

Early predictors of adverse left ventricular remodelling after myocardial infarction treated by primary angioplasty

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Abstract

Background: *Progressive left ventricular dilatation (PLVD) occurs after myocardial infarction (MI), and this may take place in the area of primary percutaneous coronary intervention (PCI). The factors predicting PLVD after primary PCI still need to be clarified. The aim of the study was to assess the prevalence and to define the baseline clinical and echocardiographic predictors of PLVD in patients with STEMI treated by primary PCI.*

Methods: *Of the 90 patients initially selected for the study 88 (29 women and 59 men, mean age 67.1 ± 5.6 years) with first ST-elevation myocardial infarction (STEMI) treated with primary PCI were examined. Echocardiographic examination was performed in all patients at discharge (M1) and after 6 months (M2). The following factors influencing PLVD were evaluated: type of infarct-related artery (IRA), infarct size expressed as wall motion score index (WMSI) ≥ 1.5 , left ventricular end-diastolic volume index (LVEDVI) ≥ 80 ml/m², ejection fraction (EF) $\leq 45\%$, restrictive pattern of transmitral flow, time to reperfusion, left ventricular mass index (LVMI) ≥ 125 g/m² and coronary risk factors.*

Results: *The overall prevalence of PLVD (according to the criterion of 20% LVEDVI increase from M1 to M2) was 24%. Univariate regression analysis revealed that the following were the significant baseline M1 predictors of adverse PLVD: left anterior descending as IRA (relative risk: $rr = 2.3$, $p < 0.05$), WMSI ≥ 1.5 ($rr = 4.29$, $p < 0.005$), EF $\leq 45\%$ ($rr = 2.89$, $p < 0.005$) and a restrictive pattern of transmitral flow ($rr = 2.4$, $p < 0.01$). Multivariate logistic analysis showed that the only independent determinant of PLVD was WMSI ≥ 1.5 .*

Conclusions: *Both regional and global left ventricular systolic dysfunction indices as well as severe left ventricular diastolic abnormalities but not left ventricular dilatation at discharge are significant predictors of adverse cardiac remodelling after STEMI in patients treated with primary PCI. However the only independent determinant of PLVD was WMSI ≥ 1.5 expressing the infarct size. (Cardiol J 2007; 14: 238–245)*

Key words: myocardial infarction, echocardiography, left ventricular remodelling

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Introduction

Complex alterations in the architecture and function of the left ventricle (LV) following myocardial infarction (MI), referred to as “LV remodelling,” can affect the patient’s prognosis [1–3]. LV remodelling is a heterogeneous process, involving both infarcted and non-infarcted zones, which affects wall thickness and chamber size, shape and function. LV dilatation following MI precedes deterioration of exercise performance and plays a role in the development of chronic heart failure [1–3].

From a clinical viewpoint LV remodelling is a dynamic process starting in the acute phase with an infarct expansion, leading to myocardial thinning and lengthening and progressing to LV dilatation [1].

It is known that early reperfusion treatment improves survival by limiting infarct size and consequently preserving LV function. Early reperfusion therapy and patency of the infarct-related artery (IRA) is crucial for reducing infarct expansion and LV enlargement [2–4].

The favourable effects of early IRA patency on LV remodelling are suggested by several studies in the thrombolysis area [2–4]. Recently there has been increased interest in the prevalence of remodelling in the area of the interventional cardiology. Some investigators have tested the hypothesis that LV remodelling occurs after percutaneous coronary intervention (PCI), despite persistent patency of the IRA, and may influence the prognosis [5, 6].

From a clinical viewpoint it is important to identify those patients at high risk of LV remodelling. The extent of the baseline myocardial damage is linked to the magnitude and the timing of LV dilatation [7]. The development of LV remodelling expressed by progressive left ventricular dilatation (PLVD) after MI is a complex process influenced by many factors. Infarct size, anterior location, the perfusion status of the IRA, a restricted pattern of LV filling and heart failure on admission have been identified as predictors of LV dilatation after MI in the thrombolysis area [2]. The factors predicting PLVD after MI treated by PCI remain to be clarified.

The purpose of the study was to assess the prevalence of PLVD patterns during the first 6 months after MI treated with primary PCI and to define at discharge the clinical, angiographic and echocardiographic predictors of the PLVD pattern.

Methods

Of the 90 patients initially selected for the study 2 were excluded owing to inadequate echocar-

diography image quality. The study group consisted of 88 patients (29 women and 59 men, mean age 67.1 ± 5.6 years) with first ST elevation myocardial infarction (STEMI), who had been referred to the catheterisation laboratory and were undergoing primary PCI. Perfusion of the infarct region by the IRA was assessed according to the criteria of the Thrombolysis in Myocardial Infarction (TIMI) Trial [8]. Primary PCI was performed < 12 h from the onset of symptoms. The patients with clinical signs of congestive acute heart failure or cardiogenic shock in the first week after MI, post-infarction angina, significant mitral regurgitation or valve disease were excluded, as well as subjects with atrial fibrillation, atrioventricular conduction abnormalities and pacemaker insertion.

Complete M-mode, two-dimensional echocardiography and Doppler ultrasound examination were performed with SONOS Image-Point at the time of discharge (M1) and 6 months after MI (M2). LV volume and ejection fraction (EF) were measured using Simpson’s formula (rule algorithm). The LV volume indices were obtained by dividing the volume of the body surface area [9]. The mean values of three measurements of the technically best cardiac cycles were taken from each examination performed by two independent inter-observers.

In each patient the wall motion score index (WMSI) was derived. The LV was divided according to a 16-segment model. For each segment wall motion was scored from 1 (normal) to 4 (dyskinetic). The ratio of the short axis cavity area to the apical four-chamber cavity area (long axis) at end-diastole was taken as a sphericity index.

LV mass was calculated by the formula introduced by Devereux et al. [10] and normalised to the body surface area to obtain the left ventricular mass index (LVMI). Left ventricular hypertrophy (LVH) was considered as $LVMI > 125 \text{ g/m}^2$.

From the echo Doppler LV diastolic filling pattern the following variables were calculated at baseline: peak velocity of early rapid filling wave E, peak velocity of atrial wave A, peak E/A wave velocity ratio and DT. A restrictive pattern was considered as $DT < 140 \text{ ms}$.

An increase in left ventricular end-diastolic volume index (LVEDVI) $> 20\%$ between M1 and M2 was considered as a PLVD pattern. According to the PLVD pattern, patients were divided into two groups: group I (n = 25) with PLVD (D+) and group II (n = 65) without PLVD (D-).

The following factors baseline influencing PLVD were evaluated:

- type of IRA: left anterior descending (LAD) vs. non-LAD;

Table 1. Baseline (M1) clinical and angiographic characteristics of patients in group I (D+) and group II (D-).

	Group I (D+), n = 25	Group II (D-), n = 63	p
Age [years]	57.5 ± 10.6	60.9 ± 11.8	Ns
Sex (female/male)	5/20	24/39	Ns
Risk factors ≥ 2	3 (12%)	19 (30%)	Ns
hyperlipidemia	8 (32%)	30 (47%)	Ns
hypertension	11 (44%)	36 (57%)	Ns
diabetes	4 (16%)	23 (36%)	Ns
Infarct-related artery–left anterior descending	12 (48%)	13 (20%)	≤ 0.05
Multivessel coronary artery disease	12 (48%)	19 (30%)	Ns
TIMI flow grade > 2	20 (80%)	58 (92%)	Ns
Percentage of stenting	20 (80%)	51 (81%)	Ns
Symptoms to balloon time ≥ 4 h	17 (68%)	32 (50%)	Ns

(D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation

- multivessel coronary artery disease;
- symptom to balloon time ≥ 4 h;
- percentage of stenting;
- 2 or more risk factors (diabetes, hypertension, smoking, dyslipidemia);
- infarct size expressed by WMSI ≥ 1.5;
- LVEDVI ≥ 80 ml/m²;
- left ventricular end-systolic volume index (LVESVI) ≥ 40 ml/m²; cut-off values of LVEDVI and LVESVI were established on the basis of the mean values plus two standard deviations in the control group;
- EF (by Simpson’s method) ≤ 45%;
- LVH, defined as LVMI ≥ 125 g/m²;
- R-restrictive patterns of transmitral flow considered as DT < 140 ms).

Statistical analysis

For each parameter mean, median and standard deviation were calculated. Statistical significance between means for different groups was calculated by the non-parametrical Wilcoxon signed rank test (the number of cases was too small to use parametrical tests). Statistical significance between frequencies was calculated by the χ^2 test with Yates’ correction or, if the expected value was less than 5, by Fisher’s exact test. Relative risk (rr) and confidence interval were also calculated. A p value of less than 0.05 was required to reject the null hypothesis. Statistical analysis was performed using the EPIINFO Ver. 3.3.2 (09–02–2005) software package. The following clinical, angiographic and echocardiographic variables were taken into account in univariate analysis: type of IRA (LAD vs. non-LAD), the presence of multivessel coronary artery disease, symptom to balloon time > 4 h, 2 or more risk factors (diabetes, hypertension, smoking, dyslipidemia), WMSI ≥ 1.5,

LVEDVI ≥ 80 ml/m², LVESVI ≥ 40 ml/m², EF ≤ 45%, LVMI ≥ 125 g/m² and the presence of restrictive patterns of transmitral flow. The variables that were significant in univariate analysis were entered into multivariate models.

Results

The clinical and angiocardiographic characteristics of patients in group I, n = 25 (D+) and in group II, n = 63 (D-) are shown in Table 1. Neither group showed any difference with regard to age, sex, hyperlipidemia, diabetes or hypertension, nor were there differences regarding the time of onset of symptoms to reperfusion. There was no difference in the groups with respect to the extent of coronary artery disease or TIMI flow grade. There was no significant difference in the percentage of stenting between the groups, 80% in group I (D+) vs. 81% in group II (D-) (Table 1).

LAD as the IRA was more common in patients with PLVD in group I (D+) with an incidence of 48% compared to 20.6% in group II (D-); p < 0.05.

The groups did not differ with respect to therapy in secondary prevention: aspirin, statins and beta-blockers, although ACE-inhibitor therapy was recommended during 6-month follow-up more often in group I (D+), 96% vs. 38% in group II (D-). We had no influence on therapy after discharge. In group I (D+) 26% of patients had revascularisation procedures during the 6-month follow-up in comparison with 20% of patients in group II (D-); NS.

The overall prevalence of PLVD, according to the criterion of a 20% LVEDVI increase from M1 to M2, was 24%. The echocardiographic baseline characteristics of patients with and without PLVD

Table 2. Baseline (M1) echocardiographic characteristics of patients in group I (D+) and group II (D-).

	Group I (D+), n = 25	Group II (D-), n = 63	p
LVEDVI [ml/m ²]	74.2 ± 16.4	69.6 ± 13.1	NS
LVESVI [ml/m ²]	36.9 ± 12.1	29.1 ± 11.1	≤ 0.005
Ejection fraction [%]	46.7 ± 8.3	55.3 ± 7.0	≤ 0.0001
Wall motion score index	1.87 ± 0.51	1.47 ± 0.34	≤ 0.0001
Sphericity index	0.639 ± 0.079	0.557 ± 0.126	≤ 0.05
Interventricular septum thickness [cm]	1.01 ± 0.25	1.2 ± 0.19	NS
Posterior wall thickness [cm]	0.90 ± 0.15	1.11 ± 0.2	NS
Left ventricular mass index [g/m ²]	201.1 ± 60.6	196.3 ± 51.9	NS
E [cm/s]	0.61 ± 0.20	0.56 ± 0.20	NS
A [cm/s]	0.58 ± 0.20	0.60 ± 0.20	NS
Deceleration time [s]	0.150 ± 0.035	0.162 ± 0.020	NS
Isovolumetric relaxation time [s]	0.120 ± 0.029	0.126 ± 0.020	NS

(D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation; LVEDVI — left ventricular end-diastolic volume index; LVESVI — left ventricular end-systolic volume index; E — peak of early filling wave; A — peak of atrial filling wave

Table 3. Six months follow-up (M2) echocardiographic indices of left ventricular remodelling pattern.

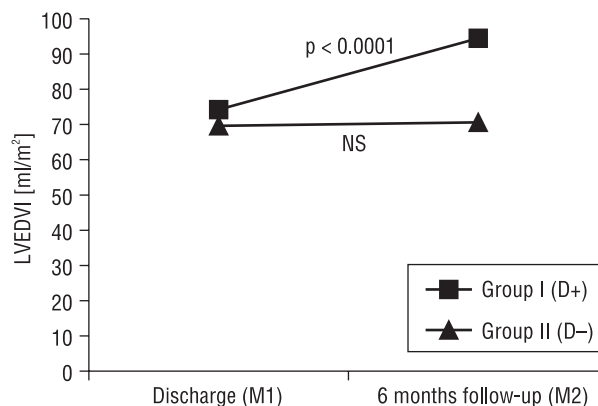
	Group I (D+), n = 25	Group II (D-), n = 63	p
LVEDVI [ml/m ²]	94.5 ± 19.3	70.6 ± 13.5	≤ 0.0001
LVESVI [ml/m ²]	45.3 ± 12.7	30.2 ± 11.4	≤ 0.0001
Ejection fraction [%]	46.4 ± 11.4	57.1 ± 7.8	≤ 0.0001
Wall motion score index	1.87 ± 0.61	1.45 ± 0.36	≤ 0.01
Sphericity index	0.646 ± 0.105	0.558 ± 0.208	NS

(D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation; LVEDVI — left ventricular end-diastolic volume index; LVESVI — left ventricular end-systolic volume index

are shown in Table 2. The 6-month follow-up measurements (M2) of the LV remodelling indices are shown in Table 3.

The LVEDVI tended to be greater at baseline in patients in group I (D+) than in group II (D-), but these differences were statistically insignificant (Table 2). At baseline both global and regional contractile functions were significantly poorer in group I (D+) than in group II (D-). Changes in LV volume indices in both groups are shown in Figures 1 and 2. A significant improvement in EF was observed in group II (D-), whereas it remained unchanged throughout the study period in group I (D+) (Fig. 3). WMSI remained unchanged during the study period in both groups (Fig. 4). In group I (D+) increasing LVEDVI and LVESVI (Fig. 1, 2) were associated with increasing distortion of the cavity shape towards the spherical (Fig. 5).

At discharge and at 6-month follow-up the sphericity index was significantly greater in group I (D+), at 0.63 ± 0.07 , than in group II (D-), at 0.55 ± 0.12 (Fig. 5).


Figure 1. Changes in left ventricular end-diastolic volume index (LVEDVI) in group I (D+) and group II (D-); (D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation

Predictors of progressive left ventricular dilatation

The univariate analysis took into account clinical, angiographic and echocardiographic variables.

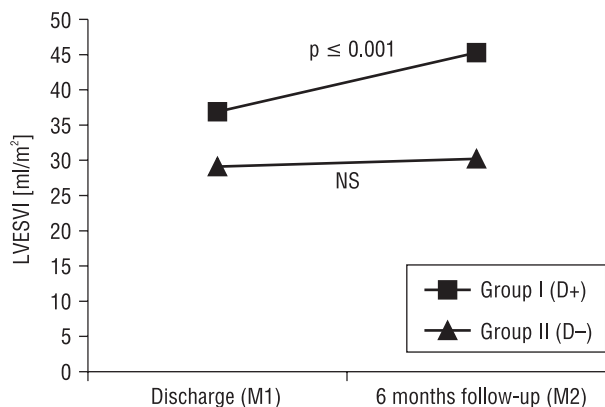


Figure 2. Changes in left ventricular end-systolic volume index (LVESVI) in group I (D+) and group II (D-); (D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation.

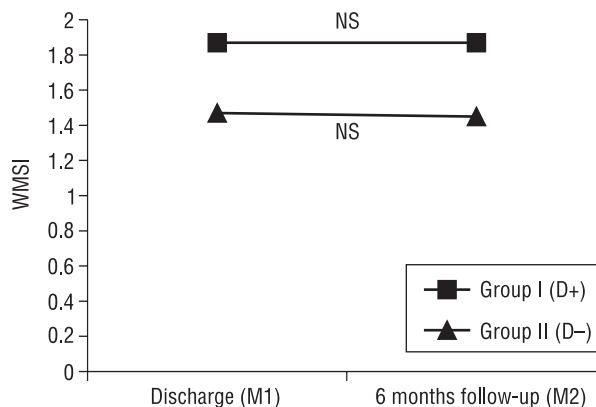


Figure 4. Changes in wall motion score index (WMSI) in group I (D+) and group II (D-); (D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation.

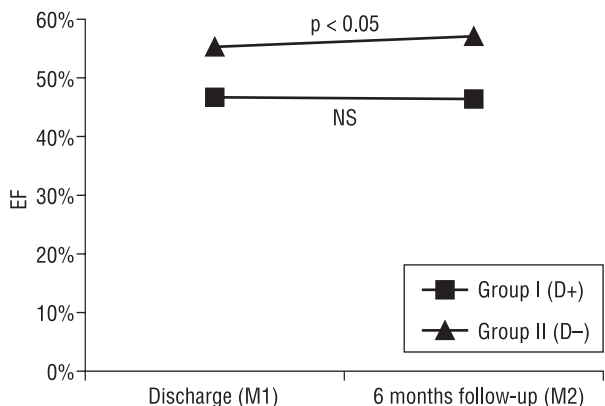


Figure 3. Changes in ejection fraction (EF) in group I (D+) and group II (D-); (D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation.

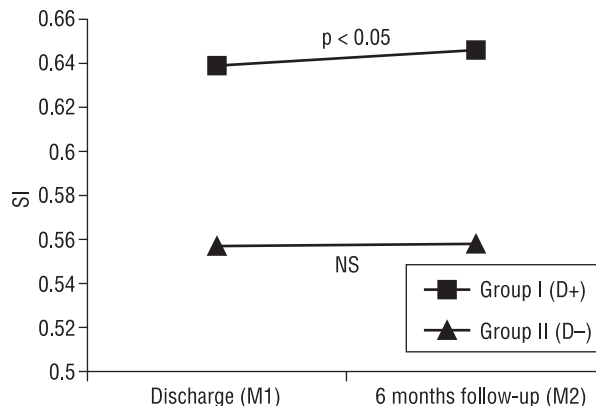


Figure 5. Changes in left ventricular sphericity index (SI) in group I (D+) and group II (D-); (D+) — patients with progressive left ventricular dilatation; (D-) — patients without progressive left ventricular dilatation.

Table 4. Baseline (M1) predictors of progressive left ventricular dilatation.

	Univariate regression analysis		p
	Relative risk	95% confidence interval	
Infarct related artery (LAD)	2.3	1.16–4.16	< 0.05
Wall motion score index ≥ 1.5	4.29	1.77–10.4	< 0.005
Ejection fraction ≤ 45%	2.89	1.62–5.18	< 0.005
Restrictive pattern of mitral flow	2.4	1.20–4.83	< 0.001

LAD — left anterior descending

The univariate regression analysis revealed that patients with LAD as the IRA, a low EF ≤ 45%, with a large infarct size expressed as WMSI ≥ 1.5 and

a restrictive pattern of transmittal flow were identified as at high risk of progressive dilatation. The significant predictors of PLVD are listed in Table 4.

In the present study neither end-diastolic volume nor end-systolic volume was a significant predictor of progressive dilatation. However, multivariate logistic regression analysis after adjustment for LAD as IRA, low EF $\leq 45\%$, WMSI ≥ 1.5 and a restrictive pattern of transmitral flow showed that the only independent determinant of PLVD was WMSI ≥ 1.5 with $rr = 4.20$, $p < 0.005$.

Discussion

LV remodelling following MI is a complex process involving changes in ventricular size, shape, and mass. LV dilatation has routinely been used as a surrogate for remodelling [2, 3]. Dilatation of the LV may play an important role in the development of chronic heart failure [1, 11].

LV remodelling after acute MI is stimulated by the interaction of a number of factors, such as a loss of contractile elements, activation of circulating neurohormones and patency of the IRA, initial MI size and LVH to normalise wall stress [1–3, 11–14].

The benefits of primary PCI over thrombolysis in improving survival have been described [4]. One could expect a low prevalence of remodelling processes following mechanical reperfusion. Our data showed that, despite early mechanical reperfusion, PLVD occurred in 24% of patients successfully treated with primary PCI, which was very close to the 34% observed in previous studies of thrombolysed patients [11].

Gaudron et al. [11] observed that 20% of patients develop progressive structural LV dilatation, which is compensatory at first but is eventually associated with progressive global cardiac dysfunction. The SAVE trial echocardiographic substudy, consisting of 512 patients who survived an acute MI with EF $< 40\%$, showed that LV dilatation occurred in more than one third within two years and was associated with a deterioration in LV function [3].

The GISSI 3 echo substudy showed that late remodelling is associated with progressive deterioration of global ventricular functions and extensive wall motion asynergy rather than significant enlargement of ventricular volume before discharge, constituting a high risk for progressive dilatation [13].

In modern clinical practice the widespread use of acute reperfusion strategies and “anti-remodelling” medications was indicated in the REVE study (Left Ventricular Remodelling After Anterior Wall Acute Myocardial Infarction in modern Clinical Practice) of 220 patients (29% treated with primary PCI, 54% thrombolysis), in which LV remodel-

ling (a 20% increase in LVEDVI) was shown in 31% patients. Peak enzyme, WMSI and systolic blood pressure have been independently associated with LV remodelling [15].

Bolognese et al. [5] showed LV dilatation at 6 months with $> 20\%$ increase in LVEDVI in 30% of a group of 284 patients undergoing primary PCI for acute MI, despite an excellent IRA patency rate. It was interesting that dilatation in general but not a specific pattern of LV dilatation was associated with a poor long term prognosis [5].

In contrast to other studies, we failed to observe the progressive deterioration of global LV functions in patients with LV remodelling. In the present study the EF in group I with (D+) remained unchanged within the 6-month follow-up. The mechanism of deteriorating LV function remains unclear. It has been suggested that systolic and diastolic dysfunctions of the non-infarcted myocardium and regional hypertrophy play a role in the deterioration of LV function [11].

The relative importance and predictive value of identifying patients at high risk for progressive dilatation and heart failure are not known. The early identification of patients at a risk of LV dilatation may have important therapeutic implications.

Our study supports the concept that LV remodelling following MI is a heterogeneous process. In univariate analysis infarct size expressed by WMSI ≥ 1.5 , type of IRA (LAD), EF $\leq 45\%$ and a restricted pattern of LV filling at discharge have been identified as predictors of LV dilatation after MI. In the present study neither LVEDVI nor LVESVI was a significant predictor of progressive dilatation. LVEDVI ≥ 80 ml/m² at discharge did not differ between groups (D+) and (D-), although multivariate logistic stepwise analysis showed the only independent determinant of PLVD to be WMSI ≥ 1.5 .

These findings are consistent with those of the GISSI 3 echo substudy in the thrombolytic area, which showed extensive wall motion asynergy rather than a significantly enlarged ventricular volume before discharge to be a high risk for progressive dilatation [13].

Our data are also in accordance with the Bolognese study, in which independent predictors of progressive dilatation were a relatively low LVEDVI and a high WMSI. These authors revealed that primary PCI progressive dilatation can be expected in patients with a large functional infarct size expressed by a higher WMSI and confirmed the concept that the adaptive compensatory nature of early remodelling (early dilatation) is not necessarily progressive [5].

The importance of LV diastolic dysfunction in the development of LV dilatation and heart failure

has been noted [6, 16]. There are few papers evaluating both systolic and diastolic functions which indicate LV dilatation predictors after MI [6].

Our findings revealed that the restrictive filling pattern appeared to be a significant predictor of LV remodelling in unifactorial analysis but was not independent of other variables in multivariate analysis. Cerisano et al. [6] showed that the restrictive pattern was an independent predictor of LV dilatation in patients treated with primary PCI and that the extent of asynergy and peak creatine kinase (as estimates of infarct size) were significantly higher in patients with a short DT. Doppler indices of LV filling are affected by a number of other physiological factors including heart rate, LV systolic functions and ventricular preload and afterload [17]. It is known that reperfusion therapy of MI causes abnormal LV stiffness and relaxation. Some studies suggest that reperfusion in MI is associated with "diastolic stunning" [18]. Obviously DT is inversely related to the LV filling pressure. LV filling pressure can subsequently influence LV dilatation owing to changes in wall stress [6].

The occurrence of diabetes was similar in the two groups. In the literature diabetes is not shown as an independent predictor of subsequent LV remodelling. Carrabba et al. [19], at 6 months after MI treatment by PCI, found a similar incidence of LV remodelling in the group with diabetes as in that without. In diabetic patients a greater LV chamber stiffness was noted, indicating that LV diastolic dysfunction may play a role in the development of heart failure [19]. In a population of patients in the SAVE study (Survival and Ventricular Enlargement) Solomon et al. [20] showed that in those with diabetes heart failure cannot be explained by an increased propensity for LV remodelling.

It has recently been noted that many factors, such as the advancement of coronary artery disease, recurrent ischemia, coronary microcirculation dysfunction and myocardial viability, can influence LV remodelling in the angioplasty area [21–23]. In contrast to our results, those of the Bolognese group [5] showed that the presence of multivessel coronary artery disease was an independent predictor of progressive dilatation, suggesting the role of progressive ischemia in triggering adverse LV remodelling. Recent studies have also confirmed that microvascular dysfunction is important in subsequent changes in LV geometry [21, 22]. Petronio et al. showed that primary PCI associated with abciximab enhanced myocardial reperfusion and reduced 6-month LV remodelling [24].

Our study, as well as others in the area of mechanical reperfusion, highlights remodelling as a heterogeneous process and suggest that factors other than infarct size and IRA patency may play a role in triggering LV remodelling after MI. This problem requires further observation and studies in order to answer the questions raised.

Patients following MI with early mechanical reperfusion and patent IRA, non-significantly dilated LV or a larger extent of wall motion abnormality at discharge are at risk of developing late adverse LV remodelling. These patients should be monitored by serial echocardiographic examinations so that therapy may be introduced to prevent or reverse the remodelling.

Limitation of the study

The main limitation of our study was a lack of knowledge of late IRA patency. We were unable to perform the control coronarography at 6-month follow-up and thus cannot exclude the possibility that recurrent ischemia may have played a role in triggering the remodelling process. The objective of reperfusion therapies should be not only to achieve rapid and sustained epicardial patency but also to restore microvascular flow and myocardial tissue perfusion. We did not evaluate the impact of microvascular damage on LV remodelling.

A further limitation is the relatively short observation period, as the remodelling process may take place over successive years.

Conclusions

Both regional and global left ventricular systolic dysfunction indices and severe left ventricular diastolic abnormalities rather than left ventricular dilatation at discharge are significant predictors of adverse cardiac remodelling in patients with ST elevation myocardial infarction treated by primary percutaneous coronary intervention. However the only independent determinant of progressive left ventricular dilatation is a wall motion score index of ≥ 1.5 expressing the infarct size.

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