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Uric Acid Levels in Individuals with Obesity: Association with Cardiovascular Disease Risk

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ABSTRACT

Introduction: Obesity is one of the main health problems of modern societies. It is known to be one of the leading causes of cardiovascular diseases, as well as diseases such as hypertension and diabetes. Here, we aimed to investigate whether the Framingham Risk Scoring (FRS) system and uric acid (UA) levels can be used as cardiovascular risk markers in individuals with obesity.

Methods: The study included 203 patients with body mass index (BMI) ≥30 kg/m^2, between the ages of 18-65 years, followed up in the obesity outpatient clinic in the last 5 years. Age, gender, chronic diseases, smoking status, systolic/diastolic blood pressure, height, weight, BMI, waist circumference, hip circumference, waist/hip ratio prescribed medications, fasting blood glucose, insulin level, hemoglobin A1c (HbA1c), total cholesterol, triglyceride, high-density lipoprotein cholesterol, low-density lipoprotein (LDL) cholesterol, and serum UA values were recorded from patient files. The FRS was calculated. Results were evaluated using SPSS.

Results: When the two groups were compared based on the median serum UA value of 5.3 mg/dL, those with serum UA <5.3 mg/dL were defined as group 1, and those with serum UA ≥5.3 mg/dL were defined as group 2. When the 2 groups were compared, there was no significant difference between the FRSs. The FRS was correlated with age, height, waist circumference, waist/hip ratio, systolic and diastolic blood pressure, fasting blood glucose, total cholesterol, LDL cholesterol, triglyceride, and insulin levels, and HbA1c level. Serum UA level was correlated with weight, waist circumference, hip circumference, BMI, systolic and diastolic pressure, triglycerides, and insulin levels. UA level was found to be associated with the FRS.

Conclusion: UA levels, in addition to traditional cardiovascular risk markers and scoring systems, can be used to predict cardiovascular risk in individuals with obesity.

Keywords: Obesity, Framingham Risk Score, serum uric acid

Introduction

Heart-related illnesses rank among the top reasons for death globally. It is thought that approximately one-third of the deaths in the world are due to cardiovascular disease (CVD) (1). Obesity is one of the main health problems in modern societies and reaches epidemic rates in many developed countries. It is known to be one of the leading causes of CVD along with many other diseases such as hypertension and diabetes (2). Given the widespread occurrence of CVD, assessing the likelihood of cardiovascular events is crucial for reducing associated deaths and illnesses (3). Framingham Risk Score (FRS) was introduced by Wilson et al. (4) in 1998 and is a risk calculator that determines the 10-year risk of developing CVD. With FRS assessment, 10-year cardiovascular risk can be estimated with 75% accuracy (5).

In humans, uric acid (UA) represents the final product of purine metabolism, whether from dietary sources or endogenous production. The liver synthesizes this compound, which is subsequently eliminated by the kidneys (6). Increased UA levels have been found to be closely associated with diabetes mellitus (DM) (7), metabolic syndrome (8), hypertension (9), and abdominal obesity (10). While the impact of UA levels on CVD development remains debatable, its involvement in inflammatory processes is well-established (11).

Our research sought to examine the correlation between serum UA concentrations and the 10-year CVD risk as calculated by the FRS system in individuals with obesity. Additionally, we aimed to evaluate the potential of UA levels as an indicator of cardiovascular risk in this population.

Methods

The research adhered to the principles outlined in the 1964 Helsinki Declaration. All participants provided their informed consent. The study received ethical clearance from University of Health Sciences Türkiye,

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Patient Population

We conducted a retrospective analysis involving 203 patients who met the inclusion criteria and had been monitored at the obesity outpatient clinic over the past 5 years. The research encompassed individuals aged 18-65, with a body mass index (BMI) ≥30 kg/m^2, who had established records at the obesity outpatient clinic within the last 5 years, possessed complete documentation, had all necessary laboratory values, and attended regular follow-up appointments. We excluded participants diagnosed with chronic renal failure, malignancy, or gout, as well as those taking medications that affect UA metabolism.

We performed a retrospective examination of the follow-up forms for patients monitored at the obesity outpatient clinic. We documented various parameters including age, gender, chronic diseases, smoking status, systolic/diastolic blood pressure, height, weight, BMI, waist circumference, hip circumference, waist/hip ratio, and prescribed medications.

Laboratory Analysis and Framingham Risk Score Calculation

The laboratory data of the patients were obtained from the outpatient clinic records of our hospital's electronic records system. Fasting blood glucose, insulin, hemoglobin A1c (HbA1c), total cholesterol, triglyceride, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, and serum UA values were recorded based on the dates of the first presentation to the obesity outpatient clinic. The FRS system was employed to assess the CVD risks of the patients. The calculation of the total risk score incorporated several factors, including gender, age, smoking status, total cholesterol levels, HDL-C values, systolic blood pressure, other risk factors, and the use of anti-hypertensive medication. For each gender, scores were assigned to these factors based on the Framingham risk table. The overall risk scores were then determined by adding up the individual scores for each risk factor. According to the FRS, those with a score lower than 10 were considered to have a low 10-year cardiovascular risk, those with a score between 10 and 19 were considered to have an intermediate risk, and those with a score greater than 20 were considered to have a high risk.

Statistical Analysis

Mean, standard deviation, median, minimum, maximum, frequency, and ratio values were used in descriptive statistics of the data. The distribution of variables was measured by the Kolmogorov-Smirnov test. Independent samples t-tests, Kruskal-Wallis tests, and Mann-Whitney U tests were used to analyze quantitative independent data. The chi-square test was used to analyze qualitative independent data. The effects were investigated by univariate and multivariate logistic regression. The SPSS 26.0 was used for the analysis. A $p<0.05$ was accepted as the significance level.

Results

The study encompassed 203 participants, comprising 166 women and 37 men. The mean age was 44.9±10.7 years and 166 (81.8%) were female. BMI, waist, and hip circumference measurements and other demographic data are shown in Table 1. Of the participants, 59 (31%) were diabetic and 63 (29.1%) had a diagnosis of hypertension. The mean UA value of the patients was 5.4±1.3 mg/dL. For the entire study population, the UA level had a median of 5.3 mg/dL.

The mean calculated FRS of the study group was 8.8±8.9. When grouped according to 10-year cardiovascular risk estimates, 138 (68%) were in the low-risk group, 46 (22.7%), in the intermediate risk group, and 19 (9.3%) in the high-risk group.

When the two groups were compared based on the median serum UA value of 5.3 mg/dL, those with serum UA <5.3 mg/dL were defined as group 1 and those with serum UA ≥5.3 mg/dL as group 2. When the 2 groups were compared, there was no significant difference between the FRS (7.8±8.2 vs. 9.8±9.5; p=0.105). The female sex ratio was higher in group 1. Height, waist circumference, systolic and diastolic blood pressure, triglyceride, and insulin levels were significantly lower in group 1 than in group 2 (Table 2).

In correlation analysis, FRS was correlated with age, height, waist circumference, waist/hip ratio, systolic and diastolic blood pressure,

Table 1. Demographic and laboratory features of the study population

	Median (minimum-maximum)	Mean
Age (years)	45 (30-71)	44.9±10.7
Female gender (n, %)	166 (81.8)	
Height (cm)	160 (143-193)	161.4±9.6
Weight (kg)	105 (70-170)	106.3±17.1
Waist circumference (cm)	120 (90-158)	119.9±11.9
Hip circumference (cm)	129 (100-158)	129.1±11.9
BMI (kg/m^2)	40 (30-65)	40.5±5.8
Smoking (n)	66 (32.5)	
Hypertension (n)	63 (31.0)	
Diabetes mellitus (n)	59 (29.1)	
Chronic disease (n)	151 (74.4)	
Systolic blood pressure (mmHg)	120 (90-190)	121.5±12.1
Diastolic blood presure (mmHg)	80 (60-190)	78±10.1
Glucose (mg/dL)	101 (71-312)	110.1±33.6
Total cholesterol (mg/dL)	207 (120-370)	210.3±41.5
Triglyceride (mg/dL)	138 (40-1047)	154.1±93.1
LDL cholesterol (mg/dL)	126.8 (14.4-264.4)	130.6±35.3
HDL cholesterol (mg/dL)	47 (25-90)	49.1±11.7
Insulin (mIU/L)	12.4 (0.7-147.5)	15.1±13.2
Uric acid (mg/dL)	5.3 (2.6-9.7)	5.4±1.3
HbA1c (%)	5.8 (4.9-11.1)	6.0±0.9
FRS (%)	5.7 (0.4-47.6)	8.8±8.9
FRS (<10%)	138 (68%)	
FRS (10-19%)	46 (22.7%)	
FRS (>20%)	19 (9.3%)	

BMI: Body mass index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, HbA1c: Hemoglobin A1c, FRS: Framingham Risk Score

fasting blood glucose, total cholesterol, LDL cholesterol, triglyceride, and insulin levels, and HbA1c level (Table 3). Serum UA level was significantly correlated with weight, waist circumference, hip circumference, BMI, systolic and diastolic pressure, triglycerides, and insulin levels (Table 3).

Table 2. Comparison of patient groups according to serum uric acid levels <5.3 (mg/dL) and ≥5.3 (mg/dL)

	Group 1, (UA <5.3)	Group 2, (UA ≥5.3)	p
Age (years)	44.9±10	44.8±11.3	0.687[m]
Female gender (n, %)	93	73	<0.001[x2]
Length (cm)	159.7±8.3	163.1±10.5	0.038[m]
Weight (kg)	103.7±16.5	108.9±17.4	0.034[m]
Waist circumference (cm)	118±11.9	121.7±11.7	0.016[m]
Hip circumference (cm)	128.2±12.1	130±11.8	0.285[m]
Waist to hip ratio	0.92±0.06	0.94±0.06	0.032[m]
BMI (kg/m^2)	40.3±6.0	40.7±5.6	0.457[m]
Smoking (n)	66		0.802[x2]
Hypertension (n)	63		0.477[x2]
Diabetes mellitus (n)	59		0.842[x2]
Chronic disease (n)	151		0.779[x2]
Systolic blood pressure (mmHg)	119.3±12.6	123.7±11.2	0.002[m]
Diastolic blood presure (mmHg)	77.5±13.1	78.5±15.8	0.022[m]
Glucose (mg/dL)	113.1±42.4	107.1±21.4	0.454[m]
Total cholesterol (mg/dL)	208.0±40.5	212.5±42.5	0.443[m]
Triglyceride (mg/dL)	146.0±108.4	162.0±73.2	0.008[m]
LDL cholesterol (mg/dL)	130.8±35.5	130.6±35.3	0.976[m]
HDL cholesterol (mg/dL)	50.0±12.4	48.2±11.0	0.482[m]
Insulin (mIU/L)	13.4±15.7	16.9±10.0	<0.001[m]
Uric acid (mg/dL)	4.4±0.6	6.4±0.9	<0.001[m]
HbA1c (%)	6.1±1.0	5.9±0.8	0.185[m]
FRS (%)	7.8±8.2	9.8±9.5	0.105[m]
FRS (<10%)	72	66	
FRS (10-19%)	23	23	0.242[x2]
FRS (>20%)	6	13	

[m]Mann Whitney U, [x2]Chi-square test, UA: Uric acid, BMI: Body mass index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, HbA1c: Hemoglobin A1c, FRS: Framingham Risk Score

In univariate logistic regression analysis of the patient groups, gender, height, weight, waist circumference, hip circumference, systolic pressure, and insulin value were found to be significantly associated with the prediction of serum UA level.

In the multivariate reduced model, height and systolic pressure values were also significantly associated with the prediction of serum UA level (Table 4).

Discussion

The main conclusion of our study is that UA levels, in addition to traditional cardiovascular risk markers and scoring systems, can be used to predict CVD risk in individuals with obesity. The FRS system was found to correlate with UA levels in the studied population.

Table 3. Evaluation of serum uric acid level and Framingham risk scoring by Spearman correlation analysis

	Framingham Risk Score		Uric acid	
	r	p	r	p
Framingham Risk Score			0.200	0.004
Uric acid	0.200	0.004	1	<0.001
Age (years)	0.780	<0.001	0.030	0.669
Lenght (cm)	-0.141	0.045	0.100	0.157
Weight (kg)	-0.009	0.900	0.229	0.001
Waist circumference (cm)	0.223	0.001	0.227	0.001
Hip circumference (cm)	0.043	0.545	0.149	0.034
Waist to hip ratio	0.251	<0.001	0.114	0.107
BMI (kg/m^2)	0.077	0.278	0.173	0.014
Systolic blood pressure (mmHg)	0.556	<0.001	0.274	<0.001
Diastolic blood presure (mmHg)	0.285	<0.001	0.145	0.039
Glucose (mg/dL)	0.517	<0.001	0.116	0.099
Total cholesterol (mg/dL)	0.413	<0.001	0.001	0.992
HDL cholesterol (mg/dL)	-0.010	0.889	-0.103	0.143
LDL cholesterol (mg/dL)	0.338	<0.001	0.213	0.480
Tryglyceride (mg/dL)	0.442	<0.001	0.213	0.002
Insulin	0.140	0.048	0.321	<0.001
HbA1c	0.540	<0.001	-0.012	0.867

BMI: Body mass index, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HbA1c: Hemoglobin A1c

Table 4. Evaluation of the factors affecting serum uric acid levels of patients by logistic regression analysis

	Univariate model			Multivariate model		
	OR	95% CI	p	OR	95% CI	p
Gender	4.62	1.99-10.70	<0.001	1.046	1,013-1,080	0.005
Lenght (cm)	1.04	1.01-1.07	0.014			
Weight (kg)	1.02	1.00-1.04	0.031			
Waist circumference (cm)	1.03	1.00-1.05	0.028			
Hip circumference (cm)	54.01	1.25-100	0.041			
Systolic BP (mmHg)	1.03	1.01-1.06	0.012	1.039	1,012-1,068	0.005
Insulin	1.03	1.00-1.06	0.049			

OR: Odds ratio, CI: Confidence interval, BP: Blood pressure

Obesity is one of the most important health problems in modern societies and has reached alarming levels in developing countries (12). Abdominal obesity increases the secretion of adipokines and insulin resistance, independent of BMI, and leads to the progression of numerous cardiometabolic risk factors (13). Obesity is not only closely associated with diseases such as hypertension, type 2 DM and metabolic syndrome, but is also an important driving factor for hyperuricemia (14,15).

UA is the last oxidation product of endogenous purine metabolism. It is made in the liver and eliminated in the kidney (6). Increased UA levels have been shown to be associated with hypertension, DM, and endothelial dysfunction, which are important risk factors for atherosclerosis (16). Large-scale studies have shown that serum UA is associated with inflammatory markers such as C-reactive protein (CRP) and interleukins (17). Although UA is known to have an antioxidant effect via scavenging free radicals (18), it is still controversial whether UA itself is a traditional cardiovascular risk factor (6). The potent antioxidant effect of urate occurs only at physiologic concentrations (19). In two separate studies, intravenous infusion of UA was shown to improve endothelial function in type 1 DM (20) and healthy adults (21). A separate meta-analysis demonstrated that elevated UA levels independently increased the likelihood of cardiovascular events, beyond the influence of conventional cardiovascular risk factors (22). In a study by Atar et al. (16), it was shown that UA was directly related to coronary calcium score on computed tomography coronary angiography, and as UA levels increased, calcium score also increased. In a study by Huang et al. (3), it was investigated whether CRP, white blood cells and UA levels differed between genders when determining the risk of cardiovascular events, and it was shown that they could only be used in male individuals for this purpose. In our study, similarly, UA levels associated with FRS and higher in males. This is generally compatible with the uricosuric effect of estrogen (23).

In a study conducted on 4,140 patients belonging to the Third Generation Framingham cohort, it was shown that UA levels were associated with femoral and carotid pulse wave velocity, which is an indicator of vascular stiffness (6). In another study conducted by Viazzi et al. (24) on hypertensive individuals with high risk of DM, the relationship of UA with metabolic syndrome and various cardiovascular risk factors was examined, and it was concluded that mild hyperuricemia was an independent indicator of metabolic syndrome in this patient group. In our study, UA levels were correlated with FRS, which is conventionally accepted as a predictor of cardiovascular events.

In another study, the effect of anti-hyperuricemic treatment on the prevalence of CVD in hypertensive patients was investigated. Among 458 hypertensive patients, some received anti-hyperuricemic therapy in addition to hypertension treatment, while others were given only anti-hypertensive therapy. At the end of the study, an increase in the number and dose of anti-hypertensive drugs was observed in the group of patients who did not receive anti-hyperuricemic treatment accompanied by a significant increase in the prevalence of CVD in this group. For patients whose serum UA was considered a variable factor, the variability was attributed to two mechanisms: the inflammatory response induced by serum UA on the smooth muscle cells in blood vessels, and the oxidative stress resulting from reactive oxygen species (25). In the study published by Li et al. (26), the relationship between serum UA level and all-cause and cardiovascular mortality in an obesity population was examined. In this study, 12,637 participants who met the inclusion criteria were prospectively observed for 15 years. While increased levels of serum UA were linked to mortality from all causes, our study found no significant relationship between these levels and deaths due to cardiovascular issues.

It is not a coincidence that the parameters showing the highest correlation with the FRS system in our study group are age, fasting blood glucose, HbA1c, and systolic blood pressure. The primary parameters constituting the FRS system are age, systolic blood pressure, the presence of diabetes, smoking status, total cholesterol, and low-density cholesterol. It is expected that patients with obesity have high atherogenic parameters. These are also the parameters that show the highest correlation with the Framingham Scoring System. Although UA levels are known to be a risk factor for atherosclerosis, a weak correlation between UA levels and the FRS system was found in our study. This may be due to the relatively small size of our study group or the possibility that patients with obesity might be using medications affecting UA levels for their chronic diseases.

Research has explored the connection between elevated serum UA levels and obesity, with findings indicating that obesity may lead to overproduction or inadequate renal elimination. The accumulation of excessive visceral fat causes a substantial influx of plasma free fatty acids into the portal vein and liver. This process triggers triglyceride synthesis resulting in the production of large quantities of UA through the activated UA synthesis pathway (27,28). A separate investigation conducted by Zeng et al. (29) tracked 15,959 individuals, over a 9-year period, determining that elevated levels of serum UA correlated with an increase in obesity. This finding lends support to the results of our research.

Study Limitations

Our study was conducted through a retrospective examination of individuals with obesity who presented to a single center over a certain period. Our UA levels are generally below the widely accepted values. Considering all parameters, the median value of the individuals in the study profile was taken as the threshold. The low significance of the values in the correlation analysis is another limitation of the study, which could be due to the limited number of cases. Our study is limited by the absence of extended patient monitoring. The research could have been more impactful in showcasing the outcomes of the FRS if we had been able to present cardiovascular events observed during their long-term follow-up of the cases.

Conclusion

Serum UA measurement is an easily applicable and inexpensive parameter that can be used as a CVD risk marker in obesity, a disease that has most of the classical CVD risk factors. Although there is not yet a scoring system that can use serum UA level for this purpose, it is obvious that this parameter is associated with many metabolic conditions that predispose to CVD. In this respect, it is thought that serum UA level

monitoring, the importance of which is supported by the literature, may contribute to preventive medicine by being put into practical use in predicting CVD risk in population with obesity, which is known to be at cardiovascular risk due to metabolic dysfunctions, as in our study.

Ethics

Ethics Committee Approval: The study received ethical clearance from University of Health Sciences Türkiye, İstanbul Training and Research Hospital (approval number: 1898, date: 28.06.2019).

Informed Consent: All participants provided their informed consent.

Footnotes

Authorship Contributions: Surgical and Medical Practices - B.D., F.A., F.S.; Concept - B.D., F.S.; Design - B.D., F.S.; Data Collection or Processing - B.D., F.A.; Analysis or Interpretation - B.D., F.A.; Literature Search - B.D.; Writing - B.D.

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