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Original Article

Epicardial fat thickness: A surrogate marker of coronary artery disease – Assessment by echocardiography

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ABSTRACT

Objective: Epicardial fat is considered as indicator of cardiovascular risk. Several studies have tested the association between epicardial fat thickness (EFT) and coronary artery disease. The aim of our study is to test the hypothesis that echocardiographic EFT is a marker of coronary artery disease.

Methods: One hundred and ten patients (70 males and 40 females with mean age of 51.5 $\pm$ 10.6 and 52.6 $\pm$ 9.6, respectively) admitted for coronary angiogram underwent assessment of epicardial fat thickness by echocardiography. Routine clinical examination, evaluation of risk factor profile, and anthropometric variables were also done. Epicardial fat thickness was measured on the free wall of right ventricle in parasternal long- and short-axis views at end-systole for 3 cardiac cycles.

Results: Mean epicardial fat thickness in angiographically normal patients and acute coronary syndromes were 4.4 $\pm$ 1.2 and 6.9 $\pm$ 1.9, respectively. Epicardial fat thickness in males and females were not statistically different. Burden of coronary arterial lesions denoted by Gensini score shows linear association with epicardial fat thickness and the severity of the coronary disease.

Conclusion: Epicardial fat is independently and linearly associated with CAD and its severity.

1. Introduction

The heart and vessels are surrounded by layers of adipose tissue, which is a complex organ composed of adipocytes, stromal cells, macrophages, and a neuronal network, all nourished by a rich microcirculation. The layers of adipose tissue surrounding the heart can be subdivided into intra- and extrapericardial fat. Their thicknesses and volumes can be quantified by echocardiography and computed tomography or magnetic resonance imaging, respectively.[1,2] The term extrapericardial fat defines thoracic adipose tissue external to the parietal pericardium. It originates from primitive thoracic mesenchymal cells and thus derives its blood supply from noncoronary sources.[3] Intrapericardial fat is further subdivided into epicardial and pericardial fat. Anatomically, epicardial and pericardial adipose tissues are clearly different.[3,4] Epicardial fat is located between the outer

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wall of the myocardium and the visceral layer of pericardium.

Pericardial fat is anterior to the epicardial fat and therefore located between visceral and parietal pericardium. Much of the importance within the epicardial fat is its anatomical closeness to the myocardium and the fact that the two tissues share the same microcirculation.

The epicardial fat layer originates from mesothelial cells and hence obtains its vascular supply from the coronary arteries. It has been shown that epicardial fat is metabolically active and a source of several adipokines and inflammatory cytokines, and there seems to be potential interactions through paracrine or vasocrine mechanisms between epicardial fat and myocardium.[5–8] This is clearly not true for the pericardial fat. Gensini scoring system is a good tool to quantify coronary stenosis.[9] Some studies correlated epicardial fat thickness with severity of coronary stenosis with conflicting results. Therefore, the aim of our study is to evaluate the hypothesis that there is a correlation between echocardiographic EFT thickness and severity of coronary artery disease.

2. Methods

2.1. *Study design*

The study was designed as an observational cross-sectional study. All the participants were informed of its objectives before the study and signed letter of consent in accordance with the Helsinki Declaration Standards was obtained. Our study cohort consisted of 110 patients, who underwent coronary angiography for suspected coronary artery disease.

2.2. *Inclusion criteria*

Patients who underwent coronary angiogram for suspected coronary artery disease and did not meet the exclusion criteria.

2.3. *Exclusion criteria*

Patients who had chest deformities, chronic lung disease, poor echo window, pericardial and/or pleural effusion on transthoracic echocardiography, previous coronary artery bypass graft surgery (CABG), percutaneous coronary intervention (PTCA), and chronic kidney disease were not included in the study. All patients underwent detailed history, clinical examination, anthropometric measurement, routine biochemistry, ECG, and transthoracic echocardiography. Some of the patients had already undergone exercise treadmill test.

2.4. *Variables*

Body mass index (BMI) was calculated as body weight in kilograms and divided by height squared. Obesity was defined as having a BMI ≥30 kg/m^2. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or requirement for antihypertensive medication.[10] Diabetes mellitus was defined according to the criteria of the American Diabetes Association or requirement for insulin or oral hypoglycemic drugs.[11] Hyperlipidemia was defined as total cholesterol higher than 220 mg/d or triglycerides ≥150 mg/dl.[12]

2.5. *Echocardiographic measurement*

Transthoracic echocardiography provides a reliable measurement of EFT.

Transthoracic echocardiography for the purpose of measuring epicardial fat thickness was done within 3 days after coronary angiography. Echocardiograms were performed with a PHILIPS HD 7 instrument according to standard techniques, with subjects in the left lateral decubitus position. Echocardiographic images were recorded onto a computerized database and videotape. The offline measurement of epicardial fat thickness was performed by 2 cardiologists who were unaware of the clinical and angiographic data. We measured epicardial fat thickness on the free wall of right ventricle from the parasternal long- and short-axis views, since it allows accurate assessment.[13,14] We preferred to measure at end-systole since compression of epicardial fat layer occurs during diastole.[6] Epicardial fat was identified as an echo-free space between the myocardium and visceral pericardium. The epicardial fat thickness was measured perpendicularly on the free wall of the right ventricle at end-systole for 3 cardiac cycles. The measurement was performed at a point on the free wall of the right ventricle where the fat thickness was highest (Fig. 1). The average value from 3 cardiac cycles for each echocardiographic view was tabulated and used for the statistical analysis.

2.6. *Coronary angiography*

In a fasting state, coronary angiography was performed using the Judkins' technique, by the femoral or radial artery approach. The severity of coronary atherosclerotic lesions was evaluated from at least 3 projections in all the patients by modified Gensini scoring system. According to this scoring system, coronary arterial system was divided into 8 segments and the most severe luminal narrowing in each coronary segment was graded with 1 to 4 points (between 1% and 49% – 1 point; 50% and 74% – 2 points; 75% and 99% – 3 points; 100% – 4 points). Each patient was evaluated with a total score between 0 and 32 points. Each point was multiplied with separate coefficients based on vessel and its segments; these coefficients were 5 for left major coronary artery, 2.5 for proximal LAD, 1.5 for middle LAD, 1.5 for distal LAD, 1 for diagonal LAD, 2.5 for proximal circumflex artery, 1 for marginal obtuse and posterolateral branch, 1.5 for right proximal coronary, 1 for posterior descending artery, and 0.5 for others. The points were added and total Gensini points were calculated for each patient.[5] Patients who had normal coronary angiograms and negative treadmill test were used as controls.

2.7. *Statistical analysis*

Statistical analysis was done using SPSS 13.0 for windows. Continuous variables are expressed as means ± SD and categorical variables as absolute numbers and percentages. Comparisons of continuous variables were performed using