

Epicardial Fat and Pericardial Fat Surrounding the Heart Have Different Characteristics

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We read with great interest the recently published article by Miyazawa et al in this issue of the Journal,¹ which examined longitudinal changes in pericardial fat volume (PFV) in a community-based cohort of Japanese men. The authors did an excellent job in demonstrating that current smoking and heart rate were significant factors that increased PFV over time. Although their work is of interest, it is important to note that the PFV they measured was not the pure volume of epicardial fat surrounding the coronary arteries. There are 2 distinct fats around the heart: the epicardial adipose tissue that adheres to the outside of the myocardium and moves with the heart, and the pericardial fat adhering to the pericardium that forms the pericardial sac (**Figure 1**).

Embryologically, epicardial fat originates from the splanchnopleuric mesoderm, whereas pericardial fat originates from the primitive thoracic mesenchyme. Although epicardial fat is an active endocrine organ, the role of pericardial fat as a source of adipokines is still unknown. Epicardial fat shares a common coronary blood supply with the myocardium,² and this fat is able to synthesize and secrete adipokines and bioactive factors that reach the myocardium through vasocrine or paracrine pathways.³ Epicardial fat may secrete cardioprotective cytokines such as adiponectin under normal conditions, but it directly

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contributes to atherosclerosis through an outside-to-inside inflammatory atherogenic signal if the epicardial fat becomes “sick fat”.^{4,5}

In the clinical setting, epicardial fat plays an important role in the development of an unfavorable metabolic and cardiovascular risk profile. Excessive epicardial fat is associated with coronary atherosclerosis and the severity of coronary artery disease (CAD).^{6,7} It is noteworthy that the epicardial fat volume (EFV) is a marker of coronary atherosclerosis even with a coronary artery calcium score of zero on coronary CT angiography.⁸ The Framingham Heart Study showed that EFV measured using CT strongly correlated with CAD, but the volume of pericardial fat did not.⁹ Their results were consistent with our study measuring the thickness of epicardial fat using echocardiography.⁷ On the other hand, pericardial fat has not yet shown all of these features. If there is evidence suggesting that pericardial fat could play an active role as a cardiovascular risk factor, then further studies will be necessary to strengthen the role of pericardial fat.

Therefore, when considering the relationship with atherosclerosis, it is necessary to evaluate the EFV. Unfor-

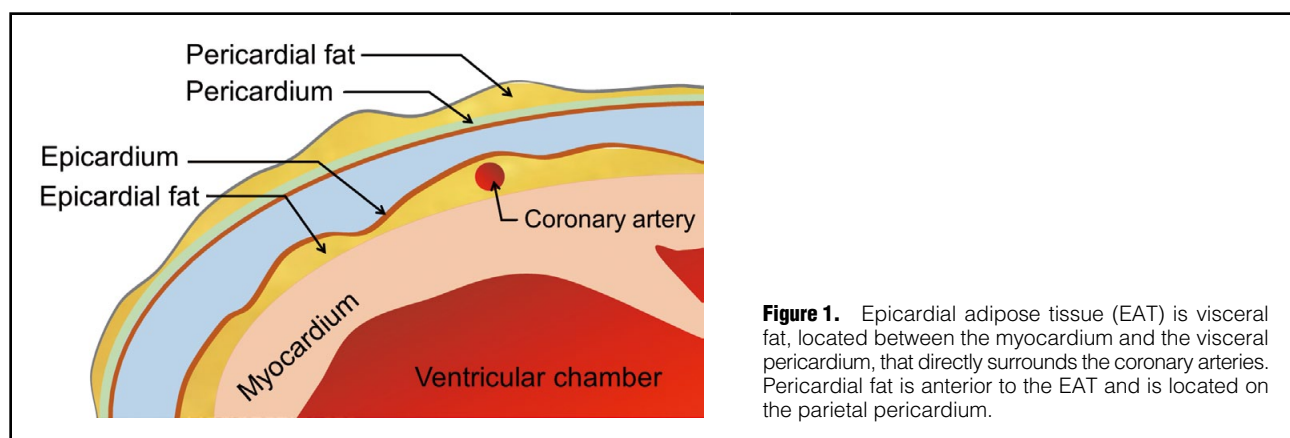


Figure 1. Epicardial adipose tissue (EAT) is visceral fat, located between the myocardium and the visceral pericardium, that directly surrounds the coronary arteries. Pericardial fat is anterior to the EAT and is located on the parietal pericardium.

The opinions expressed in this article are not necessarily those of the editors or of the Japanese Circulation Society.

Received August 16, 2018; accepted August 22, 2018; released online September 5, 2018

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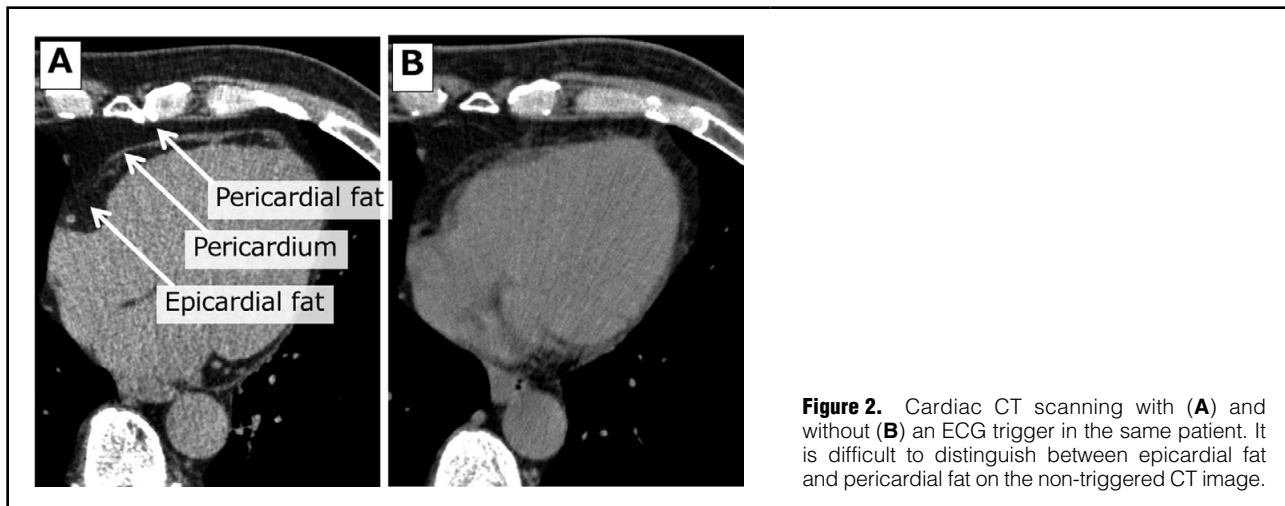


Figure 2. Cardiac CT scanning with (A) and without (B) an ECG trigger in the same patient. It is difficult to distinguish between epicardial fat and pericardial fat on the non-triggered CT image.

unately, it seemed that the method using non-contrast multidetector CT in Miyazawa's study was unable to distinguish between EFV and pericardial fat, so they reported PFV as the combined fat volume. In their results, the median PFV increased by approximately 10 mL (from 64 to 74 mL) over time. It is unclear whether the change was caused by epicardial or pericardial fat. In a previous paper, the accuracy of EFV measurement by CT was established, and there was excellent interobserver and intraobserver reproducibility.¹⁰ However, the measurement was done by CT scanning with an ECG trigger, which Miyazawa et al did not use (Figure 2). An ECG-triggered CT examination is important for measuring the volume of epicardial fat separately from that of pericardial fat.

On echocardiography, epicardial fat moves synchronously with the heart beat, but pericardial fat shows no movement during the cardiac cycle, so it is possible to clearly distinguish between them. Although echocardiography can measure the thickness of each fat type, it is difficult to measure the total volume of each type.

It is important to determine which clinical factors are related to EFV, but it is more interesting to know what clinical factors cause serial changes in the EFV. A recent study showed regression of the EFV in subjects who underwent weight loss, exercise, atorvastatin or ezetimibe administration, or SGLT2 inhibitor therapy.^{11,12} Miyazawa et al found that smoking status and heart rate may affect the PFV. Although it is interesting to speculate that the EFV can be reduced with improvement of lifestyle, such as cessation of smoking, whether this will reduce the risk of events remains to be demonstrated. The diagnostic value of the EFV and its role as a therapeutic target to modify cardiovascular risk should be analyzed in large randomized trials that include multiethnic populations.

Declaration of Conflicting Interests

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

This work was partially supported by JSPS Kakenhi Grants (Number 17K13037 to Y.H. and No. 16H05299 and 26248050 to M.S.) and a grants-in-aid from the Takeda Science Foundation (to M.S.), the

Fugaku Trust for Medical Research (to M.S.) and the Vehicle Racing Commemorative Foundation (to M.S.).

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