Nutrition 29 (2013) 122–126

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Applied nutritional investigation

Waist circumference, but not body mass index, is a predictor of ventricular remodeling after anterior myocardial infarction

Ana Lucia Cogni M.D., Ph.D., Elaine Farah M.D., Ph.D., Marcos F. Minicucci M.D., Ph.D., Paula S. Azevedo M.D., Ph.D., Katashi Okoshi M.D., Ph.D., Beatriz B. Matsubara M.D., Ph.D., Silméia G. Zanati M.D., Ph.D., Sergio A.R. Paiva M.D., Ph.D., Leonardo A.M. Zornoff M.D., Ph.D. *

Internal Medicine Department, Botucatu Medical School, São Paulo State University, Botucatu, São Paulo, Brazil

ARTICLE INFO

Article history:
Received 3 August 2011
Accepted 30 April 2012

Keywords:
Obesity
Prognosis
Ventricular dilatation
Coronary occlusion
Ischemia

ABSTRACT

Objective: The impact of obesity on ventricular remodeling after myocardial infarction (MI) is still poorly understood. Therefore, the aim of this study was to evaluate the role of waist circumference (WC) and body mass index as predictors of cardiac remodeling in patients after an anterior MI.
Methods: Eighty-three consecutive patients with anterior MI were prospectively evaluated. Clinical characteristics and echocardiographic data were analyzed at admission and at a 6-mo follow-up. Ventricular remodeling was defined as a 10% increase in left ventricular end-systolic or end-diastolic diameter at the 6-mo follow-up.
Results: In our study, 83 consecutive patients were evaluated (72% men). Ventricular remodeling was present in 31% of the patients (77% men). Patients with remodeling had higher creatine phosphokinase and creatine phosphokinase-MB peak values, a higher resting heart rate, a larger left atrial diameter, and a larger interventricular septum diastolic thickness. In addition, patients with remodeling had lower peak velocity of early ventricular filling deceleration time and ejection fraction. Patients with remodeling presented higher WC values (with remodeling, 99.2 ± 10.4 cm; without remodeling, 93.9 ± 10.8 cm, $P = 0.04$), but there were no differences in the body mass index values. In the logistic regression analysis, WC, adjusted by age, gender, ejection fraction, and creatine phosphokinase levels, was an independent predictor of left ventricular remodeling (odds ratio 1.067, 95% confidence interval 1.001–1.129, $P = 0.02$).
Conclusion: Waist circumference, but not body mass index, is a predictor of ventricular remodeling after an anterior MI. Therefore, the WC of these patients should be measured in clinical practice.

Introduction

Myocardial infarction (MI) has been associated with an increased number of hospitalizations and deaths around the world [1,2]. The prognosis after an MI has been related to several factors, including age, gender, heart rate, cardiogenic shock, infarct size, previous infarct, type 2 diabetes, hypertension, decreased ejection fraction, and signs of heart failure [3–6]. In recent years, another variable studied as a predictor of poor outcome after an MI has been ventricular remodeling [7].

Considering the relation between remodeling and outcomes, in the acute phase after MI, ventricular remodeling has been associated with ventricular rupture, aneurysmal formation, and complex ventricular arrhythmias. Similarly, chronic ventricular remodeling has been shown to play a key role in the pathophysiology of ventricular dysfunction [8,9]. Taking into consideration that the course of remodeling can be modified through several therapeutic interventions, several strategies have been used to predict the remodeling process.

Obesity has been well established as a risk factor for cardiovascular diseases [10]. However, the impact of obesity on ventricular remodeling and on outcomes after an MI is still poorly understood. Obesity has been independently associated with poor outcomes after an MI in some studies [11] but not in others [12–14]. In contrast, different reports have demonstrated a possible protective effect of obesity on outcomes (the "obesity paradox") [15–17].

In addition, taking into account the relation between excess adiposity and the adverse health consequences, several recent studies have demonstrated that anthropometric measurements

This work was supported by the Botucatu Medical School.
* Corresponding author. Tel.: +014-3822-2969; fax: +014-3822-2238.
E-mail address: lzornoff@fmb.unesp.br (L. A. M. Zornoff).

0899-9007
doi:10.1016/j.nut.2012.04.020

related to central obesity are more sensitive predictors of type 2 diabetes [18], cardiovascular diseases [19,20] and left ventricular (LV) diastolic dysfunction [21] than an index of general adiposity, such as the body mass index (BMI).

Importantly, the role of obesity in ventricular remodeling after an MI is unknown. Therefore, the objective of this study was to evaluate the role of waist circumference (WC) and BMI as predictors of cardiac remodeling in patients after an anterior MI.

Material and methods

All procedures were approved by the ethics committee of our institution, and all participants provided their written consent.

Study design

We used the formula by Fisher and Belle [22] for the sample size estimation, with the following variables: a ventricular remodeling prevalence of 30% to 60%, a 95% confidence interval, and a sample error of 10%. The result was 81 patients. Therefore, from December 2008 to July 2010, 94 consecutive patients with an anterior MI were studied. The exclusion criteria were active malignancy; infection; end-stage cardiac, pulmonary, or hepatic disease; pregnancy; an age younger than 18 y; atrial fibrillation; a previous MI; and valve disease. Three patients did not agree to participate, three patients were excluded because of an atrial fibrillation, and five patients died. Therefore, 83 patients were prospectively evaluated.

On admission, patient characteristics, including WC, BMI, age, gender, heart rate, cardiovascular risk factors, concomitant diseases, medical treatment, admission symptoms, and prehospital delay, were recorded. The BMI was calculated as body weight in kilograms divided by height in meters square. WC was measured at the umbilicus level, while the patient was standing, at end expiration [23]. Body height was measured using a stadiometer; body weight was measured using calibrated scales (Filizola, São Paulo, Brazil).

An echocardiographic assessment was completed during the index hospitalization (approximately 3–5 d after admission) and at the 6-mo follow-up. After the echocardiographic analysis, the patients were divided into two groups: those with and those without ventricular remodeling.

Definitions

Acute MI was diagnosed in the presence of the two following criteria: persistent angina pectoris for at least 20 min and an ST-segment elevation of at least 2 mm in at least two contiguous precordial leads or the presence of a left bundle branch block. An acute MI was later confirmed by the increase of cardiac enzymes to more than twice the upper limit of the normal range [24]. The infarct size was determined using the creatine phosphokinase and creatine phosphokinase-MB values.

The definition of diabetes mellitus was based on clinical features and a fasting glucose level of at least 126 mg/dL on two separate occasions or ongoing treatment of the disease. According to the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, systemic hypertension was defined as a systolic blood pressure higher than 140 mmHg and/or a diastolic blood pressure higher than 90 mmHg [25]. Dyslipidemia was identified according to the National Cholesterol Education Program Adult Treatment Panel III guidelines as total cholesterol levels of at least 200 mg/dL, a high-density lipoprotein level lower than 40 mg/dL for men and lower than 50 mg/dL for women, or a triacylglycerol level of at least 150 mg/dL [26].

Ventricular remodeling was defined as a 10% increase in LV end-systolic or end-diastolic diameter at the 6-mo follow-up [27].

Echocardiographic analysis

The echocardiograph was an HDI 5000 Sono model (Philips Medical Systems, Bothell, WA, USA) equipped with a 2.0- to 4.0-MHz probe capable of acquiring second harmonic, tissue, pulsed, continuous, and color Doppler and one- and two-dimensional mode images. With subjects positioned in the left lateral decubitus position and monitored with an electrocardiographic lead, the following echocardiographic views were obtained: the parasternal short-axis view to measure the ventricles, the aorta, and left atrium and the apical two-, four-, and five-chamber views to evaluate the cavities and the systolic and diastolic functions of the ventricles. All measurements were performed in accordance with the American Society of Echocardiography/European Association of Echocardiography [28] recommendations. The average of three measurements was calculated for each variable. Three operators assessed the echocardiograms. However, for each patient, the same operator assessed the echocardiogram at baseline and after 6 mo. Intraobserver and interobserver variabilities were lower than 3% and lower than 5%, respectively.

Left ventricular systolic function was evaluated by measuring the ejection fraction according to the Simpson method. LV diastolic function was evaluated by measuring the early and late diastolic mitral inflow velocities, the ratio between the two diastolic mitral inflow velocities, the early diastolic mitral inflow velocity deceleration time, and the isovolumic relaxation time.

Statistical analysis

The data are expressed as mean ± standard deviation or median (lower and upper quartiles). Statistical comparisons for continuous variables between the groups were performed with Student's t test for parameters with a normal distribution, which was assessed by the Kolmogorov–Smirnov test. Otherwise, comparisons between the groups were completed with the Mann–Whitney U test. Comparisons between baseline and 6 mo were performed by the paired t test. For categorical data, chi-square tests were performed. To adjust BMI and WC by gender and age, we used analysis of covariance. The logistic regression was used for the remodeling prediction. The calibration of the model was assessed by the Hosmer–Lemeshow statistic. Data analysis was completed with SigmaStat 2.03 for Windows v2.03 (SPSS, Inc., Chicago, IL, USA). $P < 0.05$ was considered statistically significant.

Results

Demographic, clinical, and laboratory data are presented in Table 1. Eighty-three consecutive patients were evaluated; 72% were men. Ventricular remodeling was present in 31% of the patients. A large proportion of patients had systemic hypertension and dyslipidemia (approximately 60% and 77%, respectively) Type 2 diabetes was present in 27% of the patients. There were no differences in these comorbidities between the two groups. Patients with remodeling presented larger WC values than patients without remodeling ($P = 0.04$). In contrast, there was no difference in the BMI between the groups ($P = 0.21$; Table 1). A comparison of the BMI and WC values when separated by male and female subjects showed no differences (Table 2).

The infarct size was larger in the remodeling group (Table 1). Reperfusion therapy was performed in 88% of the patients, and approximately 69% of patients underwent primary angioplasty (a Thrombolysis In Myocardial Infarction flow grade ≥2 was present in 96% of these patients; Table 3). In addition, all patients received acetylsalicylic acid (ASA) and clopidogrel. Patients in the left ventricular remodeling group used more spironolactone ($P = 0.007$) than the group without remodeling (Table 3).

The initial echocardiographic analysis demonstrated a greater interventricular septal diastolic thickness and a larger left atrial diameter in the remodeling group (Table 4). In addition, patients

Table 1
Demographic, clinical, and laboratory data

Variables	Left ventricular remodeling		*P*
	No ($n = 57$)	Yes ($n = 26$)	
Age (y)	59.7 ± 13.0	54.9 ± 9.8	0.10
Men	70 (40)	77 (20)	0.71
SR	56 (32)	69 (18)	0.37
DM	28 (16)	27 (7)	0.88
Dyslipidemia	77 (44)	76 (20)	0.80
Smoking	40 (23)	46 (12)	0.80
BMI (kg/m^2)	26.7 ± 4.52	28.0 ± 3.68	0.21
WC (cm)	93.9 ± 10.8	99.2 ± 10.4	0.04
CK (U/L)	1566 (1017–4623)	6168 (3593–8805)	<0.001
CK-MB (U/L)	199.0 (135.5–495.3)	514.5 (300.0–721.0)	0.002

BMI, body mass index; CK, creatine phosphokinase; CK-MB, creatine phosphokinase-MB; DM, diabetes mellitus; SR, systemic hypertension; WC, waist circumference
Data are expressed as mean ± SD, percentage (number), or median (lower and upper quartiles).

Table 2
BMI and WC separated by gender

Variables	Left ventricular remodeling		P
	No	Yes	
WC in men	94.1 ± 10.8 (40)	100.2 ± 10.0 (20)	0.039
WC women	93.7 ± 10.9 (17)	96.0 ± 12.2 (6)	0.670
BMI in men	26.6 ± 4.5 (40)	27.8 ± 3.5 (20)	0.301
BMI in women	27.0 ± 4.6 (17)	28.8 ± 4.6 (6)	0.419

BMI, body mass index; WC, waist circumference
Data are expressed as mean ± SD (number).

in the remodeling group had exhibited, on their first echocardiogram, a higher heart rate and worse diastolic and systolic functions compared with the no-remodeling group. The echocardiographic data at baseline and after the 6-mo follow-up are listed in Table 5. The MI induced increased left atrial and LV volumes and isovolumic relaxation time. In contrast, the ejection fraction increased during the 6-mo period.

An analysis of the mean BMI value demonstrated that the patients in the two groups were overweight; however, there was no difference in the BMI values between the groups. WC was larger in patients with LV remodeling ($P = 0.04$; Table 1). In the multiple logistic regression, when adjusted for age, gender, ejection fraction, and creatine phosphokinase-MB levels, WC remained an independent predictor of LV remodeling (Table 6).

Discussion

This study aimed to evaluate the role of WC and BMI as predictors of cardiac remodeling in patients after an anterior MI. Interestingly, only WC, an anthropometric measurement of central obesity, predicted LV remodeling in these patients.

After an MI, myocyte necrosis and the resultant abrupt increase in loading conditions trigger a cascade of biochemical intracellular signal processes that modulate reparative changes at the infarction site and in the non-infarcted myocardium [29]. Changes in the ventricular mass, volume, and geometry are characteristic features of ventricular remodeling. Although ventricular remodeling initially can be a compensatory process, ventricular remodeling eventually leads to progressive ventricular dysfunction, heart failure, and sudden death [8,9].

Thus, the identification of factors that influence ventricular remodeling is essential for early treatment. Several factors, such as recurrent myocardial ischemia, infarct size, pre-existing comorbidities, and ejection fraction, influence the appearance of LV remodeling and cardiac dysfunction with or without clinical heart failure after an MI [8,9]. In the present study, most patients were men, and there was a high frequency of systemic hypertension and dyslipidemia, most likely due to the specific population, with an anterior MI. However, gender or pre-existing comorbidities were not predictors of ventricular remodeling.

Considering the prevalence of ventricular remodeling in our study, it is interesting to note that 31% of the patients had remodeling despite the high rates of successful reperfusion therapy and standard care. Moreover, patients in the remodeling group already exhibited higher heart rates and worse diastolic and systolic dysfunctions on their first echocardiogram compared with the no-remodeling group. These early signs of ventricular dysfunction in the remodeling group may explain the greater use of spironolactone in these patients.

In recent years, a substantial body of evidence has demonstrated a harmful effect of excess adiposity on the cardiovascular system. Given the simplicity of the BMI as a measurement, it has

Table 3
Medication data during hospitalization

Variables	Left ventricular remodeling		P
	No ($n = 57$)	Yes ($n = 26$)	
FT	16 (9)	19 (5)	0.76
PA	65 (37)	77 (20)	0.40
ASA	100 (57)	100 (26)	1.00
Clopidogrel	100 (57)	100 (26)	1.00
GPI	47 (27)	65 (17)	0.20
UFH	26 (15)	15 (3)	0.22
Clexane	77 (44)	88 (23)	0.36
ACEI	91 (52)	96 (25)	0.66
ARB	3.5 (2)	3.8 (1)	1.00
β-Blockers	98 (56)	92 (24)	0.23
Nitrates	26 (15)	34 (9)	0.61
Spironolactone	10 (6)	38 (10)	0.007
Statins	96 (55)	100 (26)	1.00

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ASA, acetylsalicylic acid; FT, fibrinolytic therapy; GPI, glycoprotein IIb/IIIa inhibitor; PA, primary angioplasty; UFH, unfractionated heparin
Values are presented as percentage (number).

Table 4
Echocardiographic data (3 to 5 d after admission)

Variables	Left ventricular remodeling		P
	No ($n = 57$)	Yes ($n = 26$)	
LA (mm)	39.0 (38.0–43.0)	44.5 (39.5–47.0)	0.01
LVEDD (mm)	50.0 (48.0–52.0)	48.9 (44.9–55.6)	0.59
LVSD (mm)	34.0 (31.0–37.0)	32.3 (29.2–40.9)	0.39
IVS (mm)	10.0 (9.0–11.1)	11.0 (10.0–13.0)	0.05
LVWT (mm)	10.3 (9.8–11.0)	11.0 (9.9–11.7)	0.15
2 × LVWT/LVEDD	0.41 (0.38–0.47)	0.42 (0.39–0.49)	0.17
E wave (cm/s)	62.4 ± 15.6	65.6 ± 19.4	0.43
A wave (cm/s)	77.7 ± 15.5	74.2 ± 19.5	0.38
E/A wave	0.78 (0.66–0.88)	0.81 (0.63–1.26)	0.57
IVRT (ms)	116 (108–128)	108 (92–125)	0.20
EDT (ms)	238 ± 54.3	178 ± 59.6	<0.001
HR (beats/min)	72.6 ± 11.4	82.7 ± 13.5	0.001
EF (%)	48 (43.3–57.7)	38 (35.0–50.0)	<0.001

A wave, peak velocity of transmitral flow during atrial contraction; EDT, peak velocity of early ventricular filling deceleration time; EF, ejection fraction; E wave, peak velocity of early ventricular filling; HR, heart rate; IVRT, isovolumetric relaxation time; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricular end-diastolic dimension; LVSD, left ventricular systolic dimension; LVWT, left ventricular posterior wall thickness
Data are expressed as mean ± SD or median (lower and upper quartiles).

Table 5
Echocardiographic data at baseline and after 6 mo

Variables	Period		P
	Baseline	6 mo	
LA (mm)	40.0 (36.0–45.0)	41.0 (39.0–45.0)	<0.001
LVEDD (mm)	50.0 (47.0–53.0)	51.3 (48.0–54.9)	<0.001
LVSD (mm)	33.0 (30.0–37.0)	35.3 (31.0–38.9)	0.002
IVS (mm)	10.0 (9.0–12.0)	10.0 (9.0–11.0)	0.100
LVWT (mm)	10.7 (9.4–11.4)	10.0 (9.2–11.0)	0.005
2 × LVWT/LVEDD	0.42 (0.38–0.47)	0.39 (0.35–0.43)	<0.001
E/A	0.79 (0.66–0.91)	0.80 (0.68–1.08)	0.186
IVRT (ms)	116 (100–125)	120 (112–132)	0.008
EDT (ms)	216 ± 59.5	227 ± 65.0	0.062
EF (%)	46 ± 10	48 ± 10	0.034

E/A, peak velocity of early ventricular filling/peak velocity of transmitral flow during atrial contraction; EDT, peak velocity of early ventricular filling deceleration time; EF, ejection fraction; IVRT, isovolumetric relaxation time; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricular end-diastolic dimension; LVSD, left ventricular systolic dimension; LVWT, left ventricular posterior wall thickness
Data are expressed as mean ± SD or median (lower and upper quartiles).

Table 6
Logistic regression analysis for WC and BMI values as predictors of left ventricular remodeling

	OR	95% CI	*P*
WC (cm)	1.048	1.001–1.097	0.04
WC (cm)*	1.067	1.009–1.129	0.02
BMI (kg/m^2)	1.074	0.962–1.200	0.21
BMI (kg/m^2)*	1.100	0.951–1.273	0.20

BMI, body mass index; CI, confidence ratio; OR, odds ratio; WC, waist circumference

* Adjusted for age, gender, ejection fraction, and creatine phosphokinase-MB levels.

been used in clinical studies and has been recommended as a screening in the initial clinical assessment of obesity. Nevertheless, the BMI presents a poor sensitivity to reflect body fatness, given the fact that obesity is defined according to the fat percentage and not to body weight. Conversely, WC has been shown to be a simple and effective way to assess central obesity, with a good correlation with abdominal image and a strong association with cardiovascular risk [23]. However, the exact role of the BMI and WC as predictors of cardiac remodeling after an MI remains to be elucidated.

It is important to note that BMI values were not significantly different between the groups, although the BMI mean values were higher than 25 kg/m^2 in the two groups. Obesity has been known since 1965 to induce cardiac remodeling [30]; however, its influence on ventricular remodeling after an anterior MI had not been previously evaluated. Obesity has been linked to a spectrum of cardiac changes, and the primary feature was thought to be chronic volume overload owing to the high cardiac output and eccentric hypertrophy [31]. However, recent studies have reported that obesity is most often associated with concentric LV remodeling, normal LV ejection fraction, and subclinical abnormalities of systolic and diastolic functions. Moreover, there is little evidence that these abnormalities are progressive [32,33].

Taking into account the relation between excess adiposity and the adverse health consequences, epidemiologic and clinical studies have also demonstrated that obesity is associated with contradictory outcomes after an MI. Despite several pathophysiologic pathways by which obesity increases the risk of coronary artery disease and adverse cardiovascular events, some studies have demonstrated a lack of association between obesity and cardiovascular death [15–17]. This “obesity paradox” may be related to a lower lean body mass associated with lower or normal BMI values, a lack of secondary prevention therapies in patients with normal BMI values, and the inability of BMI values to adequately reflect adiposity [17].

In accordance with these assumptions, recent studies have demonstrated that anthropometric measurements related to central obesity are more sensitive predictors of comorbidities and cardiovascular risk than an index of general adiposity, such as the BMI [18–21]. This study reinforces the differences between WC and BMI values and suggests that, at least for the prediction of LV remodeling after an MI, anthropometric measurements related to central obesity should be used. Even when WC was adjusted for infarct size, systolic function, age, and gender, it remained an independent predictor of LV remodeling after an MI.

Several studies have suggested that visceral, but not subcutaneous fat, is associated with the synthesis of proinflammatory adipokines and increased levels of reactive oxygen species derived from reduced nicotinamide adenine dinucleotide phosphate oxidase activity [34,35]. Interestingly, WC is an anthropometric measurement of central obesity that is related to visceral fat. Thus, inflammation and oxidative stress are potential explanations for the independent relation between WC and ventricular remodeling after an anterior MI. Further studies are required to investigate these hypotheses.

We should considerer the major limitations of this study. Our study included a small sample. Indeed, the odds ratio for the BMI was not significant, probably because of the small sample and low statistical power. In addition, selection bias is a major source of errors and our study included patients from a single medical center. Nevertheless, we believe that our study adds important data about obesity variables and ventricular remodeling after an anterior MI.

In conclusion, our data indicate that WC, but not BMI, is a predictor of ventricular remodeling after an anterior MI. Therefore, in these patients, WC should be measured in clinical practice.

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