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Is Your Diet to Blame? Unveiling the Role of Dietary Inflammatory Index in Lipid Accumulation Product Index and Visceral Adiposity Index in Polycystic Ovary Syndrome

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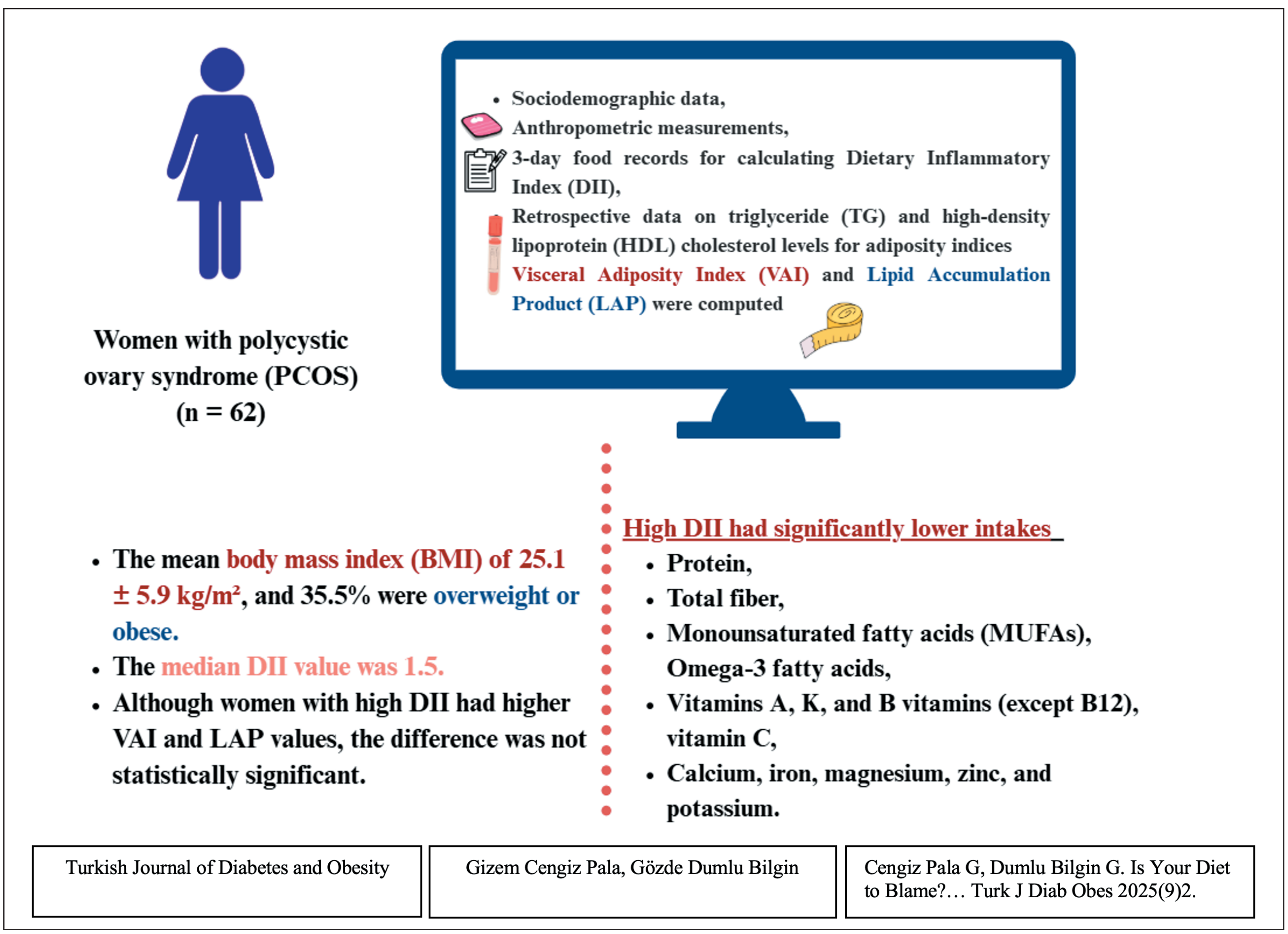
GRAPHICAL ABSTRACT



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ABSTRACT

Aim: The present study sought to examine how the Dietary Inflammatory Index (DII) is related to different adiposity indices in women diagnosed with polycystic ovary syndrome (PCOS).

Material and Methods: The study enrolled sixty-two women aged 18 - 44 years who applied to a PCOS outpatient clinic in Istanbul. Sociodemographic data, anthropometric measurements, 3-day food records for calculating DII, and retrospective data on high-density lipoprotein (HDL) cholesterol levels and triglyceride (TG) for adiposity indices were collected. Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP) were computed based on the obtained anthropometric data and specified blood parameters. Since the data were divided into low and high DII groups, the Mann-Whitney U test was employed based on the statistical distribution to evaluate group differences between continuous variables.

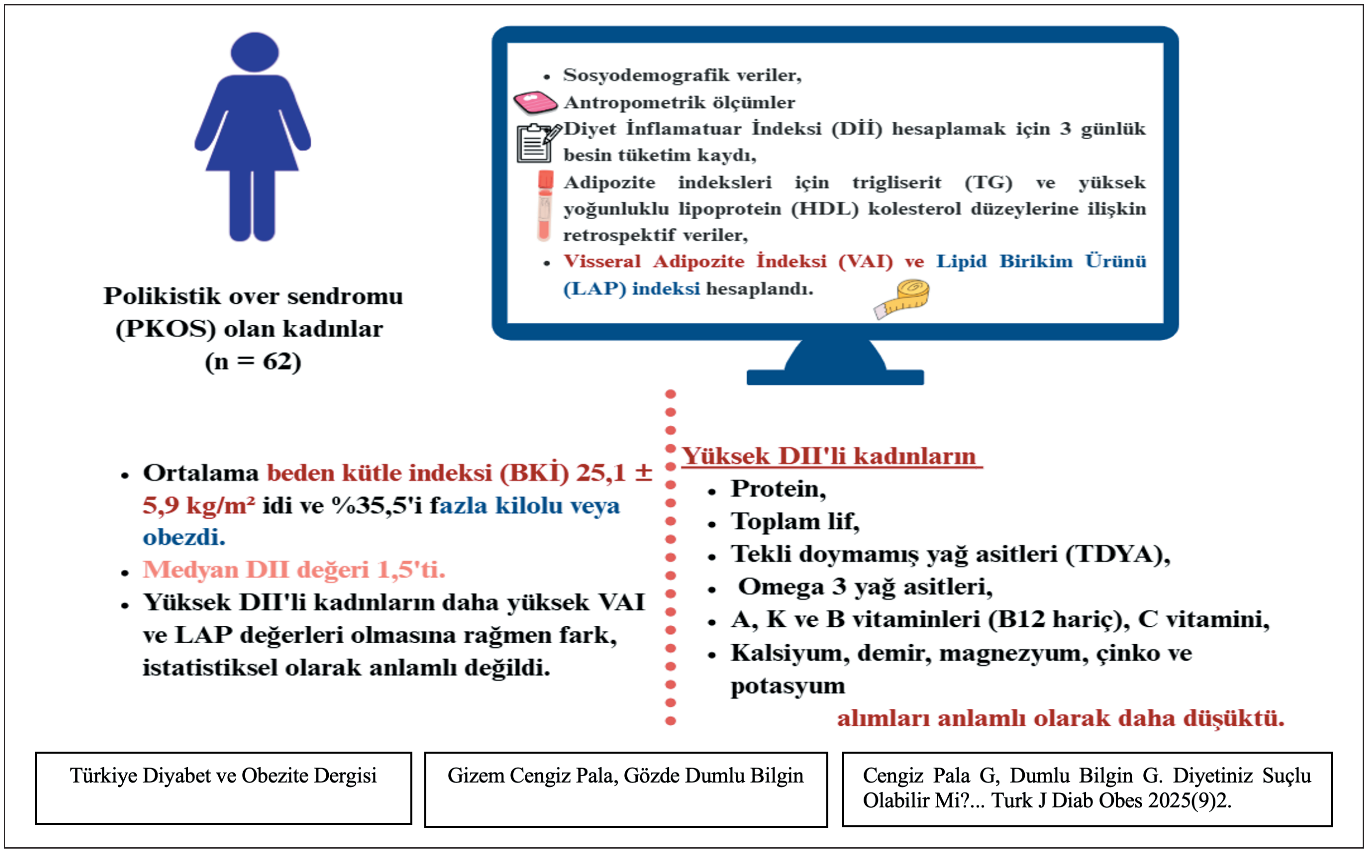
Results: The mean age of women was 26.3 ± 6.1 years, with a body mass index (BMI) of 25.1 ± 5.9 kg/m^2, and 35.5% were overweight or obese. The median DII value was 1.5. Although women with high DII had higher VAI and LAP values, the difference was not statistically significant. Additionally, women with high DII had significantly reduced dietary intakes of protein, total fiber, omega-3 fatty acids, monounsaturated fatty acids (MUFAs), vitamins A, K, and B vitamins (except B12), vitamin C, iron, calcium, zinc, potassium, and magnesium.

Conclusion: These findings suggest that diet-induced inflammation may contribute to metabolic disturbances in PCOS, emphasizing the importance of anti-inflammatory dietary approaches in managing the condition.

Keywords: *Polycystic ovary syndrome, Dietary inflammatory index, Visceral adiposity index, Lipid accumulation product, Adiposity indices, Diet*

Diyetiniz Suçlu Olabilir Mi? Polikistik Over Sendromunda Diyet İnflamatuar İndeksinin, Lipid Birikim Ürünü İndeksi ve Visseral Adipozite İndeksi Üzerindeki Rolünün İncelenmesi

GRAFİKSEL ÖZET



ÖZ

Amaç: Bu çalışma, polikistik over sendromu (PKOS) olan kadınlarda Diyet İnflamatuar İndeksi'nin (Dİİ) çeşitli adipozite indeksleriyle ilişkisini incelemeyi amaçlamıştır.

Gereç ve Yöntemler: Çalışmaya, İstanbul'da bir PKOS polikliniğine başvuran 18 - 44 yaş arası altmış iki kadın dahil edildi. Sosyodemografik veriler, antropometrik ölçümler, Dİİ'yi hesaplamak için 3 günlük besin tüketim kaydı ve adipozite indeksleri için yüksek yoğunluklu lipoprotein (HDL) kolesterol ve trigliserit (TG) düzeylerine ilişkin retrospektif veriler toplandı. Visseral Adipozite İndeksi (VAI) ve Lipid Birikim Ürünü (LAP) indeksi, elde edilen antropometrik verilere ve belirtilen kan parametrelerine dayanarak hesaplandı. Veriler, düşük ve yüksek Dİİ gruplarına ayrıldığından, sürekli değişkenler arasındaki grup farklarını değerlendirmek amacıyla, verilerin dağılımına bağlı olarak Mann-Whitney U testi uygulanmıştır.

Bulgular: Kadınların ortalama yaşı 26,3 ± 6,1 yıl, beden kütle indeksi (BKİ) 25,1 ± 5,9 kg/m² idi ve %35,5'i fazla kilolu veya obezdi. Medyan Dİİ değeri 1,5'ti. Yüksek Dİİ'li kadınların daha yüksek VAI ve LAP değerleri olmasına rağmen fark, istatistiksel olarak anlamlı değildi. Bunun yanı sıra, yüksek Dİİ'li kadınların protein, toplam lif, omega-3 yağ asitleri, tekli doymamış yağ asitleri (TDYA), A, K ve B vitaminleri (B12 hariç), C vitamini, demir, kalsiyum, çinko, potasyum ve magnezyum alımları anlamlı olarak daha düşüktü.

Sonuç: Bu bulgular, diyet kaynaklı inflamasyonun PKOS'ta metabolik bozukluklara katkıda bulunabileceğini düşündürmekte ve hastalığın yönetiminde anti-inflamatuar diyet yaklaşımlarının önemini vurgulamaktadır.

Anahtar Sözcükler: *Polikistik over sendromu, Diyet inflamatuar indeksi, Visseral adipozite indeksi, Lipid birikim ürünü indeksi, Adipozite indeksleri, Diyet*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a multifaceted female endocrine and metabolic disorder characterized by the presence of two of the following three features: oligomenorrhea, hyperandrogenism, and polycystic ovary appearance on ultrasound (1). PCOS affects approximately 10% of women of childbearing age (2) and is metabolically interrelated with several diseases, including insulin resistance, dyslipidemia, hypertension, cardiovascular diseases (CVD), psychiatric disorders, gynecological cancers, being overweight and obesity, type 2 diabetes, and increased infertility (1,3–5).

Approximately 38-88% of women with PCOS worldwide are affected by overweight or obesity (6), and this excess weight contributes to the hyperandrogenism commonly seen in PCOS through various mechanisms, including elevated secretion of androgens from the ovaries, higher levels of adipokines, and increased insulin resistance (7). The body mass index (BMI) is the most frequently employed metric for the assessment of obesity. Nevertheless, its primary function is the evaluation of generalized obesity, and it lacks the capacity to provide insight into the distribution of body fat (8). Therefore, its utility is limited in distinguishing between adipose tissue and lean mass, or in determining the distribution or functional role of distinct fat depots (9). In this regard, two novel indices have been proposed-the lipid accumulation product (LAP) index (10) and the visceral adiposity index (VAI) (11)-which are mathematical models derived from anthropometric parameters and lipid profiles, offering a more nuanced approach to assessing obesity and its associated risks. In clinical practice, these indices are highly sensitive markers in showing visceral adiposity, inflammation, insulin resistance, and cardiometabolic risk (4,7,12). In previous studies, it has been demonstrated that these scores were increased in PCOS patients (12–14). Furthermore, PCOS has been linked to chronic low-grade inflammation, which is theorized to play a critical role in the development of long-term cardiovascular risks, and dietary components also have substantial effects on inflammation (15).

It has been demonstrated that particular dietary constituents can trigger chronic inflammation through the process of oxidative damage. The Western-type diet, characterized by a high consumption of red meat, high-fat dairy products, and refined grains, has been associated with elevated levels of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and fibrinogen (16). Conversely, the Mediterranean diet, marked by a diet abundant in whole grains, fruits, green vegetables, and fish, and with limited consumption of red meat and butter, moderate alcohol intake, and olive oil, has been linked to reduced inflammation levels (17).

The Dietary Inflammatory Index (DII), conceptualized and developed by Shivappa et al., is a quantitative scale that is scored according to the intake of frequently consumed dietary components that are related to critical inflammatory blood markers (18). A number of studies have demonstrated a correlation between elevated DII scores and an augmentation in certain cardiometabolic risk indices (19,20). In a cross-sectional study conducted on individuals with type 2 diabetes, it was observed that high DII scores were associated with elevated triglyceride levels, LAP, and anthropometric measurements (21). Moreover, numerous studies have investigated the inflammatory potential of diet in PCOS patients, revealing that higher DII scores are linked to increased odds of PCOS diagnosis (22,23). Despite the existence of studies that have examined various adiposity indices within this group (9,12,24,25), addressing the relationship between the DII and adiposity indices, including LAP and VAI, in this population remains to be elucidated.

Therefore, the primary objective of this study was to clarify the interaction between the DII and certain adiposity-related measures in women diagnosed with PCOS. Since LAP and VAI are accepted as reliable proxy indicators of metabolic dysfunction and heart disease risk among PCOS patient, investigating their relationship with dietary inflammation may provide deeper insights into the underlying mechanisms linking nutrition, inflammation, and fat distribution in this population. By filling this gap, the study aims to enhance the available literature and promote the creation of more targeted dietary approaches to manage metabolic complications in women with PCOS.

MATERIALS and METHODS

Study Design and Participants

This single-center, cross-sectional study was conducted at the PCOS and Hirsutism Clinic of a University Hospital in Istanbul between April and August 2022 with women aged 18 years and older who were newly diagnosed with PCOS based on the Rotterdam criteria (26). Prior to the initiation of the study, ethical approval was obtained from the Non-Interventional Research Ethics Committee of the Faculty of Health Sciences at Yeditepe University (Approval Date and Number: 08.04.2022/16). Using *G*Power (v3.1.9.7)*, an a priori power analysis was carried out based on a presumed moderate effect size (with Cohen's d value of 0.76), a significance threshold of 0.05, and 90% power. Findings from the analysis indicated that a total of 60 participants would be required. Our study included 62 participants, which is close to this estimate and therefore considered sufficiently powered to detect medium effect sizes.

Participants were not eligible for the study if they were pregnant or lactating, under 18 years old, or had conditions, including cancer, congenital adrenal hyperplasia, Cushing's syndrome, hyperprolactinemia, or any conditions that could affect inflammatory status (e.g., infections, rheumatological diseases, inflammatory bowel diseases). Additionally, those on non-steroidal anti-inflammatory drugs, statins, or antihypertensive therapy, individuals with severe psychiatric disorders or using psychiatric medications, as well as those with endocrine, kidney, or liver diseases, or diseases requiring specific dietary interventions (such as type 1 diabetes or celiac disease) were also excluded.

Anthropometric Measurements

Participants' body weight, height, waist, and hip circumference were taken and evaluated following the World Health Organization (WHO) guideline (27). All measurements in the study were performed and recorded by a dietitian and were repeated twice. The body compositions of women were assessed using a Tanita body analyzer *(Model RD-953, Tanita Corporation, Tokyo, Japan)*, which operates based on the Bioelectrical Impedance Analysis (BIA) method. A rigid tape measure was employed to obtain the required measurements, ensuring that they were perpendicular to the body's long axis and parallel to the ground. Women's height was recorded while they were barefoot, in an upright position, and the Frankfort plane (above the auricle and the lower border of the orbital-eye socket aligned and level with the ground), with the feet side by side using a stadiometer accurate to 0.1 cm *(Seca, Germany)*.

The body mass index (BMI) was determined by applying the following formula: weight (kg)/height (m^2), based on the obtained weight and height data. According to the standards established by WHO, categorization of BMI values was conducted into the following classes: underweight (BMI < 18.5 kg/m^2), normal (18.5-24.99 kg/m^2), overweight (25.0-29.99 kg/m^2), and obese (BMI ≥ 30.0 kg/m^2) (27). The waist-to-hip ratio (WHR) and the waist-to-height ratio (WHtR) were obtained through the measurement of waist circumference (WC), hip circumference (HC), and height.

Biochemical Parameters

The high-density lipoprotein (HDL) cholesterol and triglyceride (TG) levels were retrospectively retrieved from patient records, based on routine measurements performed at the Clinical Biochemistry Laboratory of the University Hospital using standardized automated enzymatic assays performed by experienced laboratory personnel.

Dietary Assessment and Calculation of Dietary Inflammatory Index (DII)

The Dietary Inflammatory Index *(DII)* is a literature-derived dietary scoring approach designed to determine the contribution of diet to an individual's inflammatory status; a high DII score indicates a diet's pro-inflammatory properties, while a low DII score indicates a diet's anti-inflammatory properties (18). According to this index, 45 nutrients (composed of whole foods, macro and micro nutrients, and other biologically active substances) affect six inflammation markers, interleukins (IL) such as IL1β, IL-4, IL-6, IL-10, TNF-α, and CRP (28). In this study, the DII was computed based on 29 components obtained from 3-day dietary records. The intake levels of participants were assessed for energy, protein, carbohydrates, fiber, fat, saturated fatty acids (SFAs), cholesterol, monounsaturated fatty acids (MUFAs), polyunsaturated fatty acids (PUFAs), omega-3 and omega-6 fatty acids, as well as vitamins A, D, E, K, vitamin C, riboflavin, thiamine, niacin, vitamin B6, folic acid, vitamin B12, and minerals including calcium, iron, zinc, magnesium, selenium, sodium, and potassium. Other components of the original DII score (such as anthocyanidins, eugenol, flavones, flavonols, flavan-3-ols, flavanones, isoflavones, and saffron) were excluded from the analysis because data on their daily intake were missing in this dataset.

A three-day food record was obtained to identify the participants' nutritional status and calculate the DII scores. Women were instructed to keep a record of the foods and drinks they consumed for three continuous days, including two weekdays and one weekend day. The instructions on how to fill in the food consumption registration form were explained to the individuals with examples. "Food and Nutrition Photo Catalogue" (29) has been employed to identify the amount of food eaten by individuals at home in a portion, and "Standard Food Recipes" to determine the amount of nutrients in a portion of food consumed outside the home (30).

The food record analysis was conducted using the Computer Assisted Nutrition Program, Nutrition Information System (BeBis 8.2), a software developed specifically for Türkiye. BeBiS is based primarily on the German Food Code and Nutrient Data Base (Bundeslebensmittelschlüssel), and it also includes Turkish foods that have been adapted and validated for use in Turkish populations. The software has been used in multiple studies in Türkiye and has shown acceptable validity and reliability for assessing dietary intake.

The z-score values for women's daily nutrient intake were calculated using the formula: "([individual daily average consumption - standard global daily mean intake] / standard deviation)". These z-scores were then converted into percentile scores. DII, which reflects the inflammatory potential of the person's daily dietary intake, was determined by summing the scores obtained by multiplying the percentile value for each nutrient with the corresponding "specific overall inflammatory effect score" for that nutrient.

Visceral Adiposity Index (VAI)

The VAI is established as an empirically based mathematical equation, differentiated by gender, derived from basic anthropometric [BMI (kg/m^2) and WC (cm)] and metabolic indices [TG (mmol/L) and HDL cholesterol (mmol/L)], that is predictive of adipose tissue distribution and function (11).

The VAI for women is calculated using the following equation:

$$\mathrm{VAI} = \left(\frac{\mathrm{WC}}{36.58 + (1.89 \times \mathrm{BMI})}\right) \times \left(\frac{\mathrm{TG}}{0.81}\right) \times \left(\frac{1.52}{\mathrm{HDL}}\right),$$

Lipid Accumulation Product (LAP) index

The LAP index, first introduced by Kahn et al., is derived from a composite of WC (cm) and fasting TG levels (mmol/L) (10). The TG concentration in mmol/L, which is utilized in the equation, was derived by multiplying their value in mg/dL by 0.0113.

The LAP index for women is calculated using the following equation:

(WC [cm] – 58) x (TG [mmol/L])

Statistical Analysis

The statistical analyses were conducted using SPSS software (Windows version 23). The characteristics of the samples were presented as mean ± standard deviation (SD) and range (minimum-maximum) for continuous variables and as percentages (%) and numbers (n) for qualitative variables. Assessment of normality for continuous variables was conducted using the Shapiro-Wilk test, which indicated a deviation from normal distribution. Hence, non-parametric methods (Mann-Whitney U test) were employed for comparisons between two groups. To provide a more comprehensive representation of data distribution, descriptive statistics were supplemented with medians and interquartile ranges (IQR). The determination of the confidence interval was based on the 95% level of significance in all analyses, and the results were deemed statistically significant at $p < 0.05$.

RESULTS

General Characteristics of the Participants

A total of 62 adult women diagnosed with PCOS were analyzed in this study, with a mean age of 26.3 ± 6.1 years. Of these, 87.1% were single, 90.3% were university graduates or higher, and most were nulliparous (93.5%), and non-smokers (82.3%). More than half (58.1%) of the women in the study were noted to have one or more diagnosed diseases. The average sleep length of women was 7 hours, and the average age at which PCOS was diagnosed was 21.4 years. When individuals were listed based on their diagnosed disease, thyroid disease ranked first with 35.7%, followed by gastrointestinal disease with 23.8%, and migraine with 21.4%. The majority of participants (85.5%) indicated that PCOS had a notable impact on their quality of life, with the typical menstrual cycle duration falling between 21 and 40 days. The most commonly reported symptoms among women with PCOS were hairing (74.2%), acne (72.6%), and fatigue (72.6) (Table 1).

Anthropometric Measures and Adiposity Indices of the Participants

When the anthropometric characteristics of women with PCOS were examined, it was revealed that the mean BMI was 25.1 ± 5.9 kg/m^{2}, and 35.5% were overweight or obese. In addition, the average WC, WHR, WHtR, and fat mass of women are determined to be 76.2 cm, 0.76, 0.47, and 30.76% respectively. The mean values of the VAI and LAP in women were 1.19 ± 1.13 and 22.9 ± 33.3, respectively (Table 2). Median values are also presented in this table; accordingly, the median VAI was 0.79 (IQR 0.55 - 1.16) and the median LAP was 10.1 (IQR 5.5 - 24.4). Moreover, adiposity indices showed statistically significant positive correlations with key anthropometric measures, including BMI, waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) (Table 3).

Dietary Inflammatory Index (DII)

In our study, the mean DII value for women with PCOS was 1.37 ± 1.50, with a median value of 1.5 [0.18 - 2.44]. Accordingly, the DII scores were stratified into two categories: low and high, based on their position relative to the median value. All parameters were consequently evaluated following this classification (Table 4). While median VAI and LAP index values were higher in women with high DII, no statistically significant difference was observed.

On the other hand, women in the high DII group had significantly lower median (IQR) values for age [24.0 (22.0 - 26.0) vs. 27.0 (24.0 - 30.0)], protein intake [48.9 (41.5 - 62.9) vs. 63.6 (52.9 - 72.1) g/day], total fiber [12.0 (9.1 - 14.2) vs. 19.5 (15.1 - 20.9) g/day], MUFAs [27.0 (22.0 - 30.3) vs. 31.5 (26.9 - 36.5) g/day], omega-3 fatty acids [1.1 (0.9 - 1.4) vs. 1.8 (1.4 - 2.7) g/day], vitamin A [709.2 (503.1 - 895.5) vs. 1603.7 (1366.0 - 1946.3) µg RE/day], vitamin K [53.9 (31.9 - 74.1) vs. 131.4 (94.7 - 179.2) µg/day], vitamin C [53.5 (32.0 - 72.3) vs. 114.4 (89.2 - 138.6) mg/day], calcium [494.0 (418.4 - 619.0) vs. 607.7 (469.5 - 764.5) mg/day], iron [7.1 (5.9 - 8.2) vs. 10.2 (8.9 - 12.0) mg/day], magnesium [170.0 (149.4 - 206.7) vs. 245.7 (222.0 - 315.2) mg/day], zinc [7.5 (6.5 - 9.3) vs. 9.4 (7.6 - 10.6) mg/day], and potassium [1510.9 (1334.5 - 1776.6) vs. 2412.9 (2097.1 - 2686.9) mg/day] (Table 4).

Table 1: General characteristics of women

Variables	Findings (n = 62)
Age (years ± SD) (min-max)	26.3 ± 6.1 (18 - 44)
Marital status, n (%)	
Single	54 (87.1)
Married	8 (12.9)
Education level, n (%)	
Primary/Middle School	1 (1.6)
High School	5 (8.1)
Undergraduate/Graduate	56 (90.3)
Parity, n (%)	
Nulliparous	58 (93.5)
Primiparous or multiparous	4 (6.5)
Presence of disease, n (%)	
Yes	36 (58.1)
No	26 (41.9)
Diagnosed diseases*, n (%)	
Thyroid diseases	15 (35.7)
Gastrointestinal diseases	10 (23.8)
Migraine	9 (21.4)
Allergy	3 (7.1)
Asthma	2 (4.8)
Skin diseases	2 (4.8)
Depression	1 (2.4)
Smoking status, n (%)	
Non-smoker	51 (82.3)
Current smoker	6 (9.7)
Ex-smoker	5 (8.0)
Sleep duration (hours)	7.1 ± 1.5 (3 - 12)
Age at diagnosis of PCOS	21.4 ± 4.9 (12 - 33)
Symptoms related to PCOS*	
Hairing	46 (74.2)
Menstrual irregularity	37 (59.7)
Obesity	26 (41.9)
Acne	45 (72.6)
Hair loss	36 (58.1)
Fatigue	45 (72.6)
Depression	19 (30.6)
Insulin resistance	23 (37.1)
Does PCOS affect their quality of life?	
Yes	53 (85.5)
No	9 (14.5)
Menstrual cycle	
<21 day	2 (3.2)
21-40 days	48 (77.4)
> 40 days	12 (19.4)

Variables are shown as means ± standard deviations (SD) and range. Categorical variables are expressed as frequency (%)
*Participants ticked more than one option. **PCOS:** Polycystic ovary syndrome, **SD:** Standard deviation

Table 2: Anthropometric measures, Dietary Inflammatory Index *(DII)*, and adiposity indices of participants

Variables*	Findings (n = 62)		
	Mean ± SD	**Median [IQR]**	**min.-max**
Weight (kg)	65.3 ± 15.7	60.5 [54.6 - 70.9]	38.9 - 115.3
BMI (kg/m²)	25.1 ± 5.9	23.0 [21.6 - 27.8]	17.1 - 41.9
BMI classification, n (%) Underweight Normal Overweight and Obese	 3 (4.8) 37 (59.7) 22 (35.5)	-	-
WC (cm)	76.2 ± 13.8	73.5 [67.0 - 81.5]	53 - 115
HC (cm)	99.26 ± 11.34	97.5 [92.0 - 102.3]	81 - 136
WHR	0.76 ± 0.07	0.75 [0.71 - 0.80]	0.65 - 0.94
WHtR	0.47 ± 0.09		0.35 - 0.73
Fat mass (%)	30.76 ± 7.33		16.20 - 48.10
DII	1.37 ± 1.50	1.50 [0.18 - 2.44]	-1.93 - 4.64
VAI	1.19 ± 1.13	0.79 [0.55 - 1.16]	0.39 - 6.79
LAP	22.9 ± 33.3	10.1 [5.5 - 24.4]	-4.18 - 151.99
TG (mg/dL)	87.8 ± 57.2	68.5 [51.8 - 107.8]	35 - 349
HDL cholesterol (mg/dL)	62.9 ± 13.6	62.5 [55.8 - 70.0]	35 - 106

*Data are shown as "Mean ± SD, Median [IQR], minimum-maximum".

BMI: Body mass Index; (Underweight <18.5 kg/m²; Normal: 18.5-24.99 kg/m²; Overweight: BMI 25.0-29.99 kg/m²; Obesity: BMI ≥ 30 kg/m²

Data are shown as mean ± standard deviation, median [interquartile range, IQR], and minimum–maximum. Categorical variables are expressed as frequency (%).

DII: Dietary inflammatory index, **HC:** Hip circumference, **HDL:** High density lipoprotein, **LAP:** Lipid accumulation product, **TG:** Triglyceride, **VAI:** Visceral adiposity index, **WC:** Waist circumference, **WHtR:** Waist-to-height ratio, **WHR:** Waist-to-hip ratio

Table 3: Correlation analysis of Dietary Inflammatory Index *(DII)*, adiposity indices, and anthropometric measures

		1	2	3	4	5	6	7	8
1. DII	r_S	**1**							
	p	-							
2. VAI	r_S	0.059	**1**						
	p	0.651	-						
3. LAP	r_S	0.125	0.878	**1**					
	p	0.334	<0.001**	-					
4. BMI (kg/m²)	r_S	0.152	0.486	0.755	**1**				
	p	0.237	<0.001**	<0.001**	-				
5. WC (cm)	r_S	0.125	0.581	0.816	0.926	**1**			
	p	0.334	<0.001**	<0.001**	<0.001**	-			
6. HC (cm)	r_S	0.172	0.468	0.760	0.913	0.883	**1**		
	p	0.180	<0.001**	<0.001**	<0.001**	<0.001**	-		
7. WHR	r_S	0.007	0.517	0.596	0.623	0.804	0.437	**1**	
	p	0.955	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	-	
8. WHtR	r_S	0.150	0.549	0.778	0.943	0.974	0.841	0.805	**1**
	p	0.245	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	-

BMI: Body mass index, **DII:** Dietary inflammatory index, **HC:** Hip circumference, **LAP:** Lipid accumulation product, $r_{S,}$ Spearman's rank correlation coefficient, **VAI:** Visceral adiposity index, **WC:** Waist circumference, **WHtR:** Waist-to-height ratio, **WHR:** Waist-to-hip ratio

***p* < 0.01

Table 4: Distribution of dietary intakes and adiposity indices based on the categories of DII

Variables	Median-splitted DII		*p-value*
	Low < 1.5 (n = 31)	High ≥1.5 (n = 31)	
Age (years)	27.0 [24.0 - 30.0]	24.0 [22.0 - 26.0]	**0.048***
BMI (kg/m^2)	22.6 [21.1 - 26.8]	23.4 [21.6 - 28.5]	0.468
WC (cm)	72.0 [66.0 - 81.0]	74.0 [68.0 - 85.0]	0.464
HC (cm)	97.0 [92.0 - 102.0]	99.0 [92.0 - 106.0]	0.607
WHR	0.74 [0.71 - 0.80]	0.76 [0.70 - 0.80]	0.882
WHtR	0.43 [0.40 - 0.49]	0.44 [0.41 - 0.52]	0.310
VAI	0.71 [0.54 - 1.15]	0.89 [0.62 - 1.18]	0.495
LAP	10.1 [5.1 - 17.9]	10.2 [5.5 - 31.2]	0.593
Energy intake (kcal/d)	1523.4 [1311.0 - 1863.6]	1505.8 [1326.9 - 1650.4]	0.314
Protein (g/d)	63.6 [52.9 - 72.1]	48.9 [41.5 - 62.9]	**0.006***
Carbohydrate (g/d)	142.4 [108.1 - 184.6]	149.3 [100.4 - 180.3]	0.916
Total fiber (g/d)	19.5 [15.1 - 20.9]	12.0 [9.1 - 14.2]	**<0.001****
Total fat intake (g/d)	80.8 [72.2 - 97.1]	72.0 [65.1 - 86.6]	0.074
Cholesterol (mg/d)	360.1 [254.9 - 440.9]	285.6 [191.7 - 398.3]	0.113
SFAs (g/d)	26.5 [21.9 - 34.2]	27.2 [21.2 - 35.8]	0.961
MUFAs (g/d)	31.5 [26.9 - 36.5]	27.0 [22.0 - 30.3]	**0.003****
PUFAs (g/d)	14.2 [12.1 - 18.4]	13.2 [10.2 - 18.0]	0.269
Omega-3 fatty acid (g/d)	1.8 [1.4 - 2.7]	1.1 [0.9 - 1.4]	**<0.001****
Omega-6 fatty acid (g/d)	12.1 [10.2 - 14.7]	11.7 [8.7 - 16.3]	0.324
Vitamin A (μg RAE/d)	1603.7 [1366.0 - 1946.3]	709.2 [503.1 - 895.5]	**<0.001****
Vitamin D (μg/d)	4.0 [2.1 - 21.9]	3.2 [1.9 - 4.2]	0.069
Vitamin E (mg/d)	13.7 [10.9 - 18.1]	10.9 [7.8 - 17.7]	0.085
Vitamin K (μg/d)	131.4 [94.7 - 179.2]	53.9 [31.9 - 74.1]	**<0.001****
Thiamine (mg/d)	0.8 [0.8 - 1.1]	0.6 [0.5 - 0.7]	**<0.001****
Riboflavin (mg/d)	1.3 [1.0 - 1.8]	1.0 [0.8 - 1.2]	**0.001****
Niacin (mg NE/d)	13.1 [10.2 - 18.8]	9.7 [7.1 - 10.8]	**<0.001****
Folic acid (μg/d)	281.9 [230.4 - 355.0]	181.7 [160.8 - 202.8]	**<0.001****
Vitamin B6 (mg/d)	1.3 [1.0 - 1.7]	0.8 [0.6 - 0.9]	**<0.001****
Vitamin B12 (μg/d)	4.1 [2.7 - 7.1]	3.5 [2.9 - 4.8]	0.435
Vitamin C (mg/d)	114.4 [89.2 - 138.6]	53.5 [32.0 - 72.3]	**<0.001****
Calcium (mg/d)	607.7 [469.5 - 764.5]	494.0 [418.4 - 619.0]	**0.038***
Iron (mg/d)	10.2 [8.9 - 12.0]	7.1 [5.9 - 8.2]	**<0.001****
Magnesium (mg/d)	254.7 [222.0 - 315.2]	170.0 [149.4 - 206.7]	**<0.001****
Zinc (mg/d)	9.4 [7.6 - 10.6]	7.5 [6.5 - 9.3]	**0.019***
Selenium (μg/d)	12.1 [6.9 - 20.0]	9.4 [3.8 - 18.7]	0.243
Sodium (mg/d)	2163.0 [1891.0 - 2957.9]	2113.2 [1687.6 - 2543.9]	0.161
Potassium (mg/d)	2412.9 [2097.1 - 2686.9]	1510.9 [1334.5 - 1776.6]	**<0.001****

p-values were computed by the Mann-Whitney U Test for continuous variables; $^*p < 0.05$ $^{**}p < 0.01$

Data are presented as median [interquartile range, IQR].

DII: Dietary inflammatory index, **HC:** Hip circumference, **HDL:** High density lipoprotein, **LAP:** Lipid accumulation product, **MUFAs:** Mono unsaturated fatty acids, **NE:** Niacin equivalent, **PUFAs:** Polyunsaturated fatty acids, **RAE:** Retinol Activity Equivalents, **SFAs:** Saturated fatty acids, **TG:** Triglyceride, **VAI:** Visceral adiposity index, **WC:** Waist circumference, **WHtR:** Waist-to-height ratio, **WHR:** Waist-to-hip ratio

DISCUSSION

Based on existing literature, this study appears to be the first to elucidate the relationship between diverse adiposity indices and DII in women with PCOS. In this study, VAI and LAP index, known as surrogate markers of adiposity, were employed in women with PCOS. Elevated levels of visceral adiposity are hallmarks of PCOS and trigger a surge in cardiovascular mortality via several mechanisms, including underlying chronic low-grade inflammation, metabolism of sex steroids and cortisol locally in visceral fat, and a release of adipokine secretions, that directly affect the ovary (7,12,13). Several studies involving women with PCOS have demonstrated increased levels of VAI and LAP index compared with the control group (24,31). Although the present study lacked a control group, we observed notably lower mean and median values for both VAI and LAP in the PCOS sample compared to established thresholds in the literature. For instance, in India, women with PCOS exhibited higher values for both VAI and LAP, with reported averages of 9.17 and 63.49, respectively (4). Similarly, a study in Türkiye involving a comparable sample found that women with PCOS had mean values of 3.58 for VAI and 25.10 for LAP (31). Another study conducted in Türkiye with a similar study population reported mean values of 2.22 for VAI and 37.7 for LAP in women with PCOS (32). Notably, an Indian study identified VAI ≥ 2.76 and LAP ≥ 48.06 as optimal thresholds for predicting metabolic syndrome, demonstrating strong diagnostic performance (33). Wilgen et al. reported that a LAP ≥ 34.5 is considered an additional risk factor for cardiovascular disease in patients with PCOS (14). The lower values observed in our sample may reflect differences in demographic characteristics, such as age and BMI, as well as ethnic variations in adiposity distribution. Additionally, the substantial standard deviation observed in the LAP index suggests significant heterogeneity within our sample, indicating that while the mean values are low, some individuals may still be at increased metabolic risk. These findings emphasize the need to account for individual differences when evaluating metabolic risk in patients with PCOS. While VAI and LAP are valuable tools for identifying individuals at risk, their interpretation should be contextualized within the individual's overall clinical profile.

It is well-documented that the occurrence of overweight or obesity in women affected by PCOS can reach up to 61% (31); however, the majority of participants in our study had a normal BMI. Although the mean BMI of the study population was within the normal range, VAI and LAP were included due to their ability to reflect visceral adiposity and metabolic risk more precisely than BMI, particularly in populations such as women with PCOS who may have hidden metabolic risk factors despite normal weight. Supportively, a recent study suggested that LAP and VAI may have significant potential for the early detection of insulin resistance and cardiometabolic risk, and could also be valuable in assessing hyperandrogenism, even in lean women with PCOS (12). A study by Mario et al. explored the predictive capacity of various adiposity indices for identifying subclinical metabolic disruptions and cardiometabolic risk in patients with PCOS, classified as classic or ovulatory, and in healthy controls. Their findings indicated that the LAP, with a cut-off value of ≥34, and the VAI, with a cut-off value of ≥1.32, emerged as the most effective markers for PCOS of the classic and ovulatory types, respectively (34). Therefore, the critical role of heightened adiposity indices in the screening of cardiometabolic risk within these demographic groups should not be overlooked.

Emerging evidence indicates a pivotal role for pro-inflammatory and inflammatory mediators in the pathophysiology of PCOS, which appears to be distinctly associated with obesity and other factors (15,35). A substantial array of studies highlights the potent influence of diet on the escalation of inflammatory factors (17,20,35). Dietary constituents, including fruits and vegetables, dietary fiber, omega-3 fatty acids, vitamin C, and vitamin E, have been shown to decrease inflammatory activity in the human body. Conversely, certain components of the diet, such as SFAs, high glycemic index foods, and a high omega-6/omega-3 ratio of PUFAs, contribute to inflammation (22). Whilst the inflammatory potential measured using DII in our study was higher than studies conducted with similar samples in the literature (22,23,36), the study by Azarbayjani et al. reported a notably higher inflammatory potential than we observed (37). A case-control study has demonstrated that a diet with a high DII score was associated with a 1.75-fold increased risk of PCOS (22). Furthermore, in a population-based prospective study, high DII scores were characterized by an increased risk of CVD (38). Based on all this evidence, we hypothesized that adiposity indices, which are effective markers to assess metabolic disorders contributing to CVD, would be high in patients with a high DII.

Although the results are consistent with this assumption, the non-significance of the findings may be explained by the limited number of samples and different phenotypes of PCOS patients included in the studies.

We revealed that increased DII is related to a lower intake of some macro- and micronutrients such as protein, fiber, MUFAs, omega-3 fatty acids, vitamin A and K, vitamin C, B vitamins except B12, and minerals such as calcium, iron, magnesium, zinc, and potassium. The observation that anti-inflammatory components, especially omega-3 fatty acids, vitamin A, and vitamin C, are observed at lower levels in patients with a pro-inflammatory diet (high DII) is consistent with the existing literature (39). Diet has been acknowledged as one of the modifiable lifestyle factors with a significant influence on oxidative stress, and incorrect eating patterns and lack of physical activity can trigger and aggravate the symptoms of PCOS (40). Research in this area frequently emphasizes that women with PCOS tend to have suboptimal intake of essential micronutrients, including vitamins A, C, and K, as well as critical minerals such as magnesium, zinc, calcium, and potassium (41). These nutrient deficiencies may be exacerbated by the consumption of pro-inflammatory foods, contributing to a higher DII. A diet with high inflammatory potential can hinder proper nutrient absorption and disrupt metabolic processes, worsening the nutrient gaps commonly observed in women with PCOS (42). These phenomena point to a potential role for inflammation in shaping both nutrient intake and overall health outcomes in this population.

A strength of this study is its pioneering approach to investigating adiposity indices and dietary inflammatory potential. However, limitations of this study involve the small study population, the lack of a control group, and the inability to establish causal relationships, which hinder the generalizability of the results. Due to the cross-sectional nature of the study, it is not possible to determine whether diet influences adiposity or vice versa. Increased visceral adiposity may also contribute to altered dietary intake patterns or nutrient metabolism, suggesting a potential bidirectional relationship that warrants further investigation in longitudinal studies. In particular, the limited sample size-especially when participants are divided into subgroups-may have reduced the statistical power of the analyses. This might partially account for the absence of statistically significant results in certain comparisons. A post hoc power analysis or future studies with larger samples would help clarify these relationships more reliably. Furthermore, the findings of this study are based on data collected from patients at a single center and therefore reflect characteristics specific to this particular population. As such, the results may not be generalizable to other populations or settings. This limitation should be taken into account when interpreting the findings, and future multi-center studies with more diverse populations are recommended to enhance generalizability. Moreover, given that the DII was estimated through a 3-day dietary record, which may not fully capture habitual intake, future studies should consider combining dietary records with food frequency questionnaires (FFQs) to enhance the assessment of long-term dietary inflammatory potential.

In the context of our study, both VAI and LAP values among women with PCOS were found to be lower compared to previously reported values in the literature. While no statistically significant correlation was identified between DII values and adiposity indices, it is noteworthy that this may potentially confer long-term risks for these individuals. The study emphasizes the critical importance of promoting a dietary pattern that is conducive to reducing inflammation, which is a pivotal factor in the management and progression of the disease.

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Author Contributions

The study was conceptualized and designed by **Gizem Cengiz Pala** and **Gözde Dumlu Bilgin.** Data collection was carried out by **Gizem Cengiz Pala,** while data analysis and interpretation of the findings were performed by **Gözde Dumlu Bilgin,** in collaboration with **Gizem Cengiz Pala.** Both authors contributed to drafting and revising the manuscript.

All authors have critically reviewed and approved the final version of the manuscript and take full responsibility for all aspects of the study.

Conflict of Interest

The authors have declared no conflicts of interest. This study was not generated by or with the assistance of artificial intelligence tools. It is the original work of the author(s).

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Ethical Approval

This study was performed in accordance with the guidelines outlined in the Declaration of Helsinki, and all procedures were approved by the Yeditepe University Faculty of Health Science Non-Interventional Research Ethics Committee with date and number 08.04.2022/16.

Peer Review Process

Extremely and externally peer-reviewed.

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