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Serum uric acid and leptin levels in metabolic syndrome: a quandary over the role of uric acid

Jen-Der Lin[a,*], Wen-Ko Chiou[b], Hung-Yu Chang[a], Feng-Hsuan Liu[a], Hsiao-Fen Weng[a]

[a]*Division of Endocrinology and Metabolism, Department of Internal Medicine, Chang Gung Memorial Hospital, Chang Gung University, Kweishan, Taoyuan Hsien, Taiwan 333, ROC*

[b]*Department of Industrial Design, Chang Gung University, Kweishan, Taoyuan Hsien, Taiwan 333, ROC*

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Abstract

This study investigates the impact of uric acid (UA) on the risk factors associated with metabolic syndrome. In addition, this study explores the relationship between UA and insulin resistance and serum leptin levels in metabolic syndrome. A total of 470 subjects (252 women and 218 men) were recruited from the Department of Health Management at Chang Gung Medical Center (Linkou, Taiwan). Metabolic syndrome was defined using a modified Adult Treatment Panel III (ATP III) definition. The formula for the homeostasis model assessment of insulin resistance (HOMA-IR) is as follows: fasting serum insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5. Diabetes mellitus was diagnosed in 45 subjects (9.6%); 82 subjects (17.4%) had hypertension. Hyperuricemia was diagnosed in 144 subjects (30.6%). Of these subjects, 115 (63 females and 52 males) (24.5%) were diagnosed as having metabolic syndrome. Patients with hyperuricemia had increased body mass index, waist-to-hip ratio, and triglyceride (Tg) level. The subjects also had lower high-density lipoprotein and greater hypertension. Hormone assays showed an elevation of leptin, immunoreactive insulin (IRI), and HOMA-IR in the hyperuricemia group. Uric acid appeared to be better correlated with Tg, blood pressure (both systolic and diastolic), obesity, immunoreactive insulin, and HOMA-IR. Uric acid did not correlate with leptin or blood glucose levels. Metabolic syndrome and Tg/high-density lipoprotein ratio showed a statistically significant difference in HOMA-IR using 3.8 as a cutoff value. Otherwise, there was no difference in leptin value. In conclusion, serum UA is significantly related to risk factors of metabolic syndrome except for blood glucose. Waist-to-hip ratio and HOMA-IR were statistically different in subjects with and without metabolic syndrome.

1. Introduction

Serum uric acid (UA) is an important factor in cardiovascular events, but its role in metabolic syndrome is controversial [1,2]. Factors such as age group, ethnic population, and sex illustrate the importance of serum UA levels in metabolic syndrome [3,4]. Genetic characteristics, body mass structure, aging, and renal disposal ability influence serum UA levels. A recent familial clustering study of metabolic syndrome showed a significant genetic correlation between UA and body mass index (BMI), waist circumference, high-density lipoprotein (HDL) cholesterol, triglyceride (Tg), and plasminogen activator inhibitor 1 antigen [5]. The present study was conducted to investigate the impact of UA on various risk factors associated with metabolic syndrome. In addition, we examined the relationship between serum leptin levels and UA in metabolic syndrome in Chinese subjects affected by insulin resistance.

2. Subjects and methods

2.1. Subjects

A total of 470 subjects, including 252 women (mean age ± SD, 53.7 ± 12.2 years) and 218 men (mean age ± SD, 54.6 ± 13.5 years), were randomly selected from the health examination unit of the Chang Gung Medical Center in Linkou, Taiwan. All of the subjects were Chinese residents of Taiwan. Blood tests, body scanning, and evaluation of anthropometric measurements were performed on all subjects.

2.2. Metabolic syndrome definition

Blood pressure levels were classified according to the 1999 World Health Organization-International Society of Hypertension guidelines [6]. *Hypertension* was defined as a

* Corresponding author. Tel.: +886 3 3281200; fax: +886 3 3288257.
E-mail address: einjd@adm.cgmh.org.tw (J.-D. Lin).

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systolic blood pressure (SBP) of ≥140 mm Hg and/or a diastolic blood pressure (DBP) of ≥90 mm Hg. *Diabetes mellitus* (DM) was defined as a fasting glucose of ≥126 mg/dL or a postprandial glucose of ≥200 mg/dL. Metabolic syndrome was defined using a modified ATP III definition issued in 2004 by the Bureau of Health Promotion, Department of Health, ROC (Taiwan). Subjects meeting 3 or more of the following criteria were defined as having metabolic syndrome: central obesity with a waist circumference of more than 90 cm in males or more than 80 cm in females and/or BMI of ≥27 kg/m^2, hypertriglyceridemia (≥150 mg/dL or 1.695 mmol/L), low HDL cholesterol (<40 mg/dL or 1.036 mmol/L in males and <50 mg/dL or 1.295 mmol/L in females), high blood pressure (≥130/85 mm Hg), and high fasting glucose (≥110 mg/dL or ≥6.1 mmol/L).

2.3. Measurements

Venous blood was sampled after an overnight fast and centrifuged at 3000 rpm for 30 minutes at 4°C. Uric acid was detected in blood samples by using a colorimetric enzymatic method. Hyperuricemia was defined in males as more than 7.0 mg/dL (416.4 μmol/L) and in females as more than 6.0 mg/dL (356.9 μmol/L) [7]. Cholesterol and Tg levels were measured using enzymatic procedures on an Abbott VP (Abbott Laboratories, Abbott Park, IL) [8,9]. Serum lipoprotein cholesterol levels were analyzed by a combination of heparin-calcium precipitation and agar-agarose gel electrophoresis procedures [8]. Plasma glucose levels were measured by an enzymatic method using the Beckman Instant Glucose Analyzer (Beckman Instruments, Palo Alto, CA). Human immunoreactive insulin (IRI) and human leptin were evaluated with the Linco Research RIA kit (Linco Research, St Charles, MO). The formulas for the homeostasis model assessment of insulin resistance (HOMA-IR) and beta-cell function (HOMA beta-cell) are as previous described [10]. The *HOMA-IR* was defined using the following formula: fasting serum insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5. Anthropometric measurements were performed to determine BMI and waist-to-hip ratio (WHR) using a 3-dimensional scanner [11]. The results were correlated with IRI, leptin, blood pressure, blood glucose, lipid, and UA levels. The health index (HI) was determined using the following formula (as previously described [11]):

$$(\text{body weight} \times 2 \times \text{waist area})/(\text{body height}^2) \times (\text{breast area} + \text{hip area})$$

The HI combined the parameters of BMI, WHR ratio, and circumference of the chest girdle. This equation was proposed after calculation with trial and error to provide the best correlation with clinical biochemical data. Subjects were excluded from the study if they had an obvious disease and/or a body weight change greater than 10% during the last 6 months. The study protocol was approved by the institutional review board at Chang Gung Memorial Hospital (Taoyuan Hsien, Taiwan).

2.4. Statistical analysis

All data analyses were performed using the SAS software [12]. Associations between individual pairs of

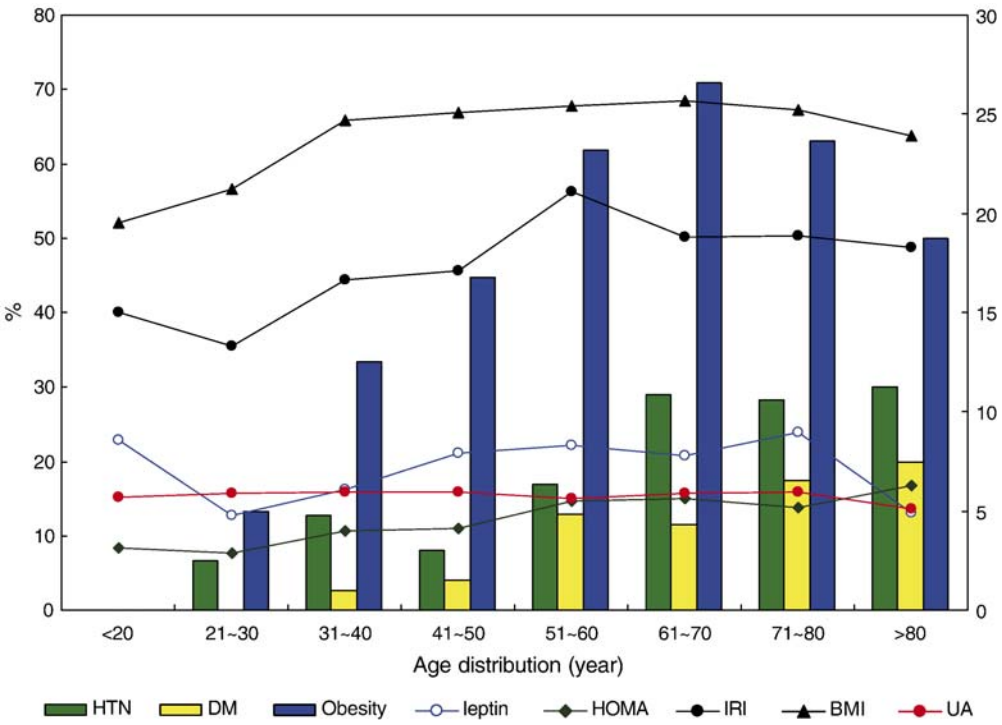

Fig. 1. The incidence of hypertension, DM, central obesity, leptin, HOMA-IR, IRI, UA, and BMI values in study patients are shown.

Table 1
Clinical features of the subjects in hyperuricemia and those with normal serum UA

	Normal UA (n = 326)	Hyperuricemia (n = 144)	*P*
Sex (female/male)	175/151	77/67	.9666
Age (y)	54.6 ± 12.6	53.1 ± 13.1	.2659
Obesity (yes/no)	168/162	98/46	.0004
BMI (kg/m^2)	24.4 ± 0.2	26.6 ± 0.3	.0001
WHR	0.89 ± 0.01	0.93 ± 0.01	.0001
Fasting glucose (mg/dL)	104.5 ± 2.3	105.1 ± 5.9	.1990
Postprandial glucose (mg/dL)	117.1 ± 3.9	115.4 ± 7.8	.8363
DM (yes/no)	35/291	10/134	.1978
Hypertension (yes/no)	44/282	38/106	.0007
SBP (mm Hg)	119.9 ± 1.0	125.8 ± 1.7	.0020
DBP (mm Hg)	75.3 ± 0.6	79.0 ± 1.0	.0009
Tg (mg/dL)	121.5 ± 4.2	166.4 ± 11.6	.0001
HDL cholesterol (mg/dL)	56.1 ± 0.8	51.5 ± 1.0	.0013
Total cholesterol (mg/dL)	201.1 ± 2.3	206.4 ± 3.5	.1990
TG/HDL (<3.8/≥3.8)	273/53	103/41	.0023
Leptin (ng/mL)	7.0 ± 0.3	9.6 ± 0.7	.0001
IRI (ng/mL)	16.2 ± 0.4	24.4 ± 4.0	.0030
HOMA-IR	4.3 ± 0.2	6.4 ± 1.0	.0032
Nonmetabolic syndrome/metabolic syndrome	263/63	92/52	.0001

Obesity denotes those meeting the criteria of metabolic syndrome as discussed in the text.

risk variables were examined using partial Pearson product moment correlation coefficients. All statistical analyses were carried out separately for men and women and for different age groups. Subjects taking antihypertensive medications were considered to have high blood pressure. Pearson product moment correlation coefficients were used to adjust for sex and age, between risk variables and by age group.

3. Results

3.1. Metabolic parameter values in study subjects

Of the 470 subjects, 115 (63 females and 52 males) (24.5%) were diagnosed as having metabolic syndrome. Forty-five (9.6%) subjects were diagnosed as having DM; 82 (17.4%) had hypertension. Hyperuricemia was identified in 144 subjects (30.6%), 30.7% (67/218) of the male subjects and 30.6% (77/252) of the female subjects. The incidence of hypertension, DM, central obesity, as well as the values of leptin level, HOMA-IR, IRI, UA, and BMI are shown in Fig. 1. The incidence of hypertension and DM increased gradually after the age of 50 years. After the age of 70 years, BMI and obesity decreased. A statistically significant difference in leptin levels was noted between male (mean, 4.9 ± 0.3 ng/mL; range, 0.9-28.7 ng/mL) and female (mean, 10.3 ± 0.4 ng/mL; range, 1.6-50.5 ng/mL) subjects (P = .0001). Further analysis of risk factors associated with metabolic syndrome in both sexes showed that females had significantly more central obesity and lower HDL levels. Men, however, exhibited more elevated blood pressure.

3.2. Correlation between UA levels and other clinical parameters

The clinical features of subjects with hyperuricemia and normal serum UA are presented in Table 1. The hyperuricemia group showed increases in BMI, WHR, Tg level, Tg/HDL ratio, lower HDL, and a greater metabolic syndrome and hypertension ratio. Hormone assays showed elevations in leptin, IRI, and HOMA-IR in the hyperuricemia group. In contrast, the ratio of DM to blood glucose revealed no significant difference. Statistical significance was noted in

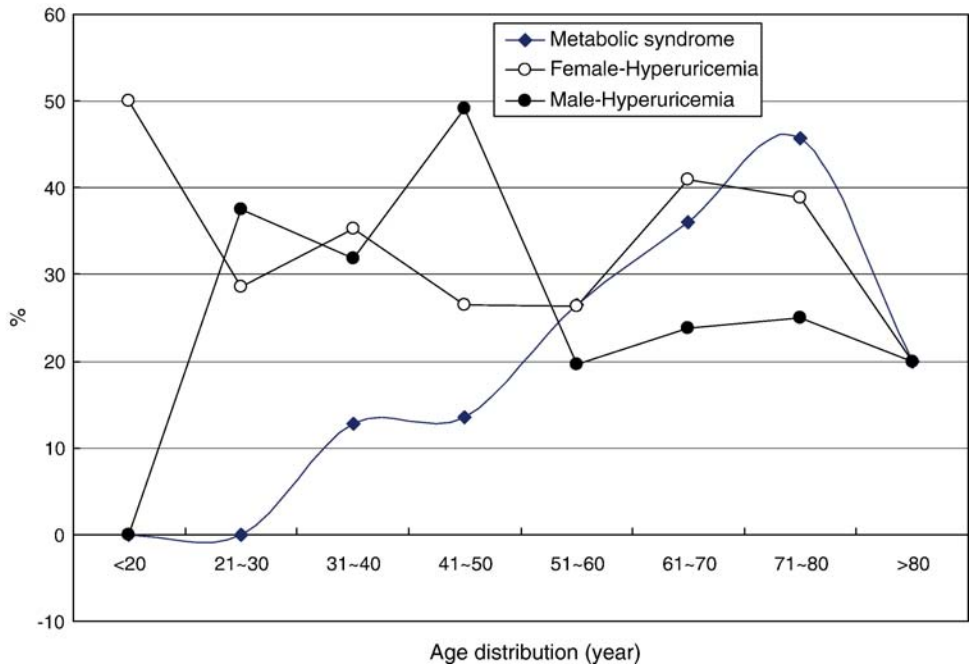

Fig. 2. The percentage of hyperuricemia in both sexes and the ratio of metabolic syndrome in different age ranges are shown in the diagram.

Table 2
Simple linear regression analysis in different sexes, with UA as a dependent variable

	Female		Male	
	r	*P*	*r*	*P*
Age (y)	0.135	.032	−0.189	.0051
Tg (mg/dL)	0.224	.003	0.029	<.0001
Fasting glucose (mg/dL)	0.074	.2402	−0.103	.1286
Postprandial glucose (mg/dL)	0.043	.4923	−0.135	.0461
HDL (mg/dL)	−0.217	.0005	−0.020	.7711
HOMA-IR	0.246	.0001	0.072	.2916
SBP (mm Hg)	0.234	.0002	0.130	.0549
DBP (mm Hg)	0.190	.0025	0.265	.0001
IRI	0.145	.0210	0.209	.0019
Leptin (ng/mL)	0.200	.0014	0.250	.0002
Height (cm)	−0.009	.8880	0.0741	.2762
Weight (kg)	0.351	<.0001	0.376	<.0001
BMI (kg/m^2)	0.383	<.0001	0.386	<.0001
Waist circumference (cm)	0.351	<.0001	0.319	<.0001
WHR	0.259	.0001	0.228	.0014

both sexes with hyperuricemia with or without metabolic syndrome (P = .0001). The incidence of hyperuricemia in both sexes and the rate of metabolic syndrome in different age ranges are illustrated in Fig. 2. The curves showed that the incidence of metabolic syndrome increased with hyperuricemia in females older than 50 years. In Table 2, simple linear regression analyses of UA in different sexes were further correlated with other parameters. In both sexes, leptin and anthropometric parameters such as BMI, waist circumference, and WHR were associated with UA levels. Different patterns of association between UA and HDL, age, and HOMA-IR were found in each sex.

3.3. Correlation between HOMA-IR values or leptin levels and other clinical parameters

Analysis of the correlation between HOMA-IR values with different parameters showed that HOMA-IR values were significantly different in subjects with central obesity, DM, and metabolic syndrome. Furthermore, leptin was higher in females and in subjects with central obesity. The Tg/HDL ratio was significantly different between high and low HOMA-IR groups using 3.8 as a cutoff value. Otherwise, there was no difference in leptin values between the groups with and without metabolic syndrome. Further analyses of the groups with and without metabolic syndrome were performed with simple and multiple regressions (Tables 3 and 4). There was a statistically significant difference between subjects with and without metabolic syndrome in UA, WHR, fasting (AC) blood glucose, postprandial (PC) blood glucose, SBP, DBP, Tg, HDL, HOMA-IR, and leptin levels upon simple linear regression study. In multiple regression analysis, the results showed that only WHR, SBP, HDL, Tg, and HOMA-IR values were significantly different between the groups.

4. Discussion

Insulin resistance estimated using HOMA-IR is the main factor to consider in the development of metabolic syndrome and cardiovascular complications [13]. This study confirmed the importance of HOMA-IR as a risk factor of metabolic syndrome. Furthermore, in the present study, subjects with hyperuricemia had a higher HOMA-IR. Analysis of subcutaneous fat obesity and visceral fat obesity by computed tomographic scan showed that visceral fat obesity is linked more closely to the overproduction of UA than to subcutaneous fat obesity [14]. Although no quantitative visceral fat study was performed in this study, WHR and BMI values were obtained. Thus, we conclude that UA level is related to central obesity.

The Framingham studies reported that UA did not have a causal role in the development of coronary heart disease, death from cardiovascular disease, or death from all causes [15]. However, during the last 10 years, epidemiologic studies related to UA and metabolic syndrome, and preliminary animal studies related to vascular endothelial function with hyperuricemia have been published [16]. A recent prevalence study of hyperuricemia in elderly Taiwanese showed that 36% of those older than 65 years had hyperuricemia [17]. This percentage is close to the incidence found in the present investigation. The role of sex is important in hyperuricemia and in metabolic syndrome during aging. In the present study, female subjects after the age of menopause consistently displayed higher

Table 3
Simple linear regression analysis

	r	*P*
UA (mg/dL)	0.206	<.0001
WHR	0.370	<.0001
Fasting glucose (mg/dL)	0.317	<.0001
Postprandial glucose (mg/dL)	0.320	<.0001
SBP (mm Hg)	0.397	<.0001
DBP (mm Hg)	0.299	<.0001
Tg (mg/dL)	0.519	<.0001
HDL cholesterol (mg/dL)	0.331	<.0001
HOMA-IR	0.409	<.0001
Leptin (ng/mL)	0.081	.0781

Dependent variable: nonmetabolic syndrome vs metabolic syndrome.

Table 4
Multiple regression coefficients

	Unstandardized coefficients	Standardized coefficients	*t*	*P*	95% confidence interval for *B*	
					Lower bound	Upper bound
Intercept	−0.922		−4.217	.000	−1.352	−0.492
UA (mg/dL)	−0.005	−0.017	−.411	.682	−0.028	0.018
WHR	0.467	0.093	2.132	.034	0.036	0.897
Fasting glucose (mg/dL)	0.000	0.040	0.353	.724	−0.001	0.002
Postprandial glucose (mg/dL)	0.000	0.040	0.353	.724	−0.001	0.002
SBP (mm Hg)	0.006	0.280	4.727	.000	0.004	0.009
DBP (mm Hg)	−0.001	−0.030	−0.515	.607	−0.005	0.003
HDL cholesterol (mg/dL)	−0.004	−0.138	−3.393	.001	−0.006	−0.002
Tg (mg/dL)	0.001	0.320	7.637	.000	0.001	0.002
HOMA-IR	0.024	0.197	3.198	.001	0.009	0.038
Leptin (ng/mL)	−0.001	−0.011	−0.253	.800	−0.007	0.005

Dependent variable: nonmetabolic syndrome vs metabolic syndrome.

rates of metabolic syndrome and hyperuricemia. These results may explain the increased risk of cardiovascular disease after menopause. In the long-term follow-up of 12 years conducted in 334 white and 243 black subjects in the Bogalusa Heart Study, elevated childhood serum UA levels were associated with increased blood pressure beginning in childhood. These higher blood pressure levels persisted into adulthood [18].

Anthropometric measurements are important factors in the prediction of metabolic syndrome [19]. In the present study, obesity, BMI, waist circumference, and WHR were associated with high serum UA levels. Other indices such as waist-to-height ratio and HI were identified as significant risk factors of metabolic syndrome and high serum UA levels [20,21]. A previous study showed that hyperuricemia was a simple important marker of insulin resistance as measured by using the euglycemic clamp [22]. Uric acid values of females were stable before the age of 18 years and decreased slightly between the ages of 19 and 44 years. Uric acid levels increased in the middle to older age groups [23]. A prospective study of 49 413 Japanese male workers with ages from 25 to 60 years investigated the relationship between hyperuricemia and health hazards [24]. Hyperuricemia showed a strong association with the relative risk of death by all causes including coronary heart disease, stroke, hepatic disease, and renal failure. In the present investigation, hyperuricemia was associated with HOMA-IR index and all the components of the metabolic syndrome, except blood glucose level. Uric acid level was not a significant risk factor of metabolic syndrome upon multiple regression coefficient analysis. Thus, the interrelationship of UA and metabolic syndrome requires more investigation.

The involvement of hyperuricemia in the pathogenesis of hypertension, cardiovascular disease, and renal disease was considered during renal involvement in one study [25]. The action of different cytokines related to metabolic syndrome and UA levels needs to be investigated. Elevated leptin levels were studied as one of the causes of elevated UA levels [26,27]. In a recent study, leptin level was found to be a risk factor of metabolic syndrome [28]. In the present analysis, leptin was significantly elevated in subjects with hyperuricemia. However, unlike the HOMA-IR value, leptin levels were not significantly different in subjects with metabolic syndrome upon multiple regression analysis. Recent data showed that the Tg/HDL ratio can predict low-density lipoprotein cholesterol particle sizes, heart rate recovery, and metabolic syndrome [29,30]. With the use of a cutoff value of 3.8 for the Tg/HDL ratio, HOMA-IR and hyperuricemia were significantly different in subjects with high vs low ratios.

In conclusion, the present study showed that UA levels correlate with most of the risk factors of metabolic syndrome, except blood glucose levels. However, UA did not show an association with metabolic syndrome upon multiple regression analysis. Significantly higher hyperuricemia was observed in subjects with metabolic syndrome. Waist-to-hip ratio and HOMA-IR were significantly different in subjects with and without metabolic syndrome.

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