Association of hyperuricemia with metabolic syndrome among university workers: sex and occupational differences

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Abstract

Background: The relationship between metabolic syndrome (MetS) and hyperuricemia is not fully understood.

Objective: To examine the association of hyperuricemia with MetS and the component of MetS that is mostly influenced by hyperuricemia among university workers.

Methods: Anthropometric measurements, blood pressure, glucose, lipid profiles, renal function tests were measured in 1198 male and 1075 female (22-60 years old) workers on annual medical examination.

Results: Hyperuricemia was 3-fold higher in males (odds ratio, OR, 2.938, 95% confidence interval, CI, 1.909-4.522, P<0.01) than females after adjustment for age, body mass index (BMI) and renal function. Overall, individuals with hyperuricemia were 3.9-fold likely to have MetS OR, 3.903; CI (2.439-6.245), P<0.01, and dyslipidemia, 2.5 times (OR, 2.501; 95% CI, 1.776-3.521, P<0.01) after adjustment for age, BMI, sex and renal function. However, no associations were found in individuals with hypertension (OR, 1.427; 95% CI, 0.996-2.205, P=0.052) and hyperglycemia (OR, 1.476; 95% CI, 0.989-2.202, P=0.057). Administrative work positively associated (OR, 1.895; 95% CI, 1.202-2.925, P<0.05) with hyperuricemia in males and not females.

Conclusion: Male workers with hyperuricemia, especially those working in administration were at risk of metabolic syndrome. It is important to screen, prevent and treat metabolic syndrome in individuals diagnosed with hyperuricemia at the workplace.

Keywords: Hyperuricemia, metabolic syndrome, uric acid, workers.

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Introduction

Metabolic syndrome (MetS) is a known risk factor for many chronic diseases including cardiovascular diseases (CVD), type 2 diabetes mellitus, chronic kidney diseases (CKD), among others[1]. The increasing prevalence of MetS in many vulnerable populations poses a serious public health problem worldwide. The prevention of MetS and its individual components among high-risk populations has been identified as an important intervention strategy for reduction of chronic diseases and the associated health costs[2]. Thus, the crucial basis for developing and implementing workplace-specific health interventions requires the identification of major MetS risk factors and their distribution patterns among workers in different occupations[3].

Current interventions towards reduction of cardio-metabolic risks in the workplace target diet and physical activity as certain occupations promote unhealthy dietary pat-

terns. Among the modulators of MetS, recent studies[4,5] focused on hyperuricemia, the elevated concentrations of uric acid (UA) in the blood. UA, the final product of purine metabolism is a very important diagnostic and prognostic factor in many disorders. The plasma level of UA is influenced by diet, cellular breakdown and renal elimination hence hyperuricemia can occur as a consequence of increased production and/or decreased excretion of UA in the kidney[6].

The relationship between MetS and hyperuricemia is suggested to be causal. Recent evidence indicated that the risk of developing MetS is higher with increased plasma levels of UA[7] and patients with MetS have higher risk of hyperuricemia than those without[8]. Although the explanation for this relationship and the underling mechanisms are currently not clear, it is partly ascribed to insulin resistance as hyperinsulinemia reduces renal excretion of UA and sodium[9]. Previous findings have also indicated that the prevalence of insulin resistance is increased in obese individuals due to elevated production of leptin hormone[10].

Both cross sectional and longitudinal studies[11] demonstrated that high plasma UA increased the prevalence of MetS by affecting its individual components. While the component of MetS that is mostly influenced by hyperuricemia is variable in many populations, it is also not established whether the relationship between MetS and hyperuricemia is specific to sex as studies showed conflicting results[12]. Thus, more studies are needed to elucidate this relationship, and to establish whether hyperuricemia is indeed an important biomarker of MetS.

Some occupations are characterized by physical inactivity, sedentariness, long working hours and work-stress which promote unhealthy dietary patterns such as consumption of high fat diet, sugar-sweetened foods and drinks and excessive alcohol[13]. These foods and lifestyle factors play an important role in the development of hyperuricemia. A fructose-rich diet can raise UA production and induce the components of MetS through mechanisms independent of energy intake or weight gain[14]. Given the close association between MetS and hyperuricemia, it is important to explore this relationship among workers as only few studies have reported the effects of occupational factors on plasma UA. Oh, et al.,[15] reported a significant association between shift work and hyperuricemia among Korean steelmaking male workers. In China, available evidence on the prevalence of hyperuricemia and its relationship with MetS focus on the general population[16] or patients, with limited data on workers. Thus, the objective of this study was to examine the association between hyperuricemia and MetS and the components of MetS that are mostly influenced by hyperuricemia among university workers.

Subjects and methods

Subjects and research design

A total of 2273 adults (male, n=1198; female, n=1075) on annual medical examination at a University Hospital participated in the study. The workers were categorized into two occupation groups: administrators including workers involved in office work and academics involved in research and teaching or both. In our previous work, we found high prevalence of MetS and its components among the male workers compared to their female counterparts, which was attributed to gender and occupational differences[17]. In the present study, we further examined whether there was an association between MetS and hyperuricemia. The study was approved by the institution's Ethics committee and hospital management. The participants consented to the study. Study protocols were conducted in accordance with the recommendations outlined in Helsinki Declaration of 1975, revised 2000.

Data collection and laboratory analysis

The standard procedures for physical assessment weight (kg) and height (m), systolic and diastolic blood pressures (mmHg), and biochemical analysis have been described previously[17]. Briefly, weight and height of the participants were measured using the stadiometer (HW-700, Zhengzhou, China) and used to compute body mass index (BMI). Blood pressure was determined using standard sphygmomanometer (YE-665 A, Jiangsu, China). Overnight fasting blood samples were obtained by venipuncture and plasma separated by centrifugation (KDC-1044, Hangzhou, China) at 1000 ×g and 4°C for 10 minutes. Immediately, lipid profiles, glucose, uric acid, creatinine and urea were determined by enzymatic methods with an Automatic Biochemical Analyser (HF 0400, Shanghai, China) at the University Hospital laboratory.

Diagnosis

Metabolic syndrome

Metabolic syndrome was defined according to the Modified criteria of the National Cholesterol Education Program Adult Treatment Panel[18]. The criteria require the

presence of at least three of the following components; central obesity, waist circumference >102 cm (males), >88 cm (females); systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg; fasting plasma glucose ≥ 5.6 mmol/L;dyslipidemia, triglycerides (TG)≥1.695 mmol/L and high density lipoprotein cholesterol(HDL) < 1.036 mmol/L (males), HDL< 1.295 mmol/L (females). Total cholesterol (TC) and HDL were used to calculate the atherosclerotic index (AI) as follows: AI= (TC mmol/L -HDL mmol/L) / HDL mmol/L.

Assessment of chronic kidney disease

The creatinine based equation that estimates glomerular filtration rate (GFR) was used to assess chronic kidney disease (CKD). This equation, in addition to plasma creatinine levels, takes into account individual differences in age, gender and race. Estimated GFR (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations[19]. The eGFR <60 ml/min/1.73 m^2 was diagnosed as CKD. The CKD-EPI equation for estimatingGFR is as follows: eGFRCKD-EPI = 141 × min (Scr/k, 1)α × max (Scr/k, 1)−1.209 × 0.993Age × 1.018 (if female) × 1.159 (if black); where k is 0.7 for females and 0.9 for males, α is −0.329 for females and −0.411 for males, "min" indicates the lesser of Scr/k or 1, and "max" indicates the greater of Scr/k or 1.

Hyperuricemia

Using plasma UA levels, hyperuricemia was defined as ≥420 μmol/L in males and ≥ 360μ mol/L in females.

Statistical analysis

Data was analyzed using the Statistical Package for Social Scientists for Windows, 18 (SPSS, Inc. Chicago, USA). Data were expressed as means ± standard deviations, correlation coefficients (r), proportions (%), standardized regression coefficients (β), odds ratios and 95% confidence interval. Independent samples t-test and analysis of variance (ANOVA) were used to compare means, and Tukey's HSD test used for post hoc analysis. Associations were assessed by Chi-square (χ2) test, Pearson correlation, and conditional forward and backward binary logistic regression. P <0.05 was considered statistically significant.

Results

Age, body mass index and renal function tests of the study participants

Table 1 presents the age, body mass index (BMI) and renal function tests of the male (1198) and female (n=1075) study participants. As shown, age, BMI, plasma levels of uric acid (UA), creatinine, urea and estimated glomerular filtration rate (eGFRCKD-EPI) were significantly (P<0.01) higher in male than female workers. Overall prevalence of hyperuricemia was 10% with prevalence being significantly higher (χ2= 75.587, p=0.001) in male (15.2%) than female (4.1%) workers. CKD prevalence was 11.7% and was higher (χ2, 220.047, P<0.01) in males (21.7%) than females (0. 4%).

Table 1. Age, body mass index and renal function tests of the study participants

Characteristic	All (n=2273)	Male (n=1198)	Female (n=1075)	p
Age (years)	42.63 ± 8.63	44.45± 8.01	40.45 ± 8.84	0.001
Body mass index (kg/m^2)	22.95 ± 3.01	24.09 ± 2.92	21.67 ± 2.57	0.001
Uric acid (μmol/L)	306.61 ± 81.24	363.98 ± 68.71	252.81 ± 56.03	0.001
Number (%) with high UA[a]	228 (10)	182 (15.2)	44 (4.1)	0.001
Creatinine (μmol/L)	67.05 ± 15.96	77.94 ± 12.72	54.98 ± 8.00	0.001
Urea (mmol/L)	4.53 ± 1.14	4.53 ± 1.14	4.88 ± 1.12	0.001
$eGFR_{CKD-EPI}$	104.02 ± 40.95	75.52 ± 22.02	135.91 ± 32.95	0.001
Number (%) with CKD[b]	234 (11.7)	230 (21.7)	4 (0.4)	0.001

Values are mean ± standard deviations; p<0.01 – statistically significant by independent samples t-test; high uric acid (UA) levels (≥ 420 μmol/L in males and ≥ 360μmol/L in females); [a]analysis by Chi (χ^2) test statistic, (χ^2= 75.587); eGFR-estimated glomerular filtration rate by CKD-EPI-Chronic Kidney Disease Epidemiology Collaboration equation; [b]CKD – chronic kidney disease (eGFR <60 ml/min/1.73 m^2) using CKD-EPI creatinine based equation for 1057 males and 941 females, χ^2, 220.047).

Plasma uric acid concentrations in different levels of metabolic parameters

Plasma uric UA concentrations were significantly (P<0.01) higher in males and females with elevated systolic blood pressure (SBP) and diastolic blood pressure (DBP), TG, AI (TC-HDL/HDL ratio), fasting plasma glucose levels (FPG) and reduced HDL (Table 2).

Table 2. Plasma uric acid concentrations in different levels of metabolic parameters

	Male			Female		
	n	Uric acid (μmol/L)	p	n	Uric acid (μmol/L)	p
SBP						
≥130 mmHg	472	363.85 ± 71.23	0.001	156	249. 93 ± 54.22	0.001
<130 mmHg	698	347.18 ± 66.13		881	270.22 ± 63.17	
DBP						
≥85 mmHg	581	364.23 ± 71.54	0.001	178	270.07 ± 62.41	0.001
<85 mmHg	589	343.71 ± 64.20		859	249.45 ± 54.07	
Triglycerides						
≥1.695mmol/L	358	378.80 ± 73.05	0.001	105	292.11 ± 64.88	0.001
≥1.695 mmol/L	814	343.19 ± 63.83		939	248.41 ± 53.20	
HDL						
<1.036 mmol/L	136	368.02 ± 75.47	0.011		-	-
≥1.036 mmol/L	1035	352.14 ± 67.59			-	-
< 1.295 mmol/L	-	-	-	170	269.18 ± 66.24	0.001
> 1.295 mmol/L	-	-	-	874	249.62 ± 53.27	
TC-HDL/HDL						
≥2.64	663	369.09 ± 67.89	0.001	241	277.20 ± 61.19	0.001
<2.64	508	334.26 ± 64.72		803	245.49 ± 52.23	
FPG						
≥ 5.6 mmol/L	269	361.51 ± 72.76	0.042	70	278.81 ± 7.89	0.001
<5.6 mmol/L	900	351.79 ± 67.38		974	250.94 ± 54.27	

Values are mean ± standard deviations; p<0.05 – statistically significant by independent samples t-test; SBP – systolic blood pressure; DBP – diastolic blood pressure; HDL – high density lipoprotein cholesterol; TC – total cholesterol; FPG – fasting plasma glucose.

Correlation between plasma UA concentrations and the metabolic parameters

Plasma UA increased with age in females and not in males (Table 3). UA associated positively (P<0.01) with BMI, SBP, DBP, TC, low density lipoprotein cholesterol (LDL), triglycerides (TG), atherosclerosis index, FPG (only females), and inversely with HDL in both sexes.

Table 3. Correlation of plasma uric acid levels with metabolic parameters

	Male (n=1198) Coefficient (r)	Female (n=1075) Coefficient (r)
Age (y)	-0.008	0.082**
BMI (kg/m^2)	0.254**	0.313**
SBP (mmHg)	0.143**	0.196**
DBP(mmHg)	0.165**	0.200**
TC (mmol/L)	0.087**	0.128**
LDL (mmol/L)	0.102**	0.189**
TG (mmol/L)	0.280**	0.259**
HDL (mmol/L)	-0.233**	-0.204**
TC-HDL/HDL	0.293**	0.293**
FPG (mmol/L)	0.009	0.141**

**p<0.01 – statistically significant by Pearson correlations; UA – uric acid; SBP – systolic blood pressure; DBP – diastolic blood pressure; TC – total cholesterol; TG – triglycerides; LDL– low density lipoprotein cholesterol; HDL– high density lipoprotein cholesterol; FPG – fasting plasma glucose.

Association of hyperuricemia with metabolic syndrome and its components

The participants were divided into two groups based on UA concentrations; hyperuricemic and normal UA group, and relationship with age, BMI, sex, renal function, MetS and each of its components examined. As shown in Table 4, hyperuricemia was 3 times significantly higher (OR, 2.938; 95% CI, 1.909-4.522, P<0.01) in males than females after adjustment for age, BMI and renal function. The MetS increased 3.7 times (OR, 3.682; 95% CI, 2.320-5.843, P<0.01), dyslipidemia, 2.5 times (OR, 2.512; 95% CI, 1.808-3.491, P<0.01) and hypertension, 1.4 times (OR, 1.421; 95% CI, 1.005-2.011, P<0.05) in hyperuricemic individuals compared to normal UA group after adjustment for age, sex and BMI. Further adjustments for kidney function increased MetS 4-fold (OR, 3.903, CI, 2.439-6.245, P<0.01) and dyslipidemia 2.5 times (OR. 2.501, 95% CI, 1.776-3.521, P<0.01). Hyperuricemia associated positively (P<0.05) with hyperglycemia (OR, 1.448; 95% CI, 1.020-2.054) only after adjustment for age and sex.

Table 4. Association of hyperuricemia with metabolic syndrome and its components in all participants

Parameter	Mode1	Odds ratio	95% CI	p
Age (y)	-	1.025	1.009-1.042	0.003*
Body mass index[a]	-	1.283	1.212-1.358	0.001**
With CKD [a]	-	0.598	0.405-0.885	0.010*
With CKD[b]	-	0.687	0.445-1.060	0.090
Sex(male)[c]	-	2.938	1.909-4.522	0.001**
Metabolic syndrome[d]	1	4.259	2.841-6.384	0.001**
	2	3.682	2.320-5.843	0.001**
	3	3.903	2.439-6.245	0.001**
Dyslipidemia[d]	1	3.211	2.396-4.303	0.001**
	2	2.512	1.808-3.491	0.001**
	3	2.501	1.776-3.521	0.001**
Hypertension[d]	1	1.812	1.330-2.469	0.001**
	2	1.421	1.005-2.011	0.047*
	3	1.427	0.996-2.205	0.052
Hyperglycemia[d]	1	1.448	1.020-2.054	0.038*
	2	1.387	0.938-2.051	0.101
	3	1.476	0.989-2.202	0.057

[a] association with hyperuricemia (dependent variable) after adjustment for age; [b] association with hyperuricemia after adjustment for age and body mass index (BMI) [c] association with hyperuricemia after adjustment for age, BMI and kidney function; [d] dependent variable; Model 1– adjustment for age and sex; Model 2 – adjustment for age, sex and BMI; Model 3 – further adjustment for kidney function; **p<0.01, *p<0.05 statistically significant by binary logistic regression; CI – confidence interval; CKD – chronic kidney disease.

Renal function tests by occupation type

The age and BMI were significantly (P<0.05) higher among administrators as compared to workers in academics for both gender (Table 5). Plasma UA and creatinine levels were higher (P<0.05) in male administrators compared to those in academics. However, there was no difference (P>0.05) in plasma UA and creatinine levels among female workers. Further, the prevalence of hyperuricemia in male administrators was significantly (P<0.05) higher (19%) than that of workers in academics (12.7%) although there was no difference in females.

Table 5. Comparison of renal function tests of male and female workers by occupation

	Male (n=1198)			Female (n=1075)		
	Administration (n=448)	Academics (n=750)	p	Administration (n=332)	Academics (n=743)	p
Age (years)	46.61±8.89	43.16±8.57	0.001	41.79±8.07	39.89±7.93	0.001
Body mass index (kg/m^2)	24.34±3.26	23.95±2.71	0.042	21.99±2.54	21.54±2.58	0.017
Uric acid (µmol/L)	360.75±71.44	349.89±66.73	0.009	255.72±55.27	251.26±56.59	0.234
% with high UA[†]	19.0	12.7	0.004	4.3	3.9	0.782
Creatinine (µmol/L)	72.87±18.72	76.59±18.51	0.001	52.72±11.65	53.88±12.55	0.156
Urea(mmol/L)	4.94±1.17	4.85±1.09	0.184	4.27±1.05	4.11±1.06	0.028
$eGFR_{CKD-EPI}$	76.69±22.13	74.58±22.09	0.125	138.82±36.93	135.63±34.35	0.188
% with CKD[‡]	19.2	23.2	0.117	0	4	0.249

Values are mean ± standard deviations; $p<0.05$ – statistically significant by independent samples t-test; high uric acid (UA) levels (≥ 420 µmol/L in males and ≥ 360µmol/L in females); [a] analysis by Chi (χ^2) test statistic, (Male, χ^2= 8.526, female χ^2 =0.077); eGFR – estimated glomerular filtration rate by CKD-EPI-Chronic Kidney Disease Epidemiology Collaboration equation; [‡]CKD – chronic kidney disease (eGFR <60 ml/min/1.73 m^2) using CKD-EPI creatinine based equation, males, χ^2, 2.453 and female, χ^2, 1.326.

Relationship between hyperuricemia and occupation in male workers

Male workers with CKD were excluded from analysis. The association between hyperuricemia and type of occupation was examined in 868 males. Hyperuricemia was 1.9-fold higher ($P<0.05$) in male administrators compared to their academic counterparts after adjusting for age and BMI (Table 6).

Table 6. Association between hyperuricemia and occupation in male workers

Model	Odds ratio	95% confidence interval	P
0	1.930	1.298-2.869	0.001
1	1.897	1.275-2.825	0.002
2	1.875	1.202-2.925	0.006

Odds ratios and 95% confidence intervals for workers in administration. Model 0 – unadjusted, Model 1 – adjustment for age; Model 2 – further adjustment for BMI; $p<0.05$ statistically significant by binary logistic regression. Individuals with chronic kidney disease were excluded from analysis.

Discussion

In the present study, hyperuricemia positively associated with MetS and the relationship was greater in males than females. Among MetS components, dyslipidemia showed greater associations with hyperuricemia after adjustments for age, sex, BMI and renal function. Moreover, administrative work was associated with a high prevalence of hyperuricemia among male workers but not in female workers. The high prevalence of hyperuricemia among male adults as compared with females (Table 1) is in consistence with reports on the general population in China[16]. This gender disparity could be attributed to age and hormonal differences as the male workers were much older than female workers who were largely pre-menopausal. High androgen levels in younger men promote renal uric acid (UA) re-absorption whereas estrogen in younger women promotes more efficient renal clearance of UA[20]. Thus, UA levels often increase after menopause in women. The disparity may also be attributed to sex differences in dietary habits and life styles.

The high prevalence of hyperuricemia may be related to increased production of UA from diet as high consumption of purine-rich foods such as meats and sea foods, sugar-sweetened food as well as alcohol consumption is common among the Chinese populations[21]. Villegas et al.,[22] also reported a direct association between seafood consumption and hyperuricemia among middle-aged Chinese men. Hyperuricemia may also be due to high intake of fructose rich foods[13]. During fructose metabolism, ATP is consumed leading to accumulation of AMP which stimulates AMP deaminase, resulting in increased UA production.

The positive correlation of UA acid with metabolic parameters supports the role of diet in hyperuricemia. While BMI, blood pressure, lipid profiles and glucose increased with elevated UA in both sexes, HDL levels reduced (Table 2 and 3). This indicates that UA levels increase with body weight and can subsequently affect lipids, blood pressure and glucose. Obesity significantly affects UA metabolism[23]. Thus, among many other mechanisms, hyperuricemia is ascribed to increased production of leptin which is known to reduce the excretion of UA from the kidney. The increase in blood pressure could be attributed to endothelial dysfunction induced by hyperuricemia[24]. The increase in plasma glucose, TG, and reduced HDL may be linked to insulin resistance. In addition, hyperinsulinemia is closely related to high TG which is usually accompanied with reduced HDL levels.

In this study, after adjustment for confounding factors such as age, BMI and renal function, hyperuricemia was strongly associated with MetS (Table 4). This occurrence may be partly explained by the ability of UA to inhibit endothelial function as well as the effects from insulin resistance as explained earlier. Another possible mechanism by which hyperuricemia may be associated with MetS is through increased oxidative stress which we previously reported in these participants[25]. Further, among the components of MetS, dyslipidemia displayed a stronger association with hyperuricemia compared to hypertension and hyperglycemia. This observation concurs with findings from a previous longitudinal study[17]. Al-Meshaweh et al.,[26] also reported that hyperuricemia was common in dyslipidemic patients. A plausible explanation may be related to dietary factors since hypertriglyceridemia and hyperuricemia are linked by a common denominator: a diet rich in fructose.

An interesting finding in this study was the high prevalence of hyperuricemia among male administrators as compared to those in academics even after adjustment for age and BMI, and excluding individuals with CKD in the analysis (Table 5 and 6). This may be explained by the nature of administrative work which is characterized by long periods of sitting and sedentariness[27] as compared to other occupations in the university[28]. Prolonged sitting is associated with insulin resistance[29], which, as explained earlier, causes reduced renal excretion of UA. The increased hyperuricemia among this occupation group might also be due to diet related factors as office-based workers may not have adequate time to eat or not able to access healthy foods[30]. Consequently, such workers may end up adopting lifestyles that promote development of hyperuricemia including consumption of convenient snacks that are high in fat, sugar-sweetened foods and drinks, and excessive alcohol[14]. Currently, there are few studies that determined the prevalence of hyperuricemia among workers in certain occupations. Oh et al.,[15] reported a significant positive association between shift work and hyperuricemia.

The limitations of this study included the cross-sectional nature of the design that does not allow for inference on the causal relationships between plasma UA concentration and MetS over time. Thus, there is a need for a longitudinal study to further confirm this relationship. The potential effects of diet on plasma uric acid levels and plasma insulin concentrations were also not measured.

Conclusion

Elevated plasma levels of uric acid were positively associated with metabolic syndrome and its components among university workers. This relationship was stronger in male than female workers. Among the MetS components, dyslipidemia was closely associated with hyperuricemia as compared to hypertension and hyperglycemia. Hyperuricemia was also common in male administrators as compared to those in academics. These findings support that hyperuricemia is a risk factor for metabolic syndrome, especially among male workers. Thus, there is a need for workplace-based health interventions aimed at reducing hyperuricemia and dyslipidemia among workers.

Conflicts of interest

Authors have no competing interests to declare.

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