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Research Letter

Epicardial fat thickness, but not intraabdominal fat, correlates with intima-media thickness in patients with metabolic syndrome
Epicardial fat and intima-media thickness

Epicardial fat is visceral fat deposited around the heart. Under normal conditions is a source of anti-atherogenic and anti-inflammatory adipocytokines [1]. However, in pathological conditions epicardial fat releases proinflammatory and proatherogenic adipocytokines [2]. An increase in epicardial fat thickness (EFT) is associated with coronary artery disease [1]. EFT can be measured by standard transthoracic, two-dimensional echocardiography (2 D) [1,2].

Carotid intima-media thickness has been directly associated with myocardial infarction and stroke; high-resolution B-mode ultrasonography provides a noninvasive method that can detect an increase in arterial wall thickness [3].

In addition to obesity extent, adiposity distribution is also a major marker of cardiovascular disease, visceral fat depots (as intraabdominal, epicardial and liver fat) are associated with higher cardiovascular risk than the subcutaneous depot [4].

Bioelectric impedance (BIA), represents an accurate, safe, cost-effective alternative to measure body composition, fat distribution and abdominal adipose tissue [5].

The aim of this study was to evaluate the relationship of EFT and abdominal visceral fat (AVF) with carotid intima-media thickness (CIMT) in patients with metabolic syndrome.

Methods

We included 80 patients with metabolic syndrome. In all of them the measurement of epicardial fat thickness was performed as described by Iacobellis, by standard transthoracic 2D echocardiography [6], with an Aloka Alfa 6 equipment (Japan) using a 3.5 MHz transducer.

CIMT was obtained using high-resolution ultrasound (ESAUTE MEGAGP. Italy), equipped with a 10-MHz linear transducer, a CIMT $\leq$ 0.9 mm was considered normal.

Abdominal visceral fat was quantified by bioelectric impedance using an Inbody 120 device (InBody Co, Ltd., Seoul Korea).

The study was approved by our Research and Ethics Committee. The register number is 208/010/016/17. Participants gave written informed consent before their inclusion in the study protocol.

Data are presented as the mean $\pm$ SD. Statistical analysis was performed with the Pearson coefficient test, and the Fisher exact test, a $P < 0.05$ was significant.

Results

Basal characteristics of patients are shown in Table 1.

Table 1
Basal characteristics of patients.

Age (years)	47.2 ± 11.3
Sex (M/F)	23/57
Body mass index	36 ± 1.2
Waist circumpherence (CM)	114 ± 12
Blood pressure (mm Hg)	134 ± 15/85 ± 9
Epicardial fat thickness (mm)	7.1 ± 1.8
Intima/media thickness (mm)	0.95 ± 0.1
Abdominal visceral fat (Kg)	20.62 ± 4.35
Glucemia (mmol/L)	6.45 ± 1.4
Tryglicerides (mmol/L)	2.03 ± 0.97
High density lipoproteins (mmol/L)	1.07 ± 0.29
Uric acid (mmol/L)	350.93 ± 71.38

We found a significative correlation between EFT and CIMT ($r = 0.71$, $r^2 = 0.5$, $p = 0.0001$) (Fig. 1). We did not find any correlation between AVF with CIMT ($r = 0.06$, $p = 0.68$).

When we evaluated the risk for a CIMT $\geq$ 0.9 mm in patients with an EFT $\geq$ 4 mm, we found a statistically significant association ($p = 0.0001$). Interestingly, only one patient with an EFT < 4 mm shown a CIMT $\geq$ 0.9 mm.

Discussion

In this work we found that CIMT correlates with EFT but not with AVF in patients with metabolic syndrome.

Although both, epicardial adipose tissue and AVF are visceral fat, we found that only epicardial fat correlated with CIMT in our patients, beyond adipose tissue localization, this fact may be due to the contiguity of myocardium with epicardial fat, which share the same microcirculation, and to the absence of fascia between them. Both tissues and the coronary arteries interact through paracrine, vasocrine and endocrine actions of the adipocytokines produced in the epicardial fat, all of them have a detrimental effect on the initiation and progression of vascular inflammation and atherosclerosis [1], compared with other fat depots, epicardial fat has a greater ability to secrete inflammatory cytokines, whereas subcutaneous fat predominantly produces adiponectin and abdominal fat mainly synthetizes leptin [7]. Indeed, evidence suggests that the regional distribution of fat depots plays an important role in the development of ischemic heart disease [4,8].

Randrianarisoa found that periaortic adipose tissue, but not peribranchial adipose tissue, correlated with CIMT [9], those findings highlight the importance of adiposity distribution in the development of arterial disease.

Adipose tissue is considered as an extremely active endocrine organ and not only an energy store [7]. In addition to the amount of adipose tissue, adiposity distribution is also a major determinant of the incidence of comorbidities [10]. We found that EFT has a greater involvement than AVF in vascular damage (evaluated as CIMT).

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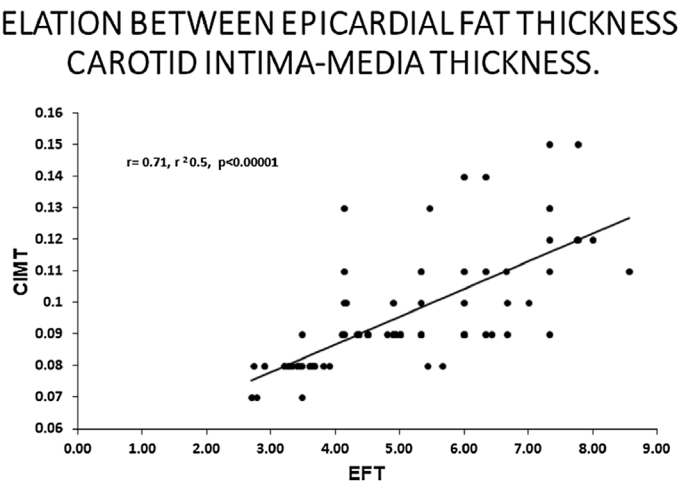

Fig. 1. Correlation between epicardial fat thickness and carotid intima-media thickness.

In conclusion, we found that EFT, but not AVF correlates with CIMT in patients with metabolic syndrome, EFT seems to be a better marker of IMT than other fat depots.

The measurement of EFT, easy and non-invasive, seems to be an independent predictor of atherosclerosis, and should be measured as a part of the global cardiovascular risk evaluation in patients with metabolic syndrome.

Conflict of interest

We do not have any financial or working relation that may lead to any conflict of interest.

Ethical statement

The authors declare that all experiments on human subjects were conducted in accordance with the Declaration of Helsinki, and that all procedures were carried out with the adequate understanding and written consent of the subjects, as described in methods section.

The authors also certify that formal approval to conduct the experiments described has been obtained from the human subjects review board of their institution. Indeed, this protocol was conducted with the approval of the Research and Ethics Committee of our hospital. The register number is 208/010/016/17, and could be provided upon request.

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