Impact of obesity and adipokines on cardiac structure and function in men with first myocardial infarction

Katarzyna Piestrzeniewicz, Katarzyna Łuczak, Marek Maciejewski, Jan H. Goch

1st Department of Cardiology, Medical University of Łódź, Sterling Memorial Hospital, Łódź, Poland

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Corresponding author:
Katarzyna Piestrzeniewicz, MD, PhD
1st Department of Cardiology
Medical University of Łódź
91-425 Łódź, Sterlinga 1/3
Poland
E-mail:
kpiestrzeniewicz@gazeta.pl

Abstract

Introduction: Alterations in cardiovascular function have been observed in obese patients. The aim of the study was to evaluate the impact of obesity and plasma concentration of the adipokines: leptin, adiponectin and resistin on cardiac structure and function.

Material and methods: Out of patients with first acute myocardial infarction (AMI) 40 obese males aged up to 65 years formed group I and 40 lean males group II. Anthropometric measurements, echocardiographic parameters and plasma adipokines concentrations were analyzed.

Results: The anthropometric measurements, leptin and resistin were significantly higher, whereas adiponectin was lower in group I. Most morphological and diastolic function parameters significantly differed between groups. Myocardial performance index was higher in obese patients and correlated with leptin and resistin. Left ventricular hypertrophy (LVH) was observed in 24 patients in group I and 10 patients in group II (60 vs. 25%, $P<0.01$). In univariate regression analysis LVH was related to obesity, hypertension and adipokines but only hypertension and leptin independently increased the probability of LVH. No relation between impaired systolic function (EF<55%) and the analyzed parameters was found. Abnormal left ventricular relaxation was present in 8 patients in group I and 3 patients in group II (20 vs. 7.5%, NS). A relation between abnormal relaxation and hypertension, diabetes and adipokines was revealed, the independent variables being hypertension and leptin.

Conclusions: Obesity is associated with alterations in cardiac structure, diastolic function and LVH. Leptin is related to LVH and abnormal left ventricular relaxation.

Key words: obesity, left ventricular hypertrophy, adipokines, myocardial performance index.

Introduction

Obesity predisposes to atherosclerosis as an element of the metabolic syndrome and a coronary risk factor [1]. It is associated with acute coronary syndromes [2] and is an independent predictor of overall and cardiovascular morbidity and mortality [3]. Surprisingly, in-hospital outcome following percutaneous coronary intervention has been shown not to be affected by slight and moderate obesity [4], and lower risk for in-hospital, 6-month, and 12-month mortality and cardiovascular events has been observed in obese patients with acute myocardial infarction (AMI) [5]. Haemodynamic overload proportional to excess body weight was found in obesity [6]. The increased metabolic demand imposed by the expanded adipose tissue results in a hyperdynamic circulation with increased blood volume and

heart rate. Several studies have shown that obesity is associated with increased left ventricular (LV) mass (LVM) [7-9] and with left ventricular hypertrophy (LVH) [10, 11]. Although LV systolic function is preserved even in severe obesity [12], a subclinical LV diastolic dysfunction has been observed in slightly and moderately obese subjects [7, 13] relatively early in life [14]. This observation is supported by haemodynamic data from invasive studies which showed an association between higher body mass index (BMI) and increased LV end-diastolic pressure, suggesting an association between obesity and diastolic dysfunction [9].

Adipose tissue has recently been recognized as an active endocrine and paracrine system. It secretes adipokines – hormone-like peptides that have an impact on glucose and lipid metabolism, the inflammatory process and other bioactivities [15]. There are only a few data concerning the impact of plasma adipokine concentrations on LVH and diastolic function [16-18].

The aim of the study was to evaluate the impact of obesity and selected adipokines – leptin, adiponectin and resistin – on cardiac structure and function in a group of patients with first AMI treated with primary percutaneous coronary intervention.

Material and methods

Study population

Out of the population of patients with first AMI successfully treated with primary percutaneous coronary intervention (TIMI flow grade 3, residual stenosis <30%), 40 obese males (body mass index BMI ≥30) aged up to 65 years, who admitted being obese for at least 5 years, were selected for the study group (group I), and 40 lean males (BMI <25), adjusted with group I for age and localization of AMI, created a control group (group II). Exclusion criteria were: atrial fibrillation, A-V or bundle branch block, temporary or permanent stimulation, significant valvular heart disease, and severe hypertension.

The study was approved by the Internal Ethics Committee of the Medical University of Łódź, and each patient gave informed consent.

Anthropometric measurements

BMI was calculated as body weight divided by height squared. Waist circumference was measured at the widest diameter between the xiphoid process of the sternum and the iliac crest. Hip circumference was measured at the widest diameter over the greater trochanters. Waist to hip ratio was then calculated.

Blood pressure measurement

Systolic (SBP) and diastolic (DBP) blood pressure were measured twice in the right arm using a standard mercury sphygmomanometer with the patient in the supine position after resting for at least 5 min. SBP and DBP were recorded as the first and fourth Korotkoff phases, respectively. Individual SBP and DBP were calculated as the mean of the two measurements. Hypertension was defined as SBP ≥140 and/or DBP ≥90 mm Hg.

Echocardiographic and Doppler examination

An echocardiographic and Doppler study was performed on the 2nd-3rd day after admission with a Sonos 5500, S3 probe. Harmonic option was used to enhance the visualization of the endocardium. Linear measurements were obtained from the M-mode, parasternal long-axis view [LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD)], LV wall thickness [interventricular septum at diastole (IVSD) and posterior wall at diastole (PWD)], aortic diameter (Ao) and left atrium diameter (LA). Following the instructions of Foppa et al. [19], who have reviewed the most common measures of left ventricular hypertrophy, we have defined endocardial border as the leading edge of each layer and used the American Society of Echocardiography formula modified by Devereux to calculate LVM:LVM = 0.8 × {1.04 × [(LVEDD + PWD + + IVSD)^3 – LVEDD^3]} + 0.6 g. The index applied for adjusting LVM was LVM/h$^{2.7}$ as the most appropriate in obese patients and LVH was identified when LV mass index exceeded 49.2 g/m$^{2.7}$ [20].

LV ejection fraction (EF) was assessed in 4-chamber and 2-chamber apical views with the biplane Simpson's formula to evaluate LV systolic function. Impaired systolic function was diagnosed in case of EF below 55%. Pulse-wave Doppler was used to detect isovolumic relaxation time (IVRT) and the following mitral inflow parameters: early (E) and late (A) transmitral peak flow velocities, E/A ratio, and E wave deceleration time (DT). Mitral inflow was recorded from the 4-chamber apical view at the tips of the mitral valve and the IVRT was assessed from the recordings made from the 5-chamber, apical view, between the left ventricular outflow and inflow. The definition of abnormal LV relaxation was made according to the criteria of the European Society of Cardiology [21] using IVRT (>100 ms between 31 and 50 years, and >105 ms over 50 years), or the combination of E/A ratio (<1 up to 50 years, <0.5 over 50 years) and deceleration time of E velocity (>220 ms up to 50, >280 over 50). Restrictive pattern of mitral inflow was defined as E/A>3.2 and DT<140 ms. Myocardial performance index (MPI), which reflects the ratio of the whole isovolumic to the ejection phase of the cardiac cycle, was calculated by subtracting LV ejection time from the period between the two consecutive mitral inflows and dividing by LV ejection time. LV outflow was recorded with

pulse-wave Doppler just below the aortic valve. Doppler recordings were stored on a magnetic disc. Measurements of E, A, DT and IVRT were made at three consecutive cardiac cycles and mean values were calculated. 2-3 pairs of similar time-length cardiac cycles (differences less than 5 ms) from inflow and outflow recordings were used to evaluate MPI [22].

Laboratory measurements

Fasting blood samples for measurements of glucose, lipid profile and adipokines were taken on the next day after admission and plasma for measurements of adipokines was frozen at –70° until analysis with the quantitative sandwich enzyme immunoassay technique (ELISA) obtained from R&D Systems Inc.

Plasma triglycerides (TG) and total cholesterol (TCH) were measured by enzymatic analytical chemistry. HDL-CH was precipitated using dextran-sulphate and measured enzymatically. The LDL cholesterol (LDL-CH) was calculated using the Friedewald equation: LDL-CH=TCH-(TG/5)-HDL-CH. Hyperlipidaemia was diagnosed in case of hypercholesterolaemia (TCH >200 mg/dl) with LDL-CH (LDL >100 mg/dl) and/or hypertriglyceridaemia (TG >150 mg/dl).

Statistical analysis

Continuous data are expressed as mean ± standard deviation (SD). Where necessary, variables were log-transformed before statistical analysis. Comparisons between the obese group and the normal weight group were analyzed with Student's t test or Mann-Whitney U-test, as appropriate. Categorical variables are presented as numbers and percentages of patients, and comparisons between the two groups were analyzed with the χ^2 test. The relationship between MPI and BMI and adipokines was examined with Pearson's correlation coefficient. To evaluate the independent contribution of univariate risk predictors of LVH, impaired systolic function and abnormal LV relaxation, stepwise multiple logistic regression analyses were used. The variables included in the univariate logistic regression analysis were: age, obesity, hypertension, smoking, hyperlipidaemia, diabetes, blood adiponectin, leptin and resistin concentrations. Only those univariate predictors with $P<0.05$ were subsequently entered into multivariate models. Results are expressed as odds ratio (OR) and confidence interval (CI). A P value <0.05 was considered statistically significant.

Results

Clinical, anthropometric and angiographic characteristics, as well as adipokine concentrations of the obese (group I) and the lean (group II) patients, are shown in Table I. The occurrence of hypertension, diabetes mellitus, smoking and hyperlipidaemia was detected in the same proportion of patients in both groups. The value of SBP was significantly higher in group I than in group II. All the assessed anthropometric measurements, plasma leptin and resistin concentrations were significantly higher in the obese group than in the control group, whereas adiponectin was significantly lower in the obese group.

Echocardiographic parameters in the analyzed groups are presented in Table II. Mean values of LVEDD, IVSD, PWD, EDV, ESV, LVM, LVM/$h^{2.7}$, Ao, LA, transmitral A velocities, IVRT and DT were significantly higher and E/A lower in obese patients.

LVH was observed in 24 patients in group I and in 10 patients in group II (Figure 1). In univariate logistic regression analysis an association between LVH and hypertension, obesity and all analyzed adipokines was revealed. Hypertension 5.5-fold (95% CI 2.05-14.96, P=0.0007) and obesity 4.5-fold (95% CI 1.73-11.69, P=0.002) increased the risk of LVH. Both leptin and resistin as biochemical independent negative factors were associated with higher probability of LVH (OR=1.05, 95% CI 1.03-1.08, P=0.0001, OR=1.12, 95% CI 1.05-1.19, P=0.0002, respectively). Increase in adiponectin by each 1 μg/dl decreased the risk of LVH by 10%. In the final model of multivariate analysis the independent predictors of LVH were hypertension (OR=6.06, 95% CI 1.84-1.92, P=0.003) and leptin (OR=1.06, 95% CI 1.03-1.09, P=0.0001). After adjusting for age and obesity, the probability of LVH was slightly reduced for hypertension and for leptin, whereas resistin appeared to be an additional predictor of LVH (Table IIIA).

There was no significant difference in the mean values of EF between the study groups. Impaired systolic function (EF <55%) was detected in 17 patients in group I and in 18 patients in group II (Figure 1). No relation between impaired systolic function and the analyzed parameters was found (Table IIIB).

In our study, a restrictive pattern of mitral inflow was not observed. Abnormal LV relaxation was present in 8 patients in group I and in 3 patients in group II (Figure 1). In the whole study group the probability of impaired LV relaxation was increased by hypertension (OR=10.90, 95% CI 1.32-89.90, P=0.0264), diabetes (OR=9.22, 95% CI 2.30-36.94, P=0.0017), leptin (OR=1.07, 95% CI 1.03-1.11, P=0.0006) and resistin (OR=1.08, 95% CI 1.02-1.13, P=0.0025) and decreased by adiponectin (OR=0.77, 95% CI 0.60-0.99, P=0.0416). In the final model of multivariate logistic regression analysis hypertension (OR=10.78, 95% CI 1.12-103.80, P=0.0396) and leptin (OR=1.07, 95% CI 1.02-1.12, P=0.0014) were the only independent factors that

Table I. Clinical characteristics of the study groups

Parameters	Obese (n=40)	Lean (n=40)	P
Age [years]	53.65±7.39	54.37±6.62	NS
Time to admission [hours]	4.05±2.55	4.70±3.01	NS
Localization of AMI			
anterior [n (%)]	16 (40%)	17 (42.5%)	NS
inferior [n (%)]	24 (60%)	23 (57.5%)	NS
Waist circumference [cm]	111.95±7.52	88.15±7.10	<0.0001
Waist-to-hip-ratio	1.03±0.05	0.97±0.03	<0.0001
Body mass index [kg/m^2]	32.26±1.97	23.87±1.40	<0.0001
Diabetes mellitus [n(%)]	11 (27.5%)	7 (17.5%)	NS
Hypertension [n(%)]	25 (62.5%)	18 (45%)	NS
Systolic BP [mm Hg]	124.12±9.33	119.00±13.26	<0.05
Diastolic BP [mm Hg]	75.50±6.18	73.13±8.37	NS
Hyperlipidaemia [n (%)]	33 (82.5%)	31 (77.5%)	NS
Current smokers [n (%)]	25 (62.5%)	27 (67.5%)	NS
Leptin [ng/ml]	46.70±18.75	15.57±11.93	<0.0001
Adiponectin [µg/ml]	6.81± 4.32	11.18±7.20	<0.001
Resistin [ng/ml]	27.84±12.15	17.35±11.08	<0.0001

Table II. Echocardiographic parameters in the study groups

Parameters	Obese (n=40)	Lean (n=40)	P
LVEDD [cm]	5.61±0.44	5.29±0.43	<0.01
LVESD [cm]	3.73±0.49	3.54±0.40	NS
IVSD [cm]	1.10±0.15	1.03±0.18	<0.05
PWD [cm]	1.02±0.14	0.94±0.13	<0.05
LVM [g]	240.77±54.40	196.85±41.92	<0.001
LVM/h$^{2.7}$ [g/m$^{2.7}$]	53.71±12.44	45.11±8.58	<0.001
LA [cm]	4.03±0.60	3.61±0.46	<0.001
Ao [cm]	3.85±0.40	3.48±0.34	<0.001
EDV [cm^3]	111.93±26.0	92.48±21.47	<0.001
ESV [cm^3]	47.65±14.72	40.68±13.60	<0.05
EF [%]	57.32±9.21	56.07±9.55	NS
E [cm/s]	70.8±14.43	70.85±13.29	NS
A [cm/s]	76.88±16.84	67.51±12.37	<0.01
E/A	0.97±0.41	1.09±0.32	<0.05
IVRT [ms]	81.45±16.12	67.51±12.37	<0.05
DT [ms]	214.38±37.36	199.52±33.50	<0.05
MPI	0.45±0.05	0.42±0.07	<0.05

increased the risk of abnormal LV relaxation. Adjustment for age and obesity increased the impact of hypertension and leptin on the risk of abnormal LV relaxation (Table IIIC).

MPI – an index of the global LV function – was significantly higher in obese patients (Table I) and it was positively related to leptin (r=0.42, P<0.001) and resistin (r=0.39, P<0.001) (Figure 2).

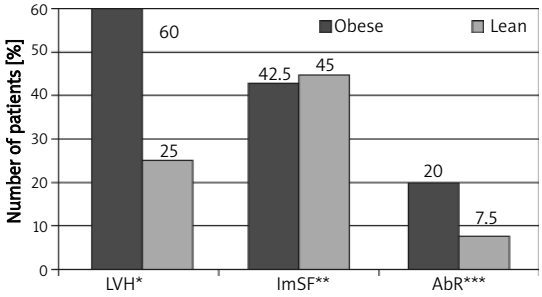

Figure 1. Incidence of left ventricular hypertrophy, impaired systolic function and abnormal relaxation in the study groups
* – Left ventricular hypertrophy
** – Impaired systolic function
*** – Abnormal relaxation

Discussion

Impact of obesity on cardiac structure and left ventricular mass

Our data show that obesity is related to several structural modifications of the heart. We have observed LA and LV remodelling, a known precursor of atrial and ventricular dysfunction. The results of our study are in compliance with several authors who have shown larger LA [8, 23-25] and LVEDD [7, 8, 24] in obese individuals. The intensity of LVH in obesity increases with its severity and duration [5] and is exacerbated by concomitant hypertension, although no synergistic influences of obesity and hypertension were revealed [10]. In our obese patients, who claimed to be obese for at least 5 years, we have similar observations to some other authors who have shown higher thickness of LV walls [7, 9, 10, 25] and higher values of LVM and indexed LVM [7, 8, 25] than non-obese subjects. Moreover, our results are in agreement with previous studies [10, 11] reporting that LVH, which is an important marker of adverse outcome in patients with first AMI [26], is more often observed in obese individuals.

Conversely, Iacobellis et al. [23] did not detect larger LV, thicker LV walls, increase of indexed LVM or signs of LVH in uncomplicated obesity. In the study of Krishnan et al. [8] LV wall thickness did not differ between obese and lean subjects despite high

Table III. Univariate logistic regression analysis and the final model of the multiple logistic regression analysis of the factors associated with left ventricular hypertrophy, impaired systolic function and abnormal relaxation

A. Left ventricular hypertrophy

Univariate logistic regression analysis for left ventricular hypertrophy				
Independent variable[a]	**OR**	**–95% CI**	**+95% CI**	**P**
Age	1.0513	0.9831	1.1243	0.1438
Obesity	4.5000	1.7314	11.6960	0.0020
Hypertension	5.5441	2.0536	14.9677	0.0007
Smoking	0.7816	0.3095	1.9742	0.6023
Hyperlipidaemia	0.6667	0.2410	1.8440	0.4347
Diabetes	2.6646	0.9061	7.8359	0.0749
Adiponectin	0.8974	0.8137	0.9898	0.0304
Leptin	1.0585	1.0310	1.0868	0.0001
Resistin	1.1207	1.0549	1.1907	0.0002
Multivariate logistic regression analysis for LVH				
Hypertension	6.0639	1.8450	19.9294	0.0030
Leptin	1.0600	1.0300	1.0910	0.0001
After adjusted for age and obesity				
Hypertension	5.6199	1.5640	20.1940	0.0082
Leptin	1.0535	1.0088	1.1001	0.0183
Resistin	1.0794	1.0056	1.1585	0.0344

[a] *– dependent variable: left ventricular hypertrophy*

Table III. Univariate logistic regression analysis and the final model of the multiple logistic regression analysis of the factors associated with left ventricular hypertrophy, impaired systolic function and abnormal relaxation – cont.

B. Impaired left ventricular systolic function

Univariate logistic regression analysis for impaired left ventricular systolic function				
Independent variable[a]	**OR**	**–95% CI**	**+95% CI**	**P**
Age	0.9973	0.9358	1.0628	0.9337
Obesity	0.9034	0.3733	2.1861	0.8217
Hypertension	0.8471	0.3492	2.0546	0.7135
Smoking	1.0575	0.4184	2.6727	0.9060
Hyperlipidaemia	1.2273	0.4386	3.4345	0.6965
Diabetes	0.7727	0.2646	2.2562	0.6372
Adiponectin	1.0492	0.9748	1.1293	0.2004
Leptin	1.0130	0.9926	1.0337	0.2131
Resistin	1.0363	0.9974	1.0767	0.0675

[a] *– dependent variable: impaired left ventricular systolic function*

C. Abnormal left ventricular relaxation

Univariate logistic regression analysis for abnormal left ventricular relaxation				
Independent variable[a]	**OR**	**–95% CI**	**+95% CI**	**P**
Age	1.0069	0.9181	1.1043	0.8833
Obesity	3.0833	0.7537	12.6132	0.1172
Hypertension	10.9091	1.3237	89.9088	0.0264
Smoking	1.5152	0.3681	6.2358	0.5649
Hyperlipidaemia	0.8718	0.2075	3.6629	0.8514
Diabetes	9.2273	2.3045	36.9462	0.0017
Adiponectin	0.7755	0.6073	0.9903	0.0416
Leptin	1.0733	1.0305	1.1178	0.0006
Resistin	1.0807	1.0277	1.1364	0.0025
Multivariate logistic regression analysis for abnormal left ventricular relaxation				
Hypertension	10.7841	1.1203	103.8090	0.0396
Leptin	1.0751	1.0285	1.1237	0.0014
After adjusted for age and obesity				
Hypertension	12.2085	1.2014	124.0641	0.0344
Leptin	1.1364	1.0398	1.2421	0.0048

[a] *– dependent variable: abnormal left ventricular relaxation*

prevalence of diabetes and hypertension in obese individuals.

A relation between BMI and LVM was revealed by several authors [27, 28]. In our study BMI correlated with LVH, but we did not confirm the observation that obesity is an independent risk factor of LVH [29].

Impact of obesity on systolic function

We have not found any differences in EF between obese and lean patients. This is in agreement with the majority of other authors, whose studies indicate that ventricular systolic function, as assessed by EF or with load independent measures such as midwall fractional shortening, is usually normal in obesity [7-9, 30]. In a few studies higher EF [23, 24] were described in obesity.

Impact of obesity on diastolic function

Our data demonstrate that in the group of obese patients with first AMI, mean value of IVRT is longer, A velocity is higher and E/A ratio is lower than in the group of lean patients. Previous studies have shown that obesity alters LV filling parameters,

although different patterns of transmitral flow have been described. Most of the authors have observed prolonged IVRT [7, 23, 25], higher A velocities [7, 23, 31] and decreased values of E/A ratio [7, 23] in otherwise healthy obese persons. However, in other studies no significant changes in IVRT [24], higher [23, 25] or normal [7, 24] E velocities, normal A velocities [24, 25], and normal values of E/A ratio [24, 25] were found. In agreement with Iacobellis et al. [23] and Malavazos et al. [25] we have detected prolongation of DT in our obese patients.

LV relaxation is the major determinant of IVRT. It is, however, dependent on the loading conditions. Prolongation of IVRT could be a result of the decrease in left atrial pressure at the onset of mitral inflow, but is unlikely in obese subjects known to have an increased blood volume [6]. The disparities of mitral inflow pattern described in obese patients could be explained by the different study populations – patients with obesity of different duration and severity, sex, presence of other deleterious conditions such as hypertension, diabetes, dyslipidaemia, coronary artery disease, as well as the different size of the study groups. Moreover, transmitral Doppler flow pattern does not exclusively depend on LV diastolic properties but also on the net atrioventricular compliance, mitral valve inertance, atrial systolic function, and atrial pressure; it is sensitive to loading conditions and change over time according to the degree of diastolic dysfunction [32].

Impact of obesity on global left ventricular function – myocardial performance index

It has been shown that two-dimensional imaging measurements are less accurate in obese than in lean subjects [33]. The Doppler derived myocardial performance index (MPI), which provides an insight into systolic and diastolic ventricular function, is an easily obtainable, non-geometric Doppler parameter [22] and seems to be an alternative to established measurements of LV function in patients with obesity [34, 35]. However, normal values of MPI have not been clearly established and depend on the method used to measure the cardiac timing intervals [36, 37]. In agreement with Malavazos et al. [34] and Levent et al. [35] we have revealed significantly higher values of MPI in obese than in lean patients.

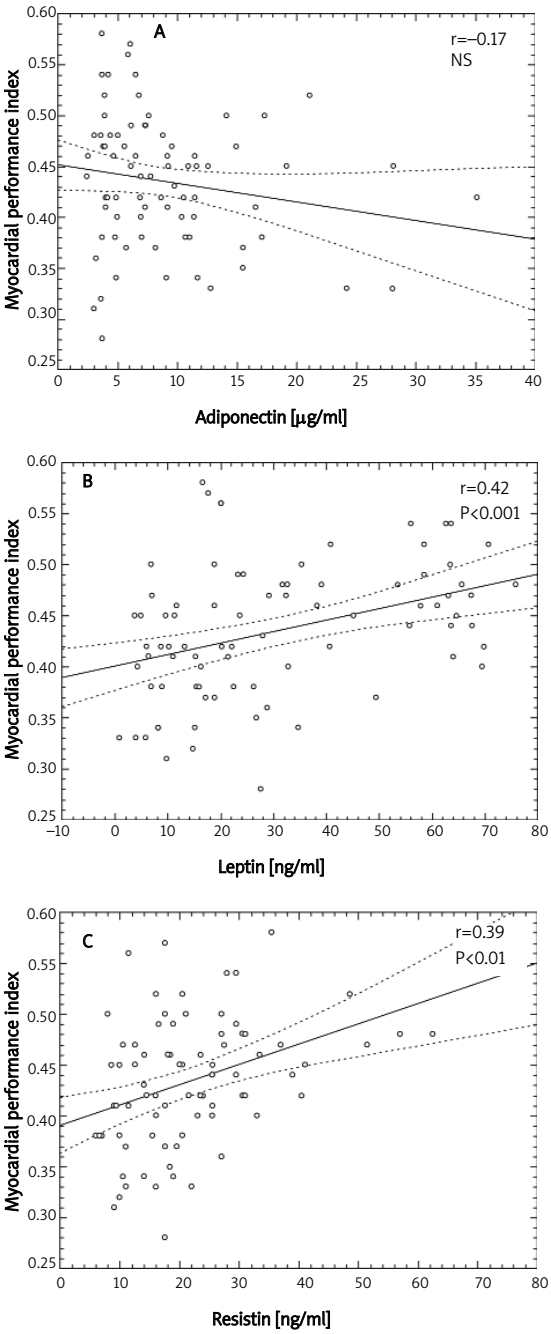

Figure 2. Correlation between myocardial performance index and adipokines

Relation of plasma adipokine concentrations to cardiac structure and function

Adiponectin, resistin and leptin are molecules involved in the induction of endothelial dysfunction, mostly by modulation of the inflammatory reaction, and have a potential role in insulin resistance, early atherogenesis and its late complications [38, 39]. An interesting finding of our study is the relation between adiponectin, resistin and leptin to LVH and abnormal LV relaxation; however, leptin was the only adipokine that apart from hypertension was a weak but independent predictor of these conditions. The power of leptin to predict abnormal LV relaxation was stronger after adjustment for obesity, suggesting that its deleterious effect is more significant in obese subjects. The impact of resistin on LVH and diastolic dysfunction has not previously been documented. A negative correlation between the "protective" adipokine adiponectin and LVM, IVRT and E/A was revealed by Hong et al. [16]. Adiponectin was inversely

and independently associated with LVH diagnosed on electrocardiography [40]. The association of leptin with left ventricular mass and hypertrophy, as well as with diastolic dysfunction, has attracted attention for the last few years [17, 18]. The possible mechanisms of these relations are not well understood. Low grade inflammation that accompanies obesity and its relation to plasma adipokine concentration have been shown to be associated with increased left ventricular mass and abnormalities of diastolic function [25]. Adipokines as the correlates of insulin resistance may be involved in its hypertensive effect and the possible but controversial insulin hypertrophic action [29, 41]. Experimental studies on rodents have demonstrated that adiponectin inhibits hypertrophic signalling in the myocardium, implying that a decrease in the blood adiponectin level could cause cardiac muscle hypertrophy. The major role of leptin secreted by adipose cells is to decrease food intake and increase energy expenditure through its action on the hypothalamus. Barouch et al. [18] have found that leptin deficiency in ob/ob mice leads to left ventricular hypertrophy. Similarly, down-regulation of the leptin receptor sensitivity that protects against excessive leptin increase paradoxically results in the relative lack of leptin signalling in the cell and accounts for its hypertrophy. Therefore, hyperleptinaemia, possibly secondary to leptin resistance, was found to correlate with LVH [42]. A protecting role of leptin against lipid overload has been elucidated and leptin resistance could possibly result in fat accumulation within and around the myocardial wall. Ectopic triglyceride location has been observed in moderately obese individuals and is related to free fatty acid exposure and LVM [43]. Moreover, leptin enhances sympathetic nervous tone, which increases vascular tone and blood pressure, although this action is counter-balanced by its direct and indirect peripheral vasorelaxation action [44].

Study limitations

Serial analysis of adiponectin in the course of AMI performed by Kojima et al. [45] showed that adiponectin significantly declines during the initial 24 hours of AMI but is relatively stable throughout the following 48 hours and then rises until the seventh day, although it does not reach values at admission. It is possible that ischaemia-reperfusion can influence release and kinetics of other adipokines, in a similar manner. However, chronic effects of obesity and adipokines on cardiovascular function could be obscured or distorted by the acute event.

In our study diastolic measurements were limited to isovolumic relaxation time and mitral inflow parameters. As no patients had a restrictive inflow pattern, only stage I diastolic abnormalities were detected. Newer echocardiographic techniques with tissue Doppler and colour M-mode velocity measurements would be helpful in diagnosis of stage II diastolic dysfunction.

At present, as the rates of obesity and its sequelae are rising steadily due to the Western lifestyle, all the aspects of the adverse effects of obesity on the cardiovascular system seem to be an important issue. Although the statistical significance of the obtained results is borderline, still the present study is another vote for the attempts to develop a specific medical approach to obesity as a health problem.

Basic research as well as long-term follow-up clinical studies that would consider the exercise component in a large number of patients are needed to fully elucidate the independent deleterious action of excess body fat apart from the cluster of cardiovascular risk factors carried by obesity.

In conclusion, obesity is associated with alterations in cardiac structure and diastolic function and with left ventricular hypertrophy. Leptin, a biochemical marker of obesity, is related to left ventricular hypertrophy and abnormal left ventricular relaxation.

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