Rupture recurrence after surgical repair of postinfarction ventricular septal rupture. Influence of early thrombolysis

Abstract  Objectives. The aim of this study was to identify factors causing rupture recurrence after surgical repair of postinfarction ventricular septal rupture and to evaluate the indication for reoperation.


Results. The mean interval between initial operation and recurrence was 3.6 days with a median of 2 days. Multivariate logistic regression analysis identified early thrombolysis after infarction (P=0.0085) as a risk factor for recurrence of the rupture. Rupture recurrence occurred more in the anterior than in the posterior infarction site, although non-significant. Reoperation was indicated in 15 patients, in 13 for postrecurrent cardiac failure. The main determinant of cardiac failure was a large postrecurrent shunt (P=0.05). The mean interval between initial operation and reoperation was 136 days with a median of 101 days. In 6 patients a combined apical ventricular septal rupture recurrence and anterior ventricular aneurysm was found, in 9 patients the recurrent rupture was proximally located, without concomitant aneurysm formation. Of 15 patients who were reoperated, one died in hospital and three after the in-hospital period. Of 10 patients treated conservatively, one died in hospital and two after the in-hospital period. One residual ventricular septal rupture closed spontaneously.

Conclusions. Rupture recurrence is mainly determined by early thrombolysis. Postrecurrent cardiac failure, as the main indication for reoperation, is dependent on postrecurrent shunt size. [Eur J Cardio-thorac Surg (1996) 10:748–753]

Key words  Myocardial infarction · Recurrent ventricular septal rupture · Surgical treatment

Introduction

Early surgical repair is the treatment of choice for postinfarction ventricular septal rupture (VSR) [5–7, 9, 10, 13, 14]. Delay in operation may result in early death for a considerable number of patients [3, 5, 12]. However, the disadvantage of early surgery is that the septal reconstruction must be performed in weak and fragile recently infarcted tissue, which may result in perioperative failure of the reconstruction or postoperative recurrence of the VSR [3, 5, 9–11, 14]. In the literature recurrence of the VSR has been seen in between 5 and 25% of the operated patients [3, 5, 9–11, 14]. Repair of rupture recurrence is indicated for cardiac failure [9–11, 14].

The present retrospective analysis was undertaken to identify factors which may influence the recurrence of VSR after surgical repair and to evaluate the outcome of conservative and surgical treatment.
Patients and methods

Patient population

From January 1980 to December 1992, 109 consecutive patients were treated surgically for postinfarction VSR. The repair was based on the techniques described by Daggett [2] using prosthetic material. Twenty-five of these patients suffered from recurrence of the VSR. A retrospective analysis was performed on the hospital charts. Follow-up data were complete for all the patients. A division was made in three time periods: 1980–1984, 1985–1988 and 1989–1992. Between 1986 and 1992, 11 patients (of 109) had been treated by early thrombolysis during the acute phase of their myocardial infarction.

Diagnostic procedures

After the reappearance of a systolic precordial bruit, recurrence of the VSR was confirmed by echocardiography or oximetry during right heart catheterization.

Site of infarction and VSR location

The patients were categorized according to the site of the infarction (anterior or posterior) and location of the VSR on the septum (proximal or distal).

Follow-up

Follow-up data were collected at the outpatient department or at the referring centers. The current status of the survivors at last study follow-up was ascertained by telephone with the patient and with the patient’s cardiologist.

Statistical analysis

Statistical analysis was performed according to standard statistical methods, incorporated in the statistical analysis program of SAS. Continuous variables are expressed as the mean ± standard deviation. Means were compared using the paired t-test. Variables not approximately normally distributed were compared using the Mann-Whitney test. For these variables the median is also reported. Proportions were compared using the chi-square test and, where appropriate, the Fisher’s exact test. Stepwise multivariate logistic regression analysis was used to relate clinically meaningful variables to dichotomous or ordinal outcome variables.

Results

Ventricular septal rupture recurrence

Table 1 demonstrates additional data for the patients with and without recurrent VSR after the primary operation with univariate analysis. Early thrombolysis in the acute phase of myocardial infarction was a significantly different variable. The number of recurrences increased in the study period 1989–1992, together with the increasing use of early thrombolysis. The early mortality rate is significantly less in the patients with rupture recurrence.

Risk factors for recurrence of the VSR are listed in Table 2. A strong predictor for recurrence in multivariate analysis

| Table 1 Clinical data for primary operation of VSR for population with and without recurrence of VSR with univariate analysis of differences (VSR ventricular septal rupture, SVD single vessel disease, DVD double vessel disease, TVD triple vessel disease, RAP right atrial pressure, PAP pulmonary artery pressure, AoCCT aortic cross clamp time) |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                  | Without recurrence | With recurrence | P value |
|                  | (n=84)            | (n=25)           |        |
| Age (yrs)        | 67.3 ± 7.3        | 66.4 ± 7.8       | 0.63   |
| Sex (male)       | 53 (63.1%)        | 16 (64.0%)       | 1.0    |
| Diabetes         | 17 (20.2%)        | 1 (4.0%)         | 0.067  |
| Study periods    |                  |                  | 0.65   |
| 1980–1984        | 16 (19.0%)        | 4 (16.0%)        |        |
| 1985–1988        | 34 (40.5%)        | 8 (32.0%)        |        |
| 1989–1992        | 34 (40.5%)        | 13 (52.0%)       |        |
| Time to VSR (days) | 4.4 ± 4.5        | 4.7 ± 4.5        | 0.67   |
| Infarction site (anterior) | 38 (45.2%) | 16 (64.0%) | 0.11 |
| VSR location (proximal) | 53 (63.1%) | 14 (56.0%) | 0.64 |
| Cardiac shock    | 48 (57.1%)        | 15 (56.0%)       | 1.0    |
| Preop creatinine (mmol/l) | 179 ± 90       | 175 ± 125        | 0.42   |
| Vessel disease   |                  |                  | 0.26   |
| SVD              | 37 (46.8%)        | 16 (64.0%)       |        |
| DVD              | 22 (27.8%)        | 6 (24.0%)        |        |
| TVD              | 20 (25.3%)        | 3 (12.0%)        |        |
| RAP mean (mmHg)  | 11.5 ± 5.2        | 9.2 ± 5.0        | 0.051  |
| PAP mean (mmHg)  | 29.2 ± 6.9        | 29.4 ± 5.7       | 0.89   |
| Left to right shunt | 3.1:1            | 3.1:1            | 0.96   |
| Right atrial O₂ saturation | 52.6 ± 12.2 | 52.4 ± 10.7 | 0.92 |
| Early thrombolysis | 5 (5.9%)         | 6 (24.0%)        | 0.017  |
| Delay VSR to operation (days) | 6.1 ± 8.4 | 3.6 ± 4.8 | 0.57 |
| AoCCT (minutes)  | 87 ± 34           | 80 ± 25          | 0.30   |
| CABG             | 36 (42.9%)        | 9 (36.0%)        | 0.65   |
| Early mortality  | 24 (33.3%)        | 2 (8.0%)         | 0.012  |

Values are numbers and percentages or mean ± SD
analysis was early thrombolysis, increasing the risk for recurrence 4.6 times. Anterior infarction site gives rise to recurrence 2 times more than the posterior infarction site, but is statistically not significant.

Clinical data after VSR recurrence

The interval between primary operation and recurrence of the VSR was 3.6 days with a median interval of 2 days (SD 4.0, range 0.5–14 days). After recurrence the mean left-to-right shunt was 2:1 (SD 0.7, range 1.3–4.0). The mean right atrial pressure was 10.3 mmHg (SD 6.0 mmHg) and the mean pulmonary artery pressure was 29.4 mmHg (SD 10.1 mmHg). Figure 1 demonstrates the relationship between values before primary operation and after recurrence for shunt size and mean pulmonary artery pressure.

Postrecurrent cardiac failure

After rupture recurrence, cardiac failure occurred in three patients. Twelve patients experienced cardiac failure after a longer interval.

Postrecurrent cardiac failure, as the main indication for reoperation, was related to variables after recurrence of the VSR. A large postrecurrent left-to-right shunt was the main predictor (odds ratio 4.39, \( P=0.0517 \)). Its relationship with postrecurrent cardiac failure is demonstrated in Figure 2. A shunt size of 1.8:1 appeared to be a breaking point.

Reoperations

A reoperation was carried out in 15 patients. In 13 of these postrecurrent cardiac failure, and in two a ventricular aneurysm, was the primary indication for reoperation; at this time the recurrent VSR was corrected as well. The mean interval between primary operation and reoperation was 136.4 days (SD 128.4 days) with a median interval of 101 days and a range of 3–426 days. Reoperation was carried out in 11 patients for a recurrent VSR after a primary anterior infarction, in four patients after a posterior infarction. In seven patients the primary VSR was proximally located and in eight distally.

Peroperative findings at reoperation

In 12 patients the recurrent VSR was directly adjacent to the primary patch and in three at a small distance from the primary patch. In the four patients with a posterior infarction, the recurrent VSR was located at the cranial site of the patch close to the valvular apparatus, in one case accompanied by a small posterolateral aneurysm. In patients with an anterior infarction, the recurrent VSR was located at the cranial site of the patch in five patients, in one case accompanied by a small ventricular aneurysm as well. In the remaining six patients, the recurrent VSR was located at the apical site of the patch, in all cases accompanied by a large antero-apical aneurysm.

Surgical technique at reoperation

The septum was approached either through the old myocardial incision or through a de novo transaneurysmal incision. The recurrent VSR was closed either by complete replacement of the patch, or by placing an extra patch over the defect, leaving the primary patch in situ, or by reattachment of the fibrous edges of the septum to the primary patch.
Fig. 2 The relationship between postrecurrent shunt size and post-
recurrent cardiac failure shows a significant difference between shunt
size with cardiac failure and without cardiac failure. A shunt size of
1.8:1 seems to be the breaking point.

In five cases the left ventricular aneurysm was corrected
by linear aneurysmectomy [1] and in three cases by a tech-
nique described by Dor [4]. In two cases the reoperation
was accompanied by coronary revascularization and in one
case by a mitral and tricuspidal valve plasty.

Postoperative outcome

Of the 15 patients who were reoperated, one died in hos-
pital (within 30 days after the primary operation) and two
after the in-hospital period, at 2 months and 1 year after
reoperation, respectively. One of them could not be weaned
from extracorporeal circulation during his second reoper-
ation for secondary rupture recurrence. One patient under-
went cardiac transplantation 4.5 years after reoperation.
The 11 survivors of reoperation are in NYHA class I or II.

Non-surgical treatment

Of the ten patients treated non-surgically, two had a period
of cardiac failure. The shunt size was 2:1 for both. They
both refused reoperation. One patient is in NYHA class III,
the other in NYHA class I. One patient died within 30 days
after primary operation due an acute untreatable ventricu-
lar arrhythmia. Two patients died after the in-hospital pe-
riod at 5 and 9 years, respectively, and seven are still alive.
Four patients are in NYHA class I and two patients are in
NYHA class II. In one patient the recurrent VSR, with a
shunt size of 1.3:1, spontaneously closed clinically and
echographically.

Discussion

Rupture recurrence

The number of recurrences of VSR after operative treat-
ment in this series (22.9%) is comparable with earlier stud-
ies [3, 5, 11, 14]. Recurrence of the VSR seems to be in-
herent to surgical repair in freshly infarcted tissue.

Early thrombolysis

The main determinant of recurrence of the VSR was early
thrombolysis in the acute phase after myocardial infar-
cion. Early thrombolysis reduces the 5-week mortality rate
after myocardial infarction by 25%. When used in conjunc-
tion with acetylsalicylic acid the reduction is 42% [8]. The reason for the higher number of recurrences of the VSR after early thrombolysis is unclear. Westaby and associates [15] described the more viable appearance of the infarcted septal tissue after early thrombolysis. This may cause underestimation of the size of the infarcted area and influence the size of septal reconstruction. Another reason might be an additional weakening or edema of the septal myocardium due to reperfusion injury after thrombolytic therapy. After disappearance of the edema, parts of the septal repair may become partially insufficient, before tight connection between patch and septum have been able to occur.

Anterior infarction site

The risk of recurrence in the anterior infarction site is twice as high as in the posterior infarction site, although not significant. This is remarkable, as the technical problems of primary repair in the posterior site, with respect to reachability of the ruptured area, seems obvious. Of the 11 recurrences after anterior site VSR repair, all six apically located rupture recurrences were accompanied by a large ventricular aneurysm. Traction on the sutures in the fresh unresected infarcted area by the aneurysm formation may have caused the recurrence. The high number of recurrences in the anterior wall may raise the question about the necessity of closing the free anterior wall in the same manner as the posterior wall, with patch reconstruction after infarction resection.

The remaining location of the recurrences, anterior and posterior, were all proximally located. This may be related to the reluctance at primary operation to make sufficiently deep stitches close to the valvular apparatus.

Clinical appearance of recurrent VSR

The difference in clinical appearance between the primary VSR and the recurrent VSR is remarkable. The degree of clinical symptoms is considerably less in patients with a recurrent VSR compared to those after a primary VSR, although the interval between primary operation and recurrence is quite short and the patients may still be considered in the acute phase of cardiac damage after their infarction [5, 11, 14]. Most of them suffered left- or right-sided ventricular failure, which was initially treated medically. The moderate severity of the clinical symptoms is also underlined by the long interval between primary operation and reoperation. It seems that, even in cases of sudden recurrence after primary operation, the partial or temporary reconstruction of the interventricular septum may allow the right ventricle to recover functionally, and therefore such an event should not be considered as a failure per se. These phenomena indicate that there is a considerable difference in approach for primary VSR versus recurrent VSR. In primary VSR, surgical repair is advocated independently of the present clinical status, while in this series of patients with a recurrent VSR, reoperation was mainly indicated for medically untreatable cardiac failure. This policy was largely determined by the hesitation to reoperate shortly after a major surgical-technically difficult, and for the patient demanding, operation.

Cardiac failure, as the main indication for closure of the recurrent VSR, was directly related to the postrecurrent shunt. This is in contrast to the relationship between primary shunt and primary clinical picture [5, 11, 13, 14]. However, before the primary operation no shunt was less than 2:1, making the two populations difficult to compare.

Follow-up

The survivors with non-operated residual VSR mainly had good functional status at the last study follow-up, apart from one patient who was in NYHA class III. In one case the residual VSR disappeared spontaneously. This phenomenon has been described before [14]. The mechanism is unclear.

Conclusions

Rupture recurrence after surgical repair of postinfarction VSR is mainly determined by the use of early thrombolysis and anterior infarction site. Aneurysm formation in unresected infarcted tissue may have caused the pronounced number of recurrences in the anterior infarction location. Postrecurrent cardiac failure, which is the main indication for reoperation, is dependent on the size of the left-to-right shunt. Rupture recurrence does not indicate a complete failure of primary VSR repair.

References

Discussion

Dr. P. Moulder (New Orleans, Louisiana). Just comment. You should not say “cardiogenic shock”. These hearts are marvelous. It is arteriovenous shunt overload failure. The second is a question, can you tell us it is arteriovenous shunt overload failure: an argument for early operation. Surgery 89:48–55

Dr. Cox: Recurrence does mostly occur close to or related to stitches in infarcted tissue, mostly in the anterior infarction site. I believe, there are not many arguments why we should not use the same technique in the repair of the anterior free wall as used in the posterior free wall, with patch repair. Recurrence rate might also decrease with an exclusion of the infarcted septum, as described by da Silva in 1989 and, recently, by David.

Dr. R. Dion (Brussels, Belgium). I would like to congratulate the group on these excellent results that we are unable to reproduce for that pathology in our center. So my question is, when do you contraindicate myocardial surgery? Do you follow the guidelines of Daggett?

For primary repair, do you apply the criteria of Daggett with regard to the level of serum creatinine? You do not seem to have any patients above 70 years of age. Do you apply some selection at primary operation or at reoperation that makes the series more homogeneous and renders the procedure more feasible?

Are you operating on patients above 70 years of age?

Dr. Cox: For primary repair the guideline is that all patients will be operated on when there is no contraindication, which is the guideline of Dr. Daggett. For secondary repair the indication was cardiac failure. At analysis of the primary population, neither serum creatinine nor age was a determinant of early mortality (unpublished data). In the primary population the oldest patient was 84 years of age.