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Epicardial adipose tissue and coronary artery disease: an article review

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ABSTRACT

Adipose tissue surrounding the heart may contribute in the progression of coronary atherosclerosis due to its proximity to the coronary arteries. In addition, epicardial adipose tissue has paracrine and endocrine functions. It can secrete numerous bioactive molecules. Most previous studies examined the relation between coronary artery disease and epicardial adipose tissue have used echocardiography and have reported controversial results, probably due to differences in measurement techniques and study populations. This study aimed to give a brief review on the value of echocardiographic assessment of epicardial adipose tissue in the prediction of coronary artery disease severity.

Epicardial adipose tissue, easily and non-invasively evaluated by transthoracic echocardiography, can be considered as an adjunctive marker to classical risk factors despite all the limitations. Moreover, it might be recommended as a useful quantitative screening examination for the prediction of the presence and the severity of coronary artery disease and the extent of atherosclerosis.

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Introduction

Recent studies have suggested that fat disposition in visceral organs and epicardial tissue could serve as a predictor of the severity of coronary artery disease (CAD) and the extent of coronary artery atherosclerosis. Most importantly, a local inflammatory effect has been proposed as EAT directly surrounds the coronary arteries (1,2).

To date, the relation between abdominal adipose tissue and predisposition to coronary artery disease has been well established.

Epicardial adipose tissue (EAT), which is thought to be a component of visceral adiposity, may contribute in the progression of coronary atherosclerosis due to its proximity to the coronary arteries (3,4). EAT has paracrine and endocrine functions. It can secrete numerous bioactive molecules including adiponectin, resistin and inflammatory cytokines. Interestingly, inflammatory mediators originating outside the coronary artery can also induce compositional changes in the inner layer of intima (5). Therefore, increased EAT might act as an inflammatory organ, which affects vascular function (6).

Most previous studies examining the relation between CAD and EAT have used echocardiography and have reported controversial results, probably due to differences in measurement techniques and study populations.

This study is a brief review on the value of echocardiographic assessment of EAT in the prediction of CAD severity.

Literature review

Wang et al. recently tested the hypothesis that epicardial adipose tissue (EAT) can be a marker

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of severe coronary artery disease in patients with acute myocardial infarction. In their work, 373 cases, whom underwent coronary angiography, were classified into 2 groups by SYNTAX score. EAT was measured by transthoracic echocardiography. They obtained a significantly greater EAT in the high-score group than in normal group. In addition, EAT had a positive correlation with SYNTAX score (7).

In a case-controlled study conducted on 124 patients, aged 40 to 91 years and a matched set of 62 symptomatic and 62 asymptomatic patients, epicardial fat thickness (EFT) was measured by transthoracic echocardiography. They reported that patients with increased EFT were at an elevated risk for developing angina, recurrent hospitalization and adverse outcomes, even with normal epicardial coronary arteries (8).

Ahn et al. studied the relationship between thickness of EAT on the free wall of the right ventricle and CAD in patients undergoing conventional coronary angiography due to chest pain. A positive correlation was found between EAT thickness and significant coronary stenosis and with the number of coronary arteries with significant stenosis (2,9).

In this study, EAT thickness was associated with abdominal Visceral Adipose Tissue(VAT), age, waist circumference, body mass index and C reactive protein. Patients with unstable angina had thicker EAT than those with stable angina or atypical chest pain (9).

Djaberi et al., in 2008, aimed at investigating the relation between EAT volume assessed by multislice computed tomography (MSCT) and presence of coronary atherosclerosis. Patients' mean age was 56.12 years with males accounting for 55% of the cases (2).

Their results showed a significantly larger mean EAT volume in patients with CAC (coronary artery calcium) and/or coronary atherosclerosis on MSCT angiogram compared to those with a CAC score>10 and/or angiographically normal coronaries. Interestingly, EAT volume was revealed to be an independent predictor for coronary atherosclerosis (2).

In another study conducted in 2012, patients in whom CAD was confirmed by multidetector computed tomography (MDCT), had a significantly greater EAT volume compared to those without CAD. Furthermore, EAT thickness was associated with metabolic syndrome whereas no correlation was found with BMI and EAT thickness>2.4 mm was the strongest independent predictor of significant CAD based on multivariate analysis. Bacher et al. concluded that an increased EAT volume might act as a marker for severe atherosclerosis in addition to be a risk factor for significant CAD in the general population (6).

In a study by Altun et al. on sixty-five Acute Coronary Syndrome (ACS) patients, mean age=57.4±12.2 years, who underwent coronary angiography, EAT thickness had a significant relationship with angiographic severity, whereas no such correlation was achieved with clinical prognosis. Therefore, they suggested echocardiographic EAT thickness as a predictive factor of severity in ACS patients (10).

Shirmani et al., in 2012, classified 315 cases underwent coronary angiography into two groups imcluding normal and CAD. EFT was quantified by echocardiography. They reported EFT thickness as an independent predictor of CAD among other well-known risk factors (11).

On the other hand, in a study conducted by Gorter et al. on a group of patients consisting of 70% men with the mean age of 61±6 years, EAT and pericoronary fat, measured by cardiac CT, were not related to severity of coronary atherosclerosis and extent of CAC in patients with suspected CAD. However, EAT volume and pericoronary fat thickness were related to a larger number of stenotic coronary vessels and more severe CAC in patients with a low BMI (12).

Chaowalit et al., in 2006, also studied 139 patients who were referred for conventional coronary angiography. No significant correlation was found between EAT thickness and the number of atherosclerotic coronary segments (2,13).

Discussion

Central obesity (visceral adipose tissue) has a strong correlation with the development of metabolic syndrome and coronary artery disease (14). Respectively, fat surrounding coronary arteries has been related to waist circumference and BMI, which are both indicators of high cardiovascular risk (9). Several studies have recently proposed echocardiographic assessment of EAT as an easy and reliable imaging indicator of VAT (14), but the association between EAT and coronary artery disease has not been well documented.

Echocardiography is a simple, noninvasive and easily accessible device for measuring fat around the heart; however, it cannot provide an adequate window of all cardiac segments and is highly dependent on acoustic windows, which are often not qualified enough for detailed assessments in obese patients (14). Moreover, echocardiographic measurement of EAT quantity is limited to the assessment of EAT thickness on the free wall of the right ventricle. On the other hand, it has been shown that the distribution pattern of EAT around the myocardium may be remarkably different among patients (2,13). This may be the

reason for the discrepancy in the results reported by various studies.

Bachar et al. study was the first to address the correlation between EAT and significant CAD in asymptomatic patients. It revealed that EAT and metabolic risk have a stronger correlation in comparison to that of other indices of systemic obesity such as BMI. Moreover, because all studied cases had more than one risk factor for atherosclerotic CAD, they suggested that EAT can be considered as a predictive factor for CAD especially before symptoms appearance in high risk patients (6). EAT was significantly correlated with CAD. Therefore, the results suggested that EAT might contribute to the development of atherosclerotic plaques. This hypothesis needs to be further addressed in future studies.

Conclusion

Taken together, despite all the limitations aforementioned and considering the available literature to date, EAT, easily and non-invasively evaluated by transthoracic echocardiography, can be considered as an adjunctive marker to classical risk factors. Moreover, it might be recommended as a useful quantitative screening examination for the prediction of the presence and the severity of CAD and the extent of atherosclerosis.

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Conflict of Interest

The authors declare no conflict of interest.

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