Early closure of postinfarction ventricular septal defects

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Myocardial infarction; Ventricular septal defect.

Background. **According to the guidelines of the American College of Cardiology/American Heart Association early closure of postinfarction septal defects is now a class I indication although it still carries a relevant morbidity and mortality. The operative risk is related both to the critical hemodynamic conditions of the patient and to the technical difficulties posed by the friable tissue of the infarcted area. The most recent techniques involving the use of pericardial patches reinforced by acrylic glue have significantly reduced the hospital mortality. The aim of this study was to discuss the reliability of an aggressive, tissue-sparing surgical approach to this complication.**

Methods. **We present a consecutive series of 12 patients operated upon between January 1998 and October 2001 within 12 hours of the onset of clinical evidence of postinfarction septal rupture. Repair was achieved with minimal septal debridement and the use of a large pericardial patch reinforced by a biological glue.**

Results. **Three cases of dehiscence required early reoperation with no hospital mortality.**

Conclusions. **This procedure is technically feasible and allows early aggressive treatment of postinfarction septal rupture with satisfactory results.**

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Introduction

The surgical treatment of postinfarction septal rupture is still a challenge both in terms of the timing of correction and the technique of repair.

The hospital mortality, which in previous reports has been reported to be as high as 35%[1], has declined mostly owing to the introduction of new surgical approaches[2,3].

These techniques, which include septal exclusion by means of a pericardial patch, overcame the necessity of delaying the operation in order to allow for the development of a more resistant fibrous tissue. The possibility of performing an early repair has not only reduced the overall morbidity and mortality related to the waiting period but has also significantly simplified the management of this severe postinfarction mechanical complication.

We present a series of 12 consecutive patients treated immediately after the diagnosis of postinfarction septal rupture with a technique which includes septal exclusion by means of an autologous pericardium and reinforcement of the necrotic tissue with the use of GRF® resorcinol-formaldehyde glue.

No in-hospital death occurred and a stable long-term hemodynamic function persists.

These 12 patients were operated upon employing an original modification of the technique of septal exclusion and with no mortality.

Methods

From January 1998 to October 2001, 12 patients (10 males, 2 females, mean age 64.4 years, range 62-83 years) with a diagnosis of postinfarction septal rupture underwent emergency surgery. The series includes all the postinfarction septal ruptures observed in the area of referral: only one 85-year-old patient died before reaching our unit.

The mean time interval between myocardial infarction and the onset of symptoms of left heart failure was 4.3 days (range 0-15 days). All patients were in NYHA functional class IV, requiring inotropic support. In all cases the diagnosis was made at transthoracic echocardiography. Coronary and ventricular angiography was performed in every patient in order to confirm the presence of the defect and to quantify the coronary involvement. During this procedure, in 6 patients with cardiogenic shock, an intra-aortic balloon pump (IABP) was inserted. The patients were operated on an urgent/emergency basis. The mean time interval from the diagnosis to

operation was 4 hours. In all cases surgery was performed promptly and none of the patients stayed in the intensive care unit overnight before being operated.

The defect was antero-septal in 5 cases and posterior in 7. In all cases there was a severe right to left shunt with a Qp/Qs ratio > 2.5:1 (mean 2.8:1). Five patients had single-vessel disease. Multivessel disease was present in 7 cases; 1 patient had been submitted to coronary bypass surgery 10 years previously and all the grafts were occluded. The left ventricular ejection fraction was depressed in all patients (mean value 34.6%, range 25-62%).

In no case was the vessel responsible for the infarct treated. In 7 cases with multivessel disease a myocardial revascularization was performed.

All patients had an IABP inserted, 6 in the catheterization laboratory, and 6 in the operating room, 3 at the time of induction of anesthesia and 3 before weaning from cardiopulmonary bypass (CPB) (Table I).

Surgical technique. The surgical technique employed is as follows: patients are monitored as usual and invasive lines are placed. In all patients a transesophageal probe is placed to check the outcome of the repair and as a guide to weaning the patient from CPB. During CPB (aortic and bicaval cannulation) the aorta is cross-clamped and myocardial protection is achieved by means of normothermic intermittent blood cardioplegia. Revascularization of all the diseased vessels is performed first. The defect is approached via a left ventriculotomy, anterior or posterior according to the location of the defect, performed on the infarcted area and with the least possible extension. The anterior ventriculotomy should be performed close to the apex in order to minimize the damage of the non-infarcted area. The most important issue in approaching posterior septal lesions is to avoid any damage to the mitral apparatus. Therefore, the posterior incision is started in the mid-paraseptal area and extended posteriorly towards the base of the heart and anteriorly towards the apex.

A careful assessment of the lesions is made: the edges of the infarcted tissue are identified and the postero-septal papillary muscle is carefully evaluated. The fragmented infarcted muscle is left in place.

A suitable patch of autologous glutaraldehyde-fixed pericardium is than secured with a 3/0 polypropylene running suture, placing the stitches deep in the healthy tissue and if necessary reinforced with a strip of pericardium. The inferior margin of the defect is approached first; the suture can be fairly loose, in order to avoid tears in the friable myocardium. Having reached the ventriculotomy site, the pocket between the patch and the infarcted septum is filled with a generous amount of GRF glue. Gentle squeezing of the area allows the transformation of the Swiss-cheese-like septum, the glue and the pericardium into a strong diaphragm.

The pericardial patch must be large enough to avoid tension and stress during the cardiac cycle. The ventriculotomy is then closed with Teflon strips and a 2/0 polypropylene suture.

If the base of the antero-septal papillary muscle is involved, this can be secured into the ventriculotomy closure.

Results

No in-hospital death occurred. The mean intensive care unit stay was 14 days (range 4-35 days).

The IABP was removed within the third postoperative day in 11 patients, while in 1 patient it was maintained for 15 days.

One patient with an anterior and 2 with a posterior defect respectively were reoperated within the tenth

Table I. Patients' data.

Patient	AMI-VSD/ VSD-Op (days)	Angiography	Surgery	EF post (%)	NYHA post/ follow-up (months)
TC	15/0	LM 40%, LAD 100%, LCx 70%, RCA 60%	IVSD + CABG2	38	II/51
BA	0/0	LAD 100%, LCx 80%	IVSD + redo	35	Ex 6/MOF
GD	2/0	RCA 100%	IVSD	60	II/49
BA	1/1	OM 70%, LCx 90%, RCA 100%	IVSD + CABG1 + redo	58	II/24
LC	5/0	LAD 100%, D1 80%, LCx 50%, PD 80%	IVSD + CABG1	25	III/38
GA	1/0	LAD 90%, LCx 90%, RCA 90%	IVSD + CABG2	30	III/31
TA	5/0	RCA 100%	IVSD	25	III/55
CB	1/0	LAD 90%, RCA 100%	IVSD + CABG1	50	I/24
BC	4/0	LAD 100%	IVSD + CABG1	40	II/29
BA	5/1	RCA 100%	IVSD	45	I/18
VF	5/0	RCA 100%	IVSD + redo	40	I/20
MI	7/0	D1 e RCA 100%	IVSD + CABG2	42	II/32

AMI = acute myocardial infarction; CABG = coronary artery bypass graft; D1 = first diagonal branch; EF = ejection fraction; IVSD = interventricular septal defect; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LM = left main coronary artery; MOF = multiorgan failure; OM = obtuse marginal branch; PD = posterior descending coronary artery; RCA = right coronary artery; VSD = ventricular septal defect.

postoperative day because of relapse of the left to right shunt. In all these cases there was a leak in the inferior portion of the pericardial patch.

The most frequent complications were acute respiratory distress (6 cases), renal failure (2 cases requiring temporary filtration), and arrhythmias (1 patient had sustained ventricular tachycardia resistant to intravenous antiarrhythmic therapy with different agents for 20 days).

The mean hospital stay was 20 days (range 10-55 days). The mean follow-up was 30 months (range 15-55 months). One patient with advanced diabetes and peripheral vasculopathy died of multiorgan failure following foot amputation for ischemic gangrene 7 months postoperatively. At necropsy the septum was perfectly consolidated.

Eleven patients are alive, 3 in NYHA functional class III, 6 in class II, and 2 in class I.

The mean ejection fraction measured at echocardiography performed during follow-up was 38%, showing a mild improvement compared to the preoperative value.

In 1 patient a mild, well tolerated residual shunt persists. One patient in NYHA functional class III has moderate mitral and tricuspid regurgitation due to annular dilation. The patient, with episodes of sustained ventricular tachycardia during the initial postoperative course, is presently in sinus rhythm without antiarrhythmic therapy.

Discussion

Postinfarction septal rupture complicates from 1 to 2% of all myocardial infarctions and only 20% of all untreated patients survive through the first month[1]. There is a great direct correlation between the size of the infarct and survival and the infarct size/left to right shunt ratio is a good predictor of survival[4]. As the longer time interval between the development of the infarct and surgery was favorably correlated with survival[5], attempts were made in the past to stabilize patients prior to surgery thus improving the outcome when stabilization was achieved but conversely excluding patients with more severe deterioration of the hemodynamic status.

In 1995 David et al.[2] introduced the concept of septal exclusion with a pericardial patch and Musumeci et al.[3], in a series of 3 patients operated upon during the first week after the development of the infarct, stabilized the patch with resorcin-formol biological glue.

In 1999 the American College of Cardiology/American Heart Association guidelines considered the diagnosis of postinfarction septal rupture as a class I indication for urgent surgery[6].

Since 1998 we operated all patients with postinfarction septal rupture referred to our units, regardless of their age[7], the clinical status, the extent of myocardial infarction[8-10], the coronary artery involvement[11,12], and the site of rupture. Our philosophy was that better results could be obtained by limiting the period of cardiogenic shock which always complements the onset of postinfarction septal rupture prior to the development of multiorgan failure as much as possible. Our surgical strategy is designed to limit myocardial trauma[13] by maintaining the ventriculotomy as small as possible[14], close to the left side of the septum, in the area of epicardial breakthrough of the necrosis. The preservation of the necrotic tissue is a critical point in the closure of the defect. With this technique the infarcted muscle becomes a sort of matrix which is strongly consolidated by the glue; the rough pleural surface of the autologous pericardium adheres to this complex and perfectly seals the septum. The most critical phase of the operation is the fixation of the pericardial patch in the lower crescent of the defect: all our recurrences were located in this area.

We strongly believe that:

- the patch must be slightly redundant in order to allow a tension-free systo/diastolic movement;
- the stitches in that area must be placed in the healthy tissue even if this implies an almost complete exclusion of the septum;
- the running suture is more willingly reinforced with a thin strip of pericardium on the muscular side.

We do not see the need of performing a right approach which can compromise the right ventricular function, does not allow the identification of the left border of the lesion, and exposes the repair to the left systolic stress.

The innovative hints of this technique include:

- the use of the autologous pericardium which combines an ideal plasticity and the absence of the potential problems related to the placement of foreign bodies. Moreover, the smooth cardiac surface of the pericardium is an ideal surface for the blood, whereas the rough pleural surface easily adheres to the muscle-glue complex;
- the preservation of the necrotic septal tissue as a matrix which melts with the glue hence creating a homogeneous strong diaphragm.

We have no evidence of problems related to the potential contact between the glue and circulating blood.

In conclusion, the absence of mortality in this consecutive unselected series involving all the patients referred to our Centers is very encouraging and demonstrates the validity of the approach. The incidence of recurrences is a disturbing factor which could be limited by a meticulous performance of every step of the surgical technique. A prompt diagnosis by means of transesophageal echocardiography in case of a clinical suspicion is mandatory so that an aggressive surgical approach can be performed. In our experience, redo operations did not present particular problems; the septum was repaired in the area of dehiscence with a supplementary small pericardial patch and the edges of ventriculotomy were strong enough to withstand a second closure.

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