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Original article

Effect of epicardial adipose tissue on diastolic functions and left atrial dimension in untreated hypertensive patients with normal systolic function

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ABSTRACT

Background: Adipose tissue is the source of many adipokines affecting the cardiovascular system either locally or systemically. Although hypertension is one of the most important factors in diastolic dysfunction (DD), the exact cause of this relationship is unknown. There is no specific study in the current literature regarding the association of epicardial adipose tissue (EAT) with left ventricular DD in patients with essential hypertension.
Methods: The present study was cross-sectional and observational, including 127 patients with untreated hypertension who underwent a complete transthoracic echocardiographic examination as well as measurements of EAT and diastolic parameters.
Results: EAT was significantly correlated with left atrial dimension, DD parameters, and left ventricular (LV) mass as well as age and blood pressure measurements. EAT was also correlated with Framingham risk score ($p < 0.001$). Age and EAT were significantly increased in patients with high grades of DD compared to those with low values ($p < 0.001$ and $p = 0.001$, respectively). Linear regression analyses revealed EAT as an independent predictor of all DD parameters. The area under the curve values of EAT were similar to age and higher than those of LV mass and mean BP for both the presence of DD and grade two DD.
Conclusion: Based on our findings, increased EAT may be associated with diastolic dysfunction and left atrial dilatation due to local or systemic effects in untreated hypertensive patients. This relationship is independent of and stronger than abdominal obesity, implicating the clinical importance of measuring EAT thickness.

Introduction

Evaluation of diastolic function in every patient undergoing echocardiography is important clinically [1]. Diastolic dysfunction, as seen in patients with hypertension (HT), diabetes mellitus, and ageing, increases the risk of heart failure and cardiovascular mortality, even if in at an asymptomatic or preclinical stage [2]. Although identifiable by Doppler echocardiography, there is no definitive treatment for diastolic dysfunction. Pathophysiological research implicates an active fibrotic process, which might lead to new therapeutic options in future [3,4].

Obesity is one of the most important features of diastolic dysfunction. Adipose tissue is the source of many adipokines affecting the cardiovascular system either locally or systemically [5]. Fat, mainly accumulated in subcutaneous tissue, is also deposited around visceral organs in the abdominal or thoracic region [6]. Fat is accumulated in three different localizations around the heart: cellular, epicardial, and pericardial. Epicardial fat is located between myocardium and visceral layer of pericardium. Pericardial fat is located between the visceral and parietal layers of pericardium [7]. Epicardial adipose tissue (EAT) thickness measured by echocardiography has a good correlation with abdominal adiposity measured by computed tomography (CT) and magnetic resonance imaging (MRI) [8,9]. Konishi et al. demonstrated a significant correlation between pericardial fat and diastolic dysfunction in patients with normal systolic function [10].

Although HT is one of the most important factors in diastolic dysfunction, the exact cause of this relationship is still unknown [11]. An increased epicardial fat mass may deteriorate cardiac function either locally or systemically by passive or active effects. There is no specific study in the current literature regarding the association of EAT with left ventricular diastolic dysfunction in patients with essential HT. Therefore, we planned this study in order to clarify this relation.

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Methods

Patient population and study protocol

The present study was cross-sectional and observational, including 127 patients with untreated HT who underwent a complete transthoracic echocardiographic examination as well as measurements of EAT and diastolic parameters. All data were prospectively collected. Resting blood pressure (BP) values were obtained at a physician's office and echocardiography room by traditional auscultatory method using a sphygmomanometer. Patients were advised to refrain from smoking or consumption of coffee or tea, and physical exercise, 30 min prior to the measurement. Before measurement, patients were seated to rest for 5 min. Two separate measurements were averaged to determine office blood pressure.

Patients with previous coronary artery disease (CAD) (17 patients), diabetes mellitus (20 patients), left ventricular systolic dysfunction (14 patients), secondary HT (2 patients, renal artery stenosis), moderate-severe valve disease (15 patients; 10 aortic stenosis, 5 mitral insufficiency), atrial fibrillation (18 patients), symptoms of CAD and equivalent findings on exercise electrocardiography and perfusion scan, or 24-h rhythm electrocardiography (20 patients; 15 positive exercise tests and 5 positive single photon emission computed tomography scans), and patients previously treated for HT (22 patients) were excluded.

Baseline characteristics of the patients were recorded. HT was defined as the documentation of blood pressure more than 140/90 mmHg. Patients who were using tobacco products on admission and those who had quit smoking within the last year were considered as smokers.

The study was performed in accordance with the principles stated in the Declaration of Helsinki and approved by the local ethics committee.

Routine measurements

Blood samples were drawn by venipuncture to measure routine blood chemistry parameters after fasting for at least 8 h. Fasting blood glucose, serum creatinine, 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). Body mass index (BMI) was determined by the following formula: BMI = weight (kg)/height^2 (m).

Echocardiography

Patients were imaged in the left lateral decubitus position with commercially available systems with a GE-Vingmed Vivid S5 (GE-Vingmed Ultrasound AS, Horten, Norway) using a 2.5–3.5 MHz transducer. Diastolic parameters were measured according to guidelines by the American Society of Echocardiography [1]. Early (E) and late mitral inflow (A) velocities, lateral and medial mitral annular velocities (Em, Am), isovolumic relaxation time (IVRT), isovolumic contraction time (IVCT), ejection time (ET), deceleration time (DT), and Tei index (also known as myocardial performance index) [MPI = (IVRT + IVCT)/ET] were calculated according to relevant guidelines [1]. Left ventricular ejection fraction was measured by Simpson's method [12]. Maximum and minimum left atrial (LA) volumes were measured by the method of discs from the apical four-chamber view at end-systole and end-diastole [13]. End-systolic measurements were obtained from the frame preceding mitral valve opening.

Left ventricular mass (LV mass, in g) was calculated according to the following formula: $1.04 \times [(\text{LVEDD} + \text{IVSD} + \text{PWD})^3 - \text{LVEDD}^3] - 13.6$ (diameter in cm) [14], where LVEDD = left ventricular end-diastolic dimension; IVSD, interventricular septal thickness at diastole; PWD, posterior wall thickness at diastole.

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 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 visceral pericardial layer and myocardium on 2-dimensional echocardiography, was measured perpendicularly in front of the right ventricular free wall at end-diastole [8,15]. We magnified each still image for better visualization and accurate measurement

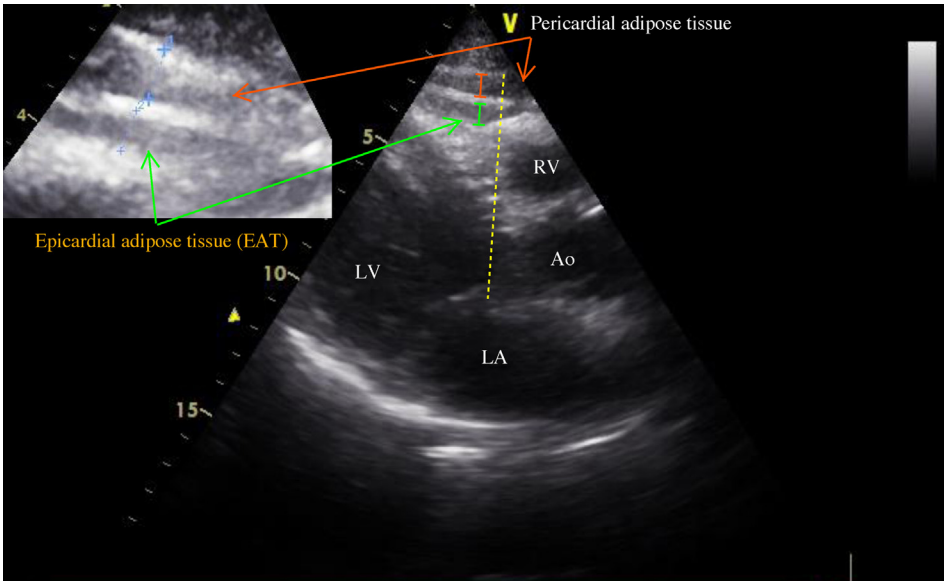

Fig. 1. Measurement of epicardial fat thickness by echocardiography. EAT, identified as an echo-free space between the myocardium and visceral pericardium from the parasternal long-axis view on 2-dimensional echocardiography, was measured perpendicularly in front of the right ventricular free wall at end-diastole. LV, left ventricle; LA, left atrium; RV, right ventricle; Ao, aorta.