ORIGINAL

Effect of Dietary Counseling on Patients with Asymptomatic Hyperuricemia

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Abstract : In Japan, hyperuricemia is on the rise. The guideline for the management of hyperuricemia and gout recommends lifestyle changes before beginning drug therapy. This study aimed to evaluate the effectiveness of dietary counseling following the guideline. Thirty-three subjects (24 men and 9 women) with asymptomatic hyperuricemia underwent dietary counseling for 6 months based on the following recommendations : (1) prevent excessive purine intake, (2) prevent excessive fructose intake, (3) limit alcohol drinking, and (4) drink sufficient water. Obese subjects were counseled on adequate energy intake. Blood sampling, anthropometric measurements, dietary surveys, and 24-h urine collection were performed at baseline and at 6 months. Serum uric acid (S-UA) levels were significantly lower at 6 months compared to baseline. Water intake and urine volume were considerably higher at 6 months than at baseline. When compared to baseline, urine UA (U-UA) levels were significantly lower, and renal fractional excretion of UA (FE_{UA}) was significantly higher at 6 months. Changes in renal function (serum creatinine, estimated glomerular filtration rate, and FE_{UA}) were significantly associated with ΔS-UA level. In this study, S-UA level was significantly decreased by dietary counseling in line with the guideline. This study illustrates the effectiveness of dietary counseling for asymptomatic hyperuricemia. J. Med. Invest. 70 : 34-40, February, 2023

Keywords **:** *asymptomatic hyperuricemia, dietary counseling, serum uric acid, guideline, renal function*

INTRODUCTION

The prevalence of hyperuricemia has increased, and the number of asymptomatic hyperuricemia patients was estimated to be over 10 million in Japan (1).

Sustained hyperuricemia has been linked to decreasing renal function and chronic kidney disease (CKD) (2, 3), and high serum uric acid (S-UA) levels are associated with obesity, metabolic syndrome, and cardiovascular disease (4-6). To avert the development of these diseases, adequate regulation of the S-UA level is essential.

Previous research suggests an association between hyperuricemia and dietary factors. Increased intake of meat and seafood, both of which are purine-rich meals, has been associated with higher S-UA levels (7, 8). Furthermore, acute purine consumption increased the incidence of recurring gout episodes nearly fivefold (9). Hence, dietary purine intake should be less than 400 mg per day. A meta-analysis of fructose intake and gout also showed an adverse association of sugar-sweetened beverages and fruit juices with the risk of gout (10). Daily alcohol use, regardless of the kind of alcoholic beverage, has been shown to increase the risk of gout in a dose-dependent manner (11-13). As a result, to prevent hyperuricemia and manage S-UA levels, it is critical to improving dietary habits. The third version of the guideline for the management of hyperuricemia and gout recommends that patients change their lifestyles before commencing drug therapy (14). According to the guidelines, patients should consume adequate energy, avoid consuming too much purine and fructose, restrict their alcohol use, and drink sufficient water. Several previous studies supported that dietary factors, such as purine-rich foods, fructose, and alcohol, are associated with hyperuricemia and gout (7-13). A previous study on treating gout patients using a stable dose of urate-lowering treatment found that dietary education did not improve S-UA level (15). However, few studies have investigated the effect of dietary counseling on patients with asymptomatic hyperuricemia.

Therefore, this study aimed to evaluate the effectiveness of dietary counseling in line with the guideline.

MATERIALS AND METHODS

Subjects

This study included 48 participants (36 men and 12 females) with asymptomatic hyperuricemia (S-UA > 7.0 mg/dL) who attended the medical clinic but had not begun anti-hyperuricemic drug treatment. Among 48 enrolled subjects, 4 withdrew owing to medication, 3 had missing data, and 8 dropped out. Finally, analyzed were 33 subjects (24 men and 9 women).

All subjects provided written informed consent. The Ethics Committee of the University of Shizuoka approved this study (approval number : 30-8), which was registered with UMIN (UMIN registration number : UMIN000045847). The study complied with the Helsinki Declaration.

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Study design

Subjects underwent monthly dietary counseling for 6 months based on the following four recommendations listed in the guideline (14) : (1) prevent excessive purine intake, (2) prevent excessive fructose intake, (3) limit alcohol drinking, and (4) drink sufficient water. Obese subjects were counseled on adequate energy intake, and their total energy intake was to be calculated by following formulas based on the guidelines for the management of obesity disease 2016 (16) : Total energy intake (kcal/day) = Ideal body weight (kg) × 25 (kcal/kg/day). Dietary counseling was conducted by well-trained registered dietitians. The well-trained registered dietitians used common nutritional guidance materials to standardize the content of dietary counseling.

Purine, fructose (fruits and soft drinks), alcohol and water intake were documented in a diary by the subjects, and these data were verified at each visit. At baseline and at 6 months, blood samples, anthropometric measures, food questionnaires, and 24-h urine collection were performed.

Measurements

Height was measured using a stadiometer at the first visit. Body weight (BW), abdominal circumference (AC), and blood pressure were measured at baseline and 6 months. BW was measured using TANITA WB-150 (Tokyo, JAPAN). AC was measured around the navel using anthropometric tape. Blood pressure was measured using OMRON automatic sphygmomanometer HEM-7251G (Kyoto, JAPAN). BMI was calculated using the following formula : BMI (kg/m^2) = weight (kg)/height (m)^2

The levels of S-UA and serum creatinine (S-Cr) were measured at baseline and at 6 months. The analyses of blood samples were performed by LSI Medience Co., Ltd (Tokyo, JAPAN).

At baseline and 6 months, urine was collected for 24 h. Subjects were instructed to discard their first-morning void the day before their visit and collect all urine for the next 24 h, including the first void the next morning. Urine was collected using a disposable urine collection box 3 L (33-4000, Atleta Co., Ltd, Osaka, JAPAN). Subjects were instructed to write down the total volume of a urine sample on a recording sheet and then collect roughly 20 mL using a dropper from the urine sample. At their visit, subjects provided the recording sheet as well as a 20 mL urine sample. UA, xanthine/hypoxanthine (Xa/Hx), Cr, albumin levels and pH were all measured in urine samples. The analyses of urine samples were performed by SRL Co., Ltd (Tokyo, JAPAN), except for the analyses of urine pH and Xa/Hx levels. The pH was measured using a portable pH meter (LAQUA act, D-71, Horiba Scientific, Kyoto, Japan). The level of Xa/Hx was measured using a Xa/Hx colorimetric assay kit (Bio Vision, USA).

We evaluated the estimated nutrient intake of subjects using a brief-type self-administered diet history questionnaire method (BDHQ) (17). The BDHQ is a 4-page fixed-portion questionnaire that asks about the consumption frequency of selected foods, but not about portion size, to estimate the dietary intake of 58 food and beverage items during the preceding month (18, 19). The BDHQ took only 20–30 min to answer. Subjects were informed about the dietary survey by well-trained registered dietitians and filled out by themselves.

Calculating formulas

We calculated the Cr clearance (C_{Cr}), UA clearance (C_{UA}), and renal fractional excretion of UA (FE_{UA}) using the following formulas (U represents urine, S means serum, and BSA denotes body surface area) (14, 20).

BSA (m^2) = Height (cm) $^{0.725}$ × Weight (kg) $^{0.425}$ × 0.007184

C_{Cr} (mL/min) = [U-volume (mL/day) × U-Cr (mg/dL)] / [S-Cr (mg/dL) × 1,440 (min)] × 1.73/BSA (m^2)

C_{UA} (mL/min) = [U-volume (mL/day) × U-UA (mg/dL)] / [S-UA (mg/dL) × 1,440 (min)] × 1.73 / BSA (m^2)

FE_{UA} (%) = [U-UA (mg/dL) × S-Cr (mg/dL)] / [U-Cr (mg/dL) × S-UA (mg/dL)] × 100

Statistical analysis

The data were presented as mean ± standard deviation. The normality of the data distribution was examined by the Shapiro–Wilk test. Parametric analysis was used for normal distribution data, and non-parametric analysis was used for data exhibiting a non-normal distribution. Changes in physical characteristics, blood parameters, the estimated nutrient intake, and urine parameters were analyzed using paired t-test or Wilcoxson signed-rank test. Correlation between changes in clinical variables at 6 months from baseline was analyzed using Pearson's product-moment correlation coefficient. Probability (p) values less than 0.05 were considered statistically significant in all analyses. All analyses used IBM SPSS statistics, version 26.0 (IBM Ltd., Chicago, IL, USA).

RESULTS

Characteristics of the subjects

Table 1 shows the characteristics of the subjects. The average age, BMI, and S-UA level at baseline were 67.1 ± 12.4 years, 24.0 ± 3.8 kg/m^2, 7.7 ± 0.4 mg/dL, respectively. The subjects included 6 subjects with diabetes, 20 subjects with hyperlipidemia, 20 subjects with decreased renal function (estimated glomerular filtration rate (eGFR) of 30–60 mL/min/1.73 m^2), 5 smokers, 11 alcohol drinkers, and 26 subjects who took the anti-hypertensive drug. The subjects were classified into subtype groups : 5 subjects were renal overload type, 27 were renal underexcretion type, and 1 was the combined type.

Changes in S-UA level and anthropometric and laboratory data

When compared to baseline, S-UA levels were significantly lower at 6 months (p<0.01), with an average reduction of 7.8%. After 6 months of dietary counseling, S-UA levels were lower in 27 subjects. Furthermore, 14 subjects were able to achieve the target range (7.0 mg/dL). Similar results were obtained when men and women subjects were analyzed separately (p<0.01, p<0.05, respectively). There was also a significant reduction at 6 months in BW and AC compared with baseline (p<0.05, p<0.01, respectively), whereas there were no significant differences in other parameters.

Nutrient intake and dietary records

Table 2 shows the estimated nutrient intakes during 6 months of dietary counseling. Compared with baseline, total fat intake and phosphorus were significantly decreased at 6 months (p<0.01, p<0.05, respectively). In the gender-stratified analysis, fat intake was decreased significantly in men (p<0.05). Alcohol tended to decrease at 6 months compared with baseline in 11 drinkers (baseline, 29.7 ± 15.9 ; 6 months, 21.0 ± 20.0 g/day, p = 0.066).

We used dietary records to estimate the intake of foods (purine-rich food, fruit, soft drink, alcohol, and water) associated with hyperuricemia. Few subjects usually consumed many purine-rich foods (≥200 mg/serving) at baseline. The soft drink and alcohol intake significantly decreased at 6 months compared with baseline (p<0.05, p<0.01, respectively). When compared to baseline, water intake significantly increased at 6 months (baseline, 1,283 ± 458 ; 6 months, 1,724 ± 621 mL/day, p < 0.01). There were no significant differences in fruit intake.

Table 1. Characteristics of the subjects with asymptomatic hyperuricemia in this study at baseline and 6 months†

		All (n = 33)		Men (n = 24)		Women (n = 9)	
		Baseline	6 months	Baseline	6 months	Baseline	6 months
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Age	(year)	67.1 ± 12.4		64.6 ± 12.0		73.7 ± 11.5	
BW	(kg)	64.2 ± 11.9	63.2 ± 11.2 *	66.5 ± 8.0	65.8 ± 7.8	58.0 ± 17.8	56.4 ± 16.1
BMI	(kg/m^2)	24.0 ± 3.8	23.7 ± 3.4	23.4 ± 1.8	23.2 ± 1.7	25.6 ± 6.6	25.0 ± 5.9
AC	(cm)	86.3 ± 10.4	84.6 ± 9.8 **	85.0 ± 6.3	83.2 ± 5.9 *	89.9 ± 17.2	88.4 ± 16.2
SBP	(mmHg)	134 ± 26	136 ± 23	131 ± 25	131 ± 21	142 ± 28	148 ± 24
DBP	(mmHg)	76 ± 17	82 ± 15	77 ± 19	82 ± 16	74 ± 10	81 ± 14
S-UA	(mg/dL)	7.7 ± 0.4	7.1 ± 0.9 **	7.8 ± 0.4	7.3 ± 0.9 **	7.4 ± 0.3	6.7 ± 0.8 *
S-Cr	(mg/dL)	0.95 ± 0.15	0.95 ± 0.13	0.98 ± 0.14	0.98 ± 0.12	0.87 ± 0.16	0.89 ± 0.14
eGFR	(mL/min/1.73m^2)	58.9 ± 13.8	58.4 ± 12.1	62.0 ± 13.6	62.0 ± 11.2	50.5 ± 11.2	49.0 ± 9.2

†Values are mean ± SD. ** ; $p < 0.01$, * ; $p < 0.05$ vs. baseline, paired t-test or Wilcoxon signed-rank test. BW ; body weight, BMI ; body mass index, AC ; abdominal circumference, SBP ; systolic blood pressure, DBP ; diastolic blood pressure, S-UA ; serum uric acid, S-Cr ; serum creatinine, eGFR ; estimated glomerular filtration rate.

Table 2. The estimated dietary nutrients assessed by BDHQ following dietary counseling for 6 months†

		All (n = 33)		Men (n = 24)		Women (n = 9)	
		Baseline	6 months	Baseline	6 months	Baseline	6 months
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Energy	(kcal)	1,657 ± 445	1,517 ± 387	1,717 ± 453	1,617 ± 334	1,497 ± 401	1,252 ± 412 *
Protein	(g)	64.2 ± 24.7	59.4 ± 18.7	65.2 ± 25.2	60.4 ± 17.6	61.8 ± 24.7	56.7 ± 22.4
Fat	(g)	51.4 ± 20.1	43.7 ± 15.0 **	52.8 ± 21.3	44.4 ± 13.8 *	47.6 ± 17.2	41.9 ± 18.7
Carbohydrate	(g)	211.6 ± 55.6	204.3 ± 64.6	215.1 ± 53.9	220.8 ± 62.8	202.3 ± 62.4	160.0 ± 48.3 *
P % Energy	(%)	15.3 ± 3.4	15.8 ± 3.2	14.8 ± 2.4	14.9 ± 2.9	16.5 ± 5.3	18.0 ± 3.2
F % Energy	(%)	27.4 ± 6.8	26.1 ± 6.3	27.0 ± 7.0	24.7 ± 5.4	28.4 ± 6.6	29.7 ± 7.2
C % Energy	(%)	52.0 ± 9.2	53.7 ± 9.2	51.1 ± 8.7	54.4 ± 9.2 *	54.4 ± 10.6	51.7 ± 9.5
NaCl	(g)	9.8 ± 3.2	9.2 ± 2.6	10.3 ± 3.3	9.8 ± 2.5	8.8 ± 3.1	7.7 ± 2.2
Potassium	(mg)	2,360 ± 982	2,357 ± 755	2,368 ± 1084	2,347 ± 794	2,341 ± 692	2,381 ± 683
Calcium	(mg)	502 ± 284	459 ± 173	494 ± 323	454 ± 174	521 ± 150	473 ± 179
Magnesium	(mg)	237 ± 93	228 ± 70	241 ± 102	233 ± 73	224 ± 68	215 ± 64
Phosphorus	(mg)	976 ± 410	887 ± 272 *	982 ± 444	898 ± 265	959 ± 324	857 ± 305 *
Alcohol	(g)	10.0 ± 16.7	7.3 ± 15.0	13.8 ± 18.3	10.0 ± 16.8	0.1 ± 0.2	0.0 ± 0.0

†Values are mean ± SD. ** ; $p < 0.01$, * ; $p < 0.05$ vs. baseline, paired t-test or Wilcoxon signed-rank test. NaCl ; sodium chloride.

Changes in 24-h urinary parameters

Table 3 shows changes in 24-h urinary parameters for 6 months. Urine volume significantly increased at 6 months compared with baseline ($p<0.05$). Urine volume at 6 months was 2,196 ± 740 mL/day, which exceeded the recommended urine volume (≥2,000 mL/day) in the guideline (14). U-Cr and U-UA levels decreased significantly at 6 months compared with baseline ($p<0.05$). In comparison to baseline, FE_{UA} increased significantly at 6 months ($p<0.05$). Other parameters did not show any significant variations. Men had similar results, while women did not show any significant differences.

Relationship between ΔS-UA and Δclinical variables

Figure 1 shows the relationship between changes in S-UA and changes in clinical variables at 6 months from baseline. A positive correlation was shown between ΔS-Cr and ΔS-UA ($R = 0.435$; $p<0.05$). ΔeGFR and ΔFE_{UA} were negatively associated with ΔS-UA ($R = -0.352$, $R = -0.368$, respectively ; $p<0.05$).

Changes in S-UA level in subgroup analyses

Table 4 shows changes in S-UA level at baseline and 6 months in subgroup analyses. Except for subgroups which were small number of subjects, we observed significant decreases in S-UA level at 6 months in most subgroups.

Table 3. Changes in 24-h urinary parameters by dietary counseling for 6 months†

		All (n = 33)		Men (n = 24)		Women (n = 9)	
		Baseline	6 months	Baseline	6 months	Baseline	6 months
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Volume	(mL)	1,815 ± 686	2,196 ± 740 *	1,875 ± 737	2,359 ± 733 **	1,656 ± 529	1,763 ± 595
pH		6.1 ± 0.5	6.2 ± 0.5	6.0 ± 0.5	6.2 ± 0.5	6.2 ± 0.6	6.0 ± 0.5
U-Cr	(mg/dL)	76.1 ± 42.6	59.4 ± 32.1 *	88.6 ± 43.5	64.9 ± 32.7 *	42.8 ± 10.3	44.7 ± 26.6
U-Alb	(mg/L)	14.1 ± 27.9	12.9 ± 23.7	9.3 ± 18.4	10.3 ± 25.2	26.8 ± 43.4	20.0 ± 18.6
U-UA	(mg/dL)	35.0 ± 16.1	28.4 ± 13.9 *	38.6 ± 16.7	29.6 ± 13.3 *	25.2 ± 9.5	25.3 ± 15.9
U-Xa/Hx	(μg/mL)	28.1 ± 21.7	35.5 ± 48.8	33.3 ± 22.4	41.3 ± 55.0	14.2 ± 12.1	19.9 ± 21.6
C_{Cr}	(mL/min/1.73 m²)	92 ± 37	88 ± 34	102 ± 35	97 ± 31	67 ± 30	65 ± 30
C_{UA}	(mL/min/1.73 m²)	5.3 ± 1.8	5.7 ± 2.1	5.6 ± 1.6	6.0 ± 1.8	4.4 ± 2.0	4.9 ± 2.5
FE_{UA}	(%)	6.0 ± 1.4	6.7 ± 1.8 *	5.7 ± 1.4	6.4 ± 1.8 *	6.7 ± 1.1	7.5 ± 1.7

†Values are mean ± SD. ** ; $p< 0.01$, * ; $p< 0.05$ vs. baseline, paired t-test or Wilcoxon signed-rank test. U-Cr ; urinary creatinine, U-Alb ; urinary albumin, U-UA ; urinary uric acid, U-Xa/Hx ; urinary xanthine/hypoxanthine, C_{Cr} ; creatinine clearance, C_{UA} ; uric acid clearance, FE_{UA} ; renal fractional excretion of uric acid.

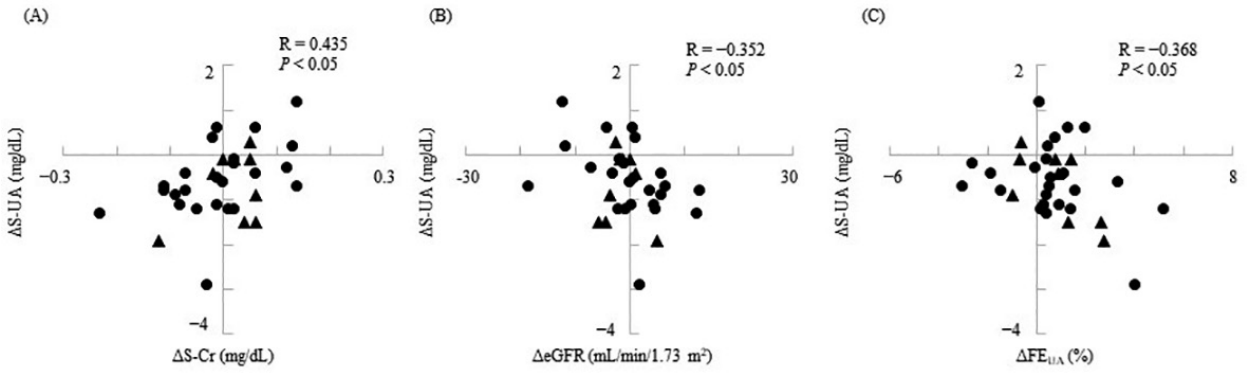

Figure 1. Relationship between ΔS-UA and Δclinical variables.
n=33. ΔS-UA and Δclinical variables were changes at 6 months from baseline.
Men is indicated by circles, women by triangle.
S-UA ; serum uric acid, S-Cr ; serum creatinine, eGFR ; estimated glomerular filtration rate, FE_{UA} ; renal fractional excretion of uric acid.
All data were analyzed by Pearson's product-moment correlation coefficient.

Table 4. Changes in S-UA level at baseline and 6 months in subgroup analyses

			Baseline	6 months
		n	Mean ± SD	Mean ± SD
Age				
	<70 years	18	7.8 ± 0.4	7.2 ± 1.0 *
	≥70 years	15	7.6 ± 0.4	7.0 ± 0.7 **
BMI				
	≥25 kg/m^2	11	7.7 ± 0.4	7.0 ± 1.2 *
	18.5−25 kg/m^2	21	7.8 ± 0.4	7.3 ± 0.6 **
Disease				
Diabetes	Yes	6	7.9 ± 0.3	7.7 ± 0.9
	No	27	7.7 ± 0.4	7.0 ± 0.9 **
Hyperlipidemia	Yes	20	7.7 ± 0.4	7.0 ± 1.1 **
	No	13	7.7 ± 0.3	7.3 ± 0.6 *
Hypertension	Yes	26	7.7 ± 0.4	7.0 ± 1.0 **
	No	7	7.9 ± 0.5	7.5 ± 0.4
Decreased renal function	eGFR 30−60 mL/min	20	7.7 ± 0.4	7.1 ± 0.9 **
	eGFR >60 mL/min	13	7.7 ± 0.4	7.1 ± 0.9 *
Classification of hyperuricemia	Renal overload type	5	7.8 ± 0.3	7.3 ± 0.8
	Renal underexcretion type	27	7.7 ± 0.4	7.1 ± 1.0 **
Lifestyle habits				
Smoking	Yes	5	7.9 ± 0.4	7.7 ± 0.7
	No	28	7.7 ± 0.4	7.0 ± 0.9 **
Alcohol drinking	Yes	11	7.9 ± 0.3	6.9 ± 0.9 **
	No	22	7.6 ± 0.4	7.2 ± 0.9 *

†Values are mean ± SD. ** ; $p< 0.01$, * ; $p< 0.05$ vs. baseline, paired t-test or Wilcoxon signed-rank test. BMI ; body mass index, S-UA ; serum uric acid, eGFR ; estimated glomerular filtration rate. One subject with BMI <18.5 and one subject with the combined type were excluded.

DISCUSSION

In this study, we examined the effect of dietary counseling for 6 months on the S-UA level and various parameters.

A previous study reported that an increase in S-UA level was associated with a decrease in eGFR and suggests that maintaining S-UA level in a normal range was essential in eGFR decline within a normal range (2, 3). Addtionally, UA-lowering therapy might retard the progression of CKD, although adequately powered randomized trials are required to evaluate the benefits and risks of UA-lowering therapy in CKD (21, 22). We observed that changes in the renal function (ΔS-Cr, ΔeGFR, and ΔFE_{UA}) were significantly associated with ΔS-UA level, and our result suggested that lowering S-UA level by dietary counseling might be associated with the maintenance of renal function.

The intestine and kidney maintain S-UA homeostasis, and approximately 70% of UA is excreted from the kidney (23). About 90% cases of hyperuricemia arise as a result of underexcretion (24), and there were 27 (82%) subjects with renal underexcretion type in this study. Thus, improving UA excretion from the kidney is vital to managing the S-UA level. This study showed that water intake and urine volume significantly increased at 6 months compared with baseline. The U-UA level decreased significantly and the FE_{UA} level increased significantly at 6 months compared to baseline. A previous study found that the state of hydration of the extracellular fluid volume (ECFV) influences net urate reabsorption. These changes are mediated by alterations in proximal tubule reabsorption rates (25). Therefore, an increase in water intake and urine volume by dietary counseling may contribute to S-UA level control through alterations in ECFV.

Furthermore, we demonstrated that at 6 months, urine volume was 2,196 ± 740 mL/day and exceeded the recommended urine volume (2,000 mL/day) in the guideline (14). A study reported that the recurrence rate was significantly lower in the intervention group that involved a high intake of water (≥2,000 mL/day) than the non-intervention group among patients with idiopathic calcium kidney stones (26). Urinary lithiasis complications are common in hyperuricemia and gout patients. It is critical to provide enough urine volume and reduce U-UA level to avoid increasing S-UA level. Our findings showed that an increased water intake and urine volume through dietary counseling might decrease U-UA levels and increase FE_{UA}.

It is well known that there is a sex difference in S-UA levels, and S-UA levels are also known to increase in women after menopause (27). In this study, the sex ratio of men/women (%) was 72.7/27.3, and the women subjects were ≥60 years although subjects in this study were intervened regardless of gender or age. We performed a subgroup analysis to evaluate sex-related differences and observed a significant decrease in S-UA level at 6 months in both sexes (Table 1). Furthermore, we also needed to

consider whether there are age-related differences. We observed a significant decrease in S-UA level at 6 months in both age groups (<70, and ≥70 years) (Table 4). These results indicated that dietary counseling is effective regardless of age or gender.

We showed that BW and AC were reduced at 6 months compared with baseline. In the subjects with reduced BW and AC, the S-UA level decreased significantly at 6 months ($p<0.01$). Hyperuricemia is closely associated with obesity and metabolic syndrome (4, 5). A previous systematic review demonstrated the beneficial effects of weight loss for overweight gout patients in terms of S-UA, achieving S-UA target, and gout attacks (28). In addition, we showed that the total fat intake decreased significantly at 6 months compared to baseline ($p<0.01$). In the subjects with decreased fat intake, BW tended to decrease (baseline, 68.9 ± 9.3 ; 6 months, 67.6 ± 8.9 kg, $p=0.05$), and the S-UA level decreased significantly at 6 months (baseline, 7.8 ± 0.4 ; 6 months, 7.1 ± 1.0 mg/dL, $p<0.01$). These results suggested that reducing fat intake by dietary counseling helps to improve obesity and reduce the S-UA level. We performed a subgroup analysis to evaluate BMI-group-related differences, and reductions of BW and AC were only seen in subjects with a high BMI group (≥25 kg/m^2) (BW : baseline, 73.7 ± 5.6 ; 6 months, 71.2 ± 6.5 kg, AC : baseline, 95.8 ± 8.2 ; 6 months, 93.0 ± 8.9 cm, $p<0.05$). Thus, this result suggested that dietary counseling by well-trained registered dietitians could bring such changes only in subjects who needed to lose weight.

Alcohol intake might be associated with an increased risk of gout in a dose-dependent manner (11). In addition, habitual alcohol intake significantly contributed to the development of hyperuricemia in Japanese men, regardless of the type of alcoholic beverage consumed (12). In a subgroup analysis, alcohol intake tended to decrease at 6 months compared to baseline in 11 drinkers ($p = 0.066$). The S-UA level was significantly decreased at 6 months in them ($p<0.01$, Table 4), and changes in the S-UA level tended to differ between drinking habits or not (drinker, −1.0 ± 0.8 ; non-drinker, −0.4 ± 0.8 mg/dL, $p = 0.069$). These results suggested that limiting alcohol intake by dietary counseling might be beneficial in lowing the S-UA level. However, some subjects were unable to decrease alcohol intake in the study, and it is necessary to consider the non-adherence of limiting alcohol intake. In a previous study, green tea catechins can enhance the excretion of UA and Xa/HX, even though alcohol is ingested (29). Thus, subjects with poor adherence need to provide an alternative approach to minimize an adverse effect of alcohol intake on the S-UA level.

Our study has several strengths. We assessed the S-UA level and various parameters by using blood and urine analyses and dietary surveys. In addition, all subjects undergo monthly dietary counseling by well-trained registered dietitians for 6 months without drug therapy to evaluate the effect of dietary counseling. In contrast, several limitations of our study should be considered. Although purine contents of many foods have been quantified (30), purine contents of all foods have not been determined. Therefore, we could not estimate purine intake in this study accurately, and it is necessary to establish a method for assessing purine intake. In addition, our study is observational, and we did not examine non-intervention conditions. It is necessary to evaluate the difference in reduction of S-UA level between dietary intervention and non-intervention to determine the effect of dietary counseling for asymptomatic hyperuricemia.

In this study, the average reduction in S-UA level was 7.8% at 6 months, and the S-UA level was significantly decreased by dietary counseling in line with the guideline. Furthermore, our result suggested that lowering S-UA level by dietary counseling might be associated with the maintenance of renal function. Before commencing drug therapy, correcting dietary habits by dietary counseling is predicted to reduce the S-UA level, prevent complications, and minimize medical costs. This study illustrates the effectiveness of dietary counseling for asymptomatic hyperuricemia.

CONFLICT OF INTERESTS-DISCLOSURE

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