Impact of Apex-Sparing Partial Left Ventriculectomy on Left Ventricular Geometry, Function, and Long-Term Survival of Patients with End-Stage Dilated Cardiomyopathy

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ABSTRACT ***Background and Aim*: Currently, partial left ventriculectomy (PLV) has not been widely accepted as a treatment option for dilated cardiomyopathy (DCM) because its results thus far have been inconsistent. In an animal study, apex-sparing PLV (AS-PLV) was shown to produce greater improvement in left ventricle (LV) function than conventional PLV in which the apex was removed. The aim of this study is to investigate the effectiveness of AS-PLV in a clinical setting. *Patients and Methods:* From September 1999 to December 2007, 13 patients with DCM underwent AS-PLV. Left ventriculotomy was made in the thinnest portion of the lateral wall without injuring the apex, the papillary muscles, and the circumflex coronary artery, which supplies the neighboring myocardium. *Results:* All patients were discharged from the hospital, except for one patient who developed refractory ventricular fibrillation on postoperative day 35. After AS-PLV, the LV diastolic dimension decreased from 71 ± 10 mm to 55 ± 9 mm; LV ejection fraction (EF) from 28% ± 8% to 39% ± 11%; and New York Heart Association (NYHA) class from 3 ± 1.7 to 1.5 ± 0.6; the differences were significant ($p < 0.01$). LV function and geometry remained unchanged 2 years after AS-PLV with LVDD of 60 ± 7 mm, LVEF of 34% ± 8%, and NYHA class of 1.7 ± 0.6, respectively (N.S vs. at discharge). Conclusions: Regardless of the etiology of LV dilatation, AS-PLV restored the ellipsoidal shape of the LV and improved LV function. AS-PLV is a feasible option for treating diseased LVs with lateral wall lesions.**
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Batista et al. introduced partial left ventriculectomy (PLV) for the treatment of patients with dilated cardiomyopathy (DCM).[1] The concept behind this procedure is that reducing the volume of the left ventricle (LV) will decrease the stress on the LV wall in accordance with Laplace's law. However, the clinical outcomes of PLV were not favorable.[2] Then, the intraoperative echo-guided volume reduction test was introduced to determine the site and the extent of PLV, which improved the clinical outcome.[3] Further, the original PLV procedure included the resection of the intact or nearly intact LV apex in addition to the LV lateral wall,[1,2] which might have damaged the continuity of muscle band forming the basal and apical loops,[4] and thus deteriorated the postoperative contractility. On the basis of this assumption, we showed in an animal study that the postoperative improvement in LV function after PLV in which the apex is spared is greater than that after PLV in which the apex is removed.[5] Here, we present the clinical results of apex-sparing PLV (AS-PLV) in patients with DCM.

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PATIENTS AND METHODS

From September 1999 to December 2007, our team performed LV-restoration surgery on 81 patients with DCM (of either ischemic or nonischemic etiology). The preoperative cardiac performance of these patients was examined by echocardiography with color kinesis (HP SONOS 5500; Hewlett-Packard Company, Palo Alto, CA, USA), cardiac catheterization including coronary angiogram, and thallium-scintigraphy for investigating myocardial viability. Our indication of AS-PLV is for patients who had a diffusely and severely hypokinetic LV wall motion with the LV end-diastolic diameter (DD) of larger than 65 mm and LV ejection fraction (EF) of less than 40%, and showed dyskinetic or akinetic laterall wall motion according to the intraoperative echo-guided volume reduction test after establishing cardiopulmonary bypass.[3] A total of 13 patients had

TABLE 1
Preoperative Patient Demographics (n = 13)

Pt. No.	Age	Sex	Dd (mm)	EF (%)	MR	Etiology	Wall Motion
1	69	M	85	26	3	NIC	Severe hypo
2	40	M	85	29	1	NIC	Severe hypo
3	54	M	69	25	3	IC	Severe hypo
4	63	F	57	33	4	NIC	Severe hypo
5	71	M	70	32	3	IC	Akinesis
6	60	M	60	40	1	IC	Akinesis
7	27	M	87	29	1	NIC	Akinesis
8	60	M	82	33	3	NIC	Akinesis
9	62	F	72	26	3	NIC	Severe hypo
10	69	M	70	24	2	IC	Akinesis
11	56	M	67	10	1	IC	Dyskinesis
12	68	M	71	37	4	IC	Akinesis
13	74	M	55	42	1	IC	Severe hypo

Pt. No. = patient number; Dd = left ventricle end-diastolic dimension (mm); EF = ejection fraction (%); MR = mitral regurgitation; NIC = nonischemic cardiomyopathy; IC = ischemic cardiomyopathy; Wall Motion = wall motion of the diseased lateral LV wall; hypo = hypokinesis.

AS-PLV (Table 1); six of these 13 patients were diagnosed with nonischemic DCM. LV lateral regional wall motion before PLV was classified as dyskinetic in one patient, akinetic in six patients, and severely hypokinetic in six patients. Three patients underwent preoperative intraaortic balloon pumping.

Under general cardiac anesthesia and monitoring, cardiopulmonary bypass was installed via ascending aortic cannulation and double venous cannulation. During the operation, LV wall motion was grossly examined, and the site for PLV was determined using transesophageal echocardiography (TEE) according to the echo-guided volume reduction test under on-pump beating heart without aortic cross-clamp.[3] Left ventriculotomy was performed at the thinnest and most dyskinetic or akinetic portion of the lateral wall with the LV cavity sufficiently vented. Care was taken not to injure the papillary muscles (PMs) and the coronary artery branches that supplied the intact myocardium. Once left ventriculotomy was performed, the LV wall was thoroughly inspected and manually palpated to precisely identify the extent of excision. The diseased LV wall between both PM bases was then excised from 2-cm distal to the atrioventricular grove to the area just proximal to the LV apex, thus sparing the LV apex (Fig. 1). The LV walls were directly closed in two layers with 0 monofilament suture with Teflon felt strips in an interrupted fashion and 2-0 polypropylene suture in a continuous fashion. Concomitantly performed procedures are shown in Table 2. PLV was performed under on-pump beating heart without aortic cross clamp, while concomitant procedures were performed under cardiac arrest with antegrade warm blood cardioplegia.

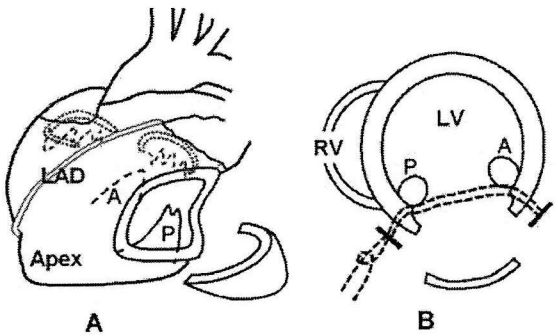

Figure 1. *Schematic illustration of AS-PLV. (A) Overview: the lateral wall of the LV between the PM bases is excised. (B) Cross-sectional view: the thin LV wall between the PM bases is excised, and the LV wall is directly closed with the LV apex intact. A = anterior PM; P = posterior PM.*

STATISTICAL ANALYSIS

All values are expressed as the mean ± standard deviation. Statistical analysis was performed to compare preoperative and postoperative variables by using the Wilcoxon signed rank test (StatView®; SAS Institute Inc., Cary, NC, USA). A p-value < 0.05 was considered statistically significant. Cumulative survival rates were calculated using the Kaplan-Meier curve.

RESULTS

The operative procedures are summarized in Table 2. Hospital mortality was 7% (1/13 patients); all patients were discharged from the hospital in good condition, except for one patient who developed refractory

TABLE 2
Concomitant Operative Procedures (Number of Patients = 13)

Coronary artery bypass	5
Mitral valve repair	12
Replacement	1
Repair	10
Alfieri	2
Tricuspid valve repair	5
Replacement	1
Repair	4

TABLE 3
Change After AS-PLV

	Before OP	After OP	P-Value
Echocardiography			
DD (mm)	71 ± 10	55 ± 9	<0.01
DS (mm)	60 ± 10	45 ± 9	<0.01
EF (%)	28 ± 8	39 ± 11	<0.01
Degree of MR	2.4 ± 1.1	0.8 ± 0.7	<0.01
Tenting depth (mm)	10.3 ± 3.7	4.3 ± 1.2	<0.01
NYHA	3 ± 1.7	1.5 ± 0.6	<0.01
BNP concentration	835 ± 686	209 ± 149	<0.05

OP = operation; DD = left ventricular end-diastolic diameter; DS = left ventricular end-systolic diameter; EF = left ventricular ejection fraction; MR = mitral regurgitation; NYHA = New York Heart Association functional class; BNP = brain natriuretic peptide.

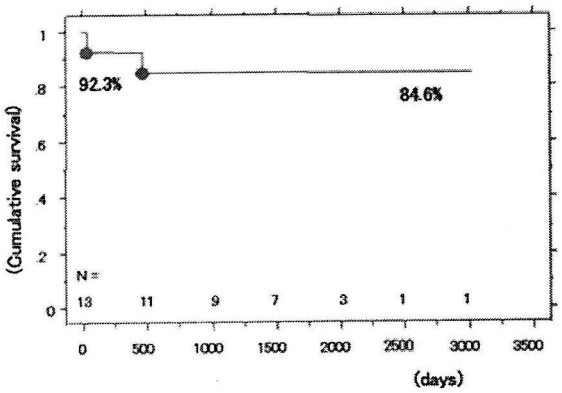

Figure 3. *Cumulative survival after AS-PLV in 13 patients.*

ventricular fibrillation on postoperative day 35. To note, the 12 survivors showed a significant improvement in LVDD, LVEF, mitral tenting depth, degree of mitral regurgitation (MR), New York Heart Association (NYHA) class, and brain natriuretic peptide levels after the operation ($p < 0.01$) (Table 3). Left ventriculography showed that in all patients, the LV had become ellipsoidal after AS-PLV (Fig. 2).

The 1 and 5-year survival rates were 92% ± 8% and 84% ± 15%, respectively (Fig. 3), with a mean follow-up period of 1523.2 ± 885.9 days and a follow-up rate of 100%. One patient died of pneumonia 1 year after operation. The remaining 11 surviving patients had no adverse cardiac events after surgery. Echocardiographic and functional data obtained 2 years after the operation showed LVDD of 60 ± 7 mm, LVEF of 34% ± 8%, and NYHA class 1.7 ± 0.6, which were not significantly different from the values recorded at discharge.

DISCUSSION

The main finding of this report is that AS-PLV can improve LV function with favorable short- and long-term survival; further, the LV becomes more ellipsoidal after this procedure.

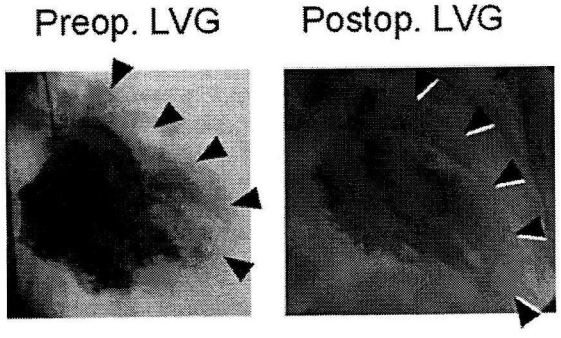

Figure 2. *Left ventriculography at end-systole before (left panel) and after (right) AS-PLV. Note that the LV has recovered its ellipsoid and natural shape after the surgery.*

Batista et al. first reported the PLV technique and its clinical outcomes: 30-day mortality; 22% and two-year survival rate, 55%[1]; these initial outcomes were not satisfactory. In our series, improvement in the outcome of PLV presented several challenges.

First, we preserved the apex while performing PLV. Torrent-Guasp et al. reported that ventricular myocardium exists as a continuous muscle band, which is oriented spatially as a helix formed by basal and apical loops. The basal loop is constituted by right and left ventricular free walls with muscle fibers wrapped around horizontally, and the apical loop is a twisted loop constituted by a descending segment and an ascending segment, the former forming the LV lateral wall including both anterior and posterior papillary muscle and the latter forming the ventricular septum. With apex-sacrificing PLV, this muscle band may result into two separate muscle band, while AS-PLV potentially retains the continuity of this muscle band (Fig. 4).[4] Indeed, in our experimental study we found that unlike apex-sacrificing PLV, AS-PLV improved the fractional area change in the basal, middle, and apical parts of the LV. This indicates that preservation of this muscle band during AS-PLV is beneficial for improving postoperative cardiac function[5] and for ensuring that after the procedure, the LV is ellipsoidal in shape. Considering the importance of this muscle band, AS-PLV may be more beneficial than apex-sacrificing PLV with regard to cardiac structure and function.

The second step that we adopted to improve the outcome of PLV was the prevention of injury to viable PMs while performing AS-PLV. We previously reported that ischemic MR is associated with increased separation of the PM tips; our technique reverses this separation, thereby eliminating the cause of MR.[6] Decreasing the LV size while maintaining valvular-ventricular continuity and geometry by sparing the PMs and the above-mentioned muscle band can restore LV contraction.

Third, PLV was performed on severely hypokinetic or akinetic lateral walls. Conventional LV-restoration surgery, particularly that in the patients with ischemic DCM, can be described as a scar excision. In most of our patients, the lateral wall of the LV was either

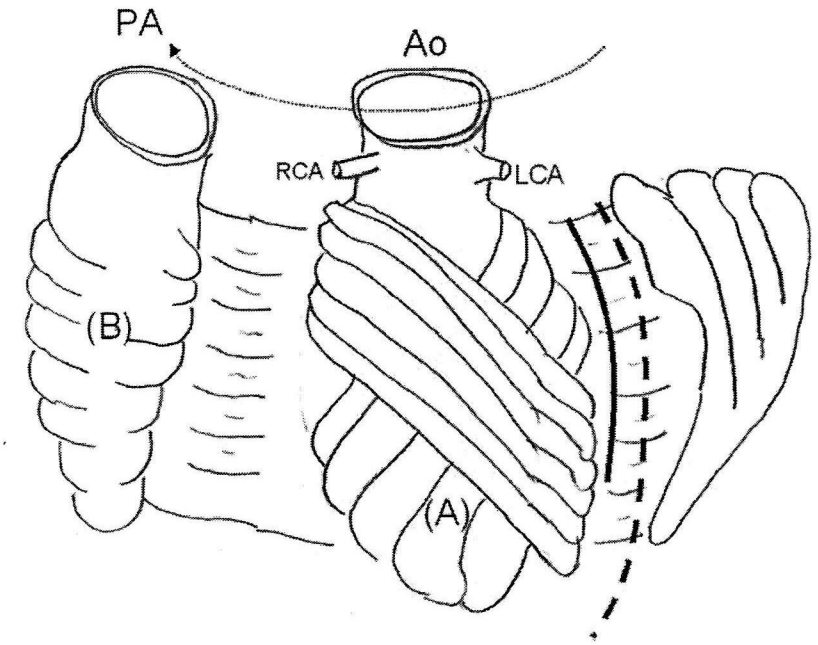

Figure 4. *Schematic presentation of two ventricular myocardial band, comprised of apical loop (A) and basal loop (B). The pulmonary artery (PA) has been separated and shifted leftward as a part of basal loop (small dotted arrow). The incision lines of apex-sacrificing PLV (large dotted line) and AS-PLV (solid line) were shown. The difference of incision extent may indicate that AS-PLV is more beneficial in preserving the integrity of ventricular muscle band. Ao = aorta; PA = pulmonary artery; LCA = left coronary artery; RCA = right coronary artery.*

akinetic or severely hypokinetic and might have contained some viable myocardium, as confirmed by intraoperative echocardiography. In patients with nonischemic DCM, the LV contractility is diffusely impaired, and the lateral LV wall thus is more or less still functioning. Therefore, the benefits of PLV may be explained by Laplace's law rather than by the loss of largely nonviable myocardium; we strongly believe that the favorable effects of AS-PLV in this series were due not to scar excision but due to decreased stress on the LV wall, as explained by Laplace's law.

With regard to the long-term outcome, one patient developed pneumonia at one year after AS-PLV; the remaining 11 showed uneventful postoperative courses. Our results showed better long-term survival after LV-restoration surgery than those in other reports from several international centers,[1,7] which indicates that sparing the LV apex while performing LV-restoration surgery may improve long-term survival.

There are several limitations of this study. First, very few patients were included. Second, the underlying etiology varied among patients. Third, we did not compare AS-PLV with apex-sacrificing PLV because the patients were too sick preoperatively to permit randomization.

CONCLUSIONS

After AS-PLV, the LV became ellipsoidal, and the LV function and survival improved, regardless of the etiology of cardiomyopathy or the degree of impairment of regional contractility. AS-PLV warrants further investigation and may be a feasible modification of PLV.

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