Epicardial fat: a novel marker of subclinical atherosclerosis in clinical practice?

In this issue of Anatolian Journal of Cardiology published on article “An increased epicardial adipose tissue is strongly associated with Carotid Intima-Media Thickness and the atherosclerotic plaque, but LDL only with the plaque,” by Kocaman et al. (1) evaluated the association between epicardial adipose tissue (EAT) and markers of subclinical carotid atherosclerosis in 252 obese patients with hypertension, diabetes and/or dyslipidemia, attending the outpatient clinic. Patients with symptoms suggestive of coronary heart disease confirmed by relevant findings on exercise electrocardiogram and perfusion scan were excluded. The authors demonstrated that EAT was strongly and independently associated with both carotid intima-media thickness (CIMT) and the presence of carotid plaques (1). In contrast, among traditional cardiovascular (CV) risk factors, age and male gender correlated only with CIMT, whereas low-density lipoprotein cholesterol (LDL-C) was related only to the presence of carotid plaques. Of note, CIMT increased with increasing LDL-C levels only in patients with EAT >5 mm. These findings highlight the potential clinical use of EAT in assessing subclinical atherosclerosis.

Overall, cardiac adiposity affects coronary circulation due to its functional and anatomical proximity, leading to myocardial dysfunction and hypertrophy, and thus to coronary heart disease and heart failure (2, 3). However, EAT may also exert systemic harmful effects due to the secretion of proatherogenic and inflammatory cytokines, as well as reactive oxygen species (2). In this context, increased EAT has been linked to type 2 diabetes mellitus (T2DM), chronic kidney disease, metabolic syndrome (MetS), non-alcoholic fatty liver disease (NAFLD), obstructive sleep apnea syndrome, erectile dysfunction, and rheumatoid diseases (2, 4–6). All of these metabolic disorders are characterized by increased CV risk (7, 8). It should be noted that EAT can be non-invasively measured by computed tomography, magnetic resonance imaging, and echocardiography, with certain advantages and disadvantages for each method, including availability, radiation exposure, reproducibility, and cost (9).

EAT has been also associated with markers of subclinical atherosclerosis. In this context, EAT was positively correlated with arterial stiffness (assessed by both pulse wave velocity and cardio-ankle vascular index) (10, 11), and negatively with flow-mediated dilatation (FMD) (12). Furthermore, EAT has been positively related to CIMT in several patient populations, including those with T2DM, NAFLD, and MetS, (13–15) as well as in children and adolescents (16). Increased EAT was also linked to both coronary and extracranial carotid artery calcification (17), as well as with the presence of carotid and aortic plaques (18). Currently, no data on EAT and ankle-brachial index have been published.

Overall, excessive peri- or intra-organ fat deposition, including EAT, has been associated with increased CV risk (9). Lifestyle interventions and certain drugs, such as anti-obesity (orlistat), hypolipidemic (statins, ezetimibe), and anti-diabetic (metformin, pioglitazone, liraglutide, and exenatide) may improve abnormal adiposity (9). Apart from CV risk, EAT has been linked to markers of subclinical atherosclerosis, including arterial stiffness, FMD, CIMT and carotid plaques (11–13, 18). As it can be easily measured during echocardiography, EAT represents an attractive surrogate to assess subclinical atherosclerosis, as well as drug effects on CV risk in clinical practice (19).

Declaration of interest
This editorial was written independently; no company or institution supported the authors financially or by providing a professional writer. NK has given talks, attended conferences, and participated in trials sponsored by Amgen, Angelini, Astra-Zeneca, Boehringer Ingelheim, MSD, Novartis, Novo Nordisk, and Sanofi-Aventis. DPM has given talks and attended conferences sponsored by MSD, AstraZeneca, and Libryte.

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