Sutureless repair for postinfarction left ventricular free wall rupture

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ABSTRACT

Objective: Left ventricular free wall rupture is a catastrophic complication of acute myocardial infarction. Sutureless repair has been reported to be an effective surgical procedure for left ventricular free wall rupture. However, the outcomes of sutureless repair remain unclear.

Methods: Between January 2001 and December 2016, 42 patients were treated for left ventricular free wall rupture at Jichi Medical University. Of them, 35 consecutive patients undergoing sutureless repair using the TachoComb (CSL Behring, Tokyo, Japan) or TachoSil (Nycomed, Zurich, Switzerland) patches were included in this study. No patient required cardiopulmonary bypass. The oozing type of left ventricular free wall rupture was observed in 33 patients (94%), and the blow-out type was observed in 2 patients (6%). The rupture sites were the anterior wall in 16 patients (46%), the posterior-lateral wall in 11 patients (31%), and the inferior wall in 8 patients (23%).

Results: The in-hospital mortality rate was 17% (6 patients). Re-rupture after sutureless repair occurred in 17% (6 patients). Of them, 4 cases (67%) of re-rupture occurred within 24 hours after surgery. The 2 patients with blow-out type left ventricular free wall rupture experienced re-rupture. Three patients required mitral valve surgery after sutureless repair during the admission. The overall survivals at 1, 5, and 10 years were 71.4%, 68.6%, and 62.9%, respectively. Multivariable analysis revealed that re-rupture was an independent predictor for decreased survival (hazard ratio, 58.6; 95% confidence interval, 4.9-701.6; $P = .001$). Postoperative pseudoaneurysm formation was not detected during the follow-up.

Conclusions: Sutureless repair using TachoComb/TachoSil patches can be a viable treatment option for left ventricular free wall rupture. Care should be taken when applying this technique in cases of the blow-out type left ventricular free wall rupture. (J Thorac Cardiovasc Surg 2019;158:771-7)

Sutureless repair for postinfarction LVFWR.

Central Message

Sutureless repair using a TachoComb (CSL Behring, Tokyo, Japan) or TachoSil (Nycomed, Zurich, Switzerland) for postinfarction LVFWR can be a safe, simple, and effective method in selected patients.

Perspective

In the treatment of postinfarction LVFWR, the outcomes of sutureless repair remain unknown because of the lack of studies on a large number of cases. Our findings show that sutureless repair using a TachoComb (CSL Behring, Tokyo, Japan) or TachoSil (Nycomed, Zurich, Switzerland) can be an easy and effective treatment option, although it requires careful surveillance postoperatively for re-rupture.

See Commentary on page 778.

Left ventricular free wall rupture (LVFWR) remains one of the life-threatening complications stemming from an acute myocardial infarction (AMI). LVFWR develops in up to 2% of the patients with AMI and accounts for 14% to

26% of the mortality after AMI.^{1,2} The mortality rate of LVFWR reportedly is as high as 42.9%, which requires prompt diagnosis and surgical repair.³

The tissue of the rupture site is usually vulnerable because of AMI, and careful surgical repair is required. Several surgical procedures for LVFWR have been developed, including direct suturing with felt, the use of pericardial patches, and sutureless repair.⁴⁻⁶ However, the

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Abbreviations and Acronyms

AMI	= acute myocardial infarction
CABG	= coronary artery bypass grafting
CPB	= cardiopulmonary bypass
LAD	= left anterior descending
LCX	= left circumflex
LVFWR	= left ventricular free wall rupture
RCA	= right coronary artery

outcomes of surgery for LVFWR have been not well studied because of the limited number of cases.

Recently, sutureless repair has gained popularity as a simple and safe surgical technique for LVFWR, resulting in a decrease in operative mortality compared with that for conventional direct closure with felt-supported sutures or infarctectomy.⁵⁻⁸ Several types of patches can be used for this procedure: an autologous pericardium, xenopericardium, synthetic materials, and an extracellular matrix.⁹ Sakaguchi and colleagues⁵ reported the clinical outcomes of 32 consecutive patients with an oozing type of LVFWR undergoing sutureless repair using an autologous pericardial patch and biological glue. However, pericardial patches can be degraded or calcified in the long term.

Sutureless repair using hemostatic collagen patches has been reported as a safe and easy method.^{6-8,10} However, the literature on sutureless repair with the collagen patches is mainly case reports. Therefore, little is known about the safety and efficacy of sutureless techniques with collagen patches. This study aimed to evaluate the outcomes after sutureless repair using collagen patches for LVFWR.

PATIENTS AND METHODS

Patient Population and Study Design

This was a retrospective study based on our institutional database. From January 2001 to December 2016, a total of 42 patients with LVFWR after AMI underwent surgical repair at Saitama Medical Center, Jichi Medical University. We excluded 5 patients undergoing direct suturing with felt reinforcement and 2 patients with patch attachment; for these 7 excluded patients, the in-hospital mortality rate was 57%. Thus, the remaining 35 patients undergoing sutureless repair using collagen sponges for LVFWR were included in this study. Preoperative patient characteristics are shown in Table 1. The diagnosis was made on the basis of clinical conditions, laboratory data, coronary angiography, and echocardiography. Our study was approved by the Institutional Review Board of Jichi Medical University, and the requirement for individual consent was waived.

Surgical Procedure

In our institution, sutureless repair was principally performed for LVFWR, except for the blow-out type of the condition, patients with aneurysmal formation, and those who concomitantly underwent ventricular septal rupture repair. In all patients, a median sternotomy was performed. Pericardiocentesis was performed when the hemodynamics

TABLE 1. Preoperative patient characteristics

Variable	Total (n = 35)
Age (y)	71.5 ± 9.5
Sex, female	14 (40.0)
Body surface area (m ²)	1.59 ± 0.17
Hypertension	25 (71.4)
Diabetes	12 (34.3)
Dyslipidemia	14 (40.0)
Atrial fibrillation	1 (2.9)
Cerebrovascular disease	3 (8.6)
Chronic kidney disease*	6 (17.1)
Preoperative coronary angiography	32 (91)
Preoperative percutaneous coronary intervention	19 (54)
Shock	25 (71.4)
Intra-aortic balloon pump	13 (37.1)
Extracorporeal membrane oxygenation	4 (11.4)

Values for continuous variables are expressed as mean ± standard deviation. Values for categorical variables are expressed as numbers (%). *Serum creatinine greater than 1.5 mg/dL.

rapidly deteriorated and we could not wait for emergency surgery. After clots and blood were removed from the pericardial space, the rupture site was carefully identified. The heart was elevated manually or using a Starfish heart positioner (Medtronic Inc, Minneapolis, Minn) if necessary. No patients required cardiopulmonary bypass (CPB). The locations of the rupture site were as follows: the anterior wall in 16 patients (46%), the posterior-lateral wall in 11 patients (31%), and the inferior wall in 8 patients (23%). According to the intraoperative findings, the oozing type was defined as no macroscopic tear on the infarcted area with blood oozing from the infarcted myocardium. The blow-out type was defined as a macroscopic defect of the epicardium with free communication between the left ventricular cavity and the pericardial space.¹¹ Thirty-three patients (94%) had the oozing-type LVFWR, and 2 patients (6%) had the blow-out type. In the latter group, sutureless repair could be performed because bleeding was minor. Subsequently, TachoComb (CSL Behring, Tokyo, Japan) or TachoSil (Nycomed, Zurich, Switzerland) ready-to-use hemostatic collagen sponges were secured to the surface of the infarcted area, with 3-minute manual compressions to widely cover the epicardial hematoma surrounding the tear (Figure 1, A and B). There was no intraoperative failure including intraoperative re-rupture. TachoSil was approved by the Food and Drug Administration in the United States in 2015. Fibrin glue was additionally applied around the collagen patches in 21 patients (60%) according to the surgeon's preference. Coronary artery bypass grafting (CABG) was concomitantly performed in 3 patients (9%). Whether concomitant CABG was performed on the nonculprit lesion was based on the target vessel, the probability of postoperative percutaneous coronary intervention, and the surgeon's preference (Video 1).

Location and Type of Left Ventricular Free Wall Rupture

Preoperative coronary angiography was performed in 32 patients (91%) when the hemodynamics were stable on admission. Nineteen patients (54%) underwent percutaneous coronary intervention before surgery for LVFWR. One patient underwent percutaneous coronary intervention postoperatively. The culprit coronary lesion was the left anterior descending (LAD) artery in 16 patients (46%), the left circumflex (LCX) artery in

FIGURE 1. Intraoperative images of sutureless repair for postinfarction LVFW. A, An oozing type of rupture of the anterior left ventricular wall caused by the occlusion of the LAD artery. The *arrow* indicates the rupture site. B, The TachoSil patches (Nycomed, Zurich, Switzerland) were secured onto the surface of the infarcted myocardium with 3 minutes of manual compressions. CPB was not used.

11 patients (31%), and the right coronary artery (RCA) in 8 patients (23%).

Follow-up

Postoperatively, echocardiography was performed daily in the intensive care unit to evaluate cardiac function, other postinfarction mechanical complications, and pericardial effusion. Follow-up data including survival and complications were obtained via a patient chart review and questionnaires mailed to patients or administered via telephone. Median clinical follow-up was 3.4 years (range, 0-13.3 years). The patient status was confirmed at least 2 years after surgery in all patients, except for those who died within 2 years after surgery or had undergone surgery within the past 2 years.

Statistical Analysis

Data were reported as mean $\pm$ standard deviation for continuous variables and as frequencies and percentages for categorical variables. Multivariable logistic regression analysis, with *P* less than .20 to include and *P* greater than .25 to exclude from the models, was performed with the stepwise method to identify independent predictors of in-hospital death and re-rupture. Variables included age, sex, body surface area, hypertension, diabetes, dyslipidemia, cerebrovascular disease, chronic kidney disease (serum creatinine >1.5 mg/dL), preoperative percutaneous coronary

intervention, shock, use of intra-aortic balloon pump or extracorporeal membrane oxygenation, blow-out type rupture, anterior AMI, posterolateral AMI, inferior AMI, and postoperative re-rupture. Variables with *P* less than .25 in the univariate analysis were subjected to multivariable analysis. Forward stepwise Cox proportional-hazards regression analysis was performed to evaluate the impact of preoperative data on survival. Low cardiac output syndrome was defined as the need for adrenaline, or a dosage of more than 5 μ g/kg/min dopamine or dobutamine, after correction of electrolytes and preload in the intensive care unit. Long-term survival was estimated using the Kaplan-Meier method. All analyses were performed with SPSS software version 25 (IBM Corp, Armonk, NY) and SAS version 9.4 (SAS Institute Inc, Cary, NC).

RESULTS

Patient Characteristics

Preoperative patient characteristics are shown in Table 1. The mean interval between the onset of AMI and the diagnosis of LVFW was 2.6 ± 2.4 days (range, 0-9 days). In 94% of cases, LVFW occurred within the first week after AMI. Three-vessel disease was found in 1 patient, 2-vessel disease was found in 5 patients, and single-vessel disease was found in 29 patients. The patient status was in shock in 71% of the cases. Fifteen patients (43%) preoperatively required an intra-aortic balloon pump or extracorporeal membrane oxygenation due to hemodynamic instability.

In-Hospital Outcomes

Early outcomes are shown in Table 2. The in-hospital mortality rate was 17% (6 patients). Causes of deaths included pneumonia in 3 patients and re-rupture with massive bleeding, arrhythmia, and heart failure in 1 patient each. Three patients underwent mitral valve surgery for severe secondary mitral regurgitation during admission, and all of them were discharged alive. Two patients (1 posterolateral and 1 inferior AMI) required mitral valve surgery for postinfarction papillary muscle rupture 12 and 5 days after sutureless repair, respectively. One patient with posterolateral AMI had severe secondary mitral regurgitation due to tethering and underwent mitral valve replacement 29 days after sutureless repair. Ventricular septal rupture occurred

VIDEO 1. An intraoperative video that demonstrates sutureless repair using TachoSil patches. Video available at: [https://www.jtcvs.org/article/S0022-5223\(19\)30359-9/fulltext](https://www.jtcvs.org/article/S0022-5223(19)30359-9/fulltext).

TABLE 2. Early outcomes

Variable	Total (n = 35)
In-hospital mortality	6 (17.1)
Reexploration for bleeding	1 (2.9)
Re-rupture	6 (17.1)
Low cardiac output syndrome	11 (31.4)
Hemodialysis	5 (14.3)
Tracheotomy	4 (11.4)
Stroke	3 (8.6)
Postinfarction ventricular septal rupture	1 (2.9)
Reoperation for secondary mitral regurgitation	3 (8.6)
Intensive care unit stay (d)	16 ± 17

Values for continuous variables are expressed as mean ± standard deviation. Values for categorical variables are expressed as numbers (%).

in 1 patient 5 days after sutureless repair and was treated surgically. Multivariable analysis showed that re-rupture was an independent predictor of in-hospital mortality as shown in Table 3.

Postoperative Re-Rupture

Re-rupture occurred in 6 patients (17%) after sutureless repair. The details of these patients are shown in Table 4. In all 5 patients with re-rupture who underwent reoperation, bleeding from the epicardium surrounding the collagen patches was noted. No bleeding through the collagen patches was found. Patch repair was performed

using a bovine pericardium with running or mattress sutures with pledgets. Direct closure was performed using mattress sutures with felt strips. Re-rupture occurred in the 2 patients with the blow-out type LVFWR. According to the type of coronary artery lesions, the rates of re-rupture were 25.0% in the RCA, 18.2% in the LCX, and 12.5% in the LAD. Although there was a decreased tendency for re-rupture to occur in infarction of the LAD area than in infarction of the LCX/RCA areas, no significant difference was detected between the rates of re-rupture and culprit coronary lesion. In 4 patients (67%) of the 6 re-rupture cases, re-rupture occurred within 24 hours after sutureless repair. The other 2 cases of re-rupture occurred 5 days and 7 days after surgery, respectively. Preoperative reperfusion by percutaneous coronary intervention was not associated with the rate of postoperative re-rupture ($P = .634$). In our cohort, no variable was identified as an independent predictor for re-rupture.

Echocardiography at Discharge and Follow-up

Echocardiography at discharge was performed in 21 patients (60%). The mean left ventricular ejection fraction was $42\% \pm 10\%$. Left ventricular diastolic and systolic dimensions were 55 ± 10 mm and 43 ± 8 mm, respectively. Among 29 patients who were discharged from the hospital, 14 (48%) had echocardiography performed at the follow-up. The mean interval between surgery and echocardiography was 2.7 ± 3.2 years. The mean left

TABLE 3. Predictors of in-hospital death

Variable	Univariate		Multivariable	
	OR (95% CI)	P value	OR (95% CI)	P value
Age, y	1.07 (0.96-1.19)	.231	1.20 (0.99-1.45)	.064
Sex (female)	1.64 (0.28-9.58)	.585	-	
Body surface area (m ²)	1.62 (0.01-297.52)	.856	-	
Hypertension	0.76 (0.12-5.01)	.777	-	
Diabetes	2.22 (0.37-13.22)	.380	-	
Dyslipidemia	1.64 (0.28-9.58)	.585	-	
Cerebrovascular disease	0.58 (0.00-7.25)	.764	-	
Chronic kidney disease*	3.13 (0.42-23.06)	.264	-	
Preoperative PCI	0.35 (0.06-2.25)	.270	-	
Shock	6.02 (0.60-1000)	.261	-	
IABP and/or ECMO	1.42 (0.24-8.26)	.699	-	
Blow-out type rupture	5.60 (0.30-104.94)	.249	-	
Anterior AMI	0.62 (0.10-3.91)	.607	-	
Posterolateral AMI	0.33 (0.03-3.18)	.336	-	
Inferior AMI	4.80 (0.74-31.08)	.100	8.77 (0.75-102.58)	.083
Re-rupture	8.67 (1.18-63.87)	.034	51.40 (1.62-1627)	.025

OR, Odds ratio; CI, confidence interval; PCI, percutaneous coronary intervention; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; AMI, acute myocardial infarction. *Serum creatinine greater than 1.5 mg/dL.

TABLE 4. Details of 6 cases of re-rupture after sutureless repair

Age, y Sex	Coronary artery lesions	Type of rupture	Timing of re-rupture	Treatment for re-rupture	Outcome	Cause of death
83 Female	LCX	Blowout	0 POD	Withdrawal	Dead	Bleeding
55 Male	LCX	Blowout	7 POD	Patch repair	Alive	-
72 Male	LAD	Oozing	5 POD	Direct closure	Dead	Stroke, pneumonia
67 Female	RCA	Oozing	0 POD	Sutureless repair	Dead	Sepsis
68 Male	LAD	Oozing	0 POD	Sutureless repair	Alive	-
58 Male	RCA	Oozing	0 POD	Patch repair	Alive	-

LCX, Left circumflex; POD, postoperative day; LAD, left anterior descending; RCA, right coronary artery.

ventricular ejection fraction and end-diastolic diameter were $50\% \pm 17\%$ and 50 ± 5 mm, respectively. No pseudoaneurysm formation was observed by transthoracic echocardiography.

Midterm Outcomes

Six late deaths occurred at follow-up. The causes of deaths included pneumonia in 3 patients and heart failure in 1 patient; the causes were unknown in 2 patients. Although few patients had long-term follow-up, the 1-, 5-, and 10-year survivals were 71.4%, 68.6%, and 62.9%, respectively (Figure 2). One patient underwent CABG and surgical ventricular reconstruction for an apical aneurysm 4 months postoperatively. Heart failure requiring admission occurred in 2 patients 34 and 94 months after surgery, respectively. As shown in Table 5, multivariable analysis revealed that age and re-rupture were independent predictors for decreased survival (hazard ratio, 1.23; 95% confidence interval, 1.07-1.43; $P = .005$, hazard ratio, 58.55, 95% confidence interval, 4.89-701.62; $P = .001$, respectively).

DISCUSSION

This is a report of a series of 35 consecutive patients undergoing sutureless repair with the collagen patch for LVFWR. Our study revealed that sutureless repair with

collagen patches is feasible and safe in patients with oozing-type LVFWR after AMI.

Sutureless repair has the advantage of mitigating the need for both suturing on the fragile infarcted myocardium and the use of CPB. Tissue in the infarcted myocardium is usually of poor quality and is difficult to suture. In severe conditions, the avoidance of CPB is expected to decrease post-CPB cytokine release and subsequently result in better recovery. Although a percutaneous intrapericardial fibrin-glue injection therapy has been reported as an alternative treatment,¹² surgical repair is the gold standard in the treatment of LVFWR. Mortality rates of traditional surgical repair have been reported to range from 34% to 43%.^{3,4} Several techniques for the sutureless repair of LVFWR have been reported with acceptable morbidity and mortality. Some authors have used the pericardium for sutureless repair.^{5,13} Sakaguchi and colleagues⁵ showed the results of 32 patients undergoing sutureless repair using the pericardium with an in-hospital mortality of 15.6%. Holubec and colleagues⁹ showed a successful case of sutureless repair using an acellular xenogeneic extracellular matrix. However, the autologous pericardium or xenopericardium generally undergoes calcification and degradation in the long term.⁹

TachoComb and TachoSil are ready-to-use sheets of fibrin tissue-adhesive collagen fleece and contain fibrinogen, thrombin, and aprotinin. Unlike TachoComb, TachoSil is free from bovine aprotinin and can be stored at room temperature. Sutureless repair with the collagen patch is fast, technically easy, and reproducible.⁶⁻⁸ In our series, no patients required CPB, even in the cases of lateral and posterior AMI. Compared with pericardial patches, the advantage of the collagen patch is that it is fast and has excellent pliability, and there is no need for stitching to the epicardium. Raffa and colleagues⁶ reported the outcomes of 6 consecutive patients who underwent sutureless repair using the collagen patch. However, as far as we know, there is no literature reporting the outcomes of this technique in a large series of cases.

Complications such as re-rupturing, left ventricular aneurysms, and pseudoaneurysm formation are concerns even after sutureless repair with TachoSil.¹⁴⁻¹⁷ Careful vital

FIGURE 2. Kaplan-Meier curve of overall survival with 95% confidence intervals after sutureless repair for postinfarction LVFWR using collagen patches. Median clinical follow-up was 3.4 years (range, 0-13.3 years).

TABLE 5. Cox proportional-hazards analysis for independent risk factors for late mortality

Variable	Univariate		Multivariable	
	HR (95% CI)	P value	HR (95% CI)	P value
Age, y	1.07 (0.99-1.14)	.058	1.23 (1.07-1.43)	.005
Sex (female)	1.20 (0.38-3.79)	.761	-	
Body surface area (m ²)	0.18 (0.01-6.35)	.343	-	
Hypertension	0.71 (0.21-2.36)	.573	-	
Diabetes	1.67 (0.53-5.31)	.384	-	
Dyslipidemia	0.48 (0.13-1.77)	.267	-	
Cerebrovascular disease	0.95 (0.12-7.35)	.959	-	
Chronic kidney disease*	3.35 (0.98-11.52)	.055	-	
Preoperative PCI	0.37 (0.11-1.23)	.105	-	
Shock	5.12 (0.66-40.08)	.120	-	
IABP and/or ECMO	1.35 (0.44-4.20)	.600	-	
Blow-out type rupture	1.77 (0.23-13.84)	.585	-	
Anterior AMI	0.63 (0.19-2.09)	.451	-	
Posterolateral AMI	0.34 (0.08-1.57)	.168	0.08 (0.01-0.86)	.037
Inferior AMI	4.34 (1.39-13.56)	.012	2.72 (0.76-9.71)	.124
Re-rupture	3.55 (1.05-12.01)	.041	58.55 (4.89-701.62)	.001

HR, Hazard ratio; CI, confidence interval; PCI, percutaneous coronary intervention; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; AMI, acute myocardial infarction. *Serum creatinine greater than 1.5 mg/dL.

checks and follow-up by echocardiography are postoperatively recommended, because all re-rupture cases occurred within 1 week after sutureless repair. In our study, 17% of patients experienced postoperative re-rupturing, and half of the patients with re-rupturing died. Therefore, the early detection of re-rupture and prompt treatment are essential for improving the surgical outcomes of LVFWR. The effectiveness of additional use of fibrin glue or generous use of the collagen patches is unclear in terms of prevention of re-rupture. Nakatani and colleagues¹⁸ reported that successful reperfusion with primary coronary angioplasty was an independent predictor of the reduced incidence of mechanical complications after AMI. In our series, preoperative coronary intervention was not associated with re-rupturing after sutureless repair.

In the myocardium, the muscle cells swirl and spiral around the chambers of the heart. They form a figure 8 pattern around the 2 ventricles and proceed toward the apex.¹⁹ This helical anatomic structure of the heart muscle makes the anterior myocardium thick, which might reduce the rate of re-rupture after AMI in the anterior wall. However, in our study, the rates of re-rupture did not differ between the culprit coronary lesions.

It remains controversial whether the collagen patch can be safely used in the blow-out type of LVFWR. Nishizaki and colleagues²⁰ reported a successful case of sutureless repair in a patient with blow-out type LVFWR with a small tear.²¹ Raffa and colleagues⁶ reported favorable outcomes of sutureless repair in 6 patients, including 2 with

blow-out type LVFWR. However, in our study, the 2 patients with blow-out type LVFWR experienced re-rupturing after sutureless repair. Therefore, we believe that case should be considered when applying this technique in blow-out type LVFWR, although multivariable analysis did not identify the blow-out type as an independent predictor for re-rupturing.

The selection of the appropriate surgical technique is crucial in each patient with LVFWR. Noteworthy intraoperative findings at the initial surgery in the re-rupture cases included an oozing of blood from multiple areas of the epicardium, bulging of the infarcted myocardium in the systolic phase, severely edematous heart, and large hematoma in the epicardium. In patients with these findings, suture or combination techniques may be better to prevent postoperative re-rupture and improve the outcomes.

In our cohort, all 3 patients undergoing concomitant CABG had 2-vessel disease, and the bypass target was not the culprit coronary artery. The culprit coronary artery usually cannot be the target of CABG during sutureless repair, because the infarcted area is broadly covered with the patches, and the effectiveness of CABG to the necrotic ruptured myocardium is unclear. Only 6 patients (17%) had multiple-vessel disease in our study, which resulted in a small number of concomitant CABGs. The need for concomitant CABG to a nonculprit lesion at the time of sutureless repair was determined by several factors, including probability of postoperative percutaneous coronary intervention and general condition.

There were 12 patients who did not undergo coronary revascularization perioperatively. The reasons why coronary revascularization was not performed included no preoperative coronary angiography due to shock and coma at arrival (3 patients), hemodynamically unstable patients during coronary angiography without time for revascularization (2 patients), onset of AMI more than 24 hours before arrival (5 patients; the intervals between the onset of AMI and admission were 1, 2, 5, and 7 days, and unknown), and the lesion causing AMI was located in the distal portion of the coronary artery (2 patients).

Midterm outcomes were acceptable. As shown in Figure 2, most mortalities occurred within 1 year after surgery, and after that, late death was not frequent. Reoperation occurred in only 1 case 4 months postoperatively. Pseudoaneurysm formation in the rupture site was not detected on echocardiography during the follow-up.

Study Limitations

Several limitations of this study should be considered. First, this was a single-center study with a small sample size. Second, the long-term outcomes after sutureless repair are not available. Third, echocardiography at follow-up was performed in only half of the patients discharged. Finally, it remains unclear if this technique is safe and effective in patients with blow-out type LVFWR. We need a larger series of patients to elucidate whether sutureless repair can be safely applied in blow-out type LVFWR.

CONCLUSIONS

Sutureless repair using the collagen patch can be an expedient, effective, and safe treatment option for LVFWR in selected cases. However, careful observation is required for postoperative re-rupturing, especially in blow-out type LVFWR.

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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