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Importance of coronary revascularization for late survival after postinfarction ventricular septal rupture

A reason to perform coronary angiography prior to surgery

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Background No consistent data are available on the specific coronary artery pathology leading to postinfarction ventricular septal rupture. The benefits and risks of coronary angiography and subsequent coronary artery bypass grafting in these patients is under discussion.

Methods Clinical and coronary angiographic factors were analysed in 109 consecutive patients treated surgically for postinfarction ventricular septal rupture between 1980 and 1992. Coronary angiography was performed in 104 patients, and 92 of the angiograms were available for complete analysis. Factors were related to late cardiac mortality in 79 patients surviving the early period.

Results Single-vessel disease was found in 58 patients (55·8%) and multiple-vessel in 46 (44·2%). In 24 patients (26·1%) there was some collateral circulation to the infarct-related coronary artery. No relationship was found between dominance, occlusion location and early or late outcome. Forty-five patients (41·3%) underwent coronary revascularization in addition to surgical closure of the ventricular septal rupture. Risk factors for late cardiac mortality in patients surviving the early postoperative period were postoperative cardiac failure ($P=0·0089$), incomplete coronary revascularization ($P=0·024$) and longer aortic cross-clamp time ($P=0·032$).

Conclusion We conclude that concomitant complete revascularization is indicated during surgical repair of postinfarction ventricular septal rupture.
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Key Words: Postinfarction ventricular septal rupture, coronary angiography, coronary artery bypass grafting.

Introduction

General consensus has been reached concerning the necessity of early surgical repair as the treatment of choice in postinfarction ventricular septal rupture[1–7]. However, there is no consensus about the indication for concomitant coronary angiography and coronary revascularization, although data have become available about the benefits of concomitant coronary angiography and subsequent coronary artery bypass grafting on direct postoperative outcome and late survival[1,2,5,8–13].

The aim of the present retrospective study was to evaluate coronary angiographic findings in a large group of patients with ventricular septal rupture, to assess if a specific pattern of coronary angiographic lesions could be identified leading to septal rupture, to assess the benefits and risks of the procedure and to evaluate the benefits of concomitant coronary revascularization on long-term survival.

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Methods

Patient population

From January 1980 to December 1992, 109 consecutive patients were treated surgically for postinfarction ventricular septal rupture in our institution. They were referred for surgery from 31 different hospitals. A retrospective analysis was performed. A division was made into three periods: 1980–1984, 1985–1988 and 1989–1992.

The patients were categorized according to the site (anterior or posterior) of the infarction and location (proximal or distal) of the rupture[7]. The follow-up data of the 60 survivors were ascertained by telephone with the patient or the patient's cardiologist. They were complete for all patients.

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Table 1 Patient data surgical repair postinfarction VSR

	Total population (n=109)	Early survivors (n=79)
Age (years)	67 1 ± 7·2	67·3 ± 7·5
Sex (male)	69 (63·3)	53 (67·1)
Prior angina	24 (22·0)	17 (21·5)
Prior infarction	6 (5·5)	4 (5·1)
Anterior infarction site	54 (49·5)	42 (53·2)
Proximal VSR location	67 (61·5)	44 (55·7)
Time to VSR (days)	4·5 ± 4·5	4·9 ± 4·7
Pre-operative shock	62 (58·7)	36 (45·6)
Pre-operative creatinine (μmol . l^{-1})	178 ± 99	167 ± 95
PAP mean (mmHg)	29·3 ± 6·7	29·6 ± 7 2
Left to right shunt	3 1:1	3·1:1
Right atrial O_2 saturation (%)	52·6 ± 11 8	54·3 ± 12·1
Delay from VSR to operation (days)	5·6 ± 7·7 median 1	6·4 ± 8·2 median 2
AoCCT (min)	86 ± 32	85 ± 33
Postoperative cardiac failure	29 (26·6)	5 (6·3)

VSR=ventricular septal rupture; PAP=pulmonary artery pressure, AoCCT=aortic cross-clamp time; Values are numbers and percentages or mean ± SD

Table 2 Coronary anatomy and pathology for 92 revised coronary angiographies

	RCA occlusion (n=48)	RDA occlusion (n=44)
Coronary dominance		
Right dominant	36 (75·0)	20 (45·4)
Balanced	11 (22 9)	14 (31·8)
Left dominant	1 (2·1)	8 (18·2)
Occlusion location		
Proximally	22 (45·8)	8 (18·2)
Distally	26 (54·2)	36 (81·8)
Vessel disease		
SVD	23 (47·9)	27 (61·4)
DVD	15 (31·3)	11 (25·0)
TVD	10 (20·8)	6 (13·6)

RCA=right coronary artery; RDA=ramus descens anterior; SVD=single-vessel disease; DVD=double-vessel disease; TVD=triple-vessel disease

Diagnostic procedures

After confirmation of the diagnosis, coronary angiography was performed in 104 patients and ventriculography in 59. Five patients were considered to be too ill to justify angiography. Ninety-two coronary angiograms were available for complete analysis. They were evaluated by means of a scoring system, which included assessment of left ventricular function, coronary artery anatomy and degree and localization of coronary artery pathology[14]. Dominance of the coronary system was estimated by counting the number of posterolateral branches. A significant coronary artery lesion was defined as a reduction of at least 70% of the luminal diameter. A coronary occlusion was labelled proximal when it was located proximal to the first major side branch (the major right ventricular branch of the right coronary artery and either the first diagonal or the first septal branch of the left anterior descending artery).

Statistical analysis

Statistical analysis was performed according to standard statistical methods incorporated in the S.A.S. program. Continuous variables were expressed as the mean ± standard deviation. Means were compared using the t-test or the one-way analysis of variance. Proportions were compared using the χ^2 test or the non-parametric Wilcoxon test, event-free proportions by means of the log-rank test. Cox's proportional hazard method was used to relate clinical variables to survival.

Results

Patients data and coronary angiography

Patient data of the total population and of the patients surviving the first 30 days are listed in Table 1. The haemodynamic status of five of the 104 patients deteriorated to some extent during angiography. Single-vessel disease was found in 57 patients (54·8%), double-vessel disease in 29 (27·9%) and triple-vessel disease in 18 (17·3%).

Analysis of coronary angiograms and ventriculograms

The angiographic data of the 92 patients, with complete analysis of their angiogram, are listed in Tables 2 and 3. No statistical significant relationship could be found between site of occlusion, pre-operative clinical picture and outcome. In 24 patients there was some evidence of collateral circulation to the infarct-related vessel, and 14 of these patients had multiple-vessel disease. No relationship was found between angiographic left ventricular wall motion score, angiographically determined diastolic and systolic volumes and outcome.

Coronary artery bypass grafting

Coronary artery bypass grafting was performed in 45 patients (41·3%). The mean number of distal anastomoses was 1·8 (SD 1·0, range 1–6). In 36 patients with double- and triple-vessel disease, complete revascularization of coronary arteries not involved in the infarcted area was provided. In 10 patients, revascularization was performed on the coronary artery involved in the infarcted area, mostly a first diagonal or acute marginal

Table 3 Analysis of occlusion site in 92 revised coronary angiographies, with univariate analysis of differences

	Location of occlusion		*P* value
	Proximal (n=30)	Distal (n=62)	
Age (years)	68·1 ± 8·7	66·8 ± 7·0	0·42
Prior angina	10 (33·3)	11 (17·7)	0·12
Prior infarction	2 (6·7)	5 (8·1)	1·0
Time to VSR (days)	4·8 ± 4·4	4·7 ± 4·9	0·70
Proximal VSR location	21 (70·0)	34 (54·8)	0·18
Pre-operative shock	20 (66·7)	33 (53·2)	0·27
PAP mean (mmHg)	28·2 ± 4·4	29·3 ± 7·2	0·48
Shunt size	3·2:1	2·9:1	0·40
Right atrial O_2 saturation (%)	48·4 ± 12·8	53·7 ± 11·4	0·047
Vessel disease			0·45
SVD	14 (46·7)	36 (58·1)	
DVD	11 (36·7)	15 (24·2)	
TVD	5 (16·7)	11 (17·7)	
Pre-operative creatinine (μmol l^{-1})	214 ± 137	158 ± 87	0·0083
Postoperative cardiac failure	11 (36·7)	18 (29 0)	0·48
Early mortality	10 (33·3)	15 (24·2)	0·45
Late cardiac mortality	5 (16·7)	6 (9 7)	0·33

VSR=ventricular septal rupture; PAP=mean pulmonary artery pressure; SVD=single-vessel disease; DVD=double-vessel disease, TVD=triple-vessel disease.

branch located proximally from the main occlusion. In 11 patients with double- (six patients) or triple-vessel disease (five patients) no or incomplete revascularization was carried out. In three patients, this was due to misjudgement of the coronary pathology and in eight patients reluctance by the surgeon to perform concomitant surgery.

To evaluate the possible benefit of concomitant coronary revascularization, the patients were divided into three groups. Group I consisted of 52 patients with single-vessel disease without revascularization, group II of 31 patients with double- or triple-vessel disease with complete revascularization, plus 10 patients with revascularization of a branch located proximal to the occlusion; and group III, 11 patients with double- and triple-vessel disease with no or incomplete revascularization. Data of these groups are presented in Table 4.

Late mortality

Nineteen patients died after the in-hospital period (30 days after primary operation). Of these, 15 (13·8%) died from cardiac, two from pulmonary and two from abdominal disease. Risk factors for late cardiac mortality are listed in Table 5. Strong predictors of late cardiac mortality were postoperative caradiac failure, increasing the risk for late cardiac death 20-fold and incomplete revascularization increasing the risk for late cardiac death 12-fold. Longer aortic-cross-clamp time was another predictor of late cardiac mortality.

Reoperation

In 14 of 25 patients with rupture recurrence, reoperation and closure was performed. Coronary revascularization was performed subsequently in three patients and aortic valve replacement in two patients. One patient underwent surgery for ventricular arrhythmias and one patient cardiac transplant. In six patients (5·5%) a permanent pacemaker was inserted for persistent atrioventricular conduction block.

Survival

Of the 60 survivors, 34 (56·7%) were in NYHA class I, 22 (36·7%) in NYHA class II and four (6·7%) in NYHA class III at the last follow-up. One patient was on medical treatment for symptomatic coronary artery disease.

Discussion

Patient selection and delay in operation

The patients were referred to our centre from 31 different hospitals. This resulted in a selection of patients beyond our scope and renders comparison of the results of overall treatment of postinfarction ventricular septal rupture impossible.

Early surgical repair is the treatment of choice for postinfarction ventricular septal rupture in our hospital[1-7]. It is therefore important to note that the

Table 4 Analysis of degree of revascularization in 104 patients with coronary angiography with univariate analysis of differences

	Degree of revascularization			*P* value
	Not indicated (n=52)	Complete (n=41)	Incomplete (n=11)	
Age (years)	66·6 ± 6 8	68·0 ± 7·2	65 2 ± 8 0	1·0
Sex (male)	34 (65·4)	25 (61 0)	7 (63·6)	0·91
Prior angina	7 (13·4)	14 (34·2)	2 (18·2)	0·055
Prior infarction	3 (5·8)	4 (9·8)	0	0·48
Time to VSR (days)	4·2 ± 4 4	4 8 ± 4·5	4 6 ± 5·2	0·72
Anterior infarction site	27 (51·9)	18 (43·9)	5 (45·5)	0·73
Proximal VSR location	33 (63·5)	22 (53·7)	9 (81·8)	0·22
Cardiogenic shock	30 (57 7)	22 (53·7)	7 (63 6)	0·82
PAP mean (mmHg)	29·4 ± 6 6	28·7 ± 6·6	27 3 ± 5·7	0·32
Shunt size	3·1:1	3·0 1	3 1:1	0·81
Right atrial O_2 saturation (%)	52 6 ± 12·8	52·6 ± 9·7	48·9 ± 14·7	0·49
Pre-operative creatinine ($\mu mol \cdot l^{-1}$)	187 ± 118	157 ± 66	175 ± 45	0·25
Delay from VSR to operation (days)	5·4 ± 8·0 *median 1*	6·0 ± 8·0 *median 2*	2·5 ± 3·6 *median 0·5*	0 37
AoCCT (min)	78 ± 29	98 ± 33	83 ± 33	0·070
Postoperative cardiac failure	15 (28·8)	13 (31·7)	4 (36·4)	0·87
Early mortality	14 (26 9)	10 (24·4)	3 (27 3)	0 73
Late cardiac mortality	8 (15·4)	7 (17·1)	3 (27·3)	0·81

VSR=ventricular septal rupture; PAP=pulmonary artery pressure; AoCCT=aortic cross-clamp time. Values are numbers and percentages or mean ± SD.

mean delay for operation was 5·6 days, with a median delay of 1 day. This may be partly explained by the different referral policies of the various hospitals. In some cases patients were referred only after their condition deteriorated. In five cases the delay was patient-related; they were suffering from ambulatory infarction and ventricular septal rupture, and only presented themselves when their clinical condition had deteriorated.

Table 5 Determinants of late cardiac mortality; Cox's proportional hazard method

	Odds ratio (± 95% CI)	*P* value
Postoperative cardiac failure	20·7 (2·1–201·3)	0·0089
Incomplete coronary revascularization	12·3 (1·4–109·1)	0·024
AoCCT	1·03 (1·00–1·06)	0·032
Gender (male)	0·10 (0·008–1·1)	0·062
Interval from AMI to VSR ≤1 day		0·10
PAP mean		0·14
VSR location		0·15
Pre-operative shock		0·25
Right atrial O_2 saturation		0·34
Pre-operative creatinine		0·37
Shunt size		0·44
Age (5 year interval)		0·62
Site of infarction		0·88
Study periods		0·92

CI=confidence interval; AoCCT=aortic cross-clamp time; AMI=acute myocardial infarction; VSR=ventricular septal rupture; PAP=pulmonary artery pressure; AV=atrioventricular.

Coronary angiography

No general consensus has been reached concerning the possible disadvantages of coronary angiography on the peri-operative period and the benefits of concomitant revascularization on late outcome and rupture closure[1,2,5,8–13]. Angiography is thought to be poorly tolerated haemodynamically and might contribute to the development of renal failure[10,11]. However, a negative effect of coronary angiography on renal function could not be found in our study. In most of our cases, the angiography was restricted to minimal contrast injections in the coronary arteries. Skillington and associates have advised refraining from angiography in ruptures located anteriorly, since this site is often associated with single-vessel disease[5]. The number of patients with single- and multiple-vessel disease in our series seemed to be more balanced. Coronary angiography was omitted in some critically ill patients to save time. Of the 104 patients who underwent coronary angiography, the haemodynamic status deteriorated to some extent in five. Whether this deterioration was related to the angiography is unclear.

No significant differences were found between proximal or distal occlusion and early and late outcome; however, there seems to be a tendency towards a more severe clinical picture with respect to pre-operative shock, shunt size, right atrial oxygen saturation, pre-operative serum creatinine levels, post-operative cardiac failure, and early mortality when the occlusion is located proximally. The difficulty of relating occlusion location to outcome may be caused by the complexity of the coronary anatomy, with respect

to dominance, vessel size, collaterals and aberrant vessel anatomy.

Coronary revascularization and late mortality

In the literature there is doubt about the necessity of performing coronary revascularization in addition to rupture repair[11,12]. Due to the fact that revascularization is performed only when indicated, it is difficult to compare early and late outcome of populations with and without revascularization. Muehrcke and associates demonstrated the benefits of coronary revascularization in late survival in univariate analysis[13]. In our series, multivariate analysis showed that incomplete revascularization was found to be a major risk factor for late cardiac mortality. Although it was assumed that revascularization was of secondary importance compared to the impact of the closure of the ventricular septal rupture, optimal revascularization of the residual viable myocardium may contribute to better recovery. The reason for withholding complete revascularization was a reluctance to perform concomitant surgery or misjudgement of coronary pathology. In our series, the length of the aortic cross-clamp time and coronary revascularization had no influence on early outcome[7].

In a previous report we identified pre-operative cardiogenic shock as the main risk factor for early mortality, which was related to proximal rupture location and right ventricular overload[7]. Postoperative cardiac failure, as the main determinant of late cardiac mortality, is likely to be related to residual left ventricular function after closure of the ventricular septal rupture.

Survival

The greatest risk for death occurs in the early postoperative period. It is remarkable that despite the severity of the peri-operative situation, patients who survive the initial period will live for a long time, with a good quality of life.

Conclusion

Coronary anatomy and pathology could not be clearly related to early and late outcome. Late cardiac death is predicted by postoperative cardiac failure and incomplete revascularization. Concomitant complete coronary revascularization is indicated during surgical repair of postinfarction ventricular septal rupture in order to improve late survival.

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