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Early Predictors of Late Dilation and Remodeling after Thrombolized Anterior Transmural Myocardial Infarction

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Summary

Background and hypothesis: Dilation of the left ventricle after myocardial infarction is associated with an adverse prognosis. There are no clinical studies on the role viable myocardium in the infarcted area assumes in relation to the development of late ventricular remodeling. The hypothesis of this study was to define the relation between remodeling and the presence of viable but akinetic myocardium in the infarct area and to identify early predictors of left ventricular (LV) dilation at 1 year.

Methods: In all, 92 consecutive patients with myocardial infarction were divided into two groups according to their ventricular volumes. Group I included 57 patients with normal volumes at discharge (9 ± 3 days after acute infarction) and after 12 months or with LV dilation at discharge who had a normalization of their volumes over a 12-month period. Group II included 35 patients who, independent of their initial volumes, developed LV dilation during follow-up. Low-dose dobutamine infusion was utilized at discharge for echocardiographic evaluation of contractile recovery of viable myocardial segments.

Results: At the first control, patients in Group I presented an end-diastolic volume index (EDVI) of 100 ± 7 ml/m^2 which decreased to 68.8 ± 6.5 ml/m^2 12 months later ($p < 0.0001$), and an end-systolic volume index (ESVI) of 47.6 ± 6.7 ml/m^2 at the first control and 30.5 ± 8.8 ml/m^2 after 12 months ($p < 0.001$). Patients in Group II presented a mean EDVI of 116.2 ± 8.1 ml/m^2 at the first control and 138.8 ± 8 ml/m^2 12 months later ($p < 0.001$), and a mean ESVI of 68.8 ± 6.5 ml/m^2 at the first control and 79.5 ± 5.4 after 12 months ($p < 0.01$). Ventricular mass index (VMI) in Group I increased from 106.4 ± 11 to 122.3 ± 15 g/m^2 ($p < 0.01$), while in Group II it decreased from 101.1 ± 10 to 98.7 ± 8 g/m^2 (p = NS). In Group I, mass-to-volume ratio was 1.15 ± 0.1 g/ml at the first control and 1.67 ± 0.1 g/ml 12 months later ($p < 0.001$), while in Group II it declined from 0.88 ± 0.1 to 0.69± 0.1 g/ml ($p < 0.01$). The multivariate analysis revealed that ejection fraction ≤40%, restrictive filling pattern, wall motion score index >2.5 in response to dobutamine infusion, and mass-to-volume ratio ≤1 g/ml, all at discharge, as well as an occluded left anterior descending artery discriminate in favor of late LV dilation and remodeling.

Conclusions: Correct use of noninvasive strategies should result in early identification of postinfarct patients who are at risk of developing LV remodeling.

Key words: myocardial infarction, thrombolytic treatment, echocardiography, left ventricular dilation, left ventricular remodeling, viable myocardium, prognosis

Introduction

Postinfarct remodeling is a dynamic process characterized by the change in regional and global volume, in ventricular shape, and in myocardial mass which occurs after an extensive transmural myocardial infarction usually involving the anterior wall of the left ventricle.[1–4] This must be seen as a continuing process which can occur months or even years after the acute event, involving both the ischemic and the residual myocardium without any modification in filling pressure.[5–7] The most important factor shown to be at the basis of the remodeling process is the acute expansion of the infarct area which means progressive increase in ventricular volume.[8,9] This process may be recognized on two-dimensional (2-D) echocardiogram[10–12] as a distortion of the ventricular silhouette caused by the lengthening of the infarct zone, tethering of the

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adjacent zones, and subsequent hypertrophy of the residual tissue.[2, 10, 13–16]

The identification of viable myocardium has important clinical relevance in this era of thrombolytic therapy.[17] Low-dose dobutamine echocardiography is currently utilized to assess reversible contractile dysfunction (stunning or hibernating myocardium) in the infarct-related artery territory after reperfusion.[18, 19] To our knowledge, there are no clinical studies reported in the literature on the role viable myocardium in the infarcted area assumes in relation to the development of late ventricular remodeling. The aim of this study was to define the relation between remodeling and the presence of viable but akinetic myocardium in the infarct area and to identify early predictors of left ventricular (LV) dilation at 1 year.

Methods

Study Population

In all, 92 consecutive patients with anterior myocardial infarction were studied. The population had a mean age of 69 ± 10 years (range 52–85 years). There were 75 (81.5 %) men and 17 (18.5%) women. The diagnosis of myocardial infarction was based on the presence of at least two of the following criteria: prolonged (>30 min) chest pain, persistent electrocardiographic (ECG) changes of myocardial ischemia and creatine phosphokinase (CPK) elevation with an MB band of >5%. The location of myocardial infarction, according to 12-lead ECG criteria was anterior in all patients who had transmural myocardial infarction with development of new abnormal Q waves in the anterior precordial leads. All patients received intravenous thrombolytic therapy within 6 h from the onset of symptoms (recombinant tissue-type plasminogen activator bolus injection of 15 mg; 50 mg for 1 h and 35 mg during the next h) in addition to standard treatment (morphine, aspirin, heparin, nitrates, beta blockers). Enrollment started in January 1993 and was concluded in September 1994; the follow-up was concluded in August 1995. Informed consent was obtained from all patients prior to entry into the study according to the protocol approved by the Ethics Committee of our university in December 1992.

The exclusion criteria consisted of:

1. Non-Q-wave infarction
2. History of previous myocardial infarction
3. Presence of significant valvular heart disease
4. Absence of sinus rhythm
5. Contraindication to thrombolytic therapy
6. Hospitalization of more than 6 h after the onset of symptoms
7. Contraindication to dobutamine infusion
8. Therapy with inotropic agents such as digoxin, ibopamin, dobutamine, or dopamine
9. Cardiogenic shock or low-output syndrome
10. New York Heart Association (NYHA) functional class III / IV at discharge.

Study Design

All patients were hospitalized for a mean period of 9 ± 3 days. At discharge, patients underwent echocardiographic and Doppler study and coronary angiography at rest and after a low-dose dobutamine infusion. A complete clinical evaluation and echocardiogram with Doppler study were repeated after 12 months.

Comparing volumes by echocardiogram at discharge with those obtained 12 months later, the study population was divided into two groups. Group I included patients with normal volumes at discharge and after 12 months and patients with LV dilation at discharge who had normalization of their volumes over a 12-month period. Group II included patients who, independent of their initial volumes, developed LV dilation during follow-up.

During follow-up, the investigators did not attempt to influence any clinical decision, in particular therapy decision. Patients' drug therapies were reported by their personal physicians. Precise information about patients' cause of death was obtained from medical personnel, hospital records, and, when available, autopsy reports (two cases).

Laboratory Analysis

Blood samples were collected before thrombolysis and after 12, 24, 36, 48, 60, and 72 h. From these samples, serum CPK and glutamic oxaloacetic transaminase (GOT) peak levels as well as the time integral of serum alpha-hydroxybutyrate dehydrogenase (alpha-HBDH), were obtained. The latter is an accepted measure of enzymatic infarct size, especially in the presence of reperfusion or thrombolytic therapy.[20] GOT peak level is an accepted prognostic indicator of acute mortality and morbidity.[21] CPK peak level was used as a noninvasive indicator of reperfusion as proposed by Shell *et al.* (CPK max <13 h from onset of chest pain)[22] and Gore *et al.* (CPK max <11 h from initial use of thrombolytic therapy).[23]

Low-Dose Echocardiographic Dobutamine Infusion and Doppler Study

A complete M-mode, 2-D, and Doppler examination were performed at rest with a commercially available phased-array imaging system, Hewlett Packard Sonos 1000 or Acuson 128XP/10c. A 2.5 MHz transducer was used for 2-D imaging and a duplex scan for pulsed-wave Doppler. The following echo-derived parameters were considered: LV end-diastolic and end-systolic volumes (biplane disk method, modified Simpson's rule),[24, 25] ejection fraction, mass (Reichek regression formula derived from 2-D measurements),[26, 27] mass-to-volume ratio and LV regional wall motion score index (WMSI). Mass-to-volume ratio represents the ratio between LV mass and end-diastolic volume. For the analysis of wall motion we used apical four- and two-chamber parasternal long- and short-axis views. The left ventricle was divided into 16 segments. The site and severity of the regional myocardial dysfunction was graded by the Herman and Gorlin

scoring system: 0 = hyperkinesia; 1 = normal; 2 = mild or moderate hypokinesia; 3 = severe hypokinesia or akinesia; 4 = dyskinesia; 5 = aneurysmal.[28] Left ventricular score index was obtained by dividing the total score by the number of segments that could be visualized.[29] Regional wall motion was also evaluated during the infusion of low-dose dobutamine. The test was performed with infusion of 5, 10 and 15 gamma/kg/min for 5 min each stage.[30] Electrocardiographic tracing was simultaneously recorded with blood pressure. Beta-blocker therapy was suspended prior to the study for at least five half-lives of the drug. All views were recorded during infusion for subsequent real time, slow motion, and frame-by-frame blinded analysis by two experienced readers. Myocardial viability was seen as an improvement in wall motion in myocardial segments judged to be severely hypokinetic, akinetic, or dyskinetic (score from 3/4 to 2/1 or 2 to 1). Disagreements between readers were resolved by mutual consensus after reevaluation of the study.

A Doppler interrogation of the mitral valve inflow was performed at rest in lateral decubitus at 30°. The following parameters were obtained:

1. The isovolumic relaxation time (IVR T ms) sampling between the mitral inflow and the aortic outflow
2. The peak velocity of early rapid filling wave (E, cm/s)
3. The peak velocity of late filling wave caused by atrial contraction (A, cm/s)
4. E to A ratio (E/A)
5. The deceleration time (Dec t, ms) obtained from extrapolating the time of the decay of the protodiastolic velocity to the zero line.

Echocardiographic views, Doppler flow velocity signals, and ECG tracing were recorded on a strip chart and on videotape for possible later evaluation. All echo-Doppler measurements were the mean of three consecutive cardiac cycles. The patients' clinical and hemodynamic data were unknown.

Study of Volumes and Ventricular Filling

To compare the volumes of the two study groups, we used the values normalized for body surface area. The variability (mean absolute difference) between measurements within one observer was measured for end-diastolic volume index (EDVI) (4.5 ml/m^2) and end-systolic volume index (ESVI) (3.5 ml/m^2), and between two observers for EDVI (7.8 ml/m^2) and ESVI (5.5 ml/m^2). We considered "normal" the following values: 70 ± 10 ml/m^2 for EDVI and 34 ± 8 ml/m^2 for ESVI. These values correspond to the mean values of a series of 55 age-matched normal control subjects. Left ventricular dilation was defined as an increase in LV volume and was considered to be significant when LV volume exceeded the mean normal values +2 standard deviation (SD).

The important role of Doppler of the mitral valve in establishing ventricular filling is well known. In 1988, Appleton[31] determined two types of abnormal filling patterns: the first type called "abnormal relaxation" is characterized by an early to late (atrial) peak velocity ratio E/A <1, an isovolumic relaxation time (IVRT) >80 ms, and deceleration time (Dec.t) ≥ 250 ms. The second type of abnormal filling, identified with the so-called "restrictive pattern," is characterized by an IVRT <80 ms, an E/A much greater than 1, and a Dec.t ≤150 ms. It is well known that the most accurate parameter of filling is Dec.t which is quite independent of respiration, heart rate, and sample volume position.[32] For Dec.t measurements, the variability between one and two observers was 12 ms and 15.2 ms, respectively.

Coronary Angiography

Selective coronary angiography was performed using the percutaneous femoral approach.

The infarct-related artery was graded according to the Thrombolysis in Myocardial Infarction Trial (TIMI) classification:[33] grade 0 (no perfusion), grade 1 (penetration without perfusion), grade 2 (partial perfusion), and grade 3 (complete perfusion). As reported by Topol,[34] only TIMI grade 3 was considered to represent true coronary reperfusion. The presence of collateral circulation was also assessed.

Statistical Analysis

Data at discharge and 12 months later were compared using Student's *t*-test for paired data. Continuous variables were compared between groups by the Student's two-tailed unpaired *t*-test. Conditional variables were compared by use of the X^2 test. Multivariate discriminant analysis, according to Wilks, was used to identify clinical, angiographic, echocardiographic, and Doppler features that were discriminant predictors of volume dilation and ventricular remodeling. A p value of < 0.05 was regarded as significant.

Results

Fifty-seven patients (61.9%) were classified in Group I and 35 (38.1%) in Group II. Tables I, II, and III show the intergroup comparison during hospital stay and at discharge (9 ± 3 days after hospital admission).

At the first control, patients in Group I presented an EDVI of 100 ± 7 ml/m^2, which decreased to 68.8 ± 6.5 ml/m^2 12 months later ($p<0.0001$), and an ESVI of 47.6 ± 6.7 ml/m^2 at the first control and 30.5 ± 8.8 ml/m^2 after 12 months ($p< 0.001$).

Patients in Group II presented a mean EDVI of 116.2 ± 8.1 ml/m^2 at the first control and 138.8 ± 8 ml/m^2 12 months later ($p < 0.001$), and a mean ESVI of 68.8 ± 6.5 ml/m^2 at the first control and 79.5 ± 5.4 after 12 months ($p < 0.01$).

Ventricular mass index (VMI) in Group I increased from 106.4 ± 11 to 122.3 ± 15 g/m^2 ($p < 0.01$.), while in Group II it decreased from 101.1 ± 10 to 98.7 ± 8 g/m^2 (p = NS).

Mass-to-volume ratio in Group I was 1.15 ± 0.1 g/ml at the first control and 1.67 ± 0.1 g/ml 12 months later ($p < 0.001$), while in Group II it declined from 0.88 ± 0.1 to 0.69 ± 0.1 g/ml ($p < 0.01$).

TABLE I Clinical characteristics of study groups during hospital stay

Parameter	Group I (n = 57)	Group II (n = 35)	p Value
Age (years)	58.3 ± 8.9	60.6 ± 8.2	NS
Male (%)	82.4 (n = 47)	79.9 (n = 28)	NS
Body surface area (m^2)	1.71 ± 0.2	1.64 ± 0.3	NS
Time delay from onset of chest pain to inital thrombolytic therapy (h)	4.5 ± 0.7	4.7 ± 0.5	NS
CPK peak level (U/l)	1426 ± 427	1522 ± 379	NS
GOT peak level (U/l)	232 ± 30	220 ± 35	NS
Alpha-HBDH time integral (U/l)	1014 ± 201	1172 ± 312	NS
Reperfusion			
Clinical signs (%)[a]	61.4 (n = 35)	62.8 (n = 22)	NS
CPK washout (%)[b]	35 (n = 20)	37.1 (n = 13)	NS

Results are expressed as mean ± standard deviation.
[a] Disappearance of chest pain and resolution of electrocardiographic changes or reperfusion arrhythmias.
[b] Fulfilled criteria of both Shell *et al.* (22) and Gore *et al.* (23)
Abbreviations: CPK = creatine phosphokinase; GOT = glutamic oxaloacetic transaminase; alpha-HBDH = alpha-hydroxybutyrate dehydrogenase, n = number.

TABLE II Clinical characteristics and echo-Doppler data. Intergroup comparison at discharge

Parameter	Group I (n = 57)	Group II (n = 35)	p Value
SBP (mmHg)	125 ± 10	128 ± 8	NS
DBP (mmHg)	78 ± 6	80 ± 8	NS
HR (beats/min)	80 ± 11	77 ± 10	NS
NYHA class	1 ± 0.5	1 ± 0.7	NS
EF (%)	47 ± 9	37.5 ± 13	0.03
EDVI (ml/m^2)	100 ± 27	116.2 ± 8.1	0.005
ESVI (ml/m^2)	47.6 ± 6.7	68.8 ± 6.5	<0.001
MI (g/m^2)	106.4 ± 11	101.1 ± 10	NS
M/V (g/ml)	1.15 ± 0.1	0.88 ± 0.1	<0.01
E/A	0.8 ± 0.2	0.75 ± 0.1	NS
IVRT (ms)	86.1 ± 8	65.8 ± 10	<0.05
Dec.t (ms)	235 ± 15	102 ± 12	<0.0001

Results are expressed as mean ± standard deviation.
Abbreviations: SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate, NYHA = New York Heart Association, EF = ejection fraction, EDVI = end-diastolic volume index, ESVI = end-systolic volume index, MI = mass index, M/V = mass-to-volume ratio, E/A = early to late (atrial) peak velocity ratio, IVRT = isovolumic relaxation time, Dec.t = deceleration time, n = number.

Ejection fraction in Group I was 47 ± 9% at the first echocardiogram and 60.3 ± 10% 12 months later ($p < 0.0001$), while in Group II it changed from 37.5 ± 13 to 30.5 ± 13% ($p < 0.05$). Figures 1 and 2 show a graph presentation of the above values for Groups I and II, respectively.

Mean NYHA functional class at discharge was 1 ± 0.5 in Group I and 1 ± 0.7 in Group II (p = NS). After 12 months, mean NYHA functional class of Group I was 1.2 ± 0.5 and 2.3 ± 0.6 in Group II ($p < 0.01$).

During the follow-up period, the majority of patients were on angiotensin-converting enzyme (ACE) inhibitors: 42.1% (24/57) in Group I and 39.9% (14/35) in Group II, p = NS. Fourteen patients (28%) in Group I and 10 (28.5%) in Group II were taking a beta blocker, p = NS. The remaining patients were not taking these two drugs. The concomitant use of calcium-channel blockers was limited: eight patients (14%) in Group I and five (14.2%) in Group II, p = NS. All patients were taking a long-acting nitrate and aspirin. Twelve patients (21%) of Group I and 7 (19.9%) of Group II had at least one episode of symptomatic ischemia (p = NS); 1 patient of Group

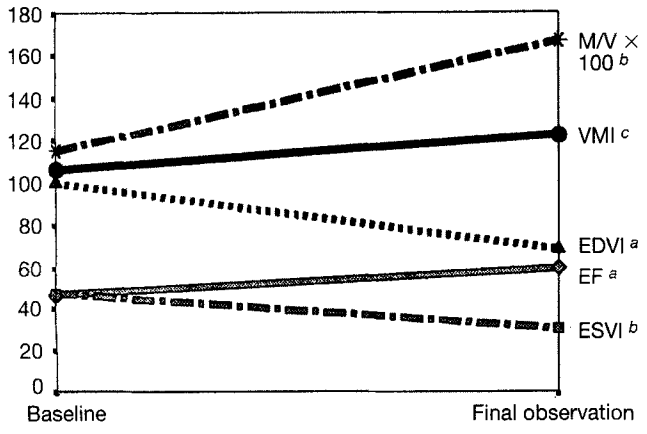

FIG. 1 Group I: Changes in left ventricular end-diastolic volume index (EDVI, ml/m^2), end-systolic volume index (ESVI, ml/m^2), ventricular mass index (VMI, g/m^2), mass-to-volume ratio (M/V, g/m^2), and ejection fraction (EF,%) between discharge (9 ± 3 days after hospital admission—baseline) and 12 months later (final observation). Note the parallel increase of mass-to-volume ratio and ejection fraction. [a] = $p<0.0001$, [b] = $p<0.001$, [c] = $p<0.01$.

TABLE III Risk factor and angiographic data: Comparison between the two study groups

Parameter	Group I (n = 57)	Group II (n = 35)	p Value
Hypertension (%)	31.5 (n = 18)	31.4 (n = 11)	NS
Diabetes (%)	24.5 (n = 14)	25.7 (n = 9)	NS
Cigarette smoking (%)	50.8 (n = 29)	51.4 (n = 18)	NS
Hypercholesterolemia (%)	35 (n = 20)	37.1 (n = 13)	NS
Patency of LADA (%)	61.4 (n = 35)	39.9 (n = 14)	<0.01
SVD (%)	42.1 (n = 24)	39.9 (n = 14)	NS
Collateral circulation (%)	24.5 (n = 14)	25.7 (n = 9)	NS

Abbreviations: LADA = left anterior descending artery, SVD = single vessel disease, NS = not significant, n = number.

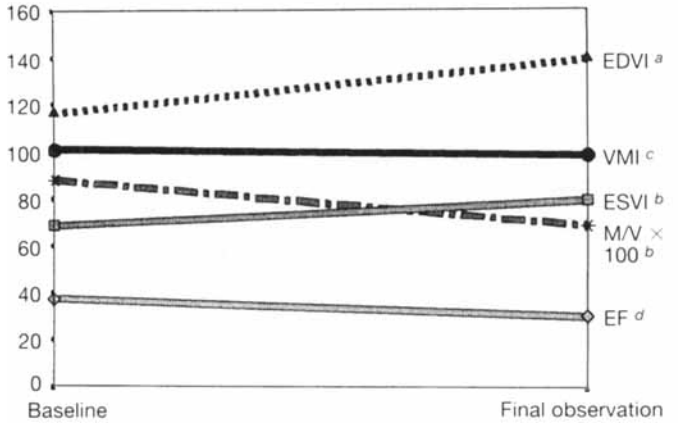

FIG. 2 Group II: Changes in left ventricular end-diastolic volume index (EDVI, ml/m^2), end-systolic volume index (ESVI, ml/m^2), ventricular mass index (VMI, g/m^2), mass-to-volume ratio (M/V, g/ml), and ejection fraction (EF,%) between discharge (9 ± 3 days after hospital admission—baseline) and 12 months later (final observation). Decrease in mass-to-volume ratio is accompanied by decrease in ejection fraction. [a] p<0.001, [b] p<0.01, [c] p = NS, [d] p<0.05.

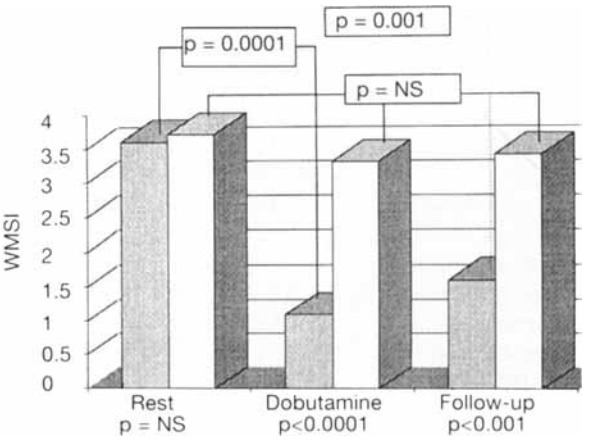

FIG. 3 The bars represent wall motion score index (WMSI) values. WMSI of Group I (shaded bars) does not vary at rest from Group II (unshaded bars). During dobutamine test, WMSI significantly decreases and mildly increases at the last observation, with values significantly lower compared with baseline in Group I. On the other hand, WMSI of Group II does not change from baseline, during dobutamine test, and at final observation.

II had a nonfatal reinfarction, and 10 patients (17.5%) of Group I and 6 (17%) of Group II underwent revascularization (p = NS). Coronary artery bypass graft surgery was done in 10 of 16 patients (62.5%) and percutaneous transluminal angioplasty in the others. Seven patients died, four of sudden death and three following a fatal reinfarction. All patients who died were in Group II.

Results of Low-Dose Dobutamine Infusion

The mean test duration was 22 ± 4 min. None of the patients had adverse effects related to the drug infusion. Pre-dobutamine and post-dobutamine systolic blood pressures in the entire study population were 132 ± 11 and 130 ± 9 mmHg, respectively (p = NS). Diastolic blood pressure changed from 74 ± 8 mmHg pre-dobutamine to 78 ± 4 mmHg post-dobutamine. Pre-dobutamine heart rate was 77 ± 14 beats/min with no significant change recorded after infusion (80 ± 11 beats/min). Of 92 patients, 33 (35.8%) had an increased WMSI in response to the 5 gamma/kg/min dose of dobutamine, 21 (22.8%) to the 10 gamma/kg/min, and only 5 of 92 patients (5.4%) to the 15 gamma/kg/min dose of dobutamine. None of the patients presented a biphasic response (improvement of WMSI at 5 or 10 gamma/kg/min followed by worsening at 10 or 15 gamma/kg/min) during dobutamine infusion. Group I patients presented a basal WMSI of 3.6 ± 0.2. During dobutamine infusion, WMSI improved to 1.1 ± 0.1 (p = 0.0001) in this group. Group II had a basal WMSI of 3.7 ± 0.2 that decreased to 3.3 ± 0.2 (p = NS) during dobutamine infusion. Figure 3 shows the variation of WMSI in the groups at rest, during dobutamine infusion, and at the final observation.

Results of Discriminant Analysis

The multivariate discriminant analysis, according to Wilks, revealed a statistically significant relevance in predicting ventricular volume dilation and the occurrence of worsening of LV function, that is, remodeling, of the following parameters: ejection fraction ≤ 40%, restrictive filling pattern, WMSI in response to dobutamine infusion >2.5, mass-to-volume ratio ≤1 g/ml all at discharge, as well as an occluded left anterior descending artery (TIMI-Flow 0–2) (Table IV).

TABLE IV Results from discriminant analysis

Steps, parameters	Equivalent F	F to remove	Wilks' Lambda	p Value
1. Ejection fraction ≤ 40%	14.615	14.615	0.84553	0.0003
2. Restrictive filling pattern	10.086	4.8526	0.79660	0.0001
3. Dobutamine wall motion score index > 2.5	5.6177	1.3199	0.73015	0.0002
4. Mass/volume ≤ 1 g/ml	6.6645	1.7708	0.74283	0.0001
5. Occluded (TIMI flow 0–2) left anterior descending artery	4.5433	1.1165	0.72991	0.0004

Discussion

Many aspects of remodeling have been established, but some remain unclear. In particular, the role of hypertrophy in determining remodeling appears to be controversial. As was demonstrated by Sonnenblick *et al.*,[35] the volume overload resulting from the loss of myocytes caused by extensive transmural acute infarction leads to hypertrophy, which is proportional to the number of lost cells, involving the remaining noninfarcted myocardial tissue. As reported by Rumberger *et al.*, rather than hypertrophy itself, it is the mutual correlation between ventricular volume and mass that determines remodeling.[36]

Our data clearly showed that patients without ventricular dilation 12 months after the acute event (Group I) developed a greater mass-to-volume ratio compared with those in Group II. The real reason why some patients, independent of risk factors, age, gender, infarct size, and type of therapy develop remodeling, is a dilemma. Nevertheless, the results of low-dose dobutamine infusion would suggest that the presence of viable myocardium in the infarct-related area should be the key to interpret this phenomenon. In fact, Group I patients, who presented an increased wall motion in response to dobutamine stimulation, showed a spontaneous improvement in WMSI, mass-to-volume ratio, and ejection fraction at the last observation. On the other hand, Group II patients did not change WMSI at rest, during dobutamine infusion, and after 12 months. In these patients we observed a progressive and significant increase in ventricular volumes, a significant decrease in mass-to-volume ratio and ejection fraction, a more important clinical deterioration, and a high mortality when compared with Group II patients.

Our results also showed that a low (≤ 40%) ejection fraction at discharge and an occluded anterior descending coronary artery were the most predictive factors for the development of late remodeling. These latter results may not appear original, since there are studies that show a poor prognosis if there is no recanalization and/or highly impaired ventricular function following the acute event.[37,38]

Another important finding in this study is that a restrictive filling pattern at discharge is a very important predictive factor of LV enlargement. The Mayo Clinic group reported a study on LV filling in patients with acute myocardial infarction, in which multivariate analysis showed that Dec.t was the powerful independent predictor of poor prognosis.[39] This can be explained by the fact that a restrictive filling pattern is the result of a reduction in LV compliance following severe ventricular dysfunction.

In conclusion, our study reveals that there are patients with viable but not contractile myocardium in the infarcted zone who maintain an adequate mass-to-volume ratio after a transmural myocardial infarct, while others, without viable myocardium, experience a progressive fall in this ratio and thus develop ventricular remodeling. This leads to a significant negative prognosis with clinical deterioration.

Limitations of the Study

The test we used cannot be considered the standard of reference for viability detection. Positron emission tomography, which is the ideal tool, is not available at our hospital. A low-dose dobutamine test is a low-cost method that is reliable and repeatable.[22,23]

In this study, patients with clinical deterioration soon after myocardial infarction were excluded; our results, therefore, are only applicable to patients without early clinical complication.

Clinical Implication and Conclusion

Our data indicate that left ventricular dilation was common even with the use of thrombolytic therapy in the acute phase of myocardial infarction. Patients at risk for significant left ventricular dilation and remodeling late after the acute event were identified adequately at discharge by the combination of echocardiographic assessment of mass-to-volume ratio, Doppler study of ventricular filling, and demonstration of viable myocardium in the infarct-related area.

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