The Association of Epicardial Fat Thickness to Cardiovascular Clinical Outcomes

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Received Date: 06 Oct 2016
Accepted Date: 13 Oct 2016
Published Date: 14 Oct 2016

INTRODUCTION

Epicardial fat is a visceral fat deposit which is located between the heart and the pericardium sharing many of the patho-physiological properties of other visceral fat deposits. There is recognition of three functional types of adipose tissue. The first type, the white adipose tissue consists of large unilocular adipocytes whose primary function is to store energy in the form of triglyceride. The second type, the brown adipose tissue which contains multicellular adipocytes with large numbers of mitochondria, this is most commonly found in young mammals and rodents. Its primary function is to generate heat via uncoupled oxidative phosphorylation. Third, the beige adipose tissue is form of brown adipocytes that arises within the white adipose depots and also has thermogenic capacity [1-7]. It is important to differentiate between the adipose tissue located on the outer surface of the fibrous pericardium (paracardial fat) from the one in the inner surface of the visceral pericardium (epicardial fat) which is in direct contact with the myocardium and the epicardial vessels, since they differ in their biochemical, molecular and vascular nutritional properties. The paracardial fat is nourished by the peri- cardiophrenic artery, a branch of the internal thoracic artery, whereas the epicardial fat is nourished by the coronary arteries [8-11]. The epicardial fat is more prominent in the atrioventricular and interventricular grooves and right ventricular lateral wall. Adipocyte infiltration into the myocardium wall as well as triglyceride infiltration into myocytes may also occur. The paracardial fat has been also called intrathoracic, mediastinal or pericardial. In addition, some other groups treat these different fat deposits as a single compartment, calling it pericardial fat [12-16]. Since several studies have observed a moderate association between EFT and cardiovascular clinical outcomes, it is important to analyze this relationship at the light of medicine based evidence.

Epicardial fat thickness (EFT) can be measured by different imaging modalities. Magnetic resonance imaging (MRI) is considered the gold standard for the assessment of total body fat and reference modality for the analysis of ventricular volumes and mass, thus making it a natural choice for the detection and quantification of EFT [17-19]. For purposes of cardiovascular risk stratification, measurement of EFT using echocardiography has generally been the study of choice, due to its lesser cost, ease of use, and absence of radiation. By echocardiography, measurements of the right ventricular free wall from both parasternal longitudinal and transverse parasternal views should be performed using the mean of three consecutive beats. These echocardiographic measurements show good correlation with the values found on MRI (r = 0.91, p = 0.001) [19]. There are some controversial issues in the EFT measurements by echocardiography. For example, there are some inconsistencies in the site of measurement due to spatial variations of the echocardiographic window, especially along the great vessels and the right ventricle. In addition, it is uncertain yet which moment of the cardiac cycle is the most suitable for measuring EFT by echocardiography. Some recommend the measurement during systole to prevent possible deformation by compression of the epicardial fat during diastole (8). On the other hand, other researchers prefer measurements in diastole to coincide with measurements of other imaging modalities like CT scans and MRI [19-21].

Although there are some studies that suggested higher cutoffs, measurements greater than 5 mm should represent a relevant cutoff to define increased EFT in low-risk population. The mean value described for EFT in systole was 6.8 mm (1.1 to 22.6 mm) [22]. In obese patients, the mean value of EFT was 9.5 mm (7.0 to 20.0 mm) for men, and 7.5 mm (6.0 - 5.0 mm) for women [23]. When measurements were performed in diastole in patients who underwent coronary angiography, the mean value was 6.4 mm (1.1 to 16.6 mm) [24]. It was also demonstrated in asymptomatic patients an EFT with a mean value of 4.7 ± 1.5 mm [25]. According to current knowledge, an EFT greater than 5 mm, or an epicardial fat volume greater than 125 mL or 68 mL/m² may be considered abnormal.

Several studies have observed a moderate association be-
between EFT and clinical outcomes. In a case-control study of incident cases during a four-year follow-up, Cheng et al. [26] compared 58 patients with major adverse cardiac events with 174 controls free of events. The patients were matched by sex and a propensity risk score that included age, risk factors and coronary calcium score. The researchers demonstrated a higher risk of events (OR = 1.74, 95% confidence interval [95% CI]: 1.03-2.95) with a two-fold increase in epicardial fat. In the MESA (Multi-Ethnic Study of Atherosclerosis) cohort, Ding et al. investigated a random sample of 998 participants and the 147 individuals who developed coronary events [16]. Epicardial fat was associated with coronary artery disease (relative risk for increase of one standard deviation = 1.26, 95% CI: 1.01-1.59) even after adjustment for cardiovascular risk factors [16]. Moreover, associations between EFT and coronary artery calcification were found both in the Framingham and in the MESA studies [15, 27]. It is well known that coronary artery calcification has been used as a marker of subclinical atherosclerosis in representative population samples. Additionally, it is speculated that the increase in EFT and fatty infiltration of the myocardium may cause other deleterious cardiovascular effects, such as interfering with diastolic relaxation, affecting the cardiac conduction system and predisposing to atrial fibrillation. EFT is inversely associated with ejection fraction and left ventricular mass [28-30].

The EFT could add significant incremental values beyond the conventional clinical and echocardiography parameters in prediction of adverse cardiovascular outcomes. EFT has been proposed to influence the development of coronary atherosclerosis owing to its endocrine and paracrine activity by secreting anti-inflammatory and pro-inflammatory cytokines and chemokines [31-35]. In patients with documented coronary artery disease, Jeong et al. demonstrated that EFT was correlated significantly with its severity of atherosclerosis [36]. Several studies also showed that age and body mass index were associated significantly with EFT. Increased EFT is strongly associated with diabetes mellitus, cardiovascular disease, visceral obesity, subclinical atherosclerosis at multiple locations, and the metabolic syndrome [34-39]. EFT is also increased in subjects with atrial fibrillation and correlates with atrial fibrillation severity and its recurrence after catheter ablation [40-42]. In non-AF patients, increased EFT is shown to be positively associated with the severity of coronary artery disease and left ventricular diastolic dysfunction and is a useful parameter in predicting adverse cardiovascular events [43-46]. An association between EFT and incident myocardial infarction and cardiovascular risk factors were observed in the general population. In the cross-section study by Aki et al. the EFT in patients diagnosed with ischemic stroke was found to be higher than those in healthy controls [35]. Therefore, EFT could provide incremental value for cardiovascular outcome prediction over traditional clinical and echocardiographic parameters.

REFERENCES


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Citation: Centurión OA, Battilana-Dhoedt JA and Garcia-Bello LB. (2016). The Association of Epicardial Fat Thickness to Cardiovascular Clinical Outcomes. M J Cardiol. 1(2): 007.


