Surgical Outcomes of a Modified Infarct Exclusion Technique for Post-Infarction Ventricular Septal Defects

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Background: Postinfarction ventricular septal defects (pVSDs) are a serious complication of acute myocardial infarctions. The aim of this study was to analyze the clinical outcomes of the surgical treatment of pVSDs.

Methods: The medical records of 23 patients who underwent operations (infarct exclusion in 21 patients and patch closure in two patients) to treat acute pVSDs from 2001 to 2011 were analyzed. Intra-aortic balloon counterpulsation was performed in 19 patients (82.6%), one of whom required extracorporeal membrane support due to cardiogenic shock. The mean follow-up duration was 26.2±18.6 months. Results: The in-hospital mortality rate was 4.3% (1/23). Residual shunts were found in seven patients and three patients required reoperation. One patient needed reoperation due to the transformation of an intracardiac hematoma into an abscess. No patients required reoperation due to recurrence of a ventricular septal defect during the follow-up period. The cumulative survival rate was 95.5% at one year, 82.0% at five years, and 65.6% at seven years. Conclusion: The use of a multiple-patch technique with sealants appears to be a reliable method of reducing early mortality and the risk of significant residual shunting in patients with pVSDs.

Key words: 1. Myocardial infarction 2. Ventricular septal defect 3. Heart failure 4. Coronary artery bypass

INTRODUCTION

Postinfarction ventricular septal defect (pVSDs) are an uncommon but serious complication of acute myocardial infarctions (MIs), which have been estimated to occur in 1%–2% of all MIs. However, with aggressive pharmacologic and interventional management of infarctions, the incidence of pVSDs drops to 0.2% [1]. For most patients, timely surgery is recommended to protect other major organs from sustained hemodynamic deterioration [2]. However, surgical mortality has been reported to be high, especially during the acute phase, due to unstable preoperative hemodynamic conditions, the fragility of the myocardium, bleeding, and depressed postoperative cardiac function [3]. In order to improve the surgical outcomes of pVSDs, a modified infarct-exclusion technique has been performed at our institution since 2001. The aim of this study was to analyze the early and late outcomes of this surgical treatment for acute pVSDs at our institution.


### Table 1. Preoperative profiles of the patients (n=23)

| Variable                          | Value |
|-----------------------------------|-------|
| Age (yr)                          | 68±9  |
| Female gender                     | 12 (52) |
| Hypertension                      | 11 (48) |
| Diabetes mellitus                 | 5 (22) |
| Coronary disease                  |       |
| Triple-vessel disease             | 4 (18) |
| Two-vessel disease                | 3 (13) |
| One-vessel disease                | 14 (61) |
| Coronary spasm                    | 2 (9) |
| Previous percutaneous coronary intervention | 4 (17) |
| Preoperative intra-aortic balloon counterpulsation | 19 (83) |
| Preoperative extracorporeal membrane oxygenator | 1 (4) |
| Emergency operation               | 20 (87) |
| Left ventricular ejection fraction (%) | 42.5±13 |

Values are presented as mean±standard deviation or number (%).

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### METHODS

1) **Patients**

Twenty-three consecutive patients who underwent surgery for acute pVSDs in Samsung Medical Center from 2001 to 2011 were analyzed retrospectively. All patients were transferred from another institution, with a diagnosis of acute MI only (n=7, 30%) or of MI with pVSD (n=16, 70%). The interval from MI onset to surgery was one day or less in 10 patients (43%), two to seven days in eight patients (35%), and longer than seven days in five patients (22%). Patients who underwent concomitant valve or aorta surgery were excluded. Preoperative coronary angiography was performed in all patients, and intra-aortic balloon counterpulsation was performed in 19 patients (82.6%), one of whom required extracorporeal membrane support due to cardiogenic shock. The preoperative patient characteristics are shown in Table 1. Our institutional review board approved the study protocol and waived the need for consent from patients or relatives (SMC 2013-08-136).

2) **Operative technique**

Median sternotomy was performed under general anesthesia. Total cardiopulmonary bypass was established by the ascending aorta and direct bicaval cannulation. Moderate systemic hypothermia was implemented. In order to prevent embolism formation, a left atrial vent was placed through the right superior pulmonary vein after clamping the aorta. Myocardial protection was achieved by initial warm-blood cardioplegia followed by intermittent antegrade and retrograde blood cardioplegia. Before unclamping the aorta, terminal warm cardioplegia was delivered in an antegrade manner.

In two patients, the pVSD was closed via a right atrial approach and left ventriculotomy. Left ventriculotomy was performed with an incision on the infarcted myocardium. The incision was typically 2–3 cm from the left anterior descending (LAD) artery or the posterior descending artery, depending on the site of the MI. The infarct exclusion technique was used in the remaining 21 patients. A patch was sutured to the non-infarcted healthy myocardium 1–2 cm from the margin of the pVSD. In four patients, the patch technique was performed, according to the operative procedure described by David [4]. The double-patch technique was used in 15 patients. In this technique, a circular piece of bovine pericardium was used to close the pVSD with an interrupted 4-0 polypropylene suture, the pericardium was then tailored into a quadrangular shape, the patch was cut into a cone shape, and the patch was then closed with running 3-0 polypropylene sutures. Before closure, a sealant (BioGlue; CryoLife Inc., Kennesaw, GA, USA) were applied to fill in the excluded ventricular cavity (Fig. 1). In two patients, the pVSD was closed using two separate patches on the right and left ventricular surfaces. Thus, three patches were used in each of these two patients. The ventriculotomy was closed with interrupted and running 3-0 polypropylene sutures, after which coronary artery bypass was performed. Four patients were treated with percutaneous coronary intervention before surgery. Seventeen patients (74%) were revascularized during surgery and LAD revascularization was performed in 14 patients, but severely unstable patients underwent rapid surgery without revascularization. The mean number of distal anastomoses was 1.7±1.0, while the mean durations of cardiopulmonary bypass and aortic cross-clamping were 194.4±36.9 minutes and 150.1±31.2 minutes, respectively. The operative data are presented in Table 2.

3) **Endpoints and follow-up**

The primary endpoint was early death, defined as death
Table 2. Operative data (n=23)

| Variable                                      | Value  |
|-----------------------------------------------|--------|
| Concomitant coronary artery bypass grafting   | 17 (74) |
| Total anastomosis sites (mean)                | 1.7 sites |
| Location of VSD                               |        |
| Anterior                                      | 19 (83) |
| Posterior                                     | 4 (17)  |
| Direct closure of VSD                         | 2 (9)   |
| Infarct exclusion technique                   |        |
| Single-patch                                  | 4 (17)  |
| Double-patch                                  | 15 (74) |
| Triple-patch                                  | 2 (9)   |

Values are presented as number (%).

VSD, ventricular septal defect.

within 30 days after surgery or during the same hospitalization. The secondary endpoint was late all-cause death and recurrence of a ventricular septal defect. Patients underwent regular follow-up at our outpatient clinic at three-month intervals; telephone and outpatient interviews were conducted if clinic visits were not conducted at the scheduled time. Follow-up ended on March 31, 2013.

In order to validate the complete follow-up data regarding mortality, information was obtained from the National Registry of Births and Deaths using each patient’s unique personal identification number. The mean duration of follow-up was 26.2±18.6 months. Echocardiographic data were obtained in the parasternal long axis view at the base, following the guidelines of the American Society of Echocardiography [5].

4) Statistical analysis

Results were expressed as mean±standard deviation or as frequency and percentage. Survival analysis was performed using the Kaplan-Meier method. Statistical analysis was carried out using PASW SPSS ver. 18.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

1) Early outcomes

The in-hospital mortality rate was 4.3% (1/23). One patient died of right heart failure two days after surgery. This patient underwent the infarct exclusion technique using a double patch and bioglue via a biventricular approach, without coronary revascularization. The mean interval from MI to surgery was 3.4±2.9 days (range, 1 to 10 days). Percutaneous coronary intervention was performed before surgery in four patients (17.4%). Early complications included atrial fibrillation, ventricular tachycardia, residual shunts, reoperations to treat bleeding, and pneumonia (Table 3).

Residual shunts were found in seven patients after surgery. Three patients underwent reoperation (single-patch technique, no bioglue), two underwent surgery using the double-patch technique, and one underwent surgery using the glue-free technique. The remaining patients were managed with supportive care alone. One of these patients died during hospitalization, and the other six patients were discharged alive.

2) Late survival

Five patients died during follow-up. The causes of death were heart failure, head trauma, pneumonia (two patients), and intestinal bleeding. No patients required reoperation due to the recurrence of a ventricular septal defect. One patient,
Table 3. Postoperative results (n=23)

| Variable                                   | Value |
|--------------------------------------------|-------|
| Operative mortality                        | 1 (4) |
| Postoperative intra-aortic balloon support (day) | 1.6±1.0 |
| Postoperative ventilator support (day)      | 2.4±1.9 |
| Intensive care unit stay (day)              | 5.1±3.1 |
| Hospital stay (day)                         | 19.2±20.2 |

Values are presented as number (%) or mean±standard deviation.

as described above, needed reoperation due to the transformation of an intracardiac hematoma into an abscess [6]. Two patients were admitted due to heart failure three months and 58 months after surgery, respectively. The remaining patients were asymptomatic and New York Heart Association functional class I or II over the course of follow-up. The cumulative survival rate was 95.5% at one year, 82.0% at five years, and 65.6% at seven years (Fig. 2).

DISCUSSION

This study showed that the rate of operative mortality in patients with pVSD was 4.3%, which is much lower than has been reported in previous studies [7,8]. Furthermore, this study highlights three important points: first, the infarct exclusion technique using multiple patches is safe and durable; second, biogluue is helpful in preventing significant residual shunts after surgery; and third, right heart failure was observed in patients who underwent the infarct exclusion procedure via the biventricular approach.

pVSDs are a severe mechanical complication of acute MI, and surgical intervention is mandatory due to its poor prognosis. However, the timing of surgical intervention remains a matter of debate. Brandt et al. [7] have argued that the operation should be deferred until three weeks after the MI if possible [7].

However, because fewer than 20% of patients survive that long [8], we favor early surgical intervention for acute pVSDs, as heart failure or other organ dysfunction might otherwise develop [2,4,8,9].

Since David [4] introduced the technique of infarct exclusion, many surgeons have adapted this technique for patients with acute pVSD. However, residual shunts remain a major complication; David [4] reported two out of 67 patients with residual shunts who were not surgically treated due to the negligible size and spontaneous closure of the shunts. Lafci et al. [9] reported no residual shunts after the infarct exclusion technique was performed using a single patch, while Labrousse et al. [10] reported an 11% rate of early recurrence of pVSDs with the single-patch technique, as well as a 66% mortality rate, despite reoperation.

We performed this operation using several distinct techniques. In the first four patients, we used a single-patch technique, resulting in two patients who required reoperation for significant residual shunts. Thereafter, we used the three-patch technique with glue to protect against excessive tension at the suture site. In order to simplify the three-patch technique, we adapted the double-patch technique, introducing a patch designed for infarct exclusion as well as applying glue to reduce tension on the suture line [11-13].

In this study, residual shunts were detected in three patients (75%) who were operated on with the single-patch technique and four patients (33%) who underwent surgery using the double- or triple-patch technique. Reoperations for residual
shunts were performed in two patients (50%) who underwent surgery using the single-patch technique and one patient (8.3%) who underwent surgery using the double-patch technique with the application of glue. Small residual shunts were detected in three patients (27.3%) who underwent surgery using the double- or triple-patch technique with glue, and none of these patients required reoperation.

Based on the findings of previous studies, we applied biological glue around the suture margin of the pericardial patch after placing gauze inside the left ventricle to even out the application of the glue. The use of glue in the double- and triple-patch techniques not only reduced the incidence of residual shunts, but also decreased left ventricular free wall rupture and bleeding from left ventriculotomy closure. However, in one patient who underwent the double-patch technique with glue, a postoperative hematoma developed into an abscess between the patch and the left ventricular cavity. The area between the patches is isolated from the blood supply, so aseptic technique is mandatory to prevent this complication. The patient’s prognosis may have been associated with the extent of the infarction and the shunt.

We performed complete revascularization when possible (in 74% of our cases), although conflicting reports exist about whether this procedure improves clinical outcomes. We acknowledge that further study is necessary, but concur with Ozkara et al. [14], who reported that myocardial revascularization, especially of the culprit artery, resulted in improved short- and long-term survival.

One interesting observation was made in a patient displaying right ventricular failure after having undergone an infarct exclusion procedure via the biventricular approach. We surmised that the right ventriculotomy may have been the cause, as patients who underwent infarct exclusion via left ventriculotomy alone did not typically display right heart failure. A prospective comparison between these two approaches will be helpful in improving clinical outcomes. This is a small retrospective review. Although the tow-patch technique was our standard technique, we included a few modifications of the infarct exclusion technique.

We conclude that the infarct exclusion technique is safe for patients with pVSD, and that the double- or triple-patch technique with biological glue is a reliable method of reducing the risk of residual shunts in these patients.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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REFERENCES

1. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation 2000;101:27-32.
2. Deja MA, Szostek J, Widenka K, et al. Post infarction ventricular septal defect: can we do better? Eur J Cardiothorac Surg 2000;18:194-201.
3. Daggett WM, Buckley MJ, Akins CW, et al. Improved results of surgical management of postinfarction ventricular septal defect. Ann Surg 1982;196:269-77.
4. David TE. Operative management of postinfarction ventricular septal defect. Semin Thorac Cardiovasc Surg 1995;7:208-13.
5. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440-63.
6. Cho YH, Kim WS, Lee YT, Park PW. Abscess transformation of intracardiac hematoma and ventricular rupture after double-patch repair of postinfarction ventricular septal defect. J Card Surg 2010;25:676-9.
7. Brandt B 3rd, Wright CB, Ehrenhaft JL. Ventricular septal defect following myocardial infarction. Ann Thorac Surg 1979;27:580-9.
8. Massetti M, Babatasi G, Le Page O, Bhoyroo S, Saloux E, Khayat A. Postinfarction ventricular septal rupture: early repair through the right atrial approach. J Thorac Cardiovasc Surg 2000;119(4 Pt 1):784-9.
9. Lafci B, Yakut N, Goktogan T, et al. Repair of post-infarct ventricular septal rupture with an infarct-exclusion technique: early results. Heart Surg Forum 2006;9:E737-40.

10. Labrousse L, Choukroun E, Chevalier JM, et al. Surgery for post infarction ventricular septal defect (VSD): risk factors for hospital death and long term results. Eur J Cardiothorac Surg 2002;21:725-31.

11. Tashiro T, Todo K, Haruta Y, Yasunaga H, Shibano R, Kawara T. Extended endocardial repair of postinfarction ventricular septal rupture: new operative technique—modification of the Komeda-David operation. J Card Surg 1994;9:97-102.

12. Tabuchi N, Tanaka H, Arai H, et al. Double-patch technique for postinfarction ventricular septal perforation. Ann Thorac Surg 2004;77:342-3.

13. Musumeci F, Shukla V, Mignosa C, Casali G, Ikram S. Early repair of postinfarction ventricular septal defect with gelatin-resorcin-formol biological glue. Ann Thorac Surg 1996;62:486-8.

14. Ozkara A, Cetin G, Mert M, et al. Postinfarction ventricular septal rupture: surgical intervention and risk factors influencing hospital mortality. Acta Cardiol 2005;60:213-7.