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Research Article

Estimation of HOMA-IR and Triglyceride-Glucose Index in Single Females with PCOS

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ABSTRACT

This study aims to estimate insulin resistance (IR) in single females with polycystic ovarian syndrome. A cross-sectional study was designed to assess the insulin hormone, blood glucose level, IR biomarker, triglyceride-glucose index, serum high-density lipoprotein, and triglyceride. The present included 45 single female participants whom a specialist gynecologist had previously diagnosed. Statistically, Females following a healthy diet showed a non-significant decrease in homeostatic model assessment for IR (HOMA-IR) and a fasting glucose-to-insulin ratio when compared with non-dieters. Regarding the menstrual cycle, the triglyceride-glucose index (a measure of IR) was considerably higher in females with irregular menstrual cycles ($P \leq 0.05$) than in those with regular cycles. A significant positive (direct) correlation was observed between HOMA-IR and the triglyceride-Glucose (TyG) index among single females. This study indicated a strong positive correlation between HOMA-IR and the Triglyceride-Glucose Index among single female patients. In conclusion, a balanced diet is crucial for mitigating IR in this condition. Unusual menstruation was closely linked to poor metabolic profiles, such as increased IR and dyslipidemia.

Keywords: Polycystic ovarian syndrome, homeostatic model assessment for insulin resistance, triglyceride-glucose index, and insulin resistance

INTRODUCTION

Polycystic ovary syndrome refers to the combination of endocrine and metabolic problems that affect a large number of fertile women. The primary organ affected is the ovary, and its reduced functioning leads to a negative reproductive outcome. This disorder typically manifests during adolescence. Insulin resistance (IR) is a decrease in the body's capacity to release the peptide hormone insulin, which is secreted by the pancreatic beta cells.[1] Polycystic ovary syndrome is a combination of metabolic and endocrine disorders among fertile women.[2] The ovary is the main organ affected, and its compromised functioning results in an unfavorable reproductive outcome, usually arising during puberty.[2,3] The cause of polycystic ovarian syndrome (PCOS) is yet unclear, but it is the combination of excessive androgen hormone levels and impaired ovulation.[3] The female diagnosis with PCOS is when at least two of the following: (i) biochemical high levels of androgen hormones, (ii) indication of oligo-anovulation, and (iii) polycystic ovarian morphology by ultrasound.[4] The main signs and symptoms are infertility, pregnancy complications, irregular menstrual cycles, hirsutism, oligomenorrhea, and severe acne.[5]

IR is a physiological disorder characterized by decreased response to high insulin hormone levels by target tissues and is caused by genetic mutations, especially those genes related to intracellular insulin hormone signaling pathways, and environmental factors such as unhealthy diet, physical inactivity, and chronic stress.[6,7] The widely used method for evaluating IR is the homeostatic model assessment for IR (HOMA-IR), which is calculated using fasting insulin and fasting glucose levels.[8]

In PCOS, IR is a major cause of the increased risk of metabolic abnormalities and reproductive dysfunction. Furthermore, it increases the risk of developing Type 2 diabetes mellitus, metabolic syndrome, and cardiovascular

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disease.[9] The complex interplay of environmental and genetic factors contributes to the development of IR in females with PCOS.[10] In polycystic ovary syndrome, hyperandrogenism causes mitochondrial dysfunction in skeletal muscle and increases the level of reactive oxygen species, which raises the risk of progressing IR.[11] In addition to that, being overweight in PCOS patients causes an increase in insulin hormone secretion from the pancreatic beta cells, leading to hyperinsulinemia and then IR.[12]

The main aim of this study is to estimate HOMA-IR and triglyceride-glucose (TyG) index in single females with polycystic ovary syndrome, and to investigate whether PCOS patients are at risk of developing IR.

MATERIALS AND METHODS

A cross-sectional study was designed to assess fasting insulin hormone, fasting blood sugar (FBS), glycated hemoglobin (HbA1c), high-density lipoprotein (HDL), triglycerides (TG), HOMA-IR, TyG index, and Glucose/Insulin ratio in single females with PCOS. The study comprised a purposive sample of 45 single females with PCOS who had previously received a diagnosis of these endocrine disorders from a specialized gynecologist. The inclusion criteria include females aged 20–40 years, diagnosed with PCOS, single marital status, and no prior diagnosis of diabetes mellitus. The exclusion criteria include females aged <20 or >40 years old, not diagnosed with PCOS, married marital status, and having diabetes mellitus or other endocrine disorders. All biochemical parameters, sample processing, and analysis were performed at Floria Laboratory, located in Erbil City.

The participants were required to be fast, and then peripheral blood samples were drawn using conventional phlebotomy techniques. Using a single-use needle, five milliliters of blood samples were extracted and placed into two tubes: An ethylenediaminetetraacetic acid tube to estimate HbA1c and a gel tube to examine biochemical parameters (glucose, TG, HDL, and insulin). Then the serum was separated by centrifugation at 5,000 rpm for 5 min.[13]

The formula used to calculate the HOMA-IR index was HOMA-IR = [glucose] (mmol/L) × [insulin] (μU/mL)/22.5 and HOMA-IR = [glucose] (mg/dL) × [insulin] (μU/mL)/405 (Lewandowski *et al.*, 2019). The fasting glucose-to-insulin ratio (G/I ratio) is a potentially flawed measure of insulin sensitivity and is calculated by dividing the fasting plasma glucose level (mg/dL) by the fasting insulin level (μIU/mL). The TyG index is calculated using serum fasting TG and glucose levels. (TyG index = ln [Fasting TG (mg/dL) × Fasting glucose (mg/dL)/2]).

Data were analyzed with the Statistical Package for the Social Science (Version 27) and GraphPad Prism 10. All selected variables were expressed as means ± standard deviations (SD) in the research study. An independent *t*-test was used for statistical evaluation of quantitative data. The probability value ($P \leq 0.05$) statistically means a significant difference, while a *P*-value more than 0.05 was considered.[14]

RESULT AND DISCUSSION

Demographic Characteristics and Lifestyle

The lifestyle and demographic characteristics of the study participants are shown in Table 1. The study's target population was reflected in the fact that every participant was female and single. PCOS was most frequently seen in early reproductive age in this group, since the majority of patients (95.6%) were between the ages of 20 and 30. This result is in line with earlier research showing that young adult women have a higher prevalence of PCOS.[15] In terms of occupation, the majority of participants (95.6%) were students, while only 4.4% were housewives. The high prevalence of physical inactivity observed in the study was 91.1% of individuals reporting not engaging in regular exercise. IR and metabolic disorders are frequently linked to PCOS. Menstrual irregularity is one of the primary clinical signs of PCOS; therefore, this is consistent with its diagnostic characteristics. About 86.7% of subjects had irregular menstrual periods, indicating a significant prevalence of menstrual irregularity. In addition, 82.2% of the participants reported having poor dietary habits. In females with PCOS, poor diet and inactivity can cause hormonal imbalance, weight gain, and metabolic issues.

IR and Dietary Influence

Table 2 shows the association between dietary habits and metabolic parameters in the study participants. Based on their

Table 1: Demographic characteristics

Variable	Frequency	Percentage
Female	45	100
Marital status		
Single	45	100
Age		
20–30	43	95.6
31–40	2	4.4
Occupation		
Students	43	95.6
Housewife	2	4.4
Smoking		
Yes	2	4.4
No	43	95.6
Exercise		
Yes	4	8.9
No	41	91.1
Cycle		
Irregular	39	86.7
Regular	6	13.3
Diet		
Yes	8	17.8
No	37	82.2
Total	45	100

Table 2: Association of biochemical parameters based on diet

Parameters	Diet		*P*-value
	Good dietary habits No. 8 (Mean±SD)	Bad dietary habits No. 37 (Mean±SD)	
HOMA_IR	3.70±2.15	4.45±5.59	0.537
Glucose/insulin ratio	6.52±3.42	7.01±3.43	0.718
TyG index	4.57±0.16	4.36±0.28	**0.012***
Fasting insulin	16.81±8.65	19.10±18.86	0.604
HbA1C	5.46±0.19	5.24±0.26	**0.029***
Fasting blood sugar	86.62±7.96	90.16±11.85	0.427
HDL cholesterol	40.61±6.25	49.25±10.48	**0.007***
Triglycerides	113.38±37.31	78.65±42.21	**0.039***

*By t-test for two independent samples. Bold values implies statistically significant differences between groups ($P < 0.05$) TyG: Triglyceride-glucose, HbA1C: Glycated hemoglobin, HDL: High-density lipoprotein, SD: Standard deviation

dietary habits, participants were divided into two groups: Those with bad dietary habits (No = 37) and those with good dietary habits (No = 8). Females who followed a diet had a slightly lower HOMA-IR (3.70 ± 2.15) than those who did not (4.45 ± 5.59); however, the difference was not statistically significant ($P = 0.537$). The same result is associated with other studies, which indicate that the diet had a role in improving IR and body composition in females with PCOS due to diet stabilizing blood sugar levels, decreasing insulin hormone overproduction from the pancreatic beta cells, and improving food quality causes fat loss.[8,16] The mean ± SD for the glucose/insulin ratio in the good diet habit group was (6.52 ± 3.42), which is slightly lower compared to the bad diet habits group (7.01 ± 3.43); nonetheless, this difference was also not statistically significant ($P = 0.718$) [Table 2]. Regarding the TyG Index, there was a significant difference between the two groups. The first-line treatment approaches for females with PCOS are lifestyle modification, which includes dietary change and physical activity that regulate the glucose and triglyceride levels.[17] There is a statistically non-significant difference in serum fasting insulin hormone levels ($P = 0.604$) and fasting blood glucose levels ($P = 0.427$) between patients who follow the diet and those who do not. A previous study found that diet programs based on low-carb diets managed blood glucose and insulin levels.[16,18] The diet PCOS group showed significantly higher HbA1C levels (5.46 ± 0.19) than the non-diet PCOS group (5.24 ± 0.26), with a $P = 0.029$, suggesting poorer long-term glucose control among those on a diet. Lipid profiles also showed significant differences. Females with bad diets had lower HDL cholesterol (40.61 ± 6.25 vs. 49.25 ± 10.48; $P = 0.007$) and higher triglyceride levels (113.38 ± 37.31 vs. 78.65 ± 42.21; $P = 0.039$) compared to those with good diets. Opposite results have been obtained from a previous study, which showed that serum levels of HDL and TG are improved by a low-calorie diet.[19]

Menstrual Cycle and Metabolic Parameters

The association between the study participants' normal menstrual cycles and metabolic markers is shown in Table 3. Only a small proportion of participants ($n = 6$) reported regular menstrual periods, whereas the majority of participants ($n = 39$) reported irregular cycles. The PCOS females with irregular cycles show higher HOMA-IR values. This result agrees with a previous study, which has shown that irregular menstrual cycles increase the risk of IR and Type 2 diabetes since an imbalance in female sex hormones affects glucose metabolism, and hyperandrogenism promotes IR by increasing insulin hormone production and secretion.[20] Other biochemical parameters, such as HDL cholesterol, FBS, and HbA1c, did not differ significantly across the groups ($P > 0.05$). The trend indicates higher IR in women with irregular menstrual cycles, which is consistent with the established link between menstrual irregularity and metabolic disturbances in PCOS, even though the glucose/insulin ratio and fasting insulin did not differ significantly between the groups ($P > 0.05$). The previous study shows no significant difference in fasting insulin hormone, fasting blood glucose, and HbA1c levels between irregular and regular menstrual cycles in single females with PCOS.[20] Another study indicated a significant difference in fasting glucose, while there was a non-significant difference in fasting insulin between females with PCOS who have menstrual disorders and females with PCOS who have no menstrual disorders.[21] Regarding HDL, the previous study found that it was significantly higher in PCOS patients with irregular menstrual cycle, which may be related to the estrogen hormone effect on lipid metabolism.[22]

Table 3: Association of biochemical parameters based on the menstrual cycle

Parameters	Menstrual cycle status		*P*-value
	Irregular No. 39 (Mean±SD)	Regular No. 6 (Mean±SD)	
HOMA_IR	4.62±5.42	2.32±1.73	0.051
Glucose/insulin ratio	6.61±3.19	8.97±4.29	0.246
TyG index	4.42±0.28	4.28±0.11	0.038*
Fasting insulin	19.62±18.38	12.61±7.32	0.113
HbA1C	5.31±0.24	5.11±0.31	0.186
Fasting blood sugar	89.66±11.33	88.72±11.79	0.427
HDL cholesterol	48.35±10.50	43.58±9.05	0.278
Triglycerides	88.66±44.92	59.88±13.10	0.003*

*By t-test for two independent samples. TyG: Triglyceride-glucose, HbA1C: Glycated hemoglobin, HDL: High-density lipoprotein, SD: Standard deviation

This study did not show any significant difference in the glucose/insulin ratio according to menstrual cycle status among single females with PCOS [Table 3]. The recent study indicated there was no significant difference between PCOS patients, with anovulatory and ovulatory, in important IR biomarkers that may be related to age, sample size, and lifestyle.[22] The triglyceride-glucose index is a biomarker used to estimate IR; the present findings showed a significant differences between females with PCOS based on menstrual cycle regularity the mean ± SD was (4.42 ± 0.28) and (4.28 ± 0.11) for PCOS females had irregular menstrual cycles and regular menstrual cycles, respectively due to PCOS patients with irregular menstrual cycles, commonly have high androgen hormone levels, which causes increased visceral

adiposity, fat redistribution, irregular metabolic process that increase triglyceride level and TyG index. The irregular menstrual cycle groups noted significantly ($P = 0.003$) higher triglyceride concentrations (88.66 ± 44.92) when compared to the regular menstrual cycle groups (59.88 ± 13.10). This result is in agreement with the results obtained by the previous studies.[23,24]

In the present study, a significant positive correlation was shown between the HOMA-IR and the TyG index among single females with PCOS. This investigation indicates that higher HOMA-IR levels are accompanied by elevated TyG index values, demonstrating the use of the TyG index as a stand-in biomarker for evaluating the metabolic status and IR [Figure 1]. These findings are consistent with previous studies that have reported a significant positive correlation between the TyG index and HOMA-IR in patients with PCOS.[9,24]

This clinical study in females with PCOS reports a non-significant positive correlation between HbA1c and HOMA-IR ($P \leq 0.05$) [Figure 2]. The previous authors reported a significant positive relationship between HbA1c and HOMA-IR in females with PCOS.[25] In females with PCOS, IR leads to chronic mild hyperglycemia that increases HbA1c.

This research showed that among single women with PCOS, there was a strong positive relationship between HOMA-IR and both fasting insulin levels and fasting blood glucose levels [Figures 3 and 4]. Fasting insulin hormone level and fasting glucose level are direct components of the HOMA-IR index; for this reason, they have a strong correlation. IR at the muscle, liver, and adipose tissue levels is common in women with PCOS. The pancreatic beta-cells must oversecrete insulin in order to overcome this resistance and preserve normal blood glucose levels. Fasting hyperinsulinemia (high fasting insulin levels) is the result of this. This finding is in line with a previous study.[26]

In the sample of single females with PCOS in this study, the TyG index illustrated a positive, statistically significant

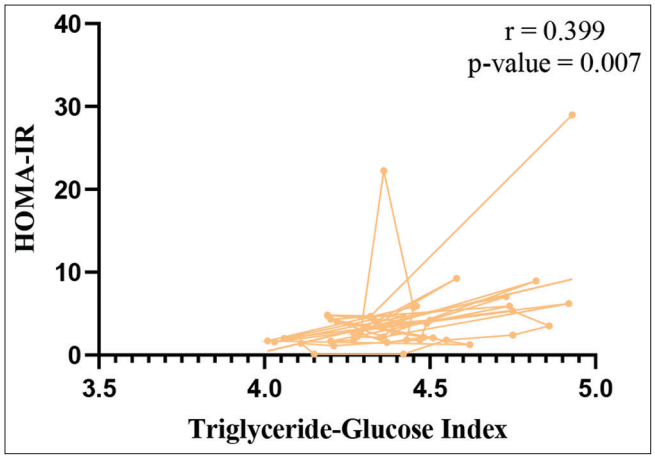

Figure 1: The positive (direct) significant correlation coefficient between homeostatic model assessment for insulin resistance and triglyceride-glucose index among single females with polycystic ovarian syndrome

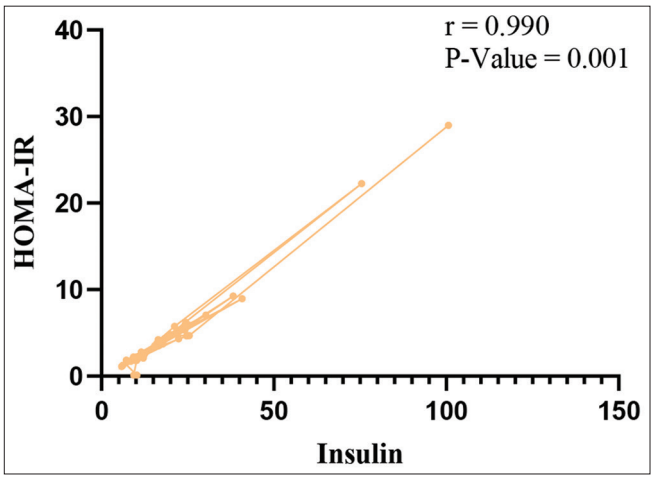

Figure 3: The positive (direct) significant correlation coefficient between homeostatic model assessment for insulin resistance and fasting insulin hormone level among single females with polycystic ovarian syndrome

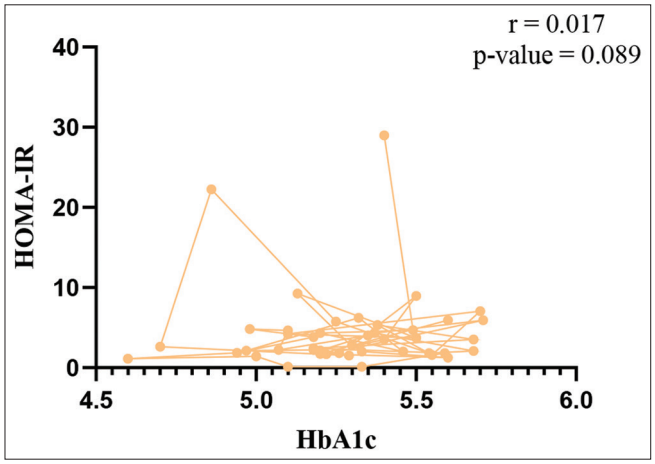

Figure 2: The positive (direct) statistically non-significant correlation coefficient between glycated hemoglobin and homeostatic model assessment for insulin resistance among single females with polycystic ovarian syndrome

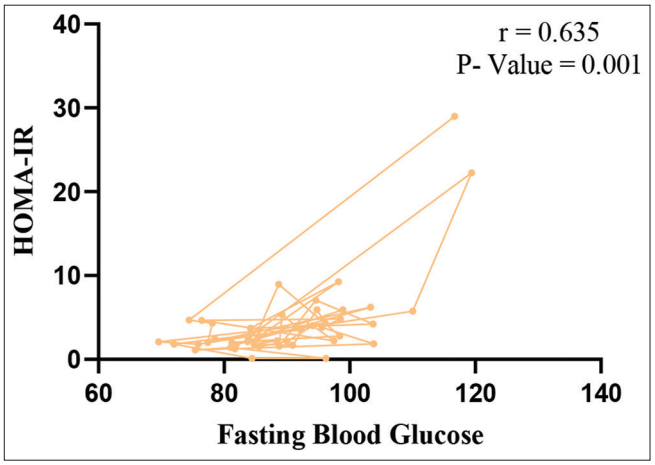

Figure 4: The positive (direct) significant correlation coefficient between homeostatic model assessment for insulin resistance and fasting blood glucose level among single females with polycystic ovarian syndrome

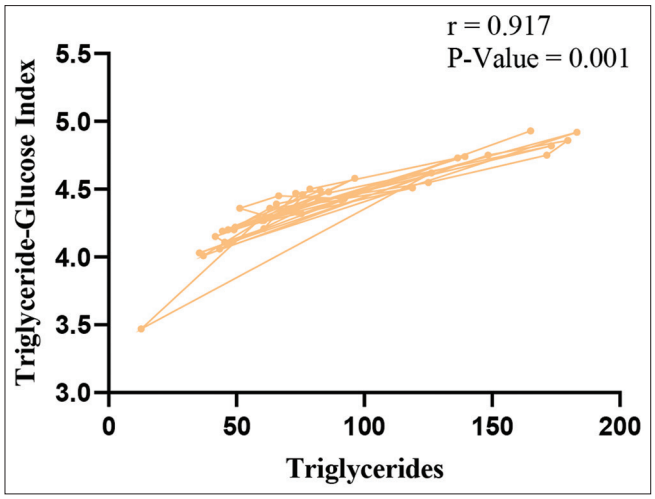

Figure 5: The positive (direct) significant correlation coefficient between the triglyceride-glucose index and fasting triglycerides level among single females with polycystic ovarian syndrome

correlation with fasting triglyceride level (TG) [Figure 5]. Normally, the insulin hormone suppresses the transport of free fatty acids from adipose tissue to the bloodstream; this suppression is affected by IR that causes the accumulation of fat in the liver and muscles. This finding is supported by recent studies.[27,28]

CONCLUSION

This research study estimated that strong interaction between metabolic health, diet, and menstrual cycle regularity in females with PCOS, and also demonstrated that irregular menstrual cycles cause IR, increase triglyceride levels, and poor glycemic control, and also investigated a strong positive correlation between HOMA-IR and the TyG index in this population. This finding concludes both the metabolic implications of menstrual health and lifestyle modifications and is essential for the management of PCOS.

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