# A Cross-sectional study to determine triglyceride-glucose index as riskmarkers of insulin resistance in polycystic ovary syndrome women in a tertiary care hospital of south India

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## Abstract:

Insulin resistance plays a central role in the development of polycystic ovary syndrome (PCOS), with the association between lipid ratios and insulin resistance varying depending on ethnicity. Some indices may not accurately predict insulin resistance in certain populations. This study aimed to assess the correlation between triglyceride to HDL-cholesterol (TG/HDL-C), total cholesterol to HDL-cholesterol (TC/HDL-C), and fasting triglyceride-glucose (TyG) indices with insulin resistance (measured by homeostasis model assessment of insulin resistance (HOMA-IR), quantitative insulin sensitivity check index (QUICKI), and fasting glucose to insulin ratio (FGIR)) among 100 PCOS subjects. Fasting insulin levels were measured using ELISA, and conventional lipid profiles were assessed using Virto's dry chemistry analyzer. Lipid ratios (TG/HDL-C, TC/HDL-C, and TyG indices) differed significantly between insulin-resistant (IR) and insulin-sensitive (IS) groups, as confirmed by HOMA-IR, FG-IR, and QUICKI values. Analysis of area under the curve (AUC) revealed that TyG, TG/HDL-C, and TC/HDL-C strongly predicted HOMA-IR with AUCs of 0.639, 0.619, and 0.623, respectively ($P < 0.05$). Additionally, TC/HDL-C was a good predictor of FG-IR with an AUC of 0.60 ($P = 0.039$). In conclusion, TyG, TG/HDL-C, and TC/HDL-C indices may serve as indicators of insulin resistance in women with PCOS.

**Keywords:** Polycystic ovary syndrome, Insulin Resistance, Homeostasis Model Assessment of insulin resistance, Quantitative insulin sensitivity check index

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# INTRODUCTION:

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders, affecting 5%-10% of women of reproductive age and is the main cause of infertility due to anovulation [1]. Epidemiological data showed evidence that PCOS is associated with an increased risk of impaired glucose tolerance, type 2 diabetes and gestational diabetes mellitus.[2]Insulin resistance (IR) outlined as a pathological condition characterized by a decreased sensitivity or responsiveness of insulin to its metabolic actions. It is a prominent feature of PCOSwith a prevalence of 35%-80%. In women with PCOS, IR plays an important role in thedevelopment and persistence of this disorder and is recognized predictor of a range of disorders. [3-4]

In steroidogenic tissues like ovary and the adrenal cortex, insulin promotes steroidogenesis by potentiating the cognate trophic hormones. The compensatory hyperinsulinemia associated with IR results in net increasing of circulating testosterone hormones. [5] An increase in insulin and androgen levels interrupts the follicular growth, which in turn leads to typical clinical features including hirsuitism, irregular menses, chronic anovulation and infertility. The persistent hyperandrogenism is associated with impaired hypothalamic–pituitary feedback, LH hypersecretion, premature granulose cell luteinization, aberrant oocyte maturation and premature arrest of activated primary follicles.[6] Thus diagnosing insulin resistance in PCOS is of critical importance for better management and prevention of complications.

Homeostasis Model Assessment of insulin resistance (HOMA-IR) method was first described by Matthews et al in 1985 [7]. HOMA is a mathematical model used to measure insulin resistance from basal glucose and insulin levels. Previous studies conducted on women with PCOS HOMA-IR have been used to assess insulin resistance [8-10]. Although the HOMA index has proven to be an accurate means to assess insulin resistance, it is difficult to perform in developing and low-economy countries due to lack of adequate equipment and high cost of insulin measurements.

Quantitative insulin sensitivity check index (QUICK) is a calculated data derived by taking both logarithms and the reciprocal of the fasting glucose and insulin values. Studies done by Hanley et al [11] and Chen H et al [12] evaluated thoroughly that QUICKI is a simple surrogate index for insulin sensitivity. Moreover, this is a calculated parameter again requires high-cost insulin measurement, which is not cost effective.

Fasting Glucose/Insulin ratio described by Legro et al [13] incorporated as a useful measure for detecting IR in obese PCOS women. However, Quon et al [14] demonstrated that the use of the fasting G/I ratio is limited in PCOS women with abnormal fasting glucose levels and this may lead to erroneous results.

These conflicting data regarding biological surrogate markers (HOMA-IR, QUICKI, and FG-IR indices) in women with PCOS limit their use in the clinical setting.

Hence an easy-to- detect surrogate marker for assessing IR representing a promising approach for maximizing treatment outcomes in women with PCOS is urgently required.

Dyslipidemia is one of the main characteristics of women with PCOS. Insulin resistanceis associated with abnormal lipid metabolism involving elevated triglycerides (TGs), low-densitycholesterol (LDL-c) levels and low high-density cholesterol (HDL-c) levels. [2] Studies have reported that superior ability of lipid ratios are better predictors of atherosclerosis and cardiovascular disease than conventional lipid parameters and are of particular clinical interest[2, 15]. Researchers have also suggested that serum lipid ratios have a significant positive correlation with IR and could be employed as surrogate indices [16, 17]. With this backdrop present study aimed to investigate whether a simple, effective and economical biological markersusing lipids and lipoproteins such as TG/HDL-C, TC/HDL-C, and TyG could be employed to determine IR in women with PCOS.

The study aimed to evaluate association between TyG ratio and other indices with IR (as measured by HOMA-IR, QUICKI, and FG-IR indices) and to evaluate the diagnostic utility of these markers in detecting IR among the women diagnosed with PCOS.

## MATERIALS AND METHODS:

A prospective cross-sectional observational study was conducted at a tertiary care teaching hospital in South India over a period of two months, from August 2022 to September 2022. The study aimed to investigate 100 diagnosed cases of Polycystic Ovary Syndrome (PCOS) among women aged 18 to 49 years who agreed to participate. PCOS diagnosis was based on the 2003 Rotterdam Criteria[18], requiring the presence of at least two of three criteria: 1) Oligo/anovulation, 2) hyperandrogenism, and 3) Polycystic ovaries observed on ultrasound imaging. Participants underwent assessments by gynecologists, and information regarding demographic characteristics, disease history, and medication use was collected. Exclusion criteria included major illness, use of specific medications (antilipidemic, anti-hypertensive, weight-loss, hormonal), hypertension, endocrine disorders, cardiovascular disease, hyperprolactinemia, pregnancy, and breastfeeding. Data analysis was performed using statistical software (Epi-Info). Quantitative data were presented as mean ± standard deviation (Min-Max), while categorical variables were expressed as frequency and percentage. Pearson's correlation coefficient was utilized for correlation analyses, considering a p-value $<0.05$ as significant. Logistic regression and ROC curve analyses were used to assess the ability of TyG parameter to predict IR in PCOS patients. Ethical clearance was obtained from the Institutional EthicalCommittee, and all participants provided informed consent before participating in the study, including history elicitation and sample collection for laboratory tests.

**Anthropometric and laboratory measurements:** Height assessment was conducted using a height measuring scale without shoes and recorded to the nearest 0.5 centimeters. Similarly, the weight of participants was measured to the nearest 0.5 kilograms without shoes and in light clothing. Body Mass Index (BMI) for each individual was calculated by dividing weight (in kilograms) by height squared (in meters) [body weight (kg)/height squared ($m^2$)].

Blood samples were drawn from the subjects after an eight-hour fasting period for the assessment of biochemical parameters, including fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C). These parameters were evaluated using a Vitros dry chemistry analyzer. The circulating levels of fasting insulin were measured using an ELISA kit, following the manufacturer's instructions (Qualisa, Zephyr Biomedicals, Goa, India). The TyG index was determined using the following calculations respectively: TG (mg/dL)/HDL-C (mg/dL), TC (mg/dL)/HDL-C (mg/dL), and Ln [TG (mg/dL) × FBG (mg/dL)]/2. Insulin resistance (IR) was definedas a HOMA-IR value of ≥2.63, an FG-IR value of < 8.25, and a QUICKI value of < 0.33. HOMA-IR was calculated as: [FBG (mg/dL)] × [fasting insulin (μU/ml)]/405[1]. FG-IR was determined as the fasting glucose (mg/dL)/fasting insulin (μU/ml). QUICKI was calculated usingthe formula: 1/(log fasting insulin [μU/ml] + log glucose [mg/dL]) [19]. Subjects were divided into insulin-resistant (IR) and insulin-sensitive (IS) groups based on HOMA-IR, FG-IR, and QUICKI indices. Clinical and biochemical parameters were compared between the two groups. Subsequently, the correlations of these parameters with FG-IR, HOMA-IR, and QUICKI indices were analyzed.

## RESULTS:

### Clinical and biochemical parameters of the study population.

Table 1. depicts the general characteristics and biochemical parameters of the subjects in the IR and IS groups, based on HOMA-IR and FG-IR values. It shows that IR exhibits a direct relationship with HOMA-IR and an indirect relationship with FG-IR and the QUICKI indices.As the analysis showed similar results with FG-IR and QUICKI index, comparison based on QUICKI index is not presented under this section.

The mean age of the study population was 28.84 ± 4.76 years, BMI was 27.58 ± 4.23 kg/$m^2$ anda mean fasting insulin level was 4.85 ± 2.38 μIU/ml. Out of 100 participants, 10% of the subjectshad a HOMA-IR value of ≥2.63, FG-IR value of < 8.25 and QUICKI value of < 0.33 categorizedas IR group. There was no difference found between the IR and the IS groups in relation with age, BMI, TG, TC and LDL-C levels as showed in Table 1. The FG-IR and QUICKI index IR groups had statistically significant higher circulating levels of fasting insulin, TC/HDL-C ratio, and HOMA-IR levels ($P < 0.05$) and statistically significant lower FG-IR and QUICKI values ($P < 0.001$) when compared to IS group counterpart. In addition, the HOMA-IR IR group exhibited increased levels of FBG, fasting insulin, TyG, TC/HDL-C, TG/HDL-C & HOMA-IR values and decreased levels of HDL-C, FG-IR and QUICKI values which was statistically significant when compared to there IS group counterpart.

**Table 1:** Clinical and biochemical parameters of the study population based to the FG-IR andHOMA-IR indices (IR & IS) in the women with PCOS.

| Clinical and biochemical parameters | Total PCOS (n=100) | FG-IR | | | HOMA-IR | | |
|---|---|---|---|---|---|---|---|
| | | IS, n = 90 (≥ 8.25) | IR, n = 10 (< 8.25) | P-value | IS, n = 90 (< 2.63) | IR, n = 10 (≥ 2.63) | P-value |
| Age (years) | 28.84 ± 4.76 | 28.80 ± 4.75 | 30.1 ± 4.51 | 0.15 | 28.81 ± 4.75 | 29.77 ± 4.53 | 0.30 |
| BMI (kg/m$^2$) | 27.58 ±4.23 | 27.58 ± 4.18 | 27.59 ± 4.51 | 0.99 | 27.66 ± 4.28 | 27.22 ± 4.01 | 0.57 |
| Insulin(μIU/mL) | 4.85 ± 2.38 | 4.12 ± 1.87 | 13.88 ± 1.46 | < 0.001 | 4.14 ± 1.86 | 13.84 ± 1.44 | < 0.001 |
| FBG (mg/dL) | 90.57 ± 9.46 | 90.58 ± 9.51 | 90.39 ± 9.12 | 0.90 | 89.69 ± 8.85 | 98.59 ± 11.14 | < 0.001 |
| TC (mg/dL) | 173.45 ±36.23 | 172.55 ±36.69 | 181.71 ± 31.09 | 0.19 | 172.58 ± 36.25 | 181.45 ± 35.68 | 0.21 |
| TG (mg/dL) | 117± 35.23 | 117±35.23 | 118±36.25 | 0.74 | 115±36.14 | 126±46.24 | 0.08 |
| LDL-C (mg/dL) | 99.53 ± 29.07 | 99.63 ± 29.53 | 98.62± 24.85 | 0.86 | 99.11 ± 29.00 | 103.37 ± 29.85 | 0.45 |
| HDL-C (mg/dL) | 45 ±5.15 | 45 ±5.15 | 44 ±5.13 | 0.19 | 45 ±6.16 | 42 ±7.22 | 0.049 |
| TC/HDL-C | 4.03 ± 1.14 | 3.98 ± 1.13 | 4.47 ± 1.16 | 0.026 | 3.97 ± 1.10 | 4.58 ± 1.35 | 0.005 |
| TG/HDL-C | 2.67± 1.15 | 2.67± 1.15 | 2.71± 1.15 | 0.76 | 2.65± 1.16 | 3.34± 2.24 | 0.035 |
| TyG | 3.96 ± 0.23 | 3.96 ± 0.23 | 3.98 ± 0.22 | 0.71 | 3.95 ± 0.22 | 4.10 ± 0.25 | 0.004 |
| FG-IR | 21.74± 8.69 | 22.76± 10.29 | 8.04± 1.06 | < 0.001 | 22.75±10.35 | 8.48± 1.44 | < 0.001 |
| HOMA-IR | 0.97± 0.69 | 0.88± 0.43 | 2.96 ± 0.40 | < 0.001 | 0.88± 0.43 | 2.99 ± 0.42 | < 0.001 |
| QUICKI | 0.38 ± 0.04 | 0.39 ± 0.03 | 0.31 ± 0.01 | < 0.001 | 0.39 ± 0.04 | 0.31 ± 0.01 | < 0.001 |

P-value < 0.05 =Significant

IR - Insulin-resistant and IS-Insulin-sensitive

**Correlation between the FG-IR, QUICKI and HOMA-IR indices with TyG and other indices**

Table 2 shows the correlations between FG-IR, QUICKI and HOMA-IR indices with TyG. TyG, TC/HDL-C, and TG/HDL-C showed negative association with FG-IR and QUICKI indices ($P < 0.05$), and positive association with the HOMA-IR index ($P < 0.05$).

**Table 2:** Correlation between FG-IR, QUICKI, and HOMA-IR indices with TyG and otherindices in the women with PCOS

| Clinical and biochemical parameters | FG-IR | P-value | QUICKI | P-value | HOMA-IR |
|---|---|---|---|---|---|
| TC/HDL-C | -0.174 | 0.002 | -0.206 | < 0.001 | 0.246 |
| TG/HDL-C | -0.14 | 0.0088 | -0.184 | 0.001 | 0.213 |
| TyG | -0.161 | 0.004 | -0.239 | < 0.001 | 0.231 |

P-value < 0.05 =Significant

**Association between the TyG index with IR**

Table 3 shows association between the lipid ratios, and TyG index between FG-IR and HOMA-IR indices with TyG through regression analysis. Due to the similar results obtained based on theQUICKI index and FG-IR index, they were not showed here. TyG (10.831, 95% CI [2.013–58.234]

**Table 3:** Association between FG-IR and HOMA-IR indices with the TyG in the women withPCOS

| Variables | FG-IR | | | HOMA-IR | | |
|---|---|---|---|---|---|---|
| | OR | 95% CI | P-value | OR | 95% CI | P-value |
| TC/HDL-C | 1.411 | 1.043-1.957 | 0.023 | 1.550 | 1.133-2.122 | 0.0059 |
| TG/HDL-C | 1.116 | 0.904-1.376 | 0.29 | 1.294 | 1.064-1.574 | 0.0088 |
| TyG | 1.323 | 0.266-6.551 | 0.71 | 10.831 | 2.013-58.234 | 0.0049 |

P-value < 0.05 =Significant

**Area under curve in predicting the HOMA-IR and FG-IR indices.**

Table 4 showed that TyG, TG/HDL-C and TC/HDL-C strongly predicted HOMA-IR with AUC values of 0.639, 0.619 and 0.623 respectively (P < 0.05). Only TC/HDL-C significantly predicted FG-IR with an AUC of 0.60 (P = 0.039). In contrast conventional lipid profile (TG, TC, LDL-C, and HDL-C) does not predict both HOMA-IR and FG-IR indices.

**Table 4:** Area under curve, sensitivity, specificity by the optimized cut-off points for TyG,TG/HDL-C and TC/HDL-C indices in predicting the HOMA-IR and FG-IR indices.

| | Predictors | AUC | P-value | Cut-off value | Sensitivity | Specificity |
|---|---|---|---|---|---|---|
| HOMA-IR | TyG | 0.639 | 0.010 | ≥4.00 | 0.61 | 0.58 |
| | TG/HDL-C | 0.619 | 0.031 | ≥2.80 | 0.58 | 0.54 |
| | TC/HDL-C | 0.623 | 0.025 | ≥4.00 | 0.58 | 0.55 |
| FG-IR | TyG | 0.51 | 0.74 | ≥3.961 | 0.55 | 0.49 |
| | TG/HDL-C | 0.54 | 0.36 | ≥2.673 | 0.48 | 0.49 |
| | TC/HDL-C | 0.60 | 0.039 | ≥4.10 | 0.58 | 0.56 |

P-value < 0.05 =Significant

## DISSCUSSION:

Insulin resistance has a pivot role in the pathophysiology of polycystic ovary syndrome (PCOS). Association between lipid ratios and IR may vary with respect to ethnicity and some of the indices are not relevant to predict IR in certain populations. The study was conducted to evaluate the correlation between fasting triglyceride-glucose (TyG) indice with IR (as measured by homeostasis model assessment of IR (HOMA-IR), quantitative insulin sensitivity check index (QUICKI) and fasting glucose to insulin ratio (FGIR)) among PCOS subjects.

High levels of fasting blood glucose (FBG), triglycerides (TG), total cholesterol (TC), and low levels of high-density lipoprotein cholesterol (HDL-C) are hallmark features of insulin resistance (IR) and metabolic syndromes. Although the IR group in our study did not exhibit dyslipidemia, lipid ratios such as TC/HDL-C, TG/HDL-C, and TyG indices were significantly elevated in subjects with a HOMA-IR value of ≥2.63 compared to those with a HOMA-IR value of < 2.63. However, only the TC/HDL-C ratio was notably higher in the IR group determined by the FG-IR and QUICKI indices. These findings align with a study conducted by Kheirollahil A et al. [1] on Iranian PCOS women, which suggested that these ratios could serve as more effective indicators of IR than traditional lipid parameters. Therefore, elevated lipid ratios serve as robust predictors of IR and its associated complications in women with PCOS [20]. To evaluate the diagnostic utility of these markers in detecting IR among the women diagnosed with PCOS AUC analysis was considered using the control cut-off values for the HOMA-IR and FG-IR indices as 2.63 and 8.25 respectively. Although, present study does not show any predictability for IR by traditional lipid parameters. On contrast, the AUC values of TG/ HDL-C, TC/HDL-C, and TyG were acceptable for predicting the HOMA-IR and FG-IR indices these findings were consistent with prior study findings [1]. Thus, these comprehensive analyses evident those lipid ratios are considered to be an effective and beneficial diagnostic indicator for IR in the PCOS.

The current study is the first to assess the diagnostic ability of TyG index among the women with PCOS. The AUC value is highest for TyG based on HOMA-IR and TC/ HDL-C ratio based on FG-IR which is similar to the study done by Kheirollahil A et al [1]. On the top of that this finding is conflicting to the results from a previous study done by Ghaffarzad et al. [16] showed that the highest AUC value is for TG/HDL-C based on HOMA-IR index. This could be due to the discrepancy in sample size recruited in research. In the present study, the optimal cut-off points of TyG, TG/HDL-C and TC/HDL-C for predicting IR based on the HOMA-IR levels were 4 (sensitivity of 61%, specificity of 58%), 2.8 (sensitivity of 58%, specificity of 54%) and 4 (sensitivity of 58%, specificity of 55%), respectively. The optimal cut-off value of TC/HDL-C using the model based on the FG-IR levelswas 4.10 (sensitivity of 58%, specificity of 56%). One of the previous studies done by Ghaffarzad et al. [16] on Iranian women with PCOS with a smaller sample size, the best cut-off point of TG/HDL-C and TC/HDL-C levels were 3.19 (sensitivity of 63.6%, specificity of 84.4%) and 4.37 (sensitivity of 69.7%, specificity of 65.6%), respectively. In another study by Song DK et al. [21] on the young Korean women with PCOS, the cut-off value of the TG/ HDL-C ratio to predict the IR as estimated by the HOMA-IR index was noted as 2.5 (sensitivity of 61%, specificity of 82%). Thus, the study is the original comprehensive study inspecting the diverse metabolic indices for assessing IR by using the HOMA-IR, FG-IR and QUICKI indices among PCOS women.

## CONCLUSION:

To conclude in the present study TG/HDL-C, TC/HDL-C, and TyG indices values showed significant difference among insulin-resistant (IR) and insulin-sensitive (IS) groups, confirmed by the HOMA-IR, FG-IR, and QUICKI values. AUC analyses showed that TyG, TG/ HDL-C, and TC/HDL-C strongly predicted HOMA-IR with area under the curve (AUC) of 0.639, 0.619, and 0.623, respectively ($P < 0.05$). Further, TC/HDL-C was a good predictor of FG-IR with AUC of 0.60 ($P = 0.039$). To conclude TyG, TG/HDL-C, and TC/HDL-C indices could be the indicators of IR among women diagnosed with PCOS.

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**CONFLICT OF INTEREST -** None

## REFERENCES

1. Kheirollahil A, Teimouri M, Karimi M, Vatannejad A, Moradi N, Borumandnia N, Sadeghi A. Evaluation of lipid ratios and triglyceride-glucose index as risk markers of insulin resistance in Iranian polycystic ovary syndrome women. Lipids in Health and Disease. 2020; 19:235 https://doi.org/10.1186/s12944-020-01410-8
2. Amisi CA. Markers of insulin resistance in Polycystic ovary syndrome women: An update. World J Diabetes. 2022;13(3):129-149.
3. Polak K, Czyzyk A, Simoncini T, Meczekalski B. New markers of insulin resistance in Polycystic ovary syndrome. J Endocrinol Invest. 2017;40:1-8 [PMID: 27473078 DOI: 10.1007/s40618-016-0523-8]
4. Amato MC, Vesco R, Vigneri E, Ciresi A, Giordano C. Hyperinsulinism and Polycystic ovary syndrome (PCOS): role of insulin clearance. J Endocrinol Invest. 2015; 38: 1319-1326 [PMID: 26294351 DOI: 10.1007/s40618-015-0372-x]
5. Divall S, Wondisford F, Wolfe A. Reproductive tissues maintain insulin sensitivity in diet induced obesity. Diabetes. 2012;61(1):114–123
6. Palomba S, Daolio J, La Sala GB. Oocyte competence in women with polycystic ovary syndrome. Trends Endocrinol Metab. 2017;28(3):186–98.
7. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia.1985;28:412-419 [PMID: 3899825 DOI: 10.1007/BF00280883]
8. Cebeci F, Onsun N, Mert M. Insulin resistance in women with hirsutism. Arch Med Sci. 2012; 8: 342-346 [PMID: 22662009 DOI: 10.5114/aoms.2012.28563].
9. Lankarani M, Valizadeh N, Heshmat R, Peimani M, Sohrabvand F. Evaluation of insulin resistance and metabolic syndrome in patients with Polycystic ovary syndrome. Gynecol Endocrinol. 2009; 25: 504-507 [PMID: 19499403 DOI:10.1080/09513590902972083].

10. Wei HJ, Young R, Kuo IL, Liaw CM, Chiang HS, Yeh CY. Prevalence of insulin resistance and determination of risk factors for glucose intolerance in Polycystic ovary syndrome: a cross-sectional study of Chinese infertility patients. Fertil Steril. 2009; 91: 1864-1868 [PMID: 18565519 DOI: 10.1016/j.fertnstert.2008.02.168].
11. Hanley AJ, Williams K, Gonzalez C, D'Agostino RB Jr, Wagenknecht LE, Stern MP, Haffner SM; San Antonio Heart Study; Mexico City Diabetes Study; Insulin Resistance Atherosclerosis Study. Prediction of type 2 diabetes using simple measures of insulin resistance: combined results from the San Antonio Heart Study, the Mexico City Diabetes Study, and the Insulin Resistance Atherosclerosis Study. Diabetes. 2003; 52: 463-469 [PMID: 12540622 DOI: 10.2337/diabetes.52.2.463]
12. Chen H, Sullivan G, Quon MJ. Assessing the predictive accuracy of QUICKI as a surrogate index for insulin sensitivity using a calibration model. Diabetes. 2005; 54: 1914-1925 [PMID: 15983190 DOI: 10.2337/diabetes.54.7.1914]
13. Legro RS, Finegood D, Dunaif A. A fasting glucose to insulin ratio is a useful measure of insulin sensitivity in women with Polycystic ovary syndrome. J Clin Endocrinol Metab. 1998; 83: 2694-2698 [PMID: 9709933 DOI:10.1210/jc.83.8.2694]
14. Quon MJ. Limitations of the fasting glucose to insulin ratio as an index of insulin sensitivity.

    J Clin Endocrinol Metab. 2001; 86: 4615-4617 [PMID: 11600512 DOI: 10.1210/jcem.86.10.7952]
15. Tangvarasittichai S, Poonsub P, Tangvarasittichai O. Association of serum lipoprotein ratios with insulin resistance in type 2 diabetes mellitus. Indian J Med Res. 2010; 131: 641-648 [PMID: 20516535]
16. Ghaffarzad A, Amani R, Mehrzad Sadaghiani M, Darabi M, Cheraghian B. Correlation of Serum Lipoprotein Ratios with Insulin Resistance in Infertile Women with Polycystic Ovarian Syndrome: A Case Control Study. Int J Fertil Steril. 2016; 10: 29-35 [PMID:27123197 DOI: 10.22074/ijfs.2016.4765]
17. Xiang SK, Hua F, Tang Y, Jiang XH, Zhuang Q, Qian FJ. Relationship between Serum Lipoprotein Ratios and Insulin Resistance in Polycystic ovary syndrome. Int J Endocrinol. 2012; 2012: 173281 [PMID: 22792101 DOI: 10.1155/2012/173281].
18. Björndahl L, Giwercman A, Tournaye H. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod. 2004;19:41–7.
19. Gutch M, Kumar S, Razi SM, Gupta KK, Gupta A. Assessment of insulin sensitivity/resistance. Indian J Endocrinol Metab. 2015;19:160
20. Traub ML. Assessing and treating insulin resistance in women with polycystic ovarian syndrome. World J Diabetes. 2011;2(3):33-40. doi: 10.4239/wjd.v2.i3.33. PMID: 21537458; PMCID: PMC3083905.
21. Song DK, Lee H, Sung YA, Oh JY. Triglycerides to high-density lipoprotein cholesterol ratio can predict impaired glucose tolerance in young women with polycystic ovary syndrome. Yonsei Med J. 2016;57:1404 –11.