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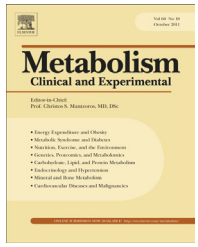

# Sagittal abdominal diameter as a marker for epicardial adipose tissue in premenopausal women

*Ana Carolina Junqueira Vasques*, José Roberto Matos Souza, Ademar Yamanaka, Maria da Saúde de Oliveira, Fernanda Satake Novaes, José Carlos Pareja, Bruno Geloneze*

*Department of Internal Medicine, Laboratory of Investigation on Metabolism and Diabetes (LIMED), School of Medicine, State University of Campinas, Campinas, Brazil*

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ABSTRACT

*Objective.* Accumulation of epicardial (EAT) adipose tissue is associated with the development of an unfavorable metabolic risk profile. Gold standard methods used to assess this fat depot are not routinely applicable in the clinic. Anthropometric measures, including the sagittal abdominal diameter (SAD), have emerged as surrogate markers of visceral obesity. We determined the relationship between EAT measurement and cardiometabolic risk parameters and the potential use of the SAD, compared with other anthropometric parameters, as a practical estimation of EAT.

*Materials/Methods.* Sixty-seven premenopausal women were evaluated. The anthropometric parameters that were measured included waist circumference, SAD, body mass index and waist-to-hip ratio. EAT was determined by echocardiogram. Visceral adipose tissue (VAT) was determined by abdominal ultrasound. Insulin sensitivity was assessed by the hyperglycemic clamp.

*Results.* The accumulation of EAT was correlated with impaired insulin sensitivity and decreased adiponectin. All of the anthropometric measurements were correlated with EAT. Interestingly, EAT was most significantly correlated with the SAD. From the ROC analysis, we found that the SAD measurements were very accurate, presenting the highest area under the curve for EAT (0.81; $p<0.01$) when compared with the other measurements. In the multiple linear regression analysis, EAT was moderately predicted by the SAD ($R^2=0.25$; $p<0.001$).

*Conclusion.* SAD, a simple anthropometric measure, accurately estimated EAT and thus represents a clinically useful non-invasive marker that can identify patients with EAT accumulation.



## 1. Introduction

The accumulation of specific fat depots such as epicardial adipose tissue (EAT) has been associated with poor metabolic outcomes [1]. Human EAT is a visceral adipose tissue (VAT), but it is a visceral thoracic fat. In pathological situations, EAT can locally affect the heart and coronary arteries through vasocrine or paracrine secretion of pro-inflammatory

*Abbreviations:* ANOVA, analysis of variances; BMI, body mass index; EAT, epicardial adipose tissue; GIR, glucose infusion rate; ISI, insulin sensitivity index; ROC analysis, receiving operating characteristic curve analysis; SAD, sagittal abdominal diameter; VAT, visceral adipose tissue; WC, waist circumference.

* *Corresponding author.* Tel.: +55 19 35218589; fax: +55 19 35218589.
E-mail address: anacarolinavasques@yahoo.com.br (A.C.J. Vasques).

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cytokines [2]. EAT has been recognized as a potential cardiac risk marker that contributes to the development of an unfavorable metabolic profile in obesity [1,3].

There is a strong correlation between generalized adiposity and the amount of EAT as well as between EAT and VAT [4]. The gold standard methods used to assess fat depots are expensive and have limited availability for routine clinical application. Anthropometric measurements are being investigated as indicators of fatty depots in many populations [5–10].

The classic anthropometric parameters used to assess obesity are body mass index (BMI), waist circumference (WC), and waist-hip ratio [5]. The sagittal abdominal diameter (SAD) is not widely used to gauge obesity, but an increasing number of studies have demonstrated excellent performance of this novel anthropometric measure in the assessment of VAT, cardiometabolic risk and mortality [6,8,9]. However, there are no reports evaluating the relationship between the SAD measured anthropometrically and EAT.

Considering the difficulty of evaluating EAT in clinical practice, and the promising findings related to the SAD, we investigated the potential use of the SAD for the practical estimation of EAT. Additionally, we assessed the relationship between EAT measurement and cardiometabolic risk parameters.

## 2. Methods

A cross-sectional study was conducted with 67 women with different glucose tolerance levels (n=29 with type 2 diabetes). The inclusion criteria were to be over 20 years old, premenopausal, and have a BMI between 18.5 and 45.0 kg/m$^2$. None were on insulin, glitazone and corticosteroids therapy. Patients with a positive history of liver and renal disease or recent neoplasia were not included. Subjects were recruited from the outpatient clinics of the Hospital School of Medicine at the State University of Campinas.

This study was approved by the Ethics Committee, State University of Campinas, Brazil, in accordance with the Declaration of Helsinki. All participants provided written informed consent before participation.

### 2.1. *Anthropometric and adiposity measures*

All of the subjects underwent a detailed anthropometric examination wearing light clothes and no shoes. The SAD was measured while the subjects were in a supine position with their knees slightly bent. The measurement was taken at the umbilicus level using the Holtain–Kahn Abdominal Caliper™. Waist and hip circumferences were measured at standing position by a measuring tape. WC was measured at the umbilicus level without clothing present in the measurement area. Hip circumference was measured at the most protuberant point between the waist and the thigh [10]. All measurements were taken in duplicate and averaged. Waist-to-hip ratio and BMI were also determined. Body fat and free fat mass were determined using a bioimpedance analyzer (model BIA 310).

Each subject underwent a transthoracic two-dimensional guided M-mode echocardiogram. Echocardiograms were performed using standard techniques on a GE instrument with the subjects in the left lateral decubitus position. EAT thickness was measured on the free wall of the right ventricle from both the parasternal long- and short-axis views. EAT appears as an echo-free space [11]. All of the measurements were taken in triplicate and averaged. The intraclass coefficients of correlation were 0.95 (95% CI 0.92–0.97) for EAT ($p<0.001$).

VAT thickness was determined in supine position 1 cm from the umbilicus by ultrasonographic procedures using a 3.5 MHz probe. VAT was the distance between the internal

**Table 1 – Clinical and metabolic parameters divided into tertiles of epicardial adipose tissue.**

| | Epicardial adipose tissue | | | |
|---|---|---|---|---|
| | Tertile 1 | Tertile 2 | Tertile 3 | *p* |
| Weight (kg) | 72.1±21.9 [a] | 84.1±12.5 [a, b] | 91.9±12.9 [b] | 0.001 |
| Height (m) | 1.62±0.06 [a] | 1.59±0.07 [a] | 1.59±0.06 [a] | 0.083 |
| Body mass index (kg/m$^2$) | 29.9±7.6 [a] | 33.7±4.6 [b] | 36.6±3.7 [b] | 0.001 |
| Visceral adipose tissue (mm) | 54.1±25.3 [a] | 67.3±20.8 [a,b] | 84.3±20.3 [b] | 0.006 |
| Systolic BP (mmHg) | 114±16 [a] | 116±17 [a] | 126±16 [b] | 0.049 |
| Diastolic BP (mmHg) | 77±12 | 80±11 | 83±10 | 0.245 |
| Total cholesterol (mg/dl) | 176.5±31.2 | 185.6±40.9 | 190.8±41.4 | 0.462 |
| HDL cholesterol (mg/dl) | 53.0±10.9 [a] | 51.2±13.1[a] | 37.9±6.3 [b] | 0.001 |
| LDL cholesterol (mg/dl) | 104.5±28.4 | 112.5±32.0 | 118.2±35.5 | 0.383 |
| Triglycerides (mg/dl) | 94.8±52.1 [a] | 137.9±78.2 [a,b] | 161.7±94.0 [b] | 0.007 |
| Uric acid (mg/dl) | 3.9±0.9 [a] | 4.4±1.0 [a] | 5.6±1.3 [b] | 0.001 |
| Fasting glucose (mg/dl) | 92.4±15.6 [a] | 119.9±51.5 [a,b] | 133.3±43.9 [b] | 0.001 |
| Glycated hemoglobin (%) | 4.7±1.0 [a] | 6.3±2.0 [b] | 6.8±1.5 [b] | 0.001 |
| ISI ($mg.kg_{ffm}^{-1}.min^{-1}.\mu U/l$) | 0.70±0.76 [a] | 0.24±0.19 [b] | 0.22±0.31 [b] | 0.001 |
| Adiponectin (ng/ml) | 3.9±1.8 [a] | 2.6±1.6 [b] | 1.6±0.7 [b] | 0.001 |
| C-reactive protein (mg/dl) | 0.33±0.53 [a] | 0.38±0.39 [a] | 0.73±0.61 [b] | 0.010 |

Data are presented as the mean±standard deviation. Abbreviations: BP (blood pressure), GIR (glucose infusion rate), ISI (insulin sensitivity index). The Kruskal–Wallis test followed by Bonferroni's post-hoc test was used for systolic and diastolic BP, triglycerides, fasting glucose and ISI. One-way ANOVA followed by Tukey's post-hoc test was utilized for the other variables.