Determinants of in-hospital and long-term surgical outcomes after repair of postinfarction ventricular septal rupture

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Objectives: Surgical repair of post–myocardial infarction ventricular septal rupture is challenging with reported early mortality being substantial. In addition, congestive cardiac failure and ventricular tachyarrhythmia frequently occur long term after the operation, although frequency and predictive factors of these events have been poorly identified.

Methods: A consecutive series of 68 patients who underwent repair of postinfarction ventricular septal rupture by 14 surgeons between 1988 and 2007 was studied. Fifty-eight (85%) patients underwent repair in an urgent setting (<48 hours after diagnosis). Coronary artery bypass grafting was concomitantly performed in 48 (71%) patients. Mean follow-up period was 9.2 ± 4.9 years.

Results: Thirty-day mortality was 35%, with previous myocardial infarction, previous cardiac surgery, preoperative left ventricular ejection fraction less than 40%, and urgent surgery being independent risk factors. Actuarial survival of 30-day survivors was 88% at 5 years, 73% at 10 years, and 51% at 15 years. Actuarial freedom from congestive cardiac failure and ventricular tachyarrhythmia was 70% and 85% at 5 years, 54% and 71% at 10 years, and 28% and 61% at 15 years, respectively. Independent predictors for congestive cardiac failure included hypertension, posterior septal rupture, residual interventricular communication, and preoperative left ventricular ejection fraction less than 40%, whereas concomitant ventricular aneurysmectomy and preoperative occlusion of the left anterior descending artery were independent predictors of ventricular tachyarrhythmia.

Conclusions: Long-term outcomes after surgical repair of postinfarction ventricular septal rupture was favorable, despite infrequent exposure by individual surgeons to the pathologic features, indicating that an aggressive surgical approach is warranted. Predictors of congestive cardiac failure and ventricular arrhythmia long term varied. (J Thorac Cardiovasc Surg 2010;140:59-65)

Ventricular septal rupture (VSR) after myocardial infarction (MI) is an infrequent condition with extremely high mortality, inasmuch as significant VSR eventually leads to congestive cardiac failure and cardiogenic shock without treatment.[1–3] Current guidelines of the American College of Cardiology and the American Heart Association recommend immediate operative intervention in patients with postinfarction VSR regardless of their clinical status.[4] Nonetheless, surgical repair of VSR is still challenging, with reported in-hospital mortality being substantial (20%–60%).[2,5–7] Preoperative hemodynamic instability, posterior septal rupture, and incomplete revascularization have been suggested as risk factors for high in-hospital mortality after repair, although they are still inconsistent.[3,6–15]

Although long-term survival after repair of postinfarction VSR has been reported to be favorable, congestive cardiac failure and ventricular tachyarrhythmia are known to frequently impair quality of life long term.[3,6,7,9,11,12,16,17] Transmural MI generating a VSR affects ventricular function and has the capacity to generate a macro-reentry circuit, inducing congestive cardiac failure and ventricular tachyarrhythmia, respectively. In addition, repair of ruptured septum with excision or exclusion of the infarcted myocardium might also alter ventricular geometry to adversely affect long-term ventricular function and electrical stability. Importantly, predictive factors of these events are poorly understood.

The goals of this study were to investigate in-hospital and long-term outcomes after VSR repair and to identify factors affecting congestive cardiac failure and ventricular tachyarrhythmia long term.

METHODS

A consecutive series of all patients who underwent surgical repair of postinfarction VSR in The Prince Charles Hospital between 1988 and 2007 was enrolled. The prospective departmental database identified that

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Abbreviations and Acronyms

CABG = coronary artery bypass grafting
LAD = left anterior descending coronary artery
LV = left ventricular
LVEF = left ventricular ejection fraction
MACE = main adverse coronary event
MI = myocardial infarction
VSR = ventricular septal rupture

68 (0.18%) patients among a total of 37,177 undergoing cardiac surgery were operated on by 14 individual surgeons during this study period. Additional information was obtained from medical records and interviews. This study was approved by the institutional ethics committee (Ref.; EC2893). Management strategy for VSR has been consistently (1) urgent preoperative angiography and (2) subsequent urgent or semiurgent VSR repair with complete revascularization over the study period

Diagnosis and Preoperative Management for Postinfarction VSR

Coronary angiography was preoperatively performed in 65 (96%) patients, whereas 2 patients with cardiogenic shock and 1 patient with impaired renal function underwent urgent VSR repair without coronary angiography. Of 65 patients who underwent coronary angiography preoperatively, 20 (31%) patients had a single diseased major coronary artery, whereas 45 (69%) patients had multiple coronary artery disease. The number of diseased coronary arteries was not significantly different over the study period. Percutaneous coronary intervention was preoperatively performed in 3 patients (4%).

Surgical Strategy for Postinfarction VSR

Fifty-eight (85%) patients underwent surgery in an urgent setting (<48 hours after diagnosis of VSR), whereas 10 (15%) patients underwent surgery in an elective setting inasmuch as the diagnosis was made incidentally without hemodynamic instability. All the operations were carried out with the aid of cardiopulmonary bypass with moderate hypothermia and cardiac arrest induced by infusing cardioplegic solutions antegradely and retrogradely every 20 minutes. Cold crystalloid predominated as the cardioplegic solution in the initial 10 years, whereas cold oxygenated blood predominated in the subsequent 10 years. Approaches to the ruptured septum were left ventriculotomy in 48 (71%) patients, right ventriculotomy in 12 (18%) patients, and both ventriculotomies in 6 (9%) patients, left atriotomy with mitral valve annulus being detached and reattached in 1 patient and right atriotomy through tricuspid valve in 1 patient, depending on the infarct area. A patch was placed to repair the ruptured septum in 67 (99%) patients, using artificial material in 50 (74%), bovine pericardium in 15 (22%), and autologous pericardium in 2 (3%), depending on the size and shape of the VSR. Infarctectomy with septal reconstruction[18] predominated in the initial 5 years, whereas minimal infarctectomy with a patch placement to exclude infracted, ruptured septum[19] predominated in the subsequent 15 years.

Coronary artery bypass grafting (CABG) was concomitantly performed in 48 (71%) patients with an average number of bypass grafts being 1.7. A side branch of the culprit coronary artery for infarct VSR was revascularized in 22 (32%) patients. In 15 patients, significantly diseased coronary arteries in the remote area from the culprit artery were not revascularized owing to ungraftable quality and/or size, defined as incomplete revascularization. The left internal thoracic artery was grafted to the left anterior descending artery (LAD) in 3 patients who were hemodynamically stable and had severe multiple coronary artery disease, whereas saphenous veins were used in the other grafts. Concomitant left ventricular (LV) aneurysmectomy was performed in 4 (6%) patients having aneurysmal formation in the LV apex. Concomitant mitral valve surgery was performed in 1 patient having moderate mitral regurgitation. A bilateral ventricular assist device was implanted for global myocardial dysfunction in 1 patient. All patients were postoperatively examined by echocardiography in the intensive care unit.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation and/or median (interquartile range). Categorical variables are shown as the percentage of the sample. Comparison between 30-day survivors and nonsurvivors was performed by the Student t test, χ^2 test, or Fisher exact test as appropriate. Independent risk factors of 30-day mortality were identified by multivariate logistic regression analysis. The potential predictors entered to logistic regression are those showing $P < .100$ at the univariate analysis (Table 1). Actuarial survival, freedom from main adverse coronary events (MACE), freedom from congestive cardiac failure, and freedom from ventricular tachyarrhythmia were estimated by the Kaplan–Meier method. Independent predictive factors for congestive cardiac failure and ventricular arrhythmia were identified by the Cox proportional hazard model. The potential predictors entered to the Cox proportional hazard model are those that showed $P < .500$ in an initial proportional hazard model including the following predictors: year of surgery, 70 years of age or more at surgery, female gender, hypertension, diabetes, previous MI, preoperative serum creatinine greater than 250 μmol/L, MI onset until surgery less than 48 hours, posterior septal rupture, preoperative LV ejection fraction (LVEF) less than 40%, 3-vessel disease, occlusion of the LAD, incomplete revascularization, not-revascularized culprit artery, concomitant aneurysmectomy, and residual interventricular communication. Statistical analysis was performed using StatView-J 5.0 (SAS Institute, Inc, Cary, NC).

RESULTS

Thirty-Day Mortality and Morbidities

Among a total of 68 patients, 24 (35%) deaths occurred within 30 days after repair of postinfarction VSR at a median of 3.5 postoperative days (0–27 days). Five (7%) patients died in the operating room owing to global myocardial dysfunction. Causes of the deaths in the patients who were successfully transferred to the intensive care unit after the operation were persistent low cardiac output in 13 patients, sepsis in 2 patients, ventricular arrhythmia in 1 patient, uncontrollable bleeding from a ventricle in 1 patient, and anemia in 2 patients who rejected blood transfusion on religious grounds. Six (10%) patients underwent re-exploration for postoperative bleeding. Three (5%) patients had permanent neurologic deficits, and 3 (5%) had transient neurologic disorders. Hemodialysis was carried out for acute renal failure in 12 (19%) patients. Of them, 7 patients died within 30 days postoperatively, whereas 5 patients survived and did not require further hemodialysis. Four patients underwent intervention of residual interventricular communication within 30 days postoperatively.

Risk Factors for 30-Day Mortality

Comparison between 30-day survivors and nonsurvivors was carried out in a univariate manner (Table 1). Of 5 patients who had previous cardiac surgery, only 1 patient

TABLE 1. Thirty-day survivors versus nonsurvivors

	Survivors (n = 44)	Nonsurvivors (n = 24)	*P* value
Background			
Year of surgery			.387
1988–1992	20 (63%)	12 (38%)	
1993–1997	13 (81%)	3 (19%)	
1998–2002	5 (50%)	5 (50%)	
2003–2007	6 (69%)	4 (40%)	
Age	66 ± 10	67 ± 8	.698
Female gender	14 (32%)	5 (21%)	.335
Hypertension	28 (64%	13 (54%)	.446
Diabetes	7 (16%)	6 (25%)	.520
Previous MI	7 (16%)	8 (33%)	.098
Previous cardiac surgery	1 (2%)	4 (17%)	.049
Preoperative condition and pathology			
Heart rate (beats/min)	103 ± 17	109 ± 17	.194
Systolic BP < 100 mm Hg	16 (36%)	21 (88%)	<.0001
Serum creatinine > 250 μmol/L	5 (11%)	8 (33%)	.050
Inotrope support	12 (27%)	13 (54%)	.028
IABP support	10 (23%)	18 (75%)	<.0001
Mechanical ventilation	2 (5%)	3 (13%)	.337
MI onset until surgery < 48 hours	14 (32%)	5 (21%)	.335
Posterior septal rupture	20 (46%)	13 (54%)	.492
LVEF < 40%	14 (32%)	16 (67%)	.006
Three-vessel disease*	16 (37%)	6 (27%)	.423
Complete occlusion of LAD*	23 (54%)	7 (32%)	.097
Perioperative variables			
Urgent operation	35 (80%)	23 (96%)	.085
Concomitant CABG	31 (71%)	17 (71%)	.974
Greater than 3 bypass grafts	3 (7%)	2 (8%)	1.000
Incomplete revascularization	5 (11%)	10 (42%)	.004
Not-revascularized culprit artery	17 (39%)	5 (23%)	.178
Concomitant aneurysmectomy	4 (9%)	0 (0%)	.289
CPB time > 150 minutes	7 (16%)	8 (33%)	.098
Cardiac arrest time > 100 minutes	7 (16%)	5 (21%)	.611

MI, Myocardial infarction; *BP*, blood pressure; *IABP*, intra-aortic balloon pumping; *LVEF*, left ventricular ejection fraction; *LAD*, left anterior descending artery; *CABG*, coronary artery bypass grafting; *CPB*, cardiopulmonary bypass. *The analysis was performed among the patients who preoperatively underwent coronary angiography (n = 43 in 30-day survivors and n = 22 in nonsurvivors).

survived; the other 4 patients died of persistent low cardiac output postoperatively. Preoperative data identifying hemodynamic instability, by low systolic blood pressure, serum creatinine level greater than 250 μmol/L, inotrope support, and intra-aortic balloon pump support, were significantly different between 30-day survivors and nonsurvivors. Of note, all 8 patients who had cardiogenic shock, including systolic blood pressure less than 100 mm Hg and oliguria with serum creatinine level elevation under inotropic support, died within 30 days postoperatively. Multivariate logistic regression analysis was performed among the patients who preoperatively underwent coronary angiography (n = 43 in 30-day survivors and n = 22 in nonsurvivors). Independent risk factors of 30-day mortality included previous MI ($P = .022$), previous cardiac surgery ($P = .013$), preoperative LVEF < 40% ($P = .046$), and urgent surgery ($P = .016$). Incomplete revascularization was not an independent risk factor ($P = .100$).

Actuarial Survival and Freedom From MACE

Actuarial survival of all patients was 67% at 1 year, 63% at 5 years, 51% at 10 years, and 36% at 15 years (Figure 1, *A*). The follow-up period of 30-day survivors was 9.2 ± 4.9 years with a median being 9.3 years (0.1–19.7 years). Among 30-day survivors, 16 (36%) deaths occurred at 7.8 ± 4.9 years with a median of 8.9 years (0.1–17.1 years) over the study period. Thirteen deaths were confirmed as cardiac death. Of them, 8 deaths were related to congestive cardiac failure. Three patients had out-of-hospital cardiac arrest owing to cardiac arrhythmia suggested by autopsy. Two patients died in the hospital after surgery for residual interventricular communication and LV apex aneurysm, respectively. Three patients died of noncardiac causes. Actuarial survival of 30-day survivors was 95% at 1 year, 88% at 5 years, 73% at 10 years, and 51% at 15 years (Figure 1, *B*). MACE included congestive cardiac failure (15 patients), ventricular tachyarrhythmia (6 patients), and redo operation for LV aneurysm (1 patient). No patients had acute coronary syndrome during the follow-up. Actuarial freedom from MACE of 30-day survivors was 91% at 1 year, 61% at 5 years, 40% at 10 years, and 19% at 15 years (Figure 1, *C*).

Congestive Cardiac Failure

A total of 20 (45.5%) patients had congestive cardiac failure at 5.4 ± 4.4 years with a median of 3.9 years (0.1–15.2 years). Among them, 7 patients were related to residual interventricular communication and underwent surgical repair. One patient had severe ischemic mitral regurgitation and underwent mitral valve replacement. Actuarial freedom from congestive cardiac failure of 30-day survivors was 93% at 1 year, 70% at 5 years, 54% at 10 years, and 28% at 15 years (Figure 2, *A*). The Cox proportional hazards model identified history of hypertension, posterior septal rupture, residual interventricular communication, and preoperative LVEF less than 40% as the predictors of congestive cardiac failure over the long term (Table 2).

Ventricular Tachyarrhythmia

Twelve (27%) patients had ventricular tachyarrhythmia treated in the hospital at 6.2 ± 4.0 years with a median 5.9 years (0.4–13.0 years). Among them, 8 patients received an automatic implantable cardiac defibrillator, whereas 4 patients who presented after 1 or 2 episodes of nonsustained ventricular tachycardia with preserved LV function were treated medically. Actuarial freedom from ventricular tachyarrhythmia of 30-day survivors was 98% at 1 year, 85% at 5

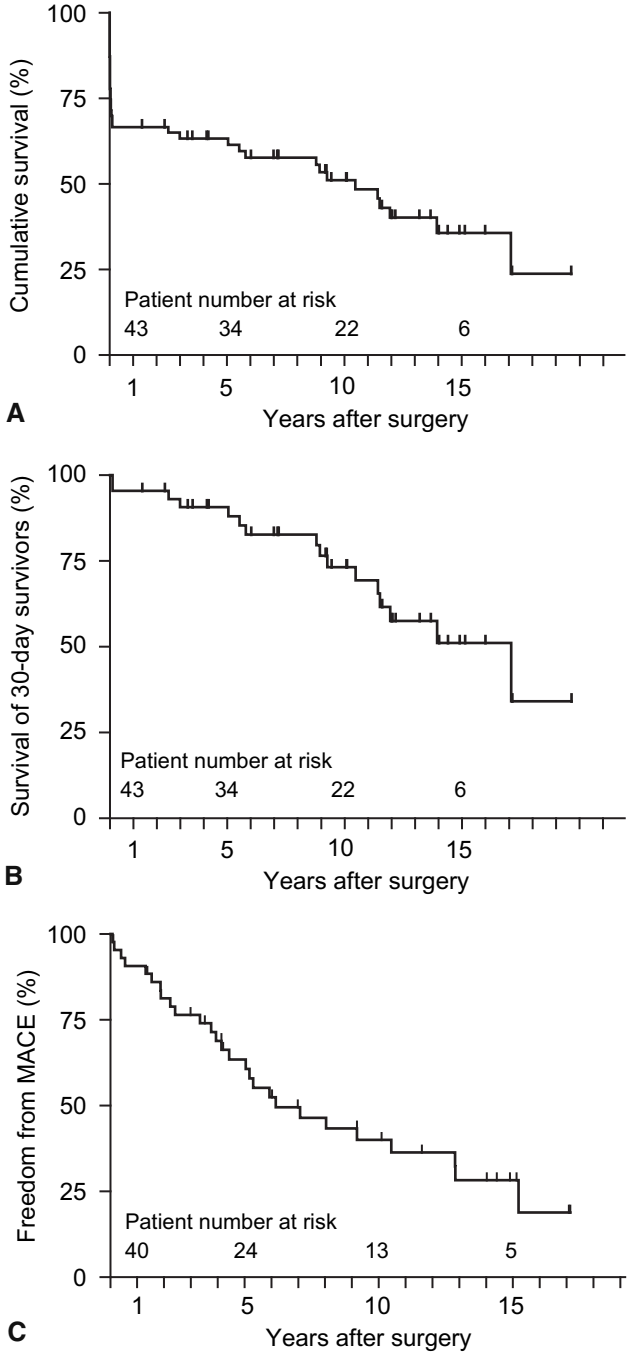

FIGURE 1. Actuarial survival of all patients (n = 68) was estimated by the Kaplan–Meier method (A). Actuarial survival (B) and freedom from main adverse coronary events (*MACE*; C) of 30-day survivors (n = 44) were also estimated by the Kaplan–Meier method.

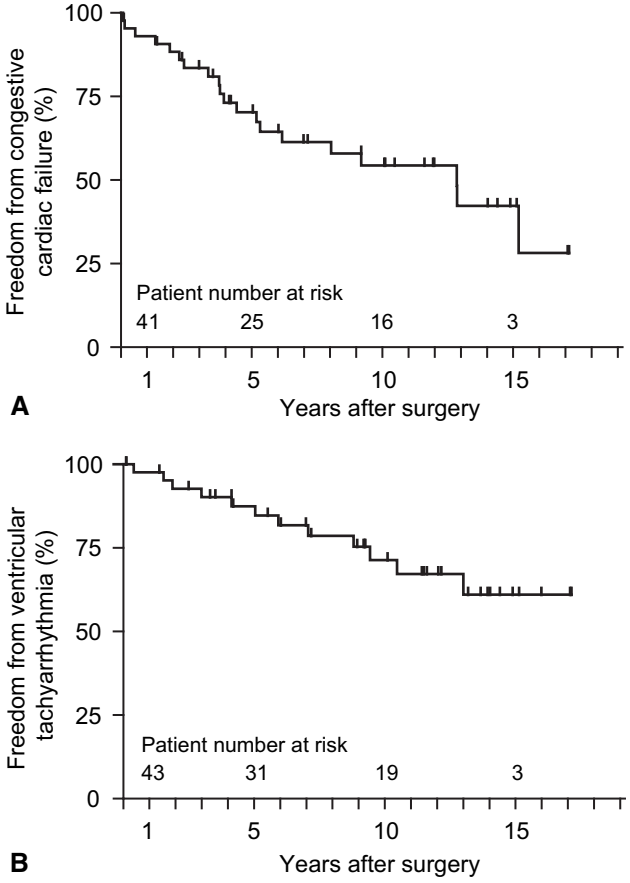

FIGURE 2. Actuarial freedom from congestive cardiac failure (A) and ventricular tachyarrhythmia (B) of 30-day survivors (n = 44) were estimated by the Kaplan–Meier method.

years, 71% at 10 years, and 61% at 15 years (Figure 2, *B*). The Cox proportional hazards model identified concomitant LV aneurysmectomy and complete occlusion of the LAD as significant independent predictors of ventricular tachyarrhythmia over the long term (Table 3).

Residual or Recurrent Interventricular Communication

Residual or recurrent interventricular communication was echocardiographically detected in 22 (35%) of 63 patients who transferred to the intensive care unit postoperatively. Three patients had persistent severe congestive cardiac failure postoperatively, which was associated with a significant interventricular communication as estimated by transesophageal echocardiography, eventually requiring reintervention in an urgent setting within 5 days after VSR repair. Among them, surgical repair was performed in 2 patients, who were discharged from the hospital without further morbidities, whereas percutaneous repair was successfully performed using an Amplatzer septal occluder[20] in 1 patient, who later died of anemia that could not be treated because of the patient's religious convictions. Six patients underwent surgical repair of residual/recurrent communication for refractory congestive cardiac failure associated with a significant interventricular communication in a semiurgent/elective setting between 24 and 202 days after VSR repair. Of them, 1 patient died in the operating room of global

TABLE 2. Independent predictors of congestive cardiac failure

Predictors	Odds ratio	95% CI	*P* value
History of hypertension	6.14	1.72–21.9	.005
Diabetes	0.35	0.03–3.62	.377
Preoperative serum creatinine > 250 μmol/L	4.62	0.52–40.8	.169
MI onset until surgery < 48 hours	2.44	0.68–8.83	.173
Posterior septal rupture	4.44	1.24–15.8	.022
Preoperative LVED < 40%	3.86	1.33–11.2	.013
Three-vessel disease	0.75	0.23–2.39	.620
Residual interventricular communicaiton	4.41	1.22–15.9	.024

CI, Confidence interval; *MI*, myocardial infarction; *LVEF*, left ventricular ejection fraction.

myocardial dysfunction, whereas the other 5 patients survived until the completion of follow-up of this study without further MACE. Two patients successfully underwent surgical repair of residual/recurrent interventricular communication at 2 years and 10 years after VSR repair without further MACE. Dehiscence between the patch and infarcted myocardium produced an interventricular communication in all patients who underwent surgical repair of residual communication. The other 11 patients who did not undergo repair of residual communication until the completion of this study had been asymptomatic with persisting minimal communication (echocardiographic pulmonary/systemic flow ratio < 1.5) and preserved LV function.

DISCUSSION

We here demonstrate 30-day and long-term (mean follow-up period of 9.2 years) clinical outcomes of 68 patients who underwent surgical repair of postinfarction VSR in a single institution that performed more than 35,000 open cardiac operations between 1988 and 2007. Thirty-day mortality was 35%, with previous MI, previous cardiac surgery, preoperative LVEF less than 40%, and urgent surgery being independent risk factors. Residual interventricular communication was echocardiographically detected in 22 patients and 11 patients underwent reintervention. Actuarial survival and freedom from MACE of 30-day survivors were 88% and 61% at 5 years, 73% and 40% at 10 years, and 51% and 19% at 15 years, respectively. Actuarial freedom from congestive cardiac failure and ventricular tachyarrhythmia of 30-day survivors were 70% and 85% at 5 years, 54% and 71% at 10 years, and 28% and 61% at 15 years, respectively. Interestingly, independent predictors were different between congestive cardiac failure and ventricular tachyarrhythmia. Independent predictors for congestive cardiac failure included history of hypertension, posterior septal rupture, residual interventricular communication, and preoperative LVEF less than 40%, whereas concomitant LV aneurysmectomy and complete occlusion of the LAD were independent predictors of ventricular tachyarrhythmia.

TABLE 3. Independent predictors of ventricular tachyarrhythmia

Predictors	Odds ratio	95% CI	*P* value
History of hypertension	0.13	0.01–1.25	.077
Previous MI	2.98	0.33–26.8	.329
Preoperative serum creatinine > 250 μmol/L	9.66	0.77–122	.079
MI onset until surgery < 48 hours	3.06	0.58–16.2	.189
Preoperative LVEF < 40%	1.97	0.52–7.51	.318
Three-vessel disease	0.41	0.09–1.94	.258
Occlusion of LAD	6.00	1.24–29.1	.026
Concomitant LV aneurysmectomy	14.80	1.64–134	.016

CI, Confidence interval; *MI*, myocardial infarction; *LVEF*, left ventricular ejection fraction; *LAD*, left anterior descending artery; *LV*, left ventricle.

Thirty-day mortality after repair of postinfarction VSR in this study (35%) was comparable with the majority of previous reports.[1,3,6,7,9,11–14,17] Consistent with previous reports,[3,6,7,9,13] all patients who had cardiogenic shock died within 30 days, suggesting that prompt diagnosis and assessment followed by urgent surgical intervention before hemodynamic deterioration would be particularly important to improve in-hospital outcomes. In addition, preoperative poor LV function was an independent risk factor, suggesting that procedures to improve LV contractility, such as CABG, might be important to improve outcomes, although incomplete revascularization was not an independent risk factor in this study. Urgent surgery was an independent predictor for 30-day mortality in this study. However, this finding not only suggests that urgent surgery elevates the risk of in-hospital mortality, but also indicates that mortality of elective intervention would be very low. Previous reports have suggested that posterior septal rupture is a risk factor of 30-day mortality owing to its more complex pathology than anterior septal rupture, including involvement of the subvalvular apparatus and right ventricular function.[11,12] This study, however, does not indicate posterior septal rupture as a risk factor. Extensive exposure of infarct area via ventriculotomy and/or atrioventricular valves to securely repair the ruptured septum might minimize residual interventricular communication and alteration in ventricular geometries, consequently improving in-hospital outcomes. A short interval between MI onset and surgical intervention has also been reported to be a risk factor of 30-day mortality, suggesting that early presentation of VSR represents extensive myocardial necrosis, leading to poor outcomes.[2,9,12,17] This study, however, indicates that MI onset until VSR repair of less than 48 hours is not a risk factor of 30-day mortality. Such a discrepancy could be explained by a difference in management strategy.[7] In our strategy, surgery was carried out urgently after the diagnosis of VSR, whereas Blanche,[17] Jones,[12] and their associates delayed the surgical repair of VSR unless hemodynamics was compromised.[12,17]

Preoperative coronary angiography has been routinely performed in patients with a diagnosis of postinfarction VSR in our institution in the past 20 years. Recently, VSR associated with multivessel coronary artery disease is reported to be more prevalent than that associated with occlusion of a single major coronary artery with other arteries being intact, which was common in the prethrombolysis/reperfusion era.[2] In our series, there were no differences in coronary artery pathologic conditions over the past 20 years, with 69% of patients having multivessel coronary artery disease. As a result, concomitant CABG was performed in 71% of total VSR patients. This finding suggests that coronary angiography is a very important preoperative diagnostic tool in postinfarct VSR to possibly improve in-hospital and long-term surgical outcomes with sufficient supporting evidence.[6,7,14,15]

Residual interventricular communication was detected in 22 (32%) patients. Of them, 11 (50%) underwent intervention for residual communication, whereas the other 11 patients were asymptomatic with a small residual communication. It is important to carefully define the suture line of the patch to repair the VSR intraoperatively, inasmuch as progressive necrosis of myocardium near the suture line leads to dehiscence, as seen in our series and previous reports.[21] Although delayed repair might allow secure repair to prevent residual/recurrent interventricular communication,[12,17] it also carries a substantial risk of hemodynamic deterioration.[4] Most important, prompt and proper diagnosis and management of residual interventricular communication are essential to improve clinical outcomes. In the early period after VSR repair, transesophageal echocardiography was particularly useful in diagnosing the cause of cardiac failure among a variety of possible causes. We successfully diagnosed and repaired residual communication in 3 patients within 5 days after VSR repair. Moreover, residual communication was the leading predictor of congestive cardiac failure long term. We performed surgical intervention in 6 patients within 1 year and 2 patients in a later period after VSR repair with 1 in-hospital mortality, illustrating that close follow-up is required for patients having residual communication. Although surgery is the standard strategy for intervention of residual communication, percutaneous device closure is also promising. Although percutaneous closure of postinfarction VSR has been suggested to carry technical difficulties, such as placement of the device without distorting ventricular geometry or causing atrioventricular regurgitation,[2] tissues where the device is placed in residual communication might be more supportive compared with necrotic myocardium in postinfarction VSR, as we successfully performed in 1 patient 5 days after VSR repair.

This study demonstrates long-term outcomes for postinfarction VSR with a mean follow-up being 9.2 years. Five- and 10-year actuarial survival of 30-day survivors in this study (88% and 73%, respectively) was consistent with the majority of previous studies.[7–9,11–14,16] Although 2 recently published reports[6,7] demonstrated long-term survival after VSR repair and Lundblad and associates[7] identified independent risk factors of long-term mortality, incidences of MACE or factors affecting MACE after VSR repair are still poorly understood.[11,16] We here clearly demonstrate substantial incidences of MACE and independent risk factors of congestive cardiac failure and ventricular tachyarrhythmia.

Congestive cardiac failure occurred in 46% of 30-day survivors. History of hypertension and residual interventricular communication were leading predictors of long-term congestive cardiac failure. Other predictors included preoperative LVEF less than 40%, which might be due to pre-existing cardiac disease or necrotic/hibernating LV myocardium. Interestingly, posterior septal rupture was also an independent predictor of congestive cardiac failure over the long term. More complex pathologic conditions in posterior rupture than anterior rupture might affect long-term systolic and diastolic ventricular function. In addition, patch repair of posterior VSR might alter geometry of the base of the ventricular myocardium and/or affect the subvalvular apparatus, resulting in ventricular dysfunction. These findings suggest that intensive medical treatment by angiotensin-converting enzyme inhibitor, β-blocker, or aldosterone blocker might need to be commenced in the early period depending on background and perioperative variables.

In this study, 27% of 30-day survivors had ventricular tachyarrhythmia, whereas Davies and colleagues[16] identified that 40% of patients showed frequent ventricular premature beats and only 5% of 43 patients had ventricular tachycardia after VSR repair. This discrepancy might be explained by difference in follow-up period (median follow-up of 114 months in this study vs 30 months in Davies' study[16]). Concomitant LV aneurysmectomy and occlusion of the LAD were predictors of ventricular tachyarrhythmia long term, suggesting that extensive myocardial infarction followed by full-thickness scar formation at the ventricle would be the most important factor to induce ventricular tachyarrhythmia.

Some limitations are due to the retrospective nature. First, preoperative right ventricular function, which has been suggested to be a risk factor of in-hospital mortality,[8,12,13,19] was not included in the analysis, inasmuch as this parameter was not consistently measured throughout the study period. Second, surgical procedures, including myocardial protection, approach, patch placement technique, or patch materials, were slightly different over the study period and operating surgeons. Third, medical treatment long term varied, although all the patients were treated by specialist surgeons or physicians. Finally, this study could not identify any differences in 30-day or long-term outcomes over the study period, despite substantial changes in perioperative managements. Considering high in-hospital mortality,

randomized controlled trials for perioperative management would be difficult. Therefore, prospective collection of data in a multicenter basis is needed to verify the results of this study and to further improve in-hospital and long-term surgical outcomes of postinfarction VSR.

In conclusion, prompt management is particularly important to improve in-hospital and long-term outcomes for postinfarction VSR. Long-term outcomes after surgical repair of post-MI VSR were favorable, despite infrequent exposure by individual surgeons to the pathologic conditions, indicating that aggressive surgical approach is warranted. As predictors of congestive cardiac failure and ventricular arrhythmia varied over the long term, intensive medical treatment depending on background or perioperative variables might be effective to improve long-term outcomes.

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