J Surg Med. 2020;4(8):664-668.
DOI: 10.28982/josam.755729

Research article
Araştırma makalesi

Journal of Surgery and Medicine
e-ISSN=2602-2079

The relation of visceral adiposity index and lipid accumulation product with metabolic, anthropometric, and hormonal parameters in patients with polycystic ovary syndrome

Polikistikover sendromlu hastalarda visseral adiposite indeksi ve lipid birikim ürünlerinin metabolik, antropometrik ve hormonal parametrelerle ilişkisi

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Ethics Committee Approval: The study protocol was approved by the Ethics Committee of Bursa Yüksek Ihtisas Education and Research Hospital (2011-KAEK-25 2019/10-02). All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.
Etik Kurul Onayı: Çalışma protokolü Bursa Yüksek İhtisas Eğitim ve Araştırma Hastanesi Etik Kurulu (2011-KAEK-25 2019/10-02) tarafından onaylandı. İnsan katılımcıların katıldığı çalışmalardaki tüm prosedürler, 1964 Helsinki Deklarasyonu ve daha sonra yapılan değişiklikler uyarınca gerçekleştirilmiştir.

Conflict of Interest: No conflict of interest was declared by the authors.
Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Financial Disclosure: The authors declared that this study has received no financial support.
Finansal Destek: Yazarlar bu çalışma için finansal destek almadıklarını beyan etmişlerdir.

Published: 8/30/2020
Yayın Tarihi: 30.08.2020

Abstract

Aim: Polycystic ovary syndrome (PCOS) is the most common endocrinopathy in women of reproductive age and is associated with glucose intolerance, central obesity, hypertension, and dyslipidemia. The Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP) are effective indices for predicting insulin resistance are associated with cardiovascular and cerebrovascular events. In this study, we aimed to investigate the relationship of VAI and LAP with metabolic, anthropometric, and hormonal parameters in PCOS patients.

Methods: A total of 106 patients with PCOS who were diagnosed according to the Rotterdam criteria and 66 healthy controls without PCOS aged 18-35 years were included in this prospective, case-control study. Patients with diabetes mellitus, Cushing syndrome, hyperprolactinemia, congenital adrenal hyperplasia, hypertension, or thyroid disorder were excluded. The VAI and LAP were calculated based on the high-density lipoprotein cholesterol (HDL-C) and triglyceride (TG) levels.

Results: There was a negative, significant correlation between LAP and HDL-C (r=-0.644), a positive, significant correlation between TG (r=0.706) and hip circumference (r=0.872), and a positive, significant correlation between VAI and waist circumference (r=0.625) in the PCOS group. There was also a positive, significant correlation between HOMA-IR and VAI in the PCOS group (r=0.462).

Conclusion: Our study results suggest that VAI seems to be a more useful index for predicting insulin resistance in PCOS patients.

Keywords: Lipid accumulation product, Polycystic ovary syndrome, Visceral adiposity index

Öz

Amaç: Polikistik over sendromu (PKOS) üreme çağında en sık görülen endokrinopati olup santral obezite, glukoz intoleransı, dislipidemi ve hipertansiyon ile ilişkilidir. Visseral adipozite indeksi (VAI) ve lipid birikim ürünleri (LAP) insülin duyarlılığı dışında kardiyovasküler ve serebrovasküler olaylar ile de ilişkili olduğu saptanmıştır. Bizler de, bu çalışmamızda PKOS ve kontrol grubu hastalarımızda VAI ve LAP ile metabolik, antropometrik ve hormonal parametreler arasındaki ilişkileri değerlendirdik.

Yöntemler: Çalışmaya 18-35 yaş aralığında 106'sı Rotterdam kriterlerine göre PKOS grubunda olmak üzere toplam 172 hasta dahil edildi. Diabetes mellitus, Cushing sendromu, hiperprolaktinemi, konjenital adrenal hiperplazi, hipertansiyon ve tiroid bozukluğu olan hastalar çalışmaya dahil edilmedi. LAP ve VAI indekslerinin hesaplanması için HDL ve trigliserid düzeylerine bakıldı.

Bulgular: PKOS'lu hasta grubunda LAP ile HDL-C (r=-0,644) arasında anlamlı negatif korelasyon saptanırken, trigliserid (r=0,706) ve kalça çevresi (r=0,872) arasında pozitif korelasyon saptandı. Yine VAI ile bel çevresi (r=0,625) ve HOMA-IR (r=0,462) arasında pozitif korelasyon saptandı.

Sonuç: Bu çalışmada PKOS hasta grubunda VAI'nın insülin direncini belirlemede yararlı bir indeks olduğu sonucuna varıldı.

Anahtar kelimeler: Polikistik over sendromu, Visseral adipozite indeksi, Lipid birikim ürünleri

How to cite/Atıf için: Aydın GA, Özgen G. The relation of visceral adiposity index and lipid accumulation product with metabolic, anthropometric, and hormonal parameters in patients with polycystic ovary syndrome. J Surg Med. 2020;4(8):664-668.

Introduction

Polycystic ovary syndrome (PCOS) is a common hormonal disorder in women of reproductive age characterized by ovulatory dysfunction, chronic anovulation, irregular menstruation, and clinic and/or biochemical hyperandrogenism [1]. A higher rate of women with PCOS suffer from insulin resistance, type 2 diabetes mellitus (DM), dyslipidemia, hypertension, and central obesity than those with regular menstruation. Insulin resistance is the main underlying cause of the pathophysiology of these metabolic disorders [2]. About 70% of women with PCOS have insulin resistance, while metabolic syndrome affects about 8 to 25% of these women with anovulatory PCOS phenotype [3,4].

Several studies have demonstrated that metabolic disorders are more frequently associated with the distribution of adipose tissue rather than absolute amount of the body fat [5]. Visceral obesity has been associated with increased insulin resistance, low-grade chronic inflammation, type 2 DM, dyslipidemia, and metabolic and cardiovascular diseases [6-8]. In about 50 to 60% of insulin-resistant PCOS patients, central adipose tissue distribution is present, irrespective of obesity [9].

Ultrasonography (USG), computed tomography (CT), and magnetic resonance imaging (MRI) are helpful imaging modalities for the assessment of visceral adiposity distribution. However, radiation exposure and excessive costs of these imaging modalities limit their use in daily clinical practice [10]. In previous studies, waist circumference (WC) and waist-to-hip ratio (WHR) were calculated for the assessment of visceral adipose tissue [11]. In more recent studies, the Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP), which are effective indices for predicting insulin resistance, were used and shown to be useful for the evaluation of cardiometabolic risk-related adipose tissue dysfunction [12,13].

To date, several studies have used VAI for visceral adiposity dysfunction in patients with metabolic syndrome, type 2 DM, and PCOS [12,14-18]. In the present study, we aimed to investigate the relationship between VAI and LAP with metabolic, anthropometric, and hormonal parameters in PCOS patients.

Materials and methods

This prospective, case-control study was conducted at the Obstetrics and Gynecology outpatient clinics between January 2019 and July 2019. The patient group included a total of 106 patients diagnosed with PCOS according to the Rotterdam criteria [19]. The control group included 66 healthy individuals without PCOS. Those with DM, Cushing syndrome, hyperprolactinemia, thyroid disorder, hypertension, or congenital adrenal hyperplasia, and patients who received oral contraceptives, anti-androgens, insulin-sensitizing agents, or statins within the past six months were excluded. A written informed consent was obtained from each participant. The protocol of the study, which was conducted in accordance with the principles of the Declaration of Helsinki, was approved by the Ethics Committee (2011-KAEK-25 2019/10-02).

Blood samples were collected after a 12-hour overnight fast between 08.00 and 10.00 AM on Days 2 and 5 of the menstrual cycle. Laboratory tests including follicle-stimulating hormone (FSH), estradiol (E_2), total testosterone, prolactin (PRL), luteinizing hormone (LH), dehydroepiandrosterone sulfate (DHEA-S), insulin, and 17-hydroxyprogesterone (17-OHP) were analyzed (ARCHITECT®, Abbott Laboratories, Singapore). Fasting blood glucose, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), HDL-C, and TG were measured using the Synchron LX20 analyzer (Beckman Coulter, CA, USA).

Insulin resistance was calculated using the Homeostatic Model Assessment Insulin Resistance (HOMA-IR) formula (fasting glucose (mg/dL) x fasting insulin (μU/mL)/405). LAP was calculated using the following formula: (WC [cm]-58) x (TG [mmol/L]) [13]. VAI was calculated using the following formula: (WC in cm/36.58 + [1.89 x BMI in kg/m2] x [TG/0.81] x [1.52/HDL-C mmol/L]) [12].

Statistical analysis

Statistical analysis was performed using the SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Descriptive data were expressed in mean (standard deviation), median (IQR) values, and number and frequency. The Kolmogorov-Smirnov test was used to check the normality assumption. The Mann-Whitney U test was performed to compare unadjusted age and BMI variables between the patient and control groups. The analysis of covariance (ANCOVA) was used to compare adjusted age and BMI variables between the patient and control groups. The Spearman's rank correlation analysis was utilized to investigate the relationship between VAI and LAP and other variables. A *P*-value of <0.05 was considered statistically significant.

Results

Of a total of 172 participants included in the study, 106 were in the PCOS group and 66 were in the control group. Descriptive demographic and clinical characteristics of the patient and control groups (unadjusted) are shown in Table 1. The mean BMI, Ferriman-Gallwey Hirsutism scores, WC, HC, WHR, LH, total testosterone, 17-OHP, insulin, fasting blood glucose, TC, and LAP ($P<0.05$ for all) were significantly higher in the PCOS group compared to the control group.

Due to a significant difference in age and BMI values between the groups, these variables were included in the model as covariates. Descriptive demographic and clinical characteristics of the patient and control groups (adjusted) are shown in Table 2. Accordingly, there was no significant difference in the VAI and LAP between the groups. However, the mean WHR ($P=0.049$), LH ($P=0.005$), 17-OHP ($P=0.001$), TC ($P=0.049$), and HOMA-IR ($P=0.049$) were significantly higher in the PCOS group compared to the control group.

Correlation analysis results are summarized in Table 3. There was a significant difference in WC between the groups ($P=0.020$), indicating a positive, significant correlation of WC with VAI in the PCOS group(r=.625). However, there was a significant difference in the HC ($P=0.022$), HDL-C ($P=0.006$), and TG levels ($P=0.016$) between the groups, indicating a positive, significant correlation of HC(r=.872), HDL-C(r=-.644), TG(r=.706) with LAP in the PCOS group. Furthermore, there was a positive, significant correlation between the HOMA-IR

(r=0.462) and VAI in the PCOS group. There was a similar correlation between HOMA-IR and LAP measurements in both groups.

Table 1: Descriptive demographic and clinical characteristics of the patient and control groups (unadjusted)

	Control group n=66		PCOS group n=106		
Variable	Mean	SD	Mean	SD	*P*-value
Age (years)	27.67	4.68	24.90	4.95	0.001
BMI (kg/m^2)	23.69	3.87	26.44	5.65	0.001
FGH score	11.13	4.56	16.29	6.52	0.001
Waist circumference (cm)	74.29	12.37	81.05	15.51	0.007
Hip circumference (cm)	99.52	8.70	103.67	12.44	0.016
Waist-to-hip ratio (cm)	0.74	0.07	0.78	0.07	0.003
TSH (µU/ml)	2.48	1.15	2.14	1.29	0.019
PRL (ng/mL)	15.79	8.45	14.65	7.78	0.461
FSH (mIU/mL)	5.93	1.88	5.21	2.35	0.002
LH (mIU/mL)	5.02	3.02	7.40	6.30	0.005
Estradiol (pg/mL)	76.16	56.58	71.68	6476	0.248
Total testosterone , (ng/dL)	16.25	16.13	21.39	19.98	0.026
DHEA-S (µg/dL)	202.97	81.29	228.52	96.84	0.067
17-OHP (ng/mL)	0.52	0.49	0.94	0.84	0.001
Insulin (µIU/mL)	9.54	13.08	10.65	6.78	0.004
HOMA-IR	1.612	.916	2.430	1.742	0.001
FBG (mg/dL)	85.35	13.04	89.56	9.20	0.023
HDL-C (mg/dL)	54.26	12.72	53.49	11.64	0.788
LDL-C (mg/dL)	97.94	28.99	108.29	29.22	0.052
TC (mg/dL)	167.83	30.47	181.71	35.10	0.015
TG (mg/dL)	90.05	49.88	97.15	56.81	0.297
VAI	3.20	2.57	3.58	2.73	0.242
LAP	16.67	21.03	25.10	24.89	0.010

PCOS: polycystic ovary syndrome, TSH: thyroid-stimulating hormone, PRL: prolactin, FSH: follicle-stimulating hormone, LH: luteinizing hormone, DHEA-S: dehydroepiandrosterone sulfate, 17-OHP: 17-hydroxyprogesterone, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, FBG: fasting blood glucose, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, TC: total cholesterol, TG: triglyceride, VAI: Visceral Adiposity Index, LAP: Lipid Accumulation Product

Table 2: Descriptive demographic and clinical characteristics of the patient and control groups (adjusted)

Variable	Control group n=66		PCOS group n=106		*P*-value
	Mean	SD	Mean	SD	
Waist circumference (cm)	77.715	8.798	78.914	8.597	0.403
Hip circumference (cm)	102.831	5.825	101.605	5.693	0.197
Waist-to-hip ratio (cm)	0.751	0.065	0.771	0.062	0.049
TSH (µU/ml)	2.608	1.300	2.058	1.277	0.010
PRL (ng/mL)	15.930	8.603	14.569	8.412	0.331
FSH (mIU/mL)	5.905	2.332	5.221	2.275	0.073
LH (mIU/mL)	4.904	5.549	7.475	5.426	0.005
Estradiol (pg/mL)	64.405	62.011	78.992	60.621	0.149
Total testosterone (ng/dL)	16.450	18.466	21.271	17.997	0.109
DHEA-S (µg/dL)	213.536	94.255	221.940	92.146	0.584
17-OHP (ng/mL)	0.462	0.772	0.975	0.752	0.001
Insulin (µIU/mL)	9.857	10.082	10.450	9.863	0.718
HOMA-IR	86.728	10.919	88.697	10.677	0.268
FBG (mg/dL)	1.843	1.44	2.287	1.76	0.049
HDL-C (mg/dL)	53.636	11.431	53.877	11.181	0.897
LDL-C (mg/dL)	98.566	30.741	107.902	30.053	0.063
TC (mg/dL)	169.374	35.388	180.749	34.604	0.049
TG (mg/dL)	95.726	55.463	93.614	54.227	0.815
VAI	3.506	2.608	3.386	2.553	0.777
LAP	21.959	18.271	21.803	17.863	0.958

Table 3: Correlation analysis results

		r	*P*-value	r	*P*-value
Control n=66	Age (years)	0.397	0.001	0.227	0.067
	BMI (kg/m^2)	0.221	0.074	0.625	0.001
	FGH score	-0.143	0.253	-0.448	0.001
	Waist circumference (cm)	0.344	0.005	0.911	0.001
	Hip circumference (cm)	0.221	0.075	0.752	0.001
	Waist-to-hip ratio (cm)	0.432	0.001	0.828	0.001
	TSH (µU/ml)	0.006	0.959	-0.269	0.029
	PRL (ng/mL)	0.197	0.113	0.362	0.003
	FSH (mIU/mL)	-0.004	0.974	0.004	0.976
	LH (mIU/mL)	-0.078	0.534	-0.100	0.426
	Estradiol (pg/mL)	-0.168	0.177	-0.429	0.001
	Total testosterone (ng/dL)	-0.169	0.179	-0.515	0.001
	DHEA-S (µg/dL)	-0.017	0.891	-0.047	0.708
	17-OHP (ng/mL)	-0.142	0.257	-0.364	0.003
	Insulin (µIU/mL)	0.268	0.030	0.386	0.001
	FBG (mg/dL)	0.077	0.538	0.348	0.004
	HDL-C (mg/dL)	-0.657	0.001	-0.318	0.009
	LDL-C (mg/dL)	0.243	0.049	0.035	0.779
	TC (mg/dL)	0.231	0.062	0.150	0.229
	TG (mg/dL)	0.844	0.001	0.455	<0.001
	HOMA-IR	0.242	0.051	0.435	<0.001
PCOS n=106	Age (years)	0.145	0.139	0.304	0.002
	BMI (kg/m^2)	0.503	0.001	0.771	0.001
	FGH score	0.010	0.919	0.102	0.300
	Waist circumference (cm)	0.625	0.001	0.905	0.001
	Hip circumference (cm)	0.617	0.001	0.872	0.001
	Waist-to-hip ratio (cm)	0.519	0.001	0.771	0.001
	TSH (µU/ml)	0.092	0.348	0.064	0.517
	PRL (ng/mL)	-0.007	0.944	0.047	0.634
	FSH (mIU/mL)	-0.010	0.921	-0.057	0.565
	LH (mIU/mL)	-0.115	0.241	-0.219	0.024
	Estradiol (pg/mL)	-0.391	0.001	-0.508	0.001
	Total testosterone (ng/dL)	-0.336	0.001	-0.488	0.001
	DHEA-S (µg/dL)	0.127	0.196	0.166	0.090
	17-OHP (ng/mL)	-0.068	0.486	0.033	0.740
	Insulin (µIU/mL)	0.463	0.001	0.525	0.001
	FBG (mg/dL)	0.251	0.009	0.306	0.001
	HDL-C (mg/dL)	-0.730	0.001	-0.644	0.001
	LDL-C (mg/dL)	0.250	0.010	0.194	0.047
	TC (mg/dL)	0.230	0.018	0.207	0.033
	TG (mg/dL)	0.909	0.001	0.706	0.001
	HOMA-IR	0.462	<0.001	0.530	<0.001

Discussion

Polycystic ovary syndrome is typically characterized by chronic anovulation, hyperandrogenism, and morphological polycystic ovaries. Irrespective of insulin resistance and obesity, increased central adipose tissue is one of the main clinical manifestations of PCOS, suggesting that PCOS is a metabolic disorder [20,21]. Even with a normal body weight, women with PCOS have usually increased visceral adipose tissue [20,21]. Visceral obesity is associated with increased adipocytokine production, proinflammatory activity, and insulin resistance and hypertension, high TG and low HDL-C levels, atherosclerosis and high mortality rates [12,22,23]. Therefore, it is of utmost importance to assess visceral adiposity in PCOS patients to diagnose and treat cardiovascular diseases, irrespective of the obesity status.

The International Diabetes Federation recommends CT or MRI for the evaluation of visceral adiposity distribution [24]. Although both imaging modalities yield reliable results, radiation exposure and excessive costs limit their use in daily clinical practice. Previous studies have also established the role of anthropometric measurements in predicting insulin resistance, metabolic syndrome, and cardiovascular risks [15,25]. Recent studies have reported that VAI and LAP are more useful tools to estimate visceral obesity and visceral adiposity functionality [12,13]. VAI and LAP seem to be helpful in identifying cardiometabolic risk in many settings including PCOS [26,27]. Review of the literature reveals several studies investigating the relationship of VAI and LAP with cardiometabolic risk factors in women with PCOS. Agrawal et al. [28] examined the possible relationship between VAI and cardiometabolic risk factors in

patients with PCOS phenotypes classified according to the Rotterdam criteria and in healthy controls. They showed higher mean VAI values in the PCOS group. Although the patient and control groups were BMI-matched, higher VAI values were attributed to the increased WC and impaired lipid functions in PCOS patients. The authors concluded that VAI showed a strong correlation with cardiometabolic risk in PCOS patients and was, therefore, a useful index. The precise cut-off reference value of VAI for assessing cardiometabolic risk in this patient group should be identified in future studies.

In another study, which evaluated the performance of adiposity indices according to phenotypes of PCOS for the first time, Mario et al. [26] examined different adiposity indices to predict preclinical metabolic alterations and cardiovascular risk in PCOS patients with classic and ovulatory phenotype and healthy controls. They observed that LAP with a cut-off value of ≥34 and VAI with a cut-off value of ≥1.32 were the best markers for classic and ovulatory PCOS, respectively. Also, both indices were more useful than other indices for screening insulin resistance in PCOS. Similarly, in the present study, we examined the relation of VAI and LAP with metabolic, anthropometric, and hormonal parameters. Our study results showed a significant correlation between LAP and HDL-C, TG, and HC and between VAI and WC in the PCOS group.

It has been well established that insulin resistance plays a key role in the formation of reproductive and metabolic alterations in PCOS patients; however, it is often challenging to diagnose [14]. In a study using hyperinsulinemic-euglycemic clamp to assess insulin sensitivity in PCOS patients, Oh et al. [14] reported that VAI with a cut-off value of 1.79 could replace visceral CT scanning as a marker for visceral adiposity defined as a visceral fat area of >100 cm^2 on CT. In another study, VAI was strongly correlated with insulin resistance, indicating an independent correlation with cardiovascular and cerebrovascular events in the general population [12]. Similarly, in our study, we observed a significant correlation between HOMA-IR and VAI in the PCOS group.

On the other hand, there are controversial reports in the literature suggesting a stronger correlation between the insulin resistance, WC and BMI than those with VAI and LAP indices. Borruel et al. [11] compared several surrogate indices of visceral adiposity with USG and determined that WC and BMI showed the strongest correlations with USG measurements of visceral adiposity, while the WHR and VAI showed a weaker, but statistically significant correlation with USG measurements of the thickness of visceral adipose tissue depots. In addition, WC and BMI showed a stronger correlation with insulin resistance than VAI. In another study, Huang et al. [9] assessed insulin resistance in Chinese women with PCOS according to the body fat indices and reported that VAI and LAP were reliable indicators of insulin resistance in the normal weight group, while LAP, BMI, and waist-to-height ratio were more sensitive in overweight/obese group. Similarly, Ramezani et al. [15] compared the validity of available indicators in PCOS patients who participated in a large population-based study and found both VAI and LAP indices to be the best indicators for predicting insulin resistance; however, WC and VAI were the best predictors for predicting metabolic syndrome.

Limitations

Nonetheless, there are some limitations to this study. First, we used the HOMA-IR for screening insulin resistance. The hyperinsulinemic-euglycemic clamp is the gold standard for the evaluation of insulin sensitivity; however, HOMA-IR shows a strong correlation with this method and is helpful in identifying cardiovascular risk [29,30]. Second, we were unable to examine the performance of these indices according to PCOS phenotypes. However, previous studies have demonstrated that cardiovascular risk factors are less frequent in patients with normoandrogenic patients than hyperandrogenic ones [31,32].

Conclusions

Our study results showed a significant correlation between the HOMA-IR and VAI in the PCOS group than the control group. These findings suggest that VAI seems to be a more useful index for predicting insulin resistance in PCOS patients. However, further large-scale studies are needed to draw a definitive conclusion.

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