Clinical Investigation

Left Ventricular Aneurysmal Repair within 30 Days after Acute Myocardial Infarction

Early and Mid-Term Outcomes

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For safe resection, left ventricular aneurysmal repair after acute myocardial infarction is usually delayed. However, delaying surgery may not be possible or prudent in some patients who are clinically unstable after acute myocardial infarction.

We retrospectively reviewed the early and mid-term outcomes of left ventricular aneurysmal repair in patients who had experienced acute myocardial infarction <30 days before the repair. From September 2001 through May 2006, 127 consecutive post-infarction patients underwent concurrent anteroapical left ventricular aneurysmal repair and coronary artery bypass grafting. In Group I (38 clinically unstable patients), the surgery was performed <30 days after myocardial infarction. In Group II, 89 patients underwent the surgery ≥30 days after infarction. The mean follow-up period was 26.16 ± 16.41 months.

One Group I patient (2.6%) died in the hospital due to graft-versus-host reaction. Three Group II patients (3.4%) died: 2 of low cardiac output and 1 of multiple-organ failure. Hospital mortality rates were not statistically significant between groups (P=0.582). All patients required similar perioperative inotropic support, intra-aortic balloon pump support, and re-exploration for bleeding or cardiac tamponade. The actuarial survival rates were 94.7% (Group I) and 94.4% (Group II). Postoperative New York Heart Association functional class improved similarly in both groups.

We infer that left ventricular aneurysmal repair with coronary revascularization <30 days after a recent myocardial infarction is a feasible procedure, with acceptable morbidity and mortality rates. Our mid-term results were comparable with those for patients who underwent this surgery ≥30 days after acute myocardial infarction. ***(Tex Heart Inst J 2007;34: 154-9)***

Key words: *Cardiac surgical procedures/methods/mortality; heart aneurysm/complications/mortality/pathology/surgery; heart ventricles/pathology/surgery; myocardial infarction/complications/surgery; reconstructive surgical procedures; retrospective studies; risk factors; survival analysis; suture techniques; time factors; treatment outcome; ventricular dysfunction, left/surgery*

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Left ventricular aneurysm (LVA) is a common complication of acute myocardial infarction (AMI). The 1st successful LVA resection was reported by Likoff and Bailey in 1954.[1] Since then, clinicians have applied various surgical methods of LVA repair, with geometric ventricular reconstruction the goal. In 1985, Jatene[2] described the circular reconstruction of LVA. Modifications of this technique were endoventricular circular plasty, developed by Dor and colleagues,[3] and endoaneurysmorrhaphy, developed by Cooley.[4] Surgeons generally prefer to delay LVA resection at least 3 months after AMI in order to enable maturation of the scarred tissue.[5] However, in some clinically unstable patients, delaying surgery is not possible or prudent, because their symptoms resist even maximal medical management.

Limited data appear in the English-language medical literature regarding the safety and efficacy of performing surgical ventricular repair early after AMI.[5-7] In this retrospective study, we evaluated the effects of LVA repair and concurrent coronary artery bypass grafting (CABG) on early and mid-term outcomes in patients with recent AMI, which we defined as an AMI experienced <30 days before the patients underwent the surgery.

Patients and Methods

From September 2001 through May 2006, 127 consecutive patients underwent concurrent anteroapical LVA repair and CABG at our institution. A computerized clinical database was used for retrospective review of the details of each patient's case. In

38 patients (29.9%) who had presented with clinical instability after AMI, the surgery had been performed <30 days after the AMI (Group I). The other 89 patients (70.1%) had undergone the surgery ≥30 days after AMI (Group II). Preoperative coronary angiography was performed in all patients. The diagnosis of LVA was made on the basis of left ventricular (LV) angiographic appearance and was then confirmed intraoperatively. Indications for surgery were angina pectoris, heart failure, cardiogenic shock, or a combination of these. Surgery was elective in all but 1 patient.

Table I shows the preoperative data and clinical characteristics of both groups of patients. The mean age and sex distributions were similar in both groups. Significantly fewer Group I patients were smokers than were Group II patients (44.5% vs 66.3%, respectively; $P=0.023$). There was no significant difference in the incidence of diabetes, hypertension, obesity (body mass index, ≥30 kg/m^2), chronic obstructive pulmonary disease, hyperlipidemia, cerebrovascular accidents, carotid artery disease, or renal dysfunction, or in the percentage of patients who had previously undergone percutaneous transluminal coronary angioplasty. The extent of coronary artery disease, and the LV ejection fraction and LV end-diastolic pressure, were similar in both groups. Significantly fewer Group I patients had stable angina (57.8% vs 79.8%; $P= 0.016$), but significantly more had unstable angina (36.8% vs 14.6%; $P=0.008$), and significantly more were in New York Heart Association (NYHA) functional classes III and IV (65.7% vs 54%; $P=0.048$). Two Group I and no Group II patients had left main coronary artery disease ($P=0.029$).

TABLE I. Preoperative Data and Clinical Characteristics of Both Patient Groups

	Group I (AMI <30 Days) No. (%)	**Group 2** (AMI ≥30 Days) No. (%)	***P* Value**
Number of patients	38	89	–
Mean age (yr)	59.9 ± 8.8	61.6 ± 10.7	0.393
Female	10 (26.3)	19 (21.3)	0.541
Risk Factors			
Smoking history	17 (44.5)	59 (66.3)	0.023*
Diabetes mellitus	9 (23.7)	18 (20.2)	0.663
Hypertension	9 (23.7)	23 (25.8)	0.798
Obesity (BMI, ≥30 kg/m^2)	5 (13.2)	11 (12.4)	0.798
COPD	3 (7.9)	18 (20.2)	0.087
Hyperlipidemia	15 (39.5)	24 (27.0)	0.162
Family history	9 (23.7)	18 (20.2)	0.663
Clinical Status			
Stable angina	22 (57.8)	71 (79.8)	0.016*
Unstable angina	14 (36.8)	13 (14.6)	0.008*
NYHA functional class III–IV	25 (65.7)	48 (54.0)	0.048*
Cardiogenic shock	1 (2.6)	–	0.299
Extent of Coronary Artery Disease			
1-vessel	7 (18.4)	26 (29.2)	0.204
2-vessel	15 (39.5)	30 (33.7)	0.534
3-vessel	16 (42.1)	33 (37.1)	0.594
Left main coronary artery	2 (5.3)	–	0.029*
Previous PTCA	4 (10.5)	4 (4.5)	0.200
Cerebrovascular accident	–	2 (2.2)	0.352
Renal dysfunction	1 (2.6)	3 (3.4)	0.827
Carotid stenosis ≥30%	6 (15.8)	18 (20.2)	0.559
Mean LV ejection fraction	0.38 ± 0.07	0.40 ± 0.09	0.144
Mean LVEDP	19 ± 6.6	18.4 ± 6.2	0.622

*Statistically significant

Means are expressed as mean ± SD.

AMI = acute myocardial infarction; BMI = body mass index; COPD = chronic obstructive pulmonary disease; LV = left ventricular; LVEDP = left ventricular end-diastolic pressure; NYHA = New York Heart Association; PTCA = percutaneous transluminal coronary angioplasty

Patients who underwent combined procedures that did not include CABG were excluded from the study.

Surgical Technique

Linear repair was performed in 21 Group I patients (55.3%), and patch endoaneurysmorrhaphy was performed in 17 (44.7%). In Group II, there were 52 linear repairs and 37 endoaneurysmorrhaphies (58.4% and 41.6%, respectively).

The standard surgical approach included general anesthesia and conventional median sternotomy. A membrane oxygenator was used for cardiopulmonary bypass (CPB). The patients were cooled to 28 to 32 °C and kept at that temperature during CPB. After the institution of CPB, the shape of the LV cavity was carefully inspected, and the diagnosis of LVA was confirmed visually. A retrograde-cardioplegia catheter was introduced under partial venous occlusion after aortic cross-clamping. For myocardial preservation, cold-blood cardioplegic solution (ratio of blood to crystalloid cardioplegic solution, 4:1) was administered at 10 °C via the MYOtherm XP® Cardioplegia Delivery System (Medtronic, Inc.; Minneapolis, Minn). After 500 mL of the solution was infused in antegrade fashion, the same volume was infused retrograde via the coronary sinus until cardiac arrest was established. Subsequently, retrograde cold-blood cardioplegic solution was administered every 20 minutes.

In the Group I patients, it was observed that intraventricular thrombi did not usually adhere to the aneurysmal walls. Therefore, the surgeons avoided manipulating the LV before applying the aortic cross-clamp, and the retrograde-cardioplegia catheter was introduced after cross-clamping in order to prevent embolic events that could arise from thrombi dislodged from the LV during catheter insertion.

Ventricular Repair

Ventricular repair was performed first. The ventricle was opened parallel to the left anterior descending coronary artery at the center of the scarred myocardium, and any clots were carefully removed. The junction (border zone) between the scarred and the viable endocardium was identified. (Of note: determining the location of a border zone can be difficult in patients who have recently experienced an AMI, such as those within our Group I. Following the definition of the thinned and friable ventricular wall is useful as a guideline.)

Linear Repair in Group II Patients. To approximate the free edges of the LV for linear repair, our surgeons used mattress sutures that were reinforced on either side with Teflon felt strips. They completed the repair by adding a 2nd layer of over-and-over suture.

Linear Repair in Group I Patients. The Group I patients were treated in the same fashion as the Group II patients, except that to prevent tearing of the unscarred and friable myocardial tissue, our surgeons placed an additional Teflon strip over the free edges of the LV before suturing the 2nd layer.

Endoaneurysmorrhaphy in Group II Patients. Using a running 2-0 polypropylene suture, our surgeons sutured an oval Teflon felt patch (diameter, 2–2.5 cm) at the border zone, then excised the excess aneurysmal wall and brought the edges of the LV over the patch by the use of a running suture.

Endoaneurysmorrhaphy in Group I Patients. A larger oval Teflon patch (diameter, 2.5–3 cm) was implanted with separate, interrupted U stitches, each reinforced with Teflon strips. These stitches were placed in sequence through the patch, the ventricular wall, and the Teflon strips, with the strips outside the wall. The ventriculotomy edges were then approximated with a continuous suture that was reinforced with an additional Teflon strip, as in the linear repair in the Group I patients.

Coronary Artery Bypass Grafting

After the LVA repair, the distal and proximal anastomoses were performed during the same period of aortic cross-clamping. In all but 2 patients, the left internal thoracic artery was used to revascularize the left anterior descending coronary artery. Before the removal of the cross-clamp, 500 mL of terminal warm-blood (hot shot) cardioplegic solution was administered. The patients were warmed to a core temperature of 36 °C before being weaned from CPB.

Follow-Up

Echocardiography was performed on all patients within the 1st postoperative month. Subsequent follow-up data were obtained from hospital records and from telephone contact with the patients or their referring physicians. Follow-up was possible with 116 of the 127 patients (91.1%).

Statistical Analysis

Statistical analysis was performed using SPSS for Windows, version 10.0 (SPSS Inc.; Chicago, Ill). The data for patient characteristics and outcomes were expressed either as percentages or as mean ± SD. Continuous variables were compared by the use of a 2-tailed *t* test (paired or unpaired, as was appropriate). Estimated actuarial survival curves were constructed using the Kaplan-Meier method (confidence limits, 95%). The statistical significance of the difference between the actuarial survival curves was examined by use of the log-rank test. A P value <0.05 was considered statistically significant.

Results

Operative factors and early clinical outcomes are summarized in Table II. The rates of linear repair and endoaneurysmorraphy were similar in both groups. There

TABLE II. Operative and Early Clinical Outcomes in Both Patient Groups

	Group I (AMI <30 Days) No. (%)	Group 2 (AMI ≥30 Days) No. (%)	*P* Value
Left ventricular thrombectomy	13 (34.2)	42 (47.2)	0.176
Aneurysmectomy type			0.470
Linear repair	21 (55.3)	52 (58.4)	–
Endoaneurysmorraphy	17 (44.7)	37 (41.6)	–
Coronary artery bypass grafting			
Arterial grafts only	12 (31.6)	23 (25.3)	0.508
Left internal thoracic artery grafts	38 (100)	87 (97.8)	0.352
Mean number of grafts per patient	2.74 ± 1	2.43 ± 0.95	0.101
Mean CPB time (min)	114.7 ± 28.5	110.5 ± 30.8	0.478
Mean aortic cross-clamp time (min)	91.4 ± 22.8	88.4 ± 25.8	0.536
Mean mechanical ventilation time (hr)	8.6 ± 3.3	8.8 ± 7.3	0.915
Inotropic support	13 (34.2)	27 (30.2)	0.667
Intra-aortic balloon pump	2 (5.3)	4 (4.5)	0.629
Atrial fibrillation	5 (13.2)	16 (18)	0.503
Re-exploration for bleeding or tamponade	2 (5.3)	3 (3.4)	0.616
Neurologic complications	–	1 (1.1)	0.352
Gastrointestinal complications	1 (2.6)	–	0.299
Renal failure	1 (2.6)	–	0.299
Mean intensive care unit stay (d)	2.9 ± 1.4	2.8 ± 1.4	0.663
Mean hospital stay (d)	7.1 ± 1.6	7.4 ± 1.9	0.410
Hospital death	1 (2.6)	3 (3.4)	0.582

Means are expressed as mean ± SD.

AMI = acute myocardial infarction; CPB = cardiopulmonary bypass

was no significant difference between the groups in the percentage of CABG (arterial grafts only) or the use of the left internal thoracic artery; in the mean number of grafts per patient; or in the mean duration of CPB, aortic cross-clamping, or mechanical ventilation. The 2 groups were similar in their requirement of inotropic support and intra-aortic balloon pump counterpulsation. The re-exploration rates for bleeding or cardiac tamponade were also similar in both groups. There was no significant difference in the incidence of neurologic, gastrointestinal, or renal complications. The in-hospital mortality rate was 2.6% in Group I (1 patient) and 3.4% in Group II (3 patients) (P=0.582). The Group I patient died due to graft-versus-host reaction. Two Group II patients died of low cardiac output; a third died of multiple-organ failure.

During the follow-up period, 1 Group I patient and 2 Group II patients died. The mean overall survival period was 26.16 ± 16.41 months (range, 1–56 mo). The actuarial survival rates were calculated as 94.74%

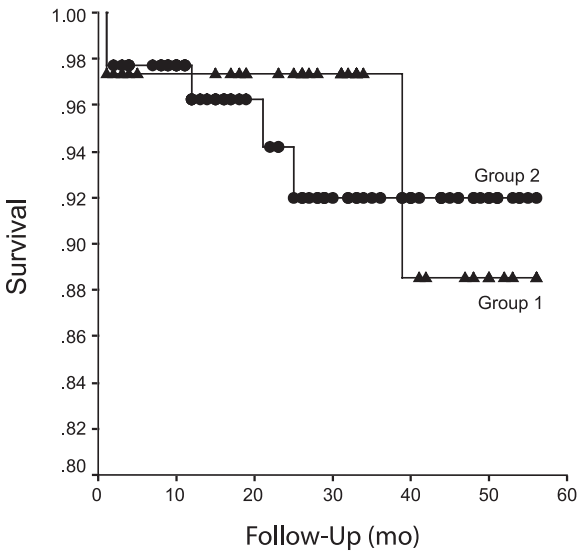

Fig. 1 *The actuarial survival rate in both groups.*

for Group I and 94.38% for Group II (Fig. 1), with no significant difference (log-rank test statistic=0.04, df=1; P=0.839). Postoperative LV ejection fraction values were similar (0.41 ± 0.12 in Group I vs 0.43 ± 0.07 in Group II; P=0.685). During the follow-up period, the percentage of patients in NYHA functional classes I and II was similar (96.9% in Group I vs 98.7% in Group II; P=0.491). One Group I patient (who was in cardiogenic shock preoperatively) and 1 Group II patient remained in NYHA functional class III after the operation.

Discussion

Long-term survival is decreased in medically treated symptomatic patients with post-infarction LVA because of recurrent myocardial infarction, congestive heart failure, arrhythmias, and thromboembolic events.[8,9] The surgical repair of LVA has been shown to provide symptomatic relief and improve long-term survival rates.[10-16] Although it is well established that surgical LVA repair is safe and effective when performed a month or longer after an AMI, only a few published clinical studies have described LVA repair that was performed soon after an anterior myocardial infarction.[5-7] Di Donato and colleagues[5] evaluated the early and late outcomes of 74 patients who had undergone surgical ventricular restoration <30 days after AMI; restoration was feasible in that patient group, with an acceptable operative mortality rate and an improved long-term outcome. Although surgeons generally prefer to postpone LVA repair for a few months or longer after a patient experiences an AMI, it may not be possible or prudent to delay surgery in patients who present with post-AMI symptoms refractory to maximal medical management. In our series, surgery could not be delayed in 38 such patients, who presented with unstable angina, heart failure (NYHA functional class III or IV), or cardiogenic shock.

Reports have shown that patients who have undergone and survived LVA repair with concurrent coronary revascularization—especially with use of the internal thoracic artery—have experienced improved hemodynamic function and clinical status.[11,17] Our institution's method of combining complete revascularization with LVA repair improved the outcomes of the Group I patients in our study.

Left ventricular remodeling, which leads to the development of systolic and diastolic dysfunction, begins soon after an acute transmural myocardial infarction and continues for months or years.[18,19] This complex and progressive process of reshaping is associated with protein changes in the extracellular matrix.[20-23] Jirmar and associates[18] showed that unsuccessful coronary revascularization led to significantly higher collagen synthesis and to degradation of infarcted myocardium, and they proved that early revascularization reduced muscular necrosis and ischemic injury to the cardiac collagen. We might then speculate that LVA repair with concurrent complete myocardial revascularization soon after an AMI prevents further remodeling of the LV in addition to providing symptomatic relief, and that the combined procedure contributes to improved early and long-term outcomes. However, we did not observe any statistically significant difference between our 2 groups with respect to postoperative LV ejection fraction. The relatively small number of patients in our study, the short follow-up period, and the lack of more reliable indicators of LV function (such as LV volume during follow-up echocardiography) may have prevented us from demonstrating the beneficial effect of early revascularization on LV remodeling.

A severe complication of LVA repair after a recent myocardial infarction is ventriculotomy-related bleeding from the friable myocardial tissues. We believe that the risk of bleeding at this site can be reduced by the use of an additional Teflon felt strip as reinforcement in both linear repair and endoaneurysmorrhaphy. In our series, the incidence of re-exploration for bleeding was acceptable, and the source of bleeding was not the site of the aneurysmal incision in any patient.

From our study, we infer that performing ventricular aneurysmal repair with coronary revascularization within 30 days after a patient's AMI is feasible and safe, with acceptable mid-term clinical outcomes. Early LVA repair incurs no more perioperative morbidity or death than does conventional post-infarction LVA repair. These results support a decision to perform early LVA repair when AMI patients are clinically unstable. Prospective trials, including large series and studies of long-term survival, are needed for further evaluation.

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