Decision-making and surgery results in postinfarction ventricular septal rupture

Arūnas Valaika *, Giedrius Uždavinys, Pranas Šerpytis, Gediminas Norkūnas, Gintaras Kalinauskas, Loreta Ivaškevičienė, Giedrė Nogiene, Vytautas Sirvydis

Centre of Cardiac Surgery, Vilnius University Hospital Santariskiu Klinikos; Clinic of Heart Diseases, Vilnius University, Vilnius, Lithuania

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Summary

Objectives: Postinfarction ventricular septal defect (VSD) remains a surgical challenge because it is technically difficult to reconstruct the septum during the acute phase of transmural myocardial infarction, and it is a relatively uncommon operative procedure.

Design and Methods: Thirty patients underwent surgical repair of a postinfarction ventricular septal defect from 1992 to 2003. 26 of the patients were in New York Heart Association functional class IV, and 9 of them – in cardiogenic shock when operated on. There were 19 females and 11 males with a mean age of 68 ± 9.5 years.

The VSD was anterior in 16 patients and posterior – in 14 patients. We used operative techniques that evolved from infarctectomy and reconstruction of the septum with Dacron patches to pericardial patch exclusion of the left ventricle and left ventricular volume reconstruction. This was accomplished by suturing a single Dacron patch to the healthy endocardium surrounding the infarcted muscle.

Results: Six patients died perioperatively, whereas the surgical mortality rate was 20%. Nineteen patients underwent coronary artery bypass grafting. Four patients developed recurrent VSD; one patient died and one patient underwent surgical closure of a defect. There were 14 survivors at 36 months of follow-up: 50% of patients were in functional class II, and 50% – in functional class III. Four patients died at a remote period.

Conclusions: Decision on terms of surgical intervention was made individually for each patient. Patch closure of the ventricular septal rupture, remodelling of the left ventricle and selective myocardial revascularisation provided acceptable results.

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Keywords: ventricular septal rupture, myocardial infarction, cardiac surgery

Rupture of the ventricular septum is a severe complication of acute myocardial infarction (AMI). The reported incidence is about 1–3%, with a high mortality rate if patients are treated medically and often with reduced cardiac function in survivors [1]. This mechanical complication usually occurs in the first week after the onset of acute myocardial infarction and is generally associated with an occluded infarct-related coronary artery, in the absence of collateral circulation [2]. As ventricular septal defect (VSD) is usually lethal (90% mortality within 3 months) [1], surgical correction should be the most appropriate treatment. We report the experience in the management of postinfarction VSD patients referred to the institution during the last decade. Although a short time from myocardial infarction to surgery has been reported to be a risk factor for mortality, the optimal timing to surgery and the most appropriate approach to VSD patients have not been established yet [3,4]. In the present study we have evaluated the clinical outcome of 30 patients treated surgically at our institution, by assessing preoperative, operative and postoperative variables.

Methods

Patient population

Our series comprised 30 consecutive patients (19 (63%) of them were women) operated on shortly after the development of postinfarction VSD, over 11 years period (01.01.1992–
were performed in all the patients for the assess-

and/or transesophageal echocardiography (TEE) 

operative transthoracic echocardiography (TTE) 

time of 9

examination. VSD was diagnosed after a mean

of a new typical systolic murmur in all the pa-

The VSD diagnosis was based on the appearance

dial infarction was present only in 4 cases (13%).

prior myocardial infarction in 15 patients (50%), infe-

electrocardiograms showed signs of anterior my-

need for intraaortic balloon pumping (IABP). The

signs of systemic hypoperfusion, oliguria, and/or

by systolic blood pressure less than 80 mmHg,

tients (30%) were in cardiogenic shock as defined

have had an acute pulmonary oedema and 9 pa-

patients (13%) – class III. Eight patients (27%)

AMI localization

AM – acute myocardial infarct; IABP – intra-aortic bal-

loon pumping; LVEF – left ventricular ejection fraction;

MVR – mitral valve regurgitation; NYHA – New York

Heart Association; VSD – ventricular septal defect.

01.01.2003) with the mean age of 68 ± 9.5 years

(Table 1). Seven patients (23%) were over 75 years

old. Medical records of all the patients were retro-

spectively reviewed and the data were analysed.

Twenty six patients (87%) were in New York Heart

Association (NYHA) functional class IV and four

patients (13%) – class III. Eight patients (27%) have had an acute pulmonary oedema and 9 pa-

patients (30%) were in cardiogenic shock as defined by systolic blood pressure less than 80 mmHg, signs of systemic hypoperfusion, oliguria, and/or need for intraaortic balloon pumping (IABP).

The electrocardiograms showed signs of anterior myocardial infarction in 15 patients (50%), inferior myocardial infarction in 13 patients (43%), and extensive anterior and inferior myocardial infarction in 2 patients (7%). Previous myocardial infarction was present only in 4 cases (13%).

The VSD diagnosis was based on the appearance of a new typical systolic murmur in all the pa-

patients and was confirmed by echocardiographic examination. VSD was diagnosed after a mean
time of 9 ± 10 days after the onset of AMI.

Post-

operative transthoracic echocardiography (TTE) 

and/or transesophageal echocardiography (TEE) 

were performed in all the patients for the assess-

ment of surgical results. Coronary angiography 

was performed in 26 patients (87%) and ventricu-

lography – in 6 patients (20%). Cardiac catheterisation data are presented in Table 2.

This study was conducted according to the 

principles of the Declaration of Helsinki.

**Operative technique**

Surgical intervention was performed after a 

mean period of 16.8 ± 14.7 days after the onset of 

AMI. Nine patients (30%) were operated on within the first 10 days, 11 patients (37%) – be-

between 10 and 30 days and 10 patients (33%) – later than 30 days after the onset of AMI. One 

patient (3%) was operated on less than 24 h after 

hospital admission, due to severe haemody-

namic instability. IABP was necessary in 11 pa-

patients (37%). The septum was approached by the 

left ventriculotomy at the level of the infarcted 

area in 30 patients (100%). Additionally, in 2 pa-

patients (7%) it was necessary to open the right 

ventricle to find a VSD. The reconstruction of the 

ventricular septum and the closure of a defect 

were performed with a Dacron or Teflon patch in 

26 patients (87%), a pericardial patch was used in 

2 patients (7%) and a direct suture of the defect 

was performed in 2 patients (7%). Associated pro-

cedures included mitral valve prosthesis in one 

patient (3%), left ventricular reconstruction for 

akinesia, dyskinesia – in 15 patients (50%) [5–7] 

and aortocoronary bypass grafting – in 19 pa-

patients (63%): a single graft – in 7 CABG patients 

(37%), two grafts – in 8 patients (42%) and three 
to five grafts – in 4 patients (21%). Crystalloid 

cardioplegia was used in 10 patients (33.3%) and 

blood cardioplegia – in 20 patients (66.7%). The 

mean extracorporeal circulation (ECC) time was 

127 ± 55 min, the mean aortic cross-clamping 
time was 73 ± 20 min; hypothermia reached a 

mean value of 30 ± 4°C.
**Statistical analysis**

All statistical analyses were undertaken using standard statistical software SPSS 10.7. Incidences were presented as percentages. Mean ± SD were given for quantitative data. For quantitative variables, the Independent-Samples T Test procedures were used to compare means for two groups of cases. For each variable sample size, mean, standard deviation, and the standard error of the mean were calculated. For the difference in means the mean, standard error, and confidence interval were estimated. The Levene’s test for equality of variances was realized to determine pooled- or separate-variances; the t-test for equality of means must be used. The Paired-Samples T Test was used to compare the means of two variables for a single group.

Whereas the sample is not large the Fisher’s exact test was used for testing the association between two qualitative variables. $P$-value < 0.05 was considered to be statistically significant. It was analysed 22 variables presented in Table 3 to assess potential predictors of early and late mortality. We compared different preoperative, operative and postoperative variables in: (1) nonsurvivors (6 patients (20%)) and survivors (24 patients (80%)); (2) the early operation group (9 patients (30%) operated on earlier than 10 days after the onset of AMI) and the later operation group (21 patients (70%) operated on later than 10 days after the onset of AMI).

**Results**

The overall operative, in-hospital mortality rate was 20% (6/30). Heart failure was the reason of death in 4 patients (13%), one patient died from sepsis and one – from polyorganic insufficiency. The timing and operative mortality is presented in Figure 1. The highest mortality rate (3 of 9 patients (33%)) was in the early (<10 days) operation group. Though the early operation time was not a predictor of mortality at logistic regression analysis ($p=0.32$), but in this group significantly more patients were in cardiogenic shock (6 of 9 patients (67%) vs 3 of 19 patients (16%), $p=0.04$) and the aortic cross-clamping time was longer (85 ± 2 vs 68 ± 19 min, $p=0.04$) comparing with the patients operated on later than 10 days after the onset of AMI.

There were more patients of advanced age and with cardiogenic shock in the non-survivors group, but only diabetes ($p=0.01$), mitral valve regurgitation (MVR) I–II$^\circ$ ($p=0.009$); ‘no CABG’ ($p=0.016$) were significantly correlated with early and late mortality (Table 4).

Preoperative echocardiographic left ventricular ejection fraction (LVEF) was significantly ($p=0.02$) higher in survivors (Table 5).

![Figure 1. Timing from complication to operation and mortality.](image_url)
Table 4.
Main preoperative and operative factors influencing early and late mortality

<table>
<thead>
<tr>
<th>Variables</th>
<th>Early mortality n = 6 (number of non-survivors/all pts. (%))</th>
<th>Fisher’s exact test</th>
<th>Late mortality n = 4 (number of non-survivors/all pts. (%))</th>
<th>Fisher’s exact test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>3/4 (75%)</td>
<td>0.01</td>
<td>0/1 (0%)</td>
<td>0.833</td>
</tr>
<tr>
<td>Without diabetes</td>
<td>3/26 (11%)</td>
<td></td>
<td>4/23 (17%)</td>
<td></td>
</tr>
<tr>
<td>With MVR</td>
<td>5/10 (50%)</td>
<td>0.009</td>
<td>1/5 (20%)</td>
<td>0.635</td>
</tr>
<tr>
<td>Without MVR</td>
<td>1/20 (5%)</td>
<td>0.0016</td>
<td>3/19 (16%)</td>
<td>0.712</td>
</tr>
<tr>
<td>CAVG</td>
<td>1/19 (5.2%)</td>
<td></td>
<td>3/18 (17%)</td>
<td></td>
</tr>
<tr>
<td>Without CAVG</td>
<td>5/11 (45%)</td>
<td>0.156</td>
<td>1/6 (17%)</td>
<td>0.283</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>4/11 (36%)</td>
<td></td>
<td>0/7 (0%)</td>
<td></td>
</tr>
<tr>
<td>Without cardiogenic shock</td>
<td>2/19 (10%)</td>
<td></td>
<td>4/17 (24%)</td>
<td></td>
</tr>
<tr>
<td>Age &gt; 70</td>
<td>5/15 (33%)</td>
<td>0.169</td>
<td>2/10 (20%)</td>
<td>0.56</td>
</tr>
<tr>
<td>Age &lt; 70</td>
<td>1/15 (6.7%)</td>
<td></td>
<td>2/14 (14%)</td>
<td></td>
</tr>
<tr>
<td>Residual VSD</td>
<td>2/4 (50%)</td>
<td>0.169</td>
<td>0/2 (0%)</td>
<td>0.668</td>
</tr>
<tr>
<td>Without residual VSD</td>
<td>4/26 (15%)</td>
<td></td>
<td>4/22 (18%)</td>
<td></td>
</tr>
<tr>
<td>Serum creatinine &gt; 200 µmol/l</td>
<td>3/8 (37%)</td>
<td>0.3</td>
<td>1/5 (20%)</td>
<td>0.635</td>
</tr>
<tr>
<td>Serum creatinine &lt; 200 µmol/l</td>
<td>3/22 (13.6%)</td>
<td></td>
<td>3/19 (16%)</td>
<td></td>
</tr>
<tr>
<td>Complication-op. time &lt; 10 days</td>
<td>3/9 (33%)</td>
<td>0.329</td>
<td>1/6 (17%)</td>
<td>0.712</td>
</tr>
<tr>
<td>Complication-op. time &gt; 10 days</td>
<td>3/21 (14%)</td>
<td></td>
<td>3/18 (17%)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1/11 (9%)</td>
<td>0.372</td>
<td>1/10 (10%)</td>
<td>0.614</td>
</tr>
<tr>
<td>Female</td>
<td>5/19 (26%)</td>
<td></td>
<td>3/14 (21%)</td>
<td></td>
</tr>
<tr>
<td>With IABP</td>
<td>3/11 (27%)</td>
<td>0.641</td>
<td>1/8 (13%)</td>
<td>0.59</td>
</tr>
<tr>
<td>Without IABP</td>
<td>3/19 (16%)</td>
<td></td>
<td>3/16 (19%)</td>
<td></td>
</tr>
<tr>
<td>LV volume restoration</td>
<td>4/15 (27%)</td>
<td>0.85</td>
<td>3/11 (27%)</td>
<td>0.38</td>
</tr>
<tr>
<td>Without LV volume restoration</td>
<td>2/15 (13%)</td>
<td></td>
<td>1/9 (11%)</td>
<td></td>
</tr>
<tr>
<td>Tepid blood cardioplegia</td>
<td>4/20 (20%)</td>
<td>0.7</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Crystalloid cardioplegia</td>
<td>2/10 (20%)</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

CABG – coronary artery bypass grafting; IABP – intra-aortic balloon pumping; LV – left ventricle; MVR – mitral valve regurgitation; VSD – ventricular septal defect.

Table 5.
Operative, preoperative variables in survivors and non-survivors

<table>
<thead>
<tr>
<th>Variable</th>
<th>Non-survivors (n = 6)</th>
<th>Survivors (n = 24)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>72.83 ± 6.7</td>
<td>66.4 ± 9.6</td>
<td>0.142</td>
</tr>
<tr>
<td>LVEF before operation (%)</td>
<td>31.67 ± 2.58</td>
<td>37 ± 7.7</td>
<td>0.013</td>
</tr>
<tr>
<td>CPB time (min)</td>
<td>142 ± 93</td>
<td>124 ± 43</td>
<td>0.483</td>
</tr>
<tr>
<td>Aortic clamp time (min)</td>
<td>76.3 ± 20</td>
<td>62.5 ± 20</td>
<td>0.150</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>59 ± 6</td>
<td>57 ± 6</td>
<td>0.57</td>
</tr>
<tr>
<td>Serum creatinine (µmol/l)</td>
<td>190 ± 83</td>
<td>162 ± 89</td>
<td>0.4</td>
</tr>
</tbody>
</table>

CPB – cardiopulmonary bypass; LVEDD – left ventricular end-diastolic diameter; LVEF – left ventricular ejection fraction.

Late results
Four patients (13%) died in the late period (26 ± 16 month). Heart failure was the major reason of death. Fourteen patients were follow-up in the late period. Seven (50%) of them were in functional class II, seven (50%) – in functional class III. Four patients developed recurrent VSD; one patient died and one patient after 14 months underwent surgical closure of a recanalisation. One patient underwent cardiac transplantation due to intractable heart failure 2.5 years after initial operation. There were no preoperative and operative factors significantly influencing late mortality. In the late period the non-survivors had lower LVEF (%) after the operation on discharge from the hospital, comparing to all the survivors (36.5 ± 5, vs 40 ± 7.5, p = 0.04).

Discussion
Rupture of the ventricular septum is estimated to occur in 1 to 3% of acute myocardial infarction and accounts for approximately 5% of all infarct-related deaths [5]. Clinical features associated with an increased risk of rupture of the ventricular septum include the lack of the development of collateral circulation, advanced age, hypertension, and possibly thrombolysis [8]. About 50% of patients with VSD are reported to die within the first week and 85% – by the end of the
second week following rupture [1]. Only about 15% of patients can be controlled by medical therapy for a period of 3 to 6 weeks after which surgery can be performed at a greatly reduced risk [1]. Our data shows that patients who were operated on 30 days after AMI had 10% mortality rate, between 10 and 30 days – 18%, and if surgery was performed earlier than 10 days after the onset of AMI, the mortality rate approached 30%. Thus, the patients who were operated on early after AMI had a significantly higher mortality rate because of the more severe clinical and haemodynamic condition. Preoperatively most of our patients had severe clinical and haemodynamic status: 87% of the patients (26 of 30) were in IV functional class, cardiogenic shock developed in one third (30%) of the patients (9 of 30) or pulmonary oedema – in 27% (8 of 30), eleven patients (37%) required IABP support. Majority of the patients of our group were females (63%). 20% of the patients (6 of 30) were older than 70 years. Ventricular septal rupture was the consequence of the first AMI in 26 patients. Angiography was performed in 87% of patients (26 of 30). We think it is important to perform coronary angiography before the operation in order to choose an adequate revascularisation strategy. During coronary angiography an occluded infarct-related coronary artery without collateral circulation was identified in about half of the patients. Most of the patients (21 of 26 (83%)) had extensive coronary artery disease (double, triple vessels or main stem). In the early stage of our experience ventriculography was performed in 6 patients. For the present we prefer to avoid this procedure as it can aggravate the clinical status of the patients. Transesophageal echocardiography provides correct diagnosis in most cases.

It is our policy to try to stabilise patients in the intensive care unit and, if no prompt stabilisation with aggressive medical therapy is achieved, we insert IABP, prior to surgical intervention. Unfortunately, stabilisation is often temporary and patients deteriorate rapidly. Virtually, all patients would die without operation [1]. In the majority of cases, haemodynamic conditions and renal failure deteriorated despite an increase in the drug regimen, so that they had to be promptly operated on.

Comparing the main preoperative and operative factors in the survivors and non-survivors groups we found that diabetes, MVR, ‘no CABG’ were significantly correlated with early mortality. We supposed the diabetic patients had diffuse coronary artery atherosclerosis, more severe functional status of other organs. Mitral valve regurgitation indirectly reflected the extent of myocardium damage. Thus, the most severe patients had these symptoms. More patients in advanced age and with cardiogenic shock were in the non-survivors group, but this difference was not significant. In our experience, age was not a predictive factor for operative mortality. The small study volume could have an influence on this data. By many authors, the impact of old age on operative mortality for post-MI VSD is considered to be important [9]. The GUSTO-I study showed that advanced age was one of the most powerful prognostic factors for increased mortality in VSD patients [10]. In the study of Williams et al [11] the mortality rate of cardiac surgery in octogenarians was significantly higher than in younger patients.

The highest mortality rate (3 of 9 (33%)) was in the early operation (<10 days) group. Though the early operation time was not a predictor of mortality at logistic regression analysis (p = 0.32), but in this group, significantly more patients were in cardiogenic shock (6 of 9 (67%) vs 3 of 19 (16%), p = 0.04), and the aortic cross-clamp time was longer (85 ± 2 vs 68 ± 19 min, p = 0.04) comparing with the patients operated later than 10 days. In the literature, a lot of authors reported that operative mortality was directly related to the interval between infarction and surgical repair [10]. The mortality is higher if the interval is shorter, obviously because the greater the myocardial damage and the haemodynamic instability, the more urgent the need for early intervention.

We found that preoperative echocardiographic left ventricular ejection fraction (LVEF) was significantly (p = 0.02) higher in survivors comparing to non-survivors and this data differed from other studies [12]. Cummings et al [13] found that right ventricular dysfunction was associated with a significantly higher mortality rate. According to our data, there was a difference in the distribution of localisation of myocardial infarction (anterior and posterior). Right ventricular infarction usually associates with posterior infarction [13].

Concomitant myocardial revascularisation was performed in 63% of patients and left ventricular reconstruction in 50% of patients, with the ECC time and aortic clamping time comparable with other studies [9,12]. Dalrymple et al [14] did not demonstrate benefits of revascularisation at the time of VSD repair. They questioned the necessity of concomitant myocardial revascularisation, advocating the increased operative time needed by this procedure, and the necessity of performing coronary angiography that can lead to or increase haemodynamic instability. Other studies [15] demonstrated an improved survival and reduced postoperative recurrence of myocardial infarction after VSD correction with concomitant
CABG. We found that CABG is a significant factor for survival in the early but not in the late period. Of course, our follow-up time was short (26 ± 16 month) and a number of investigated patients [14] was too small to detect some influence of CABG on survival in a remote period. We suppose that CABG should be performed at the time of VSD repair because the long-term prognosis in patients with multivessel coronary disease could be most likely improved by a myocardial revascularisation, as complete as possible. We detected no increased mortality rate due to associated ventricular reconstruction ($p = 0.8$). The technique for the exclusion of infarction was successfully applied in the last few patients operated on early stage of AMI [6].

Conclusions

Mitral regurgitation, diabetes and preoperative left ventricular ejection fraction were specifically important predictive data for early mortality. Coronary artery bypass grafting at the time of repair reduced operative mortality. The left ventricular ejection fraction at discharge from hospital significantly correlates with late mortality. Patients with postinfarct septal rupture should be operated immediately if conservative attempts failed to stabilise haemodynamics. Decision on terms of surgical intervention should be made individually for each patient. Patch closure of the ventricular septal rupture or exclusion of myocardial infarction, reconstruction of the left ventricle and selective myocardial revascularisation provide acceptable results.

References