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CORRESPONDENCE

Letters to the Editor

Periatrial Epicardial Fat, Local Pro- and Anti-Inflammatory Balance, and Atrial Fibrillation

We congratulate Al Chekakie et al. (1) for their interesting study on the relationship between pericardial fat volume and atrial fibrillation (AF). The authors proposed that the local effects of the proinflammatory cytokines released from the pericardial adipose tissue maybe a potential mechanism. However, 2 recent studies (2,3) indicate that periatrial epicardial fat, not periventricular fat, was associated with AF. Abed et al. (2) demonstrated that periatrial fat volume, but not periventricular fat or total fat, was associated with severity of AF in a multivariate-adjusted model including age, sex, body mass index, hypertension, obstructive sleep apnea, and left atrial area. Batal et al. (3) also showed that periatrial fat thickness between the left atrium and esophagus was an independent risk factor for AF after adjusting age, body mass index, and left atrial area. Therefore, further analysis from the data on periatrial fat volume in the current study may be warranted to clarify this issue.

Furthermore, pericardial adipose tissue has both proinflammatory and anti-inflammatory characteristics (4). Epicardial fat is a source of several proinflammatory cytokines, including tumor necrosis factor-alpha, interleukin-1, and interleukin-6. However, it also secretes adiponectin with insulin-sensitizing and anti-inflammatory effects. Very recently, Kourliouros et al. (5) showed that an increased adiponectin release from epicardial fat was associated with freedom from AF after cardiac surgery. Regulation of local proinflammatory and anti-inflammatory balance in periatrial adipose tissue may be an important therapeutic target in the prevention of AF.

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Reply

We thank Drs. Liu and Li for their remarks and interest in our work (1). The spatial distribution of adipose tissue around the heart is variable, with patches of fat adjacent to myocardium. Furthermore, significant heterogeneity of fat distribution exists among individuals, which makes it difficult to use a single regional measurement as a representative surrogate of actual overall volume or distribution. Total pericardial fat volume has been shown to be a risk factor for atrial fibrillation (AF) in our study as well as in others (1,2). A recent article on the Framingham study showed that pericardial fat was associated with prevalent AF even after correction for other risk factors (2). That study, as well as ours, did not compartmentalize the fat areas, and instead reported the entire pericardial fat volume, which is the most widely accepted method of measurement. We agree with Drs. Liu and Li that the distinction between atrial and ventricular pericardial fat is of interest and that the optimal method of measurement of adipose tissue in these specific compartments needs to be defined. Importantly, the mechanisms by which pericardial fat predisposes to AF need to be elucidated. It is reasonable to hypothesize an inflammatory mechanism based on the paracrine properties of visceral adipose tissue (3). As such, the exact regional distribution of fat may not be relevant, because there are no barriers in the pericardium separating the atria from the ventricles and thereby precluding local effects. We agree with Drs. Liu and Li that this relationship may represent an important therapeutic target for AF, and we look forward to future studies that shed light on the pathophysiological mechanisms.

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