# Epicardial Adipose Tissue Thickness in Hypertensive Patients with or without Left Ventricular Hypertrophy

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## ABSTRACT

**Background:** Epicardial adipose tissue (EAT) is a type of visceral fat located between the heart and pericardium, affecting vascular smooth muscle activity through chemical secretion. It is a risk factor for cardiovascular metabolic issues and may relate to hypertension (HT).

**Objective:** To assess the correlation between EAT thickness and HT in individuals with and without left ventricular hypertrophy (LVH).**Patients and Methods:** This observational study included 105 individuals with HT, defined as having a systolic blood pressure of ≥140 mm Hg, a diastolic pressure of ≥90 mm Hg, or being on antihypertensive medication, and 30 healthy controls. Participants underwent transthoracic echocardiography to measure left ventricle dimensions and EAT thickness. **Results:** Positive correlations were found between EAT thickness and several cardiac structural and functional parameters (Left ventricular mass index (LVMI), the ratio of early mitral inflow velocity to mitral annular early diastolic velocity (E/'e ratio), etc.), with a significant correlation between EAT thickness and both LVMI and E/'e ratio. EAT thickness above 5.25 mm predicted LVH in HT patients with 85.7% sensitivity, 82.1% specificity, and 83.9% accuracy. **Conclusions:** Compared to hypertensive patients without hypertrophy and healthy individuals, hypertensive patients with LVH had a much thicker EAT. Based on the findings, EAT may be a useful marker for HT ventricular remodeling and hypertrophy.

**Keywords:** Epicardial Adipose Tissue; Thickness, Hypertension; Left ventricular hypertrophy.

## INTRODUCTION

The accumulation of fat surrounding internal organs is only one aspect of visceral adipose tissue. Among its many metabolic and endocrine roles, it is a rich source of a small number of bioactive compounds with far-reaching effects on energy metabolism, vascular health, immunology, and inflammation [1].

EAT is a kind of visceral fat that accumulates in certain areas surrounding the coronary arteries, the right ventricle's lateral wall, the atrioventricular and interventricular sulcus, and the pericardium [2].

It is believed that its physiological functions relate to lipid storage and these processes; it is associated with metabolic syndrome [3], insulin resistance [4], coronary artery disease (CAD) [5], hypertension (HT) [6], and the production of hormones, cytokines, and chemokines [7,8]. The pathophysiology of CAD is thought to be heavily influenced by EAT. It modulates the contraction of vascular smooth muscles by releasing a variety of physiologically active chemicals [9]. Their proximity to the adventitia and extravascular bed may explain their paracrine actions [12]. New research suggests that HT may be associated with increased EAT thickness, which might be a new cardio-metabolic risk factor [10, 11].

Echo-cardiography is a common tool for diagnosing LVH, a symptom of HT-induced compensatory cardiac mechanisms carried on by an elevated chronic workload. LVH is a significant indicator of death and illness in individuals with HT. It may cause diastolic dysfunction, decrease coronary flow reserve, and promote ventricular arrhythmias [12].

Additionally, LVH serves as an indicator of underlying cardiovascular illness that may not yet be showing symptoms. The relationship between EAT and LVH remains uncertain [12]. Transthoracic echocardiography is being widely used for evaluating EAT because to its several benefits, including accessibility, affordability, lack of radiation, speed, and consistency [13].

The objective of this research was to assess the relationship between EAT thickness and the occurrence of LVH in individuals with HT.

## PATIENTS AND METHODS

An observational study was carried out on 105 individuals with hypertension, characterized by a systolic blood pressure of 140 mmHg or higher and/or diastolic blood pressure of 90 mmHg or higher [14], or using an antihypertensive medicine. Additionally, 30 non-hypertensive healthy adults were included as a control group. The study was conducted at Cardiovascular Medicine Department, Faculty of Medicine, Menoufia University Hospitals from August 2022 to september 2023.

**Exclusion criteria** included poor quality echocardiographic images, patient history of coronary artery disease (CAD), significant valvular disease on echocardiography, heart failure with reduced or mildly reduced ejection fraction, chronic obstructive pulmonary disease (COPD), and regional wall-motion abnormalities on resting echocardiography.

All patients underwent history taking, physical examination, and echocardiographic testing.

### Echocardiographic assessment

Doppler and M-mode echocardiographic evaluations were part of a comprehensive transthoracic two-dimensional echocardiographic examination that all patients underwent. Patients were positioned in the left lateral decubitus position for the acquisition of



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images, and standard echocardiographic measures were collected in accordance with established protocols. With the use of 2D-guided M-mode or direct 2D echocardiography, the area length method was used to measure the IVSd, LVPWD, and left ventricular internal end-diastolic diameter (LVEDD). This allowed for the determination of the left ventricular mass (LVM). The formula, LVM = 0.8 × 1.04 [(IVS + LVEDD + PW)3-LVEDD3] + 0.6, was used to complete the computation. Then, after adjusting for body surface area, the LVMI was determined using the following formula: LVMI = LVM / body surface area.

There is a major disparity between the sexes in terms of LVMI. The typical range for men is 49-115 g/m², whereas for women it is 43-95 g/m². Levels of LVH are defined as an LBMI of 95 g/m² or more in women and 115 g/m² or above in males. To get the E/e' ratio, one divides the peak E-wave velocity from trans-mitral pulsed-wave Doppler with the peak e'-wave velocity from annular pulsed-wave Doppler tissue imaging in the apical four-chamber view. Calculating the ratio requires contrasting the mitral valve's early inflow velocities with those of the mitral annulus and the mitral annulus during early diastole. The EAT is an echo-lucent gap that forms between the right ventricle's epicardium and the parietal pericardium. The characteristic feature of this result on transthoracic echocardiography is the presence of a wide line above the free wall of the right ventricle. Finding the end-diastolic EAT thickness on the free wall of the right ventricle was the purpose of employing the parasternal long-axis (PLAX) and short-axis (PSAX) views [6].

Two views were provided: one using the aortic root as a reference point and the other using the mid-chordal area, the tip of the papillary muscle, and the interventricular septum. We took an average after merging the PLAX and PSAX picture data **(Figure 1)**.

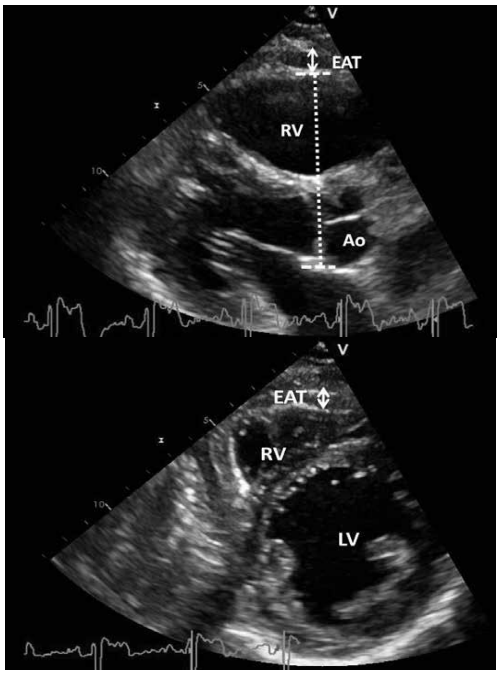


**Figure 1: Echocardiographic epicardial fat.1- PLAX view, 2- PSAX view**

**Ethical considerations**

**The study was conducted after receiving approval from the Research Ethics Committee of Menoufia University Hospitals. Written informed consent was obtained from all participants after they were briefed on the study's requirements, objectives, and potential risks. The consent form explicitly outlined their agreement to participate in the study and to allow the publication of data, ensuring the protection of their confidentiality and privacy. This research adhered to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving human subjects.**

***Statistical analysis***

The statistical study was conducted using SPSS version 26, developed by IBM Inc. of Chicago, Iowa, USA. An unpaired Student's t-test was used to compare the two groups while examining the quantitative data, which were presented using means and standard deviations (SD). The qualitative variables were measured by both frequency and percentage (%), and the data were analyzed using Chi-square test. Expected positive and negative values, as well as sensitivity and specificity, were calculated for the diagnostic test. In order to ascertain the degree of interdependence among the variables, we used the Pearson moment correlation equation, a mathematical formula representing a linear relationship between normally distributed variables. A two-tailed P value less than 0.05 was used to determine statistical significance.

**RESULTS**

The demographic data of all the participants are shown in table 1. There were 105 patients (or 77.8% of the total) in group I who served as cases, and 30 patients (or 22.2% of the total) in group II who served as controls **(Table 1)**.

**Table 1: Demographic information and medical history of the groups under study**

| | | N=135 |
|---|---|---|
| **Age (years)** | | 54.5±3.9 |
| **Gender** | **Male** | 64(47.4%) |
| | **Female** | 71(52.6%) |
| **Diagnosis** | **Cases** | 105(77.8%) |
| | **Control** | 30(22.2%) |
| **Medical history** | | |
| **DM** | | 8(5.9%) |
| **HTN** | | 105(77.8%) |
| **Family history of HTN** | | 113(83.7%) |
| **BMI** | | 29.1±2.8 9 |

DM: Diabetes Mellitus, HTN: hypertension.

Demographic data and medical history showed insignificant difference between the two groups **(Table 1)**.

**Table 2: Sociodemographic data and medical history in relation to HT in the recruited participants**

| | | Cases (N=105) | Control (N=30) | P |
|---|---|---|---|---|
| **Age (years)** | | 54.4±3.8 | 54.8±4.4 | 0.748 |
| **Gender** | **Male** | 52(49.5%) | 12(40.0%) | 0.357 |
| | **Female** | 53(50.5%) | 18(18.0%) | |
| **Medical history** | | | | |
| **DM** | | 6(5.7%) | 2(6.7%) | 0.846 |
| **Family history of HTN** | | 88(83.8%) | 25(83.3%) | 0.950 |
| **BMI (kg/m$^2$)** | | 29.3±2.8 | 28.7±2.58 | 0.294 |

DM: Diabetes Mellitus, HTN: hypertension, BMI: Body mass index.

The mean of LVMI was 110.3 ±27.5. EAT thickness from PLAX view at end diastole was 6.07±1.68. EAT thickness from PASX view at end diastole was 5.41±1.49 and EAT thickness mean was 5.7±1.49 **(Table 3)**.

**Table 3: Echocardiographic parameters in the recruited participants**

| | N=135 |
|---|---|
| **LVMI (g/m²)** | 110.3±27.5 |
| **LVESD (mm)** | 32.2±3.38 |
| **Aortic root diameter (mm)** | 30.8±2.9 |
| **LAD (mm)** | 38.04±1.6 |
| **LVEDD (mm)** | 48.4±3.9 |
| **IVSD at end diastole (mm)** | 11.7±1.8 |
| **LVPWD (mm)** | 11.8±1.8 |
| **Ejection fraction %** | 62±3.8 |
| **E/e' ratio between early mitral inflow velocity and mitral annular early diastolic velocity** | 9.2±1.74 |
| **EAT thickness from PLAX at end diastole (mm)** | 6.07±1.68 |
| **EAT thickness from PASX at end diastole (mm)** | 5.41±1.49 |
| **EAT thickness mean (mm)** | 5.7±1.49 |

LVMI: Left Ventricular Mass Index, LVESD: Left Ventricular End-Systolic Diameter, LAD = Left Atrial Diameter, LVEDD: Left Ventricular End-Diastolic Diameter, IVSD: Interventricular Septal Thickness, LVPWD: Left Ventricular Posterior Wall Thickness, EAT: Epicardial Adipose Tissue, PLAX: Parasternal Long-Axis, PASX: Parasternal Short-Axis.

In comparison to the control group, the cases group exhibited substantially higher end ventricular parameters, including as end diastolic IVSD, end EAT, end LVMI, LVPWD, and E/e' ratio. Compared to the control group, the cases group had a significantly decreased ejection %. There was no statistically significant difference between the groups when it came to LAD, aortic root diameter, or LVEDD. Compared to the control group, the group that underwent hypertrophy had a significantly elevated EAT. The hypertrophy group significantly outperformed the no hypertrophy group in various important metrics, such as the E/e' ratio, left atrial diameter, left ventricular end diastole diameter, left ventricular end diastole diameter, intraventricular septal defect (IVSD), and left ventricular wall thickness (LVWD). The ejection percentage was significantly lower in the group without hypertrophy compared to the one with hypertrophy. In terms of aortic root diameter, there was no significant difference between the two patient groups **(Table 4)**.

**Table 4: Echocardiography parameters in relation to HT in the recruited participants**

| | Cases (N=105) | Control (N=30) | P |
|---|---|---|---|
| **LVMI (g/m²)** | 119.9±22.1 | 76.7±15.1 | **<0.01*** |
| **LVESD (mm)** | 32.7±3.3 | 30.6±3.2 | **0.002*** |
| **Aortic root diameter (mm)** | 31.1±2.6 | 29.9±3.6 | 0.044* |
| **LAD (mm)** | 38.2±1.38 | 37.5±2.1 | 0.033* |
| **LVEDD (mm)** | 48.7±3.9 | 47.4±3.9 | 0.110 |
| **IVSD at end diastole (mm)** | 12.5±1.2 | 9.1±1.1 | **<0.01*** |
| **LVPWD (mm)** | 12.5±1.2 | 9.4±1.1 | **<0.01*** |
| **Ejection fraction %** | 61.5±3.5 | 64.5±3.9 | **<0.01*** |
| **E/e'** | 9.6±1.6 | 7.70±1.34 | **<0.01*** |
| **EAT thickness (mm)** | 6.2±1.4 | 4.23±0.52 | **<0.01*** |
| | **Hypertrophy (N=77)** | **No hypertrophy (N=28)** | |
| **E/e'** | 9.99±1.5 | 8.4±1.29 | **<0.01*** |
| **LVESD (mm)** | 33.3±3.2 | 30.9±2.8 | **<0.01**** |
| **Aortic root diameter (mm)** | 30.9±2.4 | 31.6±2.9 | 0.215 |
| **LAD (mm)** | 38.4±1.31 | 37.6±1.4 | **<0.01*** |
| **LVEDD (mm)** | 49.8±3.7 | 46.4±3.4 | **<0.01*** |
| **IVSD at end diastole (mm)** | 12.7±1.1 | 10.3±1.7 | **<0.01*** |
| **LVPWD (mm)** | 12.8±1.1 | 10.5±1.8 | **<0.01*** |
| **Ejection fraction %** | 61.3±3.2 | 63.4±4.3 | **<0.01*** |
| **EAT thickness (mm)** | 6.7±1.2 | 4.65±0.56 | **<0.01*** |

LVMI: Left Ventricular Mass Index, LVESD: Left Ventricular End-Systolic Diameter, LAD: Left Atrial Diameter, LVEDD: Left Ventricular End-Diastolic Diameter, IVSD: Interventricular Septal Thickness, LVPWD: Left Ventricular Posterior Wall Thickness, EAT: Epicardial Adipose Tissue, E/e': Ratio between early mitral inflow velocity and mitral annular early diastolic velocity. *: Significant.

The factors that were positively connected with each of EAT thickness, LVMI, and E/'e were LVESD, left atrium diameter, LVEDD, IVSD at end-diastole, and LVPWD at end-diastole. Additionally, significant positive relationships were found between EAT thickness and both LVMI and E/'e, and between LVMI and E/'e as well. The correlation between ejection % and

EAT thickness, LVMI, and E/'e was significantly negative. Aortic root diameter, LVMI, and E/'e did not show any statistically significant associations **(Table 5)**.

**Table 5: Correlation between EAT thickness, LVMI, E/e' with other echocardiographic parameters in recruited participants**

| | EAT thickness | | E/e' | | LVMI | |
|---|---|---|---|---|---|---|
| | r | P | r | P | r | P |
| **EAT thickness** | -- | -- | 0.645 | **<0.01*** | 0.807 | **<0.01*** |
| **E/e'** | 0.645 | **<0.01*** | -- | -- | 0.671 | **<0.01*** |
| **LVMI** | 0.807 | **<0.01*** | 0.671 | **<0.01*** | -- | -- |
| **LVESD** | 0.482 | **<0.01*** | 0.398 | **<0.01*** | 0.640 | **<0.01*** |
| **Aortic root diameter** | 0.049 | 0.575 | 0.026 | 0.766 | 0.130 | 0.133 |
| **Left atrium diameter** | 0.365 | **<0.01*** | 0.357 | **<0.01*** | 0.258 | **0.002*** |
| **LVEDD** | 0.475 | **<0.01*** | 0.312 | **<0.01*** | 0.619 | **<0.01*** |
| **IVSD at end-diastole** | 0.714 | **<0.01*** | 0.604 | **<0.01*** | 0.835 | **<0.01*** |
| **LVPWD** | 0.678 | **<0.01*** | 0.578 | **<0.01*** | 0.798 | **<0.01*** |
| **Ejection fraction** | -0.225 | **0.009*** | -0.248 | **0.004*** | -0.340 | **<0.01*** |

EAT: Epicardial Adipose Tissue, E/e': Ratio between early mitral inflow velocity and mitral annular early diastolic velocity, LVMI: Left Ventricular Mass Index, LVESD: Left Ventricular End-Systolic Diameter, LVEDD: Left Ventricular End-Diastolic Diameter, IVSD: Interventricular Septal Thickness, LVPWD: Left Ventricular Posterior Wall Thickness, r: Pearson correlation coefficient. *: Significant.

EAT thickness can significantly predict LVH in hypertensive patients (AUC = 0.927) at cut-off 5.25mm with 85.7% sensitivity, 82.1% specificity, 93% PPV, 67.6% NPV and accuracy of 83.9% **(Figure 2)**.

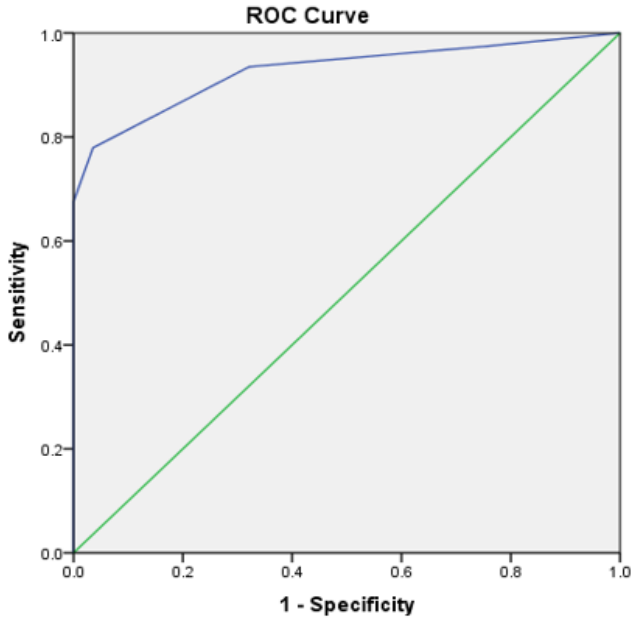


**Figure 1:** ROC curve of EAT thickness as screening for prediction of LVH.

## DISCUSSION

HT is a prevalent condition that leads to illness and death gradually. The progression of the illness is often gradual and inconspicuous. Thus, several people may be suffering from end-organ damage when they are diagnosed, and a large number of those people may have more than one disability at the same time [14].

Earlier research examined the effects of HT, insulin resistance, and aberrant fat accumulation on variations in peak systolic circumferential strain. Cardiac MRI tagging and MRI measurements of visceral and epicardial fat were used for this purpose. A correlation between early HT and increased EAT thickness was discovered by **Sironi *et al.*** [15]. The association between EAT thickness and HT was confirmed by **Sengul *et al.*** [11].

LVMI, LVESD, LVPWD, and E/e ratio were considerably greater in the HT group compared to the control group in our research. The ejection fraction was considerably lower in the HT patients group compared to the control group. LVH develops in many people with long-standing HT prior to the appearance of clinical symptoms. LVH is a significant independent risk factor for cardiovascular problems in asymptomatic individuals with HT, leading to increased morbidity and death [16]. This slightly agreed with **Wachtell *et al.*** [17] who hypothesized that HT-induced alterations in LVH were due to angiotensin II release.

Our investigation found that the EAT thickness was substantially greater in the cases group than in the control group. **Gastaldelli and Basta** [18] found a link between elevated blood pressure and the formation of ectopic fat. Visceral fat tissue and EAT were both individually linked to average blood pressure in the research.

In our study, The E/e' ratio, LVESD, left atrium diameter, LVEDD, IVSD at end-diastole, and LVPWD were all considerably greater in hypertensive individuals with hypertrophy compared to those without hypertrophy. When comparing the groups with and without hypertrophy, the ejection% was significantly lower in the former. When comparing the two groups of HT participants, researchers found no statistically significant change in aortic root diameter.

In our study, EAT thickness was significantly higher in hypertrophy group compared to no hypertrophy group. This is in harmony with **Şeker *et al.*** [19] who reported that, when contrasted with the control, normal geometry, and concentric remodeling groups, patients exhibiting hypertrophic geometric patterns, such as eccentric and concentric hypertrophy had greater EAT thickness values.

In our study, EAT thickness can significantly predict LVH in hypertensive patients (AUC = 0.927) at cut-off 5.25 mm with 85.7% sensitivity, 82.1% specificity, 93% PPV, 67.6% NPV and accuracy of 83.9%. **Natale *et al.*** [20] found that LVMI, diastolic dysfunction, increased carotid stiffness, and intima-media thickness (IMT), were all seen in patients with

EAT thickness more than 7 mm. The results were linked to the thickness of the epicardial fat. When it comes to detecting CAD, EAT is superior than other areas' visceral adipose tissues [21]. **Cavalcante *et al.*** [22] showed that EAT, even after controlling for other possible risk factors, is a predictor of LV diastolic dysfunction in apparently healthy overweight persons.

Our investigation found positive relationships between EAT, LVMI, and E/'e with several cardiac measurements in recruited individuals, including LVESD, left atrium diameter, LVEDD, IVSD at end-diastole, and LVPWD at end-diastole. Positive correlations were found between EAT thickness and E/'e as well as LVMI. Additionally, a positive correlation was identified between LVMI and E/'e. Notably, a negative correlation was found between each of EAT thickness, LVMI and E/'e with ejection fraction. Aortic root diameter was not correlated with EAT, LVMI, or E/ 'e. The findings of **Eren *et al.*** [23] about the correlations between EAT thickness and LVMI are in line with those of the prior research.

Earlier research using echocardiography and autopsy revealed a strong association between EAT thickness and LVH [24,25].

In cardiovascular disease, fat tissues enlarge, become oxygen-deprived and dysfunctional, and attract cells that engulf foreign particles, resulting in a decrease in beneficial cytokines, ultimately leading to an elevation of harmful adipocytokines and compromised heart function. **Mukherjee *et al.*** [9] discovered that elevated epicardial fat identified using computed tomography, was linked to left ventricular diastolic dysfunction, regardless of other common risk variables such age, gender, diabetes, HT, or abdominal obesity.

Abdominal obesity has a same embryological origin with eating problems. There are several areas around the heart where fat may accumulate. Pericardial fat is located between the parietal and visceral layers of the pericardium, whereas epicardial fat is found between the myocardium and the visceral layer. The EAT is a specific fat depot often associated with visceral fat rather than overall body fat. It is connected to the heart by a shared blood circulation system [26].

The study's limitations include its cross-sectional design, which hinders the establishment of a causal association between EAT thickness and LVH. The control group has a tiny sample size, potentially affecting the generalizability of the results. The research used echocardiography for heart measures, which may have limitations when compared to more modern imaging methods.

## CONCLUSIONS

Hypertensive patients with LVH had a notably greater EAT thickness in comparison to hypertensive patients without hypertrophy and persons in good health. The results emphasize EAT's potential as an indicator of ventricular remodeling and hypertrophy in HT.


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- **Conflict of Interest:** Nil.


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