Use of quantitative analysis of remote myocardial fibrosis with delayed-enhancement magnetic resonance imaging to predict outcomes after surgical ventricular restoration for ischemic cardiomyopathy

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Objective: Preserved myocardial function remote from surgical site is crucial for good outcome after surgical ventricular restoration in ischemic cardiomyopathy. We hypothesized that left ventricular scarring untouched by operation would negatively affect postoperative outcome.

Methods: In 15 consecutive patients (mean age 61 ± 12 years, mean left ventricular ejection fraction 20% ± 7.5%), left ventricular assessments by magnetic resonance imaging and right heart catheterization were performed before and after operation. Left ventricular basal scarring remote from surgical exclusion site was quantified from hyperenhancement area on preoperative delayed-enhancement magnetic resonance imaging as percentage of fibrosis (total infarct size relative to ventricular mass).

Results: Calculated percentage of fibrosis varied from 0% to 29.9% (mean 12% ± 9.6%). Percentage of fibrosis linearly correlated with significantly worse postoperative hemodynamic variables and left ventricular function recovery: left ventricular ejection fraction ($P = .0005$, $R = –0.79$), left ventricular end-systolic volume index ($P = .05$, $R = 0.51$), mean pulmonary arterial pressure ($P = .004$, $R = 0.70$), pulmonary capillary wedge pressure ($P = .009$, $R = 0.65$), and cardiac index ($P = .005$, $R = –0.69$). At mean 30-month follow-up, 4 patients with recurrent heart failure had significantly greater percentage of fibrosis than did those without recurrence (19% ± 8.2% vs 8.8% ± 8.6%, $P = .04$).

Conclusion: Amount of myocardial scarring at left ventricular base affected postoperative left ventricular function and hemodynamic improvements. Preoperative quantitative assessment of remote myocardial status with delayed-enhancement magnetic resonance imaging may predict outcomes for patients undergoing surgical ventricular restoration.

Surgical ventricular restoration (SVR) is increasingly being used as a treatment for ischemic cardiomyopathy. Several reports, including the RESTORE (the international Reconstructive Endoventricular Surgery returning Torsion Original Radius Elliptical shape to the left ventricle) group experience, have shown good midterm to long-term survivals with acceptable operative mortality.[1-5] Current relative contraindications for SVR are severe pulmonary hypertension, right ventricular dysfunction, and diastolic dysfunction with severe mitral regurgitation.[3,4,6] Several recent studies, however, have suggested that SVR is not necessarily contraindicated for patients with those presumed high surgical risks.[7-9] Although the ongoing Surgical Treatment for Ischemic Heart Failure trial may address open and controversial issues about SVR in patients with ischemic heart failure,[10] there is currently no consensus regarding indications for SVR.

Preoperative assessment of myocardial status can provide a meaningful perspective on the application of SVR. To predict whether myocardium remains stunned or hibernating or has been irreversibly infarcted, magnetic resonance imaging (MRI) is now gaining widespread use.[2,4,9,11] Especially, delayed-enhancement MRI (DE-MRI) is an important diagnostic tool to determine the nonfunctional infarcted area that should be excluded with SVR.[11,12] Moreover, it provides additional information regarding the myocardial status remote from the region of planned exclusion.[11] Because remote myocardial asynergy directly affects the surgical outcome,[3,13] assessment of remote myocardial status with DE-MRI may be critical in patient selection. No study to date, however, has quantitatively investigated how much viable remote myocardium is crucial for a good outcome.

In this study, we focused on the amount of remote myocardial fibrosis, especially left ventricular (LV) basal fibrosis, quantitatively assessed by DE-MRI, and evaluated the correlation with surgical outcomes to elucidate appropriate risk stratification.

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Received for publication Dec 23, 2007; revisions received Feb 12, 2008; accepted for publication March 23, 2008.
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J Thorac Cardiovasc Surg 2008;136:1514-21
0022-5223/$34.00

doi:10.1016/j.jtcvs.2008.03.037

Abbreviations and Acronyms

ΔEF	= change in ejection fraction
DE-MRI	= delayed-enhancement magnetic resonance imaging
LV	= left ventricular
LVEF	= left ventricular ejection fraction
MRI	= magnetic resonance imaging
NYHA	= New York Heart Association
PAP	= pulmonary arterial pressure
PCWP	= pulmonary capillary wedge pressure
SVR	= surgical ventricular restoration

MATERIALS AND METHODS

Patients

Ethical committee approval was obtained for this retrospective study, and informed consent was obtained from all patients. We included 15 consecutive patients who underwent DE-MRI study for the assessment of myocardial status before SVR between 2001 and 2007. The patients underwent MRI study 4.1 ± 1.1 weeks before the operation. Table 1 shows preoperative clinical profiles of all the patients. There were 12 men and 3 women, with a mean age of 61 ± 11 years (42–77 years). Thirteen patients (86.7%) were in New York Heart Association (NYHA) functional class III or IV. SVR was performed for large anteroseptal scars (4 dyskinetic and 11 akinetic). Seven patients had a history of another myocardial infarction in the territory of the circumflex or right coronary artery. Mean delay from first anterior myocardial infarction to the operation was 961 ± 950 days, and all patients had a chronic process of LV remodeling. No one had a preoperative history of spontaneous ventricular arrhythmia.

Operations

Operative variables are shown in Table 2. All operations were performed by a single surgeon (G.M). Conventional cardiopulmonary bypass was used with mild hypothermia. Simultaneous mitral valve repair and coronary artery bypass grafting (CABG) were performed during aortic crossclamping and with antegrade and retrograde blood cardioplegia. CABG was performed with the aim of complete revascularization with the predominant use of internal thoracic arteries. We prefer to perform SVR on the beating heart whenever possible. The decision to perform SVR on the beating or arrested heart was made on the basis of clamp time, concomitant procedures, and presence of LV mural thrombus. The most important concept in our surgical strategy for SVR is exclusion of only nonviable and noncontractile myocardium. Accordingly, surgical exclusion area was precisely planned on the basis of preoperative assessment of LV wall motion and viability with MRI (Figure 1). Nonviable tissue was determined by extent of hyperenhancement area on DE-MRI.[12] During the study period, reconstruction of the anteroseptal wall was performed with Dor-type procedure with a Fontan stitch, or with septal anterior ventricular exclusion (SAVE)[14] when the LV akinesis was broad. In the SAVE cases, a longitudinal Dacron polyester fabric patch (7 × 2.5 cm) was sutured to the transitional zone to exclude broad anteroseptal scarring without Fontan stitch, and special care was taken to create the elliptical LV shape. SVR procedures were Dor-type procedures in 4 cases and SAVE in 11. Simultaneous procedures performed during operations were CABG in 10 cases (1–4 grafts/patient), restrictive mitral annuloplasty (2 sizes smaller than measured) with a Carpentier-Edwards Physio ring (Edwards Lifesciences LLC, Irvine, Calif) for more than 2+ mitral regurgitation in 8 cases, tricuspid ring annuloplasty in 5 cases, intraventricular papillary muscle imbrication[15] in 7 cases, and maze procedure in 2 cases. Five patients who did not undergo CABG had previously undergone revascularization either percutaneously or surgically or had no viable myocardium despite the residual coronary stenosis. Mean cardiopulmonary bypass and aortic crossclamp times were 185 ± 65 and 74 ± 32 minutes, respectively.

TABLE 1. Preoperative patient's characteristics (n=15)

Age (y)	61 ± 12
Sex (male/female)	11:4
New York Heart Association functional class III or IV (No.)	13 (86.7%)
Delay from myocardial infarction (d)	961 ± 950 (75–5666)
Previous cardiac surgery (No.)	2 (13.3%)
Hyperlipidemia (No.)	5 (33.3%)
Hypertension (No.)	4 (26.7%)
Diabetes mellitus (No.)	4 (26.7%)
Chronic renal failure (No.)	1 (6.7%)
Mitral regurgitation grade (No.)	
0 or 1	5
2	3
3 or 4	7
Type of asynergy (No.)	
Dyskinetic	4
Akinetic	11
Coronary artery disease (No.)	
1 vessel	6
2 vessels	1
3 vessels	8

MRI Protocol

MRI images were acquired with a commercially available 1.5-T imager (GE Healthcare Technologies, Waukesha, Wis) with a phased-array coil with repeated breath-holding and retrospective electrocardiographically triggered gating. Steady-state free precession cine images were acquired in multiple short-axis views (every 8 mm throughout the entire LV). Temporal resolution of cine images ranged from 30 to 50 ms. The myocardial borders were planimetered on all the short-axis cine images to determine LV function. Contrast agent (gadopentetate dimeglumine [INN gadopentetic acid], 0.1–0.2 mmol/kg) was administered intravenously, and 8 to 12 DE-MRI images, depending on the cardiac size, were acquired after 10 to 15 minutes with a segmented inversion-recovery technique in the identical planes.[16] Hyperenhancement tissue on DE-MRI images was assumed to represent scarred myocardium.

Quantitative Assessment of Myocardial Scar Tissue

We specifically evaluated the LV basal scar tissue remote from the site of surgery. Quantification of the scarred myocardium was performed with the two most basal DE-MRI images just beneath the aortic valve, which represented the most basal part of the LV (Figure 2). In this study population, there were no patients in whom anteroseptal myocardium at the most basal part had been entirely infarcted with the transmural extent of

TABLE 2. Operative variables

Type of surgical ventricular restoration (No.)	
Dor	4
Septal anterior ventricular exclusion	11
Coronary artery bypass grafting (No.)	10 (66.7%)
Mitral annuloplasty (No.)	8 (53.3%)
Tricuspid annuloplasty (No.)	5 (33.3%)
Maze (No.)	2 (13.3%)
Mean bypass time (min)	185 ± 65
Mean cross clamp time (min)	74 ± 32

ACD

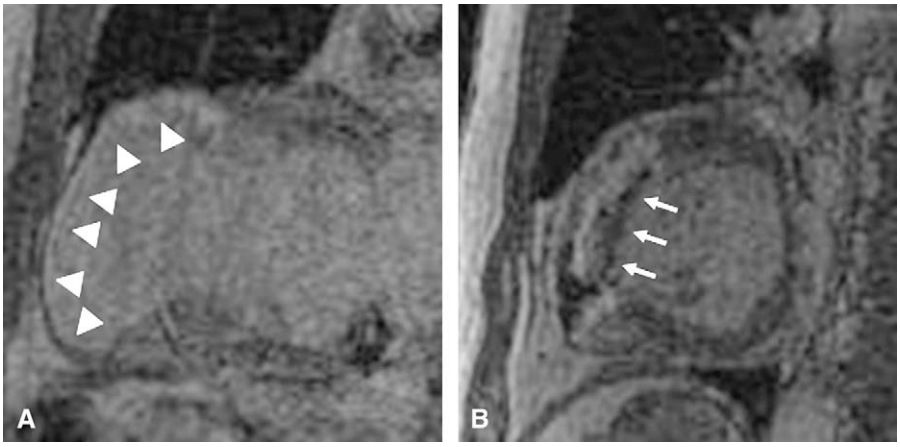

FIGURE 1. Preoperative and postoperative long-axis delayed-enhancement magnetic resonance images in patient with anteroapical dyskinetic aneurysm. Preoperative image shows transmural hyperenhancement area at anteroapical wall (A, *arrowheads*). After operation, hyperenhancement area was completely excluded with endoventricular patch (B, *arrows*).

hyperenhancement tissue. The most LV basal images were carefully defined in consensus by two observers (K.T. and G.M.). To quantify the myocardial mass of the LV, the endocardial and epicardial borders were traced for each slice. Hyperenhancement area was defined as any region with a signal intensity at least 2 SD above a reference normal myocardial region.[17] The hyperenhancement regions were automatically contoured, and the area of hyperenhancing tissue was calculated. DE-MRI analysis was verified by an experienced observer (S.H.) blinded to the surgical procedure. Measurement of hyperenhancement area was repeated three times in each case to minimize the potential errors, and the mean was used as the final value. Then the volumes of the LV wall and scarred myocardium were calculated by adding the areas and multiplying the resulting value with slice thickness. We expressed hyperenhancement mass as a percentage of the LV mass (percentage of fibrosis).

Assessments of Cardiac Function and Mitral Regurgitation

MRI was used to calculate LV ejection fraction (LVEF), LV end-systolic volume index (LVESVI), and LV end-diastolic volume index. MRI study was repeated at 3.7 ± 2.2 weeks after the operation. Preoperative and postoperative degrees of mitral regurgitation were quantified by means of echocardiographic Doppler analysis on a 5-point scale (0 to 4+) with almost the same timing as MRI study. Preoperative right heart catheterization (6.8 weeks before the operation) was used to measure mean pulmonary arterial pressure (PAP), pulmonary capillary wedge pressure (PCWP), and cardiac index. Postoperative hemodynamic parameters were obtained at 2.2 ± 1.0 weeks after the operation.

Neurohormonal Assessment

Serum brain natriuretic peptide was measured at the time of preoperative and postoperative MRI studies.

Postoperative Medical Regimens

Most patients had postoperative heart failure regimens, including β-blockers, angiotensin-converting enzyme inhibitors, or angiotension II receptor blockers, and spironolactone. Three patients who had postoperative ventricular arrhythmia develop were treated with amiodarone.

Statistical Analysis

SPSS software (version 11.0; SPSS Inc, Chicago, Ill) was used for statistical analyses. The quantitative data are presented as mean ± SD. Values obtained from preoperative and postoperative data were compared with the paired *t* test. The unpaired *t* test was used to compare the groups. The continuous variables were compared with linear regression analysis.

RESULTS

Early Outcomes

All patients tolerated the operation, and 30-day mortality was nil. Three patients required intra-aortic balloon pumping but could be weaned in the early perioperative period. One patient who had refractory ventricular tachycardia develop required an implantable defibrillator in the early postoperative period. Postoperative hemodynamic and functional parameters were improved significantly. NYHA functional class improved significantly, from 3.2 ± 0.6 preoperatively to 1.9 ± 0.8 postoperatively ($P < .0001$). Degree of mitral regurgitation was less than grade 2 in all patients postoperatively and decreased significantly, from 2.0 ± 1.5 to 0.2 ± 0.4 ($P = .002$). Serum brain natriuretic peptide level decreased significantly, from 640 ± 731 pg/mL to 358 ± 454 pg/mL ($P = .02$). In all cases, SVR improved LVEF and LV volumes. LV end-diastolic volume index decreased from 169 ± 51 mL/m^2 to 105 ± 37 mL/m^2 ($P < .0001$), and LVESVI decreased from 134 ± 50 mL/m^2 to 74 ± 34 mL/m^2 ($P < .0001$). LVEF improved from 20% ± 7.5% to 31% ± 10% ($P = .0001$). Mean change in ejection fraction (ΔEF) was 10% ± 6.9%. Mean PAP decreased from 26 ± 10 mm Hg to 22 ± 7.1 mm Hg, but this change did not reach statistical significance ($P = .06$). There were no significant differences between preoperative and postoperative values of PCWP (18 ± 7.6 mm Hg vs 15 ± 6.8 mm Hg, $P = .28$) and cardiac index (2.4 ± 0.4 L/[min · m^2] vs 2.6 ± 0.6 L/[min · m^2], $P = .28$).

Distribution of Hyperenhancement Area on DE-MRI

There was no hyperenhancement area on the LV base in 2 patients with dyskinetic anteroapical aneurysm. Among

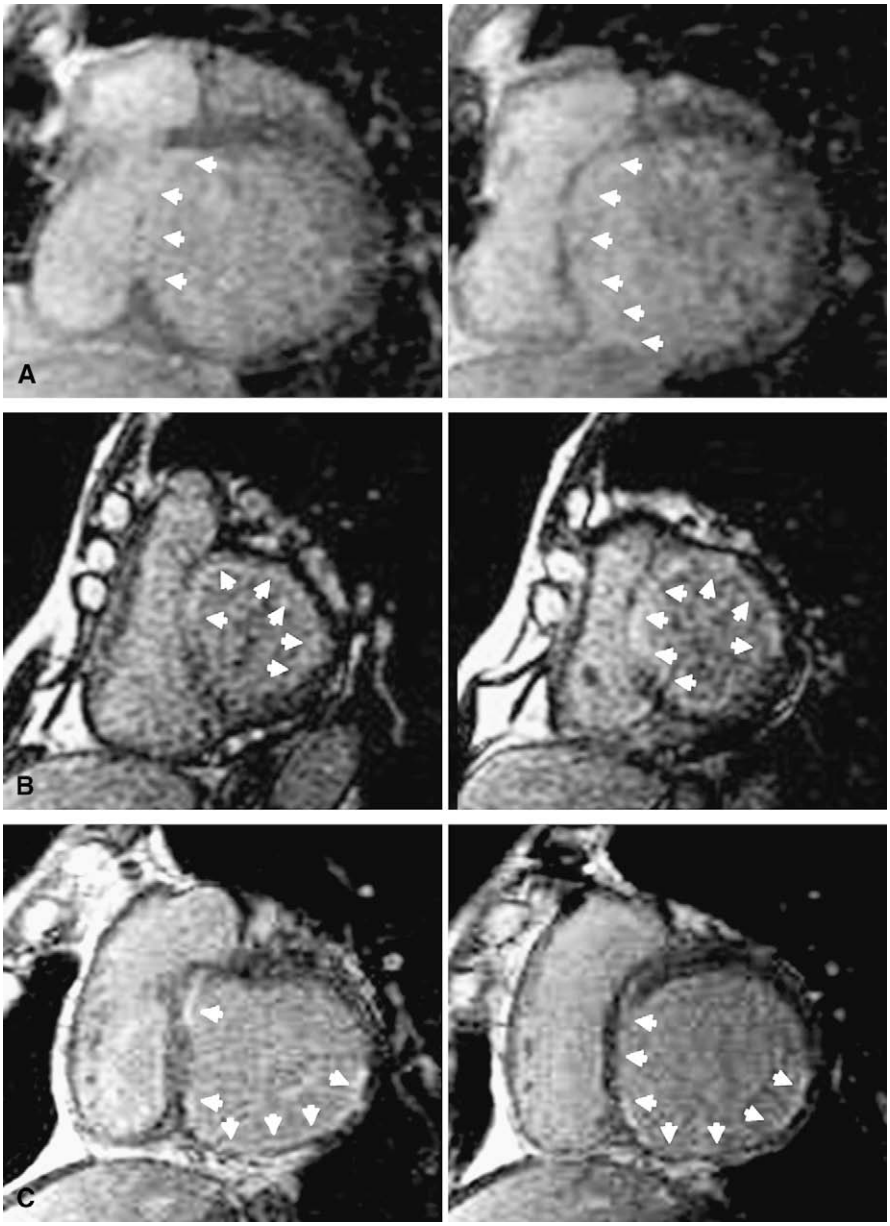

FIGURE 2. Representative short-axis delayed-enhancement magnetic resonance images of left ventricular base in patients with anteroseptal infarction (A), anteroseptal with posterolateral infarction (B), and anteroseptal with inferoposterior infarction (C). Myocardial scarring was clearly identified as hyperenhancement region (arrow).

the remaining 13 patients, hyperenhancement area was identified at the anteroseptal wall in 6 cases and at the anteroseptal with the lateral or inferior wall or both in 7 cases (Figure 3). Percentage of fibrosis ranged from 0% to 29.9%, with a mean value of 12% ± 9.6%. In particular, patients with a history of left main trunk occlusion (Figure 2, *B*) or large inferoposterior myocardial infarction (Figure 2, *C*) had a larger percentage of fibrosis. At the basal area analyzed by MRI in this study, the former group of patients had diffuse subendocardial anteroseptal to lateral scarring, whereas the latter group had near-transmural inferoposterior scarring.

Impact of Percentage of Fibrosis on Cardiac Function

There were statistically significant correlations between percentage of fibrosis and preoperative mean PAP ($P = .0001$, $R = 0.83$) and PCWP ($P = .0002$, $R = 0.82$). LV volumes (LV end-diastolic volume index $P = .23$, LVESVI $P = .14$), and LVEF ($P = .15$) had no significant correlation

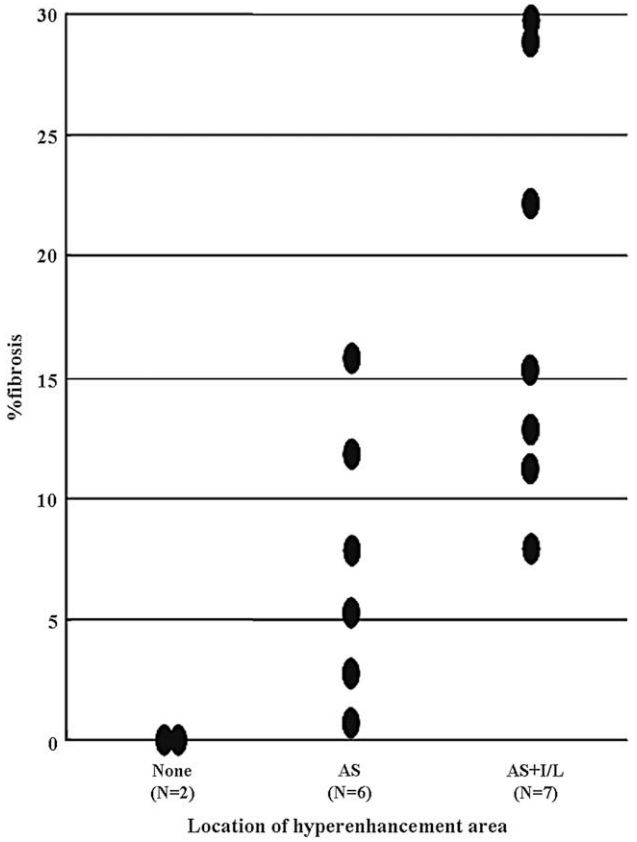

FIGURE 3. Location of myocardial scarring on LV base and percentage of fibrosis in individual patient. *AS,* Anteroseptal wall; *AS+I/L,* anteroseptal with inferior or lateral wall.

with percentage of fibrosis. Postoperative LVEF ($P = .0005$, $R = –0.79$), LVESVI ($P = .05$, $R = 0.51$), mean PAP ($P = .004$, $R = 0.70$), PCWP ($P = .009$, $R = 0.65$), and cardiac index ($P = .005$, $R = –0.69$) had significant correlations with percentage of fibrosis (Figure 4).

Table 3 compares preoperative variables between the groups with more than 10% improvement in LVEF and those with less than 10% improvement. Patients with less than 10% ΔEF (n = 6) had significantly larger preoperative percentage of fibrosis ($P = .002$).

Impact of Percentage of Fibrosis on Recurrence of Heart Failure

During a mean follow-up of 30 months, 2 patients died of recurrent heart failure at 3.7 and 57 months after the operation. The heart from a patient who died earlier was examined by pathologists (Figure 5). The analysis showed a large scarred basal inferoposterior area, compatible with the preoperative MRI study. Another 2 patients were readmitted for congestive heart failure but recovered from NYHA functional class III to II after adequate medical treatment. The others (n = 11) were well and in NYHA functional class I or II. All 4 patients with recurrent congestive heart failure were included in the group of patients with ΔEF less than 10%. Table 4 shows preoperative parameters associated with adverse postoperative outcomes tested at univariate analysis. Only percentage of fibrosis was a statistically significant predictor of recurrent heart failure ($P = .04$). The relationship between percentage of fibrosis and postoperative NYHA functional class is shown in Figure 6.

DISCUSSION

In this study, we focused on LV basal scar tissue remote from the area considered for surgical exclusion by SVR and investigated whether it affected the early and midterm postoperative outcomes. Imaging of the remote myocardial status is crucial when we plan SVR, because LV asynergy is often severe and global in ischemic cardiomyopathy. Previously, Di Donato and colleagues[3,13] reported that patients with remote myocardial asynergy had a worse prognosis because of lack of recovery of regional function after SVR. That study, however, provided no conclusive evidence to demonstrate remote myocardial status (viable vs nonviable). Viability assessment by inotropic stimulus (dobutamine stress test) is helpful to determine reserved contractility of remote segments[3]; however, it provides no quantitative data about myocardial fibrosis. We specifically quantified the degree of remote myocardial fibrosis with DE-MRI.

We found a significant correlation between percentage of fibrosis and lesser improvement of postoperative LV systolic function and symptoms (Figure 4). Our data indicate the extent of LV basal fibrosis is associated with postoperative global cardiac function. Theoretically, resection of the akinetic or dyskinetic scar tissue and reshaping of the spherical LV to a more elliptical shape by SVR reduces wall stress and produces a more helical fiber orientation, which improves regional myocardial performance in remote viable muscle.[18] Various previous studies have confirmed this theoretic benefit of SVR. Taniguchi and coworkers[19] reported that reduction of regional afterload by LV aneurysmectomy improved velocity of circumferential shortening in noninfarcted myocardial segments. With regard to remote myocardial performance, Dor and colleagues[2] and Dor and Di Donato[13] showed its importance with the centerline method on ventriculography. Some studies wtih MRI tagging methods have also shown improvements in regional myocardial function after SVR to be most marked at the base LV and in the inferior wall, remote from the surgical site.[20,21] Consequently, these studies, including our own, demonstrate that residual remote myocardial function, especially LV basal function, generates the improvement in global LV systolic function after SVR.

Our patients underwent various surgical interventions, including revascularization and reduction of mitral regurgitation, that also improve global pump function. There are still no conclusive data regarding the role of SVR in itself for ischemic cardiomyopathy because of the lack of

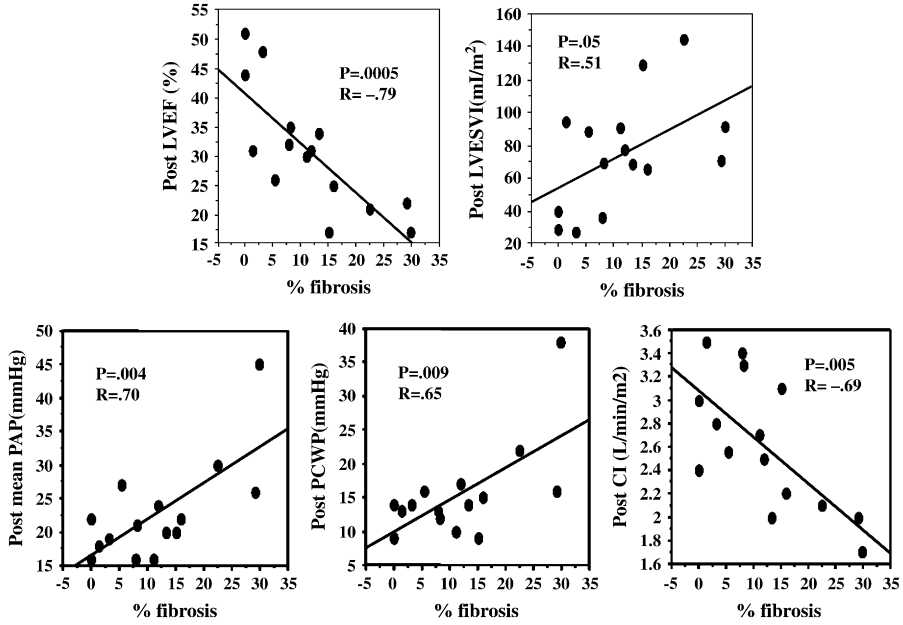

FIGURE 4. Scatter graphs showing linear regressions between percentage of fibrosis and postoperative *(Post)* left ventricular ejection fraction *(LVEF),* left ventricular end-systolic volume index *(LVESVI),* mean pulmonary arterial pressure *(PAP),* pulmonary capillary wedge pressure *(PCWP),* and cardiac index *(CI).*

randomized studies. The Surgical Treatment for Ischemic Heart Failure trial may address these controversial issues in a randomized, controlled setting.[10]

Interestingly, percentage of fibrosis had significant positive correlations with postoperative mean PAP and PCWP, which were sustained even after the aggressive correction of mitral regurgitation by restrictive annuloplasty. This finding indicates the role of the elevation of LV filling pressure as a result of reduced compliance of the LV after SVR related to the extent of remote myocardial fibrosis. Previous reports have demonstrated that SVR contributed to the rise in mean PAP by reducing compliance of the LV in some patients.[22] Most recently, Menicanti and colleagues[3] clearly showed that patients with severe diastolic dysfunction had an increased operative risk during SVR.[3] In addition to the systolic impairment, extent of infarct scarring contributed to abnormal ventricular chamber stiffness with diastolic dysfunction in an experimental animal model of myocardial infarction.[23] Our data therefore suggest that patients with broad LV basal scarring seen on DE-MRI may have substantial risks with SVR in terms of diminished compliance of the LV.

In this small series, we achieved acceptable early results with respect to improvements in LV function and functional status and decrease in brain natriuretic peptide level. The average increase in LVEF of 10% ± 6.9% is similar to values reported in other series[1,3-6]; however, there are still no obvious data on which patients will have meaningful recovery after SVR. In our series, 6 patients had less benefit in terms of systolic improvement (*Δ*EF <10%). They had significantly larger percentages of fibrosis, although their degree of preoperative LV remodeling was similar to that of patients with good improvement (*Δ*EF >10%; Table 3). This result was consistent with the previous recommendation of Menicanti and Di Donato[6] that poor basal function may be a relative contraindication for SVR. Because of poor postoperative recovery of systolic function, patients with poor basal function are likely to be a high-risk group, as previous studies have demonstrated.[1,3,4,6] Actually, in our population, patients with postoperative heart failure symptoms had significantly higher percentage of fibrosis (Table 4). Although our study did not have statistically sufficient power to clarify a risk factor for adverse outcome because of the small number of patients, our results do imply that remote myocardial fibrosis visible on DE-MRI could be a key determinant in patient selection for SVR. A further

TABLE 3. Comparison of preoperative variables according to improvement in ejection fraction

	*Δ*EF ≥10% (n = 9)	*Δ*EF <10% (n = 6)	*P* value
LV end-diastolic VI (mL/m^2)	162 ± 53	176 ± 52	.63
LV end-systolic VI (mL/m^2)	125 ± 53	144 ± 50	.51
LV ejection fraction (%)	21% ± 8.7%	19% ± 6.4%	.63
Mitral regurgitation grade	1.6 ± 1.6	2.1 ± 1.6	.54
Mean pulmonary arterial pressure (mm Hg)	21 ± 5.4	33 ± 11	.01
Pulmonary capillary wedge pressure (mm Hg)	14 ± 5.4	22 ± 7.9	.04
Cardiac index (L/[min · m^2])	2.4 ± 0.4	2.5 ± 0.4	.38
Brain natriuretic peptide (pg/mL)	726 ± 916	526 ± 433	.63
Percentage of fibrosis	5.4% ± 5.2%	19% ± 8.5%	.002

ΔEF, Change (improvement) in ejection fraction; *LV,* left ventricular; *VI,* volume index.

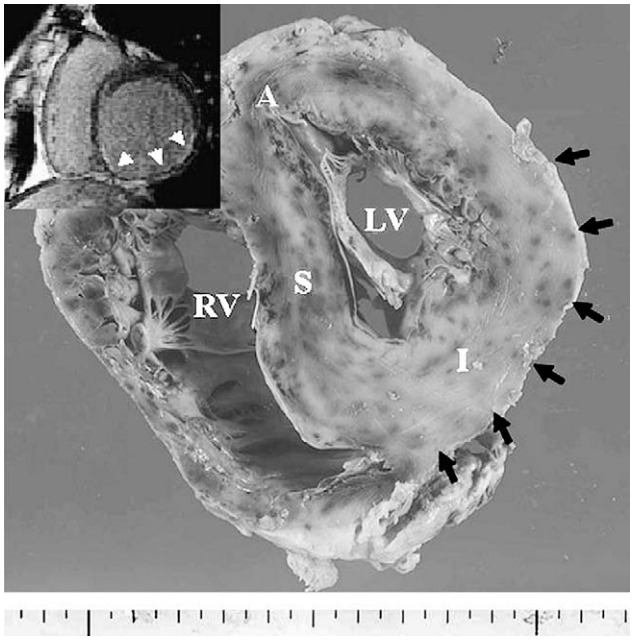

FIGURE 5. Heart specimen showing basal ventricular section 3.7 months after operation. Note broad inferoposterior transmural infarction *(black arrows)* consistent with findings on preoperative short-axis delayed-enhancement magnetic resonance imaging *(upper left inset, white arrows). A,* Anterior wall; *LV,* left ventricle; *S,* septum, *RV,* right ventricle; *I,* inferior wall.

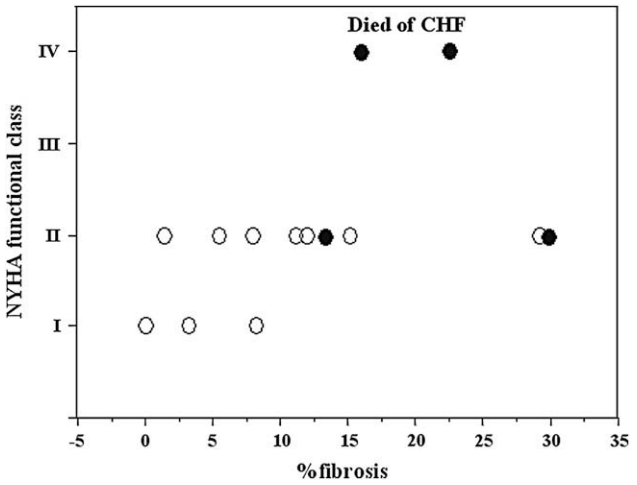

FIGURE 6. Relationship between latest New York Functional Heart Association *(NYHA)* functional class and percentage of fibrosis. *Filled circles* indicate patients with recurrent congestive heart failure *(CHF).* Two of these patients died, and 2 recovered from New York Heart Association class III to II after medical treatments.

study including a larger population is needed to elucidate an optimal cutoff value of percentage of fibrosis to predict late adverse outcomes.

LIMITATIONS

There are several limitations of this study. The number of patients was small, and selection bias is possible. Another limitation is the lack of later imaging studies. For instance, myocardium may take as long as 1 year to respond to coronary revascularization.[24] In our study, 10 of 15 patients underwent CABG concurrently with SVR. Time course of myocardial recovery may have influenced postoperative global LV function and degree of reverse LV remodeling.

Regarding the technical aspects, a variety of surgical procedures could influence outcomes. SVR is still not a standard technique, however, and the optimal choice remains to be investigated. We agree with Suma and associates[5] that the extent of visible endocardial scarring is not a reliable indicator of the borders between functioning and nonfunctioning wall, especially in broad akinetic aneurysms.[5] In this study period, with the aid of both preoperative DE-MRI evaluation of the extent of nonviable scar tissue and intraoperative myocardial inspection under the beating heart, we used the SAVE procedure for broad anteroseptal akinesia to exclude as much scar tissue as possible and to create the elliptical LV shape.[14]

Finally, quantification of myocardial hyperenhancement on DE-MRI was measured only at the LV base, because preoperative precise determination of the hyperenhancement area that is not excluded by the operation is often difficult at the mid to apical LV, especially in patients with broad anteroseptal scarring. Moreover, inferior and posterior myocardial infarctions usually extend from the basal to the apical area. Basal myocardial status thus represents extension of infarction at circumflex and right coronary artery system. That may be one explanation why the percentage of fibrosis at the basal area correlates with postoperative LV function. We therefore believe that assessment of the basal portion is the most simple and helpful method of prediction.

TABLE 4. Preoperative risk factors for late recurrence of heart failure

	No CHF (n = 11)	CHF (n = 4)	*P* value
LV end-diastolic VI (mL/m^2)	154 ± 47	206 ± 43	.07
LV end-systolic VI (mL/m^2)	119 ± 46	174 ± 39	.06
LV ejection fraction (%)	22 ± 8.0	17 ± 4.2	.22
Mitral regurgitation grade	1.7 ± 1.6	2.3 ± 1.7	.58
Mean pulmonary arterial pressure (mm Hg)	24 ± 10	30 ± 10	.33
Pulmonary capillary wedge pressure (mm Hg)	16 ± 6.6	22 ± 8.9	.16
Cardiac index (L/[min · m^2])	2.4 ± 0.4	2.5 ± 0.3	.63
Brain natriuretic peptide (pg/mL)	622 ± 794	709 ± 552	.86
Percentage of fibrosis	8.8% ± 8.6%	20% ± 8.2%	.04

CHF, Congestive heart failure; *LV,* left ventricular; *VI,* volume index.

CONCLUSION

In conclusion, the amount of LV basal scar tissue affected LV function and functional recovery after SVR. Patients with a large amount of LV basal scarring are a high-risk

group. Preoperative assessment of remote myocardial status with DE-MRI may predict surgical outcomes of patients undergoing SVR.

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