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Epicardial adipose tissue: good or bad for cardiac function?

To the editor,

I read with great interest the study of H.-X. Chang et al. [1] published in the latest issue of *Herz*. In our recent studies [2, 3], we too examined the role of epicardial adipose tissue (EAT) in patients with subclinical atherosclerosis and also in patients with coronary artery disease. In their interesting study [1], Chang and colleagues investigated the role of EAT lipolysis and the effect of EAT removal on cardiac function after myocardial infarction.

EAT is a visceral adipose tissue surrounding the heart and coronary arteries. By secreting pro-inflammatory and anti-inflammatory cytokines and chemokines, it is thought to influence the development of coronary atherosclerosis [2–5]. In our recently published study [2], we found that EAT thickness has a positive correlation with slow coronary flow in patients who have normal coronary arteries. Therefore, it can help predict the presence of subclinical atherosclerosis in patients with stable angina pectoris. Because of its close proximity, epicardial fat can affect the heart and coronary arteries locally through its vasocrine and paracrine secretions [6]. In our other study [3], we showed that patients with coronary artery disease had greater EAT thickness with higher gamma-glutamyltransferase (GGT) activity. A higher GGT activity, which is associated with atherosclerosis, was found to be independently associated with EAT thickness.

In the present study [1], removal of EAT after myocardial infarction in rats was found to improve cardiac function compared with rats retaining EAT after myocardial infarction. The authors explained this phenomenon by the weakening of the inflammatory response. Contrary to the findings of Chang et al.[1], Bière and colleagues [7] showed that in patients with ST-elevation myocardial infarction (STEMI), higher EAT was paradoxically related to smaller infarct size and patients with higher EAT had better cardiac healing. In another study [8], a lower EAT volume was found to be associated with less myocardial salvage and larger infarct size in patients with a first STEMI episode. In several studies, EAT was found to have an adverse effect on the myocardium; however, by secreting anti-inflammatory cytokines and chemokines, it is thought to supply energy and may protect the myocardium [7–9]. In the study of Chang et al. [1], similar to our findings, an EAT paradox was not found. An increase in EAT thickness independently predicts adverse cardiac events, and chronic inflammation in epicardial fat can predispose patients to these events. In my opinion, the role of EAT in cardiac function needs further investigation.

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Conflict of interest. M.R. Ege declares that she has no competing interests.

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