Original Article

Triglycerides and TG/HDL-C ratio as surrogate markers for insulin resistance in Nigerian women with polycystic ovary syndrome

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ABSTRACT

Background: Dyslipidemia is one of the most perplexing metabolic consequences in polycystic ovary syndrome (PCOS). Obesity, insulin resistance (IR), and hyperandrogenism, the pervasive features of PCOS, play significant pathophysiological roles in the lipidemic aberrations associated with the syndrome.

Objective: This study aimed to assess the diagnostic utility of triglyceride (TG) and triglyceride to high-density lipoprotein-cholesterol (TG/HDL-C) ratio as surrogate markers for identifying IR in infertile Nigerian women with PCOS.

Materials and Methods: Eighty-seven infertile women with PCOS were selected according to the Androgen Excess Society criteria and categorized into two groups. After anthropometric measurements, fasting blood samples were assayed for plasma glucose, serum insulin, total cholesterol, TG, HDL-C while lipoprotein ratios were calculated. Homeostasis model assessment for IR (HOMA-IR) was used in defining IR. The areas under the receiver operating characteristic (ROC) curve analysis were used to compare the power of the serum markers, and to obtain the optimal cutoffs of TG and TG/HDL-C with HOMA-IR.

Results: TGs correlated significantly with HOMA-IR in the obese PCOS women. However, the areas under the ROC of potential markers showed no significant marker for HOMA-IR. The highest area under the curve of ROC for TG belongs to the obese group with a sensitivity of 56% and specificity of 53% (TG $\geq$ 92.5mg/dL) as a marker of IR in obese PCOS women.

Conclusion: TG and TG/HDL-C would not be reliable markers of IR, and a concerted approach in finding surrogate markers will benefit future investigations.

Key words: Insulin resistance; Nigerian women; polycystic ovary syndrome; surrogate marker; triglyceride.

Introduction

Dyslipidemia, common in polycystic ovary syndrome (PCOS) has multifactorial causation.[1] Women with PCOS are frequently found to have atherogenic lipid abnormalities that may reflect underlying insulin resistance (IR).[2] IR plays a pivotal role by the stimulation of lipolysis and altered expression of lipoprotein lipase and hepatic lipase.[1] At the heart of the pathophysiology of PCOS is IR and hyperinsulinemia, and it may also lead to hyperglycemia and adverse profiles of cardiovascular risk factors. Although the links between IR and associated dyslipidemia, hypertension, and atherosclerosis are complex, dysregulation of fatty acid metabolism seems

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central to the pathophysiology of the IR syndrome, as it is related to cardiovascular disease.[3] Dyslipidemia in PCOS is characterized by higher TGs and lower high-density lipoprotein cholesterol (HDL-C). IR occurs in about 50–80% of women with PCOS.[1] In the insulin-resistant state, non-esterified fatty acids are mobilized from the muscle and adipose tissues to the liver, thereby increasing the substrate for TGs production.[4] The gold standard methods designed to measure insulin sensitivity are impracticable in the clinical setting since they can only be performed in specialized centers due to being complex, expensive, and time-consuming.[5] Likewise, other surrogate markers based on fasting insulin and glucose levels present important limitations related to their poor reproducibility and reliability. Besides, no clear guidelines and no universally accepted cutoffs are available for most of the main surrogate markers used.[6-8] Some studies have reported that TGs and the TGs/HDL-C ratio were closely and positively related to IR, and the two variables were recommended as surrogates for IR.[9-15] In contrast, some other studies showed that TG and TG/HDL-C ratio were not reliable markers of IR in some populations.[16-18] In developing countries, the cost of insulin assay can be a major limitation in the assessment of IR. Therefore, the utilization of surrogate markers like TG which is feasible in small centers, lower in costs, and applies to the general population can be a useful alternative. The present study aimed to assess the diagnostic utility of TG and TG/HDL-C ratio as surrogate markers in identifying IR in Nigerian women with PCOS.

Patients and Methods

This study selected 87 women with PCOS attending Infertility Clinics at the…. between April 1, 2009, and November 30, 2010. The diagnoses of PCOS were according to the Androgen Excess-PCOS Society (AES) criteria which are specifically defined by the presence of hyperandrogenism (clinical and/or biochemical), ovarian dysfunction (oligo-anovulation and/or polycystic ovaries), and the exclusion of related disorders.[19] For the AE-PCOS Society, the definition was analogous to the Rotterdam criteria but excluded women with only menstrual dysfunction and polycystic ovaries. Inclusion criteria were women with clinical and/or biochemical hyperandrogenism, ovulatory dysfunction, and/or polycystic ovaries detected by ultrasound scans. Exclusion criteria were women with hyperprolactinemia, congenital adrenal hyperplasia, thyroid dysfunction, on hormonal therapy or medications that could influence the hormonal assay. Fully informed consent was obtained from all the women. A pre-prepared standard pro forma was used in capturing the data on age, clinical and medical history, detailed anthropometry, and blood pressure measurements. Body mass index (BMI) was calculated as weight (kg)/height (m^2).

After an overnight fast, blood samples were drawn from the antecubital vein. The fasting blood samples were analyzed for baseline metabolic profiles namely, fasting plasma glucose (FG), serum total cholesterol (TC), TGs (TG), HDL-C, total testosterone (TT), luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin (PRL), thyroid-stimulating hormone (TSH), 17 hydroxyprogesterone (17-OHProg), insulin, uric acid, and high sensitivity C-reactive protein (hs-CRP). Low-density lipoprotein-cholesterol (LDL-C) was calculated using Friedewald's formula.[20] Uric acid was measured using the enzymatic method with the Randox kit (RANDOX, USA). All hormones and hs-CRP were measured using the enzyme-linked immunosorbent assay (ELISA) method with DRG kits (Marburg, Germany. Version 8.0). The detailed method has been published elsewhere.[21,22] IR was defined by using HOMA-IR, which was calculated as (fasting glucose mg/dL × fasting insulin μU/mL)/22.5.

Statistical analysis

Statistical analysis was performed using IBM Statistical Package for the Social Sciences (SPSS) Version 21.0, for Windows (SPSS, Inc., Chicago IL, USA). The mean ± standard deviation of the quantitative measurements is presented. Data for TT, LH/FSH ratio, fasting insulin (FI), HOMA-IR, fasting glucose to fasting insulin (FG/FI) ratio, hs-CRP, and uric acid were skewed, and are presented as median (interquartile range), and were log-transformed for analysis. The split data procedure was used to divide the subjects into two groups using their BMI characteristics; obese ≥ 25.0 kg/m^2 and nonobese < 25.0kg/m^2. The differences between the obese and nonobese groups were determined using independent sample student *t*-test and Chi-square test—χ^2.

The areas under the receiver operating characteristic (ROC) curves were used to compare the power of the serum markers. The standard errors of each characteristic were presented with the areas under the ROC curves. The sensitivity and specificity values for TG were also derived using the point of inflection from the areas under the ROC curve. Likelihood ratios (LR) were calculated as the ratios of sensitivity/(1 − specificity) for positive LR and (1 − sensitivity)/specificity for negative LR. Accuracy was calculated as:

$$\text{Accuracy} = \frac{TP + TN}{TP + FP + FN + TN}$$

Where TP is true positive, TN is true negative, FP is false positive, and FN is false negative.

Multiple linear regression analysis was used to evaluate the contribution of each confounding factor for HOMA-IR.

Two-tailed *P* values of < 0.05 were considered to be statistically significant all through this study.

Ethical consideration

The ethics and research committee of the…. approved the study proposal and issued the clearance certificate. Fully informed consent was obtained from all women who participated in the study.

Results

The PCOS women were categorized into two groups: nonobese (BMI < 25.0 kg/m^2) and obese (BMI ≥ 25.0 kg/m^2), with their clinical and biochemical characteristics given in Table 1. The nonobese women were 30 with a mean BMI of 23.22 ± 1.16 kg/m^2 and obese women were 57 with a mean BMI of 28.83 ± 3.23 kg/m^2. The mean ages of nonobese and obese women were 28.2 ± 5.62 years and 33.35 ± 4.86 years, respectively. The mean weight of nonobese women was 60.69 ± 5.25 kg and the mean weight of the obese group was 75.99 ± 10.82 kg. Data on some of the variables were significantly different between the nonobese and the obese groups namely age, weight, BMI, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP), mean arterial blood pressure (MABP), TT, LH/FSH ratio, hs-CRP, and uric acid. After adjustment of age [Table 1], the weight, BMI, SBP, and MABP remained similarly significant in nonobese and obese groups, while the prevalence of IR (HOMA-IR ≥ 2) was significantly higher in the nonobese group than the obese group.

Nonobese and obese women were comparable in terms of the mean values of serum total cholesterol, HDL-C, LDL-C, total cholesterol to HDL-C (TC/HDL-C) ratio, and LDL-C/HDL-C ratios ($P > 0.05$) [Table 2]. However, the serum TG and the TG/HDL-C ratio showed a significant difference between the nonobese and the obese groups. The mean values of the lipid profile variables were comparable in both nonobese and obese groups after adjustment of age ($P > 0.05$).

The areas under the ROC curves analyses [Table 3, Figures 1a and 1b] performed on the data of some variables for both obese and nonobese groups demonstrated that there was no statistically significant marker for IR among the potential markers. In the nonobese group, TG and hs-CRP had areas under the ROC curve of 0.569 and 0.608, respectively, and in obese group areas under the ROC curve of 0.540 and 0.594, respectively. However, they did not exhibit statistical significance.

In the obese group [Figure 2], TG had a significant association with HOMA-IR ($R^2 = 0.112$, $P < 0.00$) but not with the nonobese group ($R^2 = 0.014$, $P > 0.05$). When multiple linear regression analysis was used to investigate the effect of various

Table 1: Characteristics of subjects categorized by BMI

Characteristics	Total *n*=87	Nonobese (<25.0 kg/m^2) *n*=30	Obese (≥25.0 kg/m^2) *n*=57	*P**	Age-adjusted *P*$^+$
Age (years)	31.57±5.66	28.2±5.62	33.35±4.86	<0.001	-
Weight (kg)	70.72±11.79	60.69±5.25	75.99±10.82	<0.001	<0.001
Height (m)	1.62±0.060	1.62±0.065	1.62±0.058	0.673	0.669
BMI (kg/m^2)	26.89±3.80	23.22±1.16	28.83±3.23	<0.001	<0.001
SBP (mmHg)	119.43±14.25	112.00±9.61	123.00±14.8	<0.001	<0.001
DBP (mmHg)	77.47±11.12	73.00±9.52	79.82±11.26	<0.001	0.126
MBP (mmHg)	116.82±14.35	110.00 ±	120.00±14.67	<0.001	0.110
MABP (mmHg)	91.46±11.20	86.00±8.32	94.33±11.51	<0.001	<0.001
TT (ng/mL)	1.61±1.57	2.09±2.22	1.36±0.96	<0.001	0.077
LH/FSH	2.67±2.97	3.53±4.15	2.21±2.01	<0.001	0.964
FG (mg/dL)	80.69±16.21	76.30±9.60	83.00±18.44	0.067	0.257
OGTT (mg/dL)	96.60±29.31	91.73±19.46	99.16±33.22	0.264	0.633
FI (μU/mL)	10.00±10.76	10.64±9.10	2.05±2.69	0.693	0.413
FG/FI	18.12±23.8	13.31±12.85	20.65±27.66	0.173	0.291
hs-CRP (mg/L)	9.76±8.23	6.72±3.72	11.36±9.45	0.018	0.077
Uric acid (mg/dL)	5.47±1.71	4.79±1.04	5.83±1.90	0.006	0.118
HOMA-IR	2.07±2.44	2.12±1.92	2.05±2.69	0.907	0.517
HOMA-IR < 2 (%)	58 (66.7)	19 (63.3)	39 (68.4)	0.632	0.002
HOMA-IR ≥ 2 (%)	29 (33.3)	11 (36.7)	18 (31.6)	0.510	0.032

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; MBP: mean blood pressure; MABP: mean arterial blood pressure; TT: total testosterone; LH/FSH: luteinizing hormone to follicle-stimulating hormone ratio; FG: fasting glucose; OGTT: oral glucose tolerance test; FI: fasting insulin; FG/FI: fasting glucose to fasting insulin ratio; hs-CRP: high sensitivity C-reactive protein; HOMA-IR: homeostasis model assessment of insulin resistance. Data presented are mean±standard deviation. Data for TT, LH/FSH, FI, HOMA-IR, FG/FI, hs-CRP, and uric acid were skewed, and are presented as median (interquartile range) and were log-transformed for analysis. *Unadjusted *P*-value by student's t-test or χ^2 test. $^+$Age-adjusted *P*-value by analysis of covariance

Table 2: Lipid profiles of subjects categorized by BMI

Characteristics	Total $n=87$	Nonobese (<25.0 kg/m^2)	Obese (≥25.0 kg/m^2)	P*	Age-adjusted P+
TC (mg/dL)	174.49±34.66	168.20±33.34	177.81±34.97	0.219	0.815
TG (mg/dL)	79.90±31.77	70.40±20.23	84.89±35.56	0.042	0.287
HDL-C (mg/dL)	50.17±16.49	51.83±16.76	49.29±16.43	0.499	0.513
LDL-C (mg/dL)	108.33±33.19	102.23±32.58	111.54±33.34	0.216	0.697
TC/HDL-C	3.79±1.34	3.51±1.19	3.93±1.41	0.169	0.451
TG/HDL-C	1.76±0.90	1.50±0.62	1.91±1.06	0.053	0.209
LDL-C/HDL-C	2.42±1.21	2.22±1.11	2.54±1.26	0.236	0.541

TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; TC/HDL-C: total cholesterol to high-density lipoprotein-cholesterol ratio; TG/HDL-C: triglyceride to high-density lipoprotein-cholesterol; LDL-C/HDL-C: low-density lipoprotein-cholesterol to high-density lipoprotein-cholesterol ratio.Data presented are mean±standard deviation. *Unadjusted P-value by student's t-test or χ^2 test. +Age-adjusted P-value by analysis of covariance

Table 3: Comparison of areas under the ROC curves (95%) for potential markers of insulin resistance (HOMA-IR ≥ 2) of subjects categorized by BMI

Characteristics	Total		AUC (95% CI)			
			Nonobese (<25.0 kg/m^2)		Obese (≥25.0 kg/m^2)	
	$n=87$	P	$n=30$	P	$n=57$	P
TC (mg/dL)	0.602 (0.470-0.735)	0.121	0.687(0.487-0.886)	0.093	0.561 (0.386-0.735)	0.466
TG (mg/dL)	0.535 (0.397-0.673)	0.598	0.569 (0.338-0.801)	0.533	0.540 (0.364-0.716)	0.631
LDL-C (mg/dL)	0.555 (0.421-0.688)	0.407	0.593 (0.377-0.81)	0.401	0.528 (0.351-0.706)	0.731
HDL-C (mg/dL)	0.516 (0.383-0.648)	0.811	0.648 (0.423-0.874)	0.182	0.434 (0.274-0.595)	0.430
hs-CRP (mg/dL)	0.582 (0.449-0.715)	0.216	0.608 (0.373-0.842)	0.333	0.594 (0.430-0.758)	0.257
TC/HDL-C	0.504 (0.366-0.642)	0.953	0.423 (0.192-0.655)	0.491	0.558 (0.389-0.728)	0.482
TG/HDLC	0.504 (0.384-0.652)	0.787	0.423 (0.192-0.655)	0.461	0.570 (0.409-0.731)	0.400
LDL-C/HDL-C	0.506 (0.367-0.644)	0.932	0.435 (0.204-0.667)	0.561	0.561 (0.390-0.732)	0.460

ROC: receiver operating characteristics; CI: confidence interval; AUC: area under ROC curve; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; hs-CRP: high sensitivity C-reactive protein; TC/HDLC: total cholesterol to high-density lipoprotein-cholesterol ratio; TG/HDLC: triglyceride to high-density lipoprotein-cholesterol ratio; LDLC/HDLC: low-density lipoprotein-cholesterol to high-density lipoprotein-cholesterol ratio; HOMA-IR: homeostasis model assessment of insulin resistance. Data for LDL-C, TG were skewed and log-transformed for analysis

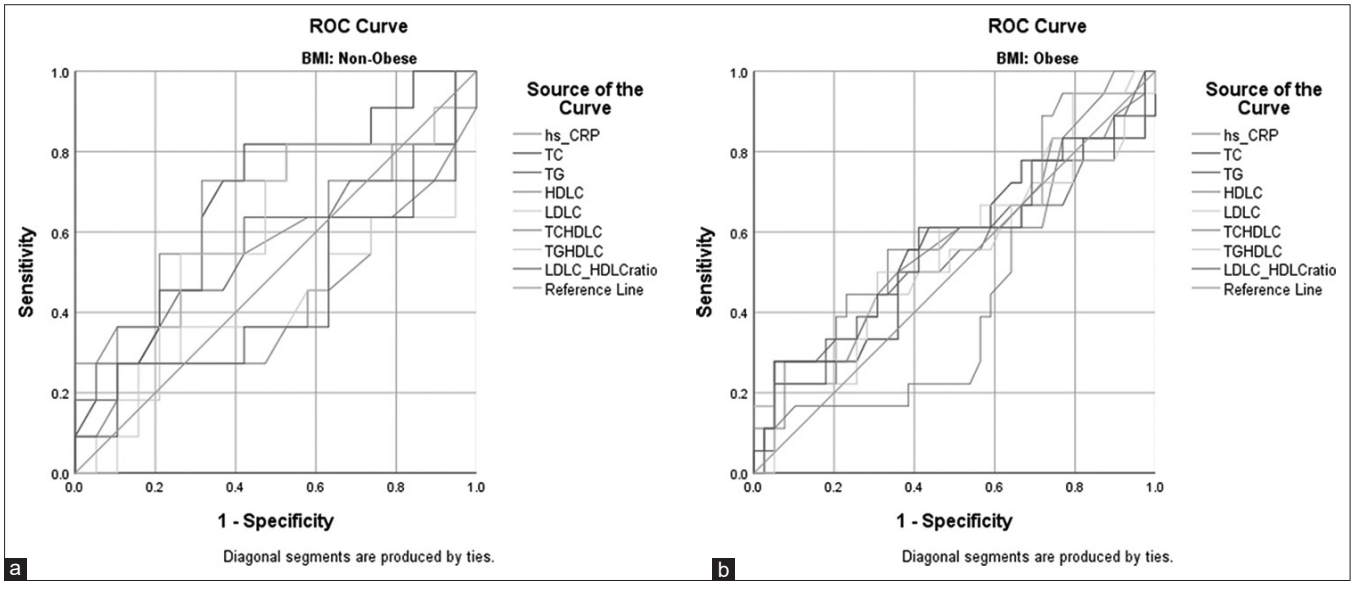

Figure 1: (a) Receiver operating characteristics (ROC) curves. Sensitivity represents the true-positive results, and 1-specificity represents the false-positive results. The best markers have ROC curves that are shifted to the left with areas under the curve near unity. Non-diagnostic markers are represented by diagnosis with areas under the ROC curves close to 0.5. (b) Receiver operating characteristics (ROC) curves. Sensitivity represents the true-positive results, and 1-specificity represents the false-positive results. The best markers have ROC curves that are shifted to the left with areas under the curve near unity. Non-diagnostic markers are represented by diagnosis with areas under the ROC curves close to 0.5. BMI, body mass index; hs_CRP, high sensitivity C-reactive protein; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TCHDLC, total cholesterol to high-density lipoprotein-cholesterol ratio; TGHDLC, triglyceride to high-density lipoprotein-cholesterol ratio; LDLC_HDLC, low-density lipoprotein-cholesterol to high-density lipoprotein-cholesterol ratio

confounding variables on HOMA-IR [Table 4], it revealed that among nonobese PCOS women, BMI, MABP, TC, and hs-CRP had positive impacts of which no variable showed any significance. Similarly, among obese PCOS women, BMI, TC, TG, and hs-CRP had no statistically significant positive impact on HOMA-IR. The joint effect of the independent variables in nonobese (β =0.290, P = 0.538) and obese (β =0.225, P = 0.171) women was not significant in predicting HOMA-IR.

The characteristic cutoff points of TG and TG/HDL-C for verifying IR are shown in Tables 5a and 5b, respectively. In nonobese women, TG of ≥ 73.5 was the cutoff point for predicting IR (HOMA-IR ≥ 2) while in obese women, TG of ≥ 92.5 was the cut-off point of IR. In nonobese and obese women [Table 5a], the LR summarized the information contained in sensitivity and specificity. In nonobese women, the positive LR value reveals that the odds of IR were increased 0.73-fold if TG was 73.5 or more. In obese women, the positive LR value demonstrates that the odds of IR were increased 1.19-fold if the value of TG was 92.5 or more. In the case of nonobese women, the negative LR showed increased odds of IR when the value of TG was lower than the cut-off

Table 4: Multiple linear regression analysis and the correlation between various factors and HOMA-IR of subjects categorized by BMI

Variables	β (*P*)		
	Total, *n*=87	Nonobese (<25.0 kg/m²) *n*=30	Obese (≥25.0 kg/m²) *n*=57
Age (years)	−0.136 (0.294)	−0.183 (0.547)	−0.206 (0.15)
BMI (Kg/m)	−0.10 (0.461)	0.249 (0.267)	0.308 (0.06)
SBP (mmHg)	−0.182 (0.424)	−0.95 (0.799)	−0.161 (0.579)
MABP (mmHg)	0.137 (0.557)	0.244 (0.422)	−0.088 (0.773)
TC (mg/dL)	0.476 (0.157)	0.606 (0.413)	0.147 (0.717)
TG (mg/dL)	0.055 (0.682)	−0.059 (0.795)	0.220 (0.211)
HDLC (mg/dL)	−0.243 (0.512)	−0.050 (0.955)	−0.28 (0.949)
hs-CRP (mg/L)	0.228 (0.061)	0.245 (0.354)	0.044 (0.750)
LDLC/HDLC ratio	−0.381 (0.376)	−0.492 (0.597)	−0.101 (0.844)
R^2	0.123 (0.307)	0.29 (0.538)	0.225 (0.171)

BMI: body mass index; SBP: systolic blood pressure; MABP: mean arterial blood pressure; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C/HDL-C ratio: low-density lipoprotein-cholesterol to high-density lipoprotein-cholesterol; hs-CRP: high sensitivity C-reactive protein; HOMA-IR: homeostasis model assessment of insulin resistance

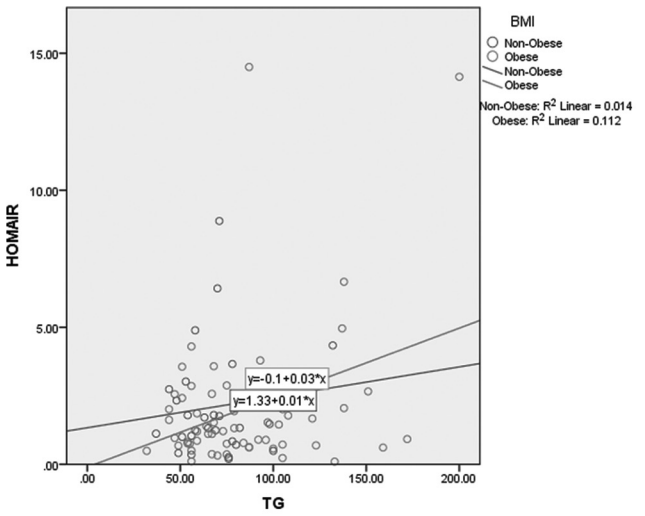

Figure 2: Correlation between TG and HOMA-IR categorized by BMI. Data for HOMAIR was skewed hence log transformed for this analysis. The lines of best fit (BMI < 25.0 kg/m²: r² = 0.014, *P* > 0.05; BMI ≥ 25.0 kg/m²: r² = 0.112, *P* < 0.00) are indicated

Table 5a: Comparison of triglycerides for predicting insulin resistance (HOMA-IR ≥ 2.0) of subjects categorized by BMI

Characteristics cutoff point	HOMA-IR		Sensitivity	Specificity	Positive LR	Negative LR	Accuracy (%)
	<2.0, *n*	≥2.0 *n*					
BMI <25.0 (kg/m²)	19	11					
TG <73.5 (mg/dL)	12	6	0.46	0.37	0.73	1.46	0.57
TG ≥73.5 (mg/dL)	7	5					
BMI ≥25.0 (kg/m²)	39	18					
TG <92.5 (mg/dL)	19	8	0.56	0.53	1.19	0.83	0.51
TG ≥92.5 (mg/dL)	20	10					

HOMA-IR: homeostasis model assessment of insulin resistance; LR: likelihood ratio; BMI: body mass index; TG: triglyceride

Table 5b: Comparison of triglycerides-HDL-C ratio for predicting insulin resistance (HOMA-IR ≥ 2.0) of subjects categorized by BMI

Characteristics cutoff point	HOMA-IR		Sensitivity	Specificity	Positive LR	Negative LR	Accuracy (%)
	<2.0, *n*	≥2.0 *n*					
BMI <25.0 (kg/m²)	19	11					
TG/HDL-C <1.84	15	7	0.36	0.79	1.71	0.81	0.63
TG/HDL-C ≥1.84	4	4					
BMI ≥25.0 (kg/m²)	39	18					
TG/HDL-C <2.0	26	9	0.50	0.67	1.19	0.75	0.61
TG/HDL-C ≥2.0	13	9					

HOMA-IR: homeostasis model assessment of insulin resistance; LR: likelihood ratio; BMI: body mass index; TG/HDL-C: triglyceride/high-density lipoprotein-cholesterol ratio

points. In the case of obese women, the negative LR revealed decreasing odds of IR when the value of TG was lower than the cut-off points.

Discussion

The present study evaluated lipid profiles and assessed the diagnostic utility of TG and TG/HDL-C ratio as surrogate markers in identifying IR in infertile Nigerian PCOS women categorized by BMI. The PCOS women in this study were classified as insulin resistant with HOMA-IR of ≥ 2.0.[23] HOMA model is widely used as a clinical and epidemiological tool since it is more cost-effective than the hyperinsulinemic-euglycemic glucose clamp. Although the model is less accurate, it showed a strong correlation with the glucose clamp method in measuring IR.[24] Thus in this study, HOMA-IR was used as the marker for IR. In nonobese and obese groups, the PCOS women who are insulin resistant were 36.7% and 31.6%, respectively. These percentages are higher compared to non-PCOS women categorized by BMI reported in the study of Apridonidze *et al*.[25] In contrast, the prevalence of IR reported among Korean obese PCOS women by Park *et al.* was high (66.0%) compared with the obese PCOS group in this study. They, however, obtained a lower rate (19.9%) in the nonobese PCOS group when compared with the present study.[15]

The findings in this study showed there was a good correlation between TG and HOMA-IR in the obese group which is consistent with the study of Park *et al.* and Lath *et al*.[15,26] However, the area under the curve (AUC) of TG was comparable with those of other potential markers and none was an acceptable marker. The ROC curve analysis reported by Park *et al*.[15] differed from our findings. Their study revealed that the best cutoff values for TG in identifying IR were ≥ 68.5 in nonobese and ≥ 100.5 in obese PCOS subjects. Furthermore, the present study obtained a positive LR of 1.19, which was the largest for TG in obese PCOS women, whereas they obtained a positive LR of 2.86, which was largest for TG in obese PCOS subjects. Park *et al.* also reported TG and TG/HDL-C as useful markers of IR in Korean PCOS patients and the BMI categorized groups.[15] However, TG is not a useful marker for predicting IR in nonobese and obese PCOS women in this study, although it showed a good correlation with HOMA-IR in obese women.

The findings in the present study are consistent with the report of Kim-Dorner *et al.* in which they indicated that TG and TG/HDL-C were poor predictors of IR, as measured by HOMA-IR, in African Americans, but acceptable markers in whites.[17] Their study revealed areas under the ROC curves of 0.625 and 0.639 for TG and TG/HDL-C respectively in African Americans, whereas in this study, we obtained areas under the ROC curves of 0.535 and 0.504, respectively. However, in whites, TG and TG/HDL-C were acceptable markers for IR with areas of 0.763 and 0.770 respectively.[17] After BMI categorization in the present study, the areas under the ROC curves for TG and TG/HDL-C were 0.569 and 0.423 respectively in nonobese women, and 0.540 and 0.570, respectively, for obese women.

Furthermore, Sumner *et al*. and Knight *et al*. reported that TG and TG/HDL-C are not reliable as markers of IR in African Americans and African descent which is consistent with the report in this study.[16,18] Li *et al*. also reported that the association of TG/HDL-C with IR in non-PCOS patients was stronger among those with BMI <25 kg/m^2 than ≥30 kg/m^2. Although TG/HDL-C could discriminate against IR in nonobese non-PCOS women, it could not in obese non-PCOS women.[27] However, McLaughlin *et al.* showed TG/HDL-C could discriminate against IR in the subjects with a BMI ≥25 kg/m^2.[9,10] Despres *et al.* report also showed that in African Americans and white women, lipoprotein lipase (LPL) activity which is responsible for clearing TG-containing lipoproteins from the circulation, was higher and this might induce lower TG levels, further causing a weak association between TG levels and IR in those population.[28] It should be noted that various study designs, races, age range, and adiposity state of the subjects could result in different findings, hence, these unavoidable different results.[7,29]

This is the first study reporting on a surrogate marker for IR among infertile women with PCOS in our region. Further research work will be necessary for finding a suitable surrogate marker that would benefit developing countries since the cost and availability of insulin assay is a major limiting factor for the assessment of IR. There is still the need to recruit a larger population of PCOS women for these studies. Additionally, funding for research for possible clinical and biological surrogate markers of IR is advocated.

Conclusion

In our population, TG and TG/HDL-C did not discriminate against IR in nonobese and obese PCOS women, and as such would not be reliable markers of IR. A concerted approach in finding surrogate markers of IR would have clinical and investigational implications.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published

and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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