Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.JournalofCardiovascularCT.com

Review Article

Quantification of epicardial fat by computed tomography: Why, when and how?

Mohamed Marwan MD, Stephan Achenbach MD*

Department of Cardiology, University of Erlangen, Ulmenweg 18, 91054 Erlangen, Germany

ARTICLE INFO

Article history:
Received 21 August 2011
Received in revised form
24 August 2012
Accepted 6 January 2013

Keywords:
Epicardial fat
CT

ABSTRACT

In the past decade, interest has grown in the relation between epicardial fat and cardiovascular disease. Several imaging modalities such as echocardiography, computed tomography, and magnetic resonance imaging can be used to quantify epicardial adipose tissue. Computed tomography provides high spatial resolution and true volume coverage of the heart; therefore, it constitutes an attractive approach to quantifying epicardial fat. An increasing body of evidence has been accumulated which shows a relation between epicardial fat volume and coronary atherosclerosis, cardiovascular outcomes, and even non–atherosclerotic heart disease such as atrial fibrillation. The association of increased epicardial fat volume with cardiac disease remains significant even after correction for weight, body mass index, and traditional cardiovascular risk factors. The mechanisms have not been reliably identified, but metabolic properties of epicardial fat may play a role. At the present time, epicardial fat quantification is not included in recommended algorithms for risk stratification. However, the available data are intriguing enough to warrant further research.

1. Introduction

Within the pericardial sac, adipose tissue surrounds the myocardium. Epicardial fat creates a smooth surface and, along with the pericardial fluid, facilitates motion of the heart relative to the pericardium. Interestingly, the amount and distribution of epicardial fat varies widely between persons and are not strictly related to body mass index (BMI) or obesity. The coronary arteries are embedded in epicardial fat over most of their course. In fact, a growing body of evidence indicates that there is a significant relation between epicardial fat and coronary artery disease (CAD).[1–6] Even for non–atherosclerotic diseases, such as atrial fibrillation, a correlation to epicardial fat has been proposed.[7] This review provides an overview of the current literature about epicardial fat, its quantification, and its relation to cardiac disease.

2. Definition of epicardial fat

The terminology used to define fat deposits surrounding the heart in the current literature is diverse and, to some extent, confusing. It includes terms such as “epicardial,” “pericardial,” “paracardiac,” and “intrathoracic” fat.[2,3,5,8–13] The term pericardial fat is frequently used in most of the published literature and refers to adipose tissue enclosed within the pericardial sac. It is hence the fat contained in the pericardial space, between the serous epicardium and the fibrous

Conflict of interest: Stephan Achenbach received research support from Bayer Schering Pharma and Siemens.

* Corresponding author.
E-mail address: mohamedmarwan@yahoo.com (M. Marwan).

1934-5925/$ – see front matter
http://dx.doi.org/10.1016/j.jcct.2013.01.002

pericardium. It is important to mention that, although the term pericardial fat is widely used in the imaging literature, the more accurate term would be epicardial fat, given its location on the epicardial surface of the heart. Paracardiac fat is the term most frequently used for adipose tissue on the outer surface of the pericardial sac, and both fat deposits together are usually referred to as intrathoracic fat (Fig. 1).

3. Epicardial fat: A metabolically active fat depot?

The importance of differentiating adipose tissue within and outside the pericardium lies in the fact that they are anatomically, embryologically, and biochemically different.[11,12] Furthermore, convincing data indicate that epicardial fat is metabolically active and functions as a source of several adipokines, which leads some researchers to assume that, through paracrine or vasocrine mechanisms, there may be a direct interaction between epicardial fat on the one hand and the closely related coronary arteries and myocardium on the other hand.[11,12] The endocrine potential of visceral adipose tissue such as epicardial fat includes the secretion of several proinflammatory and anti-inflammatory mediators and cytokines such as adiponectin, interleukin 6, and tumor necrosis factor α (TNF-α).[1,14–21] The amount of circulating plasma adiponectin decreases with increasing amounts of visceral fat,[22,23] which subsequently leads to an increase in TNF-α levels and hence to a local increase of inflammation.[18] In this fashion, a mismatch of proinflammatory and anti-inflammatory mediators and cytokines secreted by epicardial fat is suspected to have local influence on the embedded coronary arteries[24] (Fig. 2).

4. Epicardial fat and relation to coronary atherosclerosis

Because of the close proximity of epicardial fat to the coronary arteries, the hypothesis of a direct mediator-related role in the development and progression of coronary atherosclerosis has been generated, and it has received increasing support over the past decade through 2 lines of evidence. First, the amount of epicardial fat seems to be relatively independent from overall adipose body tissue but, like intra-abdominal visceral fat, shows an association to atherosclerosis. Second, histopathologic studies indicate specific patterns of mediator secretion by pericardial fat, but not by fatty tissue in remote locations, which correlate to atherosclerosis. For example, in a landmark study, Mazurek et al[16] analyzed epicardial fat and subcutaneous fat of the lower extremity in obese patients referred for coronary artery bypass grafting. They could show that the expression and secretion of several inflammatory mediators as well as chronic inflammatory cell infiltration with macrophages, lymphocytes, and basophils were increased in epicardial adipose tissue compared with subcutaneous fat.[16] Other studies found an increase of CD45 mRNA expression in the epicardial fat of subjects with CAD, representing elevated macrophage infiltration,[14] and an increase of mast cells in the adventitia of coronary lesions.[1,15] Interestingly, in a recent immunohistochemical study by Konishi et al,[25] more leucocyte common antigen–positive cells were observed in pericoronary fat of autopsy cases with CAD compared with patients without coronary disease, suggesting inflammation of pericoronary fat in the former group.[25] In a recent study by Hirata et al,[26] polarization of macrophages in epicardial fat of patients with CAD was observed compared with patients without CAD. They could show a change of the ratio of M1/M2 macrophages with abundance of the proinflammatory M1-polarized state.[26] Ishii et al[27] observed an absence of atherosclerotic intimal lesions in segments of the left anterior descending coronary artery covered by myocardium (so-called myocardial bridges), which separates the artery from the surrounding fat. They propose that the lack of contact to epicardial fat may be what protects the arteries from developing atherosclerosis.[27] In a study of 88 patients undergoing intracoronary ultrasound scanning, Prati et al[28] suggested a permissive role of epicardial fat for atherosclerotic plaques to develop positive remodeling.

In a community-based sample of the Framingham Heart Study (n = 1155), Rosito et al[5] found a significant association

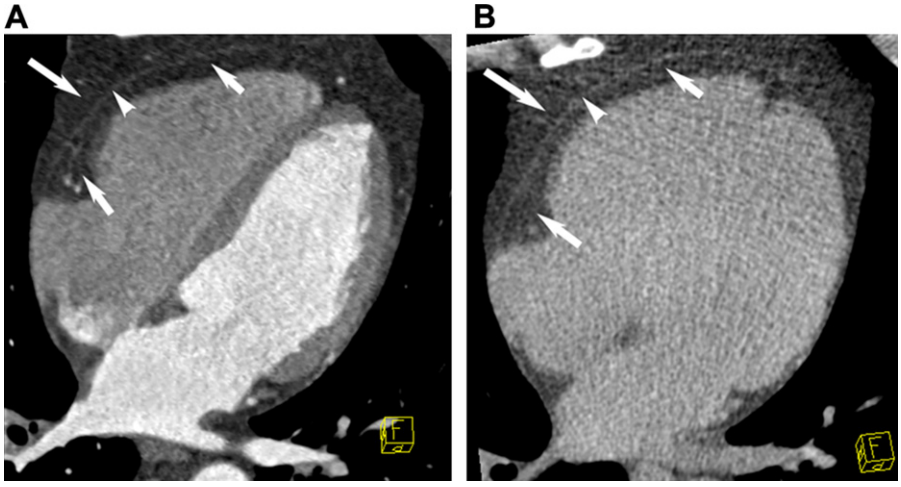

Figure 1 – Multiplanar reconstruction resembling echocardiographic 4-chamber view in contrast enhanced (A) and nonenhanced (B) CT data sets show pericardial fat (*short arrows*), paracardiac fat (*long arrow*), and the pericardium (*arrowhead*).