Early surgical anteroseptal ventricular endocardial restoration after acute myocardial infarction. Pathophysiology and surgical considerations

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Background. **The efficacy and safety of surgical anteroseptal ventricular endocardial restoration (a procedure that excludes non-contracting scarred segments) in the left ventricle with chronic dilation and remodeling secondary to an anterior myocardial infarction are well established. We present a small series and discuss the indication for early left ventricular restoration in the setting of complicated acute myocardial infarction.**

Methods. **Early ventricular restoration was performed in 8 patients (4 males, 4 females, mean age 70 ± 8 years). A postinfarction ventricular septal defect was diagnosed in 3 cases. All patients were operated, on an urgent or emergent basis, between 1 and 16 days following the onset of infarction. Surgical coronary revascularization was associated in 7 patients.**

Results. **There was one operative death. At discharge, echocardiographic morphofunctional assessment revealed: a reduction of the left ventricular end-diastolic and end-systolic volume indexes, an increase of the ejection fraction, and, most importantly, an improvement of remote myocardial shortening fraction. At a mean follow-up of 15.6 months (range 2-21 months), there were no late deaths and all survivors are in NYHA functional class I or II.**

Conclusions. **Left ventricular restoration may represent an effective adjunct to the surgical management of patients with an acute extensive anterior myocardial infarction complicated by severe heart failure, with or without septal rupture.**

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"The future has an ancient heart"
Carlo Levi (1902-1975), writer, painter,
graduate in medicine

Introduction

The efficacy and safety of surgical anteroseptal ventricular endocardial restoration (SAVER), or endoventricular patch plasty, are well established in the treatment of chronic dilation and extensive scarring of the left ventricle following an anterior myocardial infarction (MI)[1,2]. This procedure excludes non-contracting segments of the septum and free wall by the placement of an endoventricular patch sewn along the border between the fibrous tissue and healthy muscle. Successful SAVER in the setting of complicated acute MI has been described by a few authors[3,4], but has not been definitively validated.

In this report, we discuss our experience with early SAVER in 8 patients with a complicated acute MI and a failing left ventricle.

Methods

Since January 2000, SAVER was performed in 8 patients who met the following criteria: severe cardiac failure (cardiogenic shock 4/8 patients; severe congestive heart failure 4/8) and early remodeling with dilation of the left ventricular (LV) cavity at echocardiography and/or ventriculography following an acute anterior MI (1 to 16 days from onset). A postinfarction ventricular septal defect (VSD) was diagnosed in 3 cases. The patients' characteristics are reported in table I.

At the onset of MI, 4 patients, 2 of whom underwent rescue percutaneous revascularization of the left anterior descending coronary artery, received systemic thrombolysis. Because of hemodynamic instability, 4/8 patients were operated upon on an emergency basis. In such cases, intra-aortic balloon pump support was started preoperatively.

At surgery, all patients were given warm induction followed by cold blood intermit-

Table I. Preoperative data.

Patient	Sex	Age (years)	Cardiac failure	LV pathology	IABP	PAP (s/d/m) (mmHg)	Days from MI onset
1	M	60	CHF	Dyskinesia	No	45/19/31	14
2	F	69	Shock	VSD	Yes	60/27/38	4
3	M	61	CHF	Dyskinesia	No	40/18/29	12
4	F	69	Shock	VSD	Yes	51/21/33	2
5	M	70	Shock	VSD	Yes	55/23/35	1
6	M	83	Shock	Global dilation	Yes	59/25/36	3
7	F	74	CHF	Dyskinesia	No	44/20/31	16
8	F	78	CHF + angina	Dyskinesia	No	41/19/30	15

CHF = congestive heart failure; IABP = intra-aortic balloon pump; LV = left ventricular; MI = myocardial infarction; PAP = pulmonary artery pressure (systolic/diastolic/mean); VSD = ventricular septal defect.

tent antegrade/retrograde cardioplegia and substrate-enriched controlled reperfusion, according to Buckberg's principles[5]. Coronary revascularization was usually addressed first. Only in the presence of LV thrombosis at intraoperative transesophageal echocardiography, was the left ventricle opened at the beginning of the procedure to perform thrombectomy. For left ventriculotomy the incision was first started close to the apex, and then extended anteriorly and inferiorly parallel to the interventricular sulcus, in order to allow clear inspection of the entire LV chamber. The infarcted muscle margins, and, if present, the VSD were located. Encircling sutures, such as the Fontan stitch[6], were never employed to demarcate the separation between viable muscle and the infarction. An oval glutaraldehyde-fixed autologous pericardial patch was tailored (3-4 × 4.5-5 cm) and anchored by means of a deep continuous 3-0 polypropylene double suture to the border of the non-infarcted endocardium, starting from the interventricular septum and completing the suture into the antero-lateral and infero-apical LV free walls. At the point where the anterior wall joins the septum, the continuous suture of the patch was interrupted and transmural "U" stitches with teflon pledgets on the epicardial side were applied at the superior margin of the ventriculotomy, i.e., where the suture is more cumbersome. In the presence of a VSD, the patch was sutured in the septum distant from the rupture, thus excluding all necrotic muscle and the VSD from the LV cavity. The ventriculotomy was closed with a double suture over two strips of teflon felt (the first suture consisted of interrupted horizontal mattress whereas the second was a continuous suture) reapproximating the residual non-contractile ventricular muscle over the pericardial patch. Proximal anastomoses of saphenous grafts were performed on a side-biting clamp after aortic unclamping and the operation was completed according to standard technique.

Results

Surgical coronary revascularization was performed in 7/8 patients (1 had widespread non-graftable coronary disease); the left anterior descending coronary artery was grafted in 5/8 patients, while 2/8 were submitted to rescue coronary angioplasty with a good angiographic result. There was one in-hospital death related to intraoperative refractory cardiac failure (Fig. 1). All the remaining patients were weaned from cardiopulmonary bypass with inotropic support including intravenous epinephrine infusion. Intra-aortic balloon pump was used only in patients who needed this support before the operation (4/8) with a postoperative duration of 32 ± 18 hours. Assisted ventilation was prolonged (> 48 hours) in 4/7 cases, and transient renal failure was observed in 2/7. The mean postoperative stay in the Intensive Coronary Care Unit was 5.4 ± 1.5 days.

Preoperative and pre-discharge echocardiography showed a reduction of the LV end-systolic and end-diastolic volume indexes, an increase of the LV ejection fraction and an improvement of the shortening fraction in the remote myocardium (Table II).

The patients' follow-up after discharge ranged from 2 to 21 months (15.6 ± 5.4 months). There were no late deaths and all survivors are in NYHA functional class I or II.

Discussion

Pathophysiology. The LV dilation that follows an acute MI with extensive loss of contractility is initially an adaptive mechanism[7]. In about 20% of cases, LV remodeling severely impedes the systolic function during the first weeks after an extensive anterior MI. If this process is rapidly progressive, it leads to (sub)acute heart failure or cardiogenic shock, with high mortality rates[8]. Secondary papillary muscle dislocation determining restricted mitral leaflet coaptation with secondary valve regurgitation[9] and, more rarely, a VSD or an acute aneurysm (that may precede free wall rupture) worsens the patient's conditions. After an acute MI, the 30-day and 1-year mortality rates increase as LV dilation progresses, with the LV end-systolic volume index being a very important predictor of prognosis[10-12].
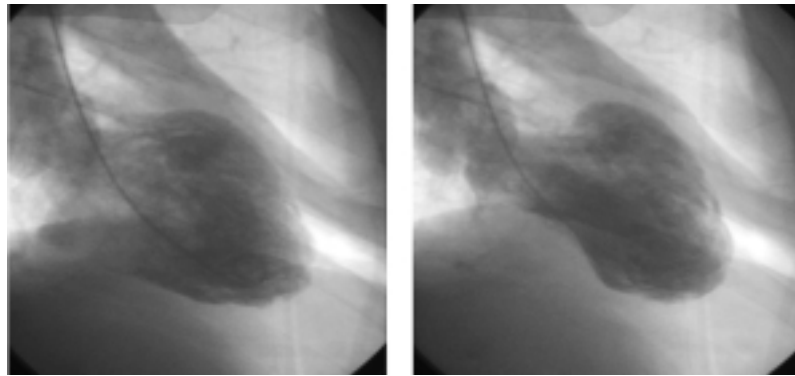
Figure 1. *Irreversible left ventricular damage or surgery too late? An 83-year-old man who developed cardiogenic shock 7 hours after the onset of an anterior myocardial infarction. Hemodynamic evaluation showed severe proximal left anterior descending coronary artery disease, widespread left ventricular akinesia and dilation (ejection fraction 20%, left ventricular end-systolic volume index 45.3 ml/m^2) and a markedly reduced cardiac index (1.8 l/min/m^2). Echocardiography revealed severe left ventricular diastolic dysfunction (E/A ratio 2.7). Intra-aortic balloon pump was started and successful rescue coronary angioplasty to the left anterior descending coronary artery was performed. After 48 hours there was no hemodynamic improvement and the patient underwent surgical anteroseptal ventricular endocardial restoration. The outcome was unsuccessful.*

Table II. Pre- and postoperative (at discharge) echocardiographic data.

LVEDVI (ml/m^2)		LVESVI (ml/m^2)		EF (%)		SF (%)	
Pre	Post	Pre	Post	Pre	Post	Pre	Post
74.3 ± 19.5	57.1 ± 13.2	51.6 ± 10.9	35.7 ± 5.9	26.3 ± 5.8	42.8 ± 2.9	28.1 ± 4.5	40.4 ± 2.1

EF = ejection fraction; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; SF = shortening fraction.

In the absence of mechanical complications, the standard treatment of cardiogenic shock complicating an acute MI includes intra-aortic balloon pump and emergency coronary angioplasty or bypass surgery. This approach improves survival, especially when performed within 24 hours of the onset of shock[13]. However, the 30-day mortality approximates 50% for patients in cardiogenic shock treated with revascularization alone (percutaneous or surgical)[13,14].

Reports concerning LV reconstruction with SAVER include only a few urgent or emergent operations, and the specific clinical setting and outcome are generally not described[15,16]. Dor et al.[15] report 11 such patients, 2 of whom with an associated VSD. Patients undergoing surgery in the acute phase after MI were also included in the multicenter SAVER study, but the outcome of this subgroup is not specified[2]. A small series of 6 patients, who developed cardiogenic shock after LV dilation in the acute phase following an anterior MI, has been recently reported[4]. All patients underwent successful SAVER associated with coronary revascularization. Since 1987, SAVER has also been performed during the acute phase of MI for the treatment of a VSD[3,17,18]. The traditional surgical approach consisting of infarctectomy and reconstruction of the ventricular septum and LV free wall with one or two prosthetic patches is associated with a high operative mortality[17,19,20]. David and Armstrong[18] report a 23% operative mortality in 26 patients with an acute anterior VSD, mostly in cardiogenic shock, who underwent SAVER between 1 and 21 days after MI. The authors related the improved clinical results to the pathophysiologic advantages of LV restoration and to the fact that SAVER leaves the right ventricular geometry unchanged, avoiding additional damage[3]. With regard to this, the right ventricle has been reported to be involved in the necrotic process in virtually all patients with a VSD[21], with severe right ventricular dysfunction often correlating with an adverse outcome[3,18].

We performed SAVER in 8 patients with an acute (1 to 16 days from onset) anterior MI complicated by severe cardiac failure (cardiogenic shock in 4/8; congestive heart failure unresponsive to medical therapy in 4/8; a VSD in 3/8). SAVER was successful in 7 cases. One patient with a very extensive infarction, no VSD, global LV dilation, and severe systo-diastolic LV dysfunction (ejection fraction 20%, E/A ratio 2.7) could not be weaned from cardiopulmonary bypass (Fig. 1).

In the setting of a severely complicated acute MI (i.e., severe cardiac failure with extensive loss of contractility, LV dilation and an altered LV geometry), SAVER appears to allow for additional benefits determining an improved LV performance, if compared to coronary revascularization alone. Various mechanisms have been advocated to explain such benefits:

• SAVER eliminates dyskinesia and reduces the akinetic region to the size of the patch;
• SAVER changes the spherical LV geometry to a more physiologic elliptical shape and realigns the muscle fiber orientation, thus allowing for a more effective systole and also for a reduction of the free wall stress and oxygen consumption that contributes to enhance the remote muscle contractility[22,23];
• when a VSD is present, SAVER obviously couples the elimination of a shunt and the functional advantages of LV reconstruction.

The usefulness of this strategy to avoid progressive LV dilation and global dysfunction in the medium or long term remains unknown. Nevertheless, preliminary experience appears to support SAVER as a surgical option for the treatment of cardiac failure secondary to acute post-infarction ventricular dysfunction and dilation unresponsive to conventional therapy.

Surgical strategy. In chronic patients, SAVER is a straightforward and reproducible technique. In the particular setting of acute MI, additional technical difficulties are related to the handling of a recently necrotic and fragile myocardium. We recommend intracardiac repair on the arrested heart through an extensive ventriculotomy to minimize the risk of patch dehiscence. In this respect, we strongly recommend that the Fontan encircling stitch not be used, in order to avoid traction on the endocardial surface.

There is consensus in the literature that in order to rebuild a more elliptical LV cavity, the patch must be oval in shape. The orientation of the patch is not the surgeon's choice, because sutures should be placed on viable muscle, often very high on the septum, resulting in an oblique direction of the patch with the creation of a new LV apex. The sizing and tailoring of the patch in the setting of acute MI, with a relatively small left ventricle, is very important to avoid the creation of a restrictive LV cavity, which may result in catastrophic diastolic dysfunction[24]. Dor et al.[1] suggest the use of an oval intraventricular balloon filled with 40-50 ml/m^2 of saline solution to determine the suture margins of the patch. We believe that the size of the patch should be related to the degree of LV dilation. In the present series, we observed much smaller preoperative LV volumes (LV end-systolic volume index 51.6 ± 10.9 ml/m^2) than those of the series of patients undergoing SAVER in the chronic setting described in the literature (LV end-systolic volume index 109 ± 71 ml/m^2)[2]. Besides, the patches were larger (3-4 $\times$ 4-4.5 cm) than those usually implanted in chronic patients (2 $\times$ 3 cm)[2]. David et al.[3,18] report the use of even larger patches (4 $\times$ 6 cm) in patients with an acute MI complicated by an anterior VSD undergoing SAVER.

Dacron patches have been largely used in the chronic setting[25], but we prefer pericardium[3,17,18] because it allows less traumatic suturing on acutely infarcted tissues. David and Armstrong[18] and Cooley[17] use glu-

taraldehyde-fixed bovine pericardium. Since 1988 we used glutaraldehyde-fixed autologous pericardium[26] without observing any rebulging or calcification at follow-up.

In conclusion, ventricular reconstruction in the acute phase of MI by exclusion of necrotic segments, the so-called SAVER, represents a surgical challenge. The results of our preliminary experience confirm the feasibility of SAVER, with an acceptable operative mortality and a good early outcome, in the treatment of patients with cardiogenic shock or refractory congestive heart failure, with or without a VSD. In our opinion, the technical reproducibility of SAVER in the presence of acutely infarcted muscle is best addressed with cardioplegic arrest. The definition of a clear indication for SAVER in the acute period following MI is very difficult. We performed the operation, primarily on the basis of the clinical picture, in patients with refractory heart failure secondary to an extensive anterior MI and a LV end-systolic volume index > 50-60 ml/m^2. Further experience is needed to establish whether SAVER may prevent the long-term morphofunctional sequelae of postinfarction LV remodeling.

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