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Roles of C-Peptide and Triglyceride as Effective Indices for Insulin Resistance Investigations in Iraqi Women with Polycystic Ovarian Syndrome

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Abstract

Objectives: To assess the insulin resistance by determination of C-peptide and triglyceride levels in women with polycystic ovarian syndrome (PCOS) and then investigation of insulin resistance by various methods and comparing them with the homeostatic model assessment method used.

Methods: A study included 120 participants (68 women have PCOS subdivide according to their BMI to 45 obese (BMI>=30) and 23 non-obese (BMI<30). The remaining 52 participants represent as apparently control group with normal weight and normal menstrual cycle. Patients with PCOS were selected from the Infertility Department, Gynecology and Obstetrics Teaching Hospital,Kerbala province, Iraq. Diagnosis of PCOS is based on 2 of 3 findings: oligo/anovulation, hyperandrogenism, polycystic ovaries in ultrasound (Rotterdam criteria). The patients were interviewed and examined for weight, height, waist circumference, and hip circumference. Venous blood samples were collected at 9 AM after an overnight fast. Measurement of serum insulin, glucose, triglyceride and C-peptide were performed using Cobas instrument and by ELISA technique.

Results: Based on HOMA-IR, the prevalence of insulin resistance in obese PCOS women was 77% while in non-obese PCOS women was 70%. HOMA-IR, QUICKI, McAuley and CPI showed significant difference between obese PCOS (4.39 ± 1.83), (0.309 ± 0.024), (3.85 ± 0.91) and (4.39 ± 1.63) respectively; and control group (2.68 ± 0.61), (0.331 ± 0.011), (4.53 ± 0.57) and (8.72 ± 1.33) respectively. CPI also showed significant difference between obese PCOS (4.39 ± 1.63) and non-obese PCOS (6.85 ± 1.74). In obese PCOS women, both QUICKI ($r = -1.00$, $P < 0.001$) and McAuley ($r = -0.81$, $P < 0.001$) were strongly correlated with HOMA-IR, whereas CPI was not. For non-obese PCOS, there was a strong correlation for both QUICKI ($r = -0.97$, $P < 0.001$), ($r = -0.62$, $P < 0.05$) and HOMA-IR, CPI was also strongly correlated with HOMA-IR ($r = -0.78$, $P < 0.001$).

Conclusion: Significant number of women with PCOS can be classified as being either insulin sensitive or insulin resistant (IR) depending on the method applied, as correlation between various IR indices is highly variable. Clinical application of surrogate indices for assessment of IR in PCOS must be therefore viewed with an extreme caution.

Keywords: C-Peptide, polycystic ovary syndrome, insulin resistance

Introduction

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder with an impact on hormonal and metabolic regulation. Women with PCOS are at increased risk of anovulation and infertility.[1] The clinical presentation is extremely variable but generally includes clinical and/or biochemical hyperandrogenism, menstrual dysfunction (oligo-amenorrhea) and polycystic ovaries on ultrasound.[2] Diagnostic criteria for PCOS mostly use the revised Rotterdam 2003 criteria.[3]

Insulin resistance (IR) is a very common finding in subjects with PCOS which not included among the diagnostic features.[4] IR is usually defined as a pathological condition characterized by a decreased responsiveness or sensitivity to the metabolic actions of insulin. In women with PCOS, IR plays an important role in the development and persistence of this disorder.[5]

IR stimulates ovarian theca cells to secrete androgens and increasing luteinizing hormone (LH) effect on ovarian androgen production. Insulin inhibits sex hormone binding globulin (SHBG) secretion, increasing free and bioactive androgen levels and worsen hyperandrogenism status.[6] Moreover, IR is critically involved in the development of metabolic syndrome and cardiovascular disease in PCOS women.[7] The need for accurate screening of IR in women with PCOS is obvious. Thus, early recognition and management would offer important preventive measures.[8]

Several methods have been developed to quantify this metabolic phenomenon. The hyperinsulinemic euglycemic clamp technique (HIEG) is generally accepted as the best available direct method to assess insulin sensitivity.[9] However, this technique is very complex and not appropriate in clinical practice. As an alternative strategy, practical surrogate markers have been proposed to measure IR; considering the concept that patients who have insulin resistance will have more insulin hormone in blood than those who does not. Homeostasis model assessment-insulin resistance index (HOMA-IR) and the quantitative insulin sensitivity check index (QUICKI) are the most widely used surrogate indices of IR, which reflect the feedback between fasting serum insulin and glucose.[10,11]

Excess adiposity and dyslipidemia may influence insulin sensitivity. Based on these factors, different indices have been developed to measure IR, which better reflect lipid profiles such as triglyceride (TG) (McAuley index).[12] The

triglyceride-glucose index is the logarithmized product of fasting triglycerides and fasting glucose and has been proposed as the alternative indicator of IR due to its relevance to dyslipidemia.[13]

The glucose insulin ratio (G/I) has also been employed as an index of IR. It has been described, as a useful measure of insulin sensitivity in obese PCOS women and has both high sensitivity and specificity for detecting IR in women.[14]

In addition, previous studies suggested that measuring C-peptide can help to determine how much of insulin a person is producing as C-peptide is secreted in equimolar amounts to insulin.[15,16] C-peptide does not undergo hepatic first-pass metabolism and has a longer half-life than insulin which affords a more stable test window of fluctuating beta cell response. Therefore, it has been suggested that peripheral C-peptide levels more precisely reflect β-cell secretory activity than peripheral insulin.[17] An increase in its levels suggests a high level of endogenous insulin which indicates worsened insulin resistant state.

The aim of the study was to assess how much IR is in both obese and non-obese PCOS women using most commonly used index of IR (HOMA-IR) and find out a correlation between HOMA-IR and the other surrogate indices: G/I, QUICKI, McAuley, triglyceride-glucose index (TyG) and C-peptide index (CPI).

Materials and Methods

A case-control study included 120 participants of which 68 women have PCOS subdivide according to their BMI to 45 obese (BMI >= 30) and 23 non-obese (BMI < 30). The remaining 52 represent the control group who were apparently healthy women with normal weight and normal menstrual cycle. Patients with PCOS were selected from the Infertility Department, Gynecology and Obstetrics Teaching Hospital, Kerbala Health Directorate/Kerbala – Iraq. Institutional ethics committee approval was sought before starting the study. Oral informed consent was obtained from subjects. PCOS was diagnosed in presence of at least two out of the three diagnostic criteria established by the revised 2003 Rotterdam European Society for Human Reproduction/American Society of Reproductive Medicine PCOS Consensus Workshop Group: i) oligo- and/or anovulation, ii) clinical and/or biochemical signs of hyperandrogenism, and iii) polycystic ovaries in ultrasound.[18]

All women underwent anthropometric assessment like measurement of weight, height, waist circumference (WC), hip circumference, waist-hip circumference ratio (WHR) and body mass index (BMI). Transvaginal ultrasound was used to identify polycystic ovaries.

Five milliliters (5 ml) of venous blood samples were collected at 9 AM after an overnight fast on the second or third day of the menstrual cycle, centrifuged and frozen immediately at –20ºC. The levels of glucose, triglycerides, cholesterol and High-Density Lipoprotein (HDL) were measured using chemistry analyzer (AU480, Beckman Coulter, USA).

Serum luteinizing hormone (LH) and follicle-stimulating hormone (FSH) were carried out with automated immunoassay system based on the enzyme linked fluorescent assay principles (ELFA) (bioMérieux, France). Free testosterone was measured by enzyme-linked immunosorbent assay (ELISA) technique.

Insulin Resistant Assessment

Fasting insulin was measured using ELISA technique. Fasting C-peptide was determined using cobas C 111 analyzer. IR was estimated as:

1. HOMA-IR = (I × G)/405
2. Fasting glucose to insulin ratio = G/I
3. QUICKI = 1/(log(I) + log(G))
4. McA = exp (2.63–0.28 × ln(I) – 0.31 × ln (TG/18)
5. TyG = ln [TG × G/2]
6. CPI = 20/(CP × G/18)

Fasting insulin (I) in (μIU/ml), fasting glucose (G) in (mg/dl), triglycerides (TG) in (mg/dl) and fasting C-peptide (CP) in (nmol/l).

The data were analyzed using the Statistical Package for Social Sciences (SPSS version 22.0). Continuous variables were expressed as means ± standard deviation (SD). Mean comparisons were made using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test. Pearson correlation analysis was used to assess the association of various IR indices with HOMA-IR. Receiver operator characteristic curves (ROC) were drawn to compare different insulin resistance/sensitivity indices. Insulin-based HOMA-IR was considered as a gold standard to define insulin resistance. HOMA-IR values less than 2.7 were considered insulin sensitive (IS) and more than that were considered as IR.

Results

The demographic and clinical features of the total study population, as well as baseline values of the various indices of insulin resistance are presented in Table 1. The non-obese PCOS patients were less in age than obese. The parameters like WC and BMI, were found to be significantly increased in obese PCOS women than other groups while WHR was increased in obese PCOS than control group. WHR was not varied between obese and non-obese PCOS women.

Both obese and non-obese PCOS had increased levels of insulin, glucose, cholesterol, triglyceride and LDL compared to the controls. Whereas HDL levels had not reached statistical significance ($P > 0.05$) among three groups.

Neither analysis of obese and non-obese PCOS women, nor controls and non-obese PCOS had significant difference regarding C-peptide values. While C-peptide was higher in obese PCOS. Compared with controls, PCOS women had elevated levels of LH, FSH, LH/FSH ratio and free testosterone. For non-obese PCOS group, there was significant difference in term of LH and free testosterone compared with controls while FSH and LH/FSH ratio were not.

The assessment of IR revealed that HOMA-IR, QUICKI, McAuley, TyG and CPI had significant difference in both PCOS groups compared with controls. G/I ratio was significantly higher in the controls than obese PCOS women. The prevalence of IR based on HOMA-IR was 80% in obese PCOS and 48% in non-obese PCOS women as shown in Figure 1.

Pearson correlation coefficient was performed to show the correlation of the different parameters. For both obese and non-obese PCOS patients, the results revealed a positive correlation of the BMI with insulin, C-peptide, HOMA-IR and

Table 1. **Difference in demographic, metabolic, hormonal features and IR status among obese PCOS, non-obese PCOS and control women**

	Obese PCOS ($N = 45$)	Non obese PCOS ($N = 23$)	Control ($N = 52$)	*P*-value
Demographic characteristics				
Age (years)	29 ± 6.06	25 ± 4.99	27 ± 5.71	
Waist circumference (cm)	108 ± 12.82	84 ± 10.06	80 ± 10.94	<0.001
WHR	0.92 ± 0.10	0.87 ± 0.10	0.82 ± 0.08	<0.001
Body mass index (kg/m^2)	35.83 ± 4.64	24.24 ± 2.56	24.09 ± 3.56	<0.001
Clinical characteristics				
Cholesterol (mg/dl)	163 ± 26.80	144 ± 35.25	117 ± 20.97	<0.001
Triglyceride (mg/dl)	103 ± 1.50	82 ± 1.57	70 ± 1.55	<0.001
HDL (mg/dl)	40 ± 6.76	39 ± 5.17	43 ± 8.95	NS
LDL (mg/dl)	97.43 ± 1.30	83 ± 1.33	56 ± 1.39	<0.001
Fasting glucose (mg/dl)	92 ± 8.25	92 ± 8.88	84 ± 5.12	<0.001
Fasting insulin (μU/ml)	18.15 ± 1.76	15.32 ± 1.70	10.74 ± 1.56	<0.001
Fasting C-peptide (nmol/l)	0.55 ± 0.38	0.50 ± 0.26	0.37 ± 0.18	<0.05
LH (IU/l)	10.23 ± 1.68	10.05 ± 1.48	6.67 ± 1.44	<0.001
FSH (IU/l)	6.55 ± 1.38	6.45 ± 1.25	5.56 ± 1.32	<0.05
LH/FSH ratio	1.70 ± 0.76	1.73 ± 0.92	1.30 ± 0.60	<0.05
Free testosterone (pg/ml)	17.57 ± 2.31	14.97 ± 2.19	4.21 ± 2.50	<0.001
Insulin resistance indices				
HOMA-IR	4.10 ± 1.78	3.47 ± 1.69	2.22 ± 1.58	<0.001
G/I	5.79 ± 2.90	6.81 ± 3.20	8.49 ± 3.31	<0.001
QUICKI	0.312 ± 0.02	0.319 ± 0.02	0.340 ± 0.022	<0.001
McAuley	3.69 ± 0.85	4.16 ± 1.00	4.76 ± 0.94	<0.001
TyG	4.58 ± 0.20	4.46 ± 0.21	4.34 ± 0.22	<0.001
CPI	8.53 ± 1.79	9.19 ± 1.90	13.01 ± 1.64	<0.05

Data was expressed as Mean ± SD. $P < 0.05$ is considered significant.

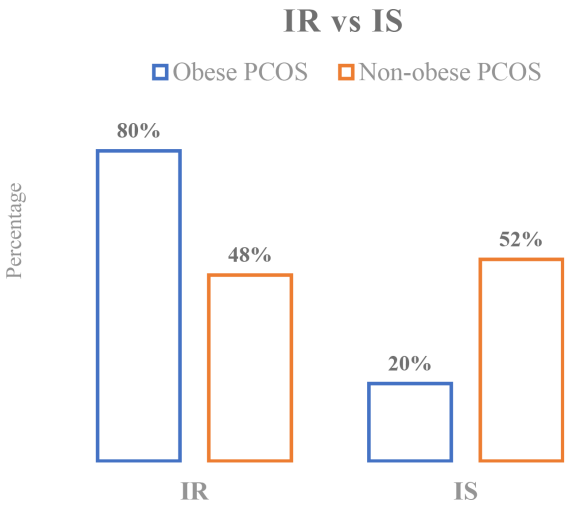

Fig. 1 **The prevalence of insulin resistance (IR)/sensitivity (IS) based on HOMA-IR in both obese and non-obese PCOS women.**

TyG index and negative correlation with G/I, QUICKI, McAuley and CPI. HOMA-IR showed significant positive correlation with insulin, C-peptide and TyG. While it shows significant negative correlation with G/I, QUICKI, McAuley and CPI. More details were demonstrated in Tables 2 and 3.

McNemar test was performed on both obese/non-obese PCOS women to check the concordance/discordance between insulin resistance indices and HOMA-IR, as shown in Tables 4 and 5.

Figure 2 shows the ROC curve for G/I ratio, QUICKI, McAuley, TyG and CPI indices as predictors for HOMA-IR. G/I ratio, QUICKI and McAuley strongly predicted HOMA-IR in both PCOS groups. TyG can be Predicted HOMA-IR in obese PCOS with AUC value of 0.79 ($P < 0.05$). CPI failed to predict HOMA-IR in both obese/non-obese PCOS, as shown in Table 6.

Discussion

The issue of IR in PCOS, though seemingly obvious, is indeed highly problematic, when supposed to be transformed from a theoretical concept into a clinical application. Some studied suggested that IR was apparent not in terms of exceeding a predefined cut-off point, but as lack in insulin sensitivity in comparison to BMI-matched non-PCOS.[19] It must be noted that there is no universal agreement as to the best cut-off point for various insulin-resistance indices. Thus, any cut-off points should be related to particular studied population, as significant ethnic differences have been reported (Wijeyaratne et al., 2002).[20]

Table 2. **Pearson correlation analysis (obese PCOS)**

		WC	WHR	BMI	Insulin	TG	G/I ratio	HOMA-IR	QUICKI	McAuley	TyG	C-peptide	CPI
WC	r	1	0.78	0.68	0.32	.266	0.51	0.32	0.48	0.50	0.35	.128	–.234
	P		.000	.000	.022	.057	.000	.023	.000	.000	.012	.366	.095
WHR	r		1	0.39	0.33	.164	0.46	0.32	0.41	0.41	.214	.058	–.075
	P			.004	.016	.245	.001	.023	.003	.002	.127	.683	.595
BMI	r			1	0.56	0.44	0.55	0.57	0.60	0.61	0.48	0.41	0.4
	P				.000	.001	.000	.000	.000	.000	.000	.002	.004
Insulin	r				1	0.48	0.83	0.99	0.88	0.79	0.49	0.7	0.54
	P					.000	.000	.000	.000	.000	.000	.000	.000
TG	r					1	0.49	0.48	0.50	0.82	0.96	0.35	0.33
	P						.000	.000	.000	.000	.000	.010	.018
G/I ratio	r						1	0.81	0.94	0.86	0.49	0.48	0.49
	P							.000	.000	.000	.000	.000	.000
HOMA-IR	r							1	0.89	0.79	0.51	0.71	0.56
	P								.000	.000	.000	.000	.000
QUICKI	r								1	0.87	0.57	0.56	0.61
	P									.000	.000	.000	.000
McAuley	r									1	0.86	0.52	0.55
	P										.000	.000	.000
TyG	r										1	0.38	0.43
	P											.006	.002
C-peptide	r											1	0.72
	P												.000
CPI	r												1
	P												

Yellow color refers to positive correlation, blue color refers to negative correlation.

Table 3. **Pearson correlation analysis (non-obese PCOS)**

		WC	WHR	BMI	Insulin	TG	G/I ratio	HOMA-IR	QUICKI	McAuley	TyG	C-peptide	CPI
WC	r	1	0.57	0.27	–0.07	0.08	–0.04	–0.10	0.08	–0.05	0.06	–0.08	0.07
	P		.004	.211	.768	.712	.863	.661	.705	.828	.796	.721	.743
WHR	r		1	–0.09	–0.15	-.021	–0.12	–0.17	0.007	–0.08	.028	.114	–.088
	P			.686	.504	.924	.571	.451	.973	.709	.900	.605	.690
BMI	r			1	0.69	0.70	0.70	0.66	0.62	0.78	0.63	0.43	–0.21
	P				.000	.000	.000	.001	.002	.000	.001	.042	.328
Insulin	r				1	0.52	0.81	0.99	0.90	0.78	0.49	0.45	–0.37
	P					.011	.000	.000	.000	.000	.017	.033	.084
TG	r					1	0.53	0.49	0.47	0.86	0.96	0.266	–0.2
	P						.009	.016	.022	.000	.000	.220	.349
G/I ratio	r						1	0.79	0.90	0.85	0.48	0.44	0.41
	P							.000	.000	.000	.020	.037	.050
HOMA-IR	r							1	0.93	0.77	0.49	0.47	0.41
	P								.000	.000	.017	.024	.055
QUICKI	r								1	0.81	0.49	0.52	0.53
	P									.000	.018	.011	.009

(*Continued*)

Table 3. **Pearson correlation analysis (non-obese PCOS)—Continued**

		WC	WHR	BMI	Insulin	TG	G/I ratio	HOMA-IR	QUICKI	McAuley	TyG	C-peptide	CPI
McAuley	r									1	0.86	0.42	0.347
	P										.000	.044	.105
TyG	r										1	0.285	–0.23
	P											.188	.300
C-peptide	r											1	0.79
	P												.000
CPI	r												1
	P												

Yellow refers to positive correlation, blue color refers to negative correlation.

Table 4. **Comparison of insulin resistance indices and HOMA-IR for assessment of IR in obese women with PCOS (cut-off for HOMA-IR > 2.7)**

IR indices		HOMA-IR				Total
		Yes N (%)		No N (%)		
G/I ratio	Yes	33	(100%)[a]	0	(0%)[c]	33 (73%)
	No	3	(25%)[b]	9	(75%)[d]	12 (27%)
QUICKI	Yes	36	(100%)[a]	0	(0%)[a]	36 (80%)
	No	0	(0%)[b]	9	(100%)[d]	9 (20%)
McAuley	Yes	35	(97%)[a]	1	(3%)[c]	36 (80%)
	No	1	(11%)[b]	8	(89%)[d]	9 (20%)
TyG	Yes	27	(93%)[a]	2	(7%)[c]	29 (64%)
	No	9	(56%)[b]	7	(44%)[d]	16 (36%)
CPI	Yes	18	(86%)[a]	3	(14%)[c]	21 (47%)
	No	18	(75%)[b]	6	(25%)[d]	24 (53%)

[a]True positive, [b]False negative, [c]False positive, [d]True negative.

Table 5. **Comparison of insulin resistance indices and HOMA-IR for assessment of IR in non-obese women with PCOS (cut-off for HOMA-IR > 2.7)**

IR indices		HOMA-IR				Total
		Yes n (%)		No n (%)		
G/I ratio	Yes	11	(79%)[a]	3	(21%)[c]	14 (61%)
	No	0	(0%)[b]	9	(100%)[d]	9 (39%)
QUICKI	Yes	11	(92%)[a]	1	(8%)[c]	12 (52%)
	No	0	(0%)[b]	11	(100%)[d]	11 (48%)
McAuley	Yes	10	(71%)[a]	4	(29%)[c]	14 (61%)
	No	1	(11%)[b]	8	(89%)[d]	9 (39%)
TyG	Yes	7	(70%)[a]	3	(30%)[c]	10 (44%)
	No	4	(31%)[b]	9	(69%)[d]	13 (56%)
CPI	Yes	8	(62%)[a]	5	(38%)[c]	13 (56%)
	No	3	(30%)[b]	7	(70%)[d]	10 (44%)

[a]True positive, [b]False negative, [c]False positive, [d]True negative.

In current study IR reported to be more prevalent in obese PCOS group than non-obese PCOS, the same finding was recorded previously, who reported that IR in PCOS had linked to obesity.[21] Although non-obese women exhibit lower IR is still a common finding in this population. Indeed, several studies have suggested IR as a pathophysiological component independent of weight.[22] Obese PCOS have a high probability of IR.[23] It was also observed a significant, but relatively weak correlation between all analyzed IR indices and adiposity indices: WC, WHR and BMI in obese PCOS group. Recent studies reported that WHR was positively correlated with the HOMA-IR.[24] A study of Šumarac-Dumanović et al., confirmed that PCOS women are more susceptible to increasing WHR regarding the development of insulin resistance.[25]

In the current study, women with PCOS had higher fasting insulin levels than controls. Similar results were found by another investigators.[26] It is also indicated that C-peptide was higher only in obese PCOS. Another study reported that C-peptide concentrations were not reached statistically significant among PCOS overweight group, PCOS obese group and healthy women. Also, it did not correlate significantly with FSH and LH serum levels within studied groups.[27] Conversely, another investigators suggested that C-peptide can be used as a surrogate marker of IR in PCOS.[28] A study by Banu et al. also indicated that assessment of C-peptide levels, along with HDL-C levels, in patients can be used to monitor IR.[29]

There is a very good correlation between indices of IR based on fasting glucose and insulin HOMA with G/I and QUICKI, also HOMA has a good correlation with McAuley that utilizes fasting triglyceride concentrations and insulin. Hence, there is an implication that fasting triglyceride concentrations can be safely used to assess IR, instead of fasting glucose. In a previous study by Lewandowski et al., McAuley

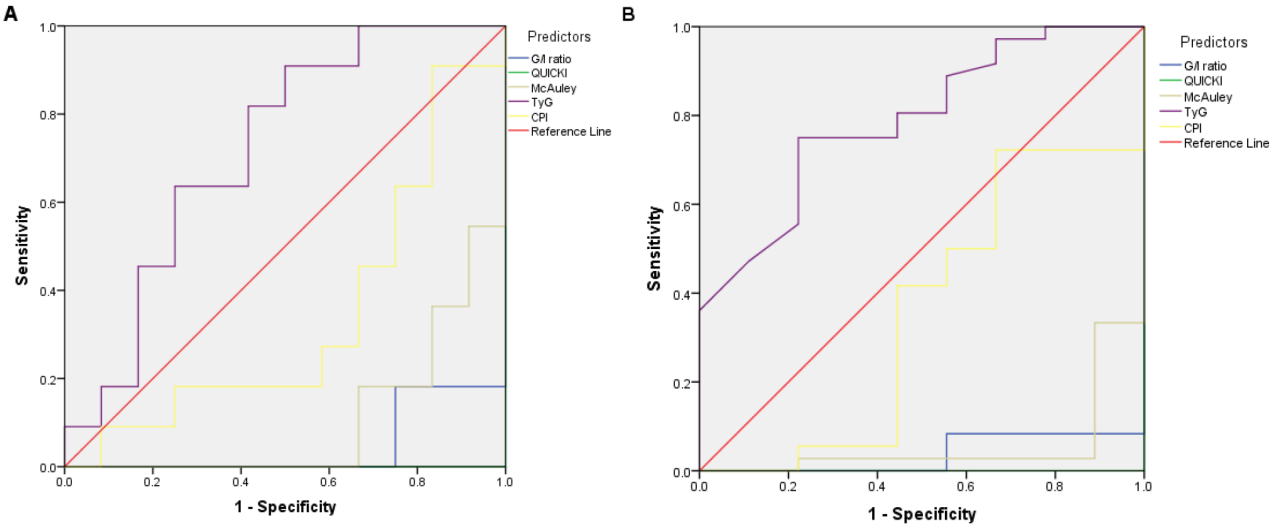

Fig. 2 **The results of ROC curve analysis regarding the predictability of G/I ratio, QUICKI, McAuley, TyG and CPI indices in classifying the IR considering HOMA-IR in (a) non-obese PCOS and (b) obese PCOS.**

Table 6. **The areas under ROC curve (AUC), sensitivity, specificity by the optimized cut-off points for IR indices in predicting the HOMA-IR**

	Predictors	AUC (95% CI)	*P*-value	Cut-off value	Sensitivity	Specificity
Obese PCOS	G/I ratio	0.04 (0.00–0.09)	0.000	7.37	92%	100%
	QUICKI	0.00 (0.00–0.00)	0.000	0.33	100%	100%
	McAuley	0.06 (0.00–0.14)	0.000	4.3	97%	89%
	TyG	0.79 (0.64–0.94)	0.008	4.51	75%	78%
	CPI	0.36 (0.14–0.57)	0.182	8.52	50%	66%
Non-obese PCOS	G/I ratio	0.05 (0.00–0.12)	0.000	7.37	100%	75%
	QUICKI	0.00 (0.00–0.00)	0.000	0.33	100%	100%
	McAuley	0.11 (0.00–0.23)	0.001	4.3	91%	66%
	TyG	0.72 (0.51–0.93)	0.074	4.51	64%	75%
	CPI	0.34 (0.11–0.57)	0.196	8.52	73%	58%

index was found to have a good correlation with HOMA-IR (r = −0.849) in large (n = 478) group of women with PCOS aged 25 ± 8.05, BMI 27.27 ± 7.18 kg/m^2 (Lewandowski et al., 2018).[30] Kheirollahi et al. suggested that TyG index strongly correlated with IR as estimated by HOMA-IR, among Iranian women diagnosed with PCOS.[31] Also, a recent study included 11,378 adults proposed TyG index as a useful surrogate measure of IR (AUC was 0.723) as reported previously.[32]

C-peptide is accepted as a better descriptor of pancreatic activity than peripheral insulin itself. In the current study, a weak correlation was found between HOMA-IR and C-peptide. Previously, Tura et al. found that an index based on insulin and glucose (IGI) strongly correlated with corresponding index for C-peptide, indicating that hepatic insulin extraction is not a confounding factor in the relationship between insulin and C-peptide-based indices.[33] Additionally, MJ found that values of fasting glucose, insulin, C-peptide and the HOMA index significantly increased with age and pubertal stage, while the QUICKI index decreased.[34]

A study by Ohkura et al. revealed that the index CPI was more strongly correlated with glucose infusion rate (GIR) derived from the glucose clamp technique, than were HOMA-IR and QUICKI, as CPI requires a single blood sample and plasma C-peptide levels better reflect insulin bioactivity in skeletal muscle, it was recommend for screening of IR.[35]

Conclusion

It can be concluded that TyG is a valuable indicator to predict IR in obese women with PCOS, partly due to its analytical and financial ease-of-access in all clinical laboratories. The use of TyG index is recommended in the assessment of IR risk among Iraqi women with PCOS. Further epidemiological research is advised.

Conflicts of Interest

None.

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