ORIGINAL ARTICLE

Anterior abdominal wall adiposity in pregnant women of adequate weight and fetal growth

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ABSTRACT

Introduction: the increase of abdominal adipose tissue in early pregnancy causes metabolic changes that promote insulin resistance, which is linked to alterations in fetal growth detectable by ultrasonography.
Objective: to determine the association of the variation of the adipose strata of the anterior abdominal wall with the presence of insulin resistance and with fetal biometry.
Methods: longitudinal analytical study in a population of 144 normopoietic pregnant women, apparently healthy, with optimal reproductive age, nulliparous, belonging to the Polyclinic "Chiqui Gómez Lubián" of Santa Clara City. The sample was 123. Theoretical, empirical and statistical methods were used.
Results: subcutaneous fat decreases as preperitoneal fat increases from the first to the second trimester, both expressed with greater intensity in pregnant women who did not have insulin resistance at the beginning of gestation. In the pregnant women who did not have insulin resistance, all the biometric variables decreased in the classification of fetuses appropriate for gestational age from the second to the third trimester and increased the frequencies of small and large fetuses, while in those who had insulin resistance, the cephalic circumference and the abdominal circumference showed stability in the identification of large fetuses.
Conclusions: the lower variation of the adipose strata of the anterior abdominal wall is related to the presence of insulin resistance at the beginning of gestation in pregnant women in whom the biometric variable abdominal circumference is associated with the identification of large fetuses for gestational age from the second trimester.
Key words: insulin resistance; abdominal adiposity; fetal growth; fetal biometry

RESUMEN

Introducción: el incremento del tejido adiposo del abdomen al inicio del embarazo suscita cambios metabólicos que promueven la resistencia a la insulina, la que se vincula a alteraciones del crecimiento fetal detectables mediante ultrasonografía.

Objetivo: determinar la asociación de la variación de los estratos adiposos de la pared abdominal anterior con la presencia de resistencia a la insulina y con la biometría fetal.
Métodos: estudio analítico longitudinal en una población de 144 gestantes normopeso, aparentemente sanas, con edad reproductiva óptima, nulíparas, pertenecientes al Policlínico "Chiqui Gómez Lubián" de la Ciudad de Santa Clara. La muestra fue de 123. Se emplearon métodos teóricos, empíricos y estadísticos.
Resultados: la grasa subcutánea disminuye en la medida que incrementa la grasa preperitoneal del primero al segundo trimestre, ambos expresados con mayor intensidad en las gestantes que no tuvieron resistencia a la insulina al inicio de la gestación. En las gestantes que no tuvieron resistencia a la insulina todas las variables biométricas disminuyen en la clasificación de fetos adecuados para la edad gestacional del segundo al tercer trimestre e incrementan las frecuencias de pequeños y grandes, mientras que en las que tenían resistencia a la insulina la circunferencia cefálica y la circunferencia abdominal muestran estabilidad en la identificación de fetos grandes.
Conclusiones: la menor variación de los estratos adiposos de la pared abdominal anterior se vincula con la presencia de resistencia a la insulina al inicio de la gestación en las gestantes en las que se asocia la variable biométrica circunferencia abdominal con la identificación de los fetos grandes para la edad gestacional desde el segundo trimestre.
Palabras clave: resistencia a la insulina; adiposidad abdominal; crecimiento fetal; biometría fetal

INTRODUCTION

Abdominal obesity in the common population generates metabolic alterations even in the absence of general adiposity, which during pregnancy promotes the increase of insulin resistance (IR),[1] a marker of cardiometabolic risk for which ultrasonography is a very useful method because it allows discriminating the adipose compartments of the abdomen at the beginning of gestation and to approach its diagnosis.[2]
Abdominal obesity is associated with metabolic alterations such as low HDL (high density lipoprotein) cholesterol levels, high serum triglyceride and total cholesterol values, glycemia alterations and hypertension.[3]
Among the adaptations of the female organism during gestation is the mobilization of reserve adipose tissue which produces an increase in adipose deposits both in the abdominal walls and in the abdominal cavity. In the cavity the major endocrine-metabolic disorders are triggered: atherogenic, prothrombotic and inflammatory alterations that significantly increase the risk of developing cardiovascular disorders.[4]
The increase in visceral adipose tissue in pregnant women leads to the dysregulated release of adipocytokines, fatty acids and inflammatory markers that provoke IR, so that the triad obesity, inflammation and pregnancy represents a dangerous relationship for maternal and fetal health.[5]
Chronic inflammation, oxidative stress and IR during pregnancy, with the consequent increase in blood glucose, have the capacity to alter fetal growth.[6]
Fetal ultrasounds, despite being the most important tests during pregnancy, have failed to demonstrate stability of fetal growth;[7] however, few longitudinal studies of intrauterine growth and of the impact of different maternal analytical variables on fetal growth have been performed.[8]

Some studies evaluate the differences in fetal growth trajectory referred to the overweight status of the pregnant woman or its consequences, but they do not discriminate whether the true causes of fetal growth impairment are overweight or associated comorbidities.[9]
A recent study proposes the alternative of using fetal weight standards and percentiles personalized to maternal characteristics, finding a greater association with the prediction of fetal weight in maternal characteristics than in the variations of biometric variables.[10]
The study of this problem constitutes a highly topical issue for the improvement of prenatal care actions, which has as an added factor the knowledge of fetal growth alterations linked to the adipose distribution of the abdomen. The present work aims to determine the association of the variation of the adipose strata of the anterior abdominal wall during the first and second trimesters with the presence of insulin resistance at the beginning of gestation and with fetal biometry.

MÉTODOS

Design and population

A longitudinal analytical study was carried out in the "Chiqui Gómez Lubián" University Polyclinic of Santa Clara City, Villa Clara Province, in a population of 144 apparently healthy pregnant women of adequate weight, with optimal reproductive age, nulliparous and singleton pregnancies, who were recruited before 12.6 weeks of gestation, in the period from November 2018 to February 2019. A non-probabilistic sample of 123 pregnant women was obtained according to inclusion criteria (Cuban citizenship, residence in the Municipality of Santa Clara and express consent to participate in the research), exclusion criteria (having achieved pregnancy as a result of ovulation stimulation, drugs or assisted reproductive technology, habitual consumption of licit or illicit drugs in a period of less than one year) and exit criteria (fetal loss during pregnancy, interruption of pregnancy due to genetic indication, transfer of health area and voluntary abandonment of the study).

Techniques and procedures for data collection

The study was carried out in the clinics of the health area scheduled for the prenatal care of the pregnant woman from the moment of enrollment. The results of the laboratory tests indicated at enrollment (glycemia and triglycerides) were obtained by means of a documentary review.
At the same time as the fetal biometry of the first and second trimesters, ultrasonographic measurements of the adipose layers of the anterior abdominal wall were performed. The results of the third trimester biometry were obtained in correspondence with the attendance to its performance. All ultrasonographic measurements were performed with a high resolution Toshiba equipment and with a 3.5 MGz linear transducer, all of them were performed by the same specialist in General Comprehensive Medicine with a diploma in Ultrasonography.
All the information was recorded in a data collection form.

Study variables

Maternal variables:

- Insulin resistance: determined through the glucose and triglyceride index (TGC). These results were obtained from the processing of blood samples in the clinical laboratory of the health area. Glucose and TGC index= Ln [TGC (mg/dl)×glucose (mg/dl)/2] (quantitative value).[11] Normal value not defined for pregnant population by this method. The variable was transformed to qualitative based on the 75th percentile value of the study population (8.43) and was considered:
  - No insulin resistance: if index is <8.43
  - If insulin resistance: if glucose index and GFR ≥8.43
- Abdominal subcutaneous fat (SCF) (minimum) (mm): panniculus adiposus of the upper half of the anterior abdominal wall above the umbilicus at the level of the linea alba (minimum subcutaneous thickness). It is measured perpendicular to the surface between the skin and the linea alba from the xiphoid appendix by moving the transducer perpendicularly in the direction of the umbilicus.
- Preperitoneal fat (PF) (maximum) (mm): layer of adipose tissue between the linea alba and the visceral sheet of peritoneum that lines the diaphragmatic side of the liver. It is measured by placing the transducer perpendicular to the body surface, at the level of the xiphoid appendix and moving it over the linea alba in the direction of the umbilicus.

Fetal biometric ultrasonographic variables:

- Biparietal diameter (BPD): measurement made between the outer surfaces of both parietal bones in the widest portion of the skull.[12]
- Fetal head circumference (HC): calculated from the BPD and fronto-occipital diameter using the formula CC= π (BPD +DFO)/2.[12]
- Fetal abdominal circumference (AC): calculated from the anteroposterior and transverse diameters of the fetal abdomen using the formula CA= π (APAD+TAD)/2.[12]
- Femoral length (FL): taken between the outer surfaces of the bone ends without measuring the trochanter.[12]

  To estimate fetal size through biometric parameters (adjusted trophic condition) it is taken into account that the population in which the weight is between the 10th and 90th percentiles of the growth curve is considered normal. This procedure allows classifying fetuses according to their gestational age as small (PEG), adequate (AEG) or large (GEG) depending on whether they are below the 10th percentile, between 10 and 90 or above 90 according to the fetal growth charts in force in Cuba.[13]

Data analysis and processing

The information was stored and processed in a file prepared in the SPSS statistical package version 20.0 for Windows according to the proposed objective.

Measures of central tendency and position (median and interquartile range) were used as summary measures for quantitative variables. Prior to the analysis of quantitative variables, normal distribution was tested and, in its absence, the nonparametric Mann-Whitney U test was used to compare

independent variables; in the case of related samples, the Wilcoxon signed-rank test was applied.
The qualitative variable is expressed by frequency distributions in absolute and relative values (number and percent). To explore the differences between the variables according to the presence of insulin resistance, the homogeneity test based on the Chi-square distribution was performed; given the limitations of the test, the exact probability available in the program was used.
For all hypothesis tests a significance level α=0.05 was prefixed for statistical decision making.
The results were expressed in statistical tables.

Ethical aspects

The research was governed by the ethical principles that guide medical research with human beings, as set forth in the Declaration of Helsinki,[14] and had the approval of the Research Ethics Committee of the health area involved and the informed consent of the pregnant women.

RESULTADOS

Table 1 describes the subcutaneous and preperitoneal anterior abdominal wall adipose strata in the first two trimesters of gestation. Subcutaneous fat significantly decreased its values between the first and second trimester; lower values were also present at the lower and upper limits of the interquartile ranges in the second trimester. Preperitoneal fat showed a median increase between the first and second trimesters. Consequently, there were higher values at the lower and upper limits of the interquartile ranges in the second trimester.
In both cases the comparison of the variable between the two trimesters when applying the Wilcoxon signed-rank test for related samples showed significant statistical differences (0.0001).

Table 1. Description of the subcutaneous and preperitoneal anterior abdominal wall adipose strata in the first and second trimesters

Adipose strata of the anterior abdominal wall	First trimesters	Second trimesters	p**
	Median Interquartile range		
Subcutaneous fat	12.2 10.0-14.8	10.6 8.5-12.7	0.0001
Preperitoneal fat	10.2 8.5-12.0	11.6 9.5-13.7	0.0001

**significance of the test for related samples of ranges with Wilcoxon signed ranks

The variation of the subcutaneous and preperitoneal anterior abdominal wall adipose strata in the first two trimesters of gestation according to the presence or absence of IR at the beginning of pregnancy is shown in Table 2. The statistical test for related samples of ranges with Wilcoxon sign yielded significant differences for both adipose strata between the first and second trimesters, regardless of the presence or absence of insulin resistance;

differences that were less significant for preperitoneal fat in pregnant women with IR.

Subcutaneous fat values in the first trimester were similar between pregnant women with and without IR, while in the second trimester their values were slightly higher in those with IR. First trimester preperitoneal fat showed slightly higher values in pregnant women with IR at the beginning of pregnancy, while in the second trimester the median values and interquartile ranges were very similar in both groups. In none of the cases were there significant statistical differences when applying the nonparametric Mann-Whitney U test (p>0.05).

Table 2. Description of subcutaneous and preperitoneal anterior abdominal wall adipose layers in the first two trimesters of gestation according to the presence of insulin resistance

Adipose strata of the anterior abdominal wall	Insulin resistance	First trimesters	Second trimesters	p**
		Median Interquartile range		
Subcutaneous fat	No (n=92)	11.6 9.9-14.8	10.4 8.3-12.7	0.0001
	Si (n=30)	11.6 10.6-14.8	10.6 9.5-13.0	0.001
p*		0.430	0.210	
Preperitoneal fat	No (n=92)	10.1 8.5-11.8	11.6 9.4-13.4	0.0001
	Si (n=30)	10.2 9.2-12.3	11.6 9.9-13.7	0.041
p*		0.564	0.686	

*significance of the nonparametric Mann-Whitney U-test for independent samples
**significance of the Wilcoxon signed ranks test for related samples

Table 3. Second and third trimester fetal biometry adjusted for trophic status according to the presence of insulin resistance

Fetal Biometry adjusted to its trophic condition		Second trimester					Third trimester				
		Insulin resistance									
		No (n=92)		Yes (n=30)		x^2 (p)	No (n=92)		Yes (n=30)		x^2 (p)
		n	%	n	%		n	%	n	%	
BPD	Small	5	5.4	0	0	3.487 (0.395)	9	9.8	0	0	9.794 (0.012)
	Suitable	86	93.5	30	100		76	82.6	30	100	
	Large	1	1,1	0	0		7	7.6	0	0	
HC	Small	10	10.9	4	13.3	1.784 (0.390)	11	12.0	1	3.3	5.295 (0.103)
	Adequate	67	72.8	24	80		65	70.6	27	90	
	Large	15	16.3	2	6.7		16	17.4	2	6.7	
AC	Small	4	4.3	2	6.7	0.796 (0.610)	7	7.6	3	10	0.542 (0.767)
	Adequate	70	76.1	24	80		68	73.9	23	76.7	
	Large	18	19.6	4	13.3		17	18.5	4	13.3	
FL	Small	5	5.4	4	13.3	1.843 (0.305)	8	8.7	1	3.3	1.987 (0.347)
	Adequate	84	91.3	25	83.4		79	85.9	29	96.7	
	Large	3	3.3	1	3.3		5	5.4	0	0	

BPD: biparietal diameter; HC: head circumference; AC: abdominal circumference; FL: femoral length

Table 3 shows the frequency of fetal biometric variables adjusted for second and third trimester fetal trophic condition according to the presence of insulin resistance at early gestation. In insulin-sensitive pregnant women all biometric

variables decrease in the classification of adequate for gestational age from the second to the third trimester and increase the frequencies of small and large, except for AC in which the large status slightly decreases (from 19.6% to 18.5%).
In pregnant women with IR at the beginning of gestation, the behavior of the variables varies: BPD variable identifies all fetuses in the condition of adequate for gestational age in both trimesters, HC decreases the identification of small (from 13.3% to 3.3%) with stability in the frequency of large (2, 6.7%), AC increases the frequency of small (from 6.7% to 10%) and large remains stable (four, 13.3%), FL decreases in both small (from 13.3% to 3.3%) and large (from 3.3% to zero).

DISCUSSION

Abdominal adipose distribution, specifically in the visceral compartment, is recognized as an important determinant of cardiometabolic diseases,[15] determined by the dysfunction of the adipose tissue in which changes in the secretory profile and hypertrophy of adipocytes occur, as well as infiltration of the adipose tissue by inflammatory cells, causing alteration of communication with other organs.[16]
The mobilization of reserve adipose tissue during gestation is related to an important part of the changes that occur, fundamentally with the increase in abdominal adiposity, which when it occurs in the intra-abdominal compartment is the cause of the main dysregulations of glucose, insulin, lipid and amino acid metabolism that can cause perinatal effects.[17,18]
The decrease in subcutaneous fat and increase in preperitoneal fat from the first to the second trimester found in the present study is indicative of the migration that occurs in adipose tissue from one compartment to another.
There are differences in the deposition of visceral adipose tissue during gestation in pregnant women of different nutritional status.[19] They describe a pattern of evolution of abdominal adipose tissue in which preperitoneal fat increases significantly throughout gestation, while subcutaneous fat decreases, although the latter decreases at a more accelerated rate in normopese pregnant women.
The accumulation of intra-abdominal fat, expressed at this stage of gestation by preperitoneal fat, is greater than the accumulation of subcutaneous fat, from which it is inferred that the changes in lipid metabolism and insulin sensitivity that occur are a reflection of the greater accumulation of preperitoneal fat.[20]
Correspondingly, the results of the present study demonstrate the link between insulin resistance at the beginning of gestation and the evolution of the subcutaneous and preperitoneal adipose strata of the anterior abdominal wall, with a more physiological decrease in abdominal subcutaneous fat and an increase in preperitoneal fat in the absence of IR at the beginning of gestation.
The loss of insulin sensitivity during gestation translates into an increased supply of nutrients such as glucose and lipids to the fetus, causing fetal overgrowth and obesity.[21] A recent study reflects the impact of central adiposity on perinatal morbidity and infant anthropometry by associating the ultrasonographic measurement of visceral adipose tissue with excessive fetal

growth and development and with the trophic condition of large for gestational age at birth.[22]

In the present study, in the presence of insulin resistance from early gestation, the biometric variables HC and AC showed greater stability in the detection of large fetuses for gestational age. Fetal AC, being an indicator of abdominal organ growth and subcutaneous fat deposition, is strongly influenced by maternal nutritional status[23] and its association with serum triglyceride levels and with neonatal anthropometric measurements has been demonstrated,[24] so it is considered useful in the evaluation of fetal growth in specific populations.

The present research contributes to the explanation of the evolution of the adipose strata of the anterior abdominal wall and its association with insulin resistance in pregnant women who begin pregnancy with adequate weight, as well as the variations in fetal growth and development expressed in the variations of the basic anthropometric variables.

CONCLUSIONS

The variation of the adipose layers of the anterior abdominal wall between the first and second trimester, due to a decrease in subcutaneous fat and an increase in preperitoneal fat, is less marked in pregnant women with insulin resistance at the beginning of gestation, in whom the fetal biometric variable abdominal circumference is associated with the identification of large-for-gestational-age fetuses from the second trimester.

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

AUTHORSHIP CONTRIBUTION

CRM: conceptualization, data curation, research, fund acquisition, project management, methodology, visualization, writing (review and editing).
NLSM: conceptualization, data curation, methodology, monitoring, validation, visualization, writing (review and edit).
EÁGG: conceptualization, data curation, resources, research, visualization, writing (review and edit).
OCL: visualization, supervision, writing (review and edit).
AAS: conceptualization, data curation, methodology, writing original draft.
COM: data curation, formal analysis, writing (reviewing and editing).