Visceral adiposity index was independently associated with hyperuricemia in patients with polycystic ovary syndrome

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Abstract

Objective: The current study aimed to explore the prevalence rate of hyperuricemia in women with polycystic ovary syndrome (PCOS) and investigate the relationship between Visceral adiposity index (VAI) and hyperuricemia in PCOS.

Methods: In this cross-sectional study, 318 PCOS women were evaluated between November 2018 to September 2020. Of them, 256 subjects with complete anthropometric and the serum uric acid (SUA) level data were analyzed. Multivariable linear regression and logistic regression analyses were performed to determine the associations of VAI with the SUA level and hyperuricemia.

Results: The prevalence rate of hyperuricemia was 56.3% in women with PCOS and was gradually increased across tertiles of VAI, which was 2.6%, 21.3%, 22.4%, respectively. Obese subjects had significantly higher levels of systolic blood pressure (SBP), waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR), body fat percentage (BFP), triglycerides (TG), low-density lipoprotein cholesterol (LDL-c), VAI (P<0.001) and lower high-density lipoprotein cholesterol (HDL-c) (P<0.001). Pearson correlation analysis showed the SUA level was positively correlated with BMI, BFP, WHR, log (TG), log (LDL-c), SBP, and log (VAI) and negatively correlated with HDL-c. In addition, with adjustment for potential confounding factors, multivariable linear regression and logistic regression analyses showed that VAI significantly associated with the SUA level and hyperuricemia, with the coefficient (95% confidence interval (CI)) of 9.20 (2.85-15.56, P=0.005) and the adjusted odds ratio (95% CI) of 1.32 (1.05-1.65, P=0.018), respectively.

Conclusion: The present study indicates that VAI was independently associated with hyperuricemia, even with adjustment for BMI and other potential confounding factors.

Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disease affecting up to 21% of reproductive-age women[1]. Women with PCOS accompanying with metabolic disorders such as insulin resistance (IR), abdominal obesity, obesity, hyperuricemia, dyslipidemia and abnormal glucose[2]. The prevalence of obesity in women with PCOS is about 30%~70%, and obesity can worsen metabolic disorders in PCOS patients[3].

Guidelines for the management of hyperuricemia and gout highlighted the benefits of weight loss for overweight / obese patients[4]. Previous studies have developed a number of weight management indices, including body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and body fat percentage (BFP). BMI and WC are the common measurements used to identify obesity in clinical practice and have been demonstrated as predictors of metabolic and Cardiovascular diseases (CVDs), but they cannot completely distinguish between visceral adipose tissue and subcutaneous adipose

tissue[5]. Studies have shown that there is a stronger correlation between the SUA level and visceral adipose tissue[6]. In recent years, as an indicator of the function of visceral adipose tissue, Visceral adiposity index (VAI) has been introduced to reflect metabolic abnormalities[7]. VAI is a model that can be easily calculated by both anthropometric (BMI and WC) and laboratory (triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C)) parameters. It has been demonstrated as a novel and accurate indicator of visceral fat distribution[8]. Moreover, studies have shown that the VAI has high accuracy in predicting metabolic disease such as IR, Type 2 Diabetes Mellitus (T2DM) and prediabetes[9-11].

Among those metabolic disorders in patients with PCOS, a growing evidence suggested that hyperuricemia was closely related to obesity [1, 12]. Hyperuricemia is a disease of impaired uric acid metabolism. A recent study indicated that the prevalence of hyperuricemia in PCOS women is more than 25%, while 58.75% of obese PCOS women approximately suffer from hyperuricemia, which was almost three folds higher than that in PCOS women with normal BMI[13]. Elevated SUA level not only increased the risk of gout, but also increased cardiovascular risk by promoting inflammation, oxidative stress and proliferation in PCOS women[14]. Also, hyperuricemia put PCOS women at a higher risk of fetal outcome and adverse maternal events [15]. Therefore, although it is unclear whether the SUA level is a cause or risk factor for obesity, we ought to pay more attention to women with high uric acid levels, especially obese PCOS women. However, there are limit studies on the association between visceral adipose tissue and hyperuricemia in women with PCOS, in which there have been some contradictory views [16, 17]. Therefore, the objective of the present study was to explore the relationship between VAI and hyperuricemia in women with PCOS.

Materials And Methods

Study population

The cross-sectional study was carried out at Department of Endocrinology and Diabetes, the First Affiliated Hospital of Xiamen University, Xiamen, China, from November 2018 to September 2020. A total of 318 patients aged from 18 to 40 years with PCOS were screened in this study. Of 318 PCOS patients, a total of 256 (80.5%) with serum urate data were left for further analysis. The diagnosis of PCOS is based on Rotterdam criteria[18], which has been detailed described in our previous report[19]. The ethics committee of the First Affiliated Hospital of Xiamen Medical University approved this study protocol and written informed consent was obtained from each participant.

Anthropometric and Laboratory Measurements

The socio-demographic status included age, medical history and drug use, smoking and drinking, etc. All clinical, anthropometric and laboratory indicators were measured by a properly trained healthcare workers

following standardized protocols and performed as described in the previous report[19]. Height, WC, Hip circumference (HC) and body weight were measured by using a calibrated scale with lightweight clothing. The analysis of BFP was performed using bioelectrical impedance analysis (Tanita BC-420MA; Tanita, Tokyo, Japan). This analyzer is a simple and validated method for assessing body composition[20]. After sitting quietly for at least 15 minutes, the participants measured their blood pressure (BP) using an Omron electronic sphygmomanometer ((OMRON Healthcare)).

All subjects received blood testing for reproductive hormonal and metabolic parameters in the morning after an overnight fasting. All biochemical measurements were tested in the central laboratory of the First Affiliated Hospital, Xiamen University. Total cholesterol [6], TG, HDL-c were determined on a HITACHI 7450 analyzer. SUA level and creatinine were measured by the autoanalyzer (COBAS INTEGRA 400 plus, Roche, Basel, Switzerland). Testosterone (T) were quantified using chemiluminescent immunoassay analysis (SIEMENS ADVIA Centaur XP Immunoassay System; Siemens Healthcare Diagnostics Inc.).

Calculation and definition of indexes

Anthropometric indices were calculated using the following equation: BMI = weight [kg] / (height [m])^2; The WHR =WC [cm] /HC [cm]; and VAI = $\left[\frac{WC[cm]}{(36.58+1.89\times BMI)}\right]\times(\frac{TG[mmol/l]}{0.81})\times(\frac{1.52}{HDL-c[mmol/l]})$ [21]; The glomerular filtration rate (eGFR) was estimated using the following formula: eGFR (mL/min/1.73m^2) = 175×Scr (mg/dL)$^{-1.234}$ × age (year)$^{-0.179}$ ×0.79[22].

Hyperuricemia was defined as the SUA level exceeding 360 μmol/L (6 mg/dl) in women[23]. BMI categories were defined as normal weight (BMI < 24kg/m^2), overweight (24 ≤ BMI < 28 kg/m^2) and general obesity (BMI ≥ 28 kg/m^2)[24]. And abdominal obesity was defined as a WC≥ 80 cm for females[24].

Statistical analyses

The statistical analyses of the data were performed using SPSS version 21.0 software (IBM Corporation, Armonk, NY). Skewness and kurtosis tests for normality. Continuous variables are expressed as the mean

± standard deviation (SD) or as median (inter-quartile range, IQR)，while categorical variables are presented as number and percentage. All subjects were stratified by the BMI and the SUA level, respectively. Differences between two groups were analyzed on continuous variables using the Student's t test for those with normal distribution and Mann-Whitney U test for those with skewed distribution and on categorical variables using chi-square test. Differences between the three groups were analyzed on continuous variables using one-way ANOVA for those with normal distributions and Kruskal-Wallis test for those with skewed distributions and on categorical variables using chi-square test.

Pearson's correlation analysis was used to investigate the correlation of the SUA level with age, Systolic blood pressure (SBP), diastolic blood pressure (DBP), BMI, WHR, lipid profiles, BFP, T(log-transformed), and VAI (log-transformed). Stepwise multivariable linear regression was used to assess the association between various factors associated with the SUA level. Multivariable linear regression was used to analyze the association of VAI with the SUA level. And multivariable logistic regression analysis was used to calculate the adjusted odds ratios (OR) and 95% confidence interval (CI) of VAI for hyperuricemia in different models with adjustment for potential confounders. For both the multivariable linear regression and logistic regression analyses, model 1 was adjusted for age and occasional drinking. SBP, DBP, and eGFR were adjusted for in model 2; TC, LDL-c, T, and BMI were further adjusted for in model 3. All p-values were two-sided and p-value<0.05 was considered statistically significant.

Results

After excluding 62 participants without SUA data, 256 PCOS patients were included in this study. The mean (±SD) of SUA was 376.84 ± 87.95 μmol/L for all subjects and their media (IQR) of age was 27.5 (24.3-31.0) years old. The prevalence rate of hyperuricemia was 56.3%, with the mean (±SD) of SUA 438.91 ± 58.95 μmol/L. Patients with hyperuricemia had higher BMI and VAI (Table 1), and with the increase of BMI and VAI, the level of SUA and the prevalence of hyperuricemia also increased (Table 2, Figure 1).

Characteristics of study population

The detailed anthropometric and metabolic characteristics of females with PCOS categorized by the existence of hyperuricemia were shown in Table 1. Compared with the normouricemia group, the group with hyperuricemia had significantly greater values for SBP (116±11 vs. 120 ± 13, P=0.013), TC (4.94 ±0.90 vs. 5.19 ±0.92, p=0.028), TG (1.26 [0.86 - 1.84] vs. 1.61 [1.17 - 2.19], P<0.001), LDL-c (2.62 [2.19 - 3.18] vs. 2.85 [2.56 - 3.44], P=0.001), and creatinine (52.29 ± 8.33 vs. 55.57 ± 9.26, p=0.004) and lower values for HDL-c (1.32 [1.12 - 1.57] vs. 1.18 [1.03 - 1.33], P<0.001) and eGFR (150.96 ±27.74 vs. 141.44 ±31.12, p=0.011). The obesity indices such as BMI (25.50 ± 4.74 vs. 29.64 ± 4.61, P<0.001), WHR (0.85±0.07 vs.0.88±0.06, P=0.001), WC (84.9 ±12.5 vs. 93.8 ± 11.3, P<0.001), HC (99.3± 9.6 vs. 106.8±9.6,

P<0.001), BFP (34.23 ±7.35 vs. 39.57 ±6.51, P<0.001) and VAI (1.88 (1.18 - 2.79) vs. 2.54 (1.81 - 3.73), P<0.001)) were higher in the hyperuricemia group than that in the normouricemia group. However, there was no significant differences in age, DBP, and T between two groups.

Besides, in order to further study the relationship between obesity and other indicators, subjects were categorized into three groups (normal weight, overweight and obesity) in Table 2. As the degree of obesity increased, the level of SUA (312.6 ± 71.16, 372.57 ± 84.86, 410.93 ± 78.76, respectively, p<0.001) and the prevalence rate of hyperuricemia also increased (p<0.001). In addition, subjects with higher BMI were more likely to be older and occasional smoker and had significantly higher levels of SBP, WC, HC, WHR, BFP, VAI (1.45 (0.86 - 2.08), 2.19 (1.42 - 3.46), 2.65 (1.93 - 3.77), P<0.001, respectively), TG, HDL-c and LDL-c. However, there was no significant difference in DBP, TC, creatinine, eGFR and T.

Figure 1 showed the distributions of SUA levels and prevalence of hyperuricemia according to tertiles of VAI in patients with PCOS. As the tertiles of VAI increased, the level of SUA (p<0.001) and the prevalence rate of hyperuricemia also increased (2.6%, 21.3%, 22.4%, respectively, p<0.001).

Correlations of SUA level with clinical characteristics

We performed Pearson's correlation to investigate the correlation between SUA with clinical characteristics. We found that there were significant association between the obesity indexes (BMI, BFP and WHR), lipid profiles (log (TG), log (LDL-c) and log (HDL-c)), SBP, and log (VAI) (r=0.346, p<0.001) with SUA. However, further stepwise linear regression analysis indicated that SUA level were only positively associated with BMI (β= 0.325, p < 0.001) and Log (VAI) (β = 0.243, p < 0.001) in PCOS women (Table 3).

Association of VAI and SUA level

In addition, multivariate linear logistic regression analysis was used to further assess the association between VAI and SUA level (Table 4). In model 1 with adjustment for age and occasional drinking, VAI was significantly associated with SUA level, and the coefficient (95% CI) was 16.52(10.18-22.85, P<0.001). In model 2 with further adjustment for SBP, DBP, and eGFR, the significant association of VAI with SUA level remained and the coefficient (95% CI) was 14.74 (8.45-21.03, P<0.001). Further, after adjusted for additional TC, LDL-c, T, and BMI in model 3, VAI was still significantly associated with the SUA level, and the coefficient (95% CI) was 9.20 (2.85-15.56, P=0.005).

Association of VAI and hyperuricemia

Multivariate logistic regression analysis was performed to explore the association between VAI and hyperuricemia. The following three models were performed with same adjustments as those in multivariable linear regression analyses. In model 1, VAI were significantly associated with hyperuricemia, and the adjusted OR (95% CI) was 1.56 (1.26 - 1.92, P<0.001). In model 2, the associations of VAI with hyperuricemia remained significant, with the adjusted OR (95% CI) of 1.53 (1.24 - 1.89, P<0.001). In model 3, the significant associations between VAI and hyperuricemia still existed, and the adjusted OR (95% CI) was 1.32 (1.05-1.65, P=0.018) (Table 4).

Discussion

In the present study, the prevalence of hyperuricemia was 56.3% in PCOS women. The obesity indices such as BMI, WHR, WC, HC, BFP and VAI in PCOS women with hyperuricemia were significantly higher than those in normouricemia patients. Stepwise linear regression analysis showed that only BMI and VAI were significantly and positively associated with SUA level. Furthermore, VAI was significantly associated with hyperuricemia after adjusting for potential confounding factors including BMI in multivariable linear regression and logistic regression analyses. Additionally, SUA level and the prevalence of hyperuricemia increased along with tertiles of VAI in women with PCOS.

Previous studies have shown increasing SUA levels were positively associated with increased cardiovascular mortality and increased the risk of adverse pregnancy outcomes in PCOS patients, therefore SUA level determination is valuable for women with PCOS[15, 25]. Several studies had reported that the SUA levels and prevalence of hyperuricemia increased greatly in obese PCOS women[13], which was similar to present results. However, the underlying mechanism had not been clear until now, there were some hypotheses as following: body fat accelerated SUA production and the synthesis of triglyceride[26] and hyperinsulinemia could decrease renal UA excretion[27]. Since as derivatives of purine inhibiting oocyte maturation[28, 29], the elevated SUA levels in patients with PCOS could further aggravate the adverse pregnancy outcome. Besides, some guidelines have emphasized the benefits of weight loss in overweight or obese population with hyperuricemia [4, 30]. Therefore, more attention should be paid on women with high level of SUA, especially in obese PCOS women. To our knowledge, this is the first cross-sectional study to comprehensively evaluate the relationship between six obesity indicators (WC, HC, BMI, WHR, BFP, VAI) and hyperuricemia in PCOS women.

Obesity, especially visceral obesity not only aggravates reproductive outcomes in patients with PCOS, but also accelerates progression of cardiovascular disease and cardiovascular events[31]. Moreover, visceral obesity leads to IR and compensatory hyperinsulinemia by impairing the action of insulin, which plays a major role in the pathophysiological process of PCOS[32]. Therefore, to future evaluate and treat

metabolic and reproductive disorders in women with PCOS it is critical to determine whether visceral obesity is independent of overall obesity. Although the gold standard for evaluating visceral fat accumulation is magnetic resonance imaging (MRI) and CT[33]. However, considering the cost of MRI and radiation exposure, some newly developed indicators such as VAI has been introduced [34]. In recent years, VAI as surrogate marker of visceral fat dysfunction have been widely used in clinical practice and it is independently correlated with cardio-metabolic risk in the general population[35]. Recent study indicated that as VAI levels increased, the severity of anovulation, IR, dyslipidemia and the risk of type 2 diabetes also increased along with in PCOS patients[36-38]. Further Oh et al. [39] reported that VAI could replace visceral CT scanning to evaluate visceral adiposity and could predict IR in young PCOS patients, and also determined that VAI >1.79 was the optimal cutoff point for visceral adiposity. A recent cross-sectional study of 1328 general population showed that visceral fat accumulation was positively correlated with the risk of hyperuricemia[40]. Similarly, the results in the current study also suggested that VAI, as an indicator of visceral adipose accumulation, was a significant risk factor of hyperuricemia in patients with PCOS independent of potential confounding factors, which was consistent with the study conducted by Huang et al. [40]. Also, we observed that the prevalence of hyperuricemia trended to increase with the elevation of VAI in PCOS women. Overall, VAI appears to provide more valuable information beyond other obesity indices in assessing the SUA level and may be used as a potential risk marker for hyperuricemia in PCOS women.

The major advantage of current study was that it comprehensively evaluated the associations of various obesity indices with hyperuricemia in women with PCOS for the first time, and to further determine the relationship between VAI and SUA level and hyperuricemia. However, there were also several limitations to the present study. First, being a cross-sectional study, these data could not determine the causal relationship between VAI and hyperuricemia. The second limitation was that the sample size was relatively small and might not have enough power to reflect a significant correlation between VAI and the SUA level, which calls for further verification in larger PCOS samples. Third, we did not evaluate visceral fat by more accurate methods such as CT or MRI. Therefore, it remains to develop a well-designed epidemiological study to explore the predictive value of VAI for hyperuricemia in PCOS women and to determine its pathogenesis.

Conclusion

In summary, VAI could be used as a potential hyperuricemia risk marker in PCOS women, beyond the general index of obesity. the present study indicates that VAI was independently associated with hyperuricemia in women with PCOS, even with adjustment for BMI and other potential confounding factors. The high level of VAI was related to an elevated SUA level and the prevalence of hyperuricemia, suggesting that visceral fat accumulation may be related to uric acid metabolism. Future studies are necessary to clarify its underlying mechanism in PCOS patients.

Abbreviations

PCOS, Polycystic ovary syndrome; SUA, serum uric acid; SBP, systolic pressure; DBP, diastolic pressure; BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; VAI, visceral adiposity index; BFP, body fat percentage; TC, total cholesterol; TG, triglycerides; HDL-c, high density lipoprotein cholesterol; LDL-c, low density lipoprotein cholesterol; CI, confidence interval; IR, insulin resistance; CVDs, Cardiovascular diseases; T2DM, Type 2 Diabetes Mellitus; eGFR, estimated glomerular filtration rate; T, testosterone; UA, uric acid; SD, standard deviation; IQR, inter-quartile range; OR, odds ratios.

Declarations

Ethical approval and consent to participate

The study received approval and was carried out in accordance with the approved guidelines of the ethics committee of the First Affiliated Hospital of Xiamen Medical University

Consent for publication

Not applicable.

Availability of supporting data

Not applicable

Competing interests

The authors declare that they have no conflict of interest.

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Author Contributions

The study concept and design were framed by CL and XY. CY, YH, HY, XZ, JY, DM, ZC, XZ, and XS collected data. CY, YH and CL conducted the statistical data analysis and drafted the manuscript. XY, CL and XY contributed to discussion and revision. All authors read and approved the final manuscript.

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Tables

Table 1 Anthropometric information and biochemical characteristics in women with PCOS.

	all	Normouricemia	Hyperuricemia	p value
n	256	112(43.7%)	144(56.3%)	
Age (years)	27.5 (24.3-31.0)	28.0 (25.0 - 30.8)	27.0 (24.0 - 31.0)	0.877
Occasional drinking (n, %)	23 (8.98%)	7 (6.25%)	16 (11.11%)	0.179
SBP (mmHg)	118 ± 12	116 ± 11	120 ± 13	0.013
DBP (mmHg)	80 ± 10	79 ± 9	80 ± 11	0.197
WC (cm)	89.9 ± 12.6	84.9 ± 12.5	93.8 ± 11.3	<0.001
HC (cm)	103.5 ± 10.3	99.3 ± 9.6	106.8 ± 9.6	<0.001
BMI (kg/m^2)	27.8 ± 5.1	25.50 ± 4.74	29.64 ± 4.61	<0.001
WHR	0.86 ± 0.07	0.85 ± 0.07	0.88 ± 0.06	0.001
TC (mmol/L)	5.08 ± 0.92	4.94 ± 0.90	5.19 ± 0.92	0.028
TG (mmol/L)	1.47 (1.01 - 2.00)	1.26 (0.86 - 1.84)	1.61 (1.17 - 2.19)	<0.001
HDL-c (mmol/L)	1.23 (1.06 - 1.43)	1.32 (1.12 - 1.57)	1.18 (1.03 - 1.33)	<0.001
LDL-c (mmol/L)	2.77 (2.34 - 3.37)	2.62 (2.19 - 3.18)	2.85 (2.56 - 3.44)	0.001
UA	376.84 ± 87.95	297.04 ± 43.24	438.91 ± 58.95	<0.001
Creatinine	54.14 ± 9.00	52.29 ± 8.33	55.57 ± 9.26	0.004
eGFR	145.60 ± 29.80	150.96 ± 27.24	141.44 ± 31.12	0.011
T (ng/DL)	41.36 (32.09 - 53.46)	40.21 (32.22 - 50.84)	44.32 (31.83 - 56.34)	0.359
BFP	37.36±7.34	34.23 ±7.35	39.57 ±6.51	<0.001
VAI	2.24 (1.50 - 3.32)	1.88 (1.18 - 2.79)	2.54 (1.81 - 3.73)	<0.001

Note: Data were presented as mean ± SD or median (interquartile ranges) for continuous variables, and numbers (proportions) for categorical variables

Abbreviations: PCOS, Polycystic ovary syndrome; SBP, systolic pressure; DBP, diastolic pressure; BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; VAI, visceral adiposity index; BFP, body fat percentage; TC, total cholesterol; TG, triglycerides; HDL-c, high density lipoprotein cholesterol; LDL-c, low density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; T, testosterone; UA, uric acid;

Table 2 Participant characteristics according to BMI category.

	Normal BMI<24 kg/m^2	Overweight 24≤BMI<28 kg/m^2	Obesity BMI≥28 kg/m^2	p value
n	63(24.61%)	66(25.78%)	127(49.61%)	
Age (years)	29.0 (26.0 - 31.0)	26.0 (23.0-30.0)	27.0 (24.0 - 31.0)	0.039
Hyperuricemia (n, %)	14 (5.5%)	37 (14.5%)	93 (36.3%)	<0.001
Occasional drinking (n, %)	6 (9.52%)	1 (1.51%)	16 (12.60%)	0.035
SBP (mmHg)	113 ± 9	117 ± 12	122 ± 13	<0.001
DBP (mmHg)	79 ± 8	79 ± 10	80 ± 11	0.387
WC (cm)	74.7 ± 5.4	87.0 ± 5.5	99.1 ± 9.3	<0.001
HC (cm)	91.5 ± 4.3	100.5 ± 5.1	111.1 ± 7.5	<0.001
WHR	0.81 ± 0.05	0.86 ± 0.05	0.89 ± 0.07	<0.001
BMI (kg/m^2)	21.35 ± 1.80	26.07 ± 1.16	31.95 ± 3.23	<0.001
TC (mmol/L)	5.03 ± 1.11	4.92 ± 0.81	5.20 ± 0.85	0.118
TG (mmol/L)	1.06 (0.76 - 1.69)	1.40 (1.00 - 2.02)	1.59 (1.25 - 2.31)	<0.001
HDL-c (mmol/L)	1.46 (1.17 - 1.66)	1.18 (1.07 - 1.34)	1.20 (1.03 - 1.33)	<0.001
LDL-c (mmol/L)	2.45 (2.14 - 2.90)	2.77(2.30-3.36)	2.91 (2.60 - 3.46)	<0.001
SUA (μmol/L)	312.60 ± 71.16	372.57 ± 84.86	410.93 ± 78.76	<0.001
Creatinine(μmol/L)	52.76 ± 8.01	53.99 ± 7.99	54.89 ± 9.88	0.309
eGFR (mL/min/1.73m^2)	148.23 ± 27.35	145.80 ± 27.10	144.22 ± 32.30	0.685
T (ng/dL)	42.04 (33.69 - 55.83)	40.16 (32.10 - 56.74)	41.55 (31.08 - 53.39)	0.654
BFP (%)	27.20 ± 4.45	35.02 ± 2.58	42.48 ± 4.75	<0.001
VAI	1.45 (0.86 - 2.08)	2.19 (1.42 - 3.46)	2.65 (1.93 - 3.77)	<0.001

Note: Data were presented as mean ± SD or median (interquartile ranges) for continuous variables, and numbers (proportions) for categorical variables

Abbreviations: BMI, body mass index; SBP, systolic pressure; DBP, diastolic pressure; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; TC, total cholesterol; TG, triglycerides; HDL-

c, high density lipoprotein cholesterol; LDL-c, low density lipoprotein cholesterol; SUA, serum uric acid; eGFR, estimated glomerular filtration rate; T, testosterone; BFP, body fat percentage; VAI, visceral adiposity index;

Table 3 Pearson's correlation and stepwise linear regression of determinants of serum uric acid

	Pearson's correlation		Stepwise linear regression	
Variables	r	p value	Standardized β	p value
Age (years)	-0.034	0.591	-	
SBP (mmHg)	0.229	<0.001	-	
DBP (mmHg)	0.102	0.104	-	
BMI (kg/m^2)	0.438	<0.001	0.325	<0.001
WHR	0.278	<0.001	-	
TC	0.116	0.065	-	
Log (TG)	0.285	<0.001	-	
Log (HDL-c)	-0.294	<0.001	-	
Log (LDL-c)	0.193	0.002	-	
Log (T)	0.008	0.907	-	
BFP	0.406	<0.001		
Log (VAI)	0.346	<0.001	0.243	<0.001

Abbreviations: SBP, systolic pressure; DBP, diastolic pressure; BMI, body mass index; WHR, waist-to-hip ratio; TC, total cholesterol; TG, triglycerides; HDL-c, high density lipoprotein cholesterol; LDL-c, low density lipoprotein cholesterol; T, testosterone; BFP, body fat percentage; VAI, visceral adiposity index;

Table 4. Associations of visceral adiposity index and body fat percentage with serum uric acid level and hyperuricemia in patients with PCOS.

	Multivariable Linear regression on serum uric acid level			Logistic regression on hyperuricemia		
	Coefficient	95%CI	P value	ORs	95%CI	P value
VAI						
Model 1	16.52	10.18-22.85	<0.001	1.56	1.26-1.92	<0.001
Model 2	14.74	8.45-21.03	<0.001	1.53	1.24-1.89	<0.001
Model 3	9.20	2.85-15.56	0.005	1.32	1.05-1.65	0.018

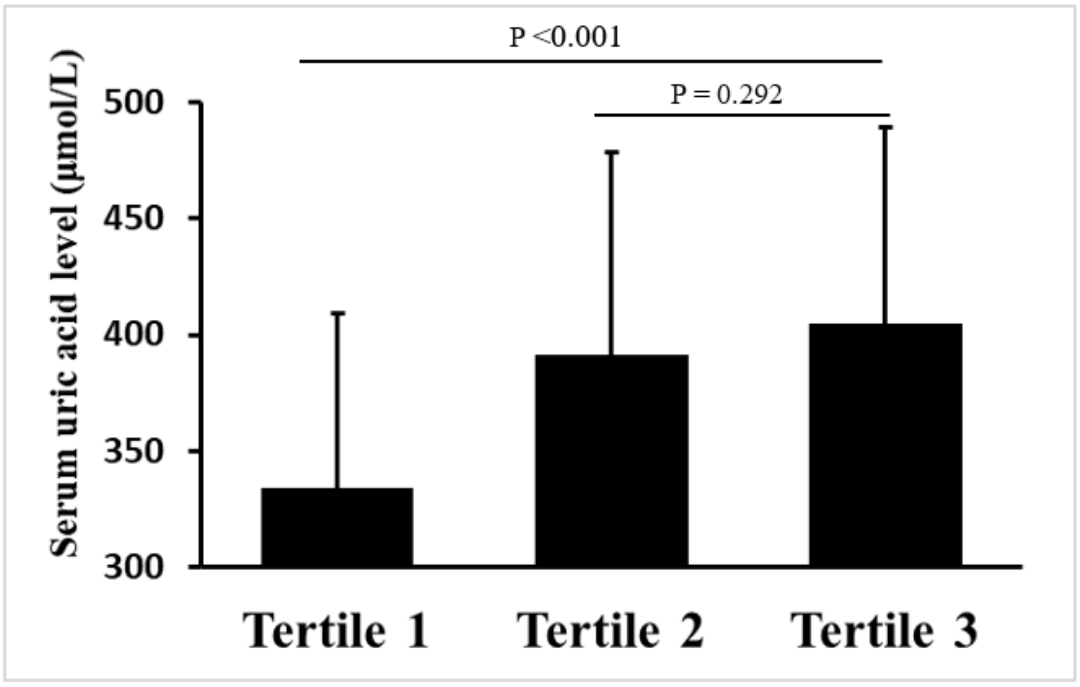
Model 1 was adjusted for age, occasional drinking.

Model 2 was further adjusted for SBP, DBP, and eGFR

Model 3 was further adjusted for TC, LDL-c, T, and BMI

Abbreviations: PCOS, Polycystic ovary syndrome; BFP, body fat percentage; VAI, visceral adiposity index; SBP, systolic pressure; DBP, diastolic pressure; eGFR, estimated glomerular filtration rate; TC, total cholesterol; LDL-c, low density lipoprotein cholesterol; T, testosterone; BMI, body mass index;

Figures



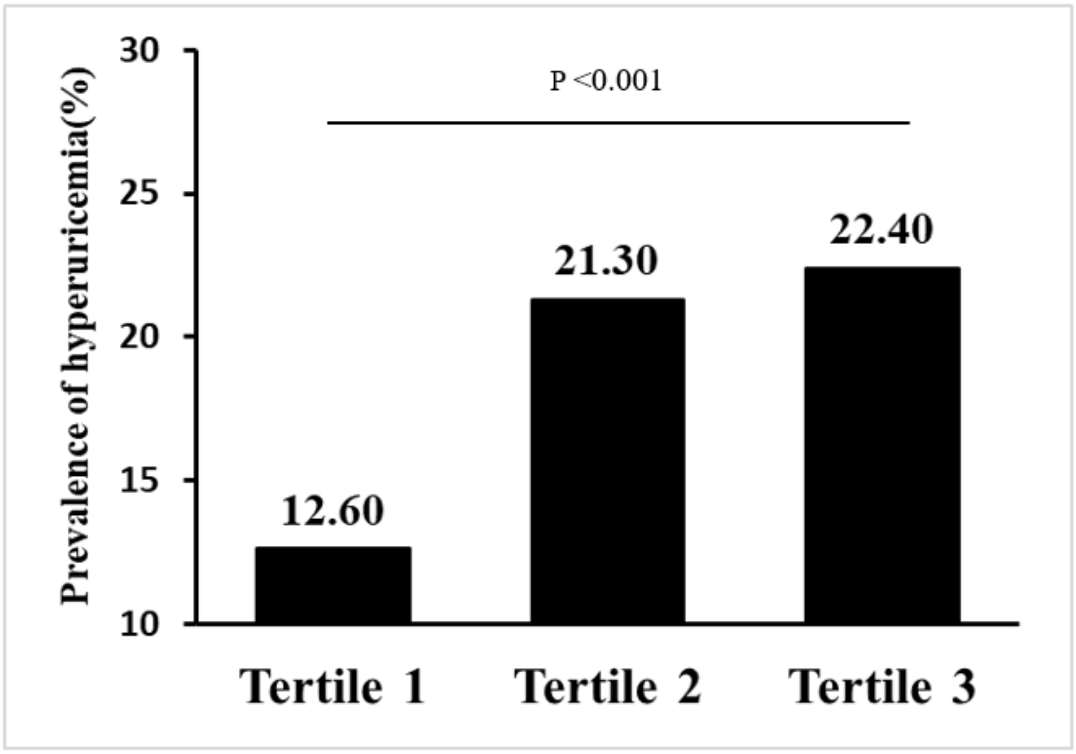


Figure 1

Distributions of serum uric acid levels stratified by VAI tertiles and prevalence of hyperuricemia in patients with PCOS. Abbreviations: PCOS, Polycystic ovary syndrome; VAI, visceral adiposity index