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Can Uric Acid Be A Marker For Metabolically Unhealthy Obesity in Children and Adolescents?

Ürik Asit Metabolik Sağlıksız Çocuk ve Adolesanlarda Ayırt Edici Bir Marker Olabilir mi?

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ABSTRACT

Objective: In this study it was investigated whether there is a difference between metabolically healthy (MHO) and unhealthy (MUO) obese children regarding laboratory results, especially serum uric acid levels.

Material and Methods: Data of 220 individuals diagnosed with obesity were collected from medical records. Obese individuals without cardiovascular risk factors (fasting serum lipids, blood pressure, and glucose) were classified as MHO (n=124). Individuals meeting one or more criteria of cardiovascular risk factors were classified as MUO (n=96). The control group was composed of 111 healthy individuals.

Results: The mean age of the participants was 12.14±3.28 years, including 44.4% (n=147) were males. While there was statistical significance regarding uric acid between obese and healthy individuals (308.11±71.97 umol/L (5.18±1.21 mg/dl), 251.6±70.78 umol/L (4.23±1.19 mg/dl), Z=6.670, p<0.001, respectively). No statistical significance was found between MHO and MUO groups (302.16±69 umol/L (5.08±1.16 mg/dl), 315.84±74.94 umol/L (5.31±1.26 mg/dl), Z=1.265, p=0.206, respectively). However, uric acid had a significant correlation with many variables, such as weight, height, body mass index, triglyceride, high-density lipoprotein and serum insulin levels, but the strongest being with weight (Spearman r=0.525, p<0.001). Moreover, there was no difference between these 3 groups concerning platelet count, mean platelet volume, and platelet distribution width (H; p, 3.620; 0.164, 1.624; 0.444, and 1.948; 0.378, respectively).

Conclusion: This study showed that uric acid level is higher in obese than healthy controls and the most significant correlation with weight. However, uric acid alone is not a good indicator between MHO and MUO groups.

Key Words: Adolescent, Metabolically Healthy Obesity, Metabolically Unhealthy Obesity, Children, Uric Acid

ÖZ

Amaç: Bu çalışmada metabolik olarak sağlıklı ve sağlıksız obez çocuklar arasında laboratuvar değerlerinin, özellikle serum ürik asit düzeyleri açısından bir fark olup olmadığı araştırılmıştır.

Gereç ve Yöntemler: Obezite teşhisi konulan çocuk ve adolesan 220 kişinin verileri tıbbi kayıtlardan toplandı. Kardiyovasküler risk faktörleri (açlık serum lipidleri, kan basıncı ve açlık glukoz yüksekliği) olmayan obez bireyler sağlıklı obez (n = 124) olarak sınıflandırıldı. Bir veya daha fazla kardiyovasküler risk faktörü kriterini karşılayan kişiler sağlıksız obez (n = 96) olarak sınıflandırıldı. Kontrol grubu 111 sağlıklı bireyden oluşturuldu.

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Bulgular: Çalışmaya alınan olguların yaş ortalaması 12.14 ± 3.28 yıl, %44.4'ü (n = 147) erkekti. Obez bireyler ile sağlıklı kontroller arasında ürik asit düzeyleri arasında istatistiksel anlamlı olarak fark saptandı (sırasıyla, 308.11±71.97 umol/L (5.18±1.21 mg/dl), 251.6±70.78 umol/L (4.23±1.19 mg/dl); Z=6.670, p<0.001), bu fark metabolik olarak sağlıklı ve sağlıksız obezlerde saptanmadı (sırasıyla, 302.16±69 umol/L (5.08±1.16 mg/dl), 315.84±74.94 umol/L (5.31±1.26 mg/dl); Z=1.265, p=0.206). Ancak kilo, boy, vücut kitle indeksi, trigliserid, yüksek yoğunluklu lipoprotein ve insülin serum seviyeleri gibi ürik asit düzeyi üzerine etkili birçok faktör saptandı, fakat en güçlü oranda korelasyon vücut ağırlığı ile ürik asit düzeyi arasında saptandı (Spearman r=0.525, p <0,001). Ayrıca trombosit sayısı, ortalama trombosit hacmi ve trombosit dağılım genişliği açısından bu 3 grup arasında fark yoktu (sırasıyla H; p, 3.620; 0.164, 1.624; 0.444 ve 1.948; 0.378).

Sonuç: Bu çalışma, obezlerde ürik asit düzeyinin sağlıklı kontrollere göre daha yüksek olduğunu, ürik asit düzeyleri ile en güçlü korelasyon gösteren parametrenin kilo olduğunu göstermektedir. Ancak ürik asit tek başına metabolik olarak sağlıklı ve sağlıksız obezlerde iyi bir gösterge değildir.

Anahtar Sözcükler: Adolesan, Metabolik sağlıklı obezite, Metabolik sağlıksız obezite, Çocuk, Ürik asit

INTRODUCTION

Obesity is generally characterized by increased body adipose tissue when energy intake is more than energy expenditure. Excessive body fat in childhood and adolescence initiates the cardiometabolic complication process. At the end of this process, cardiovascular dysfunction (CVD), hypertension, dyslipidemia, and type 2 diabetes develop, if measures are not taken (1,2). However prevention and treatment of obesity are not easy due to its multifactor nature. Therefore, it is a serious public health problem with an increasing frequency.

Despite all this, it does not mean that the risk factors listed above will develop in all obese patients. Those who are metabolically fit despite being overweight or obese are not expected to have a high risk of developing cardiovascular disease or cancer (3). When obesity is classified as metabolically healthy and unhealthy, metabolically healthy obesity (MHO) is less prone to have typical obesity-associated metabolic disorders (4,5). Although waist circumference, fasting glucose levels, blood lipid levels, and hypertension values are used to determine the metabolic condition in obesity (6,7), it is difficult to reach a consensus, especially in children and adolescents.

Sometimes, anthropometric measurements (i.e., body dimensions, such as waist circumference, body mass index (BMI), fatty body mass, and free-fat mass) remain at the research level and are not used frequently in practice (8). That is why studies focus on identifying new biomarkers that can distinguish different phenotypes of obesity to achieve improvements in the early period of life in obese adults, adolescents, and children (6, 8). Recently, biomarkers, such as serum uric acid (UA) are claimed to be related to the development of metabolic syndrome, type 2 diabetes mellitus, and cardiovascular events in metabolically unhealthy obesity (MUO) in children and adolescents (6). However, it is emphasized that more studies are needed on this subject.

On the other hand, as the platelet count and mean platelet volume (MPV) play a role in the development of vascular inflammation and atherosclerosis, they have been researched in both children and adults (9-11). Increased MPV may be a possible cause of increased cardiovascular risk in obese patients. Studies are supporting this opinion, as well as research showing that there is no difference between healthy and obese children (9-12)

This study aimed to determine serum UA levels in both children and among obese individuals with MHO and MUO as well as normal healthy children to investigate its potential use as a predictor marker.

MATERIAL and METHODS

The data of this descriptive cross-sectional study were obtained from the records of the cases followed up in the pediatric endocrinology department between 01 January, 2017 and 01 February, 2020. The study approval was obtained from local ethics committee (2020/14-165). Written consent for participation was obtained from adolescents and their parents. Study reporting was done per the STROBE guideline.

The hospital where this study is done is the largest hospital in its region with a capacity of 1090 beds, of which 128 are in the intensive care unit. The department of pediatrics has 2nd and 3rd-level pediatric intensive care, pediatric palliative care, and 2nd and 3rd-level neonatal intensive care units.

Children aged 6-18 years were included in this study. Those with a body mass index above the 95th percentile were considered obese. Obese individuals were divided into two categories as metabolically healthy obese (MHO) and metabolically unhealthy obese (MUO) according to the following criteria: Dyslipidemia, hypertension and high fasting blood glucose were used as metabolic syndrome marker, individuals meeting one or more of these criteria were classified as MUO (6). i) Dyslipidemia was defined as high triglycerides (TG) and low HDL (TG level was defined as higher than the 95% percentile according to age and gender, while HDL level was determined as below 5% percentile by age and gender, according to the American Academy of Pediatrics) (13), ii) hypertension (Hypertension was defined as a blood pressure value higher than the 95th percentile according to age, gender and height) (14), iii) impaired fasting glucose (IFG) (defined as fasting serum glucose ≥100 mg/dl (6). While obese children with at least one of the cardiovascular risk

Table I: Metabolic healthy status.

Criteria	Cardiovascular risk factors (CRF)	MHO	MUO
	Hypertension SBP ≥ 95th percentile or DBP ≥ 95th percentile	No CRF	< 2 of the CRF
	Dyslipidemia TG ≥ 95th percentile or HDL< 5th percentile		
	Impaired fasting glucose ≥ 100 mg/dL		

MHO: *Metabolically healthy Obesity,* ***MUO:*** *Metabolically unhealthy Obesity,* ***SBP:*** *Systolic blood pressure,* ***DBP:*** *Diastolic blood pressure,* ***TG:*** *Triglycerides,* ***HDL:*** *High density cholesterol*

factors were classified as MUO, those without hypertension, dyslipidemia, and IFG were classified as MHO (6). This situation is summarized in table I.

The primary outcome of the study was uric acid levels. The other studied variables were age, sex, puberty period (prepubertal/pubertal/postpubertal), weight (kg), height (cm), weight standard deviation (SD) score, height SD score, BMI, BMI SD score, alanine transaminase (ALT) (U/L), aspartate transaminase (AST) (U/L), TG (mg/dl), high-density lipoprotein (HDL) (mg/dl), systolic blood pressure (mmHg), diastolic blood pressure (mmHg), low-density lipoprotein (LDL) (mg/dl), cholesterol (mg/dl), glucose (mg/dl), Homeostasis Model Assessment-Insulin Resistance (HOMA-IR), insulin (µU/ml), uric acid (mg/dl), albumin (g/dl), glycated hemoglobin (HbA1c), hemoglobin (g/dl), hematocrit (%), WBC (µl/ml), thrombocyte (x10^9/l), mean platelet volume (fl), and platelet distribution width (PDW) (%). The parameters which were measured as conventional units were converted to SI units.

A SECA stadiometer (Seca, 216, Hamburg, Germany) and the Charder Medical MS-3400 digital 300 kg×100g (Charder Electronic, Taiwan) were used to measure height and weight, respectively. The Turkish national database (www.ceddcozum.com) was used to determine standard deviation scores (SDS) for anthropometric measurements, such as height, weight, and BMI (15). Normal and obese individuals were classified according to BMI. Those below the 10th percentile (-1.23 SDS) were classified as weak, those in the 10-85th percentile (-1.23 and 1.04 SDS) as normal, those in the 85-90th percentile (1.04-1.65 SDS) as overweight, and those above the 95th percentile (1.65 SDS) as obese. Pubertal development was assessed according to the definition of the Tanner stage of breast development in girls and genital stage in boys. Pubertal development was divided into 3 pubertal stages (Tanner stage I was classified as prepubertal; Tanner phase II, III, and IV as pubertal; Tanner stage V as postpubertal) (16).

After at least 10 hours of fasting, complete blood count, blood sugar, insulin, uric acid, lipid profiles (HDL, LDL, cholesterol, TG), and liver function tests (ALT, AST) were evaluated as part of the routine examination process in the hospital. Biochemical parameters were measured with the Abbott Architect 1600 clinical chemistry analyzer (Abbott Diagnostics, Abbott Park, IL, USA). Complete blood counts were measured using a Sysmex XP 100 automatic counter (Sysmex Corp., Hyogo, Japan).

Insulin resistance (HOMA-IR) was calculated according to the following formula: fasting insulin (µU/ml)×fasting glucose (mg/dl)/405 (17). Triglyceride, total cholesterol, and HDL-cholesterol levels were determined via a Coulter AU 5800 (Beckman Coulter, Inc., California, USA) analyzer, using enzymatic end-point analyses, enzymatic cholesterol esterase-cholesterol oxidase-peroxidase assay, and immunoinhibition assay, respectively. LDL cholesterol was calculated by Friedewald's Formula (18). SI units were obtained by converting from conventional units.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS for Windows, Version 25.0, Chicago, IL, USA) program was used for statistical analysis. Numerical variables were presented as mean and standard deviations, while categorical variables as

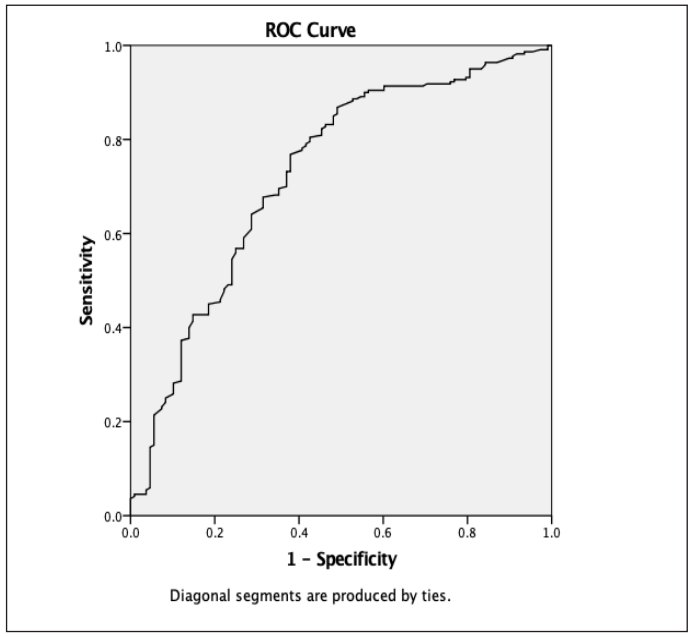

Figure 1: ROC analysis of uric acid in predicting the presence or absence of obesity.

ROC analysis demonstrated an area under the curve of 0.727 for uric acid in predicting the presence or absence of obesity). A cut off point of 4.32 mg/dl provided a sensitivity of 76.8%, a specificity of 62.0%, and a likelihood ratio of 2.0.

frequency and percentage. Normal distribution was evaluated by the Kolmogorov-Smirnov test. Multiple group comparisons were performed via the Kruskal-Wallis test, while binary group comparisons were conducted with the Mann-Whitney U test. However, categorical variables were compared using the Chi-square test. Relationships between variables were analyzed with Spearmen's rho test. ROC analysis was performed to check for the predictive capacity of uric acid in determining obesity (Figure 1). A p-value of <0.05 was considered sufficient for statistical significance.

RESULT

The mean age of the participants was 12.14±3.28 years. When the demographic characteristics of the participants were examined, there was no statistical difference between the study groups (Table II-III). When the laboratory findings of the groups were examined, metabolically unhealthy children had the highest BMI and uric acid (H=195.393, p<0.001; H=45.869, p<0.001, respectively (Table III). The difference between the groups was originating from the obese and control groups

Table II: Comparison of the characteristics of the participants.

	MHO*	MUO**	Control***	χ^2	p
	n (%)	n (%)	n (%)		
Sex					
Male	53 (43.1)	46 (47.9)	48 (43.2)	0.623	0.745
Female	70 (56.9)	50 (52.1)	63 (56.8)		
Puberty					
Prepubertal	40 (32.3)	32 (33.3)	35 (31.5)	0.656	0.959
Pubertal	44 (35.5)	30 (31.3)	36 (32.4)		
Postpubertal	40 (32.3)	34 (35.4)	40 (36.0)		

: Consists of 124 cases, **: Consists of 96 cases, *: Consists of 111 cases, **χ^2:** Chi-Square test value, **MHO:** Metabolically healthy obese, **MUO:** Metabolically unhealthy obese.*

Table III: Comparison of the groups regarding laboratory findings and anthropometric measurements.

	Groups*						H	p
	MHO		MUO		Control			
	Mean	SD	Mean	SD	Mean	SD		
Age	12.29	3.28	12.09	2.73	12.04	3.71	0.396	0.820
Weight SD score	2.66	0.81	2.77	0.89	0.08	0.55	220.557	<0.001
Height SD score	0.64	1.13	0.70	1.18	-0.09	0.78	37.268	<0.001
BMI	28.78	4.77	29.37	4.56	19.40	3.21	195.393	<0.001
BMI SD score	2.48	0.50	2.57	0.50	0.16	0.46	221.672	<0.001
Systolic BP (mmHg)	106	10	108	12	107	6	0.591	0.744
Diastolic BP (mmHg)	69	6	69	7	67	6	2.602	0.272
Glucose (mg/dl)	86.00	8.00	87.00	7.00	85.00	8.00	1.373	0.503
ALT (U/L)	21.00	9.00	22.00	11.00	19.00	6.00	3.841	0.147
AST (U/L)	22.00	6.00	22.00	7.00	23.00	8.00	2.359	0.308
Triglyceride (mmol/L)	0.98	0.27	1.89	0.87	0.91	0.3	138.979	<0.001
HDL (mmol/L)	1.32	0.28	1.1	0.23	1.26	0.29	34.776	<0.001
LDL (mmol/L)	2.54	0.6	2.75	0.6	2.23	0.67	35.047	<0.001
Cholesterol (mmol/L)	4.3	0.65	4.69	0.75	3.86	0.78	53.025	<0.001
Uric acid (umol/L)	302.26	69.02	315.95	74.97	251.69	70.8	45.869	<0.001
HbA1c (%)	5.47	0.34	5.53	0.40	NA	NA	0.463	0.496
Thrombocyte (x10^9/L)	345153	74289	344937	63317	332928	79018	3.620	0.164
MPV (fl)	10.11	1.03	9.98	0.97	10.04	0.85	1.624	0.444
PDW (%)	11.69	2.00	11.61	1.93	11.31	1.69	1.948	0.378

: The MHO group consists of 124 and the MUO group consists of 96 people, while the control group consists of 111 subjects, **H: Kruskal-Wallis test value, **SD:** Standard deviation, **MHO:** Metabolically healthy obese, **MUO:** Metabolically unhealthy obese, **BP:** Blood pressure, **MPV:** Mean platelet volume, **PDW:** Platelet distribution width.*

Table IV: Distribution of the numerical variables between the obese and control groups.

	Group*	Mean	SD	Z	p
Weight SD score	Obese Control	2.71 0.08	0.85 0.55	14.849	<0.001
Height SD score	Obese Control	0.67 -0.09	1.15 0.78	6.103	<0.001
BMI	Obese Control	29.03 19.40	4.67 3.21	13.954	<0.001
BMI SD score	Obese Control	2.52 0.16	0.50 0.46	14.855	<0.001
Triglyceride (mmol/L)	Obese Control	1.38 0.91	0.76 0.3	6.570	<0.001
HDL (mmol/L)	Obese Control	1.22 1.26	0.28 0.29	0.920	0.357
LDL (mmol/Ll)	Obese Control	2.63 2.22	0.6 0.68	5.384	<0.001
Cholesterol (mmol/L)	Obese Control	4.47 3.88	0.72 0.78	6.230	<0.001
Uric acid (umol/L)	Obese Control	308.21 251.69	72 70.8	6.670	<0.001

Z: *Mann-Whitney U test value,* ***SD:*** *Standard deviation,* ***HDL:*** *High-density lipoprotein,* ***LDL:*** *Low-density lipoprotein,* ***BMI:*** *Body-mass index,* ***HOMA:*** *Homeostatic model assessment insulin resistance,* ****:*** *Obese group consists of 220, control group consists of 111 subjects*.

(Table IV). When the MHO and MUO groups were compared, no statistically significant difference was found regarding either metabolic outcomes or anthropometric characteristics (Table V). It was determined that uric acid had a significant correlation with many variables, the strongest being with weight (r= 0.525, p<0.001) (Table VI). A uric acid cut off point of 256.95 umol/L (4.32 mg/dl) provided a sensitivity of 76.8%, a specificity of 62.0%, and a likelihood ratio of 2.0 to predict the presence of obesity (Figure 1).

DISCUSSION

Prevention of obesity is as crucial as the treatment of accompanying metabolic problems. For this reason, the focus has been on the parameters that can be used to distinguish between MUO and MHO status in obesity beginning in early childhood. In this context, measurements of body dimensions, radiological imaging methods, and biochemical parameters, such as uric acid, have been investigated (6,19-21). Furthermore, it has been argued that genetic factors play a role in the development of MHO and MUO phenotypes. The relationship between genetic loci and BMI was shown in some studies (22,23). While some of these parameters are only at the research level, uric acid levels seem to be the most useful. Measuring UA levels require no extra equipment, and the results are easy to interpret. UA, correlated with BMI, is higher in obese and lower in normal-weight individuals. However, the question to be addressed is whether uric acid levels differ in obese individuals depending on their metabolic health status.

Some observations emphasize that UA protects erythrocytes as an antioxidant. It is argued whether it is responsible for the increase in plasma antioxidant activity in healthy individuals (8,24,25). In contrast, serum uric acid has been associated with cardiovascular risk, particularly by causing endothelial damage through decreased nitric oxide production and anti-proliferative effects on the endothelium (8,26). In adipose tissue cells, uric acid induces intracellular reactive oxygen species production and reduces nitric oxide bioavailability (26-28). Considering that oxidative stress in adipocytes plays an essential role in the development of insulin resistance, increased oxidative stress due to hyperuricemia may play a role in the development of these dysregulations. If uric acid is considered to play a crucial role in developing metabolic syndrome, it is essential to identify factors that increase serum uric acid levels.

While uric acid has been investigated more in adult obese individuals, studies in children and adolescents are limited. A recent study conducted on children and adolescents showed that MHO people had higher uric acid levels than healthy individuals, and MUO had higher uric acid levels than MHO (6). However, it is certain that more research is certainly needed in this area. Besides several parameters, such as high visceral fat stores, waist circumference, adiponectin, insulin resistance, nuchal subcutaneous adipose tissue thickness, and arteriosclerosis of the carotids, it has been emphasized that uric acid level can be used in the differentiation of MUO and MHO (6,19,20). Similarly, without distinction of healthy or unhealthy metabolic status, obese and healthy children were compared for uric acid levels, and high uric acid was detected in obese individuals (29). In this study, although high uric acid levels were found in obese children than controls, no statistical significance

Table V: Comparison of metabolic healthy and unhealthy obese children concerning the metabolic outcomes.

	Group*	Mean	SD	Z	p
Weight SD score	MHO MUO	2.66 2.77	0.81 0.89	0.334	0.738
Height SD score	MHO MUO	0.64 0.70	1.13 1.18	0.096	0.923
BMI	MHO MUO	28.78 29.37	4.77 4.56	1.041	0.298
BMI SD score	MHO MUO	2.48 2.57	0.50 0.50	1.502	0.133
Triglyceride (mmol/L)	MHO MUO	0.98 1.89	0.27 0.87	10.197	<0.001
HDL (mmol/L)	MHO MUO	1.32 1.1	0.28 0.23	5.808	<0.001
LDL (mmol/L)	MHO MUO	2.54 2.74	0.59 0.59	2.705	0.007
Cholesterol (mmol/L)	MHO MUO	4.3 4.7	0.66 0.75	4.221	<0.001
Insulin (µU/ml)	MHO MUO	15.32 16.61	12.53 10.24	1.382	0.167
HOMA IR	MHO MUO	3.34 3.63	2.99 2.44	1.445	0.149
Uric acid (umol/L)	MHO MUO	302.26 315.95	69.02 74.97	1.265	0.206

Z: *Mann-Whitney U test value,* ***SD:*** *Standard deviation,* ***MHO:*** *Metabolically healthy obese,* ***MUO:*** *Metabolically unhealthy obese.* ****:*** *MHO group consists of 124, MUO group consists of 96 subjects.*

Table VI: Relationship of uric acid with anthropometric and laboratory findings.

	Uric acid	
	r	p
Age	0.377	<0.001
Weight	0.525	<0.001
Weight standard deviation score	0.331	<0.001
Height	0.460	<0.001
Height standard deviation score	0.040	0.472
Body mass index	0.493	<0.001
Body mass index standard deviation score	0.366	<0.001
Triglyceride	0.292	<0.001
High-density lipoprotein	0.228	<0.001
Systolic blood pressure	0.282	<0.001
Diastolic blood pressure	0.273	<0.001
Low-density lipoprotein	0.139	0.119
Homeostatic model assessment insulin resistance	0.107	<0.001

r=Spearman r

was seen between MHO and MUO. According to previous studies (6,19,20), there is a statistical significance regarding BMI SDS between MHO and MUO groups. There is a strong correlation between BMI SDS and UA, which is emphasized in the mentioned studies. Beyond other relationships between UA and the metabolic and anthropometric variables, this correlation was observed in this our study too. Although the serum uric acid level in children correlates with many factors associated with obesity, it is not much useful in predicting metabolic syndrome (30). However, our study revealed that serum uric acid levels can be a useful marker for predicting the presence of obesity.

To study the relationship between UA and obesity, not only BMI but also body compositions, such as body fat mass and free-fat mass, should be taken into account. The amount of change in body dimensions related to UA and its contribution to the persistence and aggravation of metabolic syndrome should be better explored, which is not an easy task. Furthermore, due to controversies regarding the definition of metabolic syndrome and the lack of consensus thresholds for the single components in children and adolescents (6,31), there is no internationally accepted diagnostic pathway for metabolic syndrome available. To avoid conflicts concerning the definition of metabolic syndrome, much clearer, and practically applicable guidelines are needed.

In addition to hypercholesterolemia, diabetes mellitus and hypertension, MPV as platelet function and activation, is increased in certain vascular disease and in obese individuals (32-34). Platelet activation may play a central role in the development of cardiovascular disease (34). Besides, in patients with cerebral stroke, an increased MPV has been observed signaling increased platelet activity (32). It has been reported that patients with metabolic syndrome have increased MPV values and thus, have an increased cardiovascular risk (35). However, there is limited information about the relationship between MPV levels in obese patients. We found no difference between obesity and normal healthy individuals, as did other

similar studies (10,12,36). Therefore, we conclude that more studies are needed on this subject.

Study Limitations

The lack of information about the duration of obesity and the absence of data on the follow-up findings can be considered as limitations of this study. On the other hand, this is a retrospective file-based study. Inclusion of laboratory results on some markers such as adiponectin, visceral fat stores, and atherosclerosis could provide further inputs about metabolism and obesity.

CONCLUSION

This study showed that uric acid plays an essential role in obesity, increases significantly in obese children, and is associated with many obesity-related factors. It has been evaluated that serum uric acid levels can be used as an indicator of obesity, even in early adolescence and childhood. However, no statistically significant difference was found between metabolically healthy and unhealthy obese children. This may be attributed to the duration of obesity. Further studies, including information about follow-up findings of the patients, are required to clarify the relationship of UA with other metabolic indices.

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