Surgical repair of postinfarction ventricular septal rupture: Risk factors of early and late death

Runar Lundblad, MD, PhD,[a] Michel Abdelnoor, PhD,[b] Odd R. Geiran, MD, PhD,[a,c] and Jan L. Svennevig, MD, PhD[a,c]

Objective: The aim of the study was to identify risk factors of early and late death after surgical repair of postinfarction ventricular septal rupture.

Methods: During a 25-year period, from May 1981 to August 2006, 102 patients underwent repair of postinfarction ventricular septal rupture. Data were collected on clinical, angiographic, and echocardiographic findings; operative procedures; early morbidity; and survival time. Univariable and multivariable analyses were performed to identify risk factors of 30-day mortality and total mortality.

Results: Thirty-day mortality was 33% altogether and decreased from 45% in the first half to 21% in the second half of the period ($P = .01$). Follow-up was a mean of 5.2 ± 6.2 years and a median of 2.9 years (range, 0–26.3 years). Five- and 10-year cumulative survival was 50% and 32%, respectively. Shock at surgical intervention and incomplete coronary revascularization were strong and independent risk factors of both 30-day mortality and poor long-term survival.

Conclusions: Early outcome after repair of ventricular septal rupture improved significantly during time, with 30-day mortality being 21% in the last decade. Five- and 10-year cumulative survival was 50% and 32%, respectively. Shock at surgical intervention and incomplete coronary revascularization were strong and independent predictors of poor early and late survival.

Ventricular septal rupture (VSR) is a fatal complication after acute myocardial infarction (AMI) and represents a major surgical challenge. Thrombolytic therapy and acute percutaneous coronary intervention (PCI) have led to a 5- to 10-fold reduction in the incidence of VSR,[1-4] but the operative risk remains high. Because of advanced age, heart transplantation and assist devices as a bridge to transplantation are seldom proposed. Although prognosis is extremely poor in medically treated patients,[1,5,6] it is important to identify patients with excessive risk in whom surgical intervention can be avoided. Several predictors of poor outcome have been identified, including large AMI, preoperative shock, right ventricular infarction, posterior VSR, and large left-to-right shunt. In this single-center, retrospective study of 102 consecutive patients undergoing VSR repair, we describe predictors of 30-day mortality and long-term survival.

MATERIALS AND METHODS

This retrospective study was approved by the institutional review board.

Patients

During a 25-year period, from May 1981 to August 2006, 106 consecutive patients from the south of Norway were referred to our hospital with postinfarction VSR. Four patients died before reaching surgical intervention, and therefore 102 patients underwent VSR repair and were included in the study for follow-up. The preoperative cardiologic assessment was done in our institution and included 2-dimensional Doppler echocardiographic analysis and selective coronary angiographic analysis for all patients. Details of revascularization were collected from the operative records and correlated to the angiograms, and each patient was categorized into ''yes'' or ''no'' categories for the 2 variables of (1) complete global coronary revascularization and (2) revascularization of the culprit artery (infarct-related artery). Preoperative and operative risk factors of 30-day mortality are summarized in Table 1. There were 76 men and 26 women with a mean age of 67 ± 8 years. Ninety patients had not experienced AMI previously, and 12 patients had a history of 1 previous AMI. Thrombolysis was performed in 19 cases, and acute PCI was performed in 5 cases. Time from AMI to diagnosis of VSR was a median of 5 days and did not change throughout the study period. Time from VSR to surgical intervention decreased from a median of 3 days in the first half to 1 day in the second half of the study period ($P = .02$). Shock at surgical intervention was present in 14 patients and was strictly defined as hypoperfusion leading to acute oliguria/anuria or acute increase in creatinine value to greater than 200 μmol/L. None of the patients had preoperative chronic renal failure that could confound this definition. Posterior VSR was slightly more common than anterior VSR. Shunt size was estimated in 78 cases by means of echocardiographic analysis or right cardiac catheterization with oximetry, and two thirds of them had Qp/Qs ratios of greater than 2.5. All patients had significant coronary lesions, and for the whole cohort, the frequency of 1-, 2-, and 3-vessel disease was approximately equal. There was a tendency toward more 1-vessel disease in anterior VSR and more 3-vessel disease in posterior VSR ($P = .10$). The left anterior descending coronary artery was the infarct-related artery in all patients with anterior VSR. The right coronary artery system was the infarct-related artery in all patients with posterior VSR, except in 1 patient with occlusion of a dominant circumflex.

Surgical Procedure

The operation was performed through a median sternotomy by using cardiopulmonary bypass (CPB) and moderate systemic hypothermia. Antegrade crystalloid solution (St Thomas II) was infused for cardioplegic arrest in all cases except 2, in which the repair was performed on the beating heart. The VSR was approached through the infarction area of the left

From the Department of Thoracic and Cardiovascular Surgery,[a] Rikshospitalet, Oslo, Norway; the Research Forum, Unit of Clinical Epidemiology and Biostatistics,[b] Ullevål University Hospital, Oslo, Norway; and the Faculty Division,[c] Rikshospitalet, University of Oslo, Oslo, Norway.

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Address for reprints: Runar Lundblad, MD, PhD, Department of Thoracic and Cardiovascular Surgery, Rikshospitalet, N-0027 Oslo, Norway (E-mail: rulund@rikshospitalet.no).

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Abbreviations and Acronyms

AMI = acute myocardial infarction
ASAT = aspartate aminotransferase
CABG = coronary artery bypass grafting
CPB = cardiopulmonary bypass
IABP = intra-aortic balloon pump
PCI = percutaneous coronary intervention
VSR = ventricular septal rupture

ventricle and was repaired with the traditional technique of Daggett and associates[7] or the infarct-exclusion repair of David and colleagues.[8] Daggett repair (n = 67) dominated in the first half and David repair (n = 35) dominated in the second half of the series. In Dagget repair the VSR was patched (Dacron or polytetrafluoroethylene patches) in all cases except 1, in which the defect was directly closed with buttressed interrupted sutures. The ventriculotomy was closed with buttressed sutures or, in a few cases, resected myocardium was replaced with prosthetic material. In David repair the infarcted myocardium and VSR were excluded with a large patch of bovine pericardium and in a few cases supplemented with a second patch directly on the VSR. The ventriculotomy was closed without infarctectomy by using buttressed sutures. Glue was not used. No valve procedure was performed. Coronary artery bypass grafting (CABG) was performed when significant coronary artery disease was present and the vessel periphery was deemed suitable for revascularization. The left internal thoracic artery and saphenous vein were used for grafts. Distal anastomoses were performed on the cardioplegic heart, and finally, proximal anastomoses were performed on a beating heart. Sixty-six patients underwent revascularization: CABG in 61 patients, acute PCI of the culprit artery in 3 patients with 1-vessel disease, and acute PCI of the culprit artery followed by CABG in 2 patients with 3-vessel disease. The mean number of distal anastomoses was 1.8. The infarct-related coronary artery was revascularized in 24 patients (left anterior descending coronary artery in 11 patients and right coronary artery in 13 patients). Of 64 patients with disease in remote coronary territories, 39 underwent complete remote revascularization, 17 had incomplete remote revascularization, and 8 had no remote revascularization. Accordingly, 25 patients left the operating room with remote myocardium at ischemic risk as a consequence of incomplete or no revascularization of non–infarct-related arteries. Altogether, 22 patients underwent complete coronary revascularization (culprit artery plus remote arteries). An intra-aortic balloon pump (IABP) was used in 91 patients and was started preoperatively (>24 hours) in 21 patients and perioperatively in 70 patients. IABP was used for a median of 4 days (range, 0–12 days) after the operation.

Study Design, Data Collection, and Statistical Analysis

The study is a cohort analysis of 102 consecutive patients undergoing operations for postinfarction VSR in our hospital during a 25-years period, from May 1981 to August 2006. This dynamic cohort had different entry times (date of operation), and the common closing date was July 1, 2008. Data were collected from patient records, and survival data were entirely based on information from the Norwegian Death Registry. Follow-up was 100% complete and was a mean of 5.2 ± 6.2 years and a median of 2.9 years (range, 0–26.3 years). Continuous data are presented as the mean ± standard deviation or median (range), and categorical data are frequencies or fractions of patients. End points were 30-day mortality and total mortality (all deaths including 30-day deaths).

Univariable analysis of 30-day mortality was performed with 2 × 2 tables and χ^2 or Fisher's exact tests for categorical data and the 2-tailed *t* test or the Mann–Whitney test for continuous data. Survival curves were plotted according to the Kaplan–Meier method, and differences between curves were pinpointed by using the Breslow test and the log-rank test.[9]

Multivariable analysis was performed for variables that demonstrated statistical significance ($P < .05$) or marginal significance ($P < .2$) in the univariable analysis or that were considered clinically or pathophysiologically important. Accordingly, the following variables were included in the logistic regression analysis and the Cox model: age, sex, diabetes mellitus,

TABLE 1. Patient data and risk factors of 30-day mortality

Variable	All (n = 102)	Survivors (n = 68)	Nonsurvivors (n = 34)	*P* value
Age (y)	67 ± 8	66 ± 8	68 ± 9	>.2
Male/female sex	76/26	49/19	27/7	>.2
BMI	24.9 ± 2.7	25.0 ± 2.7	24.7 ± 2.8	>.2
Diabetes mellitus	12	8	4	>.2
Previous AMI	12	7	5	>.2
Thrombolysis/acute PCI	24	17	7	>.2
Left main stenosis	9	7	2	>.2
1/2/3-Vessel disease	38/30/34	25/18/25	13/12/9	>.2
Anterior/posterior VSR	47/55	34/34	13/21	>.2
AMI to VSR (d)	5 (1–27)	5 (1–27)	2 (1–14)	.20
VSR to surgical intervention (d)	2 (0–60)	2 (0–60)	1 (0–30)	.09
$ASAT_{max}$ (U/L)*	470 ± 355	349 ± 190	665 ± 468	.01
Shunt size >2.5/<2.5†	50/28	30/24	20/4	.02
Shock at surgical intervention	14	5	9	.01
Aortic crossclamp time (min)	64 ± 31	60 ± 29	71 ± 33	>.2
CPB (min)	116 ± 45	109 ± 40	130 ± 53	.04
Coronary revascularization	66	47	19	.19
Complete revascularization	22	18	4	.09
Culprit revascularization‡	24	20	4	.05

Values are presented as means ± SD or medians (ranges) or the number of patients or fractions of patients. *BMI*, Body mass index; *AMI*, acute myocardial infarction; *PCI*, percutaneous coronary intervention; *VSR*, ventricular septal rupture; *ASAT*, aspartate aminotransferase; *CPB*, cardiopulmonary bypass. *Maximal aspartate aminotransferase after acute myocardial infarction measured in 42 patients. †Qp/Qs ratio measured in 78 patients. ‡Revascularization of the infarct-related artery.