Focus on Medical Sciences Journal Original Article

May 2021, Volume 7, Issue 2

Epicardial Fat Thickness Correlates with the Triglyceride Glucose Index, But Not with Visceral Adiposity Index in Patients with Metabolic Syndrome

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Submitted: 08.05.2021

Accepted: 08.17.2021

Keywords:
Epicardial Fat
Triglycerides/Glucose Index
Visceral Adiposity Index
Cardiovascular Risk
Metabolic Syndrome

Abstract

Introduction: Epicardial fat is true visceral fat deposited around the heart. Visceral fat is the main risk factor for the development of the metabolic syndrome. Transthoracic echocardiography provides a reliable measurement of EFT. Visceral adiposity index (VAI) has been associated with cardiovascular risk. Triglyceride and glucose index (TGI) is a validated marker of insulin resistance. This study aims to evaluate the relationship of EFT with VAI and TGI in patients with metabolic syndrome.

Methods: We assessed 123 patients with metabolic syndrome who underwent echocardiography; EFT was measured with an Aloka alfa 6 equipment; by 2 cardiologists who were unaware of the clinical data. Also, glycemia, lipid profile, and serum uric acid were measured in all patients.

In all of them, ITG and VAI were calculated. Statistical analysis was performed with the Pearson coefficient test.

Results: We did not find a positive any correlation between EFT with VAI (r= 0.152, p= 0.93). However, we found a significant positive correlation between EFT with TGI (r= 0.388, p=0.0001).

Conclusions: Our results agree with those reports that have shown that triglycerides are useful tools in the evaluation of risk for the development of metabolic complications.

INTRODUCTION

Epicardial fat is real visceral fat deposited around the heart, between the outer wall of the myocardium and the visceral layer of the pericardium. Under normal conditions, it plays an important role in the maintenance of cardiovascular functions, provides protection to the myocardium, and has an important role in energy supply, and is a source of anti-atherogenic and anti-inflammatory adipocytokines [1, 2]. However, in pathological conditions, epicardial fat releases proinflammatory and proatherogenic adipocytokines (Table 1) [1, 2]. The increase in epicardial fat thickness (EFT) is associated with a greater risk for metabolic syndrome and coronary artery disease [3].

Due to their contiguity, to the absence of fascia between them, and as myocardium and epicardial fat share the same microcirculation, both tissues interact through paracrine and vasocrine actions of those adipokines that freely diffuse towards the heart [2].

Epicardial fat thickness may be measured on the free wall of the right ventricle, this method provides a simple, cheap, and readily available assessment of the epicardial adipose tissue [4]. As adipokines released by epicardial adipose tissue may lead to insulin resistance and endothelial dysfunction, an association between epicardial fat and glucose metabolism has been proposed, as well as a role in the lipid profile of patients with metabolic syndrome [5].

Visceral adiposity index is a mathematical model that calculates visceral adiposity based on anthropometric measures and the lipid profile and is considered a marker of adipose tissue function, it has been associated with metabolic syndrome and cardiovascular risk [5]. Triglyceride glucose index is a validated marker for insulin resistance and metabolic syndrome and has been also associated with cardiovascular risk [6].

Table 1: Adipokines Released by The Epicardial Fat	
Released Adipokines	
Inflammatory actions	
Anti-inflammatory	Adiponectin, omentin.
Proinflammatory	Tumor Necrosis Factor α, Interleukins (1β, 6,8), Resistin, Angiotensinogen, C reactive protein.
Atherogenic	
Proatherogenic	Tumor Necrosis Factor α, Resistin, Angiotensinogen, C reactive protein.
Antiatherogénic	Adiponectin, omentin
Metabolic effects	Adiponectin, Resistin, Leptin, Omentin, Adrenomedullin, Apelin, Tumor Necrosis Factor α, Angiotensin.
Others	Plasminogen -1 activator inhibitor.

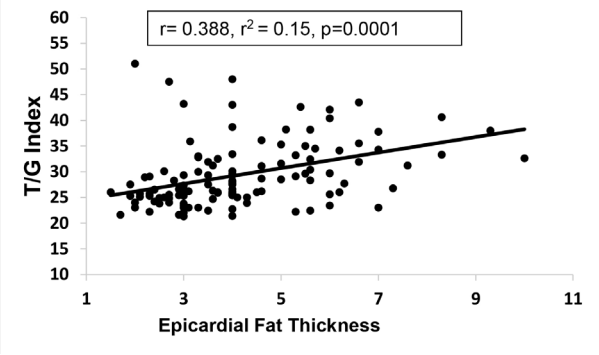

Figure 1: Correlation Between Triglyceride/Glucose Index and Epicardial Fat Thickness

Table 2: Basal Characteristics of Patients	
Variables	**Values**
Age (Years)	61.2 ± 11
Sex (m/f)	49 / 74
Body Mass Index (Kg/m²)	29.4 ± 5
Abdominal Circumference(cm)	100.4 ± 12
Blood pressure (mm Hg)	131 ± 14 / 76 ± 10
Glycemia (mg/dl)	111 ± 23
Total Cholesterol (mg/dl)	192.2 ± 32.4
High density Lipoproteins (mg/dl)	43.4 ± 9.5
Triglycerides (mg/dl)	176.3 ± 71.6
Uric acid (mg/dl)	6.33 ± 1.5
Epicardial fat thickness (mm)	4.14 ± 1.3
Visceral adiposity index	4.02 ± 2.1
Triglycerides/glucose index	29.4 ± 5
Background of type 2 diabetes	109 (51.6%)
Background of arterial hypertension	113 (53.55%)

Both indexes are simple, cheap, useful and ready for the evaluation of metabolic health in patients. This study aims to evaluate the relationship of EFT with VAI and TGI in patients with metabolic syndrome.

METHODS

We assessed 123 patients with metabolic syndrome who underwent echocardiography in our facility; The selection of the sample was determined as non-probabilistic at convenience because it is useful for this study. In all of them, the measurement of the epicardial fat thickness (EFT) was performed. Epicardial fat thickness was identified as the echo-free space between the outer wall of the myocardium and the visceral layer of the pericardium. It was measured on the free wall of the right ventricle, perpendicularly at end-systole from the parasternal long-axis views of 3 cardiac cycles by standard transthoracic 2D echocardiography, as described by Iacobellis [4], with an Aloka Alfa 6 equipment (Japan) using a 3.5 MHz transducer, by 2 cardiologists who were unaware of the clinical data.

In all patients, serum glucose (glucose oxidase), creatinine (JAFFE), low-density lipoprotein, high-density lipoprotein (CHODPAP), and triglycerides (Triglyceride-pap) were measured by personal unaware of the study. All venous samples were collected in the morning, after 12 hours overnight fast. The triglyceride glucose index (TGI) was calculated with the next formula:

Ln (Triglyceride [mg/dL] X glucose [mg/dL] / 2).

Visceral adiposity index (VAI) was calculated with the next formula:

Abdominal circumference (cm) / (39.68+(1.88 X BMI)) X (TG/1.03) X (1.31/HDL) for male, and

Abdominal circumference (cm) / (36.58+(1.89 X BMI)) X (TG/0.81) X (1.52/HDL) for female

(BMI = Body mass index. TG= tryglycerides, HDL = High density lipoproteins)

Patients with any of the following diagnoses were excluded from the study: Decompensated diabetes mellitus (glucose ≥ 250 mg/ml), heart, hepatic, or renal failure, evidence of valvular heart disease, heart block or cardiac arrhythmia, acute coronary syndrome, or cerebrovascular disease six months before the baseline of the study. There also were excluded subjects with autoimmune disease, pericardial effusion, pregnancy, malignancy, and alcohol or psychotropic drugs abuse.

The study was conducted with the approval of the Research and Ethics Committee of our hospital, in accordance with the Helsinki declaration. The register number is 208/010/18/16. Participants gave written informed consent before their inclusion in the study protocol.

Statistical analysis was performed with the Pearson coefficient test, which was calculated by the researchers in the excel program.

RESULTS

The baseline characteristics of the patients are shown in Table 2. Briefly, alterations of carbohydrate metabolism, dyslipidemia, and hyperuricemia were common in our patients. We did not find a positive correlation between EFT with VAI (r= 0.152, p= 0.93). However, we found a significant positive correlation between EFT with TGI (r= 0.388, p=0.0001) (Fig. 1). The Odds ratio for a TGI > 8 in patients with an EFT ≥ 4 mm, was statistically significant (4.27, CI_{95} 1.71 - 10.7)

DISCUSSION

In this paper we found that epicardial fat thickness correlates with the T/G index, but not with the visceral adiposity index in

patients with metabolic syndrome, Epicardial fat is a metabolically active tissue that secretes adipokines that can affect the metabolic media throw several pathways [2].

An increased body mass index has been associated with increased cardiometabolic risk; however, the usefulness of this measurement is limited because do not allow discrimination between muscle mass from fat, nor visceral fat from subcutaneous fat, as a consequence, new tools have been implemented as an alternative for measuring abdominal visceral fat [7]**.** The visceral adiposity index is considered a surrogate marker of visceral adipose tissue function, its normal value is 1, a VAI greater than 2.2 is associated with an increased cardiometabolic risk, and may lead to the development of type-2 diabetes mellitus [8].

TGI is mainly a marker of insulin resistance, recently, it has been also linked with increased cardiovascular risk, values greater than 8.0 are associated with subclinical atherosclerosis, this index is related to visceral fat depots too [9], as in our study, where TGI had a significant correlation with EFT. Besides the amount of visceral fat, the localization of visceral fat depots has a role in the development of cardiovascular disease, in fact, visceral fat (epicardial fat included), generates greater cardiovascular risk than subcutaneous fat [10].

Yang et al found that VAI was not better than triglyceride values for the prediction of new-onset type-2 diabetes mellitus, and Elizalde et al found that VAI has the same capacity as triglycerides, for the prediction of new-onset type-2 diabetes mellitus [11]. Those results may reflect that VAI is not a good marker of metabolic risk, which is in accordance with our results, as we did not find a correlation between VAI and EFT. Narvaez et al reported an association between an EFT > 3 with the presence of metabolic syndrome [12], in fact, epicardial adipose tissue is considered a marker of the metabolic status of the patient, is associated with the metabolism of glucose too, this fact agrees with our results, a correlation between EFT with a marker of insulin resistance, as the T/GI.

Interestingly, both, increased EFT [13] and TGI [9], are surrogate markers of subclinical atherosclerosis. Our results may have therapeutical implications, the use of agents that reduced EFT may reduce insulin resistance and cardiovascular risk, this fact requires further investigation.

The routinary measurement of epicardial fat thickness, and the evaluation of T/GI, are good tools for the calculation of global cardiovascular risk, in patients with metabolic syndrome.

ACKNOWLEDGMENTS

None.

ETHICAL STATEMENT

The study was conducted with the approval of the Research and Ethics Committee of our hospital, in accordance with the Helsinki declaration. The register number is 208/010/18/16. Participants gave written informed consent before their inclusion in the study protocol.

CONFLICTS OF INTEREST

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

FUNDING

The paper was self-financed by our group.

AUTHORS' CONTRIBUTIONS

CGG, AFRG, DRBM, JLNR**,** contributed equally to this work; SARR, SHR. participated in the acquisition, analysis, and interpretation of the data, and drafted the initial manuscript; All authors revised the article critically for important intellectual content and approved this final version.

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