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Factors Related to Epicardial Adipose Tissue Thickness

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Dear Editor,

We read the article by Akyüz et al. [1] with great interest. In their study, the authors concluded that the amount of epicardial adipose tissue (EAT) was higher in subjects with xanthelasma than in subjects without. We appreciate their investigation concerning the relationship between xanthelasma and EAT thickness. However, a number of well-known factors may independently affect EAT thickness. These include the metabolic syndrome independent of body mass index, and the correlation between visceral adipose tissue and waist circumference [2, 3]. EAT thickness is significantly higher in patients with nonalcoholic fatty liver disease compared to controls [4], and patients with subclinical hypothyroidism seem to have a significantly increased EAT thickness [5]. Treatment with statins could induce EAT regression independently of low-density lipoprotein lowering [6]. Hence, all these factors may separately influence EAT thickness.

In conclusion, xanthelasma may be associated with an increased EAT thickness. However, to define the exact relationship between xanthelasma and EAT thickness, all those factors that may independently have an effect should be taken into consideration.

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Dear Editor,

We thank Eyuboglu and Karakoyun for their comment on our paper [1]. Several studies have shown an important relation between xanthelasma palpebrarum (XP) and atherosclerotic cardiovascular risk factors [2, 3]. Christoffersen et al. [2] suggested that, independent of other well-known cardiovascular risk factors, XP could cause a higher risk for myocardial infarction and total death. The aim of our study was to test the hypothesis that patients with XP might have increased epicardial fat that could lead to a potentially increased rate of cardiovascular events in this population [1]. We certainly agree that there are some other well-known factors independently affecting the epicardial adipose tissue (EAT) thickness. However, we mentioned the limitation that our study population was small and its design did not allow us to discuss in detail the pathological mechanisms of increased EAT in this population. Furthermore, our study was designed as a pilot study and included some confounding factors. Therefore, further studies are required to investigate the impact of other factors on EAT in patients with XP.

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