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ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2023;12(1):332-7

Association of left atrial ejection force and obesity: A prospective study of middle-aged adults

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Received 17 February 2023; Accepted 01 March 2023
Available online 01.03.2023 with doi: 10.5455/medscience.2023.02.026

Abstract

Increased hemodynamic load in obese individuals leads to alterations in cardiac geometry and function, including the left atrium (LA). Left atrial ejection force (LAEF) is the force required for late diastolic filling of the left ventricle and has been shown to be associated with age, hypertension, glucose level and weight. Our aim in this study was to investigate the relationship between obesity and LAEF in middle-aged-adult individuals. For this prospective study, a total of 104 individuals, 52 healthy normal weight (BMI < 25) and 52 healthy obese (BMI>30), who were admitted to the cardiology clinic with cardiac complaints were enrolled. Detailed physical examination, anthropometric measurements, glucose levels, lipid levels, interventricular septal thickness (IVSD), left ventricular end-diastolic diameter (LVEDD) on echocardiography, left ventricular posterior wall thickness (LVPWD), left ventricular mass index (LVMI), left ventricular ejection fraction (LVEF), left atrial diameter (LAD), left atrial volume index (LAVI) and left atrial ejection force (LAEF) were calculated. Univariate and multivariate analyses were performed to determine the factors affecting LAEF. Age, male ratio, smoking rate, arterial blood pressure and lipid parameters were similar in both groups. BMI value of the normal weight group was 23.7±1.2 and 38.9±3.7 in the overweight group (p<0.001). The obese group had higher IVSD, LVEED, PWD, LVMI, LAD, LAVI and LAEF compared to the normal weight group (p<0.001 for all values). Pearson correlation analysis showed a significant and moderate correlation between BMI and LAEF (r = 0.582, p<0.001). Regression analysis also showed that BMI was a significant and independent predictor factor for LEAF (p<0.001). Higher BMI is associated with increased LAEF in middle-aged-adult healthy obese individuals.

Keywords: Echocardiography, left atrium, obesity

Introduction

Obesity can lead to various hemodynamic alterations, resulting in morphologic and functional disorders of the heart. These obesity-related disorders can eventually lead to heart failure even without co-morbid conditions such as hypertension and coronary artery disease. Although according to our traditional knowledge, these disorders are attributed to hemodynamic changes caused by obesity, recent studies provide data supporting that this condition may also develop due to neurohormonal and metabolic abnormalities [1]. Left ventricular (LV) diastolic dysfunction is especially common in severely obese individuals [2]. Pulmonary capillary wedge pressure and LV peak diastolic pressure are elevated in class II and class III obese individuals, especially during exercise, and this elevation is attributed to the decrease in

CITATION
Kaplangoray M, Toprak K, Cicek OF, Cekici Y. Association of left atrial ejection force and obesity: A prospective study of middle-aged adults. Med Science. 2023;12(1):332-7.

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DOI: 10.5455/medscience.2023.02.026

LV compliance [3].

The left atrial ejection force (LAEF) is a known indication of both LV diastolic function and the mechanical function of the atrium. LAEF is a force applied during atrial systole to push blood through the mitral valve into the LV. In accordance with Newton's principles, LAEF is determined by multiplying the mass and velocities of the blood entering the left atrium (LA) during atrial systole. In the Strong Heart Study, LAEF was correlated with age, body mass index (BMI), hypertension, serum glucose, creatinine and insulin levels. Moreover, it has been demonstrated that an elevated LAEF is a standalone predictor of cardiovascular events [4-5].

We are unaware of any investigation examining the association between obesity and LAEF in the middle-aged obese population. This investigation was carried out to look into this connection.

Material and Methods

Study Population

For this prospective study, patients admitted to the Cardiology Outpatient Clinic of Health Sciences University Şanlıurfa Mehmet Akif Inan Training and Research Hospital due to cardiac complaints were included. After detailed physical examination and tests, according to the criteria recommended by the World Health Organization [6], 52 obese individuals with a BMI > 30 kg/m2, no other co-morbidities and 52 healthy individuals with a BMI < 25 kg/m2 a total of 104 participants were included. Cardiovascular system examinations were also performed in all patients. Blood pressures of the participants were measured in the sitting position in both arms with a mercury sphygmomanometer with a suitable cuff. Body weights, heights and waist circumferences were calculated. Using the Quetlet index, the BMI was computed by dividing the body weight by the square of the height in meters. Fasting glucose, lipid parameters, thyroid and renal function tests, and hemogram parameters were determined from venous blood samples obtained from the participants after 12 hours of fasting. All subjects underwent transthoracic echocardiographic examination and 12-lead electrocardiography (ECG).

Exclusion Criteria

•Patients with a history of coronary artery disease

•Hypertension

•Diabetes mellitus

•Metabolic syndrome

•Left ventricular wall motion defect, left ventricular ejection fraction <50%

•Valvular heart disease

•Primary cardiomyopathy

•Bundle branch block,

•Atrioventricular conduction abnormality on ECG

•Anemia

•Electrolyte imbalance

•Renal failure

•Pulmonary disease

•Poor echocardiographic appearance

In accordance with the Declaration of Helsinki, the study protocol was approved by the Harran University Faculty of Medicine Ethics Committee (HRU/22.12.22). All participants gave their written consent after being fully informed.

Conventional echocardiographic examination

All patients included in the study underwent 2-dimensional, M-mode, pulsed-wave and color flow Doppler echocardiographic examination by two cardiologists blinded to the study criteria. 2-dimensional and conventional Doppler examination was performed in the parasternal long axis and apical four spaces according to the recommendations of the American Society of Echocardiography (ASE) [7-9]. The mean value of three consecutive cycles was taken for all parameters. Left ventricular end diastolic diameter (LVEDD), interventricular septum (IVS), posterior wall (PW) and LV ejection fraction (LVEF) were calculated by M-mode echocardiography. LV mass was calculated using the Devereux formula. LV mass index (LVMI) was calculated as LV mass divided by body surface area. LA volume was calculated using the two-plane area-length method at the end of systole (maximal left atrium size) in apical four and apical two-chamber imaging, and LA volume index (LAVI) was calculated by dividing this value by body surface area [10]. 2D-mode Pulsed-wave-Doppler recordings were obtained from LV inflow velocity as recommended by ASE guidelines. Early diastolic wave (E), late diastolic wave (A), the ratio of two waves (E/A), isovolumic relaxation time, and deceleration time were calculated.

Calculation of LAEF

According to Newton's principles of motion, the product of mass and acceleration gives force. Following this logic, Manning et al [11] proposed that LAEF is equal to the product of the amount of blood passing through the mitral valve during atrial systole and the acceleration of the blood. They proved that LAEF can be calculated with the formula LAEF= 1/2 × MVA × A2. However, although Newton's law applies to solids, it can lead to miscalculation if used for liquids. It has been shown in many studies that the modified version of this formula, LAEF= 1/3 × MVA × A2, can be used for liquid bodies and gives more accurate results [12]. This formula was also used in this study. In this formula, MVA is the mitral valve area and A is the velocity measured during atrial systole. The mitral annulus diameter (d) was measured manually from the apical four-chamber view and MVA was calculated as $\pi d2/4$ assuming the shape of the annulus to be circular.

DOI: 10.5455/medscience.2023.02.026

Statistical Analysis

The software program Statistical Program for the Social Sciences (SPSS for Windows, version 22.0, IBM Corp., Armonk, NY, U.S., 2016) was used to perform statistical analyses on the research data that had been gathered. Descriptive statistics for continuous variables were presented as mean ± standard deviation and median values in the presence of normally and non-normally distributed data, respectively.

The chi-square or Fischer's exact test was used to compare categorical variables that were reported as percentages. Whether the data was normally distributed was evaluated with the Kolmogorov-Smirnov test. Two groups were compared with the independent-samples t-test for continuous data conforming to the normal distribution. Non-normally distributed data were compared with the Mann-Whitney U test. Pearson's correlation coefficient was used for the correlation analysis between the independent indicators of LAEF. The univariate and multivariate logistic regression analyses were used to identify the independent predictors of LAEF. P≤0.05 was considered statistically significant.

Results

Baseline characteristics, echocardiographic and laboratory results of the study participants are summarized in Table 1.

Table 1. Clinical and laboratory characteristics of the study population

	Controls (n=52)	Obese (n=52)	P
Age (year)	39.2±3.5	39.9±4.1	0.312
Male, n (%)	27 (51.9%)	26 (50 %)	0.844
Smoking, n (%)	14 (26.9 %)	13 (25 %)	0.089
Weight (kg)	67.2 ±10.3	107.1 ±11.6	**<0.001**
Waist circumference (cm)	86.5±5.6	114.2±8.1	**<0.001**
BMI	23.7±1.2	38.9±3.7	**<0.001**
Systolic blood pressure (mmHg)	115.4±11.3	116.3±11.2	0.696
Diastolic blood pressure (mmHg)	77.8±5.2	79.3±4.1	0.110
Glucose (mg/dl)	85.8±10.7	92.5±7.1	**<0.001**
Total cholesterol (mg/dl)	165.4±11.9	169.2±12.9	0.130
LDL-cholesterol (mg/dl)	131.9±17.4	139.6±24.1	0.066
HDL-cholesterol (mg/dl)	50.2±5.7	48.9±7.7	0.348
Triglycerides (mg/dl)	139.8±14.6	144.3±15.9	0.139
TSH (mIU/L)	1.94±0.98	1.87±0.99	0.278
Creatinin (mg/dl)	0.95±0.8	0.88±0.2	0.278
WBC (×103/µL)	7.3±1.8	8.43±0.8	0.216
Hemoglobin(g/dl)	13.6±1.5	14.1±1.6	0.101
Platelet (×103/µL)	274.26±86.3	244.46±65.1	0.091

LDL: low-density lipoprotein, HDL: high-density lipoprotein,BMI:body mass index, WBC: White blood cell count, TSH:tyroid stimulating hormone

The mean age of the study population was 39.6±3.9 years and the male rate was 51%. Participants were divided into two groups according to their BMI, with those with a BMI above 30 defined as obese and those with a BMI below 25 defined as healthy. Weight (107.1±11.6 vs. 67.2±10.3, p<0.001), waist circumference (114.2±8.1 vs. 86.5±5.6, p<0.001), BMI (38.9±3.7 vs. 23.7±1.2, p<0.001) and glucose level (92.5±7.1 vs. 85.8±10.7, p<0.001) were higher in the obese group than in the normal weight group (Table 1). When the groups were compared in terms of echocardiographic parameters, IVSD (1.05±0.04 vs. 0.92±0.12, p<0.001), LVEDD (5.01±0.3 vs. 4.4±0.3, p<0.001), LVMI (89.5±13.8 vs. 77.3±16.8, p<0.001), LAD (4.1±0.2 vs. 3.5± 0.3, p<0.001), LAVI (37.4± 6.9 vs. 20.3± 1.9, p<0.001) and LAEF (7.2±1.5 vs. 5.3±1.4, p<0.001) were found to be higher compared to normal weight individuals (Figure 1).

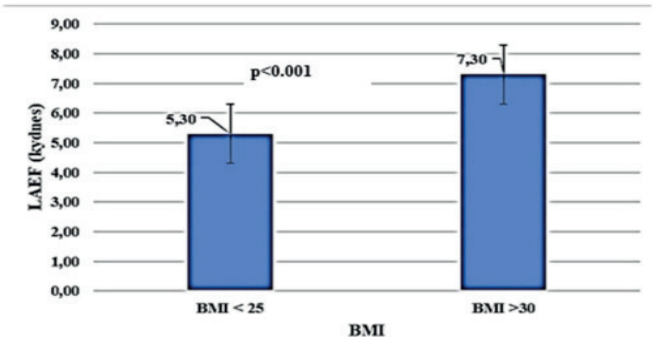

Figure 1. Comparison of LAEF of the groups

While the LVEFs of both groups were similar, peak A velocity was higher in the obese group and peak E velocity was higher in the control group. The E/A ratio was lower in the obese group (0.97±0.2 vs. 1.2±0.3, p<0.001) (Table 2).

Table 2. Echocardiographic parameters of the study population

Echocardiographic parameters	Controls (n=52)	Obese (n=52)	P
IVSD (cm)	0.92±0.12	1.05±0.04	**<0.001**
LVEDD (cm)	4.4±0.3	5.01±0.3	**<0.001**
Posterior wall thickness (cm)	0.98±0.4±	1.07±0.4	**<0.001**
LVMI	77.3±16.8	89.5±13.8	**<0.001**
LVEF (%)	60.1±5.2	62.3±4.5	0.089
LAD (cm) (anterior-posterior)	3.5±0.3	4.1±0.2	**<0.001**
Peak E velocity (cm/s)	70.01±6.4	62.8±7.4	**<0.001**
Peak A velocity (cm/s)	57.4±7.3	66.1±6.9	**<0.001**
E/A ratio	1.2±0.3	0.97±0.2	**<0.001**
Deceleration time (sec)	183.8±14	217.2±22.9	**<0.001**
LAVI	20.3±1.9	37.4±6.9	**<0.001**
LAEF(kdynes)	5.3±1.4	7.2±1.5	**<0.001**

IVSD: interventricular septal thickness, left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction,LAD: left atrium diameter, BMI:body mass index, LAVI:left atrial volume index, LAEF: left atrial ejection force,LVMI: LVMI, left ventricular mass index

DOI: 10.5455/medscience.2023.02.026

The results of the correlation analysis of LAEF with echocardiographic, anthropometric and laboratory results are summarized in Table 3.

Table 3. Pearson's correlation of echocardiographic, anthropometric and laboratory parameters with LAEF

Variables	r value	p value
Waist circumference	0.516	**<0.001**
BMI	0.582	**<0.001**
LAD	0.442	**<0.001**
LAVI	0.511	**<0.001**
IVSD	0.383	**<0.001**
LVEDD	0.498	**<0.001**
Glucose	0.446	**<0.001**
LVMI	0.564	**<0.001**

Abbreviations: see Table 1 and Table 2

According to Pearson's correlation analysis results, a moderately significant correlation was observed between LAEF and BMI ($r = 0.582$, $p<0.001$), waist circumference ($r = 0.516$, $p<0.001$), LAVI ($r = 0.511$, $p<0.001$) and LVMI ($r = 0.564$, $p<0.001$), while was a significant weak correlation observed between LAD ($r = 0.442$, $p<0.001$), IVSD ($r = 0.383$, $p<0.001$), LVEDD ($r = 0.498$, $p<0.001$) and glucose level ($r = 0.446$, $p<0.001$). Logistic regression analysis revealed that waist circumference, BMI, LAD, LAVI, IVSD thickness, glucose level and LVMI were independent and significant parameters determining LAEF (Table 4).[Waist circumference: hazard ratio (HR): 1.182, 95% confidence interval (CI): 1.054-1.327, p<0.004; BMI: HR: 1.388, 95% CI: 1.087-1.772, p=0.009; LAD: HR: 1.327, 95% CI: 1.121-1.435, p<0.028; LAVI: HR: 0.801, 95% CI: 0.671-0.793, p=0.024; IVSD: HR: 1.356, 95% CI: 0.809-1.987, p=0.024; glucose level: HR: 1.197, 95% CI: 1.053-1.362, p=0.006; and LVMI: HR: 1.096, 95% CI: 1.026-1.171, p=0.007 respectively].

Table 4. Univariate and multivariate logistic regression analyses of the predictors of LAEF

	Univariate analysis		Multivariate analysis	
Variables	**OR (95% CI)**	**P-value**	**OR (95% CI)**	**P-value**
Waist circumference	1.143 (1.087-1.201)	**<0.001**	1.182 (1.054-1.327)	**0.004**
BMI	1.232 (1.140-1.331)	**<0.001**	1.388 (1.287-1.772)	**<0.001**
LAD	1.030(1.020-1.040)	**<0.001**	1.327 (1.121-1.435)	**0.028**
LAVI	1.089 (1.040-1.140)	**<0.001**	0.801 (0.671-0.973)	**0.024**
IVSD	11.801 (2.397-15.903)	**<0.016**	1.356 (0.809-1.987)	**0.049**
LVEDD	13.662 (3.652-51.113)	**<0.001**	1.805 (0.175-18.662)	0.620
Glucose	1.108 (1.053-1.166)	**<0.001**	1.197 (1.053-1.362)	**0.006**
LVMI	1.090 (1.050-1.132)	**<0.001**	1.096 (1.026-1.171)	**0.007**

Abbreviations: see Table 1 and Table 2

Discussion

Data from postmortem investigations, echocardiograms, and recently developed cardiac magnetic resonance imaging studies have been the primary sources for information on the impact of obesity on heart morphology [1]. These studies have predominantly enrolled obese individuals with comorbid diseases such as coronary artery disease (CAD) and hypertension, and thus the overlap between these co-morbid conditions and the changes in cardiac morphology caused by obesity itself has been inevitable. In light of the data from experimental studies, the effect of obesity on cardiac morphology on its own has started to be discussed [13]. Obesity causes many functional changes in addition to changes in cardiac morphology. The most common functional change is LV diastolic dysfunction [3]. Previous studies have demonstrated that impairment in transmitral inflow flow occurs with weight gain and in another study, obesity was associated with increased LAEF, cardiac output and increased left ventricular mass index in hypertensive obese individuals [14,15]. In a simple definition, LAEF is the mechanical power expended by the LA to maintain end-diastolic filling of the LV in the setting of impaired LV diastolic dysfunction [12].In this study, we investigated the relationship between obesity and LAEF in healthy obese middle-aged adults and to our clinical knowledge, this is the first study in this aspect. The demographic and anthropometric parameters of normal weight and obese individuals in our study were similar and the individuals in both groups had no other comorbidities other than obesity. Therefore, we think that this study is important in terms of clearly demonstrating how obesity can affect LA functions. Our study revealed that LAEF was higher in obese patients. There are several speculations about this situation. LV diastolic dysfunction is a very common condition in obese individuals [16]. It is only possible to compensate for the increase in LV diastolic filling pressure secondary to LV diastolic dysfunction and to maintain LV ventricular filling by increasing LAEF [17]. LV diastolic dysfunction is also thought to increase the preload of the LA, leading to a Frank-Starlig mechanism in the LA and eventually to an increase in LAEF [18]. In addition, previous studies have also shown that total blood volume and cardiac output increase in association with obesity. Volume increase may lead to an increase in all cardiac cavities including the left atrium and left ventricular cavities [19]. In parallel with this situation, in our study, obese individuals had higher LAD, LAVI, LVEDD and LVMI values compared to normal-weight individuals. Studies on obese patients have shown an increase in LVM including left ventricular hypertrophy [1] and the findings in this study were in this direction.

M. Chinali et al. [20] in a subgroup of overweight adolescents in "The Strong Heart Study" demonstrated that changes in cardiac function and morphology, including LVH, occur in proportion to weight starting from the early period. These changes are thought to be the result of increased hemodynamic load. In the early period of weight gain, LVH and LV diameters increase, whereas

DOI: 10.5455/medscience.2023.02.026

in the later period, LAEF increases secondary to increased LV filling pressure. In our study, LVMI was found to be higher in the obese group although LVH was not at a pathologic level, which is parallel to these findings. In parallel with this, obese individuals were also found to have higher LAEF.

Ayer JG et al. [21] demonstrated that LAD increased in a correlated manner with BMI independent of left ventricular diameter and thickness in their large study. However, the fact that LAEF was not used in this study can be considered as a limitation and the results of the correlation analysis performed in our study revealed the presence of a significant correlation between LAEF and BMI. In addition, according to the logistic regression analysis performed in our study, waist circumference, BMI, LAD, LAVI, IVSD, glucose level and LVMI were found to be independent and significant predictive factors for LAEF. These findings are parallel with those of the Strong Heart Study [22].

Limitations of the study

The single center and small volume of the study is a limitations that can be counted first and foremost. In addition, due to the small number of patients, obese individuals were not restricted according to the degree of obesity and the changes in cardiac morphology according to obesity class could not be investigated. As is known, the measurement of LAV by three-dimensional echocardiography (3DE) provides more objective values than measurement by two-dimensional echocardiography. We think that the absence of 3DE in our clinic and its lack of use in this study is also a deficiency.

Conclusion

This study demonstrated that BMI is an independent and important factor for LAEF in middle-aged adults. This study also demonstrated that weight alone, even in the absence of comorbidities such as hypertension, DM and metabolic syndrome, which are frequently associated with obesity, can cause changes in LAD, LAVI and LVMI, especially LAEF. LAEF may provide additional insights into the pathophysiology of early diastolic dysfunction in obese individuals and the treatment of associated symptoms. LAEF, which is associated with mortality and morbidity and can be calculated noninvasively, is high in middle-aged-adult individuals in the early period of obesity and may be a guiding parameter for early action in the fight against obesity.

Conflict of interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical approval
The Harran University Ethics Committee for Clinical Trials (HRÜ/22.12.22) gave its approval for the project.

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DOI: 10.5455/medscience.2023.02.026

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