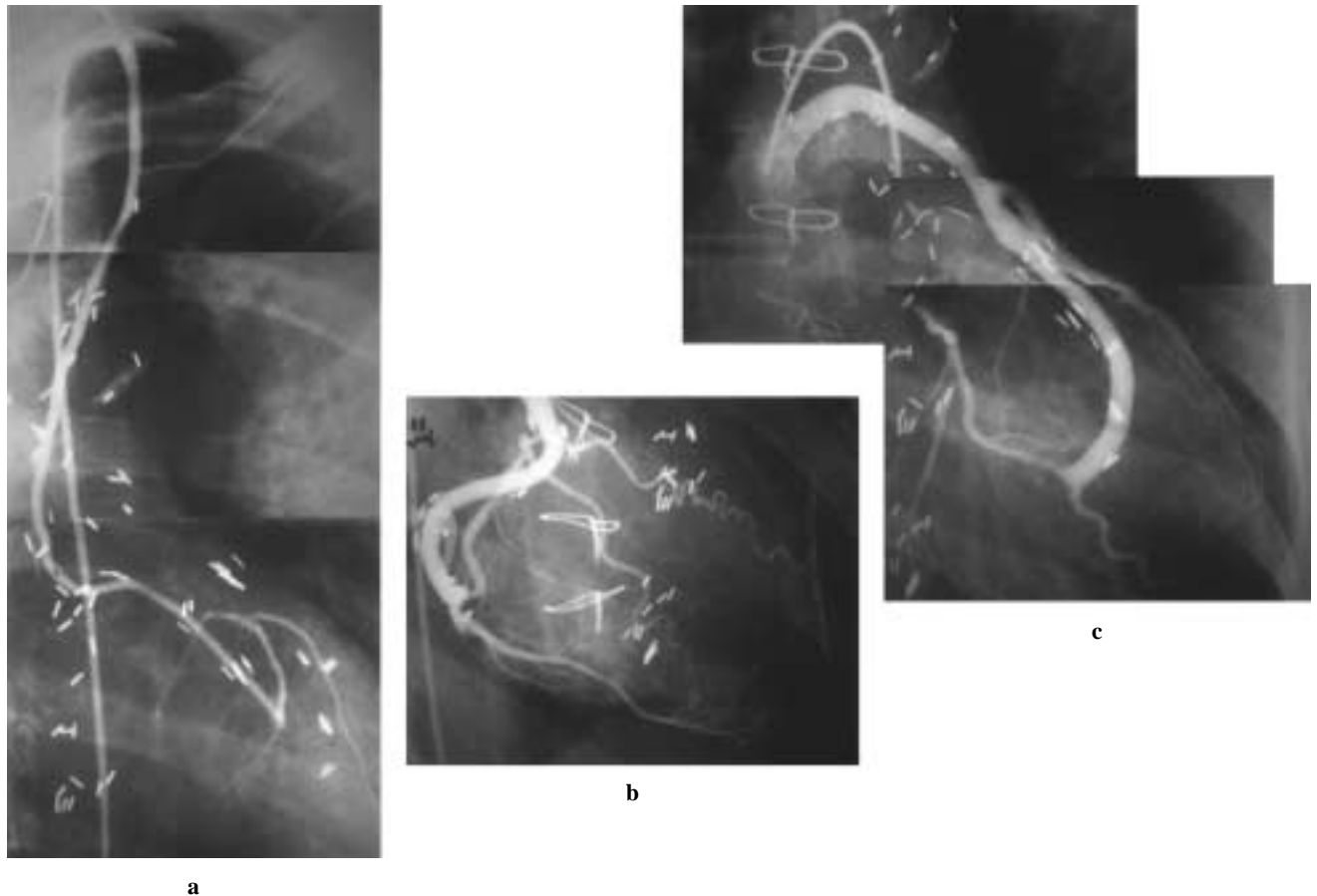
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and excludes the akinetic or dyskinetic areas, including the interventricular septum.<sup>3-8)</sup> This technique of left ventricular reconstruction using endoventricular circular patch plasty (EVCPP: Dor operation) has been utilized increasingly for patients with end-stage ischemic cardiomyopathy, who were previously listed for heart transplantation.<sup>9)</sup> In a case with combined lesions such as valvular disease and coronary artery disease, a simultaneous procedure may be required. In this report, we describe a case of aortic valve stenosis, complicated with severe left ventricular dysfunction secondary to end-stage ischemic cardiomyopathy. The patient was treated successfully with aortic valve replacement combined with EVCPP.

## Case Report

The patient was a 58-year-old woman. In 1984, she was admitted to a hospital for acute myocardial infarction. She had been followed with medication until 1990, when



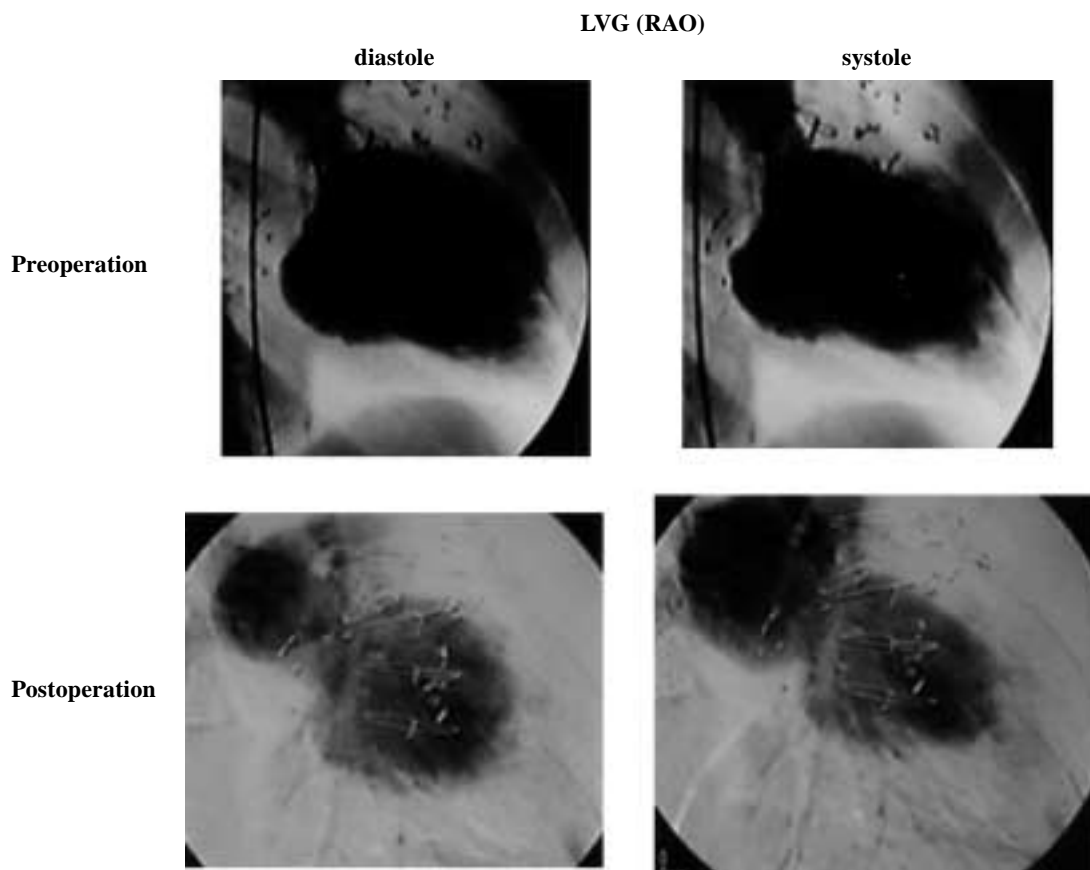
**Fig. 1.** Coronary angiogram performed before the operation demonstrated patent grafts.

a: Left internal thoracic artery (LITA)-LAD, b: saphenous vein graft (SVG)-right coronary artery (RCA), c: SVG-obtuse marginal artery (OM) and first diagonal (Y graft).

she developed sustained anterior chest pain. She underwent coronary angiography, which demonstrated triple vessel disease with total occlusion of the left anterior descending artery (LAD). The left ventriculogram showed severely depressed left ventricular systolic function with antero-septal myocardial infarction. Left ventricular ejection fraction (LVEF) was 26%. On May 28, 1990, she underwent quadruple coronary artery bypass grafting (CABG) to the LAD, the first diagonal, obtuse marginal artery, and the right coronary artery (RCA). Her postoperative course was uneventful and she was discharged at the end of June 1990. In October 1998, she developed symptoms of severe congestive heart failure. The resting 12-lead electrocardiogram showed the negative T wave in III and V5-V6, and abnormal Q wave in III, V1-V4. She was admitted emergently and underwent cardiac catheterization. Although all the grafts were patent (Fig. 1), left ventricular systolic function was severely depressed with an elevated left ventricular end diastolic pressure of up to 28 mmHg. The left ventricle

was severely dilated with an akinetic antero-septal and apical area. The other area showed severely hypokinetic wall motion. The left ventricular end-diastolic volume (LVEDV) was 203 ml (LVEDVI: 144 ml/m<sup>2</sup>), the left ventricular end-systolic volume (LVESV) was 155 ml (LVESVI: 110 ml/m<sup>2</sup>), and the left ventricular ejection fraction (LVEF) was 24% (Fig. 2, Table 1). Echo-doppler study revealed an 85-mmHg pressure gradient across the aortic valve with 3+ aortic regurgitation.

Surgery was performed on December 9, 1998. Under cardioplegic cardiac arrest, the aortic valve was inspected. The valve was tricuspid and all leaflets were severely thickened and calcified. The orifice admitted a 20 mm Hegar dilater and an 18-mm prosthetic aortic valve (ATS-AP, ATS Medical Inc., Minneapolis, MN) was implanted. After the aortotomy was closed, the left ventricle (LV) was opened in the center of the depressed anterior wall area produced by aspiration through a LV vent catheter. The endocardial scar characterized with a whitish appearance was extensively identified in the antero-septal



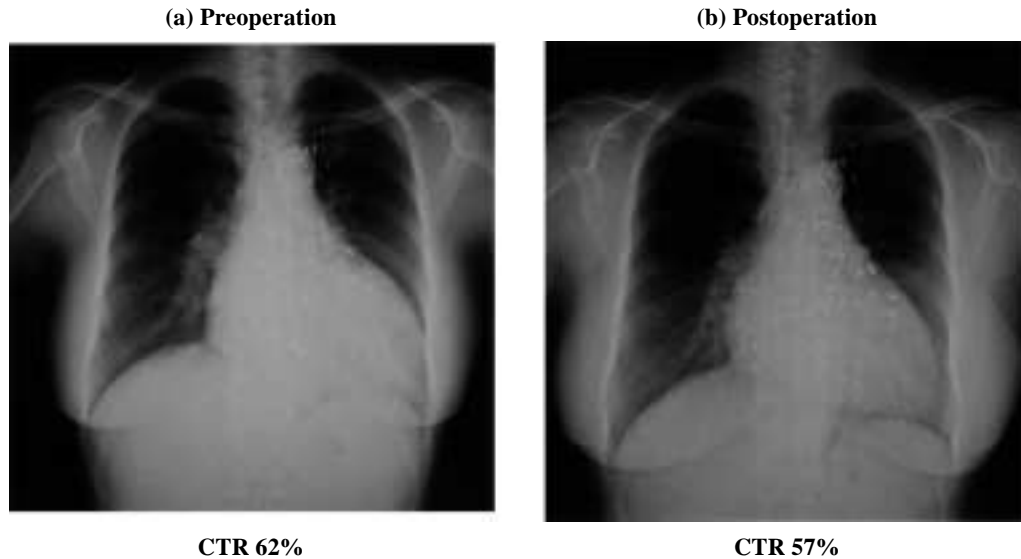
**Fig. 2.** Preoperative left ventriculogram showed a markedly enlarged left ventricular cavity with a large akinetic anteroseptal area and severe hypokinetic inferolateral area. Postoperative left ventriculogram showed a reduced ventricular volume and generally improved contractility.

and lateral area. The endoventricular circular suture was passed in the plane including the mid-point of the inter-ventricular septum, the apex, and a line with a 1.5-cm distance from the anterolateral papillary muscle. By tying down this suture, the opening of the LV was plicated from a 5 to 2.5-cm diameter. A Hemashield patch was

fixed on this line with a 2-0 prolene monofilament suture. After hemostasis was secured, the excluded myocardial edge was re-approximated directly above the patch. The suture line was reinforced by incorporating stripes of bovine pericardium. The cardiopulmonary bypass was discontinued uneventfully with an intraaortic balloon pump (IABP), which was inserted prophylactically. Her initial postoperative course was unremarkable and she was extubated on the day of the operation. She developed sustained ventricular tachycardia on the next day, and was eventually placed on percutaneous cardiopulmonary support (PCPS). After the arrhythmia was controlled, the patient was weaned from PCPS and IABP in five and eight days respectively. Postoperative cardiac catheterization performed at approximately two months after the operation showed improved pulmonary artery and wedge pressure (Table 1). Left ventriculogram demonstrated decreased left ventricular volume and improved ejection fraction. LVEDV was 122 ml (LVEDVI: 90 ml/m<sup>2</sup>), LVESV was 83 ml (LVESVI: 61 ml/m<sup>2</sup>), and the left ventricular ejection fraction (LVEF) was 32% (Fig.

**Table 1. Hemodynamic data**

	Preoperation	Postoperation	
RA	(5)	(2)	(mmHg)
RV	34 / ~5	33 / ~2	(mmHg)
PA	50 / 19 (33)	26 / 10 (15)	(mmHg)
PCW	(25)	(10)	(mmHg)
LV	183 / ~28	N.A.	(mmHg)
Ao	125 / 75	120 / 72	(mmHg)
CO	3.52	3.79	(L/min)
CI	2.46	2.80	(L/min/m <sup>2</sup> )
LVEDV (I)	203 (144)	122 (90)	(ml) (ml/m <sup>2</sup> )
LVESV (I)	155 (110)	83 (61)	(ml) (ml/m <sup>2</sup> )
EF	24	32	(%)
Lown	IVb	II	
NYHA	III	II	



**Fig. 3.** a: Preoperative chest X-ray showed an enlarged cardiac shadow with 62% of CTR, b: Postoperative chest X-ray showed reduced CTR (57%).

3, Table 1). The patient was discharged two months after surgery with New York Heart Association (NYHA) Class II symptoms. Histological examination of the excluded tissue in the LV showed extensive fibrosis between the residual myocardium, especially in the endocardial side. The aortic valve showed severe calcification and diffuse fibrosis. She remains well with NYHA Class I symptoms for about 2 years of follow up. No arrhythmic events have been identified by holter electrocardiogram.

## Discussion

Large anterior transmural myocardial infarction causes both early and late distortion of the structure of the heart. The post infarction left ventricular scar may result in an akinetic region or occasionally a dyskinetic scar, and will not contract to support cardiac output. Dilatation of the ventricular cavity raises wall tension, produces mechanical disadvantages, and further promotes adverse remodeling. This may cause chronic heart failure, because the contractility of remote non-ischemic muscle is reduced by increased wall tension over time. The EVCPP (Dor operation) technique restores the circular ventricular architecture by excluding the antero-septal scar, reduces the cavity to restore wall tension toward normal by means of the Laplace relationship, and thereby improves hemodynamic status.<sup>3-6</sup> Among several evolving surgical strategies for end-stage heart failure, the EVCPP has become a promising alternative for patients with ischemic cardiomyopathy, who previously have been indicated for

heart transplantation.<sup>9</sup>

In the present case, aortic valve stenosis, which had progressed after the initial CABG procedure, together with ischemic myocardial damage caused severe congestive heart failure. The indication for aortic valve stenosis is to be justified without much controversy. However, EVCPP may be the matter of concern. Studies of patients with aortic valve stenosis and depressed LV function have shown that aortic valve replacement (AVR) improves LV systolic function if LV dysfunction is caused by afterload mismatch, but there has been a cohort of patients who did not improve with AVR.<sup>10</sup> Connolly et al. have reported that change of ejection fraction (EF) after aortic valve replacement was inversely related to coronary artery disease.<sup>11</sup> Therefore, severe ischemic myocardial damage, as seen in the present case, may be the factor precluding functional improvement with AVR in the patients with LV dysfunction. This fact prompted us to perform the Dor procedure with aortic valve replacement. Among 781 cases with EVCPP reported by Dor et al., only 4 cases underwent concomitant aortic valve replacement.<sup>4</sup> However, as shown in the present case, reduction of intraventricular pressure by removing aortic valve stenosis is effective in the setting of ischemic cardiomyopathy. The reduction of wall tension by EVCPP may have further promoted the recovery of systolic function in the non-ischemic area. This fact suggested that aggressive application of AVR may be effective for marginal aortic stenosis in patients with ischemic cardiomyopathy, who otherwise are not indicated for replacement.

The initial postoperative course was complicated with intractable ventricular arrhythmia. As Dor has recommended, cryoablation on the border zone between the scar and viable myocardium was a reasonable option to prevent this complication.<sup>7)</sup> We did not perform this procedure for the following two reasons. First, in the heart with akinetic dilatation, the border zone is not necessarily clear. Secondly, this transitional ventricular arrhythmia immediately after the operation is largely related to hemodynamic instability and resultant dilatation of the ventricular cavity. Therefore, this could be avoided with more appropriate circulatory management. We reported here, a case with aortic valve stenosis with severe LV dysfunction secondary to ischemic cardiomyopathy. Aortic valve replacement combined with EVCPP was effective in this complicated situation.

## References

1. Faxon DP, Myers WO, McCabe CH. The influence of surgery on the natural history of angiographically documented left ventricular aneurysm: the coronary artery surgery study. *Circulation* 1986; **84**: 110.
2. Dor V, Di Donato M, Sabatier M, Toso A, Montiglio F, Maioli M. Efficacy of endoventricular patch plasty in large postinfarction akinetic scar and severe left ventricular dysfunction: comparison with a series of large dyskinetic scars. *J Thorac Cardiovasc Surg* 1998; **116**: 50–9.
3. Dor V, Kreitmann P, Jourdan J. Interest of “physiological” closure (circumferential plasty on contractile areas) of left ventricle after resection and endocardectomy for aneurysm or akinetic zone: comparison with classical technique about a series of 209 left ventricular resections. *J Cardiovasc Surg* 1985; **26**: 73 (abstr).
4. Dor V, Saab-M, Coate P, Sabatier M, Montiglio F. Endoventricular patch plasties with septal exclusion for repair of ischemic left ventricle: technique, results and indications from series of 781 cases. *Nippon Kyoubu Geka Gakkai Zasshi* 1998; **46**: 389–98.
5. DiDonato M, Sabatier M, Dor V, Toso A, Maioli M, Fantini F. Akinetic versus dyskinetic post infarction scar: relation to surgical outcome in patients undergoing endoventricular circular patch plasty repair. *J Am Coll Cardiol* 1997; **29**: 1569–75.
6. Dor V. Reconstructive left ventricular surgery for post ischemic akinetic dilatation. *Semin Thorac Cardiovasc Surg* 1997; **9**: 139–45.
7. Dor V. The treatment of refractory ischemic ventricular tachycardia by endoventricular patch plasty reconstruction of the left ventricle. *Semin Thorac Cardiovasc Surg* 1997; **9**: 146–55.
8. Suma H, Isomura T, Horii T, et al. Dor operation for end-stage ischemic cardiomyopathy. *J Cardiol* 1998; **31**: 165–70.
9. McCarthy PM, Young JB, Starling RC, et al. Anterior infarct exclusion surgery for ischemic cardiomyopathy. *Circulation* 1999; **100** (suppl): 514–5.
10. Rediker DE, Boucher CA, Block PC, et al. Degree of reversibility of left ventricular systolic dysfunction after aortic valve replacement for isolated aortic valve stenosis. *Am J Cardiol* 1987; **60**: 112–8.
11. Connolly HM, Oh JK, Orszulak TA, et al. Aortic valve replacement for aortic stenosis with severe left ventricular dysfunction prognostic indicators. *Circulation* 1997; **95**: 2395–400.