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Triglycerides and glycated hemoglobin for screening insulin resistance in obese patients

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ABSTRACT

Keywords:

Insulin resistance
Obesity
Triglycerides
Dyslipidemias
Lipids
Glycated hemoglobin

Objective: Assessment of insulin resistance (IR) is essential in non-diabetic patients with obesity. Thus study aims to identify the best determinants of IR and to propose an original approach for routine assessment of IR in obesity.

Design and patients: All adult with obesity defined by a body mass index ≥ 30 kg/m², evaluated in the Nutrition Department between January 2010 and January 2015 were included in this cross-sectional study. Patients with diabetes were excluded. IR was diagnosed according to the HOMA-IR. Based on a logistic regression, we determined a composite score of IR. We then tested the variables with a principal component analysis and a hierarchical clustering analysis.

Results: A total of 498 patients with obesity were included. IR was associated with grade III obesity (OR = 2.6[1.6–4.4], $p < 0.001$), HbA1c $\geq 5.7\%$ (OR = 2.6[1.7–4.0], $p < 0.001$), hypertriglyceridemia > 1.7 mmol/l (OR = 3.0[2.0–4.5], $p < 0.001$) and age (OR = 0.98[0.96–0.99], $p = 0.002$). Exploratory visual analysis using factor map and clustering analysis revealed that lipid and carbohydrates metabolism abnormalities were correlated with insulin resistance but not with excessive fat accumulation and low-grade inflammation.

Conclusions: Our results highlight the interest of simple blood tests such as HbA1c and triglyceride determination, which associated with BMI, may be widely available tools for screening IR in obese patients.

1. Introduction

Obesity is classically associated with a constellation of metabolic pattern including insulin-resistance (IR), dyslipidemia and low-grade inflammation, now clustered as cardio-metabolic risk factors [1]. Face to patients with obesity, a major public health challenge is to identify individuals at high risk of both type 2 diabetes and cardiovascular diseases [1,2]. It is well accepted that a higher prevalence of IR is associated with abdominal obesity measured by waist circumference in routine [3]. Abdominal obesity is a major component of the metabolic syndrome, but it appears that it is not synonymous with IR [2,4]. Likewise, a subgroup of metabolically healthy individuals with obesity who might not be at increased risk of cardiovascular events, has been clinically recognized even if no universally accepted criteria exist to

define it [5]. Identifying IR phenotype has important clinical implications for interventional programs such as those aiming to delay the onset of diabetes. Most definitions of metabolic health take into account, insulin sensitivity, especially in non-diabetic obese patients [6]. Euglycemic hyperinsulinemic clamp is the gold standard for assessment of insulin resistance, but is cumbersome, making it difficult to use in clinical practice [7]. In obese subjects, it was reported that fasting insulin should be taken into account in the definition of metabolic normality [8]. Homeostasis model assessment (HOMA-IR) provides generally reliable measures of IR and it has been widely used in clinical studies as an index of IR based on fasting measurements including serum insulin [9,10]. However, insulin testing still suffers from a lack of standardization making the definition of a HOMA-IR cut-off value difficult and limiting its use for screening patients in clinical practice [11].

Abbreviations: IR, insulin-resistance; HOMA-IR, homeostasis model assessment; BMI, body mass index; HbA1c, glycated hemoglobin A1c; hsCRP, high sensitivity C-reactive protein.

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While the metabolic heterogeneity of non-diabetic patients with obesity is of clinical interest, routine assessment of IR remains a challenge. Accordingly, the aim of this cross-sectional study was to identify the best determinants of IR in our non-diabetic patients with obesity using an original statistical approach including logistic regression, principal component analysis and hierarchical model of classification.

2. Material and methods

2.1. Patients and methods

All consecutive patients with obesity (body mass index (BMI) ≥ 30 kg/m²) aged of 18 years or more, evaluated in the Nutrition and Diabetes Department between January 2010 and January 2015 were included in a cross-sectional study. Patients with diabetes, defined according to American Diabetes Association [12], were excluded from the study. None of the included patients had undergone bariatric surgery.

Height and weight were measured wearing underclothing and no shoes. Waist circumference was recorded to the nearest 0.1 cm midway between the last rib and the iliac crest using a non-stretch tape measure on lightly clad participants. Blood pressure was measured after 20 min of rest. Tobacco use was based on self-reports: current smoker was defined as patients with current tobacco smoking or former smoker who had quit for < 1 year. We used criteria of metabolic syndrome according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) definition [2] to define elevated waist circumference (waist circumference > 88 cm in women or > 102 cm in men). Hypertension was defined using blood pressure $\geq 130/85$ mmHg or treatment for hypertension. The study was performed according to the principles of the Declaration of Helsinki and was approved by the local ethics committee. All patients were entered into a registry with data collected from their hospitalization including anthropometric, clinical and biological information (N^oDC-2009-1052).

2.2. Laboratory procedures

Blood samples were collected after an overnight fast for measurement of total cholesterol, triglycerides, HDL-c, glucose, glycated hemoglobin A1c (HbA1c), insulin and high sensitivity C-reactive protein (hs-CRP). The LDL-cholesterol was calculated by the Friedwald formula. The majority of biochemical parameters were determined by standard methods on the AU2700 analyzer (Olympus, Rungis, France) until July 2012 and on the Cobas 8000 (Roche Diagnostic, Grenoble, France) analyzer from August 2012 with good correlation according to ISO15189 (glucose: $y = 1.01x - 0.014$, $r = 0.999$; triglycerides: $y = 0.9753x + 0.0915$, $r = 0.981$). Insulin was measured by ElectroChimiLuminescence (ECL) on the Cobas 6000 e601 (Roche Diagnostic, Grenoble, France) analyzer since January 2010. HbA1c was measured by HPLC (Menarini HA-8140V before June 2011 and G8, Tosoh Bioscience, Lyon, France from July 2011) and standardized against DCCT standard. The both methods were correlated and appeared interchangeable ($y = 0.975x - 0.0775$, $r = 0.982$) [13]. The cut-off of HbA1c ($\leq 5.7\%$) was chosen according to American Diabetes Association [12]. All parameters were measured immediately from each blood sample except insulin levels that were measured later from plasma stored at -80°C . A trained and qualified personnel was in charge of laboratory and anthropometric measurements during all period of enrollment at study. The coefficients of variation (CV) of each measurement were calculated for the six-year period, based on 2 levels of daily internal quality control and were lower than 3.0% (total cholesterol: 1.7%, HDL-c: 2.3%, triglycerides: 2.4%, plasma glucose: 1.9%, plasma insulin: 2.6%, hs-CRP: 3.0%, HbA1c: 1.9%).

IR was assessed with the HOMA-IR, calculated as followed: $[\text{glucose (mmol/l)} \times \text{insulin (UI/l)}] / 22.5$ [14]. Because of a lack of validated cut-off value, IR was diagnosed if HOMA-IR value was higher than the value of the 75th percentile of patients of the cohort with normal glucose regulation according to American Diabetes Association [12]. A cut-

off of 3.3 was defined for HOMA-IR among patients with obesity in our institution. In previous studies, the cut-off used for HOMA-IR were ranged between 2.7 and 7.84, which are not comparable because of the validity of HOMA-IR that is limited to the specific assay used for measurement [15]. Criteria of metabolic syndrome were used to define hyperglycemia, hypertriglyceridemia and low high-density lipoprotein-cholesterol (HDL-c) respectively, fasting blood glucose ≥ 6.1 mmol/l (but < 7.0 mmol/l) and serum triglycerides ≥ 1.7 mmol/l and HDL-c < 1.03 mmol/l in men or < 1.30 mmol/l in women, or in presence of lipid lowering drugs. Low-grade inflammation was defined as hs-CRP ≥ 2 mg/l [16]. Each participant underwent a total body scan using dual-energy x-ray absorptiometry (Dual Energy X-ray absorptiometry, Hologic QDR 4500 A) with a radiation densitometer for assessment of total body fat. In the clustering analysis, we defined adiposity as a median total fat mass > 40%.

2.3. Statistical analysis

Categorical data are reported as numbers and proportions and continuous data as median values with interquartile ranges. Comparisons of categorical data were performed using a Chi square or Fisher exact test as appropriate; continuous variables were compared using a Kruskal-Wallis rank sum test.

A logistic regression was used to identify independent risk factors for insulin resistance status in the cohort. A multivariate model was established to predict insulin resistance status. Variables were selected if p -value was < 0.20 in the univariate analysis and a stepwise procedure was used to select the final model. To establish a composite score, we gave a score to each of the variables included in the final prediction model in relation to each one's β parameter (logistic regression coefficient) in that model.

The explorative analysis was performed using principal component analysis. Linear combinations of the variables formed new independent variables or components. Results were visualized through a graph or map, in which the original variables or individuals are displayed, allowing easy visual interpretation of their relationships. Then, a cluster analysis based on the significant components of the principal component analysis was performed in individuals. Cluster analysis was performed using Ward's method. Next, the variables of the clusters of patients with obesity were compared. All the statistical analysis was performed using R 2.15 (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Clinical and metabolic parameters of the cohort

A total of 498 obese non-diabetic patients (101 male and 397 female) were included. Age range was between 18 and 81 years old. The characteristics and metabolic parameters of the patients are presented in Table 1 and Table 2. One hundred thirty-nine (27.9%) patients had grade I obesity ($30 \leq \text{BMI} < 35$ kg/m²), 186 (37.3%) had grade II obesity ($35 \leq \text{BMI} < 40$ kg/m²) and 173 (34.7%) had grade III obesity ($\text{BMI} \geq 40$ kg/m²). This repartition was not different between genders. Less of a half of the patients (198, 39.8%) were IR according to HOMA-IR broken down as follows: 37 (26.6%) with grade I obesity, 70 (37.6%) with grade II obesity and 91 (52.6%) with grade III obesity ($p < 0.001$). Almost all the patients met the criteria of abdominal obesity measured by waist circumference without significant different prevalence between insulin-resistant (IR) and insulin-sensitive (IS) patients (98.5% vs. 94.0%, NS). Grade III obesity was predominant in IR patients (41.6% vs. 30.3%, $p < 0.001$). Fasting hypertriglyceridemia was more prevalent in IR patients (51.7% vs. 29.3%, $p < 0.001$) as well as glycated hemoglobin (60.9% vs. 41.7%, $p < 0.001$) and low-grade inflammation (87.8% vs. 76.3%, $p = 0.016$). Statin treatment and tobacco consumption were not different between IR and IS patients.

Table 1
Baseline characteristics of the study subjects according to insulin-sensitivity status.

	Overall	Insulin-resistant	Insulin-sensitive	p values
	n = 498 (100%)	n = 198 (39.8%)	n = 300 (60.2%)	
Gender, Male (%)	101 (20.3%)	48 (24.2%)	53 (17.7%)	0.094
Age, years	48 (34–58)	47 (31–57)	49 (37–58)	0.099
BMI, kg/m ²	37.8 (34.5–42.2)	39.4 (36.1–44.2)	36.7 (33.5–40.2)	< 0.001
Waist Circumference, cm	112 (103–122)	117 (109–127)	108 (100–119)	< 0.001
Total fat mass, %	44.1 (40.7–47.8)	44.4 (41.6–48.2)	44.0 (40.1–47.3)	0.201
SBP, mm Hg	120 (115–130)	121 (115–130)	120 (115–130)	0.529
DBP, mm Hg	70 (65–80)	70 (65–80)	70 (64–78)	0.224
RAAS inhibitors	96 (19.3%)	40 (20.2%)	56 (18.7%)	0.757
Current smoking	78 (18.7%)	29 (17.4%)	49 (19.6%)	0.656
Statin treatment	55 (11%)	20 (10.1%)	35 (11.7%)	0.69

Values were described using proportions for categorical variables and median (IQR) for quantitative variables.

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; IQR, interquartile range; RAAS, Renin angiotensin aldosterone system; SBP, systolic blood pressure.

3.2. Validation of a simple predictive score for IR status

We performed a composite score in order to find the features of IR in our cohort. The risk score of the logistic regression model to predict occurrence of IR, assessed by HOMA-IR, was: $Z = -0.822 + 0.462 * (\text{presence of grade II obesity}) + 0.968 * (\text{presence of grade III obesity}) + 0.957 * (\text{HbA1c} \geq 5.7\%) + 1.090 * (\text{presence of hypertriglyceridemia}) - 0.023 * (\text{age})$. In this multivariate model, gender was tested but has no influence on the prediction model and was not selected. IR was associated with grade III obesity (OR = 2.6 [1.6–4.4], $p < 0.001$), HbA1c $\geq 5.7\%$ (OR = 2.6 [1.7–4.0], $p < 0.001$), hypertriglyceridemia (OR = 3.0 [2.0–4.5], $p < 0.001$) and age (OR = 0.99 [0.96–0.99], $p = 0.002$).

3.3. Principal component analysis

We used a principal component analysis to visualize the link between obesity-related components. Fig. 1 shows the projection of the cardio-metabolic components on dimension 1 (Dim 1) and dimension 2 (Dim 2). The set of orthogonal variables are uncorrelated and the bigger is the arrow, the more reliable is the parameter. We observed contributions of hypertriglyceridemia, low HDL-c, hypertension and HbA1c $\geq 5.7\%$ on the x-axis (modules near dimension 1). Dimension 2 was defined by elevated waist circumference, grade III obesity, elevated fat

Table 2
Metabolic parameters of the study subjects according to insulin-sensitivity status.

	Overall	Insulin-resistant	Insulin-sensitive	p values
	n = 498 (100%)	n = 198 (39.8%)	n = 300 (60.2%)	
Total cholesterol, mmol/l	5.29 (4.62–5.91)	5.21 (4.62–6.02)	5.31 (4.64–5.88)	0.663
LDL-c, mmol/l	3.25 (2.68–3.84)	3.21 (2.66–3.90)	3.25 (2.71–3.82)	0.689
HDL-c, mmol/l	1.24 (1.06–1.47)	1.16 (1.01–1.34)	1.29 (1.11–1.54)	< 0.001
Triglyceride, mmol/l	1.50 (1.19–1.97)	1.72 (1.30–2.25)	1.38 (1.13–1.83)	< 0.001
Fasting glycemia, mmol/l	5.0 (4.6–5.4)	5.3 (4.9–5.8)	4.9 (4.5–5.2)	< 0.001
Fasting insulin, $\mu\text{UI/ml}$	13.4 (8.5–19.6)	21.2 (18.2–26.5)	9.2 (6.9–12.3)	< 0.001
HOMA-IR	2.9 (1.8–4.6)	5.0 (4.3–6.1)	2.0 (1.4–2.6)	< 0.001
HbA1c, %	5.7 (5.4–6.0)	5.8 (5.5–6.0)	5.6 (5.3–5.9)	< 0.001
hs-CRP, mg/l	4.7 (2.6–9.2)	5.3 (3.2–9.8)	4.4 (2.2–8.1)	0.003

Values were described using proportions for categorical variables and median (IQR) for quantitative variables.

Abbreviations: HbA1c, glycated hemoglobin A1c; HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment - insulin resistance; hs-CRP, high sensitivity C-reactive protein; IQR, interquartile range; LDL-c, low-density lipoprotein cholesterol.

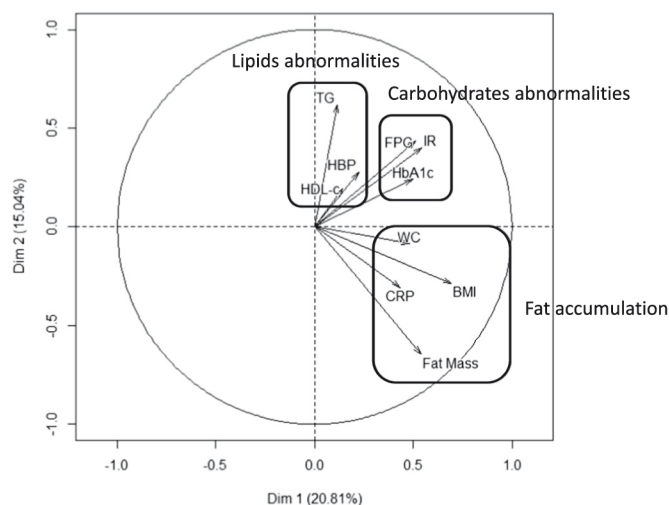


Fig. 1. Factor map of cardio-metabolic abnormalities obtained in all patients (n = 498) by principal component analysis. The original variables are projected in a reduced dimension space defined by component 1 (x-axis) and component 2 (y-axis). The set of orthogonal variables are uncorrelated.

Abbreviations: BMI, Grade 3 obesity; CRP, low-grade inflammation; FPG, fasting hyperglycemia; HDL-c, low high-density lipoprotein cholesterol; HbA1c, glycated hemoglobin A1c $\geq 5.7\%$; HBP, hypertension; IR, Insulin-resistance; TG, hypertriglyceridemia; WC, elevated waist circumference.

mass and low-grade inflammation. In summary, exploratory visual analysis revealed three subgroups of redundant variables: lipid metabolism abnormalities characterized the first subgroup; carbohydrates metabolism abnormalities the second subgroup, and surrogates of fat accumulation characterized the last one. The first two groups were correlated but were independent of the latest. Interestingly, low-grade inflammation was only correlated to fat accumulation.

3.4. Clustering analysis and classification of the subjects

The patients were classified using a hierarchical model of classification, which generated a grouping of the individuals. The optimal grouping was obtained with three clusters (Table 3). The characterization of the three clusters revealed that level of total fat mass, hypertriglyceridemia and elevated HbA1c were the key components for classification. Patients with higher adiposity, evaluated by clinical and imaging assessment, higher prevalence of low-grade inflammation and hypertension characterized clusters 1 and 2. The key difference between clusters 1 and 2 was the greater prevalence of hypertriglyceridemia and HbA1c $\geq 5.7\%$ in cluster 2 that displayed the larger proportion of IR patients. Cluster 3 included patients with lower level of total fat mass and lower prevalence

Table 3
 Repartition of metabolic features among our patients after a hierarchical cluster analysis.

	Cluster 1	Cluster 2	Cluster 3	
	Higher adiposity	Higher adiposity with insulin resistance	Lower adiposity	
	n = 324 (65.1%)	n = 153 (30.7%)	n = 21 (4.2%)	p values
Insulin-resistant, n (%)	62 (19.1%)	133 (86.9%)	3 (14.3%)	< 0.001
Age	46 [32.1–55.4]	53.6 [44.2–60.4]	44.7 [24.3–49.7]	< 0.001
Grade III obesity	94 (29.1%)	79 (51.6%)	0 (0.0%)	< 0.001
Elevated waist circumference	324 (100.0%)	152 (99.3%)	0 (0.0%)	< 0.001
Median total fat mass (%)	44.1 [41.0–47.4]	45.2 [41.7–48.9]	38.2 [35.0–41.5]	< 0.001
Hypertension	156 (48.1%)	99 (64.7%)	7 (33.3%)	0.001
Fasting hypertriglyceridemia	111 (34.3%)	76 (49.7%)	3 (14.3%)	< 0.001
Low HDL-c	175 (54.0%)	88 (57.1%)	11 (52.4%)	0.766
HbA1c \geq 5.7%	106 (32.7%)	135 (88.2%)	4 (19.0%)	< 0.001
Fasting hyperglycemia	4 (1.2%)	94 (61.4%)	2 (9.4%)	< 0.001
Low-grade inflammation	254 (78.3%)	135 (88.2%)	13 (61.9%)	0.010

Abbreviations: HbA1c, glycated hemoglobin A1c; HDL-c, High-density lipoprotein cholesterol. Adiposity was defined as a total fat mass > 40%.

of metabolic abnormalities than the other clusters. Regarding low HDL-c, clusters did not differ.

4. Discussion

The results of this cross-sectional study indicate that, in non-diabetic patients with obesity, hypertriglyceridemia, HbA1c \geq 5.7% and grade III obesity are the best determinants of IR, highlighting the interest of triglycerides and HbA1c measurement in the assessment of obese non-diabetic patients in clinical practice.

Our population was characterized by a young median age and an elevated median BMI but was homogenous regarding BMI distribution, given that each class of obesity was equally represented. Gender distribution did not reveal significant difference among the IR and IS patients but younger age gives greater risk to be of an IR phenotype in the composite score even. However the weighting of age in the score was less strong than metabolism abnormalities surrogates. This can be statistically explained by the high prevalence of obesity among young women (age 18–25) as shown in a large cohort study of the general population in France [17]. We reported a lower prevalence of IR (39.5%) in our French cohort of patients with obesity compared to similar studies in which 88.7% and 83.1% of patients with obesity were IR defined respectively by a HOMA-IR \geq 2.5 and HOMA < 90th percentile [18,19]. However, it should be noted that the prevalence of IR is varying according to definitions. In these studies, diabetic patients were not excluded, suggesting that a consistent part of IR patients with obesity had previously diagnosed diabetes. Thus, a main strength of our study is to have carefully excluded patients with referral diagnosis of diabetes or newly diagnosed diabetes according to the definition of ADA, based on an oral glucose tolerance test and HbA1c.

In our study, we investigated the influence of several cardiometabolic parameters on IR in non-diabetic patients with obesity. Hierarchical analysis further underlined that the association of markers of fat accumulation, HbA1c \geq 5.7% and dyslipidemia conspire to IR. Higher total fat mass is a clear determinant of both Cluster 1 and 2 and is closely related to low-grade inflammation. Further, the principal component analysis demonstrated that low-grade inflammation is

highly correlated to excessive fat accumulation but neither with carbohydrates metabolism abnormalities as assessed by fasting hyperglycemia, HOMA-IR or HbA1c, nor with lipid metabolism abnormalities. Surprisingly, low-grade inflammation was not correlated with IR. In agreement with these data, a recent study found a similar cardiovascular risk in patients with abdominal obesity and metabolic abnormalities regardless of presence or not of a low-grade inflammation [20]. Thus, systemic low-grade inflammation could strongly be linked with lipid storage in adipose tissue rather than with IR. This might be associated with the ability of adipose tissue to be infiltrated by pro-inflammatory immune cells and to secrete pro-inflammatory factors [21]. Finally, hsCRP might not be a discriminating factor in patients who already have metabolic abnormalities.

The main result of our statistical analysis is that HbA1c \geq 5.7% and hypertriglyceridemia are the best determinants (Table 3, Cluster 2) of IR when taken together, independently of markers of fat accumulation (Fig. 1). HbA1c is recognized as a more accurate than fasting glucose predictive test of increased risk for diabetes in general population in association with oral glucose tolerance test and no analytical interference with hypertriglyceridemia are described [12,22]. Although data regarding HbA1c in obese patient without diabetes are scarce, one epidemiological study, including overweight and obese inpatients, showed that simplifying screening by measuring only fasting plasma glucose or HbA1c fails to identify the same subjects as does the oral glucose tolerance test [23]. Concerning our data, we propose to combine HbA1c with triglycerides testing to identify IR obese individuals. Hypertriglyceridemia is a common disturbance in type 2 diabetes that has also been described as an independent coronary heart disease risk factor [24]. Hypertrophied intra-abdominal adipocytes are characterized by a hyperlipolytic state that is resistant to the antilipolytic effect of insulin [3]. Excess fatty acids entering pancreatic β -cells likely are one cause of increased insulin secretion and hyperinsulinemia found in patients with obesity. Over time, ectopic lipid may destroy β -cells through overstimulation of insulin secretion and lipotoxicity that could account for the apparent insulin exhaustion commonly observed in patients with type 2 diabetes [21]. In another side, it has been shown that glucose can induce apolipoprotein CIII transcription, which may represent a mechanism linking hyperglycemia and hypertriglyceridemia [25]. The link between hypertriglyceridemia and insulin resistance in obesity has been studied before but the role of plasma triglycerides level alone is still debated [26–28]. Hypertriglyceridemia and increased large triglyceride-rich lipoprotein have regained interest because of their link with glucose metabolism and a higher risk of cardiovascular disease [29,30]. Several indices where triglycerides or lipids might be used to screen IR were proposed. Blackburn et al. argued hypertriglyceridemia is a screening approach to identify individuals with deteriorated cardio-metabolic risk markers, when associated with waist circumference [31,32]. This phenotype was called “hypertriglyceridemic waist” but was not studied specifically in obese patients. Moreover, waist circumference is not discriminating in obese patients, especially with BMI \geq 35 kg/m² as the majority of patients of our cohort [8,33]. A lipid-parameter-based index including HDL-C and non esterified fatty acids in addition to measure of insulin was developed as an estimate of IR in non-diabetic obese women but as difficult as HOMA-IR and routine practice [34]. Recently, the “TyG index”, a new formula not using insulin testing but a product of fasting glucose and triglyceride levels has emerged as a simple measure reflecting IR and predicting the development of type 2 diabetes with a good correlation with the euglycemic hyperinsulinemic clamp [35–37]. However, among obese post-menopausal women, it showed only a moderate correlation to the gold standard [38]. In line with these studies, we found that HbA1c \geq 5.7%, well known to be more accurate than fasting glucose, associated with fasting hypertriglyceridemia might be an easy way for identifying IR among patients with obesity, especially grade III obesity.

The main limits of our study are the absence of follow-up of patients

to assess the incidence of diabetes and the fact that we did not use the gold standard method (euglycemic hyperinsulinemic clamp) for assessment of insulin resistance because this technique is quite invasive, expensive as well as time consuming and thus poorly suitable for large-scale trials. The results of the present study may be considered preliminary but they may stimulate interest in the need for greater participant characterization in research studies. According to our findings, hsCRP might not be discriminating in obese patients in contrast to HbA1c \geq 5.7% and hypertriglyceridemia that appears to be widely available screening tools for identifying IR obese patients, especially in grade III obesity. The role of triglycerides in the obesity-related risk has recently re-emerged, in agreement with the concept that this lipid dysmetabolism may precede peripheral IR state, while the link with glycated hemoglobin has never been studied in this population [29,39,40]. This study restores the interest of a simple blood test as triglycerides and HbA1c determination, which could be an easy and available method to help physicians identify obesity related insulin resistance for appropriate prevention.

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