Epicardial adipose tissue and atrial fibrillation: The other side of the coin

To the Editor,

Epicardial adipose tissue, a specialised visceral adipose tissue, produces numerous pro-inflammatory and pro-atherogenic mediators that promote the initiation and progression of coronary atherosclerosis (1). Increased epicardial adipose tissue is related to the presence and angiographic severity of coronary artery disease and coronary plaque vulnerability and independently predicts major adverse cardiovascular events (2). Furthermore, in visceral obesity, the epicardial adipose tissue undergoes conformational and functional changes, leading to the secretin of pro-inflammatory and pro-atherogenic adipokines (e.g., interleukin-6, tumor necrosis factor α, adiponectin, leptin, and plasminogen activator inhibitor) (2), which are involved in a causal relationship between inflammation and atrial fibrillation (3). Consequently, beyond classical cardiovascular risk factors, a causative link between the epicardial adipose tissue and atrial fibrillation has also been suggested because of the structural and functional interplay between atrial fibrillation and the epicardial adipose tissue and the existing evidence of abnormal atrial architecture, adipocyte infiltration, and atrial fibrosis that predispose the myocardial tissue to arrhythmic genesis (4).

In their very interesting and well-conducted clinical research article entitled "An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque" recently published in the Anatolian Journal of Cardiology 2017; 17: 56-63, Kocaman et al. (2) emphasized that the epicardial adipose tissue had a stronger association with carotid intima-media thickness than other risk factors. The epicardial adipose tissue has a complex pathophysiological function; potential direct interactions through paracrine or vasocrine mechanisms between the epicardial adipose tissue and myocardium are strongly suggested because of its metabolically active role as a source of several both pro- and anti-inflammatory adipokines (5). Therefore, it is reasonable to assume its additional role in the modulation of biochemical and metabolic triggers leading to atrial fibrillation (5). The association between the epicardial adipose tissue amount and atrial arrhythmia is supported by a consistent body of evidences suggesting a strong relationship; moreover, the presence of other cardiovascular risk factors does not weaken this link, clearly indicating that the epicardial adipose tissue depot can play a role in the complex pathophysiological scenario of atrial fibrillation (5).

Hence, one could hypothesize that the role of epicardial adipose tissue as a novel cardiovascular risk predictor involves both coronary artery disease and atrial fibrillation. Considering that this probable role in providing continuous pro-atherogenic and pro-inflammatory stimuli could be involved in both the initiation and progression of atherosclerosis, in addition to that a modulator in the arrhythmia genesis and as a possible substrate or trigger, this relationship is not clinically negligible and should be considered a very important element in the prevention/management of cardiovascular disease. In conclusion, based on these evidences, we can suggest that the epicardial adipose tissue is a novel and comprehensive surrogate of cardiovascular risk. Therefore, further consensus on the definition and method to assess and quantify the epicardial adipose tissue should be reached; the epicardial adipose tissue can become a therapeutic target, and evaluating the epicardial adipose tissue amount can become a major need, both for the diagnostic work up and for the assessment of therapy response.

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