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Fat deposits and their relationship with type 2 diabetes in patients with metabolic syndrome

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Abstract

Aim: In Mexico, 18.3% of patients over 20 years of age suffer from type 2 diabetes mellitus (T2DM). Visceral fat has been identified as a risk factor for developing T2DM, considering that its effect depends mainly on the amount, it has been proposed that its location also participates in the genesis of T2DM. This paper aims to analyze whether the location of fatty depots participates in the appearance of T2DM in patients with metabolic syndrome.

Methods: A total of 101 patients with metabolic syndrome according to the criteria of the International Diabetes Federation were included. In all of them, epicardial fat (EF) was measured by echocardiography, according to the Iacobelis criteria. Preperitoneal fat (PreF), mesenteric fat (MF), and perirenal fat (PF) were also measured by ultrasound. The statistical methods used were the odds ratio (OR) and Fisher's exact test.

Results: We found a significant association between EF thickness (OR 8.28, 95% CI 2.16–31.70, $p = 0.001$) and MF (OR 4.27, 95% CI 1.63–11.13, $p = 0.0037$) with T2DM, 60% of patients with increased PF presented T2DM. Interestingly, no patient with a PF less than 10 mm presented with T2DM. We found no association between PreF and T2DM (OR 0.88, 95% CI 0.32–2.40).

Conclusions: The results suggest that the location of adipose tissue plays an important role in the development of T2DM. This may be due to the type of adipocytokines secreted by the various fat deposits and their metabolic effects.

Keywords

epicardial fat, type 2 diabetes mellitus, perirenal fat, mesenteric fat, preperitoneal fat, metabolic syndrome

Introduction

The National Survey on Health and Nutrition 2022 found that in Mexico, 18.3% of the population over 20 years old suffers from type 2 diabetes mellitus (T2DM) [1]. The same survey found an obesity prevalence of 36.9% in the same age group [2].

There is a well-established relationship between obesity and T2DM, as both conditions share common pathways, such as the accumulation and dysfunction of adipose tissue, insulin resistance and increased proinflammatory and diabetogenic adipocytokines, such as resistin [3, 4], leading to β-cell dysfunction, multi organ insulin resistance, endothelial dysfunction, hypercoagulability, and abnormal lipid metabolism, ultimately leading to the clinical manifestations [3, 4]. Chronic inflammation and hyperinsulinemia are also proposed to underlie the increased risk of other chronic diseases conferred by obesity [2].

In addition to the amount of fat, its distribution has been linked to cardiovascular disease. For example, Rubio-Guerra and colleagues found that epicardial fat (EF) correlates more than intra-abdominal fat with increased intima-media thickness in subjects with metabolic syndrome [5] and that an EF thickness > 5 mm is associated with atherosclerosis and arterial stiffness [6].

Other fat depots have also been associated with cardiometabolic risk; indeed, a perirenal fat (PF) thickness > 13 mm increases the risk of hypertension, and a mesenteric fat (MF) thickness > 10 mm increases the risk of cardiovascular disease [7].

Lee and colleagues [8] found that intra-abdominal fat and intrahepatic fat correlate with cardiometabolic risk factors to a greater extent than subcutaneous, pericardial, intramuscular, and other fat deposits.

The aim of this work is to evaluate whether the location of fatty depots participates in the appearance of T2DM in patients with metabolic syndrome.

Materials and methods

Methods

One hundred and one patients diagnosed with metabolic syndrome, according to the International Diabetes Federation definition [9], were studied, of whom 63 (62.38%) suffer T2DM. EF was measured by echocardiography as the echo-free space between the myocardial wall and the visceral layer of the pericardium, in the free wall of the right ventricle at end-systole, in 3 cycles from a parasternal window, with an Aloka alfa (Japan) 6 device, with a 3.5 MHz transducer, by 2 echocardiographers who were blinded to the details of the study [5, 6]. We use a cutoff point of 5 mm in thickness because we have found that from that value, the risk of cardiovascular disease increases [6].

Preperitoneal fat (PreF) was measured by ultrasound. The recording was made longitudinally, with the transducer perpendicular to the skin, along the midline of the abdomen, between the xiphoid process and the umbilicus, in triplicate, and the average of the 3 greatest thickness measurements was used for the analysis [10].

MF was measured from the paraumbilical area, identifying the mesenteric layers as elongated structures free of peristalsis between peritoneal surfaces that reflected echoes. When different mesenteric layers were visualized, the maximum thickness was measured. The mean of the three thickest mesenteric layers, and an average of 3 measurements, were used for the analysis [10]. We used 10 mm thickness as the cutoff point because it has been reported that, from that value onward, the risk of cardiovascular disease increases [7].

PF was recorded with the patient in the supine position. The transducer was placed vertically against the lateral skin surface over the kidney. Longitudinal scans of the kidney were taken gently, and PF thickness was measured from the kidney surface to the inner side of the abdominal musculature. The average of the maximum thickness values on both sides, taken three times, was used for analysis [11]. We selected a cutoff point of 13 mm because that value is associated with hypertension [7].

All the measurements were performed by a certified sonographer, before noon, who was unaware of the patients' characteristics.

In all patients, blood glucose (glucose oxidase), serum creatinine (Jaffe), lipid profile (CHODPAP), and triglycerides (triglyceride-PAP) were measured. All samples were collected by venipuncture after a 12-hour fast in tubes without anticoagulant, before 9 a.m. The samples were centrifuged at 800 rpm for 15 min, and the serum was then separated for immediate processing. The measurements were performed by personnel who were blinded to the nature of the study.

Exclusion criteria

Patients with renal insufficiency (creatinine > 2.5 mg/dL), hypothyroidism, type 1 diabetes mellitus, hyperthyroidism, hepatic insufficiency (serum aminotransferases more than twice the upper limit of normal), ascites, pericardial effusion, malignancy, a cardiovascular or cerebrovascular event within the last 6 months, or a history of alcohol or psychotropic drug abuse were excluded.

Data statistical methods

The statistical methods used were odds ratio (OR; for the association of EF, MF, and PreF and the coexistence of diabetes mellitus), Mann-Whitney *U* test for the comparison of items between groups, and Fisher's exact test for the association between PF and the coexistence of diabetes mellitus. Data are presented as mean ± standard deviation, and a p-value < 0.05 was considered statistically significant.

Ethical approval

The study was approved by our hospital's research and ethics committee, with registration number 208010-005-2021, and was conducted in accordance with the Declaration of Helsinki. Patients gave their informed consent prior to their inclusion in the study.

Results

The basal characteristics of patients are shown in Table 1. Briefly, patients have an increased abdominal circumference and a body mass index in the range of obesity when compared with subjects without T2DM.

Table 1. Basal characteristics of patients.

Items	Values
age (years)	55.00 ± 10.00
Weight (kg)	77.86 ± 11.40
Height (cm)	155.88 ± 13.20
Gender (*n*, male/female)	34/67
Waist circumference (cm)	100.30 ± 14.50
Body mass index (kg/m²)	32.04 ± 1.50

We found a significant risk of coexisting T2DM when EF > 5 mm (OR 8.28, 95% CI 2.16–31.70, p = 0.001), and MF > 10 mm (OR 4.27, 95% CI 1.63–11.13, p = 0.0037); we also found a significative association between PF thickness and diabetes mellitus (p = 0.0076), interestingly, no patient with PF < 10 mm presented T2DM (Table 2). We found no association between PreF and T2DM (OR 0.88, 95% CI 0.32–2.40).

Table 2. Association between type 2 diabetes mellitus (T2DM) and visceral fat depots.

Fat depot	Odds ratio, 95% CI	*p*
Epicardial fat	8.28, 95% CI 2.16–31.70	0.001
Mesenteric fat	4.27, 95% CI 1.63–11.13	0.0037
Perirenal fat	-	0.0076
Preperitoneal fat	0.88, 95% CI 0.32–2.40	> 0.05

In Table 3, we present the differences between patients with diabetes mellitus and those without diabetes, briefly; the coexistence of hypertension was higher in patients with diabetes mellitus. Serum glucose, triglycerides, abdominal circumference, EF thickness, MF thickness, and PF thickness were higher in patients who suffer from T2DM.

Table 3. Differences between groups

Items	Diabetes mellitus	Without diabetes	*p*
Sample, *n*	63	38	-
Gender (*n*, male/female)	19/44	15/23	-
Age (years)	59.20 ± 7.10	54.00 ± 13.00	0.082
Coexistence of hypertension (*n*, %)	46 (73.02%)	11 (28.95%)	0.00001
Weight (kg)	78.00 ± 12.00	77.00 ± 11.00	0.48
Heigth (cm)	156.00 ± 11.00	158.00 ± 14.00	0.09
Waist circumference (cm)	105.20 ± 12.00	98.80 ± 9.30	0.022
Glucose (mg/dL)	171.00 ± 8.90	96.40 ± 5.00	0.001
Triglycerides (mg/dL)	207.00 ± 9.20	180.00 ± 6.50	0.008
Epicardial fat (mm)	6.20 ± 0.90	5.40 ± 1.00	0.016
Preperitoneal fat (mm)	14.60 ± 4.30	15.30 ± 3.60	0.18
Mesenteric fat (mm)	41.80 ± 13.00	23.50 ± 9.00	0.00001
Perirenal fat (mm)	18.00 ± 3.00	14.80 ± 3.50	0.00021

Discussion

In this study, we found that the increase in the thickness of fat deposits in epicardial, mesenteric, and perirenal locations is significantly associated with the coexistence of type-diabetes in subjects with metabolic syndrome. The design of the protocol, with the measurement of these fat deposits by personnel who were unaware of the study, allows us to have reliable data to perform an unbiased analysis that leads us to valid results.

Visceral fat exhibits greater lipolytic activity than subcutaneous fat, which is accompanied by an excessive release of free fatty acids. This promotes insulin resistance and a higher cardiovascular risk [7]. Furthermore, the increase in the amount of visceral fat tissue leads to a greater release of pro-inflammatory, pro-atherogenic, and hyperglycemic adipocytokines, and a decrease in the synthesis of others, such as adiponectin, which has a cardioprotective role and reduces serum glucose [4, 5]. All these changes contribute to the development of T2DM. EF, MF, and PreF are all types of visceral fat, each with its own distinguishing characteristics [7].

EF is twice as metabolically active as other white fat deposits, releasing a greater quantity of fatty acids and vasoactive adipocytokines [12]. Increased EF thickness promotes left ventricular hypertrophy and ischemic heart disease [6], and releases adipocytokines with primarily pro-inflammatory, atherogenic activity that promote insulin resistance [5]. As EF shares coronary circulation with the myocardium and there is no fascia between the two tissues, these adipocytokines diffuse directly into the left ventricle and the systemic circulation [5, 6].

MF has a metabolically active behavior, showing an adipocytokine secretion pattern typical of visceral fat; when its thickness increases, it exhibits a detrimental effect on insulin sensitivity [7]. Our study is consistent with the findings of Wang and colleagues [13], who found that abdominal visceral fat (measured by magnetic resonance) shows a greater association with the onset of diabetes than other fat deposits such as liver fat, muscle fat, and subcutaneous fat.

The fact that adipocytokines produced by EF pass directly into the coronary and systemic circulation [5], while those produced by MF may pass through the liver filter via the portal circulation [7], and that the production of adipocytokines by increased EF thickness consists mainly of pro-inflammatory molecules, while the production by abdominal visceral fat is mainly based on leptin [5], would explain the higher risk of coexistence of T2DM with increased EF compared to increased MF, as found in our study.

PF has a secretion of adipocytokines similar to other fat deposits, which promotes insulin resistance and the development of diabetes mellitus [12]. Interestingly, the fact that no patient with PF < 10 mm Hg developed T2DM reflects the absence of diabetes risk in subjects with values below that thickness. To our knowledge, this is the first study to associate PF thickness with the risk of developing type 2 diabetes.

Liu et al. [14] found an association between MF, but not PreF, with intima-media thickness. The role of PreF as a non-portal adipose tissue may explain its lack of association with intima-media thickness and with the coexistence of T2DM, which requires further investigation.

Karaevren et al. [15] found thar hepatic steatosis and fatty pancreatic disease are associated with newly diagnosed T2DM, and with elevated fasting plasma glucose, whereas Zheng et al. [16] found that visceral adipose index and fatty liver index, contributed to insulin resistance in subjects with T2DM, both papers reflect the importance of visceral fat its depots on the development of metabolic complications and T2DM.

This study provides information for a more complete assessment of patients with metabolic syndrome, since measuring the thickness of fat tissue deposits at the epicardial, mesenteric, and perirenal levels could be a valuable tool to define the risk of coexisting T2DM [12], and perhaps guide the physician to the use of drugs such as glucagon-like peptide-1 (GLP-1) receptor agonist in those patients, which, in addition to promoting weight loss, reduce visceral fat, particularly the thickness of EF [17], that in addition to the benefits described, could prevent the development of diabetes mellitus [18]. This fact requires confirmation in clinical studies.

It is important to say that our study only reports association, not causality.

Conclusions

Our results show that it is useful to include the measurement of EF, PF, and MF deposits in the comprehensive assessment of patients with metabolic syndrome, as it provides valuable information for establishing the overall risk of comorbidities. Furthermore, these procedures are simple, non-invasive, safe, and have proven to provide reliable information that can guide therapeutic decision-making.

Abbreviations

EF: epicardial fat

MF: mesenteric fat

OR: odds ratio

PF: perirenal fat

PreF: preperitoneal fat

T2DM: type 2 diabetes mellitus

Declarations

Author contributions

IGNO, JLNR, DRBM, AFRG, CGG, AMS: Conceptualization. Data curation, Formal analysis, Investigation, Methodology, Writing—original draft, Writing—review & editing. All authors read and approved the submitted version.

Conflicts of interest

The authors declare that there are no conflicts of interest.

Ethical approval

The study was approved by our hospital's research and ethics committee, with registration number 208010-005-2021, and was conducted in accordance with the Declaration of Helsinki.

Consent to participate

Informed consent to participation in the study was obtained from all participants.

Consent to publication

Not applicable.

Availability of data and materials

The datasets that support the findings of this study are available from the corresponding author upon reasonable request.

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