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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The relationship between serum uric acid levels and angiographic severity of coronary heart disease

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Abstract

Background: Many studies have shown that the serum uric acid (SUA) level is associated with atherosclerosis.

Aim: To determine the relationship between the SUA level and the presence and severity of coronary heart disease (CHD).

Methods: A total of 705 patients who underwent coronary angiography were included in this study. All patients were assessed for the presence of cardiovascular risk factors and ongoing medications. SUA levels were measured in all patients before the procedure after 12 h of fasting. The severity of CHD was assessed by the SYNTAX score. The independent association between the SUA and the severity of CHD was statistically evaluated using IBM SPSS Statistics 21 for Windows.

Results: The mean age of the study population was 60.2 ± 11.0 years. 252 were female (35.7%) and 453 were male (64.3%). Of the patients, 59.0% had significant CHD, 34.6% had diabetes mellitus, 67.7% had hypertension, 55.3% had hyperlipidaemia, and 45.4% were current smokers. The mean SYNTAX score was 10.6 ± 12.9. According to the SYNTAX score, 289 of the patients (41%) had normal coronary arteries and non-significant CHD (controls, SYNTAX score: 0), 236 of the patients (33.5%) had mild CHD (SYNTAX score: 1–22), 97 (13.8%) had moderate CHD (SYNTAX score: 23–32), and 83 (11.8%) had severe CHD (SYNTAX score: ≥ 33). The mean SUA values were 5.3 ± 1.5 mg/dL in the control group, 5.6 ± 1.4 mg/dL in the mild CHD group, 6.2 ± 1.6 mg/dL in the moderate CHD group, and 6.5 ± 1.7 mg/dL in the severe CHD group. According to Spearman's rho analysis, a positive correlation between the SUA levels and the SYNTAX score was determined to be statistically significant ($p < 0.001$, $r = 0.239$; $p = 0.002$, $r = 0.148$ in men; $p = 0.001$, $r = 0.204$ in women).

Conclusions: In this study, we found a positive correlation between the SUA level and the SYNTAX score. Therefore, this routine biochemical test can be used for the evaluation of the severity of CHD besides other risk factors in clinical practice. However, larger scale randomised studies are needed to show the effects of SUA on the severity of CHD.

Key words: SYNTAX score, uric acid, coronary heart disease

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INTRODUCTION

Coronary heart disease (CHD) is now the leading cause of death worldwide. Several tests are used to predict atherosclerotic CHD. Serum uric acid (SUA) is a final enzymatic product of purine metabolism in humans. It was previously reported that SUA levels were associated with the risk of hypertension [1, 2], atherosclerosis [3], and cardiovascular diseases [4, 5]. SUA may contribute to the atherosclerotic process through the induction of endothelial dysfunction [6] and inflammation [7]. A recent meta-analysis demonstrated that hyperuricaemia may increase the risk of CHD events independently of traditional CHD risk factors [8]. In the present study, we aimed to investigate the association between SUA levels and the complexity of CHD as evaluated by the SYNTAX score.

METHODS

The study population was derived from a population of 1071 consecutive patients who underwent coronary angiography due to positive noninvasive stress test results. A total of 366 patients were excluded from the study because they met the exclusion criteria (n = 320) or did not fulfil the inclusion criteria (n = 46). Finally, 705 patients were enrolled (mean age 60.2 ± 11.0 years; 64.3% men). Our institutional review board approved the study and we obtained informed consent from all subjects. The inclusion criteria were age greater than 18 years, a coronary angiogram clear enough to enable evaluation of the cause of stress-induced chest pain, and the patient's consent. The exclusion criteria were current pregnancy, cardiomyopathy, previous myocardial

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Table 1. Baseline characteristics for the severity of coronary heart disease (CHD)

	Control group	Mild CHD	Moderate CHD	Severe CHD	P
Sex (M/F)	138/151	171/65	83/14	61/22	< 0.001
Hypertension	58.8%	72%	73.2%	79.5%	< 0.001
Hyperlipidaemia	46%	61.4%	59.8%	65.1%	0.001
Diabetes mellitus	25.3%	39.0%	41.2%	47.0%	< 0.001
Smoking	38.1%	55.1%	52.6%	34.9%	< 0.001
Age [year]	56.5 ± 11.3	61.1 ± 9.7	63.3 ± 9.9	66.9 ± 10.2	< 0.001
BMI — F [kg/m^2]	22.3 ± 3.2	23.2 ± 4.9	24.6 ± 4.6	23.3 ± 4.1	0.215
BMI — M [kg/m^2]	25.6 ± 4.9	25.9 ± 4.8	26.3 ± 4.1	26.7 ± 5.3	0.125
Waist circumference — F [cm]	80.5 ± 4.5	81.4 ± 5.9	82.1 ± 6.2	82.7 ± 5.1	0.118
Waist circumference — M [cm]	98.5 ± 4.8	99.1 ± 3.9	100.8 ± 5.1	101.3 ± 4.8	0.222
Fasting blood glucose [mg/dL]	104.6 ± 27.3	109.2 ± 30.1	116.5 ± 39.3	130.8 ± 57.1	< 0.001
Creatinine [mg/dL]	0.9 ± 0.3	1.0 ± 0.4	1.2 ± 0.3	1.3 ± 0.4	0.014
LDL-C [mg/dL]	121.3 ± 37.2	121.6 ± 38.8	124.3 ± 39.9	128.1 ± 38.7	0.012
HDL-C [mg/dL]	45.1 ± 11.9	41.5 ± 11.1	41.3 ± 12.1	38.9 ± 10.1	< 0.001
Total cholesterol [mg/dL]	191.4 ± 43.3	190.1 ± 44.7	192.3 ± 47.7	199.4 ± 46.4	0.006
Triglyceride [mg/dL]	150.5 ± 90.0	160.9 ± 86.5	173.4 ± 121.1	155.0 ± 110.4	0.198
Total cholesterol/HDL	4.7 ± 1.5	4.8 ± 1.5	4.7 ± 1.6	5.2 ± 1.9	0.130
Serum uric acid [mg/dL]	5.4 ± 1.5	5.6 ± 1.4	6.2 ± 1.6	6.5 ± 1.7	< 0.001
Haemoglobin [g/dL]	13.8 ± 1.7	14.6 ± 6.8	14.1 ± 1.7	13.5 ± 1.8	0.004
Platelet count [$\times 10^3/\mu$L]	248.0 ± 66.0	236.1 ± 69.6	237.1 ± 59.1	228.3 ± 75.8	0.010

Severity of CHD was determined by SYNTAX score; M — male; F — female; BMI — body mass index; LDL-C — low-density lipoprotein cholesterol; HDL-C — high-density lipoprotein cholesterol

infarction or any revascularisation procedure, unstable angina pectoris, history of congenital heart disease, renal dysfunction (GFR < 30 mL/min), being on any SUA-lowering therapy, and history of any uric acid metabolism disorder.

Two experienced cardiologists blinded to the study analysed the angiograms with a validated quantitative coronary angiographic system (General Electric Innova 3100 angiographic system, Buc Cedex, France). Selective coronary angiography was performed by the femoral approach using the Judkins technique. Multiple views were obtained, with visualisation of the left anterior descending and the left circumflex coronary artery in at least four projections, and the right coronary artery in at least two projections. Coronary angiograms were recorded on compact discs in DICOM format. The severity of the CHD was evaluated according to the SYNTAX score, which was calculated by a computer program consisting of sequential and interactive self-guided questions. The algorithm consists of 12 main questions. The total SYNTAX score was composed of the individual scores for each separate lesion with a diameter stenosis of ≥ 50% in a vessel ≥ 1.5 mm in diameter by visual assessment, as previously reported [9]. The 705 patients were divided into four groups on the basis of the SYNTAX scores: control group (SYNTAX score: 0), mild CHD group: group I (SYNTAX score: 1–22), moderate CHD group: group II (SYNTAX score: 23–32), severe CHD group: group III (SYNTAX score: ≥ 33). Blood samples were collected by venipuncture to perform routine blood chemistry after at least 12 h of fasting. Fasting SUA levels were measured at baseline on the clinical analyser utilising a uricase-based commercial kit.

Statistical analysis

The data were analysed with IBM SPSS Statistics 21 for Windows. The normal distribution of variables was verified with the Kolmogorov-Smirnov test. Spearman's rho correlation was used when one or both of the variables was/were not normally distributed. We used the Kruskal-Wallis test to account for the differences among the groups, but in order to analyse the specific sample pairs for significant differences we used the Conover-Inman test. χ^2 test was used to investigate whether the distributions of categorical variables differed within the groups. Moreover, binary logistic regression analyses were conducted according to diabetes mellitus (DM), hypertension (HT), hyperlipidaemia (HL), smoking, and SUA. Patients' characteristics are summarised as mean ± standard deviation values or as percentages. $P < 0.05$ was considered to be statistically significant.

RESULTS

The average age of the study population was 60.2 ± 11.0 years, and 64.3% of them were male. Table 1 shows the baseline

characteristics and biochemical examinations of the study participants according to SYNTAX score groups. Of the patients, 59.0% had significant CHD, 34.6% had DM, 67.7% had HT, 55.3% had HL, 52.1% had a family history of CHD, 45.4% of them were current smokers, and 7.8% had a history of smoking. Mean SYNTAX scores were 8.3 ± 3.9, 24.3 ± 1.9, and 37.7 ± 5.2 in the mild, moderate, and severe CHD groups, respectively. Higher SYNTAX scores were calculated in men than in women (12.8 ± 13.5, 6.5 ± 10.9, respectively; $p < 0.001$). According to the SYNTAX score, 289 of the patients (41%) had normal coronary arteries or non-significant CHD (controls, SYNTAX score: 0); 236 (33.5%) had mild CHD (SYNTAX score: 1–22); 97 (13.8%) had moderate CHD (SYNTAX score: 23–32); and 83 (11.8%) had severe CHD (SYNTAX score: ≥ 33). Diabetic, hypertensive, hyperlipidaemic patients, and smokers had more severe CHD than the controls ($p < 0.001$, $p < 0.001$, $p < 0.001$, $p = 0.001$, respectively). Mean SUA values were 5.4 ± 1.5 mg/dL in the control group; 5.6 ± 1.4 mg/dL in the mild CHD group; 6.2 ± 1.6 mg/dL in the moderate CHD group, and 6.5 ± 1.7 mg/dL in the severe CHD group (Fig. 1). According to Spearman's rho analysis, a positive correlation between the SUA levels and the SYNTAX score was determined to be statistically significant ($p < 0.001$, $r = 0.239$; $p = 0.002$, $r = 0.148$ in men; $p = 0.001$, $r = 0.204$ in women). A significant relationship was observed between the severity of CHD and SUA ($p < 0.05$ was considered to be statistically significant after Conover-Inman test) (Table 2). Higher SUA levels were measured in men than in women (6.0 ± 1.5; 5.1 ± 1.6, respectively; $p < 0.001$). Table 3 shows the SUA distribution by gender. Cutoff values of SUA for predicting severe CHD are shown in Figure 2. After adjustment according to traditional risk factors including age, DM, HT, and smoking status, the correlation between SUA and SYNTAX score maintained its significance ($p < 0.001$, $r = 0.239$). Furthermore, a significant relationship was found between the SUA levels and treatment modality after coronary angiography ($p < 0.001$) (Table 4). In the logistic regression analysis, well-established risk factors for CHD, such as DM, HT, HL, smoking, and SUA, were the covariates. SUA was found to be an independent predictor of CHD (CHD was defined as SYNTAX score > 1) ($p < 0.001$, odds ratio [OR]: 1.252; 95% confidence interval [CI] 1.128–1.389); DM, HL, HT, and smoking were also found to affect the severity of CHD (Table 5).

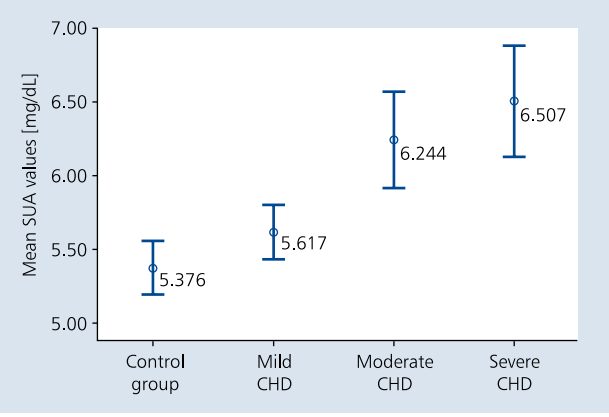

Figure 1. Mean serum uric acid (SUA) values according to the severity of coronary heart disease (CHD) groups

DISCUSSION

Uric acid is a heterocyclic compound of carbon, nitrogen, oxygen, and hydrogen, with the formula $C_5H_4N_4O_3$. It forms

Table 2. Binary comparisons according to the severity of coronary heart disease (CHD)

	Controls–Mild CHD (p)	Controls–Moderate CHD (p)	Control–Severe CHD (p)
Serum uric acid	0.169	**< 0.001**	**< 0.001**
Platelet count	**0.035**	0.840	**0.043**
Fasting blood glucose	0.349	**0.025**	**< 0.001**
TC/HDL-C	0.193	0.894	**0.025**
Triglyceride	0.058	0.256	0.749
LDL-C	0.174	**0.007**	0.429

Conover-Inman test was performed for the binary comparisons among the groups; $p < 0.05$ was considered to be statistically significant. Severity of CHD was determined by SYNTAX score; TC — total cholesterol; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol

Table 3. Serum uric acid (SUA) distribution by gender

SUA [mg/dL]	Control group	Mild CHD	Moderate CHD	Severe CHD	P
Female (n = 252)	4.8 ± 1.4	5.2 ± 1.7	5.9 ± 2.2	6.1 ± 1.7	0.002
Male (n = 453)	5.9 ± 1.5	5.8 ± 1.3	6.3 ± 1.5	6.6 ± 1.7	0.003

Severity of coronary heart disease (CHD) was determined by SYNTAX score.

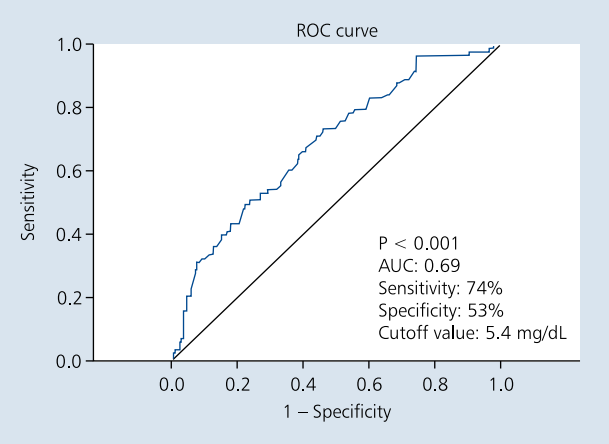

Figure 2. Cutoff value of serum uric acid for predicting severe coronary heart disease. Receiver-operating characteristic (ROC) curve for SYNTAX score ≥ 33; AUC — area under the curve

Table 4. The relationship between treatment modality and serum uric acid (SUA) levels; p < 0.001

	SUA [mg/dL]
Medical therapy	5.5 ± 1.5
PCI	5.9 ± 1.5
CABG surgery	6.4 ± 1.6

CABG — coronary artery bypass graft; PCI — percutaneous coronary intervention

Table 5. Results of binary logistic regression analysis of various risk factors effective for the presence of coronary heart disease (CHD)

	ß	P	OR	95% CI
Diabetes mellitus	0.535	0.004	1.708	1.192–2.447
Hypertension	0.439	0.013	1.551	1.098–2.192
Hyperlipidaemia	0.425	0.011	1.529	1.100–2.126
Smoking	0.504	0.002	1.655	1.202–2.279
Serum uric acid	0.225	< 0.001	1.252	1.128–1.389

CHD was defined as SYNTAX score > 1; OR is statistically significant (CI does not include 1); OR — odds ratio; CI — confidence interval

ions and salts known as urates and acid urates such as ammonium acid urate [10, 11]. SUA is the metabolic end product of purine metabolism in humans, and excess accumulation can lead to various diseases. A high SUA level is a prerequisite for gout and is also associated with metabolic syndrome and risk factors for cardiovascular disorders [12–14].

Increased SUA levels are usually caused by hereditary reasons. High SUA levels have been reported in diabetic, hypertensive, obese, and dyslipidaemic patients. A multiple risk factor clustering syndrome has been considered to be responsible for increased SUA levels in CHD, questioning the role of SUA level as an independent risk factor [15]. The last two reactions of its production catalysing the transformation of hypoxanthine to xanthine and the second to SUA are catalysed by the enzyme xanthine oxidoreductase, which may attain two inter-convertible forms, namely xanthine dehydrogenase or xanthine oxidase. The latter uses molecular oxygen as an electron acceptor and creates superoxide anion and other reactive oxygen molecules. The role of SUA in conditions associated with oxidative stress is not completely clear. It has previously been reported that increased SUA level is a risk factor for atherosclerotic cardiovascular disease where oxidative stress plays an important pathophysiological role [16]. Demir et al. [17] has shown renewed interest in the inhibition of xanthine oxidase system for preventing the progression of atherosclerosis. Allopurinol, a xanthine oxidase inhibitor, is being extensively studied for its role as an add-on therapy in stable atherosclerotic CHD [18]. It acts by reducing vascular oxidative stress resulting in endothelial stabilisation. Its benefits in prolonging survival in patients with heart failure have been proven in the literature [19]. Hyperuricaemia is an independent predictor for early atherosclerosis [20]. SUA can easily be measured through a blood test and is a potentially important target for intervention not only to reduce the risk of gout, but even more importantly, the more serious threat of cardiovascular death.

In this present study, the mean SUA levels are consistent with the literature [21]. However, in some studies SUA levels were higher than those measured in our study [22, 23]. Consistent with the literature, CHD was more common and more severe in men than in women in our study [24]. The relationship between hyperuricaemia and CHD according to sex is controversial in the literature. The NHANES study observed SUA to be predictive of CHD mortality in women but not in men [25]. Among women in the Gothenburg Study [26] and the Framingham Study [27], SUA was independently associated with all-cause mortality but not with CHD incidence. In contrast to these findings, an independent association between hyperuricaemia and a trend for more severe CHD was reported in men, but not in women, in an Iranian sample undergoing coronary angiography [28]. Unlike these studies, it has been reported that elevated SUA is associated with the presence and severity of CHD in both men and women [29]. In accordance with this literature, our study showed that the SUA levels in the high SYNTAX-score group were significantly higher than those in the low-moderate SYNTAX-score groups and those measured in the controls for both sexes. Logistic regression analysis demonstrated that increased SUA level was an independent risk factor for the presence of CHD in our study. Therefore, a higher baseline of SUA level was independently associated with the severity and complexity of CHD as assessed by the SYNTAX score. Elevated SUA levels may act as a biomarker of underlying myocardial ischaemia and serve as another possible explanation for the associa-

tion between hyperuricaemia and high SYNTAX score. The underlying mechanisms linking SUA levels and cardiovascular mortality have not yet been clearly demonstrated. Although it has antioxidant properties at low serum levels, when it is above 6 to 6.5 mg/dL (for females and males, respectively) these properties change into a pro-oxidant state. Contrary to our findings, Lu et al. [30] reported that there was no relationship between SUA levels and severity of CHD.

We also found an association between treatment modality after coronary angiography and SUA levels in our study. Therefore, increased level of SUA was often associated with the choice of percutaneous coronary intervention and coronary artery bypass grafting surgery. In contrast to our study, Lin et al. [31] found that there was no association between the SUA levels and the selected treatment modality for CHD.

Limitations of the study

Our study has some limitations. In the current study the patients did not undergo intravascular ultrasonography to assess the atherosclerotic coronary plaque burden. Secondly, patients undergoing coronary angiography who visit a single centre do not represent the whole community. Thirdly, it is a cross-sectional study, so we were unable to examine the impact of hyperuricaemia over time. It would also be interesting to follow up the patients for prognostic significance and analyse the effect of varying uric acid levels. Large-scale prospective studies are needed to obtain further information.

CONCLUSIONS

In conclusion, the SUA levels were independently associated with the severity and complexity of CHD in our study. With regard to the association between the severity of CHD and the SUA levels, this simple biochemical test can be used to determine the cardiovascular disease burden besides other risk factors during routine clinical practice. Large-scale prospective, randomised clinical trials are needed to see whether the SUA levels obtained during routine testing are of greater value in terms of diagnosis, risk stratification, and treatment evaluation in patients with atherosclerotic CHD.

Conflict of interest: *none declared*

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Zależność między stężeniem kwasu moczowego w surowicy a nasileniem zmian angiograficznych w chorobie wieńcowej

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Streszczenie

Wstęp: W wielu badaniach wykazano, że stężenie kwasu moczowego w surowicy (SUA) wiąże się z występowaniem miażdżycy.

Cel: Celem niniejszej pracy było określenie zależności między stężeniem SUA a występowaniem i nasileniem choroby wieńcowej (CHD).

Metody: Do badania włączono ogółem 705 chorych poddanych koronarografii. Wszystkich uczestników badania oceniono pod kątem obecności czynników ryzyka sercowo-naczyniowego i stosowanej farmakoterapii. Przed koronarografią u wszystkich chorych zmierzono stężenie SUA na czczo (po 12 godzinach nieprzyjmowania pokarmów). Nasilenie CHD określano za pomocą skali SYNTAX. Niezależny związek między stężeniem SUA a nasileniem CHD oceniano statystycznie przy użyciu oprogramowania IBM SPSS Statistics 21 do systemu Windows.

Wyniki: Średnia wieku badanej grupy wynosiła 60,2 ± 11,0 lat. Wśród uczestników były 252 (35,7%) kobiety i 453 (64,3%) mężczyzn. U 59,0% osób rozpoznano istotną CHD, u 34,6% — cukrzycę, u 67,7% — nadciśnienie tętnicze, u 55,3% — hiperlipidemię, a 45,4% pacjentów paliło tytoń. Średnia punktacja w skali SYNTAX wynosiła 10,6 ± 1,9. Zgodnie z punktacją w skali SYNTAX 289 (41%) chorych miało prawidłowe tętnice wieńcowe i nieistotną CHD (grupa kontrolna, punktacja w skali SYNTAX: 0), 236 chorych (33,5%) — łagodną CHD (punktacja w skali SYNTAX: 1–22), 97 chorych (13,8%) — umiarkowaną CHD (punktacja w skali SYNTAX: 23–32), a 83 (11,8%) osób — ciężką CHD (punktacja w skali SYNTAX: ≥ 33). Średnie stężenie SUA wynosiło 5,3 ± 1,5 mg/dl w grupie kontrolnej, 5,6 ± 1,4 mg/dl w grupie z łagodną CHD, 6,2 ± 1,6 mg/dl w grupie z umiarkowaną CHD i 6,5 ± 1,7 mg/dl w grupie z ciężką CHD. Na podstawie analizy współczynnika rho Spearmana wykazano, że dodatnia korelacja między stężeniem SUA a punktacją w skali SYNTAX była statystycznie istotna ($p < 0,001$; $r = 0,239$; u mężczyzn: $p = 0,002$; $r = 0,148$; u kobiet: $p = 0,001$, $r = 0,204$).

Wnioski: W niniejszym badaniu stwierdzono dodatnią korelację między stężeniem SUA a punktacją w skali SYNTAX. Zatem to rutynowe badanie biochemiczne może być stosowane w praktyce klinicznej w celu oceny stopnia ciężkości CHD obok innych czynników ryzyka. Potrzebne są jednak badania z randomizacją obejmujące większą grupę chorych, aby wykazać wpływ stężenia SUA na nasilenie CHD.

Słowa kluczowe: skala SYNTAX, kwas moczowy, choroba wieńcowa

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