Original Research Article

Association of triglycerides/high density lipoprotein cholesterol ratio with insulin resistance in polycystic ovary syndrome

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Received: 07 October 2018
Accepted: 29 October 2018

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ABSTRACT

Background: Insulin resistance (IR) is frequently observed in women with polycystic ovary syndrome (PCOS). Recent studies advocated that triglyceride to high-density lipoprotein cholesterol ratio (TG/HDL-C) can be used as a simple clinical indicator of IR. Hence, the present study was performed to investigate the use of TG/HDL-C and its association with IR in PCOS.

Methods: Forty-one patients with PCOS and 40 healthy age matched women were randomly enrolled. Demographic and clinical characteristics were obtained. Insulin resistance was defined by the homeostasis model assessment for insulin resistance (HOMA-IR) and quantitative insulin sensitivity check index (QUICKI).

Results: In PCOS group, the insulin, HOMA-IR and TG/HDL-C ratio were significantly higher (p=0.001) than controls while, QUICKI was lower (p=0.001). Insulin, HOMA-IR were positively correlated with TG/HDL-C (r=0.303, p=0.006 and r=0.312, p=0.005 respectively) while, QUICKI was negatively correlated (r=-0.698, p=0.001). In receiver operating characteristic (ROC) analysis, area under the curve (AUC) for model based on QUICKI levels was better 0.898 (95% CI: 0.811-0.955, p=0.001) than HOMA-IR 0.636 (95% CI: 0.522-0.740, p=0.03). A cut-off value 3.23 for TG/HDL-C is proposed from the model based on QUICKI with best combination of sensitivity 83.3% and specificity 86.7%.

Conclusions: Results of present study support that TG/HDL-C ratio may be a simple indicator of IR in PCOS patients which helps clinicians to identify IR in small centers, where the assays for insulin measurement are not available.

Keywords: HOMA-IR; insulin resistance, QUICKI, PCOS, TG/HDL-C ratio

INTRODUCTION

Poly Cystic Ovary Syndrome (PCOS) is the most common form of chronic anovulation associated with androgen excess, occurring in 5-10% of reproductive age women.1 PCOS is associated with increased cardiometabolic risk factors.2 Insulin resistance (IR) has been shown to be the determinant of cardiovascular risk independent of obesity in PCOS women and is also seen in non obese women with PCOS.2,3 However, obesity is known to exacerbate the underlying insulin resistance in PCOS women.4

The gold standard methods designed to measure insulin sensitivity are the hyperinsulinemic euglycemic clamp and intravenous glucose tolerance test (FSIVGTT). These are impractical in the clinical setting since it requires intravenous infusion and can only be performed in specialized centers.5 Other surrogate markers based on fasting insulin and glucose levels have been proposed...
such as the fasting insulin levels (FIL), the whole body insulin sensitivity index (WBISI), the homeostasis model assessment of IR (HOMA-IR), quantitative insulin sensitivity check index (QUICKI), Matsuda index, Avignon index, Stumvoll index, and the new simple index assessing insulin sensitivity using oral glucose tolerance test (SIsOGTT).6 These indices present important limitations related to their poor reproducibility and reliability.7 In addition, no clear guidelines and no universally accepted cutoffs are available for most of the main surrogate markers used.5

Hypertriglyceridemia and low HDL-cholesterol are two key metabolic abnormalities associated with IR states.8,9 Similar to fasting serum insulin levels, the triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio was found to be correlated with IR and hence is considered to indicate the concomitant presence of IR and dyslipidemia.10 TG/HDL-C has been shown to predict IR.11 In developing countries, the availability and cost of insulin assay can be a major limiting factor for the assessment of insulin resistance. Hence, use of alternate markers like the TG/HDL-C ratio which is feasible even in small centres and is cost-effective can be a useful alternate. The present study was thus taken up to study IR and its correlation with TG/HDL-C ratio in PCOS women and to assess the diagnostic utility of TG/HDL-C ratio in identifying IR in PCOS women.

METHODS

Forty-one PCOS patients attending the Endocrinology outpatient Department of Sri Venkateswara Institute of Medical Sciences, Tirupati, Andhra Pradesh, aged 20-38 years and diagnosed with PCOS based on National Institutes of Health (NIH) consensus 1990 criteria were included after informed consent.12 The diagnostic criteria according to the NIH consensus 1990 criteria were oligomenorrhea (≤9 menses/year) or amenorrhea (no menstrual periods for 3 or more months), hyperandrogenism and/or hyperandrogenaemia and after exclusion of related disorders with similar presentation like hypothyroidism [thyroid stimulating hormone (TSH) >5mIU/mL], hyperprolactinaemia (serum prolactin >100ng/mL), Cushing’s syndrome (cortisol >2µg/dL), adrenal hyperplasia and androgen secreting tumours (testosterone levels greater than 3 times the upper reference limit associated with relevant clinical features).Women with virilization, pregnancy, those on oral contraceptives, glucocorticoids, anti-androgens, ovulation inducing agents, antidiabetic drugs or obesity drugs or other hormonal drugs during the previous 6 months were excluded from the study. Forty age-matched healthy females from among the hospital staff were taken as controls. The criteria for healthy control group were absence of menstrual irregularities, hirsutism and major medical illness. Sample size calculation was done based on data from previous studies. The study was approved by Institutional ethics committee.

Sample collection

Around 5ml of venous blood was collected from both controls and PCOS women, following 12hr of fasting. The plain samples were allowed to stand for half-an-hour and centrifuged at 3000rpm for 15min whereas the samples from anticoagulant bottle were centrifuged immediately and the plasma was separated. The serum and plasma samples obtained were stored at -80°C until biochemical analysis.

Biochemical analysis

The plasma glucose was determined by glucose oxidase-peroxidase (GOD-PDO) method (Autospan, Gujarat, India). Lipid profile was estimated using commercial kits from Aspen laboratories (Delhi, India) for TC, Accurex (Thane, India) for TG by enzymatic methods. HDL-C kits were obtained from Beckman Coulter (Galway, Ireland). All the parameters were analyzed on Beckman CX-9 fully automated analyzer (Galway, Ireland). LDL-C levels were calculated using Friedewald’s formula.13 Serum Insulin was determined by ELISA method using commercial kit (Dia source kit, Belgium). Insulin Resistance was calculated as Homeostasis Model Assessment-Insulin Resistance (HOMA-IR) using the formula: HOMA-IR = fasting insulin (µU/mL) x fasting glucose (mmol/L)/22.5.14 Insulin sensitivity is calculated as quantitative insulin sensitivity check index (QUICKI) formula: 1/[log (insulin µU/mL)+log (glucose mg/dL)].15

Statistical analysis

Kolmogorov-Smirnov test was used to evaluate the distribution of continuous variables. Data was expressed as mean and standard deviation or median (IQR, interquartile range), depending on the data distribution. Statistical comparisons of the groups were made using an unpaired t-test or Mann Whitney U test, as appropriate. Spearman rank correlation was used to explore the associations among the variables (TG/HDL-C ratio with insulin, HOMA-IR and QUICKI). Logistic regression analysis was performed to determine the association of TG/HDL-C ratio with, HOMA-IR and QUICKI as dependant variable. Statistical analysis was performed using Microsoft excel spreadsheet sheet and SPSS for windows version 11.5. A ‘p’ value of <0.05 was considered statistically significant.

RESULTS

Table 1 shows the baseline and biochemical characteristics of the PCOS women and controls. There was no significant difference in age, BMI and fasting glucose between PCOS women and controls. The TG/HDL-C ratio was found to be significantly elevated in PCOS women compared to controls. Table 2 shows the insulin resistance markers i.e., insulin, HOMA-IR and QUICKI. Among these, insulin, HOMA-IR levels were significantly elevated in PCOS women. Concomitantly,
insulin sensitivity marker QUICKI was significantly decreased. TG/HDL-C ratio showed a significant positive correlation with insulin and HOMA-IR with ($p$=0.006; $p$=0.312, $p$=0.005). On the other hand, QUICKI showed a significant negative correlation with TG/HDL-C ratio ($p$= -0.698, $p$=0.001) (Table 3).

### Table 1: Baseline and biochemical characteristics of the study subjects.

| Characteristic | Controls | Cases | $p$-value |
|---------------|----------|-------|-----------|
| Age (years)   | 24.33±5.06 | 22.75±5.60 | 0.210 |
| BMI (kg/m²)   | 24.80±2.64 | 24.05±2.16 | 0.195 |
| FBG (mg/dL) | 92.50±5.63 | 93.02±11.39 | 0.794 |
| Total cholesterol (mg/dL) | 148.73±26.43 | 171.66±37.15 | 0.002* |
| Triglycerides (mg/dL) | 107.25±38.29 | 151.24±63.97 | 0.001* |
| HDL-C (mg/dL) | 45.15±4.16 | 36.73±3.65 | 0.001* |
| LDL-C (mg/dL) | 82.13±26.89 | 105.76±37.91 | 0.001* |
| VLDL-C (mg/dL) | 21.45±7.65 | 30.25±12.79 | 0.001* |
| TG/HDL-C | 2.41±0.97 | 4.15±1.71 | 0.001* |

Data was expressed as mean and standard deviation; BMI - body mass index; FBG - fasting blood glucose; HDL-C - high density lipoprotein cholesterol; LDL-C - low density lipoprotein cholesterol; VLDL-C - very low density lipoprotein cholesterol; TG/HDL-C - ratio between triglycerides and HDL cholesterol; HOMA-IR - homeostatic model assessment of insulin resistance; QUICKI - quantitative insulin sensitivity check index; * - statistically significant.

### Table 2: Insulin resistance markers in the study subjects.

| Variable            | Controls       | Cases        | $p$-value |
|---------------------|----------------|--------------|-----------|
| Insulin (µIU/mL)**  | 7.17 (4.99-14.91) | 15.30 (10.82-25.81) | 0.001* |
| HOMA-IR**           | 1.57 (1.06-3.74) | 3.64 (2.51-5.54) | 0.001* |
| QUICKI              | 0.34 ± 0.03    | 0.28 ± 0.04  | 0.001* |

Data was expressed as mean and standard deviation; ** median (IQR, inter quartile range); HOMA-IR - homeostatic model assessment of insulin resistance; QUICKI - quantitative insulin sensitivity check index; * - statistically significant.

### Table 3: Correlation between TG/HDL-C ratio and insulin resistance markers in PCOS patients.

| Parameter | $p$-value | $p$-value |
|-----------|-----------|-----------|
| Insulin   | 0.303     | 0.006*    |
| HOMA-IR   | 0.312     | 0.005*    |
| QUICKI    | -0.698    | 0.001*    |

$r$ = spearman’s correlation coefficient, TG/HDL-C-ratio between triglycerides and HDL cholesterol; HOMA-IR - homeostatic model assessment of insulin resistance; QUICKI - quantitative insulin sensitivity check index; * statistically significant.

Receiver operating characteristic curve analysis was performed for TG/HDL-C ratio to discriminate those who were insulin resistant from those who were insulin sensitive using control cutoff values for HOMA-IR and QUICKI (i.e. 2.84 and 0.34 respectively). The AUC for the model based on QUICKI was superior 0.898 (95% CI: 0.811-0.955, p=0.001) than HOMA-IR 0.636 (95% CI: 0.522-0.740, $p=0.03$). Cutoff value with the best combination of sensitivity and specificity was obtained from the model based on QUICKI levels. A cutoff value of 3.23 for TG/HDL-C ratio was proposed with sensitivity 83.3% and specificity 86.7% (Figure 1, 2).
important which women (p=0.001). was significant compared insulin insulin from this study, standardized Model TG/HDL C coefficient; Model 1: HOMA-IR as a dependent variable, Model 2: QUICKI as a dependant variable, B-unstandardized coefficient; β-standardized coefficient; *-statistically significant

**Figure 2:** Receiver operating characteristics (ROC) curve analysis for TG/HDL-C ratio cutoff value based on the QUICKI model.

Logistic regression analysis using TG/HDL-C ratio (with cutoff value of 3.23) as independent variable and insulin resistance markers i.e., HOMA-IR and QUICKI as dependent variables showed significant negative association of QUICKI (standard coefficient β= -0.895, p<0.001), while no association was found with insulin and HOMA-IR (Table 4).

**Table 4: Logistic Regression analysis.**

| Independent variable | B   | Std. Error | β     | t-value | p-value |
|----------------------|-----|------------|-------|---------|---------|
| Model 1 TG/HDL-C     | -1.201 | 0.913      | -0.206 | -1.314  | 0.196   |
| Model 2 TG/HDL-C     | -0.007 | 0.001      | -0.895 | -12.39  | 0.001 * |

**DISCUSSION**

In the present study PCOS women were found to be insulin resistant as evident from increased levels of serum insulin and increase in HOMA-IR in PCOS women compared to controls (p=0.001). On the other hand, a significant decrease in insulin sensitivity marker QUICKI was seen in PCOS women compared to controls (p=0.001). This is in agreement with previous reports. Mechanisms underlying insulin resistance in these women are unclear. Multiple factors have been implicated; of which hyperandrogenemia and obesity which are commonly seen in these women along with alteration of adipose tissue morphology seems to play an important role. Hyperandrogenism produces its effect through increased lipolysis. Another mechanism put forth is the central/visceral distribution pattern of body fat favored by hyperandrogenemia. The increased lipolytic activity of visceral fat results in increased free fatty acid flux leading to skeletal muscle insulin resistance. Androgens also seem to have an inhibitory effect on lipoprotein lipase activity and women with PCOS have been shown to have lower lipoprotein lipase activity. Insulin resistance is an important cause of dyslipidemia in PCOS women.

In the present study, PCOS women had significantly increased triglycerides, and lower HDL-C when compared to controls (p = 0.001). TG/HDL-C in PCOS women was found to be significantly higher than those of the age-matched healthy women (p = 0.001). This is in agreement with previous reports. The dyslipidemia in the setting of polycystic ovary syndrome can occur due to multiple causes. The increased prevalence of obesity, insulin resistance and hyperandrogenemia have all been proposed to be involved in the lipoprotein disturbances observed in PCOS women. Increased lipogenesis, decreased clearance, reduced oxidation of fatty acids and their increased availability and an increased secretion of very low density lipoprotein (VLDL) particles by the hepatocytes contribute to the increased triglyceride levels in the presence of insulin resistance.

A significant positive correlation was observed between insulin, HOMA-IR with TG/HDL-C ratio (r=0.303, p=0.006; r=0.312, p=0.005). On the other hand, QUICKI showed a significant negative correlation with TG/HDL-C ratio (r=-0.698, p=0.001) (Table 3). The relationship between the TG/HDL-C ratio and a direct measure of IR was first reported by Mc Laughlin T et al and they proposed a cutoff value of 3.0 for TG/HDL-C ratio which showed 57% sensitivity and 71% specificity for detecting IR. However, different cut-off values have been proposed in different ethnic groups.

Racial differences in lipoprotein lipase (LPL) activity may be responsible for racial differences in TG. African-Americans have been reported to have higher LPL levels than the caucasians. Insulin resistance causing impairment in lipoprotein lipase activity thereby leading to higher TG levels has been reported in caucasians, while no such impairment has been seen in African-Americans. Indian studies evaluating surrogate markers of IR found a good correlation of TG/HDL-C ratio with fasting glucose-to-insulin ratio (G/I ratio) in adolescent girls with PCOS. In the present study, the IR was measured using HOMA-IR and QUICKI and included women in the range of 20-38 yrs.

To assess the diagnostic ability of TG/HDL-C ratio to discriminate those who were insulin resistant from those who were insulin sensitive, receiver operating characteristic curve analysis using control cutoff values for HOMA-IR and QUICKI (i.e. 2.84 and 0.34 respectively). The AUC for the model based on QUICKI

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CONCLUSION

To conclude, the present study shows a significant association between TG/HDL-C ratio and the IR markers i.e., HOMA-IR, QUICKI in the PCOS group and thus TG/HDL-C ratio can be used as a marker of IR. The TG/HDL ratio can thus serve as a simple, convenient and inexpensive surrogate marker which can be used to screen PCOS women for IR in Indians.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Ilhan T, Berrin C, Zeynep C, Erdem T. The plasma homocysteine concentrations and relationship with insulin resistance in young women with polycystic ovary syndrome. Turkish J Endocrinol Meta. 2005;1:23-8.
2. Mather KJ, Kwan F, Corenblum B. Hyperinsulinemia in polycystic ovary syndrome correlates with increased cardiovascular risk independent of obesity. Fertil Steril. 2000;73:150-6.
3. Morales AJ, Laughlin GA, Butzow T, Maheshwari H, Baumann G, Yen SS. Insulin, somatotropic, and luteinizing hormone axes in lean and obese women with polycystic ovary syndrome: common and distinct features. J Clin Endocrinol Meta. 1996;81:2854-64.
4. Nestle JE. Obesit, insulin, sex steroids and ovulation. Int J Obes Relat Metab Dis. 2000;24(Suppl)2:S71-3.
5. Levy-Marchal C, Arslanian S, Cutfield W, Sianko S, Druet C, Marcovecchio ML, et al. Insulin resistance in children: consensus, perspective, and future directions. J Clin Endocrinol Meta. 2010;95:5189-98.
6. Pisprasert V, Ingram KH, Lopez-Davila MF, Munoz AJ, Garvey WT. Limitations in the use of indices using insulin and insulin levels to predict insulin sensitivity: impact of race and gender and superiority of the indices derived from oral glucose tolerance test in African Americans. Diabetes Care. 2013;36:845-53.
7. Anderwald C, Anderwald-Stadler M, promptinter M, Prager G, Mandl M, Nowotny P, et al. The clamp like index: a novel and highly sensitive insulin sensitivity index to calculate hyperinsulinemic clamp glucose infusion rates from oral glucose tolerance test in non diabetic subjects. Diabetes Care. 2007;30:2374-80.
8. Lewis GF, Uffelman KD, Szeto LW, Steiner G. Effects of acute hyperinsulinemia on VLDL triglyceride and VLDL apoB production in normal weight and obese individuals. Diabetes. 1993;42:833-42.
9. Van Linthout S, Spillmann F, Schultheiss HP, Tschope C. High-density lipoprotein at the interface of type 2 diabetes mellitus and cardiovascular disorders. Curr Pharm Des. 2010;16:1504-16.
10. Li C, Ford ES, Meng YX, Mokdad AH, Reaven GM. Does the association of the triglyceride to high density lipoprotein cholesterol ratio with fasting serum insulin differ by race/ethnicity? Cardiovas Diabetol. 2008;28:7:4.
11. Mc Laughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, et al. Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? Am J Cardiol. 2005;96:399-404.
12. Zawadzki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In: Dunaif, A, Givens, JR, Haseltine, FP, Merriam, GE, editors. Current Issues in Endocrinology and Metabolism: Polycystic ovary syndrome. Boston: Blackwell Scientific Publications;1992:377.
13. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18:499e502.
14. 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;28:412-19.
15. Duncan MH, Singh BM, Wise PH, Carter G, Alagband-Zadeh J. A simple measure of insulin resistance. Lancet. 1995;346:120-1.
16. Tarkun I, Arslan BC, Cantürk Z, Türemen E, Sahin T, Duman C. Endothelial dysfunction in young women with polycystic ovary syndrome: relationship with insulin resistance and low-grade chronic inflammation. J Clin Endocrinol Meta. 2004;89:5592-6.
17. Mai K, Bobbert T, Reinecke F, Andres J, Maserc-Gluth C, Wudy SA, et al. Intravenous lipid and heparin infusion-induced elevation in free fatty acids and triglycerides modifies circulating
androgen levels in women: a randomized, controlled trial. J Clin Endocrinol Metab. 2008;93:3900-6.
18. Kahn BB, Flier JS. Obesity and insulin resistance. J Clin Invest. 2000;106:473-81.
19. Mannerås-Holm L, Leonhardt H, Kullberg J, Jennische E, Odén A, Holm G, et al. Adipose tissue has aberrant morphology and function in PCOS: enlarged adipocytes and low serum adiponectin, but not circulating sex steroids, are strongly associated with insulin resistance. J Clin Endocrinol Metab. 2011;96:E304-11.
20. Hong Y, Yang D, Liu W, Zhao X, Chen X, Li L. Dyslipidemia in relation to body mass index and insulin resistance in Chinese women with polycystic ovary syndrome. J Biol Regul Homeost Agents. 2011;25:365-74.
21. Dunaf A. Insulin Resistance and the Polycystic Ovary Syndrome: Mechanism and Implications for Pathogenesis Endocrine Reviews. 1997;18:774-800.
22. Shoaiab OM, Mustafa SM, Nourein IH. Serum lipid profile of polycystic ovary syndrome in Sudanese women. Inter J Med Sci Pub Heal. 2015;4:1605-10.
23. Mc Laughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G. Use of metabolic markers to identify overweight individuals who are insulin resistant. Ann Intern Med. 2003;139:802-9.
24. Giannini C, Santoro N, Caprio S, Kim G, Lartaud D, Shaw M, et al. The triglyceride to HDL cholesterol ratio. Diabetes Care. 2011;34:1869-74.
25. Kim-Dorner SJ, Deuster PA, Zeno SA, Remaley AT, Poth M. Should triglycerides and the triglycerides to high-density lipoprotein cholesterol ratio be used as surrogates for insulin resistance? Metabolism. 2010;59:299-304.
26. Gasevic D, Frohlich J, Mancini GB, Lear SA. The association between triglyceride to high-density-lipoprotein cholesterol ratio and insulin resistance in a multiethnic primary prevention cohort. Metabolism. 2012;61:583-9.
27. Sumner AE, Finley KB, Genovese DJ, Criqui MH, Boston RC. Fasting triglyceride and the triglyceride-HDL cholesterol ratio are not markers of insulin resistance in African Americans Arch Intern Med. 2005;165:1395-1400.
28. Eckel RH, Yost TJ, Jensen D. Alterations in lipoprotein lipase in insulin resistance. Int J Obes. 1995;19(suppl 1):S16-S21.
29. Sumner AE, Vega GL, Genovese DJ, Finley KB, Bergman RN, Boston RC. Normal triglyceride levels despite insulin resistance in African Americans: role of lipoprotein lipase. Metabolism. 2005;54:902-9.
30. Anuradha K, Sreekumaran N, Lavanya R. Association of obesity and insulin resistance with Dyslipidemia in Indian women with polycystic ovarian Syndrome. Indian J Med Sci. 2006;60:447-53.

Cite this article as: Sreenivasulu K, Kiranmayi VS, Prasad NR, Bifta ARR, Sachan A. Association of triglycerides/high density lipoprotein cholesterol ratio with insulin resistance in polycystic ovary syndrome. Int J Res Med Sci 2018;6:4028-33.