doi: 10.48095/cccg2025457

Status of a lipid accumulation product, triglycerides/glucose index and visceral adiposity index on cardiometabolic risk factors in young polycystic ovary syndrome patients

Vliv produktů akumulace lipidů, triglyceridového/glukózového indexu a viscerálního adipozitního indexu na kardiometabolické rizikové faktory u mladých pacientek se syndromem polycystických ovarií

G. Arslan[1], G. Karakelleoğlu[1], C. Karadağ[2]

[1]Department of Obstetrics and Gynecology, Okan University Hospital, Istanbul, Turkey
[2]Department of Obstetrics and Gynecology, Medicana Hospital, Istanbul, Turkey

Summary: Objective: To determine whether the triglycerides/glucose index (TyG-index), lipid accumulation product (LAP), and visceral adiposity index (VAI) would differ in young polycystic ovary syndrome (PCOS) patients when compared to the non-PCOS group, and to investigate the relationship between these markers and cardiovascular disease (CVD) risk in PCOS patients. **Materials and methods:** One hundred and ninety two women with PCOS, and 148 age and body mass index (BMI) matched healthy women without PCOS were enrolled. Levels of serum androgens, sex hormones, lipids, fasting glucose, fasting insulin, and N-terminal pro-brain natriuretic peptide (Nt-probnp) were determined. The 75 g oral glucose tolerance test was performed. The Homeostatic Model Assessment-Insulin Resistance Index (HOMA–IR) and Matsuda insulin sensitivity index were calculated. Levels of TyG-index, LAP, and VAI were determined. **Results:** TyG, LAP, and VAI were significantly higher in PCOS patients than in the control group (P = 0.001 vs. P = 0.001 vs. P = 0.001, resp.). HOMA-IR was significantly higher in PCOS patients and Matsuda ISI was significantly lower in PCOS patients (P = 0.001 vs. P = 0.001, resp.). Levels of Nt-probnp were significantly higher in PCOS patients (P = 0.001). Serum total testosterone and androstenedione levels had significant correlations with TyG, LAP, and VAI. Nt-probnp was significantly correlated with TyG, LAP, and VAI. LDL was positively correlated and HDL was negatively correlated with TyG-index, LAP, and VAI. **Conclusion:** PCOS patients have increased values of TyG-index, LAP, and VAI. TyG index, LAP, and VAI may indicate an increased risk of CVD and hyperandrogenism in PCOS patients.

Key words: polycystic ovary syndrome – triglycerides/glucose index – lipid accumulation product – visceral adiposity index – cardiovascular disease

Souhrn: Cíl: Zjistit, zda se index triglyceridů/glukózy (TyG-index – triglycerides), produkt akumulace lipidů (LAP – lipid accumulation product) a index viscerálního tuku (VAI – visceral adiposity index) liší u mladých pacientek se syndromem polycystických ovarií (PCOS – polycystic ovary syndrome) ve srovnání se skupinou bez PCOS, a zkoumat vztah mezi těmito markery a rizikem kardiovaskulárních onemocnění (CVO – cardiovascular disease) u pacientek s PCOS. **Materiály a metody:** Do studie bylo zahrnuto 192 žen s PCOS a 148 zdravých žen bez PCOS s daným věkem a indexem tělesné hmotnosti (BMI). Byly stanoveny sérové androgeny, pohlavní hormony, lipidy, glukóza nalačno, inzulin nalačno a hladiny N-terminálního mozkového natriuretického peptidu (Nt-probnp). Byl proveden perorální glukózový toleranční test se 75 g glukózy. Byl vypočítán index homeostatického modelu – inzulinová rezistence (HOMA-IR) a Matsudův index citlivosti na inzulin. Byly stanoveny hladiny TyG-index, LAP a VAI. **Výsledky:** TyG, LAP a VAI byly u pacientek s PCOS signifikantně vyšší než v kontrolní skupině (p = 0,001 vs. p = 0,001 vs. p = 0,001). HOMA-IR byl signifikantně vyšší u pacientek s PCOS a Matsuda ISI byl signifikantně nižší u pacientek s PCOS (p = 0,001 vs. p = 0,001). Hladiny Nt-probnp byly u pacientek s PCOS signifikantně vyšší (p = 0,001). Hladiny celkového testosteronu a androstendionu v séru signifikantně korelovaly s TyG, LAP a VAI. Nt-probnp signifikantně koreloval s TyG, LAP a VAI. LDL pozitivně a HDL negativně koreloval s TyG-indexem, LAP a VAI. **Závěr:** Pacientky s PCOS mají zvýšené hodnoty TyG-indexu, LAP a VAI. TyG index, LAP a VAI mohou naznačovat zvýšené riziko kardiovaskulárních onemocnění a hyperandrogenizmu u pacientek s PCOS.

Klíčová slova: syndrom polycystických ovarií – triglyceridový/glukózový index – produkt akumulace lipidů – index viscerální tukové tkáně – kardiovaskulární onemocnění

Introduction

Polycystic ovary syndrome (PCOS) is a comman endocrinological disorder in women of reproductive age, affecting 5–20% of women worldwide [1]. The main characteristics of PCOS are oligo or anovulation, clinical or biochemical hyperandrogenism, and detection of polycystic ovaries via ultrasonography. Increased insulin resistance is a prevalent finding for patients with PCOS [2]. Obesity is also a common feature, approximately affecting 50% of PCOS patients, generally presented in women with upper body obesity which has been related to menstrual disorder [3]. Upper body obesity is associated high insulin levels, increased insulin resistance and metabolic abnormalities [4].

The triglycerides/glucose index (TyG--index) is a simple marker that can determine insulin resistance by evaluating fasting serum glucose and triglyceride levels in the blood steam [5]. The TyG-index has commonly been used as a surrogate insulin resistance marker [5,6]. Recent studies have suggested this marker as an indicator of cardiometabolic disorders and metabolic syndrome (MetS) [7]. Studies have also shown the association between TyG-index and increased risk of cardiovascular disease (CVD) [8]. Women with PCOS have an increased risk for CVD which is correlated with insulin resistance [9]. Thus the TyG-index may be an effective marker for identifying CVD risk women with PCOS.

The lipid accumulation product (LAP) is an index that can report central fat accumulation and is recommended for determining the risk of insulin resistance, metabolic syndrome, and CVD [10]. Visceral adiposity index (VAI) is also a new anthropometric marker that can determine the grade of insulin resistance and risk of cardiometabolic disorders [11]. Increased VAI levels indicate lower insulin sensitivity, higher adipocytokine production, dyslipidemia, diabetes, and hypertension [12]. Previous studies reported an association between VAI and an increased risk of CVD and hyperandrogenemia in PCOS patients [13].

The aim of this study was to determine whether the TyG index, LAP, and VAI would differ in young PCOS patients when compared to non-PCOS group, and to investigate the relation between these markers and CVD risk in PCOS patients.

Materials and methods

Study population

Our study consisted of 192 women with PCOS and 148 age and body mass index (BMI) matched subjects without PCOS. The study was conducted at the Marmara University School of Medicine Hospital from April 2017 to February 2019. The ethics committee of Marmara University approved this study (approval number: 09.2016.121), and the study was designed accordance with the ethical standards described in an appropriate version of the 1975 Declaration of Helsinki, as revised in 2000. Written informed consent was taken from all participants.

The 2003 Rotterdam criteria was performed for PCOS diagnosis [14]. Patients who have at least two of the following three criteria were diagnosed as PCOS:

1) oligoovulation or anovulation: presence of oligomenorrhea or amenorrhea;
2) clinical or biochemical hyperandrogenism: a hirsutism score of 8 or more according to the modified Ferriman-Gallwey (mFG). acne, alopecia, oily skin or serum total testosterone level higher than 0.68 ng/mL;
3) polycystic ovaries: presence of 12 or more follicles (2–9 mm) or higher than 10 mL of each ovarian volume.

Participants with virilizing tumors (ovarian or adrenal), high levels of prolactin, Cushing's syndrome, or congenital adrenal hyperplasia were excluded from the study. Patients with hypo- or hyperthyroidism, hypo- or hyperparathyroidism, or diabetes mellitus were also excluded. PCOS patients who had received hormonal treatment were not included in the study. The control group was composed of 126 healthy women with normal regular menstrual cycles (24–35 days) and without features of hyperandrogenism.

Measurements and laboratory analysis

Waist circumference (WC) and hip circumference (HC) of participants were measured. The measurement be made at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest for WC. HC measurement was taken around the widest portion of the buttocks. To calculate the waist/hip ratio (WHR) WC divided by HC. BMI (kg/m^2) of participants were determined. Systolic (SBP) and diastolic blood pressure (DBP) values of all participants were also noted.

Blood samples were taken from all participants at the 3rd, 4th or 5th days of their menstrual cycle after fasting for 8 hours. To determine insulin sensitivity and insulin resistance of patients 75 g oral glucose tolerance test was performed and all participants gave blood samples at 0, 30, 60, 90, and 120 minutes. The Homeostatic Model Assessment–Insulin Resistance Index (HOMA–IR) was calculated as fasting glucose (mmol/L) × fasting insulin (mU/L)/22.5 to determine insulin resistance. The Matsuda insulin sensitivity index (Matsuda-ISI) was used according to this formula: 10,000/ √ (fasting glucose × fasting insulin) × (mean glucose × mean insulin during OGTT) [15].

The UniCel D×I 800 Access Immunoassay System (Beckman Coulter Inc., Brea, CA) was used to determine the levels of serum thyroid stimulating hormone (TSH), follicle-stimulating hormone (FSH), ACTH, cortisol, total testosterone (T), sex hormone-binding globulin (SHBG), and dehydroepiandrosterone sulfate (DHEAS). The electrochemiluminescence immunoassay (ECLIA) (Elecsys 1010/2010/modular Analytics E170 (Elecsys module), Roche Diagnostic Gmbh,

Mannheim/Germany) method was used to evaluate the levels of luteinizing hormone (LH), insulin (Ins), prolactin (Prl), and 17b-estradiol (E2). An enzymatic UV test (hexokinase method, AU5800, Beckman Coulter Inc., Brea, CA)was used to determine serum glucose levels. Serum androstenedione levels were measured using a chemiluminescent assay (Immulite 2000, Siemens Healthcare Diagnostics, Marburg, Germany). RIA (Schering, Berlin, Germany) was used to determine serum 17 hydroxyprogesterone (17OHPG). N-terminal pro-brain natriuretic peptide (Nt-probnp) levels were measured by ECLIA on an Elecsys 2010 analyser (Roche, Heidelberg, Germany). The levels of serum total cholesterol (T. chol), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and TG were calculated by the Roche Modular P chemistry analyzer (Roche Diagnostics Inc., Mannheim/Germany)

The TyG index was calculated as :
TyG = Ln [fasting TG (mg/dL) × fasting glucose (mg/dL)/2] [16].
LAP was calculated as:
LAP = (WC [cm] – 58) × TG (mmol/L) [17].
VAI was calculated using the following formula:
VAI = [WC/(36.58 + (1.89 × BMI))] × × (TG/0.81) × (1.52/HDL-C) [12].

Statistical analysis

All statistical analyses were performed by SPSS version 20.0 (SPSS, Chicago, IL, USA). Student T-test was used to compare the numeric data with the parametric distribution. For data with non-parametric distribution Mann-Whitney U-test was used. Spearman and Pearson tests were used for correlation analyses where appropiate. All P-values < 0.05 were considered statistically significant. Data were shown as the mean ± standard deviation.

Results

The demographic data and laboratory results of the participants are shown in Tab. 1. The WC and W/H ratio of the PCOS group were significantly higher than the control group (P = 0.009 vs. P = 0.012, resp.). Moreover, the SBP and DBP of the PCOS patients were significantly higher than the control group (P = 0.001 vs. P = 0.022, resp.).

Tab. 1. Demographic parameters and laboratory results of the groups.
Tab. 1. Demografické parametry a laboratorní výsledky skupin.

Group	PCOS (N = 192)	Controls (N = 148)	P
age (years)	28.26 ± 4.94	29.12 ± 5.03	0.204
BMI (kg/m^2)	26.04 ± 3.02	26.66 ± 3.74	0.368
weight (kg)	69.27 ± 14.72	67.48 ± 13.22	0.166
WC (cm)	88.14 ± 12.45	79.13 ± 9.72	0.009
W/H	0.81 ± 0.07	0.76 ± 0.05	0.012
mFG score	16.05 ± 4.72	4.41 ± 1.02	0.001
SBP (mmHg)	122.18 ± 7.72	109.11 ± 7.03	0.001
DBP (mmHg)	76.43 ± 5.02	69.18 ± 4.32	0.022
LH (mIU/mL)	9.92 ± 4.23	5.98 ± 2.37	0.001
FSH (mIU/mL)	5.23 ± 2.01	6.47 ± 1.92	0.021
E2 (pg/mL)	28.83 ± 11.45	33.11 ± 14.21	0.078
androstenodione (ng/mL)	3.88 ± 1.36	2.64 ± 0.75	0.001
T (ng/mL)	0.74 ± 0.23	0.51 ± 0.11	0.001
DHEAS (µg/dL)	292.72 ± 49.60	273.14 ± 66.47	0.062
SHBG (nmoL/L)	33.48 ± 13.66	56.31 ± 19.54	0.001
Prl (ng/mL)	20.59 ± 4.83	17.55 ± 6.42	0.096
TSH (µIU/mL)	2.18 ± 0.63	2.16 ± 0.45	0.346
LDL (mg/dL)	112.63 ± 28.42	102.76 ± 25.34	0.011
T.chol (mg/dL)	184.46 ± 40.42	160.51 ± 32.61	0.001
TG (mg/dL)	116.68 ± 54.60	82.31 ± 33.02	0.001
HDL (mg/dL)	49.82 ± 15.23	61.58 ± 20.04	0.001
Nt-probnp (pg/mL)	62.82 ± 25.22	46.22 ± 18.46	0.001
fasting glucose (mg/dL)	93.46 ± 12.61	84.44 ± 9.96	0.001
fasting Ins (µU/mL)	15.22 ± 7.56	10.02 ± 4.99	0.001
HOMA-IR	3.14 ± 1.93	2.07 ± 1.25	0.001
matsuda ISI	4.97 ± 2.38	7.72 ± 3.96	0.001
TyG	5.45 ± 0.48	3.47 ± 0.19	0.001
LAP	29.15 ± 18.48	15.45 ± 9.48	0.001
VAI	2.25 ± 1.34	1.52 ± 0.79	0.001

BMI – body mass index, DPB – diastolic blood pressure, DHEAS – dehydroepiandrosterone sulfate, E2 – 17b-estradiol, FSH – follicle stimulating hormone, HOMA-IR – Homeostatic Model Assessment-Insulin Resistance index, Ins – insuline, LA – lipid accumulation product, LH – luteinizing hormone, matsuda ISI – insulin sensitivity index, mFG – modified Ferriman Gallwey, Ntprobnp – N-terminal fragment of brain natriuretic protein, PCOS – polycystic ovary syndrome, Prl – prolaktin, SBP – systolic blood pressure, SHBG – sex hormone binding globulin, T – total testosterone, T. chol – total cholesterole, TSH – thyroid stimulating hormone, TyG – triglycerides/glucose index, VAI – visceral adiposity index, WC – waist circumference, W/H – waist/ hip ratio

T and androstenedione levels in PCOS patients were higher than in the control group (P = 0.001 vs. P = 0.001, resp. Levels of SHBG were found significantly lower for PCOS patients than controls (P = 0.001).

T. chol, LDL, and TG levels were significantly higher in PCOS patients than

Tab. 2. Correlation between cardiovascular risk factors, and serum lipids and androgens for PCOS patients.
Tab. 2. Korelace mezi kardiovaskulárními rizikovými faktory a hladinami lipidů a androgenů v séru u pacientek s PCOS.

	TyG	LAP	VAI	HOMA-IR	Matsuda ISI
BMI	R: 0.511**	R: 0.567**	R: 0.612**	R: 0.423**	R: 0.504**
WC	R: 0.587**	R: 0.628**	R: 0.682**	R: 0.523**	R: 0.594**
T	R: 0.456**	R: 0.544*	R: 0.612**	R: 0.304*	R: –0.472**
androstenedione	R: 0.516**	R: 0.556**	R: 0.542**	R: 0.285*	R: –0.402**
Nt-probnp	R: 0.756**	R: 0.844**	R: 0.868**	R: 0.524**	R: –0.653**
SBP	R: 0.306*	R: 0.378*	R: 0.456**	R: 0.094	R: –0.075
DBP	R: 0.112	R: 0.098	R: 0.297*	R: 0.103	R: –0.092
T. chol	R: 0.674**	R: 0.892**	R: 0.886**	R: 0.312*	R: –0.543**
LDL	R: 0.572**	R: 0.756**	R: 0.887**	R: 0.156	R: –0.167
HDL	R: –0.432**	R: –0.316*	R: –0.556**	R: –0.156	R: 0.202
TyG		R: 0.642**	R: 0.789**	R: 0.351**	R: –0.475**
LAP	R: 0.642**		R: 0.871**	R: 0.242*	R: –0.338**
VAI	R: 0.789**	R: 0.871**		R: 0.234*	R: –0.414**
HOMA-IR	R: 0.351**	R: 0.442**	R: 0.434**		R: –0.654**
matsuda ISI	R: –0.475**	R: –0.338**	R: –0.414**	R: –0.654**	

**P < 0.01, *P < 0.05
BMI – body mass index, DPB – diastolic blood pressure, HOMA-IR – Homeostatic Model Assessment-Insulin Resistance index, LAP – lipid accumulation produc, matsuda ISI – insulin sensitivity index, Ntprobnp – N-terminal fragment of brain natriuretic protein, PCOS – polycystic ovary syndrome, SBP – systolic blood pressure, T – total testosterone, T. chol – total cholesterole, TyG – triglycerides/glucose index, VAI – visceral adiposity index, WC – waist circumference

in the control group (P = 0.001 vs. P = 0.011 vs. P = 0.001, resp.). HDL levels in the PCOS patients were significantly lower than the control group (P = 0.001). Nt-probnp is a high predictive marker for CVD, especially in patients without overt CVD [18], and the levels of Nt-probnp were significantly higher in PCOS patients (P = 0.001).

HOMA-IR was significantly higher in PCOS patients and the Matsuda ISI was significantly lower in PCOS patients (P = 0.001 vs. P = 0.001, resp.). TyG, LAP, and VAI were all found to be significantly higher in PCOS patients than in the control group (P = 0.001 vs. P = 0.001 vs. P = 0.001, resp.).

The correlations between the parameters are shown in Tab. 2. There were moderate positive correlations between serum androgens (T and androstenedione) and TyG, LAP, and VAI. The R-value of these correlations was higher than the r value of the correlations between HOMA-IR and androgens. NT-probnp was significantly correlated with TyG, LAP, and VAI, separately. Similarly, the R value of the correlations between Nt-probnp and TyG, LAP, and VAI was higher than the r value of the correlation between Nt-probnp and HOMA-IR. There was a negative significant correlation between Nt-probnp and Matsuda ISI. The association between SBP levels and VAI, TyG and LAP were minimal in a positive way. While LDL and HDL showed moderate to good positive correlations with TyG-index, LAP, and VAI, there was very weak correlation between LDL and HDL and HOMA-IR or Matsuda ISI.

Discussion

In this study, we investigated the association of TyG, LAP, and VAI with cardiometabolic and hormonal parameters for PCOS patients. We found PCOS patients had higher TyG, LAP, and VAI values. These markers positively correlated with serum lipid levels (T. chol, LDL), and were negatively correlated with HDL. TyG, LAP, and VAI were also positively correlated with NT-probnp and SBP. Serum androgens (T and androstenedione) were found to be positively correlated with TyG, LAP, and VAI. We also found a strong correlation between serum androgens and the tested markers when compared to the correlations between serum androgens and HOMI-IR or Matsuda ISI.

Many studies have demonstrated the relation between PCOS and CVD [19]. The increased risk of CVD is commonly related with elevated insulin resistance, dyslipidemia, and hypertensive disorders in PCOS patients [20]. Recent evidence has suggested that the TyG-index could be a surrogate marker for insulin resistance , and metabolic disorders [7]. Lambrinoudaki et al. investigated the rassociation between TyG index and cardiometabolic disorders, and reported that TyG can be a new marker for CVD [21]. In our study, we found PCOS patients had increased TyG values. The TyG-index showed a weak correlation

with SBP levels. Hyperandrogenism is considered to be another factor that may increase CVD risk for PCOS patients [22]. Moreover, the TyG-index was also moderately correlated with serum androgen levels in our study. This increased TyG values could be a sign of an increased CVD risk or MetS risk in PCOS patients.

LAP is another marker that can indicate an increased risk of insulin resistance, MetS, and cardiometabolic disorders [10]. Macut et al. [23] evaluated 222 PCOS patients to investigate the association between LAP and metabolic syndrome. They reported that LAP was an independent clinical marker for MetS and CVD risk in PCOS patients, with a cut-off value of 25.9. Similarly, Wiltgen et al. [24] found an increased LAP value in PCOS patients and they determined that PCOS patients with a LAP > 34.5 had an increased risk of CVD. Additionally, Abruzzese et al. [25] reported LAP as an effective marker for indicating the risk of metabolic disturbances and CVD in PCOS patients. In our study, LAP value of PCOS patients were higher. There were moderate to good associations between LAP and Nt-probnp, androgens and SBP, which are related to the increased CVD risk. Our results demonstrate LAP could be an effective marker for identifying about the risk of CVD for PCOS patients.

TGs and fatty acids are elevated by visceral fat as a results of increased insulin resistance and hyperandrogenism [13]. Visceral adiposity is also related with an increased risk of CVD, menstrual disorders, anovulation, and other metabolic disorders in PCOS patients [26,27]. BMI is not a sufficient measurement to assess CVD risk or other metabolic disorders [28]. WC is a more accurate risk predictor of CVD, metabolic and hormonal disorders, and also metabolic syndromes, which are the main components of VAI [12]. VAI is an indicator of fat distribution and its function is related to cardiometabolism and CVD [12]. It was shown that VAI was superior in indicating CVD risk when compared to WC, WHR, and BMI [13]. Moreover Amato et al. found VAI values are higher in PCOS patients with oligomenorrhea [29]. Likewise Oh et al. reported that VAI positively correlated with visceral fat area, insulin resistance, SBP, and DBP, and negatively correlated with insulin sensitivity [30]. In our study, we found higher VAI levels in PCOS patients. VAI moderately correlated with NT-probnp, SBP, DBP, T. chol, and LDL, and but the correlation was weak and negative with HDL and Matsuda ISI. These may be associated with an increased risk of CVD in PCOS patients. Hyperandrogenism also can contribute to CVD risk in PCOS patients [22]. VAI is also linked with hyperandrogenism. In our study, we found a positive and moderate correlation between VAI and serum androgen levels.

Study limitations

There are some limitations to our study. Firstly, PCOS is a complex disorder with many phenotypes. We did not categorize the study group according to these phenotypes. Secondly we only included women in their reproductive period. Further investigation of the association between the TyG index, LAP, and VAI and older PCOS women might show different results than ours. Third, since the long-term clinical features to establish the effectiveness of these markers were not an objective our study, we did not follow those women for a longer period.

Conclusion

PCOS patients have increased values of TyG-index, LAP, and VAI. These markers were positively correlated with androgens, dyslipidemia, SBP, and Nt-probnp levels. TyG index, LAP, and VAI may indicate an increased risk of CVD and hyperandrogenism in PCOS patients. These could be beneficial markers for identifying cardiometabolic disorders in women with PCOS. Nevertheless further long-term studies should be conducted in order to determine the clinical consequences in PCOS patients later in their lives.

References

1. Azziz R, Carmina E, Chen Z et al. Polycystic ovary syndrome. Nat Rev Dis Primers 2016; 2: 16057. doi: 10.1038/nrdp.2016.57.
2. Dunaif A, Book CB. Insulin resistance in the polycystic ovary syndrome. Clinical research in diabetes and obesity. Springer 1997: 249–274.
3. Liou TH, Yang JH, Hsieh CH et al. Clinical and biochemical presentations of polycystic ovary syndrome among obese and nonobese women. Fertil Steril 2009; 92(6): 1960–1965. doi: 10.1016/j.fertnstert.2008.09.003.
4. Lord J, Thomas R, Fox B et al. The central issue? Visceral fat mass is a good marker of insulin resistance and metabolic disturbance in women with polycystic ovary syndrome. BJOG 2006; 113(10): 1203–1209. doi: 10.1111/j.1471-0528.2006.00973.x.
5. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab 2010; 95(9): 3347–3351. doi: 10.1210/jc.2010-0288.
6. Abbasi F, Reaven GM. Comparison of two methods using plasma triglyceride concentration as a surrogate estimate of insulin action in nondiabetic subjects: triglycerides× glucose versus triglyceride/high-density lipoprotein cholesterol. Metabolism 2011; 60(12): 1673–1676. doi: 10.1016/j.metabol.2011.04.006.
7. Khan SH, Sobia F, Niazi NK et al. Metabolic clustering of risk factors: evaluation of Triglyceride-glucose index (TyG index) for evaluation of insulin resistance. Diabetol Metab Syndr 2018; 10: 74. doi: 10.1186/s13098-018-0376-8.
8. Sánchez-Íñigo L, Navarro-González D, Fernández-Montero A et al. The TyG index may predict the development of cardiovascular events. Eur J Clin Invest 2016; 46(2): 189–197. doi: 10.1111/eci.12583.
9. Cristello F, Cela V, Artini PG et al. Therapeutic strategies for ovulation induction in infertile women with polycystic ovary syndrome. Gynecol Endocrinol 2005; 21(6): 340–352. doi: 10.1080/09513590500463360.
10. Mirmiran P, Bahadoran Z, Azizi F. Lipid accumulation product is associated with insulin resistance, lipid peroxidation, and systemic inflammation in type 2 diabetic patients. Endocrinol Metab (Seoul) 2014; 29(4): 443–449. doi: 10.3803/EnM.2014.29.4.443.
11. Brończyk-Puzoń A, Jagielski P, Kulik-Kupka K et al. Usefulness of a new anthropometric indicator-VAI (Visceral Adiposity Index) in the evaluation of metabolic and hormonal disorders in women with polycystic ovary syndrome. Adv Clin Exp Med 2017; 26(5): 825–828. doi: 10.17219/acem/61100.
12. Amato MC, Giordano C, Galia M et al. Visceral adiposity index: a reliable indicator of visceral fat function associated with cardiometa-

bolic risk. Diabetes Care 2010; 33(4): 920–922. doi: 10.2337/dc09-1825.
13. Zheng SH, Li XL. Visceral adiposity index as a predictor of clinical severity and therapeutic outcome of PCOS. Gynecol Endocrinol 2016; 32(3): 177–183. doi: 10.3109/09513590.2015.1111327.
14. ESHRE TR, Group A-SPCW. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004; 81(1): 19–25. doi: 10.1016/j.fertnstert.2003.10.004.
15. Matsuda M, DeFronzo RA. Insulin sensitivity indices obtained from oral glucose tolerance testing: comparison with the euglycemic insulin clamp. Diabetes Care 1999; 22(9): 1462–1470. doi: 10.2337/diacare.22.9.1462.
16. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord 2008; 6(4): 299–304. doi: 10.1089/met.2008.0034.
17. Onat A, Avci GŞ, Barlan M et al. Measures of abdominal obesity assessed for visceral adiposity and relation to coronary risk. Int J Obes Relat Metab Disord 2004; 28(8): 1018–1025. doi: 10.1038/sj.ijo.0802695.
18. Olsen MH, Wachtell K, Tuxen C et al. N-terminal pro-brain natriuretic peptide predicts cardiovascular events in patients with hypertension and left ventricular hypertrophy: a LIFE study. J Hypertens 2004; 22(8): 1597–1604. doi: 10.1097/01.hjh.0000125451.28861.2a.
19. Wild RA, Carmina E, Diamanti-Kandarakis E et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. J Clin Endocrinol Metab 2010; 95(5): 2038–2049. doi: 10.1210/jc.2009-2724.
20. Fauser BC, Tarlatzis BC, Rebar RW et al. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertil Steril 2012; 97(1): 28.e25–38.e25. doi: 10.1016/j.fertnstert.2011.09.024.
21. Lambrinoudaki I, Kazani MV, Armeni E et al. The TyG index as a marker of subclinical atherosclerosis and arterial stiffness in lean and overweight postmenopausal women. Heart Lung Circ 2018; 27(6): 716–724. doi: 10.1016/j.hlc.2017.05.142.
22. Jones H, Sprung VS, Pugh CJ et al. Polycystic ovary syndrome with hyperandrogenism is characterized by an increased risk of hepatic steatosis compared to nonhyperandrogenic PCOS phenotypes and healthy controls, independent of obesity and insulin resistance. J Clin Endocrinol Metab 2012; 97(10): 3709–3716. doi: 10.1210/jc.2012-1382.
23. Macut D, Antić IB, Bjekić-Macut J et al. Lipid accumulation product is associated with metabolic syndrome in women with polycystic ovary syndrome. Hormones (Athens) 2016; 15(1): 35–44. doi: 10.14310/horm.2002.1592.
24. Wiltgen D, Benedetto I, Mastella L et al. Lipid accumulation product index: a reliable marker of cardiovascular risk in polycystic ovary syndrome. Hum Reprod 2009; 24(7): 1726–1731. doi: 10.1093/humrep/dep072.
25. Abruzzese GA, Cerrrone GE, Gamez JM et al. Lipid accumulation product (LAP) and visceral adiposity index (VAI) as markers of insulin resistance and metabolic associated disturbances in young argentine women with polycystic ovary syndrome. Horm Metab Res 2017; 49(1): 23–29. doi: 10.1055/s-0042-113463.
26. Cascella T, Palomba S, De Sio I et al. Visceral fat is associated with cardiovascular risk in women with polycystic ovary syndrome. Hum Reprod 2008; 23(1): 153–159. doi: 10.1093/humrep/dem356.
27. Androulakis II, Kandaraki E, Christakou C et al. Visceral adiposity index (VAI) is related to the severity of anovulation and other clinical features in women with polycystic ovary syndrome. Clin Endocrinol (Oxf) 2014; 81(3): 426–431. doi: 10.1111/cen.12447.
28. Franzosi MG. Should we continue to use BMI as a cardiovascular risk factor? Lancet 2006; 368(9536): 624–625. doi: 10.1016/S0140-6736(06)69222-2.
29. Amato MC, Verghi M, Galluzzo A et al. The oligomenorrhoic phenotypes of polycystic ovary syndrome are characterized by a high visceral adiposity index: a likely condition of cardiometabolic risk. Hum Reprod 2011; 26(6): 1486–1494. doi: 10.1093/humrep/der088.
30. Oh JY, Sung YA, Lee HJ. The visceral adiposity index as a predictor of insulin resistance in young women with polycystic ovary syndrome. Obesity (Silver Spring) 2013; 21(8): 1690–1694. doi: 10.1002/oby.20096.

ORCID of author

G. Arslan 0000-0002-3332-3178

Submitted/Doručeno: 7. 8. 2025
Accepted/Přijato: 16. 9. 2025

Gaye Arslan, MD
Department of Obstetrics and Gynecology
Okan Universitesi Hastanesi
İçmeler, Aydınlı Yolu Caddesi. No: 2
34947 Tuzla, Istanbul
Turkey
gaye.arslan@okan.edu.tr

Publication ethics: The Editorial Board declares that the manuscript met the ICMJE uniform requirements for biomedical papers.
Publikační etika: Redakční rada potvrzuje, že rukopis práce splnil ICMJE kritéria pro publikace zasílané do biomedicínských časopisů.
Conflict of interests: The authors declare they have no potential conflicts of interest concerning the drugs, products or services used in the study.
Konflikt zájmů: Autoři deklarují, že v souvislosti s předmětem studie/práce nemají žádný konflikt zájmů.