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Association between visceral fat and components of metabolic syndrome in young Mexicans: a preliminary study

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ABSTRACT

Introduction: Obesity in young adults is a worldwide growing concern, increasing the risk of metabolic syndrome (MetS) and type 2 diabetes. This study explores whether healthy young individuals with a visceral fat area over 100 cm² show early metabolic alterations and investigates potential molecular mechanisms, including RGS2 expression, to better understand the onset of insulin resistance before clinical disease appears.

Objectives: Since a wide percentage of people who develop metabolic syndrome come from being overweight or obese, it is important to identify whether a higher area of visceral fat could be a diagnostic strategy associated with insulin resistance and higher levels of RGS2 in the young population.

Methods: Healthy male and female participants underwent routine medical evaluations and were grouped based on visceral fat area (VFA). Individuals with VFA < 50 cm² were classified as the Low VFA group, while those with VFA > 100 cm² comprised the High VFA group. Anthropometric assessments and serum biochemical analysis were performed to estimate insulin resistance, waist-to-height ratio, triglyceride-to-HDL ratio, and MetS severity score. Finally, mRNA was extracted to calculate the SERCA pump and RGS2 gene expression through qRT-PCR.

Results: Young participants with High VFA exhibited MetS signs, higher HOMA-IR values, and elevated waist-to-height and triglyceride-to-HDL ratios. The MetS z-score and a strong positive correlation between VFA and MetS severity ($r = 0.8307$, $p < 0.0001$) further support the relationship between central adiposity and metabolic dysfunction. Additionally, young participants with High VFA tended to have higher levels of RGS2 gene expression, targeting this protein as a potential therapeutic target.

Conclusion: Our findings demonstrate the role of exacerbated visceral adiposity in the metabolic risk in young people, despite their apparent healthy condition.

KEYWORDS

Obesity, visceral fat area, insulin resistance, metabolic syndrome, regulator of G-protein signaling-2.

INTRODUCTION

Obesity constitutes a serious public health concern worldwide. According to the latest National Health and Nutrition Survey[1] in Mexico, the levels of overweight and obesity are elevated in more than a third of the adolescent and young adult population. Overweight and obesity are conditions that increase the risk of developing non-communicable metabolic diseases, such as hypertension, cardiovascular diseases, metabolic syndrome (MetS), and type 2 diabetes mellitus (T2DM), by disturbing several molecular mechanisms affecting metabolic health. Given the health risks associated with MetS, it's essential to identify its clinical signs as early as possible to enable timely intervention and potentially reverse its effects to prevent its clinical evolution to T2DM.

For instance, the etiology of MetS is generally recognized as multifaceted; still, its essential components are insulin resistance and obesity[2], and some studies underscore the importance

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of metabolic status, independent of body mass index, in predicting cardiovascular risk[3]. In adults, a visceral fat area (VFA) over 100 cm² is widely recognized as an increased risk threshold for metabolic and cardiovascular diseases[4,5]. However, in young adults, although visceral fat accumulation is concerning and is associated with cardiovascular risk factors, there are no defined thresholds or international consensus for the clinical use of VFA as a risk predictor[6].

In this respect, excessive visceral fat accumulation is associated with alterations in lipid metabolism and the development of dyslipidemia. Although the molecular mechanisms involved in the pathogenesis of MetS have not yet been fully resolved, it is known that increased levels of fatty acids can impair the action of insulin, further contributing to insulin resistance[7,8]. In this sense, previous studies have highlighted alterations in the expression of the G protein signaling regulator 2 (RGS2) and sarco/endoplasmic reticulum calcium ATPase (SERCA) as possible mechanisms that contribute to the development of insulin resistance and type 2 diabetes mellitus (T2DM) due to saturated fatty acids[8,9]. However, to focus on RGS2 and SERCA as potential therapeutic targets, it is crucial to determine whether the expression of these two proteins would be altered in young, Apparently healthy individuals with insulin resistance before the onset of MetS or T2DM.

Accordingly, it is of great importance to understand the establishment and evolution of metabolic disorders in the young population, considering most of the studies are directed at the description of molecular mechanisms of obesity and its associated disturbances in the elderly population, so several aspects of the pathogenesis are not well understood in young people[11-13]. Thus, this study aimed to evaluate whether apparently healthy young people with a visceral fat area greater than 100 cm² present metabolic alterations characteristic of MetS, as well as to investigate the possible molecular causes involved.

MATERIALS AND METHODS

Ethical Statement

The protocols carried out in the present study were previously approved by the Hospital General 5 de Diciembre of ISSSTE Mexicali, Mexico, Ethics Committee (Circular letter number 0985/2017), following the principles of the Declaration of Helsinki, as revised in 2000. The participants' informed consent was obtained and documented before initiating the research protocol.

Subjects

Participants were recently admitted students at the Sports School of the Universidad Autónoma de Baja California, who underwent a medical examination as a requirement to enroll in sports activities. The present double-blind study considered 47 apparently healthy male and female patients. The inclusion criteria required participants to be between 18 and 28 years old and to show no signs or symptoms of disease, while the exclusion criteria disqualified anyone with a medical condition or who was pregnant. After a cluster analysis based on MetS criteria, the participants were divided into two experimental groups per their visceral fat area (VFA) content: Low VFA when it was less than 50 cm² given that they were all participants of both gender who did not present any metabolic alteration and will act as control; and High VFA when over 100 cm². In the search for the problem group, there is solid data showing that a VFA greater than 100 cm² is associated with metabolic alterations characteristic of obesity in both women and men; therefore, this group was classified as High VFA[5] (Figure 1).

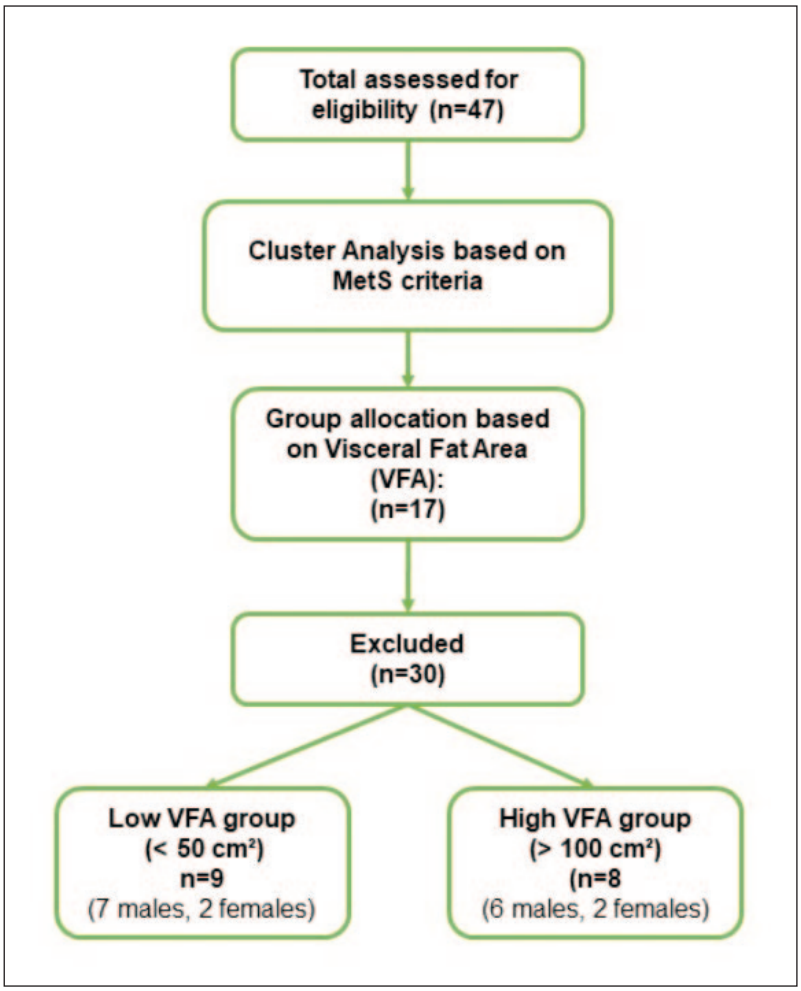


Figure 1. Flowchart of the study selection process

Determination of body and serum parameters

The body mass index (BMI) was calculated by dividing weight by height squared. Height measurement was determined with a portable SECA 213 stadiometer. Waist circumference was measured with millimeter precision using a SECA 201 ergonomic tape. The body fat percentage and the visceral fat area were determined by bioelectrical impedance (InBody 720, Hennock Road East, Marsh Barton, UK). Blood pressure was registered after at least 10 minutes of rest using a Welch Allyn OSZ 5 digital monitor.

For the serum parameters, after fasting overnight, 5 mL of peripheral blood was collected into polypropylene tubes with sodium citrate (Vacutainer System, BD Biosciences). Blood was centrifuged at 4000 r.p.m. for 15 minutes to obtain the serum, which was used to determine total cholesterol, glucose, triglycerides, LDL-Cholesterol, and HDL-Cholesterol in an automatic analyzer (Spin 120, SpinReact, Barcelona, Spain). HOMA-IR was calculated by multiplying the fasting insulin level (in micro-units per milliliter) by the fasting glucose level (in milligrams per deciliter) and dividing the result by 405. Waist-to-height ratio was calculated by dividing the waist circumference by the height, both in centimeters. The triglyceride to HDL ratio is calculated by dividing the level of triglycerides by the level of HDL cholesterol, both in mg/dL. MetS severity score was calculated using the software MetS Calc following the authors' indications[14,15].

Insulin and adiponectin levels determination

Insulin and adiponectin serum levels were determined by enzyme-linked immunosorbent assay (ELISA) using a specific kit for each protein and following the manufacturer's instructions (Insulin IN374S, Calbiochem, CA, USA; Adiponectin EZHADP-61K, Merck Millipore, Darmstadt, Germany). Optical density was detected at 450 nm using a microplate absorbance reader (iMark, BioRad).

Quantitative reverse transcriptase polymerase chain reaction (qRT-PCR) assay to determine RGS2 and SERCA mRNA levels

Total RNA was isolated from blood tissue using TRIZOL reagent (Life Technologies, 15596026, USA). A reverse transcription was performed using the iScript gDNA Clear cDNA synthesis kit (Bio-Rad, 1725035, USA). The amplification primers for RGS2 were forward 5′-CCT CAA AAG CAA GGA AAA TAT ATA CTG A-3′ and reverse 5′-AGT TGT AAA GCA GCC ACT TGT AGC T-3′ [16]. The amplification primers for SERCA were forward 5´-TTA AAG CAA CTG TCT ATT TCT GCT G-3´ and reverse 5´-AGT CAG AAA AAG CAA AAC AAA ATC TA-3´ [17]. The conditions for cDNA synthesis were 25°C for 5 min, 46°C for 20 min, and 95°C for 1 min. Real-time PCR was performed to measure gene expression using SYBR® Green Supermix (Bio-Rad, 1725270, USA). PCR reactions were performed in volumes of 20 µl; the detailed PCR reaction was as follows: for the initial denaturation, it was subjected to 95 °C for 30 seconds, followed by 35 cycles of denaturation, alignment, and extension at temperatures of 95°C for 10 s, 57°C for 30 s, and 65°C for 5 s, respectively. The final extension was performed at 95°C for 5 minutes, followed by cooling to 4°C. Relative gene expression levels were determined by RT-qPCR, normalized to GAPDH. The amplification primers for GAPDH were forward 5′- GTC TCC TCT GAC TTC AAC AGC G-3′ and reverse 5′- ACC ACC CTG TTG CTG TAG CCA A-3′ [18].

Statistical Analysis

Average values were analyzed using unpaired t-tests, while a Spearman correlation analysis (r) and test were performed to correlate the raw data. GraphPad PRISM version 9.5.0 for macOS (GraphPad Software, Inc., San Diego, CA, USA) was used for both analyses. A p-value < 0.05 was considered statistically significant, and data are presented as the mean ± standard error (SE).

RESULTS

High VFA in young participants is associated with metabolic syndrome components.

Evaluation of body composition and serum parameters demonstrated that individuals with a High VFA (greater than 100 cm²) exhibited at least three metabolic syndrome-related alterations, such as high blood pressure, increased waist circumference, and dyslipidemia (Supplementary table 1). In comparison, those with a Low VFA (less than 50 cm²) showed no metabolic abnormalities (Table 1).

High VFA in young participants is associated with insulin resistance and increased metabolic risk

To determine insulin resistance and metabolic risk depending on VFA, we have determined the blood concentration of adiponectin and calculated some indices based on body composition and blood data (Figure 2). Concerning the waist-to-height ratio (Figure 2A) and the triglycerides to HDL-cholesterol (Figure 2B), both indices were significantly increased in the High VFA group, indicating a higher metabolic risk in this group. A Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) analysis was conducted to estimate the insulin resistance state, revealing a higher HOMA-IR in the High VFA group than the Low VFA group (Figure 2C). In this sense, adiponectin, a cytokine produced by the adipose tissue that exerts metabolic protective effects in several tissues and regulates insulin sensitivity, did not change significantly between groups; it did show a downward trend in the High VFA group (Figure 2D).

High VFA in young participants is associated with increased metabolic syndrome severity

Interestingly, the MetS z-score calculation indicated that those in the Low VFA group had a lower degree of MetS

Table 1. Participants´ general characteristics

PARAMETERS	GROUP		*P value*
	Low VFA	High VFA	
BMI (kg/m^2)	20.01 0.5	33.63 1.7 **	0.000001
Waist circumference (cm)	71.29 1.8	105.59 4.3 **	0.000002
Body fat percentage (%)	12.79 1.8	39.31 2.8 **	0.000001
Visceral fat area (cm^2)	24.07 4.5	154.52 10.9 **	0.00000001
Systolic blood pressure (mm Hg)	110.67 1.4	138.75 8.6*	0.004
Diastolic blood pressure (mm Hg)	64.56 1.5	86.00 3.5 **	0.00003
Glucose (mg/dL)	84.33 3.1	98.88 3.6 *	0.01
Triglycerides (mg/dL)	80.56 4.2	152.75 17.8 *	0.001
Total cholesterol (mg/dL)	133.00 6.0	169.13 11.6 *	0.01
HDL-cholesterol (mg/dL)	45.78 2.5	40.75 6.3	0.45
LDL-cholesterol (mg/dL)	87.22 6.1	128.38 7.8 *	0.001
Insulin (IU/mL)	6.58 1.6	14.76 3.3 *	0.04

Values are mean ± S.E.M of N=9 for Low VFA and N=8 for High VFA. BMI, body mass index; HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; VFA, visceral fat area. *P ≤ 0.05 and **P ≤0.0001 vs. Low VFA.

severity than the average, while the High VFA group presented a positive MetS severity score, meaning more risk for future metabolic disease (Figure 3A). Indeed, there was a robust and statistically significant positive correlation between MetS severity and VFA ($r = 0.8307$, $p < 0.0001$), suggesting that individuals with higher VFA tend to have more severe manifestations of metabolic syndrome (Figure 3B).

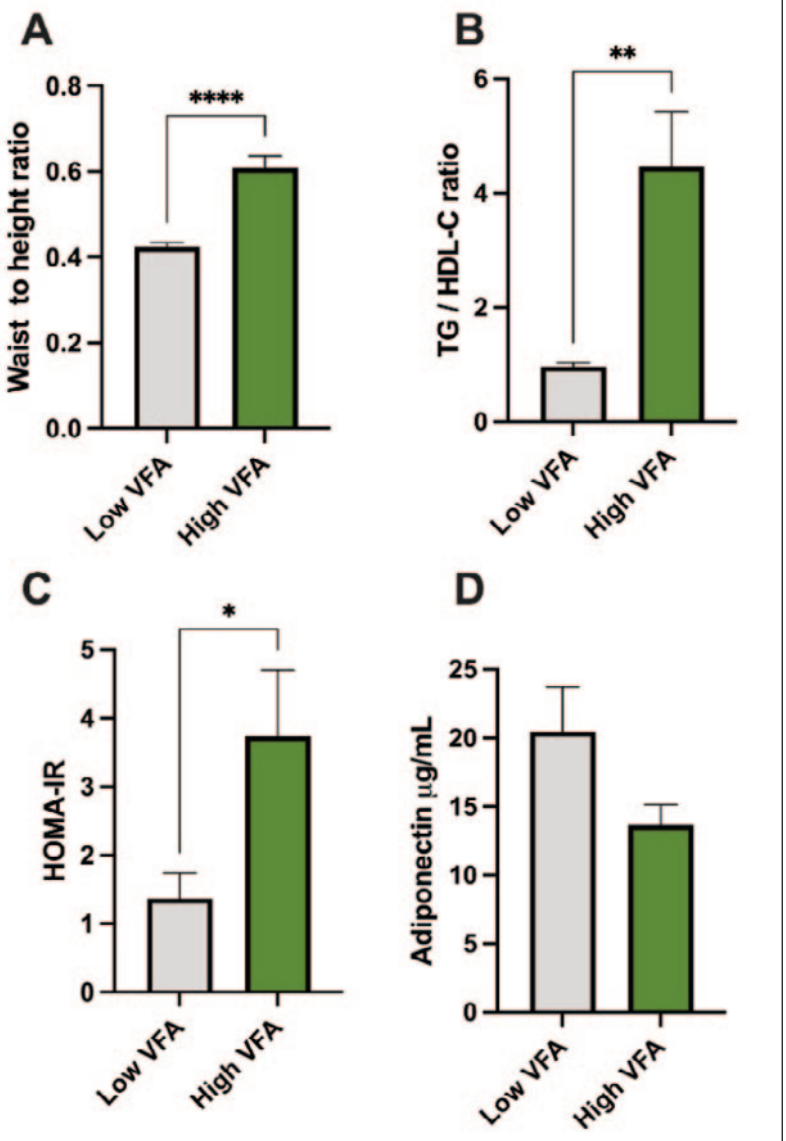
Young participants with High VFA tended to express higher levels of RGS2 mRNA.

In order to identify the molecular mechanisms associated with the genesis of type 2 diabetes, we aimed to determine whether there were alterations in the gene expression of RGS2 and the SERCA pump, given these proteins' association in insulin resistance molecular mechanisms. According to our RT-qPCR findings, there was no difference in SERCA pump gene expression between the Low and High VFA groups (Figure 4A), while for RGS2 there was a trend for the High VFA group to present a 3-fold increase in RGS2 gene expression compared to the Low VFA group (Figure 4B).

DISCUSSION

Despite the group being apparently healthy young people, the difference in visceral fat content proved to be a key factor for metabolic health in this population. In this sense, a study demonstrated the correlation of an increased abdominal circumference with insulin resistance, blood pressure, and dyslipidemia in pediatric patients[19]. Visceral fat accumulation is characterized by increased free fatty acid release, reactive oxidative species overproduction, an increase in proinflammatory cytokines such as IL-6 and TNF-α, and a decrease in anti-inflammatory cytokines like adiponectin. These physiological responses to obesity contribute to systemic inflammation, oxidative stress, and insulin resistance[20]. As depicted, the High VFA group requires increased insulin concentrations to maintain homeostatic glucose levels, as evidenced by the increased HOMA-IR data. So, based on the Murguía-Romero et al. report, which indicates that a HOMA-IR value greater than 2.6 is linked to insulin resistance in the Mexican population, the High VFA group presents insulin resistance[21].

Furthermore, the insulin resistance in the High VFA group was accompanied by a disturbed metabolic state as represented by the serum parameters results; in fact, the High VFA group presented at least three alterations related to MetS in contrast to the Low VFA group, which did not have any signs for MetS diagnosis. Related to this, the determination of some metabolic risk markers confirmed that the High VFA group is more likely to present conditions like cardiovascular diseases, T2DM, and MetS. In this regard, the MetS z-score revealed a healthier metabolic profile and lower disease risk for the Low VFA group, in contrast to the increased risk for MetS incidence in the High VFA group. Given the age of the participants, as has been



Bar graphs representing A. Waist to height ratio, B. Triglyceride-to-HDL-C ratio, C. Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), and D. Adiponectin levels of participants classified in Low VFA (visceral fat area) and High VFA groups. All values are presented as mean ± SE, obtained from N=9 for the Low VFA group and N=8 for the High VFA group. (*) p<0.05, (**) p<0.001, (****) p<0.0001 vs. Low VFA.

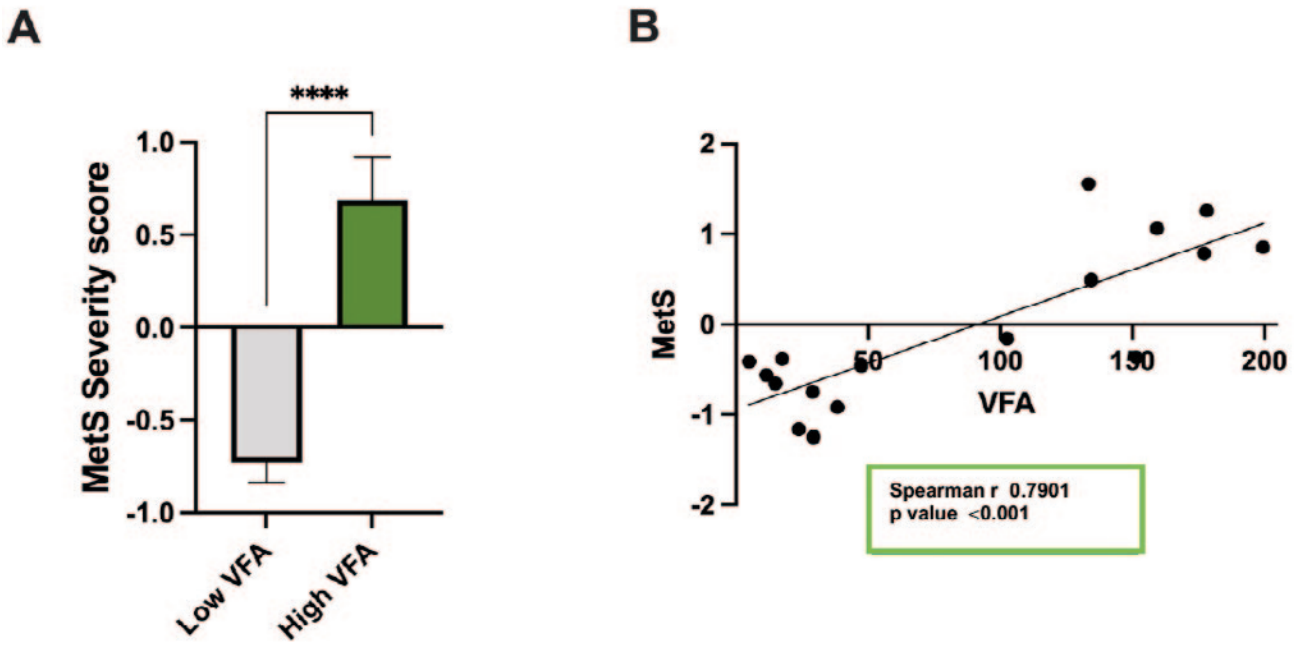
Figure 2. Metabolic risk indicators in Low VFA and High VFA young participants

shown in other studies, a higher MetS z-score in childhood predicted a greater likelihood of developing cardiovascular disease and T2DM years later[22]. The use of a z-score based on the sum of components to define MetS favors the specificity of the diagnosis in the face of factors such as gender or race, which have a greater influence on the clinical picture, providing the potential opportunity to identify young patients at higher risk, and encourage them to change their lifestyle, and track treatment progress over adulthood[23]. Moreover, our results demonstrate the strong correlation between the VFA and the MetS z-score in the young participants, confirming the report of Summer et. al. that establishes abdominal obesity as a strong predictor of higher MetS z-scores in US adolescents[24].

On the other hand, previous work from our laboratory has demonstrated the determining role of the RGS2 protein in the pathogenesis of the state of insulin resistance, both in cellular models of metabolic alterations and in platelets from patients with T2DM[9,25]. In fact, rodents fed a high-fat diet are reported to have increased visceral fat and higher levels of RGS2.[4,26]. Hence, to determine whether RGS2 is elevated in the platelets of individuals who have insulin resistance but have not developed T2DM, we identified individuals with a VFA greater than 100 cm², as it has been reported that these individuals present metabolic abnormalities such as insulin resistance despite appearing healthy. When RGS2 expression levels were analyzed in the final phase, it was found that while the High VFA group expressed RGS2 mRNA at higher levels, these increases were not statistically significant. Hence, factors other than increased RGS2 expression may contribute primarily to the development of insulin resistance in young humans. In this matter, it is essential to emphasize that obesity is a preceding stage of insulin resistance and T2DM development and that the increase in RGS2 expression is probably one of the numerous mechanisms limiting insulin action. However, it is noteworthy that many medications intended to promote glucose homeostasis reduce RGS2 expression due to these facts[27]. This study has some limitations that should be considered when interpreting the results. The small sample size limits the generalizability of the findings to other populations. Although a trend toward increased RGS2 expression was identified in the high-visceral fat group, this did not reach statistical significance. Finally, important variables such as lifestyle, diet, or physical activity level, which could influence metabolic outcomes, were not considered.

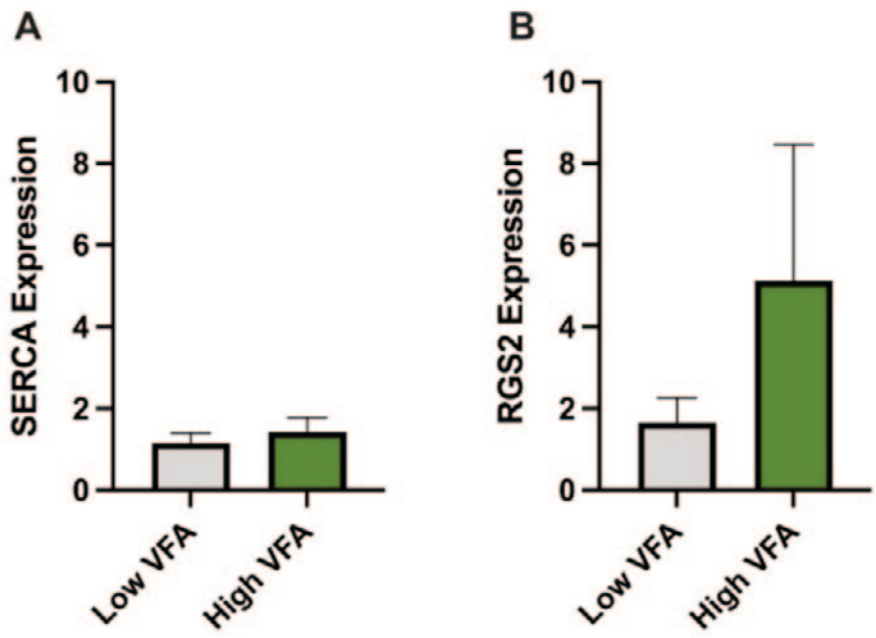
CONCLUSION

Our study demonstrates that a high visceral fat area in young individuals is strongly associated with multiple MetS



A. Metabolic syndrome severity score (Z-score) of participants classified in the Low VFA (visceral fat area) and the High VFA groups.
B. Dispersion graph representing the correlation between metabolic syndrome severity score (Z-score) and VFA of young participants. N= 17.

Figure 3. MetS severity score and visceral fat area correlation in Low VFA and High VFA young participants



SERCA and RGS2 mRNA levels in the blood of young individuals with Low VFA and High VFA. All values are presented as mean ± SE, obtained from N=9 for the Low VFA group and N=8 for the High VFA group.

Figure 4. mRNA expression levels of SERCA and RGS2 in Low VFA and High VFA young participants

Supplementary table 1. Anthropometric, biochemical and clinical data of apparently healthy university students participating

	Year to age	Gender	BMI (kg/m^2)	Waist circumference (cm)	Body fat percentage	Visceral fat area (cm^2)	Blood pressure mmHg		Glucose (mg/ml)	Triglycerides (mg/ml)	Total cholesterol (mg/ml)	HDL cholesterol (mg/ml)	LDL cholesterol (mg/ml)	Insulin (IU/mL)
							Systolic	Diastolic						
Low VFA	19	M	21.1	79	16.6	47.4	115	69	91	82	169	44	125	0.6
	20	M	17.9	66	10.1	14.9	105	67	95	76	136	42	94	13.7
	19	M	19.7	71	9.5	17.5	115	69	78	102	127	39	88	2.7
	18	M	20.3	69.1	12.5	23.7	127	78	68	68	107	49	58	5.2
	18	F	18.4	65.5	21.0	29.3	112	63	83	72	140	63	77	6.9
	19	M	22.1	78	12.2	29	114	64	87	69	126	47	79	14.0
	18	F	21.7	68	20.2	38.3	106	65	76	97	148	49	99	10.0
	18	M	19.7	78	6.7	11.5	106	54	84	70	119	38	81	4.4
High VFA	23	M	29.7	101	29.3	102.6	171	90	110	155	236	81	155	31.2
	24	M	29.3	104.5	29.5	133.4	140	90	105	215	183	23	160	14.2
	18	M	30.5	97.2	35.9	134.5	135	83	98	130	139	39	100	15.2
	18	F	30.7	91	40.9	151.4	109	70	91	80	186	48	48	7.9
	21	M	42.3	125	45.7	199.5	179	98	87	100	156	36	120	13.3
	27	M	35.6	124	44.6	178.2	124	82	114	152	146	33	113	24.6
	18	F	38.5	101	51.6	159.4	122	82	87	224	137	30	107	11.0
	18	M	32.4	101	36.8	177.2	125	82	99	166	170	36	134	0.8

components, insulin resistance, and increased metabolic risk. Additionally, a trend toward increased RGS2 gene expression in the High VFA group suggests a potential molecular link to early insulin resistance. Overall, these results highlight the importance of evaluating visceral adiposity and related biomarkers to identify at-risk individuals in the young population to prevent the appearance of clinical metabolic disorders by stopping their evolution.

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