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Prevalence and predictors of ventricular remodeling after anterior myocardial infarction in the era of modern medical therapy

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
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Summary

Background: The consequences of aggressive therapy following a myocardial infarction (MI) on ventricular remodeling are not well established. Thus, the objective of this study was to analyze the prevalence, clinical characteristics, and predictors of left ventricular remodeling in the era of modern medical therapy.

Material/Methods: Clinical characteristics and echocardiographic data were analyzed in 66 consecutive patients with anterior infarction at admission and at 6-month follow-up. Ventricular remodeling was defined as an increase of 10% in ventricular end-systolic or end-diastolic diameter.

Results: In our study, 58% of patients presented with ventricular remodeling. Patients with remodeling possessed higher total plasma creatine kinase (CPK), MB-fraction (CPK-MB), heart rate, heart failure, shortness of breath, and reperfusion therapy than patients without remodeling. In contrast, patients with remodeling had a smaller ejection fraction, E-Wave deceleration time (EDT), and early (E' Wave) and late (A' Wave) diastolic mitral annulus velocity (average of septal and lateral walls), but a higher E/E' than patients without remodeling. Patients with remodeling used more diuretics, digoxin, oral anticoagulants and aldosterone antagonists than patients without remodeling. In the multivariate analyses, only E' Wave was an independent predictor of ventricular remodeling. Each 1 unit increase in the E' Wave was associated with a 59% increased odds of ventricular remodeling.

Conclusions: In patients with anterior MI, despite contemporary treatment, ventricular remodeling is still a common event. In addition, diastolic function can have an important role as a predictor of remodeling in this scenario.

key words: **predictors; remodeling; ventricular dilation**

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Background

Cardiac remodeling may be defined as changes in the size, geometry, shape, composition, and function of the heart [1–4]. After acute myocardial infarction (MI), this process is clinically characterized by an increase in the ventricular cavity. In the acute phase, ventricular dilation is a result of the infarction expansion process, whereas late cavity dilation is the result of the eccentric hypertrophy process [4,5].

Ventricular remodeling is associated with cardiac rupture, ventricular aneurysm, an increased risk for progressive ventricular dysfunction, and cardiovascular death after MI. Therefore, several variables have been used to predict the remodeling process in the acute phase of MI, such as infarct size, infarct location, previous infarct, wall stress, neurohumoral activation, diabetes mellitus, hypertension, decreased ejection fraction, and signs of heart failure [6–9].

In recent years there have been significant advances in the treatment of patients with MI, in particular in the use of anti-remodeling strategies, including reperfusion therapy, angiotensin converting enzyme inhibitors, and beta-blockers [10]. Therefore, the objective of this study was to analyze the prevalence, clinical characteristics, and predictors of left ventricular remodeling in the era of modern medical therapy.

Material and Methods

All procedures were approved by the ethics committee of our institution, and all participants provided their written consent. From January 2008 to November 2009, consecutive patients with anterior myocardial infarction were prospectively recruited.

Acute MI was diagnosed in the presence of 2 of the following criteria: persistent angina pectoris for ≥20 min and ST-segment elevation of ≥2 mm in ≥2 contiguous precordial leads or the presence of a new or presumably new left bundle branch block. Acute MI was later confirmed by the elevation of cardiac enzymes of more than twice the upper limit of the normal range.

Exclusion criteria were active malignancy, infection, end-stage cardiac, pulmonary or hepatic disease, pregnancy, age <18 years, atrial fibrillation, previous myocardial infarction, and valve disease.

At admission, data on patient characteristics, including waist circumference, body mass index, age, sex, heart rate, cardiovascular risk factors, concomitant diseases, adverse events, medical treatment and data regarding symptoms and pre-hospital delay, were recorded. Our definition of diabetes mellitus was based on clinical features and a fasting glucose level of ≥126 mg/dL on 2 separate occasions or ongoing treatment for the disease. Systemic arterial hypertension was considered to be present if the systolic blood pressure was >140 mm Hg and/or diastolic blood pressure was >90 mm Hg or the patient was already maintained on antihypertensive drug therapy. Dyslipidemia was identified according to the National Cholesterol Education Program (NCEP) III guidelines as total cholesterol levels ≥200 mg/dL, or HDL <40 mg/dL for men and <50 mg/dL for women, or a triglycerides level ≥150 mg/dL. Obesity was defined as a body mass index (BMI) ≥30 kg/m^2.

For the adverse events during the follow-up period, stable angina was diagnosed in the presence of cardiac symptoms in a pattern that remained constant in presentation, frequency, character and duration over time, and coronary disease was diagnosed using coronary angiography. Unstable angina was diagnosed in the presence of new cardiac symptoms and positive electrocardiogram (ECG) findings with normal biomarkers or a changing pattern of symptoms and positive ECG findings with normal biomarkers and coronary disease at coronary angiography. All other prespecified definitions utilized in this study were similar to previous clinical trials [11].

The echocardiogram assessment was completed by the same operator during the index hospitalization (approximately 3–5 days after admission) and at the 6-month follow-up. The echocardiograph was an HDI 5000 Sono CT model (Philips Medical Systems, Bothell, Washington, USA) equipped with a 2.0 to 4.0 MHz probe capable of acquiring second harmonic, tissue, pulsed, continuous, and color Doppler, as well as one- and two-dimensional mode images. With individuals positioned in the left lateral decubitus and monitored with an electrocardiographic lead, the following echocardiographic views were obtained: parasternal short-axis to measure the ventricles, aorta and left atrium and the apical 2-, 4- and 5-chambers to evaluate the cavities and the systolic and diastolic functions of the ventricles. All measurements were performed in accordance with the recommendations of the American Society of Echocardiography/European Association of Echocardiography [12]. The average of 3 measurements was calculated for each variable. In the study group, intraobserver and interobserver variabilities were <3% and <5%, respectively.

The left atrium volume was obtained using the Simpson method from the apical 2- and 4-chamber views. LV systolic function was evaluated by measuring the ejection fraction according to the Simpson method. LV diastolic function was evaluated by measuring the early (E-Wave) and late (A-Wave) diastolic mitral inflow velocity, the E- to A-Wave ratio, the E-Wave deceleration time (EDT), the isovolumic relaxation time (IVRT), the early (E' Wave) and late (A' Wave) diastolic mitral annulus velocity (the average of the septal and lateral walls) using tissue Doppler, and the E/E' ratio. Ventricular remodeling was defined as an increase of 10% in the LV end-systolic or end-diastolic diameter at the 6-month follow-up [13].

The comparisons between the groups were completed with Student's *t* tests when the data presented a normal distribution. For a non-normal distribution, the comparisons between the groups were completed using Mann-Whitney *U* tests. The data were expressed as the mean ± standard deviations or the median with the 25th and 75th percentiles. A chi-squared test was used to compare categorical variables. The predictive values were analyzed using a multivariate logistic regression. Data analysis was completed with SigmaStat for Windows v2.03 (SPSS Inc, Chicago, IL). The significance level was considered to be 5%.

Results

Seventy-six consecutive patients were evaluated. Three patients presented with atrial fibrillation, 1 patient had valve disease and 6 patients died. Thus, 66 patients were analyzed at admission and at the 6-month follow-up.

Table 1. Demographic, clinical and laboratory data.

Variables	Left ventricular remodeling		P value
	Yes (n=38)	No (n=28)	
Age (yrs)	57±11	61±14	0.238
Male (%)	71	82	0.454
HP (%)	60	53	0.754
DM (%)	29	25	0.939
Dyslipidemia (%)	82	89	0.498
Smoking (%)	45	29	0.280
BMI (kg/m^2)	27±4	26±4	0.416
CPK (U/L)	6851 (3963–8734)	1525 (841–4364)	<0.001
CPK-MB (U/L)	512 (318–664)	183 (107–454)	0.002
HR (beats/min)	85±14	72±16	0.001
Heart failure (%)	64	27	0.007
SB (%)	18	0	0.018
Reperfusion (%)	94	75	0.030
TIMI ≥2 (%)	88	96	0.384

HP – hypertension; DM – diabetes mellitus; BMI – body mass index; SB – shortness of breath; CPK – creatine phosphokinase; CPK-MB – creatine phosphokinase – MB; TIMI – Thrombolysis In Myocardial Infarction grade. Data are expressed as the mean ±SD or the median (including the lower and upper quartiles).

Table 2. Medication data.

Variables	Left ventricular remodeling		P value
	Yes (n=38)	No (n=28)	
FT (%)	18	17	0.792
ASA (%)	100	100	1.00
Clopidogrel (%)	100	100	1.00
Heparin (%)	95	93	1.00
ACE i (%)	95	93	1.00
Beta-blockers (%)	97	100	1.00
Nitrates (%)	37	30	0.282
Digoxin (%)	63	36	0.051
Spironolactone (%)	40	14	0.050
Diuretics (%)	66	36	0.030
Statins (%)	100	93	0.176

FT – fibrinolytic therapy; ASA – acetylsalicylic acid; ACE i – angiotensin converting enzyme inhibitor.

In our study, 58% of patients demonstrated ventricular remodeling. The patients were divided in 2 groups using the clinical and echocardiographic data – patients with remodeling and patients without remodeling.

The clinical characteristics are shown in Table 1. Patients with remodeling presented with higher total plasma creatine kinase (CPK) levels, MB-fraction (CPK-MB), heart rate, incidence of heart failure, shortness of breath, and reperfusion therapy. The remaining variables showed no differences between the groups.

The medications utilized during the hospitalization are shown in Table 2. Patients with remodeling used more

Table 3. Initial echocardiographic data.

Variables	Left ventricular remodeling		P value
	Yes (n=38)	No (n=28)	
LA (mm)	41.0 (37–46)	40.0 (38–44)	0.668
LVDD (mm)	48.0 (45–53)	50.0 (49–52)	0.114
LVSD (mm)	32.0 (29–37)	33.0 (31–36)	0.508
E' wave (cm/s)	8.3 (5.5–9.4)	10.5 (9.5–11.7)	0.002
A' wave (cm/s)	11.6 (10.0–13.9)	14.8 (13.8–16.0)	0.001
E/E'	8.5 (5.8–11.2)	6.1 (5.1–6.8)	0.001
E/A	0.79 (0.65–1.00)	0.78 (0.69–0.89)	0.791
IVRT (ms)	111±21	115±16	0.350
EDT (ms)	170±56	238±51	<0.001
EF (%)	37.0 (35–50)	48.0 (43–58)	<0.001

LV – left ventricle; LA – left atrium; LVDD – LV end-diastolic dimension; LVSD – LV systolic dimension; E' wave – early diastolic mitral annulus velocity (average of septal and lateral walls); A' wave – late diastolic mitral annulus velocity (average of septal and lateral walls); IVRT – isovolumetric relaxation time; EDT – E-Wave deceleration time; EF – ejection fraction. Data are expressed as the mean ±SD or the median (including the lower and upper quartiles).

Table 4. Echocardiographic data after 6 months.

Variables	Left ventricular remodeling		P value
	Yes (n=38)	No (n=28)	
LA (mm)	43.0 (40.0–47.0)	40.8 (39.0–42.5)	0.027
LVDD (mm)	52.6 (49.1–57.0)	50.0 (48.0–51.0)	0.007
LVSD (mm)	36.6 (34.1–44.0)	32.0 (30.2–35.7)	<0.001
E' wave (cm/s)	9.0 (8.0–11.0)	11.0 (9.3–11.0)	0.013
EF (%)	45.0 (40–48)	53.0 (49–61)	<0.001

LV – left ventricle; LA – left atrium; LVDD – LV end-diastolic dimension; LVSD – LV systolic dimension; E' wave – early diastolic mitral annulus velocity (average of septal and lateral walls); EF – ejection fraction. Data are expressed as the mean ±SD or the median (including the lower and upper quartiles).

diuretics, digoxin, oral anticoagulants and aldosterone antagonist than patients without remodeling. The remaining variables showed no differences between the groups. After 6 months, the rates of patients using aspirin, angiotensin-converting enzyme inhibitors, and beta-blockers were 95%, 86% and 82%, respectively. Importantly, after 6 months, considering angiotensin-converting enzyme inhibitors, 76% of patients with remodeling and 78% of patients without remodeling continued with medication. Considering beta blockers, 76% of patients with remodeling and 93% of patients without remodeling continued with medication.

The initial echocardiographic data are shown in Table 3. Patients with remodeling presented with smaller ejection fractions, EDTs, E' Waves, and A' Waves than patients without remodeling. In contrast, patients with remodeling presented with higher E/E' ratios than patients without remodeling. The remaining variables showed no differences between the groups. The main echocardiographic data after 6 months are shown in Table 4.

In the multivariate analyses, only the E' Wave was an independent predictor of ventricular remodeling (Table 5). Each 1 unit increase in the E' Wave was associated with a 59% increased odds of ventricular remodeling. In addition, Figure 1 shows the ROC curve for ventricular remodeling with cutoff ⩽9; sensitivity=67.6%; specificity=89.3% (AUC=0.822; 95% CI=0.708-0.906; p=0.0001).

Discussion

The goal of this study was to analyze the prevalence, clinical characteristics, and predictors of left ventricular remodeling after coronary occlusion in the era of modern medical therapy. Despite aggressive treatment, including a high percentage of reperfusion and anti-remodeling strategies, ventricular enlargement is common in patients with anterior MI. E' Wave assessed using tissue Doppler is an independent predictor of remodeling at 6-month follow-up.

Table 5. Multiple logistic regression for ventricular remodeling prediction.

Variables	Odds ratio 95% CI		P value
E' wave (cm/s)	0.629	0.473–0.836	0.001
HR (beats/min)	1.019	0.981–1.058	0.340
CPK/100 (U/L)	1.014	0.992–1.036	0.210
EF (%)	0.035	0.001–12.613	0.265

E' wave – early diastolic mitral annulus velocity (average of septal and lateral walls); HR – heart rate; CPK – creatine phosphokinase, EF – ejection fraction.

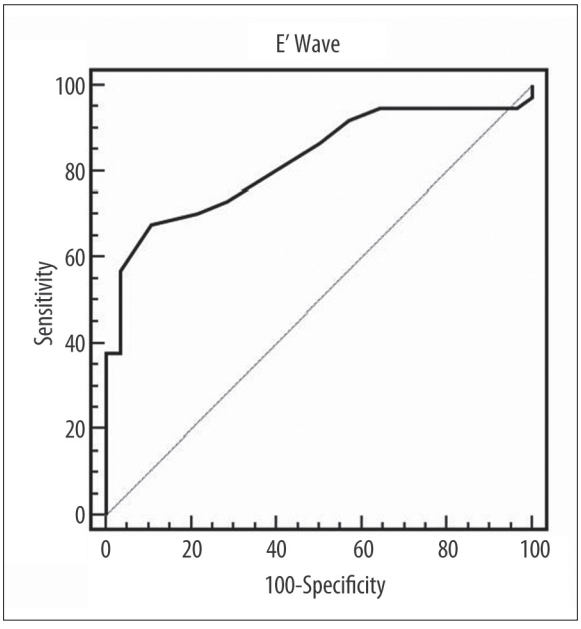

Figure 1. ROC curve for ventricular remodeling. Cutoff ≤9; sensitivity=67.6%; specificity=89.3% (AUC=0.822; 95% CI=0.708–0.906; p=0.0001).

Classically, experimental and clinical evidence suggest that ventricular remodeling is a frequent event after MI. Indeed, studies completed in dogs and rats following coronary occlusion found an expansion of 81% and 65%, respectively [14,15]. Likewise, left ventricular enlargement was present, ranging from 40% to 50%, in patients after MI [6,16–19]. Importantly, another study demonstrated that 61% of patients with anterior myocardial infarction dilated compared with 33% of patients with inferior infarction [6].

Recent improvements in medical therapy and the management of acute MI could impact the incidence and extent of left ventricular remodeling post-MI, including reperfusion therapy [20], angiotensin-converting enzyme inhibitors [21], and beta-blockers [22]. In fact, in the 17 patients with anterior MI, there was a significant increase in left ventricular end-diastolic volume from 2 weeks to 1 month; however, no significant change occurred thereafter [23]. Likewise, progressive left ventricular dilation occurred in 24% of patients after MI in the 86 patients treated with primary percutaneous coronary intervention [24]. In contrast, for patients with anterior MI treated with reperfusion and medications to prevent remodeling, approximately 32% presented with left ventricular dilation after 1 year of follow-up [9]. In 82 patients with MI reperfused within 12 hours of symptoms, 32% of patients developed significant left ventricular dilation, which is defined as a ≥20% increase in left ventricular end-diastolic volume from hospitalization to the 6-month follow-up. In this study, patients with both anterior and inferior infarction were included [25]. In another study of consecutive patients ≥70 years old with MI, the 6-month prevalence of remodeling was 34% [26]. Therefore, the prevalence of ventricular remodeling after anterior myocardial infarction in the era of modern medical therapy is still unclear.

In this study, 58% of patients presented with ventricular remodeling. An important finding was that more than 86% of patients were submitted to reperfusion therapy, and more than 83% of patients presented a thrombolysis in myocardial infarction (TIMI) grade ≥2. In addition, the majority of patients were treated with angiotensin-converting enzyme inhibitors and beta-blockers at hospitalization and after 6 months. Therefore, despite contemporary treatment, our data suggest that ventricular remodeling is still a frequent event, at least in patients with anterior MI. It is important to consider that adequate reperfusion in patients with acute myocardial infarction salvages myocardium and reduces mortality. However, successful restoration of epicardial coronary artery patency does not always lead to adequate reperfusion at the microvascular level (phenomenon of no-reflow). In addition, there is a relationship between no-reflow and ventricular remodeling after MI. In our study, despite more reperfusion, the remodeling group presented bigger infarct size than the group without remodeling. Therefore, we conclude that patients with remodeling might present more no-reflow phenomenon than the group without remodeling.

Another important issue is the remodeling prediction. The importance of identifying patients at risk for progressive dilation is well known. If left ventricular dilation were diagnosed at an early stage, a more aggressive therapeutic approach could be undertaken to potentially improve the prognosis after MI.

Previous studies have established that infarct size, anterior location, coronary patency and some anti-remodeling medications are independent predictors of progressive left ventricular dilation [4–8]. However, the role of systolic function variables as predictors of remodeling is less clear [6,8,9]. Recently, a restrictive pattern of diastolic dysfunction was found to be a predictor of remodeling after MI in some [27] but not all [22] studies. In this study, the ejection fraction was smaller in patients with remodeling compared to patients without remodeling. However, in the multivariate analysis,

ejection fraction did not predict remodeling. In agreement with our results, 20% of patients in the HEART study demonstrated complete recovery of function during the first 2 weeks; this suggests that the predictive value of an early assessment of left ventricular function may be limited [28]. In contrast, infarct size and early diastolic mitral annulus velocity were independent predictors of ventricular remodeling. Therefore, our data suggest that diastolic function may be a stronger predictor of remodeling than is systolic function.

Finally, we should considerer the major limitations of this study. Our study included a small sample size and patients from a single medical center. In addition, we did not study the phenomenon of no-reflow. Despite that, we believe that our study adds important data about ventricular remodeling after anterior myocardial infarction in the era of modern medical therapy.

Conclusions

In conclusion, our data demonstrate that in patients with anterior MI, despite contemporary treatment, ventricular remodeling is still a common event. In addition, diastolic function can have an important role as a predictor of remodeling in this scenario.

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