Mid-term results for the use of the extended sandwich patch technique through right ventriculotomy for postinfarction ventricular septal defects

Soh Hosoba*, Tohru Asai, Tomoaki Suzuki, Hiromitsu Nota, Satoshi Kuroyanagi, Takeshi Kinoshita, Noriyuki Takashima and Masato Hayakawa

Division of Cardiovascular Surgery, Department of Surgery, Shiga University of Medical Science, Otsu, Japan

* Corresponding author. Division of Cardiovascular Surgery, Department of Surgery, Shiga University of Medical Science, Setatsukinowacho, Otsu, Shiga 520-2192, Japan. Tel: +81-77-5482244; fax: +81-77-5442901; e-mail: sohosoba@belle.shiga-med.ac.jp (S. Hosoba).

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Abstract

OBJECTIVES: Postinfarction ventricular septal defect (VSD) is a rare, but feared, complication after acute myocardial infarction. Although numerous techniques and materials have been used, the best technique has not yet been settled upon. We present a novel technique of VSD closure through the VSD via right ventricular (RV) incision and assess short- and mid-term outcomes.

METHODS: Between April 2008 and March 2012, 15 consecutive patients presenting with postinfarction VSD underwent surgical repair using this technique in our department.

RESULTS: Thirty-day mortality was 20% (3 patients). Two patients died from low cardiac output. No early complications related to the VSD repair were observed, such as shunt recurrence, severe septal dyskinesia or pseudoaneurysmal change in the left ventricular myocardium. The left ventricle was contracted well without mitral regurgitation. The mean follow-up period was 17 ± 15 months. The Kaplan–Meier estimate of 3-year cumulative survival is 76%. At the mid-term stage, one trivial residual leak was noted, but no patient required reoperation. RV function was within the normal range after the operation.

CONCLUSIONS: This method of VSD repair using right ventricle incision and trans-VSD approach is safe and simple and reduces the postoperative recurrence of VSD.

Keywords: Acute myocardial infarction • Postinfarction ventricular septal defect • Double-patch repair

INTRODUCTION

Postinfarction ventricular septal defect (VSD) is one of the most serious complications associated with acute myocardial infarction (MI). The frequency of postinfarction VSD has been reported to be 0.2–2% [1–3]. VSD is seen with equal frequency in both anterior and posterior infarction [4, 5]. In patients with cardiogenic shock after this complication, the prognosis is fatal without urgent surgery [6]. In spite of the development of numerous techniques and materials [7–11], the mortality rate and risk of recurrence remain high.

We have introduced a novel technique of VSD closure through the VSD via right ventricular (RV) incision [12]. Our method creates a ‘sandwich’ double patch using large interrupted horizontal mattress sutures only, with no incision in the left ventricle (LV) wall.

We studied a series of 15 patients who underwent VSD repair with this technique to analyze the outcome of the operation in terms of operative and postoperative complications and early to mid-term outcome.

MATERIALS AND METHODS

Between April 2008 and March 2012, 15 consecutive patients presenting with postinfarction VSD underwent VSD repair by three surgeons at the Shiga University Hospital. Approval was obtained from the institutional review board of Shiga University of Medical Science, Shiga, Japan. Data were reviewed retrospectively. Preoperative patient characteristics are shown in Table 1. Preoperative coronary angiography was undertaken in 13 (87%) patients before transfer to our hospital. The number of diseased vessels was 2.3 ± 1.0. Six (40%) patients were operated on within 24 h after the onset of VSD, 8 (53%) within 2–7 days and 1 (7%) 35 days after the onset of VSD. The mean interval from the onset of VSD to operation was 43 ± 41 h. At the time of admission, 8 (53%) patients were in cardiogenic shock. An intra-aortic balloon pump was inserted in 12 (80%) patients and percutaneous cardiopulmonary support in 2 (13%) before the operation.
Surgical technique

Cardiopulmonary bypass was established via the ascending aorta with bicaval cannulation. Myocardial protection was achieved with antegrade and retrograde cold-blood cardioplegia.

Repair of anterior ventricular septal defect

An incision is made in the RV wall 10–15 mm from, and parallel to, the distal left anterior descending artery (Figs. 1 and 2) [12]. Two patches are tailored to overlap the VSD margin. Each patch is octagonal and about 50 mm in diameter for most patients. The first patch is prepared with eight 3-0 Prolene sutures with an MH needle (Ethicon, Inc., Somerville, NJ, USA) at each end, and the sutures are placed trans-septally or transmurally from the LV cavity via the VSD and spaced equally at 15 mm intervals around the VSD. The first patch is then inserted through the VSD into the LV. Sutures brought outside of the heart from both RV and LV are secured using same large Teflon felt pledgets (Meadow Medical, Inc., Oakland, NJ, USA). Sutures brought into the RV are passed through the second Dacron patch. A surgical adhesive is applied to the defect between the two patches. The RV wall is closed with two Teflon felt strips.

Repair of posterior ventricular septal defect

The principle is very similar: an incision is made in the inferior wall of the RV about 10 mm from, and parallel to, the midportion of the posterior descending artery (PDA; Figs. 3 and 4).

Table 1: Patient characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Data</th>
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</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>76 ± 10</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>8 (53)</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>8 (53)</td>
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<tr>
<td>Smoking, n (%)</td>
<td>4 (27)</td>
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<tr>
<td>Hyperlipidaemia, n (%)</td>
<td>6 (40)</td>
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<td>Diabetes mellitus, n (%)</td>
<td>7 (47)</td>
</tr>
<tr>
<td>Previous myocardial infarction, n (%)</td>
<td>1 (7)</td>
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<tr>
<td>Preoperative IABP, n (%)</td>
<td>12 (80)</td>
</tr>
<tr>
<td>Preoperative PCPS, n (%)</td>
<td>2 (13)</td>
</tr>
<tr>
<td>Anterior infarction, n (%)</td>
<td>5 (33)</td>
</tr>
<tr>
<td>Inferior infarction, n (%)</td>
<td>10 (67)</td>
</tr>
<tr>
<td>Cardiogenic shock, n (%)</td>
<td>8 (53)</td>
</tr>
<tr>
<td>Primary PCI, n (%)</td>
<td>8 (53)</td>
</tr>
<tr>
<td>Interval from onset of VSD to OR, h</td>
<td>43 ± 41</td>
</tr>
<tr>
<td>Left-to-right shunt, %</td>
<td>55 ± 14</td>
</tr>
</tbody>
</table>

IABP: intra-aortic balloon pumping; PCPS: percutaneous cardiopulmonary support; PCI: percutaneous coronary intervention; VSD: ventricular septal defect; OR: operating room.

Figure 1: Repair of anterior VSD. (A) Transmural suture is placed via VSD. (B) First patch is introduced into the LV cavity. (C) Second patch is fixed by trans-septal sutures. (D) Sutures are tied following the insertion of glue into defect between two patches. LAD: left anterior descending; RV: right ventricle.
Statistical analysis and data collection

Results are expressed as mean ± standard deviation. The survival rate was determined by the Kaplan–Meier method. All statistical analysis was performed using the SPSS 11.0 software (SPSS, Inc., Chicago, IL, USA). The follow-up period ranged between 2 and 44 months.

Echocardiographic examinations were performed at key times: during hospitalization and at the early postoperative (2 weeks) and late postoperative stages. RV function was assessed quantitatively using echocardiographic analysis as the percentage of change in the cavity area from end diastole to end systole. RV fractional area change (RVFAC) was defined using the formula (end-diastolic area × end-systolic area)/end-diastolic area × 100.

RESULTS

The mean operation time, cardiopulmonary bypass time and aortic cross-clamp time were 272 ± 44 (range 198–338 min), 140 ± 29 (range 95–186 min) and 100 ± 20 (range 73–139 min). Eleven (73%) patients had concomitant coronary artery bypass grafting with 1.5 anastomoses on average. The left internal thoracic artery was used in 5 (33%) patients, the saphenous vein in 9 (60%) and the right internal thoracic artery and gastroepiploic artery in 1 (7%) each. Concomitant aortic valve replacement was performed in 1 (7%) patient with bicuspid stenotic aortic valve. Concomitant repair of papillary muscle rupture and of free wall rupture was each performed in 1 (7%) patient. In the early postoperative term, no patient required re-exploration for bleeding
or tamponade. There were 3 (20%) cases of 30-day mortality (Table 2). Two patients with preoperative deep cardiogenic shock died of persistent low cardiac output and multiple organ failure 2 days after surgery. One patient died 17 days after surgery due to cerebral haemorrhage. Thirty-day mortality occurred in 3 cases in the posterior VSD group (30% of posterior VSDs) and in no cases in the anterior VSD group.

The median intensive care unit stay for survivors was 5.5 (range 1–23 days). The median total ventilation time was 79 (range 18–715 h). The median hospital stay for survivors was 30 (range 14–55 days).

On predischarge echocardiographic examination, the LV was contracting well without mitral regurgitation (Table 3). No early complications related to the VSD repair were observed, such as shunt recurrence, severe septal dyskinesia or pseudoaneurysmal change in the LV muscle.

The mean follow-up period was 17 ± 15 months. The follow-up rate was 100%. There were two late deaths, both caused by pneumonia. The Kaplan–Meier estimate of 3-year cumulative survival of all the survivors is 76%. At the mid-term stage, echocardiography was undertaken at the time of outpatient routine examination. No residual leak was noted except in 1 case in which the residual shunt was trivial (Qp/Qs = 1.1), and actual measured pulmonary artery systolic pressure was <30 mmHg. Complications related to severe septal dyskinesia, or pseudoaneurysmal change in the LV muscle, were not observed. No patient required reoperation for recurrent VSD.

On echocardiographic examination at the last annual follow-up point (n = 10), we measured two-dimensional parameters of LV and RV functions. We used RVFAC to estimate RV function (Table 3). Estimated RV function was within the normal range after the operation.

**DISCUSSION**

VSD following MI has been a challenge to cardiac surgeons. Postinfarction VSD is still a high-mortality disease, and the rate of recurrence after VSD repair remains high.

A technique currently adopted seems to be based on patch repair with infarct exclusion through LV incision. The running suture usually has to cover a large region, and even partial dehiscence can cause massive, and possibly lethal, leakage. The area patched in that design (covering the whole infarction, including the LV incision) might be so large as to compromise residual LV contraction. Our technique eliminates those disadvantages.

Anatomically, the blood supply to the RV does not come from the PDA. Branches originated from the right dominant coronary artery supplied most part of the RV free wall. We therefore proposed a new approach through the VSD itself via RV incision without LV incision. There have been similar reports of double-patch VSD repair or patch repair via right ventriculotomy [9, 10, 11]. Our strategy excluded the weak infarcted area, which can extend to the anterior or posterior free wall, and prevented muscular tear at the suture point. It also prevented ventricular septal dyskinesia by ensuring equal distribution of the tension.
There are 3 cases of 30-day mortality. Two patients with pre-operative deep cardiogenic shock died of low cardiac output and multiple organ failure 2 days after surgery. Both of them were referred without diagnosed with VSD, after percutaneous coronary intervention for multivessels with mechanical circulatory support.

The reported incidence of residual VSD varies from 28 to 43%, and the reported incidence of reoperation for residual VSD is around 10% [4, 5, 13]. Our method, which creates an almost leak-free 'sandwich' double patch, should provide slightly more extended and better-anchored coverage of the VSD margin using large interrupted horizontal mattress sutures only. A low incidence of VSD recurrence would be associated with a better survival rate [4].

Postoperative echocardiography demonstrated slight, if any, impairment of cardiac function. The approach to this repair technique is a single right ventriculotomy through the VSD, which allows for a maximal sparing of viable myocardium and reduced scar tissue formation. RV dysfunction is related to several factors, such as left ventricular dysfunction, RV infarction or ischaemia, and RV volume. RV function is an important risk factor after MI [14–16]. Anavekar et al. demonstrated that RVFAC might be a better echocardiographic descriptor of RV systolic function. In the present study, impairment of RV function, possibly due to the right ventriculotomy, was very slight in the early term and not seen in the mid-term.

Several limitations of the present study must be considered. The study investigates a small number of patients retrospectively. A larger number of patients are necessary to corroborate our findings.

We have employed this method since 2008. Although this is a limited series, there was no residual leakage requiring reoperation, and LV and RV functions were preserved. The VSD repair with this method is simple and secure and reduces invasiveness on LV function following a catastrophic event and surgery.

Conflict of interest: none declared.

REFERENCES


