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ORIGINAL ARTICLES

The relationship of visceral adiposity with endothelial functions and subclinical atherosclerosis in obese individuals

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Abstract

Introduction: On the cardiovascular system, obesity accelerates atherosclerosis progression, inducing pathophysiological changes that are detectable already from young adults. Endothelial dysfunction is one of the earliest vascular alterations observed in obesity. In this study, we aimed to determine endothelial functions and carotid intima-media thickness in patients with obesity without overt cardiovascular disease.

Methods: The study was conducted with 112 individuals with obesity without overt cardiovascular disease and any chronical diseases (BMI > 30 kg/m^2) (84 female, 28 male, mean age: 46.3±11.2 years) and 49 healthy individuals with no diseases (33 female, 16 male, mean age: 44.6±10.2 years). All patients were examined for endothelial functions by the flow-mediated dilatation (FMD) method and carotid intima-media thicknesses (CIMT). All measurements were performed by the same imaging specialist, averaging 3 different measurements. In addition to the body mass index and waist circumference visceral adiposity index (VAI) and triponderal mass index (TPI) also calculated.

Results: The percentage of FMD obtained by brachial artery ultrasound was significantly lower, visceral adipose tissue, perirenal adipose tissue thicknesses measured by abdominal ultrasound and CIMT were significantly thicker in Group 1 compared to Group 2. FMD had a negative significant correlation with body mass index, visceral adipose tissue thickness, perirenal adipose tissue thickness, and waist and hip circumferences, and carotid intima-media thickness and CIMT had a significant correlation with visceral adipose tissue thickness, perirenal adipose tissue thickness, VAI, TPI and waist, hip circumferences.

Conclusion: Individuals with obesity have impaired endothelial functions and greater carotid intima-media thicknesses compared to healthy individuals. This impairment in endothelial functions is proportional to the amount of visceral and perirenal fat accumulation. Parameterss reflecting visceral fat distribution such as VAI and TPI are also related with these impairment.

Keywords: Obesity, endothelial functions, adiposity, flow-mediated dysfunction, carotid intima-media thickness.

What is new? What is important?

Our study investigates the relationship between visceral adiposity with endothelial functions and subclinical atherosclerosis in obese individuals without overt cardiovascular disease.
Endothelial functions were impaired and carotis intima media thickness were increased in obese individuals and these parameters were related with visceral adiposity. Visceral adiposity index and triponderal mass indices were also related with these impairment. Visceral adiposity may be target parameters for primary prevention in these patients.

INTRODUCTION

Obesity is a disease with high mortality and morbidity due to its close association with various chronic diseases such as diabetes and hypertension, and it constitutes an important public health problem in modern societies [1]. Adiposity is associated with various cardiovascular disease (CVD) risk factors; however, different fat storages indicate varied degrees of CVD risk [2]. Growing evidence in recent years has shown that the relationship between obesity and cardiovascular diseases is not

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only related to the total amount of body fat [3,4] but also related to body fat distribution, and visceral fat accumulation is more important for the development of metabolic syndrome, insulin resistance, and Type 2 Diabetes Mellitus [5,6].

The natural course of atherosclerosis occurs when the disease is diagnosed after a frequent cardiovascular event or at a later stage after a prolonged subclinical phase. Subclinical atherosclerosis (SCA) indicates atherosclerotic stenosis in the coronary, cerebral, renal, and peripheral arteries without significant clinical symptoms [7].

Obesity has an accelerating effect on atherosclerosis in the cardiovascular system, and endothelial dysfunction is the earliest vascular manifestation where endothelial cells detected in obesity transform into pro-atherogenic phenotype [2]. Various studies have been conducted on the relationship between visceral adipose tissue and endothelial functions [8,9]; however, the relationship between the two clinical conditions is not clearly known.

In our study, we aimed to investigate the relationship of visceral adipose tissue thickness with endothelial functions revealed by flow-mediated dilatation and subclinical atherosclerosis demonstrated by carotid intima-media thickness measurement in overweight-obese individuals and without a diagnosed cardiovascular disease and overt diabetes. In addition, to support the study, the relationship of parameters such as visceral adiposity index (VAI) and triponderal index (TPI) with endothelial functions and subclinical atherosclerosis was also examined.

MATERIAL AND METHODS

STUDY POPULATION

Our study is a cross-sectional study and it was conducted with 161 individuals (119 female, 42 male, mean age: 45.8±10.9 years), who presented to the cardiology and internal medicine outpatient clinics with an any complaint and did not have a cardiovascular pathology detected by physical examination tests, and imaging methods. Study was conducted in 6 months period. Detailed anamnesis of all individuals included in the study were obtained, physical examinations were performed, and the medications were recorded. Height (m) and body weights (kg) of the individuals were measured at the time of presentation and BMI was calculated in kg/m². Waist and hip circumferences of the patient and control groups were measured. The waist circumference was measured based on the umbilical point and the hip circumference was measured based on the level of the greater trochanter. Waist/hip and waist/height ratios were calculated. VAI which was based on sex, WC, BMI and lipid parameters, calculated by using the formula proposed by Amato and collaborators: VAI=(WC(cm)/39,68+(1.88*BMI)*(TG/1.03)*(1.31/HDL) for men and VAI = (WC(cm)/36,58+(1.89*BMI)*(TG/0.81)*(1.52/HDL) for women [10]. TPI was calculated as the weight (kg) divided by height cubes in meters (m^3) [11].

Blood pressure and heart rates were measured and recorded. Electrocardiograms were performed for all patients. The suspected patients were examined for coronary artery disease by echocardiography, effort test or, if necessary, myocardial perfusion. All patients were questioned about cardiac complaints and symptoms. Electrocardiograms were taken for all of them, and it was ensured that they did not have cardiovascular disease by using exercise tests and myocardial perfusion scintigraphy when necessary. Patients diagnosed with heart failure or patients with moderate-advanced valvular valve pathology on echocardiography, patients with decreased ejection fraction (EF<50%), patients with diagnosed coronary artery disease, patients with arrhythmia, patients using drugs with proarrhythmic effects, patients with hypertension, patients using drugs that may positively and negatively affect endothelial functions, patients who mentioned claudication and who were found to have peripheral artery disease (including carotid vertebral atherosclerosis) as a result of examinations, patients with Type 1 Diabetes mellitus, patients, who used drugs for insulin resistance such as oral anti-diabetics and patients who did not agree to participate in the study, were excluded from the study. Study groups were formed as group 1 including 112 patients with obesity (body mass index >30 kg/m²) (84 female, 28 male, mean age: 46.3±11.2 years), and group 2 as the control group including 49 individuals with normal weight and no diagnosed disease (33 female, 16 male, mean age: 44.6±10.2 years).

A signed informed consent form was obtained from all participants of the study. The experimental protocols and the process of obtaining informed consent were approved by the review committee of the institution. The study started upon receiving approval from the local Ethics Committee (approval no 2105).

LABORATORY PARAMETERS

Fasting insulin (FI), triglyceride (TG), total cholesterol (TC), low-density cholesterol (HDL-C), high-density cholesterol (LDL-C), and c-reactive protein (CRP) levels, liver enzymes (aspartate aminotransferase, alanine transaminase), and HbA1C levels were measured from venous blood obtained from all participants after minimum 8 hours of fasting and kidney function tests (urea, creatinine) were performed (Roche Cobas 8000-Roche Diagnostics; Switzerland). HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) was calculated according to the relevant formula.

HOMA-IR: (Fasting insulin) X (Fasting glucose)/405 [12]

FLOW-MEDIATED DILATATION (FMD)

Endothelial dysfunction was examined in all patients by ultrasonographic measurement technique (FMD) from the brachial artery in the nondominant arm. Attention was paid to ensure that patients did not exercise, smoke, consume caffeinated or alcoholic beverages, and eat within 12 hours before the procedure. The procedure started by measuring the systolic and diastolic blood pressure of the patients after ten minutes of rest in an environment at twenty-five ºC. Patients were placed in the supine position and palpated in the antecubital fossa in the brachial artery longitudinal plane and the brachial artery was viewed longitudinally in the region where the best image was taken along the trace using a Philips EPIQ 7 (Philips Healthcare, 3000 Minuteman Road, Andover, MA, USA) echocardiography device and a 5.0-1.0 MHz linear transducer. For two-dimensional imaging, a segment was selected between the lumen and the vessel wall where the anterior and posterior intimal faces could be clearly evaluated and the diameter of the brachial artery was measured three times according to ECG monitoring at the end of the diastole and the mean of these three measurements was obtained. This value was recorded as the diameter of the basal brachial artery. After the baseline values were recorded, the cuff pressure was increased to 50 mmHg above the patient's systolic blood pressure to completely interrupt the arterial flow, and the ischemia was developed by keeping the cuff in this position for 5 minutes. 60 seconds after the sleeve was lowered, images of the brachial artery were obtained and the mean of three different measurements was recorded as the diameter of the brachial artery lumen (endothelial-dependent vasodilator response) after flow. FMD was expressed as % (percentage) increase compared to basal vessel diameter (BVD) and was calculated with FMD=[(EBVC-BVD)/BVD] × 100 equation [13].

ULTRASONOGRAPHIC EXAMINATION

Abdominal ultrasound was performed in both groups to measure the thickness of the visceral and perirenal adipose tissue. Ultrasound measurements were performed by the same radiologist using the Toshiba Aplio 300 ultrasonography device to minimize interpersonal measurement errors. Measurement of the perirenal adipose tissue thickness was performed with 3.5 MHz convex probe and visceral adipose tissue thickness was measured with 7.5 MHz linear probe.

Measurements were performed while the patient was in supine position. Perirenal adipose tissue thickness was measured by calculating the mean of 3 measurements obtained from the widest part of the axial plane around the right and left kidneys.

Visceral adipose tissue thickness was measured as the distance between the interior surface of the rectus abdominis muscle and the anterior wall of the aorta at the level of the umbilicus. 3 measurements were made from the thickest point of the visceral adipose tissue and their mean was calculated.

CAROTID INTIMA-MEDIA THICKNESS

Carotid intima-media thickness is defined as the distance between the lumen-intima interfaces of the common carotid arteries and the intima-media. For the measurements, both sides of the neck were scanned and measurements were performed from the posterior walls of the common carotid arteries just proximal to the carotid bulb. 3 separate measurements were performed for each side and their mean was obtained.

STATISTICAL ANALYSIS

Mean, standard deviation, lowest median, highest median, frequency, and ratio values were used for the descriptive statistics of the data. The distribution of the variables was measured by the Kolmogorov Smirnov test. Independent Samples t-Test and Mann-Whitney U Test were used in the analysis of the quantitative independent data. Chi-square test was used in the analysis of the qualitative independent data and Fisher’s exact test was used when the chi-square test requirements

were not met. The Pearson's and Spearman's correlation tests were used for the correlation analyses. SPSS 22.0 software was used to analyse the data obtained. P<0.05 was considered significant.

RESULTS

There was no difference between the groups in terms of age, gender ratio, and smoking habits (Table 1). The family history of heart disease was significantly higher in Group 1 compared to Group 2. Participants with a family history of coronary artery disease were significantly higher in Group 1 compared to Group 2 (Table 1).

Systolic blood pressure and basal heart rates were significantly higher in Group 1 compared to Group 2. Height, weight, waist circumference, hip circumference, and BMI were significantly higher in Group 1 compared to Group 2 (Table 1). VAI and TPI were significantly higher in Group 1 compared to Group 2.

Fasting insulin, HbA1C, triglyceride, LDL-C, ALT, and HOMA-IR levels were significantly higher in Group 1 compared to Group 2 (Table 1). HDL-C levels were significantly lower in Group 1 compared to Group 2 (Table 1). There was no difference between the other laboratory values (Table 1).

The percentage of flow-mediated dilatation obtained by brachial artery ultrasound was significantly lower in Group 1 compared to Group 2 (Table 1). Visceral adipose tissue and perirenal adipose tissue thicknesses measured by abdominal ultrasound were significantly thicker in Group 1 compared to Group 2 (Table 1). Carotid intima-media thickness was significantly greater in Group 1 compared to Group 2 (Table 1).

Table 1

Demographic, laboratory features, endothelial functions, carotis intima media thickness, visceral and perirenal fat thickness of the groups

	Group 1 (n=112)	Group 2 (n=49)	p
Age (years)	46.3±11.2	44.6±10.2	0.367
Gender (F,n)	84	33	0.318
Smoking (n)	38	22	0.187
Hypertension (n)	17	0	<0.001
Diabetes mellitus (n)	28	0	<0.001
Systolic blood pressure (mm Hg)	130.3±18.2	113.9±10.6	<0.001
Diastolic blood presure (mm Hg)	78.5±12.9	68.6±8.7	<0.001
Heart rate (beat/min)	81.9±11.6	77.3±6.5	<0.001
BMI (kg/m^2)	34.6±3.4	22.2±1.7	<0.001
Waist circumference (cm)	106.5±10.8	84.6±7.6	<0.001
Hip circumference (cm)	117.6±8.1	98.3±5.8	<0.001
VAI	2.85±1.96	1.42±0.84	<0.001
TPI (kg/m^3)	21.4±2.3	13.2±1.1	<0.001
Glucose (mg/dL)	110.1±48.9	91.0±8.7	0.007
Total cholesterol (mg/dL)	209.2±44.7	198.3±40.9	0.149
Triglyceride (mg/dL)	160.2±118.9	88.2±50.7	<0.001
LDL cholesterol (mg/dL)	129.0±37.1	125.2±35.1	0.542
HDL cholesterol (mg/dL)	50.2±15.3	55.6±13.6	0.034
Urea (mg/dL)	27.8±8.3	26.4±6.1	0.286
Creatinine (mg/dL)	0.67±0.16	0.73±0.18	0.073
AST (U/L)	22.7±9.2	20.2±7.5	0.010
ALT (U/L)	26.2±16.8	17.4±9.0	0.106
HbA1C (%)	6.16±1.29	5.38±0.29	<0.001
HOMA-IR	2.78±2.10	1.16±0.66	<0.001
Flow mediated dilatation (%)	6.8±3.3	12.1±3.1	<0.001
Visceral fat thickness (mm)	5.68±2.88	2,28±0,86	<0.001
Perirenal fat thickness (mm)	6.16±1.84	2.20±0.95	<0.001
Carotis intima media thickness (mm)	0.184±0.381	0.029±0.005	<0.005

Abbrevations: BMI; body mass index, LDL; low density lipoprotein, HDL; high density lipoprotein, AST; aspartate transaminase, ALT; alanine transaminase, HOMA-IR; homeostasis model assessment-estimated insulin resistance

When the relationship between FMD and various parameters was examined, a negative significant correlation was found with body mass index, visceral adipose tissue thickness, perirenal adipose tissue thickness, waist and hip circumferences, and carotid intima-media thickness (Table 2). Additionally, FMD had a weak correlation with HbA1C and HOMA-IR (r=-0.37; p<0.001, r=-0.31; p<0.001, respectively). When various parameters were examined concerning CIMT, a significant correlation was found with visceral adipose tissue thickness, perirenal adipose tissue thickness, and waist and hip circumferences (Table 3). VAI and TPI were significantly corelated with FMD and IMT (Table 3).

In ROC analysis, area under the ROC curve value of VAI, VFT and PFT for obesity were 0.81 (95% CI: 0.73-0.89, P < .001), 0.93 (95% CI: 0.89-0.97, P < .001) and 0.97 (95% CI: 0.95-1.00, P < .001) respectively (Figure 1). The best cut-off value for VAI, VFT and PFT obtained by the ROC curve analysis were >1.515, >2.99 mm and >3.85 mm for the prediction of obesity respectively (VAI: sensitivity: 78.6%, specificity: 69.0%; VFT: sensitivity: 91.3%, specificity: 81.0%; PFT: sensitivity: 94.2%, specificity: 88.1%) (Figure 1).

Table 2

Spearman correlation analysis between FMD and several parameters

	r	P
Age	-0.23	0.004
Waist circumference	-0.53	<0.001
Hip circumference	-0.45	<0.001
Body mass index	-0.48	<0.001
CIMT	-0.46	<0.001
Visceral fat thickness	-0.47	<0.001
Parerenal fat thickness	-0.51	<0.001
VAI	-0.33	<0.01
TPI	-0.55	<0.001

Abbrevation: CIMT; carotis intima media thickness

Table 3

Spearman correlation analysis between CIMT and several parameters

	r	P
Age	0.36	<0.001
Waist circumference	0.71	<0.001
Hip circumference	0.47	<0.001
Body mass index	0.61	<0.001
Visceral fat thickness	0.62	<0.001
Parerenal fat thickness	-0.59	<0.001
VAI	0.54	<0.001
TPI	0.58	<0.001

Abbrevation: CIMT; carotis intima media thickness

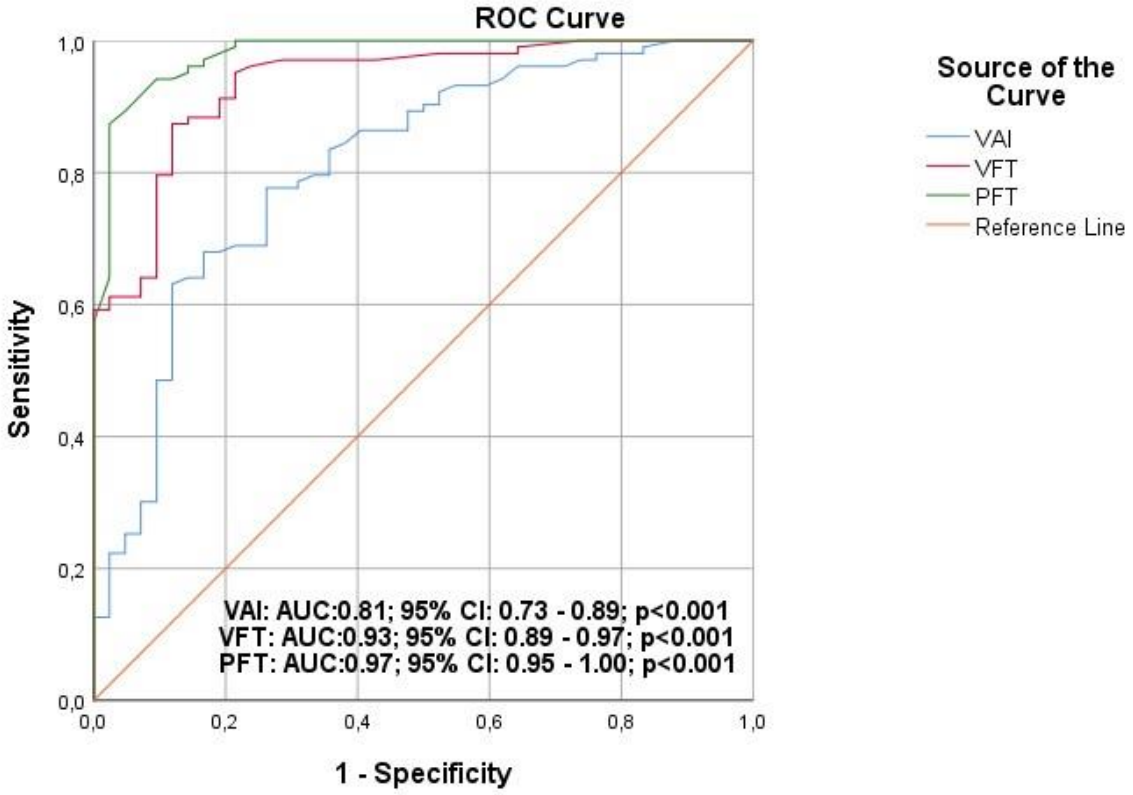


Figure 1. Figure showing receiver operating characteristic (ROC) analysis, area under the ROC curve (AUC) values of VAI, VFT and PFT for obesity.

DISCUSSION

The main result of our study is that endothelial functions were impaired and carotid intima-media thickness was increased in obesity without overt cardiovascular changes compared to healthy individuals. The impairment in endothelial functions was related to the visceral and perirenal adipose tissue obtained by ultrasonography as well as body composition indices. Likewise, visceral and perirenal adipose tissues were associated with carotid intima-media thicknesses.

The vascular endothelium is not only a barrier in the body, but also a key regulator of vascular hemostasis, which can be affected by various environmental and genetic effects. This function is achieved by producing a wide range of factors that regulate vascular tone, cellular adhesion, smooth muscle cell proliferation, resistance to platelets, and vascular wall inflammation in response to physical and chemical signals [2]. Endothelial dysfunction develops as a result of imbalance in the production of vasodilators such as nitric oxide (NO), prostacyclin (PGI2), endothelial-derived hyperpolarizing factors (EDHF) and vasoconstrictor molecules such as endothelin-1 (ET-1), and angiotensin (ATII) [14]. During obesity, this delicate balance is often impaired and it has been suggested that low-grade chronic vascular inflammation is a key event in the pathogenesis of endothelial dysfunction and CVD [15]. Negative changes in the balance predispose the vascular endothelium to prothrombotic and proatherogenic conditions, exposing it to conditions that result in platelet activation, vasoconstriction, pro-oxidation, mitogenesis, vascular inflammation, atherosclerosis, and subsequent thrombosis resulting in cardiovascular diseases [14]. It has been demonstrated that low-grade chronic vascular inflammation and decreased bioavailability of nitric oxide due to increased oxidative stress in obesity play an important role in endothelial dysfunction [16,17]. Increased plasma concentrations of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and other inflammatory cytokines have also been associated with the presence of endothelial dysfunction in patients with obesity [18]. Visceral adipose tissue (VAT) is an important key tool affecting glucose metabolism, blood pressure, lipid profile, and inflammatory profiles, and is associated with a worse cardiometabolic profile, including impaired lipid

profile and high fasting insulin, even in healthy young adults who appear to lack traditional risk factors, whether overweight or not [19,20]. The visceral adiposity index (VAI), a predictor of visceral dysfunction is an uncomplicated marker specific to the gender that includes anthropometric data and lipid profiles to provide [10]. Similarly TPI a suitable parameter in determining obesity, has been shown to discriminate body fat distribution more accurately than BMI [21]. In our study, we found that the endothelial functions of individuals with obesity without obvious cardiovascular disease were impaired according to the flow-mediated dilatation method. In addition, impairment of existing endothelial functions was also associated with visceral and perirenal adipose tissue thicknesses. In addition; VAI and TPI values are also associated with these impairment.

Various studies were conducted in the past to examine the relationship between obesity and endothelial functions. In a study conducted by El Assar *et al.* on morbid obese patients (32 with insulin resistance, 20 without insulin resistance), endothelial-dependent relaxation and bradykinin were evaluated within mesenteric micro vessels with wire myograph. They suggested that increased mitochondrial superoxide formation and increased systemic inflammation mediated by TNF-α could cause deterioration in endothelial functions in these patients and this deterioration could only occur in the presence of insulin resistance [15]. In our study, HbA1C and HOMA-IR were significantly higher in the group with obesity compared to the control group and there were correlations with FMD.

In a study conducted on healthy individuals in Korea, the relationship between anthropometric factors, factors that may be associated with endothelial dysfunction, and the amount of abdominal adipose tissue measured by computed tomography was examined. As a result of the study, it was concluded that central obesity was associated with parameters that could indicate high CRP levels, insulin resistance, and endothelial dysfunction [22]. In our study, we demonstrated that the visceral and perirenal adipose tissue thicknesses obtained by ultrasonography were associated with the endothelial functions demonstrated with FMD. Similarly, we found a relationship between HbA1C and HOMA-IR, which would indicate insulin resistance. In another small-scale study explaining the relationship between weight gain and endothelial functions, 43 individuals with normal weight were divided into groups as weight gain (4 kg on average) and individuals who maintained their weight, and endothelial functions were examined using the FMD method. It was revealed that there was no change in endothelial functions in individuals whose weight gain and endothelial functions were stable. In addition, it was demonstrated that endothelial functions recovered after weight loss in individuals, who regained weight [23]. In an experimental study conducted in 2016, renal blood flow glomerular filtration rate, perirenal adipose amount, and endothelial functions in renal arteries were investigated ex vivo after a diet that could cause obesity and metabolic imbalances in pigs for 16 weeks. It was demonstrated that the amount of perirenal fat increased in pigs with negative nutrition and obesity, and that endothelial functions deteriorated in these pigs compared to pigs fed without fat. They concluded that increased proinflammatory macrophage infiltration in perirenal adipose tissue was associated with increased TNF alpha expression [24]. In our study, we demonstrated that FMD levels were impaired in individuals with obesity and thicker perirenal adipose tissue, and this impairment was associated with perirenal adipose tissue thickness. VAI and TPI which are indicators of body fat distributions were also related with FMD according to our results.

Atherosclerosis is a chronic progressive inflammatory disease thought to begin in the early stages of life and has a possible long subclinical phase [26]. It is quite difficult to reveal the true prevalence; however, studies have suggested that it is present in approximately 1/3 of female and male individuals [25,26]. Therefore, it is necessary to diagnose pathology early to prevent the progression of the disease and to administer treatment in the early period. CIMT, which can be easily diagnosed with carotid ultrasound, a non-invasive imaging method, is a predictor of future cardiovascular diseases and is also associated with markers of traditional cardiovascular diseases such as age, cholesterol level and diabetes [27]. It has been demonstrated to be a screening tool in individuals with high risk of heart disease [28].

In a study conducted on 3079 patients, by Alvey *et al.* examined the relationship between visceral, subcutaneous, and pericardial adipose tissue densities measured by computed tomography with the amount of coronary and abdominal aortic

calcium. As a result, they concluded that low visceral and subcutaneous adipose tissue densities were associated with lower subclinical atherosclerosis [29]. Unlike Alvey *et al.*, we determined visceral and perirenal adipose tissue thicknesses with ultrasonography and subclinical atherosclerosis with CIMT measurement in our study. Similarly, we found a significant correlation between the amount of visceral adipose tissue and CIMT. In addition, some studies demonstrated that carotid ultrasonography was more sensitive compared to the coronary artery calcification score in the detection of subclinical atherosclerosis [30]. In a study conducted by Gast *et al.* involving 2451 patients, the effects of visceral adipose tissue and total body fat on subclinical atherosclerosis were examined. While subclinical atherosclerosis was primarily associated with insulin resistance, it was demonstrated that total body fat was associated with subclinical atherosclerosis in both males and females, and that visceral adipose tissue was associated with subclinical atherosclerosis, especially in females. They presented the idea that VAT had a promotor effect on early atherosclerosis [31]. In our study we found significant relation between CIMT and visceral and perirenal adipose tissue. As a result of our study, the fact that parameters such as VAI and TPI were closely related to CIMT supported our results.

In a similar study, Wang et al. examined the relationship between the visceral adipose tissue thicknesses obtained by magnetic resonance imaging and CIMT in a group of 1005 people without cardiovascular disease. They concluded that overweight individuals or individuals with obesity had thicker CIMT as well as carotid atherosclerosis in both healthy individuals and overweight individuals or individuals with obesity with visceral fat accumulation $\geq$80 cm^2 [32]. This study also demonstrated that there was a relationship between excess weight and subclinical atherosclerosis even in individuals without cardiovascular disease. The amount of visceral fat is effective in demonstrating subclinical atherosclerosis.

Sturm *et al.* examined the relationship between visceral and subcutaneous fat accumulation and CIMT, endothelial functions, and hepatic fat amount measured by ultrasonography in 64 severely overweight individuals (BMI 42.3±4.3 kg/m^2). They demonstrated that FMD was negatively correlated and CIMT was positively correlated with visceral fat accumulation. In their study, while there was a relationship between the degree of Liver fat and FMD, there was no relationship with CIMT. As a result of the study, they concluded that the amount of visceral adipose tissue was an essential determinant of endothelial dysfunction in individuals with obesity [8]. Similar to Sturm *et al.*, we measured visceral adipose tissue with ultrasonography and obtained similar results in our study. However, we demonstrated that perirenal adipose tissue thickness, which was measured differently, could also be associated with endothelial functions.

STUDY LIMITATIONS

Firstly, our study was cross-sectional and it was conducted by including individuals who admitted to our hospital at a certain period. The results of the study may not be generalized to individuals in different geographical locations and different sociocultural groups. Since visceral fat accumulation is not a homogeneous accumulation, ultrasonographic adipose tissue thickness measurements may be considered potentially erroneous. Therefore, the measurements were obtained by the same specialist, separately from both abdomens and by taking the mean of 3 separate measurements to reduce the margin of error. Another limitation was that the results of the study could have been confirmed in case the patients were followed up for a long time and imaged according to their weight gain and loss status. However, we did not have the opportunity to follow-up the patients. Tomography could have been a better method to measure the amount of perirenal and visceral adipose tissue. We did not consider it appropriate to administer an examination with X-rays to patients without any indication.

CONCLUSION

As a result, individuals with obesity have impaired endothelial functions and greater carotid intima-media thicknesses compared to healthy individuals. This impairment in endothelial functions and increase in carotid intima-media thicknesses are proportional to the amount of visceral and perirenal fat accumulation. Parameters reflecting visceral fat distribution such as VAI and TPI are also related with these impairments. These changes develop before the development of overt cardiovascular disease.

Introducere: Obezitatea accelerează progresia aterosclerozei, inducând modificări fiziopatologice care sunt deja detectabile de la adulții tineri. Disfuncția endotelială este una dintre cele mai timpurii modificări vasculare observate în obezitate. În acest studiu, ne-am propus să determinăm funcțiile endoteliale și grosimea intima-media carotidiană la pacienții cu obezitate fără boală cardiovasculară evidentă.

Metode: Studiul a fost realizat pe 112 persoane cu obezitate, fără boală cardiovasculară evidentă și orice boală cronică (IMC>30 kg/m^2) (84 femei, 28 bărbați, vârsta medie: 46,3±11,2 ani) și 49 indivizi sănătoși fără afecțiuni (33 femei, 16 bărbați, vârsta medie: 44,6±10,2 ani). Toți pacienții au fost examinați pentru funcțiile endoteliale prin metoda dilatației mediate de flux (FMD) și grosimea intima-media carotidiană (CIMT). Toate măsurătorile au fost efectuate de același specialist în imagistică, cu o medie a 3 măsurători diferite. În plus față de indicele de masă corporală și circumferința taliei, indicele de adipoziție viscerală (VAI) și indicele de masă triponderal (TPI) au fost calculate.

Rezultate: Procentul de FMD obținut prin ecografie de artera brahială a fost semnificativ mai mic, țesutul adipos visceral, grosimile țesutului adipos perirenal măsurate prin ecografie abdominală și CIMT au fost semnificativ mai groase în grupul 1 comparativ cu grupul 2. FMD a avut o corelație semnificativă negativă cu indicele de masă corporală. grosimea țesutului adipos visceral, grosimea țesutului adipos perirenal și circumferința taliei și șoldului și grosimea intima-media carotidiană și CIMT au avut o corelație semnificativă cu grosimea țesutului adipos visceral, grosimea țesutului adipos perirenal, VAI, TPI și circumferința taliei, șoldului.

Concluzie: Persoanele cu obezitate au funcții endoteliale afectate și grosime intima-media carotidiană mai mare în comparație cu indivizii sănătoși. Această afectare a funcțiilor endoteliale este proporțională cu cantitatea de acumulare de grăsime viscerală și perirenală. Parametrii care reflectă distribuția grăsimii viscerale, cum ar fi VAI și TPI, sunt, de asemenea, legați de această afectare.

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