Epicardial adipose tissue is independently associated with increased left ventricular mass in untreated hypertensive patients: an observational study

Epikardiyal adipoz doku tedavi edilmemiş hipertansif hastalarda artmış sol ventrikül kitlesi ile bağımsız ilişkilidir: Gözlemsel bir çalışma

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ABSTRACT

Objective: Epicardial adipose tissue (EAT) secretes various inflammatory mediators and growth factor, and has endocrine and paracrine effects on myocardium and body. We planned the present study in order to evaluate the possible relationship between EAT and left ventricular mass (LVM), a potent predictor of cardiovascular mortality and morbidity, independent of age, blood pressure and the metabolic parameters in patients with hypertension (HT).
Methods: The present study was cross-sectional and observational, including consecutive 107 untreated essential hypertensive patients who underwent a complete transthoracic echocardiographic examination as well as measurements of LVM and EAT. Blood pressure, routine blood chemistry, C-reactive protein, and patient characteristics were also recorded. Univariate and then multiple linear regression analyses were used for analysis of independent variables associated with EAT.
Results: LVM significantly correlated with waist circumference, EAT, glucose, uric acid, high-density lipoprotein (HDL) cholesterol, and systolic and diastolic blood pressure. When we divided study population into two groups according to median mean blood pressure (BP) (Mean BP ≤116 vs. >116 mmHg), EAT was the only associated factor for LVM in patients below median BP (Beta: 0.518, p<0.001). Linear regression analyses revealed EAT to be independently associated with LVM (Beta: 0.419; p<0.001) and LVM index (Beta: 0.384, p<0.001) as well as high-density lipoprotein (Beta: -0.264, p=0.006).
Conclusion: EAT was related to increased LVM independent of BMI, waist circumference, weight, systolic and diastolic blood pressure and other risk parameters, in patients with HT. Determination of increased EAT by echocardiography may have an additional value as an indicator of cardiovascular risk and total visceral adipose tissue. *(Anadolu Kardiyol Derg 2013; 13: 320-7)*
Key words: Left ventricular hypertrophy, epicardial adipose tissue, hypertension, obesity, visceral fat, blood pressure, regression analysis

ÖZET

Amaç: Epikardiyal adipoz doku (EAD) çeşitli enflamatuvar mediyatörler ve büyüme faktörleri salarak miyokart ve vücut üzerine endokrin ve parakrin etkilere sahiptir. Bu çalışmada hipertansiyonlu (HT) hastalarda EAD ve kardiyovasküler morbidite ve mortalitenin güçlü bir öngörücüsü olan sol ventrikül hipertrofisi (SVH) arasındaki olası ilişkiyi yaş, kan basıncı ve metabolik parametrelerden bağımsız olarak araştırmayı amaçladık.
Yöntemler: Bu çalışma EAD ve SVH ölçümleri yanında tam bir ekokardiyografik değerlendirmesi yapılan 107 ardışık, tedavi almamış esansiyel HT hastasında yapılan kesitsel ve gözlemsel bir çalışmadır. Kan basıncı (KB), rutin biyokimya, C-reaktif protein ve diğer hasta karakteristikleri ölçüldü. Tek değişkenli ve ardından çoklu lineer regresyon analizleri EAD ile bağımsız ilişkide olan değişkenlerin analizinde kullanıldı.
Bulgular: Sol ventrikül kitlesi (SVK) bel çevresi, EAD, glikoz, ürik asit, yüksek-dansiteli lipoprotein (HDL) kolesterol, ve sistolik ve diyastolik kan basıncı ile anlamlı olarak ilişkiliydi. Çalışma popülasyonu ortanca ortalama kan basıncına göre iki gruba ayrıldığında (ortalama KB ≤116 vs. >116 mmHg), medyan KB değeri altındaki hastalarda EAD SVK'nin tek nedensel faktörüydü (Beta: 0.518, p<0.001). Lineer regresyon analizleri EAD'yi SVK (Beta: 0.419; p<0.001) ve SVK indeksi (Beta: 0.384, p<0.001) için HDL kolesterolün azaltıcı etkisi yanında (Beta: -0.264, p=0.006) tek bağımsız ilişkili değişken olarak belirledi.

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Sonuç: EAD hipertansiyon hastalarında artmış SVK ile VKİ, bel çevresi, kilo, sistolik ve diyastolik KB ve diğer risk parametrelerinden bağımsız olarak ilişkiliydi. Ekokardiyografi ile artmış EAD'un belirlenmesi total visseral yağ dokusu ve kardiyovasküler riskin öngörülmesinde bir belirteç olarak ek bir değere sahip olabilir. *(Anadolu Kardiyol Derg 2013; 13: 320-7)*

Anahtar kelimeler: Sol ventrikül hipertrofisi, epikardiyal adipoz doku, hipertansiyon, obezite, visseral yağ, kan basıncı, regresyon analizi

Introduction

Hypertension (HT) causes compensatory processes in heart due to increased chronic workload manifested as left ventricular hypertrophy (LVH), which is frequently diagnosed by echocardiography (1). LVH is an important predictor of mortality and morbidity in patients with essential HT (2-4). Moreover, LVH is a marker of subclinical cardiovascular disease (5). Decreased left ventricular hypertrophy is associated with lower cardiovascular mortality independent of blood pressure reduction (6-9).

Another cardiovascular risk factor for LVH in addition to increased blood pressure is obesity (10). Even though a higher total body fat is observed in obese patients, cardiovascular events are mainly related to increased visceral fat tissue (11). Epicardial adipose tissue (EAT), localized between myocardium and visceral pericardium, is a true visceral fat that correlates well with total visceral adipose tissue (12-15). EAT increases with obesity (16,17). Moreover, EAT secretes various inflammatory mediators and growth factor and works as an endocrine, paracrine and autocrine effected organ (18-22). EAT is strongly associated with increased cardiovascular risk, and increased LVM (23) documented by both autopsy and echocardiography (12, 24). Iacobellis et al. (24) reported that this association was independent of systolic and diastolic blood pressure. However, they revealed this result in a limited number of patients with heterogeneous characteristics. Moreover, recent studies also documented that EAT is also associated with diastolic dysfunction (25), non-dipper status (26), increased left atrial size and lower ejection fraction (27), arterial stiffness (28), higher coronary calcium score (29), and reduced regional systolic motion (30) in various populations.

Clinical studies support a robust relationship between EAT and LVH (24, 26). Essential HT is frequently observed in obese patients during daily clinical practice, which suggests that EAT may also play a crucial part in LVH.

Therefore, we planned the present study in order to evaluate the relationship between EAT and LVH in patients with HT.

Methods

Study design

The present study was observational and cross-sectional in design.

Study population and protocol

Our study included 107 consecutive untreated essential hypertensive patients who underwent a complete transthoracic echocardiographic examination as well as measurements of EAT. Patients with previous coronary artery disease (CAD), diabetes mellitus, left ventricular systolic dysfunction, pulmonary hypertension, right ventricular hypertrophy, secondary hypertension, moderate-severe valve disease, atrial fibrillation, symptoms of CAD and equivalent findings on exercise ECG and perfusion scan, and patients previously treated for hypertension were excluded. Patients with non-optimal echocardiographic image quality and patients who did not give informed consent were also excluded. Ambulatory blood pressure (BP) measurements were recorded in all patients. The study was performed in accordance with the principles stated in the Declaration of Helsinki and approved by the local Ethics Committee.

Clinical and laboratory assessment

Baseline characteristics of the patients were recorded. HT was defined as a sustained systolic BP > 140 mmHg, and/or diastolic BP >90 mmHg (with high blood pressure after two documented office reading). Patients who were using tobacco products on admission and those who quitted smoking within the last year were considered as smokers.

Blood samples were drawn by venipuncture to measure routine blood chemistry parameters after fasting for at least 8 hours. Fasting blood glucose, serum creatinine, uric acid levels, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglyceride levels were recorded. Glucose, creatinine, and lipid profile were determined by standard methods. Serum C-reactive protein (CRP) was analyzed using a nephelometric technique (Beckman Coulter Image 800; Fullerton, CA, USA; normal range 0-0.8 mg/dL). Weight and height were measured while the subjects were fasting and wearing only their undergarments.

BP values were obtained both at office by traditional auscultatory method using a sphygmomanometer and at home by ambulatory blood pressure monitoring. Ambulatory BP measurements (PhysioQuant Ambulatory Blood Pressure Monitor system, EnviteC-Wismar, Germany) were recorded in all patients.

Echocardiography

Images were acquired in the left lateral decubitus position of patients with a GE-Vingmed Vivid S5 (GE-Vingmed Ultrasound AS, Horten, Norway) using a 2.5-3.5 MHz transducer by two experienced cardiologists. The echocardiographic study required recording of ≥10 cycles of 2-dimensional parasternal long-axis views and ≥10 cycles of M-mode with optimal cursor beam orientation in each view (31, 32). Interventricular septum (IVS), posterior wall (PW), left ventricular end-diastolic diameter (LVEDD), and left ventricular end-systolic diameter (LVESD) were measured and noted. Body mass index (BMI) was determined by the follow-

ing formula: BMI=weight (kg)/height^2 (m). Waist circumference was measured while the subjects were standing with their heels together. Body surface area (BSA) was calculated according to formula BSA (m^2) = 0.007184 x Height $(cm)^{0.725}$ x Weight $(kg)^{0.425}$, LVM was calculated according to 1.04 [(LVEDD + PW + IVS)^3-(LVEDD)^3] -13.6 g formula (33), left ventricular mass index (LVMI) was calculated as LVMI=LVM/BSA formula.

Evaluation of epicardial adipose tissue

EAT was evaluated on the free wall of right ventricle from the parasternal long-axis view, using aortic annulus as an anatomic reference (Fig. 1). We preferred the area of above the right ventricle to measure EAT thickness, because this area is known to have the thickest EAT layer. EAT, identified as an echo-free space between the myocardium and visceral pericardium on 2-dimensional echocardiography, was measured perpendicularly in front of the right ventricular free wall at end-diastole (16, 34). We magnified each still image for better visualization and accurate measurement of EAT thickness and measured the thickest point of EAT in each cycle. To standardize the measuring axis, we used the aortic annulus as anatomical reference. The measurement was performed at a point on the free wall of the right ventricle along the midline of the ultrasound beam, perpendicular to the aortic annulus. The average value comprising three cardiac cycles of each echocardiographic view was used for the statistical analysis. Inter-observer and intra-observer variability on epicardial fat thickness measurement was excellent: intra-class coefficient of correlation was 0.92 and 0.96, respectively.

Statistical analysis

The SPSS statistical software (SPSS 15.0 for Windows, Inc., Chicago, IL, USA) was used for all statistical calculations. Continuous variables are given as mean±standard deviation; categorical variables are defined as percentages. Data were tested for normal distribution using the Kolmogorov-Smirnov test. The Chi-square test was used for the categorical variables among the groups. Mean values were compared by ANOVA followed by the Tukey's HSD test among different groups. Pearson correlation coefficient was used to analyze the relationship between study variables. Linear regression analysis with enter method was used for all relevant independent variables which were included if they were significantly different in the univariate analyses. Further, the analysis was repeated after a pre-elimination with Stepwise method for the independent variables. All tests of significance were two-tailed. Statistical significance was defined as $p<0.05$.

Results

Clinical characteristics

The clinical characteristics of the study population are detailed in Table 1.

Associations of EAT with study parameters

BMI ($p<0.001$), waist circumference ($p<0.001$) and blood pressure measurements (systolic; $p=0.001$, diastolic; $p=0.004$) were significantly elevated in high EAT group compared to low EAT group. Age and C-reactive protein had only a tendency to increase with EAT ($p=0.093$ and $p=0.061$, respectively). LVM and LVM index were significantly higher in patients with increased EAT (Table 1).

Correlations of left ventricular mass with study parameters

Left ventricular hypertrophy significantly correlated with waist circumference ($r=0.342$, $p=0.001$), EAT ($r=0.534$, $p<0.001$) (Fig. 2), glucose ($r=0.205$, $p=0.045$), uric acid ($r=0.242$, $p=0.022$), HDL ($r=-0.435$, $p<0.001$), and systolic and diastolic blood pressure ($r=0.230$, $p=0.018$ and $r=0.277$, $p=0.004$, respectively). These correlations were also valid for LVM index (Table 2).

CRP, LVM and EAT

Although CRP was related to BMI ($r=0.485$, $p<0.001$), waist circumference ($r=0.368$, $p=0.001$) and EAT ($r=0.315$, $p=0.003$), CRP did not correlate with LVM and LVM index.

The association of EAT and LVM

When we divided study population into two groups according to median mean blood pressure (Mean BP ≤116 vs. >116 mmHg), EAT was the only associated factor for LVM in patients below median BP. However, in patients above median BP, diastolic BP was also a factor for LVM (Table 3). Linear regression analyses revealed that EAT was significantly associated with LVM and LVM index as well as high-density lipoprotein (Table 4).

Discussion

We revealed an independent relationship between LVM, increased EAT and decreased high-density lipoprotein in patients with HT in the present study.

Evidence suggests that visceral fat accumulation increases cardiovascular risk and susceptibility to ischemic heart disease (35). Waist circumference and BMI measurement is currently utilized in routine clinical practice to estimate visceral fat tissue. BMI and waist circumference display similar correlations to total, visceral, and subcutaneous fat areas (36). Computed tomography (CT scan) and magnetic resonance imaging (MRI) are currently the gold standard for quantitative assessment of intra-abdominal adipose tissue (37). EAT, an intra-thoracic visceral fat tissue, is embryologically similar to abdominal visceral fat tissue (19). Iacobellis et al. (16, 34) first reported echocardiographic measurement of epicardial fat in 2003. Echocardiographically measured epicardial adipose tissue thickness is a marker for visceral adiposity, adiposity related metabolic and cardiovascular risk (16, 34). Epicardial adipose tissue has been shown to be very closely related to intra-abdominal adiposity, a marker of entire body visceral adiposity, according to various magnetic resonance imaging studies (16, 34). Echocardiographic measurement of EAT,

Table 1. Baseline characteristics of the study population

Variables (N=107)	Epicardial fat pad thickness			F	†p
	<6 mm (n=41)	6-8 mm (n=31)	>8 mm (n=35)		
Age, years	47±7	51±10	51±10	2.43	0.093
Gender, male, %	44	52	60	-	0.375
BMI, kg/m^2	29.8±3.8	31.0±3.8	34.5±4.3*	13.9	<0.001
Waist circumference, cm	98.4±9.5	103.5±8.5	112.8±8.5*	20.5	<0.001
Smoking, %	42	40	57	-	0.284
Hyperlipidemia, %	39	33	34	-	0.861
Glucose, mg/dL	97±18	102±15	102±16	0.95	0.392
Creatinine, mg/dL	0.80±0.22	0.83±0.12	0.78±0.10	0.79	0.456
Uric acid, mg/dL	4.87±1.45	5.46±1.22	5.64±1.46	2.65	0.076
Total cholesterol, mg/dL	212±42	227±29	227±42	1.65	0.198
LDL, mg/dL	134±32	142±33	143±38	0.71	0.494
HDL, mg/dL	51±15	48±12	46±10	1.12	0.307
Triglyceride, mg/dL	142±81	186±126	189±124	1.89	0.157
CRP, mg/dL	0.41±0.42	0.67±0.55	0.72±0.64	2.88	0.061
Epicardial fat pad thickness, mm	4.8±1.1	6.9±0.7*	9.9±1.5*	192.7	<0.001
Blood pressure measurements					
Office					
Systolic BP, mmHg	147±16	158±15*	161±17*	7.14	0.001
Diastolic BP, mmHg	93±10	100±6	99±11*	3.72	0.004
Pulse pressure, mmHg	54±12	58±14	62±13*	3.07	0.051
ABPM (1 month after treatment)					
Total Systolic BP, mmHg	138±13	146±13	146±13	2.94	0.060
Total Diastolic BP, mmHg	93±12	96±9	96±10	0.87	0.422
Mean BP, mmHg	108±12	113±9	113±10	1.67	0.199
Day Systolic BP, mmHg	140±12	148±14	148±12	3.29	0.043
Day Diastolic BP, mmHg	103±13	105±9	105±12	2.08	0.667
Mean day BP, mmHg	110±12	116±10	115±10	1.97	0.147
Night Systolic BP, mmHg	134±14	140±14	140±15	1.37	0.262
Night Diastolic BP, mmHg	90±13	88±9	88±11	0.06	0.944
Mean night BP, mmHg	103±13	105±9	105±12	0.41	0.667
Echocardiography					
Ejection fraction, %	65±3	65±4	64±4	1.94	0.149
LVEDD, mm	45±8	45±4	47±4	1.15	0.362
LVESD, mm	28±3	29±7	30±5	1.81	0.169
IVSD, mm	11.4±1.2	12.2±1.7	13.8±1.8*	22.0	<0.001
PWD, mm	10.7±1.2	11.5±1.2*	12.6±1.5*	22.5	<0.001
LVM, gr	211±65	231±50	293±84*	14.2	<0.001
LVM index, gr/m^2	111±34	118±25	143±40*	8.59	<0.001

Data are presented as mean±SD and percentages
†Chi-square and ANOVA test
*post-hoc Tukey HSD test - when compared to first group (<6mm) p<0.05
ABPM - ambulatory blood pressure monitoring, BMI - Body mass index, BP - blood pressure, CRP-C - reactive protein, HDL - High-density lipoprotein, IVSD - interventricular wall thickness, LDL - Low-density lipoprotein, LVEDD - left ventricular end-diastolic dimension, LVESD - left ventricular end-systolic dimension, LVM - left ventricular mass, PWD - posterior wall thickness

Table 2. Correlations of epicardial fat pad thickness with LVM and other study parameters

Variables	LVM (gr)		LVM index (gr/m²)	
	r	†p	r	†p
Age, years	0.176	0.066	0.251	0.008
BMI, kg/m²	0.121	0.206	-0.014	0.886
Waist circumference, cm	0.342	0.001	0.170	0.110
EAT, mm	0.534	<0.001	0.446	<0.001
Glucose, mg/dL	0.205	0.045	0.152	0.139
Creatinine, mg/dL	0.173	0.090	0.237	0.020
Uric acid, mg/dL	0.242	0.022	0.180	0.089
CRP, mg/dL	0.039	0.715	0.062	0.561
LDL, mg/dL	-0.121	0.244	-0.092	0.376
HDL, mg/dL	-0.435	<0.001	-0.362	<0.001
Triglyceride, mg/dL	0.186	0.070	0.096	0.351
Systolic BP, mmHg	0.230	0.018	0.215	0.027
Diastolic BP, mmHg	0.277	0.004	0.247	0.011
Pulse pressure, mmHg	0.121	0.220	0.126	0.200

†Pearson correlation analysis
BMI - Body mass index, BP - blood pressure, CRP-C - reactive protein, EAT - epicardial adipose tissue, HDL - high density lipoprotein, LDL - low-density lipoprotein, LVM - left ventricular mass

Table 3. Correlations of EAT and blood pressure parameters with LVM in two groups according to median mean blood pressure

The patients with mean BP ≤116 mmHg (median value)				
	LVM (gr)		LVM index (gr/m²)	
Variables	r	p	r	p
EAT, mm	0.518	0.001	0.480	0.001
Systolic BP, mmHg	0.019	0.904	-0.077	0.626
Diastolic BP, mmHg	-0.055	0.731	-0.162	0.304
Mean BP, mmHg	-0.028	0.863	-0.148	0.349
The patients with mean BP >116 mmHg (median value)				
Variables	**r**	**p**	**r**	**p**
EAT, mm	0.484	<0.001	0.347	0.006
Systolic BP, mmHg	0.173	0.173	0.186	0.141
Diastolic BP, mmHg	0.405	0.001	0.398	0.001
Mean BP, mmHg	0.349	0.005	0.354	0.004

†Pearson correlation analysis
BP - blood pressure, EAT - epicardial adipose tissue, LVM - left ventricular mass

an objective, noninvasive, readily available, and certainly less expensive measure of visceral fat than MRI or CT, offers a more sensitive and specific index of true visceral fat content by avoiding the possible confounding effect of subcutaneous abdominal fat (38, 39).

There is not a real fascia separating EAT from the myocardium, thus both share similar microcirculation (40). Mediators

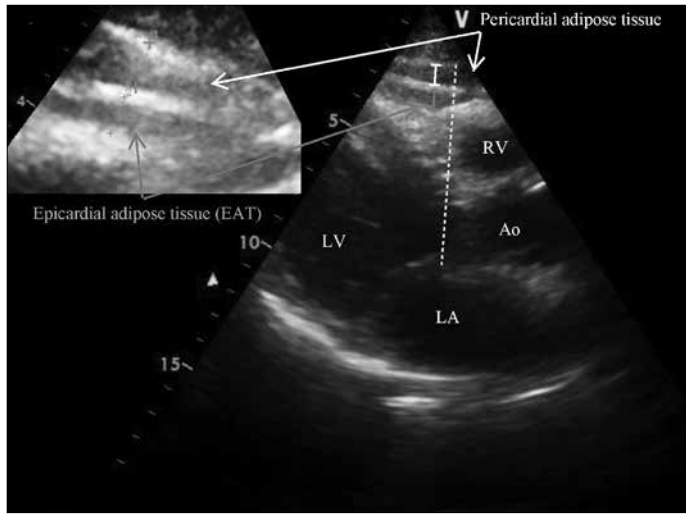

Figure 1. The measurement of epicardial fat thickness by echocardiography
EAT - identified as an echo-free space between the myocardium and visceral pericardium from the parasternal long-axis view on 2-dimensional echocardiography, was measured perpendicularly in front of the right ventricular free wall at end-diastole

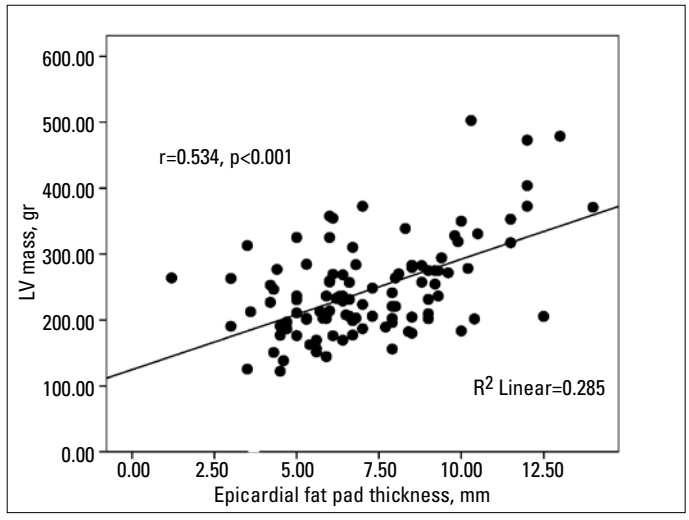

Figure 2. The correlation of epicardial adipose tissue thickness with left ventricular mass
*Pearson correlation analysis

secreted from epicardial fat tissue may affect myocardial tissue by vasa vasorum or direct diffusion (40). Previous studies demonstrated that EAT secretes several inflammatory mediators and growth factors (41, 42). Of these mediators, interleukine-6 (IL), transforming growth factor-β and macrophage chemotactic factor-1 (MCP-1) induce myocardial hypertrophy in cell cultures (20-22, 43). On the other hand, increased EAT may cause myocardial inflammation which disrupts local collagen metabolism, resulting in increased left ventricular mass (44). We hypothesize that both proposed theoretical mechanisms justify the notion that EAT might increase left ventricular mass, independent of blood pressure values. The present study and the previously reported studies regarding this association did not reveal specific mechanisms. Therefore, more studies are required to clarify this issue. However, since we could not document a signifi-

Table 4. Linear regression analysis of factors associated with increased LVM

Linear regression analysis	Dependent variable: LVM				Dependent variable: LVM index			
Independent variables	***p**	**Beta (standardized)**	**†p**	**Beta (standardized)**	***p**	**Beta (standardized)**	**†p**	**Beta (standardized)**
Age, years	0.138	0.162			0.109	0.164		
Epicardial fat pad thickness, mm	0.012	0.357	<0.001	0.419	<0.001	0.463	<0.001	0.384
Waist circumference, cm	0.733	-0.038			0.103	-0.191		
Glucose, mg/dL	0.506	0.065			0.734	0.033		
Creatinine, mg/dL	0.176	0.150			0.012	0.258		
Systolic BP, mmHg	0.807	-0.032			0.638	-0.060		
Diastolic BP, mmHg	0.029	0.302	0.062	0.165	0.066	0.243		
HDL, mg/dL	0.001	-0.358	0.001	-0.301	0.003	-0.296	0.006	-0.264
Triglyceride, mg/dL	0.886	0.014			0.739	-0.034		
Uric acid, mg/dL	0.876	-0.018			0.938	-0.010		
Constant	0.569	-	0.095	-	0.615	-	<0.001	-
Adjusted R^2	0.473		0.367		0.413		0.244	

Linear regression analysis with enter method was used for all relevant independent variables which were included if they were significantly different in the univariate analyses*.
Further, the analysis was repeated after a pre-elimination with Stepwise method for the independent variables†.
BP - blood pressure, HDL - high-density lipoprotein, LVM - left ventricular mass

cant association between LVM and anthropometric calculations, indirect indicators of visceral fat tissue; we, like Corradi et al. (12), think that local effects of EAT have an important role in left ventricular hypertrophy. On the other hand, HDL had an independent negative relationship with LVM in our analyses. HDL is a metabolic syndrome (11) parameter and may represent it; therefore, absence of MS may be protective for LVM (45,46).

Another important result of our study is that creatinine, an indirect marker of renal function, increased LVM, which suggests importance of subclinical end-organ damage as a co-factor (47). However, this association was not strong enough to be independent of EAT and HDL. Additionally we revealed an independent negative association between LVM and HDL potentially related to metabolic syndrome and/or sedentary life style (46,48-51). Interestingly we could not document a significant association between LVM and systolic or diastolic blood pressure in patients with mean BP ≤116 mmHg, possibly due to relatively lower systolic and diastolic blood pressure values of this patient group, in which EAT was only predictor for LVM. However, increased left ventricular mass correlated with higher blood pressure values in patients with mean BP >116 mmHg.

Study limitations

Magnetic resonance imaging is the gold standard diagnostic method for measuring epicardial fat thickness now. Not using MRI in our research is a study limitation. Although epicardial fat is readily visualized with the high-speed computed tomography and MRI, widespread use of these methods for assessment of EAT is not practical. Echocardiography provides an objective, noninvasive, readily available method and is certainly less expensive than MRI or computed tomography for measuring epicardial fat. In present study, we did not study the local in tissue level or systemic markers of EAT; therefore this was an important limitation to link findings of echocardiographically measured EAT thickness and LV hypertrophy in hypertension.

Conclusion

In conclusion, EAT is related to increased LVM independent of BMI, waist circumference, weight, systolic and diastolic blood pressure and other risk parameters, in patients with essential HT. Thus, determination of increased EAT by echocardiography may have an additional value as an indicator of predicting cardiovascular risk and total visceral adipose tissue. The patients with increased EAT may have an increased risk for LV hypertrophy and other cardiovascular events, and EAT can predict them and it can be a clinical tool for the determination of high-risk patients. Moreover, interventions decreasing epicardial adipose tissue may also reduce cardiovascular risk by concomitant reductions in left ventricular mass.

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Peer-review: Externally peer-reviewed.

Authorship contributions: Concept - T.E.; Design - S.A.K.; Supervision - S.A.K.; Resource - A.Ç.; Material - Y.U.; Data collection&/or Processing - E.E.; Analysis &/or interpretation - S.A.K.; Literature search - M.Ç.; Writing - M.E.D.; Critical review - S.A.K.

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