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Orjinal Araştırma / Original Article

Association Between Hyperuricemia and Non-Traditional Adiposity Indices in Pre-Menopausal Women

Menapoza Girmeyen Kadınlarda Hiperürisemi ve Geleneksel Olmayan Adipozite Belirteçleri Arasındakı İlişki

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Abstract

Aim: Hyperuricemia is a risk factor for hypertension, type 2 diabetes mellitus, dyslipidemia, metabolic syndrome, cardiovascular diseases. Body adiposity index (BAI), lipid accumalation pruduct (LAP), cardiometabolic index (CMI) and visceral adiposity index (VAI) are non-traditional parameters used to evaluate visceral obesity. There are not enough studies on the relationship between non-traditional adiposity markers and hyperuricemia. In this study, we aimed to investigate the relationship between hyperuricemia and non-traditional adiposity markers in pre-menopausal women.

Material and Method: 86 premenopausal women were included in the study. Height, weight, waist circumference (WC) and hip circumference measurements were made, Body mass index (kg/m^2) was calculated. Blood pressure was measured from both arms after 10 minutes of rest. Blood samples were taken after 12 hours of fasting.

Rusults: 43 women (%50) had hyperuricemia. In the group with hyperuricemia, traditional and non-traditional (BAI, LAP, VAI, CMI) adiposity markers were increased. A positive correlation was found between serum uric acid (UA) levels and adiposity markers. WC, LAP and CMI were found to be independent adiposity markers of serum UA.

Conclusion: In our study, we determined that WC, LAP and CMI were independent adiposity markers for serum uric acid value.

Keywords: Cardiometabolic index, lipid accumalation pruduct, visceral adiposity, uric acid

Öz

Amaç: Hiperürisemi hipertansiyon, tip 2 diyabet, dislipidemi, metabolik sendrom ve kardiyovasküler hastalıklar için bir risk faktörüdür. Vücut yağ indeksi (VYI), lipit biriktirme ürünü (LBÜ), kardiyo metabolik indeks (KMI) ve viseral adipozite indeksi (VAI) viseral adipoziteyi değerlendirmek için kullanılan geleneksel olmayan parametrelerdir. Hiperürisemi ve geleneksel olmayan adipozite belirteçleri arasındaki ilişkiyi inceleyen yeterli sayıda çalışma yoktur. Biz bu çalışmada menapoza girmemiş kadınlarda hiperürisemi ve geleneksel olmayan adipozite belirteçleri arasındaki ilişkiyi incelemeyi amaçladık.

Gereç ve Yöntem: Çalışmaya 86 menapoza girmemiş kadın dahil edildi. Boy, kilo, bel çevresi (BÇ), ve kalça çevresi ölçümleri yapıldı. VKI (kg/m^2) hesaplandı. Kan basıncı 10 dakikalık dinlenmeden sonra her iki koldan ölçüldü. Kan örnekleri 12 saatlik açlıktan alındı.

Bulgular: 43 kadında (% 50) hiperürisemi tespit edildi. Hiperürisemi grubunda, geleneksel ve geleneksel olmayan (VYI, LBÜ, VAI, KMI) adipozite belirteçleri artmış olarak bulundu. Serum ürik asid ve adipozite belirteçleri arasında pozitif korelasyon tespit edildi. BÇ, LBÜ ve KMI serum ürik asidin bağımsız adipozite belirteçleri olarak bulundu.

Sonuç: Çalışmamızda, BÇ, LBÜ ve KMI in serum ürik asid için bağımsız adipozite belirteçleri olduğunu belirledik.

Anahtar Kelimeler: Kardiyo metabolik indeks, lipid biriktirme ürünü, viseral adipozite, ürik asit

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INTRODUCTION

Hyperuricemia is a risk factor for hypertension (HT), type 2 diabetes mellitus (DM), dyslipidemia, metabolic syndrome, cardiovascular diseases (CVD).[1-4] In addition, hyperuricemia increases cardiovascular mortality.[5]

Since uric acid is an antioxidant, increased plasma uric acid (UA) concentration is thought to have a compensatory role in response to oxidative stress.[6] While UA has antioxidant activity in extracellular environment; It has harmful effects after entering the cells, including vascular smooth muscle cells and adipocytes.[7]

The most commonly used clinical parameter today to determine visceral obesity is the waist circumference (WC).[8] Visceral obesity is associated with increased adipocytokin production, proinflammatory activity, impaired insulin sensitivity, increased risk of DM, high triglyceride (TG)/low HDL cholesterol, HT, and atherosclerosis.[9]

Waist circumference, body mass index (BMI), waist hip ratio (WHR) and waist height ratio (WHtR) are traditional parameters used to classify and define obesity. Body adiposity index (BAI), lipid accumulation pruduct (LAP), cardiometabolic index (CMI) and visceral adiposity index (VAI) are non-traditional parameters used to evaluate visceral obesity. Since visceral fat measurement rather than obesity is more important for cardiovascular risk, these new parameters is developed.[10]

Although methods such as magnetic resonance (MR) imaging, computed tomography (CT), dual X-ray absorptiometry (DEXA) are used to evaluate visceral adiposity, these methods are not useful because they are costly and time consuming.[11,12] In recent studies, it has been shown that new parameters (BAI, LAP, CMI, VAI) created with mathematical models including both anthropometric (BMI, WC) and atherosclerotic (TG and HDL) parameters can be used to evaluate visceral obesity.[10,13]

There are not enough studies on the relationship between traditional and non-traditional adiposity markers and hyperuricemia. In this study, we aimed to investigate the relationship between hyperuricemia and traditional and non-traditional adiposity markers in premenopausal women.

MATERIAL AND METHOD

86 premenopausal women aged 18-50 years, who applied to our hospital's Internal Diseases, Endocrinology and Metabolic Diseases polyclinics, were included in the study. Consecutive subjects who agreed to participate in the study were included in the study. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Ethics committee approval was obtained from the local ethics committee with the number 2016/692 prior to the study. Volunteers were given detailed information about this study and those who wanted to participate were included in the study after the informed consent form was signed.

An anamnesis was received in detail from all volunteers who wanted to participate in the study. The patients were evaluated in terms of known and previous diseases, smoking, alcohol, drug and drug history. After the anamnesis, physical examination of all women was done. They were primarily evaluated for any focus of infection. Height, weight, WC and hip circumference (HC) measurements were made, BMI (kg/m^2) was calculated. Blood pressure was measured from both arms after 10 minutes of rest and recorded as systolic (SBP) and diastolic (DBP) blood pressure. Blood samples were taken to measure triglyceride (TG), HDL-cholesterol, UA, glucose and insulin after 12 hours of fasting. Hyperuricemia was defined as the serum UA level higher than 6.0 mg/dL. The cases were divided into 2 groups according to their UA levels. Cases with UA level ≥ 6 mg/dL were determined as high UA group, and cases with UA level < 6 mg/dL were determined as normal UA group.

Exclusion criteria;

- Under 18 and postmenopausal women,
- Pregnant or breastfeeding women,
- Those who use drugs that affect the level of UA. (allopurinol, thiazide etc)
- Those with known DM or those taking antidiabetic medication,
- Those who use drugs that affect insulin sensitivity,
- Those who use steroid or immunosuppressive therapy, lipid-lowering drugs, antihypertensive drugs, hormone replacement therapy,
- An active infection focus was detected in the physical examination,
- Those with known malignancies, kidney failure, liver failure, rheumatological disease or gout disease, hypothyroidism
- Women using cigarettes and alcohol.

Biochemical Analysis

Glucose (used glucose hexokinase method), HDL-cholesterol (used antigen-antibody complex method), TG (used enzymatic reaction method) and UA (used enzymatic uricase method) were studied an Olympus AU 5800 (Becman Coulter Inc. USA) device. Insulin Chemiluminescence method and Immulite 2000 Immunoassay System (Siemens Health Care Diagnostic, Germany) was studied.

Data Analysis

- Homeostasis Model Assessment of Insulin Resistance (HOMA-IR):
- (Fasting glucose (mg/dL) x fasting insulin (µU/mL))/22.5
- Body Mass Index (BMI): Body Weight (kg)/length^2 (m^2)
- Visceral Adiposity Index (VAI) (female): (WC/(36.58+(1.89xBMI))]) x [(TG mmol/l/0.81) x (1.52/HDL mmol/l)]
- Body Adiposity Index (BAI): ((HC cm)/Height1.5 m) -18
- Lipid Accumulation Product (LAP) (female): ((WC cm) -58) * TG mmol/l
- Cardiometabolic Index (CMI): (TG mmol/l/HDL mmol/l) * (WHtR cm)

Statistical Analysis

The SPSS 22.0 package program was used for statistical data analyses Descriptive statistics were shown for normal distribution of continuous variables as mean±standard deviation, while numerical parameters without normal distribution were shown with median (minimum-maximum). Categorical variables were represented by numbers and percentages. Continuous numerical variables were checked by the Kolmogorov-Smirnov Test to determine normality of distribution. In the comparison of the two groups, those with normal distribution were performed with the T-test, and those with abnormal distribution were performed with the Mann Whitney U test. Spearman correlation analysis and lineer regresyon analysis was used to determined the parameters related with serum UA levels. Logistic multivariate regression analysis was performed to determine traditional and non-traditional adiposity parameters associated with hyperuricemia. Independent parameters included in Backward linear regression analysis were WC, WHR, BMI, LAP, CMI, BAI and VAI. P<0.05 was considered significant in all analyses.

RESULTS

Of the 86 women included in the study, 43 women (%50) had hyperuricemia. In the group with hyperuricemia, traditional (BMI, WC, WHR) and non-traditional (BAI, LAP, VAI, CMI) adiposity markers, SBP, DBP, HOMA-IR, insulin, fasting blood glucose and TG values were increased (in all of them p <0.001) (**Table 1**). In the correlation analysis of serum UA level with the above parameters; A positive correlation was found between serum UA levels and all parameters. In addition, a negative correlation was found between serum UA level and HDL-cholesterol (**Table 2**). In addition,;WC, LAP and CMI were found to be independent adiposity markers of serum UA in the multivariate linear regression analysis (**Table 3**). WHR, BMI, BAI and VAI were not found to be significant in multivariate linear regression analysis. No correlation was detected between the parameters included in the regression analysis. The WC was only found to be independent adiposity marker of hyperuricemia in binary logistic regression analysis (OR=0.902, p=0.003) (**Table 4**).

DISCUSSION

The relationship between hyperuricemia and obesity and especially visceral obesity has been shown in previous studies.[14-17] In recent years, new markers (LAP, CMI, BAI, VAI) have been identified that determine visceral obesity. A limited studies have been conducted on the relationship between these new non-traditional visceral adiposity markers and UA, especially in Asian communities.[10,18,19] To our knowledge, our study is one of the first to exclude factors that may affect serum UA in the western population.

Hyperuricemia is a risk factor for HT, type 2 DM, dyslipidemia, metabolic syndrome, and CVD.[1-4] In addition, hyperuricemia increases cardiovascular mortality.[5]

Table 1. Demographic and laboratory characteristics of women with and without hyperuricemia.

Parameters	High UA UA ≥ 6 mg/dL (n=43)	Normal UA UA < 6 mg/dL (n=43)	p
Age (years)	35.4±10.2	35.2±7.1	0.923
BMI (kg/m²)	36.3±6.0	25.8±6.4	<0.001
WC (cm)	102.2±12.8	77.2±12.8	<0.001
WHR	0.87±0.06	0.77±0.05	<0.001
SBP (mmHg)	126.3±12.4	113.0±11.9	<0.001
DBP (mmHg)	80 (56-97)	70 (58-91)	<0.001
Glucose (mg/dL)	100.5±12.8	87.8±12.9	<0.001
İnsülin (µU/mL)	14.6 (2.0-49.1)	7.4 (2.0-32.6)	<0.001
HOMA-IR	68.4 (8.0-257.5)	28.5 (5.9-180.3)	<0.001
Uric acid (mg/dL)	6.7 (6.0-9.9)	4.0 (2.0-5.8)	<0.001
HDL (mg/dL)	44 (29-70)	49 (30-83)	0.039
TG (mg/dL)	148 (69-512)	85 (33-456)	<0.001
VAİ	2.71 (1.13-9.86)	1.28 (0.32-7.96)	<0.001
LAP	71.2 (21.8-280.8)	13.9 (0-123.6)	<0.001
CMI	0.88 (0.35-3.01)	0.31 (0.08-2.20)	<0.001
BAI	37.7 (28.9-54.0)	28.5 (22.4-51.0)	<0.001

BMI: Body mass index, WC: Waist circumference, WHR: waist to hip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, TG: Triglycerides, VAI: Visceral adiposity index, BAI: Body adiposity index, LAP: Lipid accumulation pruduct, CMI: Cardiometabolic index, UA: Uric acid.

Tablo 2. Spearman's correlation of determinants of serum UA

Parameters	r
BMI (kg/m²)	0.753**
WC (cm)	0.766**
WHR	0.662**
VAİ	0.598**
BAI	0.694**
LAP	0.687**
CMI	0.640**
HOMA-IR	0.526**
Glucose (mg/dL)	0.523**
İnsülin (µU/mL)	0.486**
SBP (mmHg)	0.583**
DBP (mmHg)	0.484**
TG (mg/dL)	0.544**
HDL-Cholesterol (mg/dL)	-0.407**

BMI: Body massindex, WC: Waistcircumference, WHR: waist to hip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, TG: Triglycerides, VAI: Visceral adiposity index, BAI: Body adiposity index, LAP: Lipid accumulation pruduct, CMI: Cardio metabolic index. ** p<0,001 for spearman's correlation

Tablo 3. Backward linear regression analysis of determinants of serum UA (Adjusted R2= 0.602)

Parameters	r	Standardized β	p
WC (cm)	0,766**	0.620	0.001
LAP	0.687**	- 0.574	0.023
CMI	0.640**	0.565	0.007

WC: Waistcircumference, LAP: Lipid accumulation pruduct, CMI: Cardio metabolic index. Independent parameters included in linear regression analysis were WC, WHR, BMI, LAP, CMI, BAI and VAI.

Table 4. Binary logistic Regression Analysis for hyperuricemia

	Model 1 (Backward) R^2=0.503			Model 6 (Backward) R^2= 0.495		
Parameters	**p**	**Exp (B)**	**CI (%95)**	**p**	**Exp (B)**	**CI (%95)**
Constant	0.011	1.436E+10	-	<0.001	2803538271	-
WC cm	0.455	0.925	(0.753-1.135)	0.003	0.902	(0.843-0.965)
WHR	0.206	0.000	(0.000-10089.159)	0.058	0.000	(0.000-1.718)
BMI	0.318	0.878	(0.669-1.139)			
BAI	0.804	1.039	(0.766-1.411)			
LAP	0.861	1.004	(0.958-1.052)			
CMI	0.708	5.670	(0.001-49632.351)			
VAI	0.649	0.579	(0.055-6.101)			

BMI: Body massindex, WC: Waist circumference, WHR: Waist to hip ratio, VAI: Visceral adiposity index, BAI: Body adiposity index, LAP: Lipid accumulation pruduct, CMI: Cardio metabolic index.

The first study to report that non-traditional visceral adiposity markers (LAP, CMI and BAI) are independent risk factors for hyperuricemia have been conducted very recently with Wang H et al.[19] In this study, it was reported that Chinese female individuals with 5937 hyperuricemia were older and BMI, WC, WHR, WHtR, BAI, LAP, CMI, SBP, DBP, fasting blood sugar and TG levels were higher than those without hyperuricemia. However, hyperuricemia has been reported to be higher in patients with DM (619 patients), HT (2841 patients), and patients with a history of heart disease (701 patients). While these comorbid conditions known as risk factors for hyperuricemia were not included in our study; the traditional (BMI, WC, WHR, HC) and non-traditional (BAI, LAP, VAI, CMI) adiposity markers, SBP, DBP, TG, fasting blood glucose, insulin and HOMA-IR values showed strong positive correlation with serum UA levels (r> 0.500, p <0.001 in all individuals). In addition, we determined that WC, LAP and CMI were adiposity markers for serum UA level.

In another recent study in China; Liu XZ et al.[10] investigated 174698 individual. Similarly, in this study, all traditional and non-traditional adiposity markers (except BMI and VAI) were found to be significantly higher in hyperuricemic individuals. In the correlation analysis in women; While only positive correlation was detected between LAP and CMI and serum UA level (r=0.235, r=0.264 p, respectively, p <0.001), no correlation was found with other non-traditional adiposity markers (BMI, VAI). In another Chinese study last year; Huang X et al.[18] examined the relationship between hyperuricemia and visceral adiposity in 1284 individuals over 40 years old without malignancy and chronic kidney disease. They reported that independent determinants of serum UA level increased age, VAI and LAP.

The first study investigating the relationship between non-traditional adiposity markers (VAI and BAI) and hyperuricemia was performed with Dong H et al.[20] and they were reported that VAI has a strong relationship with hyperuricemia. Then, in the second study in men who did not have metabolic syndrome; It has been reported that VAI is strongly associated with hyperuricemia.[21]

Yamada et al.[22] investigated the relationship between visceral adiposity and hyperuricemia. The study included 801 Japanese men who did not use any medication that affected uric acid levels (antidiabetic and antihypertensive etc), and did not have any kidney, cardiovascular or malignant diseases. Visceral adipose tissue and hepatic adipose tissue were evaluated using computed tomography. Visceral adipose tissue and hepatic adipose tissue have been shown to be independently associated with hyperuricemia.

In a retrospective study involving 1498 patients by Amato et al.[13] it has been reported that VAI is an independent risk factor for cardiovascular and cerebrovascular events. However, the same relationship could not be shown for WC and BMI. This effect is thought to be due to the indirect reflection of non-traditional risk factors such as the production of cytokines, lipolytic activity and increased plasma free fatty acids. This study shown that VAI is an important indicator of visceral adipose tissue function and insulin sensitivity and that VAI is strongly associated with cardiometabolic risk.

There are important differences between the subcutaneous adipose tissue and the visceral adipose tissue in the abdominal cavity. Visceral adipose tissue contains more inflammatory cells and a larger percentage of adipocytes than subcutaneous adipose tissue, Visceral adipose tissue carries more glucocorticoid and androgen receptors than subcutaneous adipose tissue. Visceral adipocytes are metabolically more active, more sensitive to lipolysis and have more insulin resistance. For this reason, visceral adipose tissue is more associated with cardiometabolic mortality.[23] In a study by Takir et al.[24] the relationship between lowering uric acid level and insulin resistance was investigated in individuals with asymptomatic hyperuricemia without DM. 73 people were included in the study and 40 people were administered allopurinol for 3 months. As a result of the study, it was found that administration of allopurinol in hyperuricemic individuals reduces uric acid levels and improves insulin resistance. In our study, we found that HOMA-IR, which is an indicator of insulin resistance, was higher in the group with hyperuricemia.

The most important of our study's limitations may be the low number of cases. However, since we exclude all possibilities that may affect the serum uric acid level in our study, the number of cases can be considered sufficient. Our second limitation; Since it is a cross-sectional study, we cannot show the cause-effect relationship exactly.

CONCLUSION

As a result; in our study, we determined that WC, LAP and CMI were independent adiposity markers for serum uric acid value. We also found that the independent adiposity marker for hyperuricemia is WC.

ETHICAL DECLARATIONS:

Ethical approval: Ethics committee approval was obtained from the local ethics committee with the number 2016/692 prior to the study.

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer reviewed.

Conflicts of interest: There are no conflicts of interest.

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Author Contributions: All of the authors declared that they have all participated in the design, execution, and analysis of the paper, and they have approved the final version.

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