Off-pump anteroapical aneurysm plication following left ventricular postinfarction aneurysm: effect on cardiac function, clinical status and survival

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Accepted for publication
Nov. 7, 2011

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DOI: 10.1503/cjs.022111

Background: In patients with coronary disease and aneurysm, ventricular reconstruction with revascularization is a surgical option. Details of patient selection and optimal surgical technique are still debated. We report our results with off-pump aneurysm plication after ventricular aneurysm with relative wall thinning.

Methods: We retrospectively reviewed the records of 248 patients who had an operation for postinfarction left ventricular aneurysm. Reconstruction was accomplished by off-pump anteroapical aneurysm plication. The following variables were recorded: preoperative clinical, angiographic and echocardiographic findings and operative procedures. Outcomes were early mortality, long-term survival and poor 5-year result, defined as the need for transplantation or repeated hospitalization for congestive heart failure. Risk factors were pinpointed using the *t* test and survival curves. Independent risk factors were identified using Cox regression methods.

Results: Hospital mortality was low (2.0%). Mean follow-up was 5.8 (standard deviation [SD] 3.8) years. Actuarial survival at 1 and 5 years was 94% and 84%. Among the 232 survivors, 200 were in functional class I or II, and the average increase in ejection fraction was 14.0% (SD 3.1%). As determined by multivariable analysis, factors predicting poor outcome were advanced age, ejection fraction less than 0.35, conicity index less than 1, end-systolic volume index greater than 80 mL/m^2, advanced New York Heart Association functional class and congestive heart failure.

Conclusion: Using wall thinning as a criterion for patient selection, the technique of off-pump anteroapical aneurysm plication can be performed with low operative mortality and provides good symptomatic relief and long-term survival.

Contexte : Chez les patients atteints de coronaropathie et d'un anévrisme, la reconstruction ventriculaire avec revascularisation est une option chirurgicale envisageable. Les détails concernant la sélection des patients et la technique opératoire optimale ne font pas l'unanimité. Nous faisons état des résultats que nous avons obtenus avec une technique de plicature de l'anévrisme sans circulation extracorporelle (CEC) après la survenue d'un anévrisme ventriculaire associé à un amincissement relatif de la paroi.

Méthodes : Nous avons analysé de manière rétrospective les dossiers de 248 patients qui ont subi une intervention pour correction d'anévrisme ventriculaire gauche consécutif à un infarctus. La reconstruction a été réalisée par plicature antéroapicale de l'anévrisme sans CEC. Les variables suivantes ont été enregistrées : signes cliniques, angiographiques et échocardiographiques préopératoires et protocole opératoire. Les résultats ont été mortalité précoce, survie prolongée et résultats insatisfaisants à 5 ans, définis par le recours à la greffe ou des hospitalisations répétées pour insuffisance cardiaque congestive. Les facteurs de risque ont été reconnus à l'aide du test *t* et des courbes de survie. La méthode de régression de Cox a permis de relever les facteurs de risque indépendants.

Résultats : La mortalité hospitalière a été faible (2,0 %). Le suivi médian a duré 5,8 (écart-type [ET] 3,8) ans. La survie actuarielle à 1 et à 5 ans a été de 94 % et de 84 %. Parmi les 232 survivants, 200 présentaient une classe fonctionnelle I ou II et l'augmentation moyenne de leur fraction d'éjection était de 14,0 % (ET 3,1 %). Selon l'analyse multivariée, les facteurs prédicteurs de piètres résultats ont été l'âge avancé, une fraction d'éjection inférieure à 35 %, un indice de conicité inférieur à 1, un indice du volume télésystolique supérieur à 80 mL/m^2, une classe fonctionnelle avancée selon la *New York Heart Association* et l'insuffisance cardiaque congestive.

Conclusion : En utilisant l'amincissement de la paroi comme critère pour la sélection des patients, on peut appliquer la technique de plication anévrismale antéroapicale sans CEC, car elle donne lieu à un faible taux de mortalité peropératoire, procure un bon soulagement des symptômes et une survie prolongée.

Surgical ventricular restoration has been performed for many years, and a number of different surgical techniques and modifications have been developed to restore left ventricular (LV) shape and to improve LV function.[1–3] The most commonly used techniques are endoventricular repair, with or without the use of an endoventricular patch, introduced by Dor in 1989,[4] and linear repair. Previous studies have demonstrated that a dyskinetic aneurysm, in contrast to an akinetic aneurysm, is associated with better postoperative improvement in LV function and lower operative mortality.[5] However, the terms dyskinetic and akinetic are difficult to quantify, and studies had obvious limitations in the detection of these wall motion abnormalities by cineangiography.[6] A critical, quantitative analysis of ventricular dimensions and function is lacking in the published data, and in most series, the type and degree of asynergy are qualitatively based on the cardiologist's reading of cineangiography. Recently, real-time 3-dimensional echocardiography (RT3DE) was proposed as a potential alternative to magnetic resonance imaging (MRI) for this evaluation, as it provides an accurate and comprehensive assessment, including quantification of LV size, shape, global systolic function, regional wall motion and myocardial scar together with precise evaluation of the severity of mitral regurgitation.[7] Excellent correlation ($r = 0.97$, $p < 0.001$) and agreement were found between RT3DE and MRI for quantification of LV volumes and ejection fraction. In a segment-to-segment comparison, RT3DE was shown to be accurate also for the analysis of wall motion abnormalities (k = 0.62) and LV regional thickness (k = 0.56) as a marker of myocardial scar.[7] Controversy has existed over whether patients with less discrete akinetic aneurysms will benefit from wall excision and repair. Anterior myocardial infarction (MI) results in either akinetic scar or dyskinetic aneurysm; there is no doubt about the extent of thinning. Using wall thinning as a criterion for patient selection, we have used aneurysm scar plication for aneurysm repair in patients with both akinetic and dyskinetic aneurysms. This report updates our experience and highlights long-term results achieved with this simple, reproducible technique. We used preoperative and postoperative echocardiography to provide evidence of improvement in ventricular function, and independent risk factors were identified using logistic regression and Cox regression methods.

Methods

Patient population

We retrospectively reviewed the records of 248 consecutive patients who underwent LV aneurysm repair performed by a single staff surgeon (C.G.) between March 2002 and May 2008. Patients with previous anterior myocardial infarction and secondary LV dilatation were selected for surgery. The primary and common indication for LV reconstruction was dyskinetic or akinetic areas of relative thinning following myocardial infarction in the left anterior descending artery, heart failure, angina and/or a combination of the 3. Associated severe mitral regurgitation was a relative contraindication. Exclusion criteria were a posterior myocardial infarction LV dilatation and the need for mitral valve repair or replacement, and for removal of the intracavity clot, those with right ventricular hypokinesis or severe pulmonary hypertension (pulmonary artery pressure at the systemic level) and those with diffuse disease in all coronary distributions were considered inoperable. The patients provided informed consent to participate in the study, and our study protocol was approved by our local ethics committee.

Echocardiography assessment

Echocardiography assessment involved transthoracic echocardiography preoperatively and before discharge (7–10 d after the operation). End-diastolic aneurysm wall thickness was measured by RT3DE. A late echocardiography study was performed at least 6 months after the operation in patients who returned to the hospital for clinical evaluation ($n = 198$).

Standard 2-dimensional echocardiography assessment

Patients were imaged in the left lateral decubitus position using a commercially available system (iE33; Philips Medical Systems) equipped with a 3.5 MHz transducer. The LV end-systolic volume and LV end-diastolic volume were determined from the conventional apical 2- and 4-chamber views, and LV ejection fraction was calculated using the biplane Simpson technique.[8] As a measure of LV shape, we calculated the sphericity index (SI) by dividing LV end-diastolic volume by the volume of a sphere whose diameter was derived from the major end-diastolic LV long-axis. The LV long-axis (the longest distance between the centre of the mitral annulus and the endocardial apex in the 4-chamber view) and the conicity index (CI) apical to short axis ratio were calculated. Finally, using standard and off-axis views, we evaluated the presence of LV thrombus, especially in the apical region, without contrast administration.

Real-time 3-dimensional echocardiography for the preoperative assessment of patients with left ventricular aneurysm

Patients were imaged with the same system (iE33; Philips Medical Systems) equipped with an X3 fully sampled matrix transducer. Apical full-volume data sets were obtained combining, within 1 breath-hold, 7 small real-time subvolumes to provide a larger pyramidal volume (up to 101° × 104°) and to ensure a complete capture of the left ventricle. We considered a 3-D data set to be unsuitable for analysis if more than 2 segments could not be visualized or if it contained visible stitch artifacts due to irregular heart rate or breathing movements. To assess the severity of mitral regurgitation, apical full-volume colour Doppler data sets were also acquired with a Nyquist limit set between 30 cm/s and 50 cm/s. All 3-D data sets were stored digitally, and quantitative analysis was performed offline using a semiautomated contour tracing algorithm (Q-Laboratory Version 6.0; Philips Medical Systems).

During postprocessing of the 3-D data set, the software automatically displayed the apical 4- and 2-chamber views and the parasternal short-axis view. After initial identification of the apex and mitral annulus with 5 reference points on the end-diastolic and end-systolic frames, a preconfigured ellipse was fitted to the endocardial border for each frame. A manual adjustment of the endocardial border was performed if required. An LV 3-D model was automatically generated, and LV volumes and LV ejection fraction were calculated; however, the use of 2 near orthogonal cutplanes within the 3-D data set allowed for the exact location of the endocardial apex. Furthermore, by slicing the 3-D data set, several LV short- and long-axes could be obtained (at the basal, midventricular and apical level) and analyzed to assess the 3-D global wall motion score index, as with 2-dimensional echocardiography. With the same approach, we also evaluated the presence of an LV thrombus, particularly at the apical level. From the same short-axis slices, the endocardial and epicardial contours were traced manually at end diastole, with the papillary muscles included in the cavity. We then used the traced contours to calculate LV regional thickness according to the standard 16-segment model. In particular, as previously described,[9] severely thinned regions (end-diastolic thickness < 6 mm) were considered to be representative for transmural scar.

In this study, diagnosis of LV thrombus was based on routine clinical echocardiographic readings performed by a level-3 reader as part of the patient's preoperative evaluation. Finally, unlike in prior studies, antiplatelet agents and anticoagulation were routinely used. Anticoagulant therapy may substantially decrease the incidence of embolic events by 33%.[10]

Operative technique

All patients underwent median sternotomy; details of the surgical technique have been reported previously.[11,12] All operations were performed on a beating heart. Complete coronary artery bypass grafting (CABG) was first performed, almost always with the left internal mammary artery on left anterior descending and sequential venous grafts on the right and circumflex arteries, with the goal of complete revascularization. After CABG was completed, the size and shape of the LV cavity were carefully examined. In many patients it was not possible, using ventriculography or echocardiography, to determine whether akinesis or dyskinesis corresponded to an area of hibernating myocardium that would benefit from revascularization or to an area of scarred, thinned myocardium that should be resected; we believed the decision to plicate a portion of nonfunctioning wall was best made at the time of the operation. If the area of previous infarction consisted of a mixture of viable muscle and scar and there was doubt about the extent of thinning, with the beating heart, the surrounding wall was palpated, and its ability to contract (regional wall thickening) could be reliably assessed. All thinned, nonfunctioning portions of the wall were considered for plication.

The transition between infarcted aneurysm and uninfarcted myocardium was palpated, and the LV aneurysm was plicated between 2 strips of Dacron felt. Polypropylene sutures (2–0 Prolene; Hangzhou Aipu Medical Products Co.) were passed through the felt, through the aneurysm at its border and through the opposite strip of felt in a horizontal mattress fashion. Care was taken not to pass the sutures through the left anterior descending artery or the right ventricle. Plication was planned to remove as much nonfunctioning wall as possible while restoring ventricular size and shape using the modified linear closure. The resulting LV chamber had no patch and only a thin rim of noncontracting scar. In this manner, the LV "apex" was recreated adjacent to the right ventricular apex, and ventricular shape was restored (Fig. 1A and B). In cross-section, the anterior infarct was excluded, and the reconstructed LV chamber was surrounded by viable LV muscle and reinforced with the mattress sutures (Fig. 1C and D).

Follow-up

Follow-up was completed in May 2010 by means of telephone contact with patients and the referring physician or office visits when indicated. All patients were followed up in our outpatient clinic 6 months after surgical intervention and thereafter at yearly intervals. Of the 248 patients, 198 had complete clinical and echocardiographic follow-up. Preoperative, intraoperative and postoperative clinical data were obtained, and the results of echocardiograms and poor 5-year results (e.g., the need for transplantation or hospital admission for congestive heart failure [CHF]) were reviewed to assess LV function and to identify predictive factors.

Statistical analysis

We compared patient characteristics using the *t* test for continuous variables and the χ^2 or Fisher exact test for categorical variables. Continuous variables are reported as means and standard deviations (SD). Survival curves for all-cause mortality were estimated using Kaplan–Meier analysis, stratified by baseline characteristics, and were compared using log-rank tests. A good long-term result, defined as 5-year survival without the need for transplantation or repeated hospital admissions for heart failure was evaluated multivariately using a Cox proportional hazards model with the following building strategy. Variables with a univariate $p < 0.25$ or values with a known biologic importance that failed to meet the critical level were submitted for consideration to the multivariate analysis by stepwise selection. Statistical analyses were performed using SPSS version 13.0 (SPSS Inc.).

Results

We included 212 men and 36 women, with a mean age of 57 (SD 9) years. Ninety percent of the patients had sustained 1 or 2 MIs preoperatively, and 10% had more than 2 MIs. Mean delay from infarct to repair was 16 (SD 8) months. All patients had symptoms from their LV aneurysm, and 77% were in New York Heart Association functional class III or IV. Indications for surgery were angina pectoris (25%), CHF (23%), angina pectoris combined with CHF (40%) and ventricular arrhythmia combined with angina pectoris or CHF (12%). Substantial mild or moderate mitral regurgitation was present in 78 (31.5%) patients. Double or triple vessel disease was present in 245 (98.8%). The demographic, clinical, echocardiographic and procedural data are summarized in Table 1.

The entire procedure was successfully carried out on

Fig. 1. (**A**) Transthoracic echocardiographic analysis, 4 chambers. The scar involves the septum and the apex. (**B**) The scar is limited to the septoapical portion of the left ventricle (LV). The resultant LV chamber has no patch and only a thin rim of noncontracting scar. The LV "apex" is recreated adjacent to the right ventricular apex. (**C**) Drawing of the cross-section of the anterior infarct is excluded, and the reconstructed LV chamber is surrounded by viable LV muscle, with the exception of a thin rim of scar where the polypropylene sutures were plicated and reinforced with the mattress sutures. (**D**) Intraoperative picture of anteroapical aneurysm placation.

the beating heart, but 180 (72.6%) patients required inotropic support for more than 24 hours, and 78 (31.5%) required an intra-aortic balloon pump. The mean number of distal anastomoses was 2.8 (SD 1.4). Early mortality (in-hospital or < 30 d mortality) was 2.0% (5 hospital deaths). The causes of hospital deaths were low output syndrome postoperatively in 2 (0.8%) patients, adult respiratory distress syndrome in 2 (0.8%) and acute MI in 1 (0.4%). Five (2.0%) patients had perioperative infarcts. Eight (3.2%) patients required reoperation for bleeding. There were 3 (1.2%) sternal infections, 2 (0.8%) transient ischemic attacks and 1 (0.4%) stroke. Independent risk factors for early mortality were advanced New York Heart Association functional class, ejection fraction less than 0.35, CHF, age older than 70 years and interval between infarction to repair less than 3 months (Table 2).

Follow-up was 5.8 (SD 3.8, range 1.6–13.8) years, and total observation time was 861 years. There were 11 late deaths. The causes of late deaths were CHF in 4 patients, ventricular arrhythmias in 1, MI in 2, chronic obstructive pulmonary disease in 1 and unknown in 3 patients. The actuarial survival curve (including early mortality) is shown in Figure 2. Overall survival was 96% at 1 year and 84% at 5 years; survival curves stratified by dichotomized baseline characteristics, with significant difference in survival, are shown in Figure 3. Independent risk factors for overall mortality were advanced age (> 70 yr), CHF, low LV ejection fraction, advanced New York Heart Association functional class, conicity index (CI) less than 1 and end-systolic volume index greater than 80 mL/m^2 (Table 3). The following factors had no influence on early or overall mortality: sex, diabetes, angina pectoris, left main stenosis and number of distal coronary anastomoses. Among the 232 survivors, 200 (88.1%) were in functional class I or II, and the average increase in symptom class was 1.3 (SD 1.1) classes per patient ($p < 0.001$).

In all, 198 patients had complete clinical and echocardiographic follow-up. Data concerning LV diastolic diameter, LV systolic diameter, end-diastolic volume and end-systolic volume are presented in Table 4. There was a significant increase in ejection fraction (paired t test), with a mean change of 14.0% (SD 3.1%; $p = 0.006$). The mean preoperative end-diastolic volume was 133 (SD 52) mL preoperatively versus 103 (SD 35) mL postoperatively ($p = 0.002$) and the mean preoperative end-systolic volume was 97 (SD 49) mL versus 65 (SD 31) mL postoperatively ($p = 0.004$). The end-diastolic and end-systolic volumes were significantly reduced immediately after the operation

Table 1. Preoperative patient characteristics and indications for surgery (*n* = 248)

Variable	Mean (SD) [range]*
Age, yr	57 (9) [35–68]
Male:female, no.	212:36
Diabetes mellitus, no.	78
Hypertension, no.	189
Renal insufficiency, no. (%)†	56 (22.5)
SYNTAX Score‡	16 (8)
Interval after infarction, median (range) mo.	16 (8) [0–360]
< 3 mo, no. (%)	62 (25)
> 3 mo, no. (%)	186 (75)
Multiple myocardial infarctions, > 3	25
Left ventricular ejection fraction	28 (6) [25–48]
Significant coronary lesions, no.	
2-vessel	34
3-vessel	214
Mitral regurgitation	78
Mild	56
Moderate	22
EuroSCORE	7.2 (2.6) [3–16]
New York Heart Association functional class	
III, no. (%)	116
IV, no. (%)	75
Indication for surgery	
Angina + CHF + ventricular tachycardia	30 (12)
CHF	57 (23)
Angina + CHF	99 (40)
Angina	62 (25)

CHF = congestive heart failure; SD = standard deviation.
*Unless otherwise indicated.
†Renal insufficiency was defined as creatinine level greater than 1.5.
‡An emerging tool developed to characterize the coronary vasculature in more detail with respect to the number of lesions and their complexity, functional impact and location.

Table 2. Risk factors for early mortality, by multivariate analysis, in 248 patients*

Variable	Level	β (SE)	OR (95% CI)	*p* value
Age > 70	Continuous (Δ = 10 yr)	0.6 (0.2)	2.7 (0.9–10)	0.08
Interval†	Yes/no	2.5 (0.5)	7.5 (2.1–23)	0.029
CHF	Yes/no	1.7 (0.4)	10.2 (1.3–2)	0.008
NYHA IV	Yes/no	2.1 (0.6)	12.1 (0.4–27)	0.016
EF < 0.35	Yes/no	3.4 (0.5)	13.5 (0.6–34)	0.032
Constant		–10.3 (2.3)	0.001	

CHF = congestive heart failure; CI = confidence interval; EF = ejection fraction; NYHA = New York Heart Association; OR = odds ratio; SE = standard error.
*Logistic regression with manual, backward elimination.
†Interval between infarction to repair (< 3 mo).

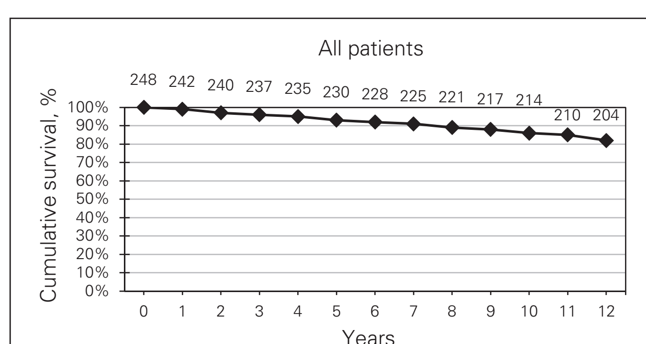

Fig. 2. Overall actuarial survival after left ventricle aneurysm plication. Figures on the curve indicate the number of patients left at risk at the time of follow-up.

and increased again in the intermediate postoperative stage. The mean diastolic sphericity index was 0.78 (SD 0.06) preoperatively versus 0.64 (SD 0.11) postoperatively (p = 0.005). Average systolic CI was 0.73 (SD 0.06) preoperatively versus 0.95 (SD 0.07) postoperatively (p = 0.003).

Discussion

This article presents short- and long-term results achieved in a large series of patients who underwent aneurysm repair by means of an off-pump anteroapical aneurysm plication technique. The series includes patients with akinetic and dyskinetic aneurysms, both large and small. We have outlined the selection criteria used in choosing patients for operative intervention. With this approach, we have achieved excellent surgical results: early mortality (in-hospital or < 30 d mortality) of 2.0% and 5-year survival of 84%. Mortality in the present study compares favourably with rates reported in other studies using linear or more complex repair techniques.[13,14]

Left ventricular aneurysmectomy has been performed for many years. However, controversy still exists about the optimal repair technique for postinfarction dyskinetic LV aneurysm. The linear closure of the ventriculotomy was introduced in 1958 by Cooley and colleagues,[15] and with its various modifications,[16] it has remained the technique of choice in most instances. Based on theoretic considerations,[17] patch remodelling of postinfarction LV aneurysm has been advocated to improve ventricular geometry and thereby early and late outcomes. Retrospective clinical studies have opposed linear to patch repair and reported varying results.[18,19] Kesler and colleagues[20] compared the results of linear plasty and patch endoventriculoplasty and found no significant difference in mortality, angina, CHF or echocardiographic variables. Laboratory studies on animals carried out by Nicolosi and colleagues[21] did not reveal a difference in the systolic and diastolic functions of LV between LV linear plasty and plasty with a synthetic patch; hence, various types of LV plasty are sufficiently effective during surgical treatment of cardiac aneurysms, and the postoperative improvement of the patients' clinical state does not depend on the type of LV plasty used.

The aim of the plicating technique is to spare the LV lateral wall and preserve systodiastolic expansions of the viable lateral myocardial wall, which frequently shows normal contractile activity in patients with a previous anteroseptal infarction. This repair shortens LV equatorial diameter and the distance between the lateral wall and septum, preserving lateral contractility. Like other surgical ventricular restoration techniques, the plicating repair improves symptoms and LV ejection fraction by reducing LV volumes and diameters. Our echocardiographic data has provided

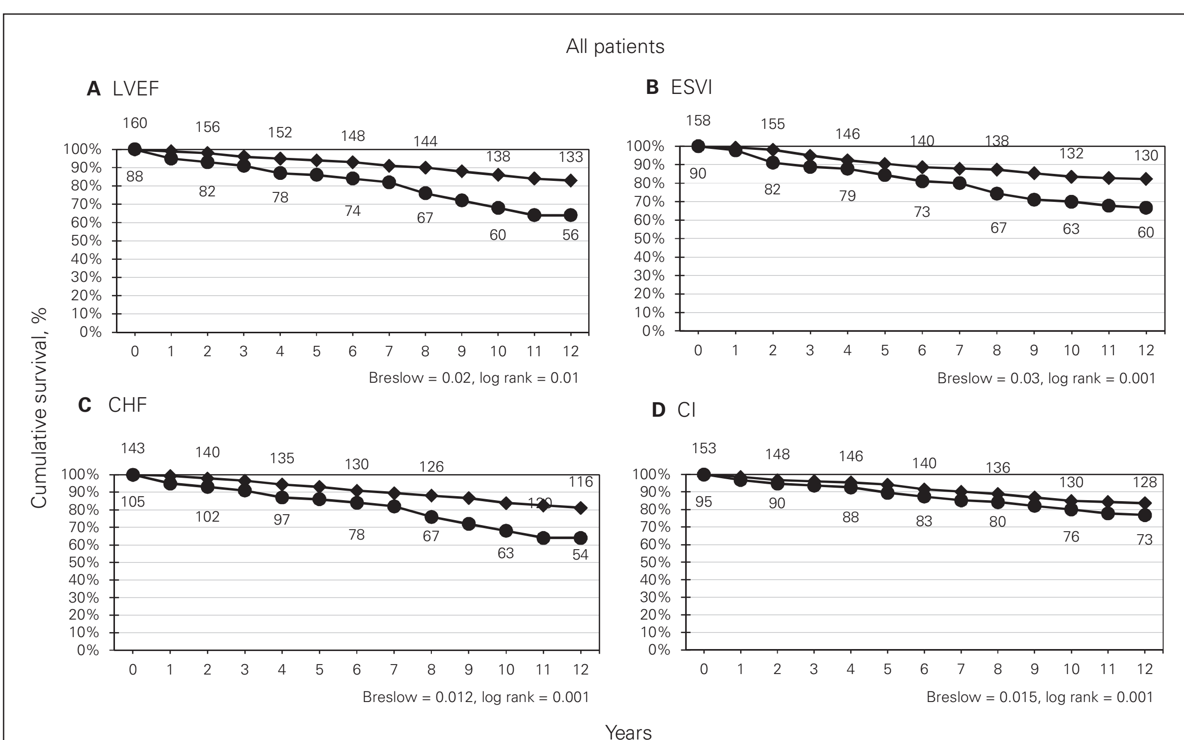

Fig. 3. Effect on cumulative survival after left ventricular (LV) aneurysm repair. (**A**) Squares show LV ejection fraction (LVEF) greater than 0.35, and circles show LVEF less than 0.35. (**B**) Circles show end-systolic volume index (ESVI) greater than 80 mL/m^2, and squares show ESVI less than 80 mL/m^2. (**C**) Circles show congestive heart failure (CHF) greater than III, and squares show CHF less than III. (**D**) Circles show conicity index (CI) less than 1, and squares show CI greater than 1. Numbers on the curves indicate the number of patients at risk.

evidence of improved LV function after the operation. The average increase in ejection fraction of 14.0% (SD 3.1%) is similar to reports of 10%–14% in other series.[14,22]

An important aspect of our approach, especially in patients with akinetic aneurysms, is assessment of wall thickness and function in the beating ventricle, which allows us to make decisions about resection versus revascularization without relying on viability studies. Furthermore, we believe that carrying out the repair on the beating heart without cardiopulmonary bypass has the advantages of no ischemic time and easier decision-making with regard to optimal restoration of LV geometry. By plicating nonfunctioning wall, excluding aneurysmal portions of the left ventricle and plicating the length of the thin-wall aneurysm in the repair, we attempt to decrease chamber dimensions, wall tension and wall stress and to restore LV geometry. This avoids creating too small a cavity with the repair; with inadequate correction we may not have a long and narrow LV after linear plasty.

Previous studies have demonstrated that a dyskinetic aneurysm, in contrast to an akinetic aneurysm, is associated with better postoperative improvement in LV function and lower operative mortality.[4,5] Furthermore, other reports have suggested that only patients with clear-cut dyskinesis will benefit from aneurysm excision in terms of symptom relief, improved ejection fraction and long-term survival. It has been assumed that a dyskinetic aneurysm produces a greater volume of stress on the contractile myocardium; therefore, its resection should predict an improvement in LV function after aneurysmectomy, whereas the absence of paradoxic systolic motion correlates with higher operative mortality and no improvement in function. However, the terms dyskinetic and akinetic are difficult to quantify, and studies have obvious limitations in the detection of these wall motion abnormalities using cineangiography.[6] In the present study, standard 2-dimensional echocardiography and RT3DE for the preoperative assessment of patients with LV aneurysm have shown excellent correlation ($r = 0.97$, $p < 0.001$) and agreement between RT3DE and standard 2D echocardiography for quantification of LV volumes, ejection fraction and sphericity index. In a segment-to-segment comparison, RT3DE has been shown to be accurate for the analysis of wall motion abnormalities and LV regional thickness as a marker of myocardial scar.[7] We have demonstrated that surgical outcome, both in terms of functional improvement and early mortality, does not depend on the presence or absence of paradoxic motion. In these patients, there was a significant increase in ejection fraction (paired t test).

We defined a poor 5-year result as death or the need for transplantation or repeated hospital admission for CHF. Multivariable predictors of poor 5-year outcome were poor LV function (ejection fraction ≤ 35%) and New York Heart Association functional class III/IV with combined with CHF. Age older than 70 years, marked ventricular enlargement (end-systolic volume index > 80 mL/m^2) and CI less than 1 also predicted poor outcome. In contrast, we did not find the extent of coronary artery disease (number of diseased vessels, coronary score:SYNTAX Score ratio) or left main coronary stenosis to be a risk factor for death. The extent of revascularization performed was not found to be predictive of risk, and this is probably due to a policy of complete revascularization in our patients. Moderate or severe mitral regurgitation and ventricular arrhythmias

Table 3. Risk factors for overall mortality, by multivariate analysis

Variable	Level	β (SE)	OR (95% CI)	p value
Age > 70	Continuous (Δ = 10 yr)	0.9 (0.4)	2.4 (1.0–12)	0.004
EF < 0.35	Yes/no	2.1 (0.8)	7.9 (1.6–40)	0.013
ESVI > 80mL/m^2	Yes/no	1.3 (0.7)	3.7 (1.0–14)	0.042
CHF	Yes/no	1.5 (0.8)	4.4 (1.0–19)	0.047
Conicity index	Yes/no	0.8 (0.3)	2.2 (1.2–4.3)	0.023
Constant		−10.8 (3.3)	0.0001	0.001

CHF = congestive heart failure; CI = confidence interval; EF = ejection fraction; ESVI = end-systolic volume index; OR = odds ratio; SE = standard error.

Table 4. Changes in left ventricular shape and cardiac status changes at different intervals

	Time;* mean (SD) [median]		
Variable	Preoperative, n = 248	Early postoperative, n = 243	Late postoperative, n = 198
End-diastolic volume index, mL	133 (52) [145]	103 (35) [116]†	98 (20) [128]†‡
End-systolic volume index, mL	97 (49) [111]	65 (31) [72]†	76 (30) [80]†‡
Ejection fraction, %	33 (10) [33]	40 (10) [39]†	39 (10) [39]†
Sphericity index	0.78 (0.06)	0.64 (0.11)†	0.75 (0.09)
Conicity index	0.73 (0.06)	0.95 (0.07)†	0.92 (0.06)†‡
LV diastolic diameter, mm	73.3 (7.5)	56.9 (9.3)†	58.1 (6.9)
LV systolic diameter, mm	50.8 (10.2)	42 (11.1)†	45.1 (9.2)
New York Heart Association functional class	2.7 (0.9)	2.1 (0.6)	1.6 (0.7) [1.0]†

LV = left ventricle; SD = standard deviation.
*Early postoperative period from 10 days to 1 month; late postoperative period from 1 year.
†$p < 0.01$ versus preoperatively.
‡$p < 0.01$ versus early postoperatively.

have been found in other series to be related to increased operative mortality; however, there were too few patients in our study to show a significant association. Nevertheless, our in-hospital mortality was low compared with that reported in other series (2% v. 7%–13%); case selection and operative technique may account for these differences.

Pasini and colleagues[23] reported an 8.6% incidence of sudden deaths after aneurysm repair. Survival is reduced in patients who are older and have poor LV function, CHF and CI less than 1. After aneurysm resection, close follow-up and careful adjustment of medications are necessary to optimize symptomatic improvement. During follow-up, 200 of 232 (86.1%) survivors were in functional class I or II, and no patients were referred for additional procedures because of CHF. Our 5-year survival rate was 84%, which compares with the results of other reported series at 2 and at 5 years.[24]

Ventricular arrhythmias after MI are associated with high mortality and risk for sudden cardiac death. The Multicentre Automatic Defibrillator Trial II (MADIT-II)[25] reported that prophylactic implantation of a defibrillator improved survival in patients with a prior MI and severe LV dysfunction. It also showed that new or worsened heart failure requiring hospitalization was more frequent in the defibrillator group than in the conventional therapy group and that back-up ventricular pacing may impair LV function.[25] Myocardial stretch has been reported to be arrhythmogenic.[26] It is plausible that ventricular reconstruction, restoring near-normal LV size and form and thus reducing wall tension, could have a beneficial effect on electrical instability. It has recently been reported that surgical ventricular restoration creates a mechanical intraventricular resynchronization in patients with ischemic cardiomyopathy and with no preoperative electrical conduction delay.[27] It has also been shown in patients with ischemic cardiomyopathy that cardiac resynchronization therapy reduces both inducibility of ventricular tachycardia and frequency of ventricular tachycardia episodes. Thus, it seems that intraventricular resynchronization, either by surgical ventricular restoration or biventricular pacing, reduces ventricular arrhythmias in the dilated heart. The mechanism for this improvement may be related to the beneficial effects on LV synchrony, because improved synchrony will not only improve LV hemodynamics but also homogenize regional wall stress and reduce regional prestretch, which is potentially arrhythmogenic.[28] For these reasons, in our opinion, a thorough preoperative assessment and a focused surgical strategy can improve outcomes in these patients. This concept was introduced by Dor and colleagues[4] and consists of complete revascularization to relieve ischemia, ventricular reconstruction to restore normal shape and volume to reduce LV wall tension and improve hemodynamics. In patients with postoperatively confirmed spontaneous or inducible ventricular tachycardia, we recommend using a prophylactic implantable cardioverter defibrillator. There was only 1 arrhythmia-related death during follow-up in our series, suggesting that over time this treatment policy is valid.

We observed a trend toward redilation. The causes of the postoperative remodelling are unclear but may include progression of the underlying biologic disease process and failure of the repair to reduce wall stress in the border zone and remote uninfarcted myocardium. Di Donato and colleagues[29] revealed that some changes of LV shape after the procedure contribute to the enlargement of LV sphericity. More importantly, the sphericity index in their series increased linearly after the operation, potentially causing a progressive increase in LV wall tension and energy consumption. We found out that LV tends to have a greater elliptical shape after plication plasty. Average systolic CI was 0.73 (SD 0.06) preoperatively versus 0.92 (SD 0.06) postoperatively.

Conclusion

Our experience with the technique of aneurysm scar plication for ventricular aneurysm with relatively thinning wall has shown that aneurysm repair can be performed even in patients with severe coronary artery disease and poor ventricular function with a low operative mortality, good symptomatic improvement and excellent 5-year survival. We believe that the technique can be accomplished without aortic cross-clamping and is advantageous because of its simplicity, reproducibility and lack of ischemic time.

Competing interests: None declared.

Contributors: C. Gu and Q. Zhou designed the study. X. Huang and C. Gu acquired the data. X. Huang, C. Gu, J. Yang, H. Wei and J. Li analyzed the data. X. Huang wrote the article. C. Gu, J. Yang, H. Wei, J. Li and Q. Zhou reviewed the article. All authors approved its publication.

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