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# Analysis of Correlation between 24-Hour Urinary Sodium and the Degree of Blood Pressure Control in Patients with Chronic Kidney Disease and Non-Chronic Kidney Disease

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We investigated the association between 24-hr urinary sodium (24UNA) and adequacy of blood pressure (BP) control in patients with chronic kidney disease (CKD) and nonCKD. All data were collected retrospectively by accessing the electrical medical records in patients with 24-hr urine collection and serum creatinine. Enrolled 400 subjects were subgrouped by the amount of 24UNA, or CKD stage. The appropriate BP was defined as BP < 130/80 mmHg for subjects with proteinuria, and BP < 140/90 mmHg for subjects without proteinuria. The mean level of 24UNA was 166 ± 76 mEq/day. The 24UNA group was an independently related factor to diastolic BP as a continuous variable. The rate of appropriate BP control in patients with proteinuria was highest in 24UNA < 100 mEq/L ($P$ = 0.012). The odds to fail achievement of BP target in subjects with 24UNA ≥ 90 mEq/day was 2.441 (1.249-4.772, $P$ = 0.009) higher than that of 24UNA < 90 mEq/day among participants with proteinuria. There was difference in the amount of 24UNA between CKD and non-CKD except each stage of CKD group. In conclusion, salt intake estimated by 24-hr urine sodium excretion is a risk factor to achieve appropriate BP control.

**Keywords:** Salt; Hypertension; Blood pressure; Renal insufficiency

## INTRODUCTION

High sodium intake increases blood pressure (BP) and proteinuria, induces glomerular hyperfiltration (1), and negatively affects renal outcome (2). Lowering salt intake not only reduces BP, but also lowers albuminuria. A low salt diet (5 g per day) significantly reduced 24-hr urinary protein excretion by 19% and decreased systolic BP (SBP) and diastolic BP (DBP) by 8 mmHg and 3 mmHg, respectively (3). However, few studies have investigated the direct effect of salt on the incidence of end-stage renal disease, a hard end-point of renal function.

According to guidelines for diabetic and non-diabetic patients with chronic kidney disease (CKD), an albuminuria < 30 mg/day necessitates a BP target consistently below 140/90 mmHg and an albuminuria level over 30 mg/day induces a BP target consistently below 130/90 mmHg (4). Except for anti-hypertensive medication, modifiable risk factors to increased blood pressure are life styles such as salt intake, obesity, and exercise. The recommended amount of daily salt intake for individuals with cardiovascular risks is < 90 mM ( < 2 g) of sodium (corresponding to 5 g of sodium chloride) in adults (4).

The 24-hr urinary sodium (24UNA) analysis is a standard method for estimating the daily sodium intake. A few studies have investigated the association between 24UNA and surrogate markers of renal outcome. In patients with metabolic syndrome, the salt intake estimated by 24UNA was associated with hypertension (5). In patients with hypertension, there was also an association of 24UNA and blood pressure (6). However, no study has examined the association between 24UNA and surrogate markers for renal outcome in CKD. Therefore, we estimated the salt intake by 24UNA and observed the degree of BP controlled by the daily salt intake.



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## MATERIALS AND METHODS

### Populations

All data were collected retrospectively by accessing the electrical medical records of patients in Seoul National University Hospital. Among 1,363 outpatients whose serum creatinine and 24-hr urinary sodium were measured, we selected outpatients who collected 24-hr urine, appropriately. We defined an appropriate urine collection as the ratio of measured 24-hr urinary creatinine to estimated 24-hr urinary creatinine, 0.75-1.25. We also excluded patients who had been prescribed diuretics or fluid therapy, which may influence 24-hr urinary sodium excretion. The number of enrolled patients for this analysis was 400.

### Definitions

The 24UNA groups were defined by the level of 24-hr urinary sodium (group 1: < 100 mEq/day, group 2: ≥ 100 mEq/day, < 200, group 3: ≥ 200 mEq/day). CKD was defined as 24-hr urine protein 150 mg/day (proteinuria) or more and/or estimated GFR < 60 mL/min/1.73 $m^2$ (7). The GFR was estimated by the equation of the 2009 CKD-EPI creatinine equation (8). CKD groups were classified by the estimated GFR (CKD group - non-CKD, CKD stage 1, CKD stage 2, CKD stage 3a, CKD stage 3b, CKD stage 4, and CKD stage 5).

We used two BP criteria for appropriate BP control according to KDIGO guideline (4). The BP target 1 with SBP < 130 and DBP < 80 mmHg was applied to patients with proteinuria and the other BP target 2, with SBP < 140 and DBP < 90 mmHg, for patients without proteinuria. Hypertension was defined as SBP of ≥ 140 mmHg, DBP of ≥ 90 mmHg, a self-reported history of HTN, or use of antihypertensive medications. Diabetes mellitus (DM) was defined as a fasting glucose of 126 mg/dL, a self-reported history of DM, or use of hypoglycemic agents. The amount of 24-hr urinary creatinine was estimated by the equation ([28-(0.2 × age)] × body weight (kg), { × 0.85 if female}, [mg/day] (9).

### Statistics

We used Student's *t*-test or one-way ANOVA test for continuous data according to the number of subgroups and Fischer's exact chi-square test for categorical data. The continuous data were summarized as mean ± standard deviation values. Pearson's correlation coefficients were calculated to determine the related factors for BP targets. Multiple logistic regression analyses were conducted to determine the independent risk factors to target BP adjusted related factors. The blood pressures were estimated among groups by a covariate analysis (ANCOVA) adjusted with related factors to blood pressure. Statistical analyses were performed with SPSS version 20.0 (SPSS Inc., Chicago, IL, USA). Two-sided *P* values were used in all statistical analyses. A *P* value < 0.05 was considered statistically significant.

### Ethics statement

This study was approved by the institutional review board of the Seoul National University Hospital (IRB No. H-1306-030-496). Informed consent was exempted by the board.

## RESULTS

### Participants' characteristics

The mean age was 48.8 ± 15.0 yr. The female:male ratio was 204:196. The proportion of DM patients was 17.3% (69/400) and that of patients with hypertension was 58.0% (232/400). The mean estimated GFR was 85.2 ± 34.3 mL/min/1.73 $m^2$. The mean amount of 24UNA was 166 ± 76 mEq per day. The mean ratio of 24-hr urinary creatinine and estimated creatinine was 1.01 ± 0.13. The 24UNA groups 1 (24UNA < 100 mEq/L), group 2 (24UNA 100-199 mEq/day) and group 3 (24UNA ≥ 200 mEq/L) comprised of 71,223, and 106 subjects, respectively. There was no difference in the prevalence of DM, hypertension, coronary heart disease, and cerebrovascular disease. The proportion of men and obesity and the levels of DBP, ALT, serum albumin, glucose, estimated GFR, daily urine volume, and 24-hr creatinine were highest in 24UNA group 3 (Table 1). There was no difference in the proportion of patients taking anti-hypertensive medication among groups (*P* = 0.776).

### The risk factors have an effect on blood pressure

The variables associated with SBP independently were age, BMI, serum glucose, ALT and the amount of 24-hr urinary protein. There was no correlation with 24UNA (Table 2). Estimated SBP was 122 (118-126) mmHg in group 1, 125 (123-127) mmHg in group 2, and 125 (122-128) mmHg in group 3 by ANCOVA adjusted with those related factors (*P* = 0.381) (Fig. 1).

The variables associated with DBP independently were CKD, serum glucose, and 24UNA group. The estimated DBP was 73 (70-76) mmHg in group 1, 74 (72-75) mmHg in group 2, and 77 (74-79) mmHg in group 3 by ANCOVA adjusted with those related factors (*P* = 0.050). There was a difference between groups 1 and 3 (*P* = 0.030) by post-hoc analysis by Fisher's least significance difference (Fig. 1).

### The association between 24UNA and achieved targets for blood pressure

*For patients with proteinuria; the degree of blood pressure control (target BP < 130/80 mmHg)*

The frequency of BP below 130/80 mmHg was 70.4% (50/71) in 24UNA group 1, 59.6% (130/218) in group 2 and 48.1% (50/104) in group 3 (*P* = 0.012) (Fig. 2). We demonstrated the association of the following risk factors with not target BP: history of cerebrovascular disease and cardiovascular disease, DM, BMI, serum glucose, ALT, AST, and 24-hr urinary protein. In multiple logistic regression analysis adjusted with these variables, sub-


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**Table 1.** Clinical characteristics of 24UNA group

| Variables | 24-hr urinary Na (mEq/day) | | | *P* value |
|---|---|---|---|---|
| | < 100 | 100-199 | ≥ 200 | |
| Number | 71 | 223 | 106 | Ns |
| Age (yr) | 48.9 ± 15.9 | 50.1 ± 15.0 | 46.1 ± 13.9 | 0.077 |
| Male (%) | 45.1 | 45.3 | 67 | 0.001 |
| DM (%) | 18.3 | 15.2 | 20.8 | 0.450 |
| HTN (%) | 64.8 | 55.2 | 59.4 | 0.337 |
| CHD (%) | 4.2 | 0.9 | 4.7 | 0.069 |
| CVD (%) | 4.2 | 3.1 | 2.8 | 0.868 |
| BMI (kg/m$^2$) | 22.3 ± 3.1 | 24.0 ± 3.5 | 25.4 ± 3.6 | < 0.001 |
| SBP (mmHg) | 121 ± 14 | 125 ± 16 | 127 ± 17 | 0.061 |
| DBP (mmHg) | 73 ± 11 | 74 ± 10 | 74 ± 13 | 0.008 |
| ALT (U/L) | 17 ± 8 | 20 ± 14 | 24 ± 16 | 0.007 |
| AST (U/L) | 20 ± 9 | 21 ± 8 | 22 ± 8 | 0.289 |
| Cholesterol (mg/dL) | 190 ± 61 | 182 ± 41 | 185 ± 37 | 0.457 |
| Glucose (mg/dL) | 95 ± 15 | 96 ± 23 | 110 ± 58 | 0.002 |
| Protein (g/dL) | 6.9 ± 0.9 | 7.1 ± 0.6 | 7.1 ± 0.7 | 0.067 |
| Albumin (g/dL) | 4.0 ± 0.7 | 4.2 ± 0.5 | 4.2 ± 0.5 | 0.031 |
| Creatinine (mg/dL) | 1.38 ± 1.33 | 1.26 ± 1.31 | 1.03 ± 0.73 | 0.126 |
| GFR (mL/min/1.73 m$^2$) | 80.3 ± 38.5 | 83.2 ± 33.9 | 93.0 ± 33.9 | 0.023 |
| ≥ 90 | 50.0 | 53.4 | 66.7 | 0.166 |
| 60-89 | 20.0 | 22.6 | 15.2 | |
| 45-59 | 11.4 | 6.8 | 8.6 | |
| 30-44 | 2.9 | 6.8 | 3.8 | |
| 15-29 | 8.6 | 5.0 | 4.8 | |
| < 15 | 7.1 | 5.4 | 1.0 | |
| 24 hr urine (/day) | | | | |
| Volume (L) | 1.28 ± 0.63 | 1.96 ± 1.52 | 2.76 ± 2.25 | < 0.001 |
| Creatinine (g) | 0.98 ± 0.30 | 1.05 ± 0.30 | 1.30 ± 0.40 | < 0.001 |
| Na (mEq) | 65.9 ± 23.7 | 153.3 ± 27.7 | 262.5 ± 51.0 | < 0.001 |
| Protein (g) | 1.83 ± 5.25 | 0.78 ± 1.36 | 1.15 ± 2.49 | 0.021 |
| Protein ≥ 150 mg/day (%) | 66.2 | 58.0 | 61.0 | 0.464 |
| Non-CKD | 25.7 | 37.6 | 39.4 | |
| CKD stage 1 | 32.9 | 25.2 | 29.8 | |
| CKD stage 2 | 11.4 | 12.8 | 12.5 | |
| CKD stage 3a | 11.4 | 6.9 | 8.7 | 0.351 |
| CKD stage 3b | 2.9 | 6.9 | 3.8 | |
| CKD stage 4 | 8.6 | 5.0 | 4.8 | |
| CKD stage 5 | 7.1 | 5.5 | 1.0 | |

CHD, coronary heart disease-stable or unstable angina, acute or old myocardial infarction, CVD, hemorrhagic or non-hemorrhagic cerebrovascular disease

**Table 2.** Correlation between BPs and other factors by multiple linear regression model

| Variables | | B | 95% CI for B | | *P* value |
|---|---|---|---|---|---|
| For SBP* | Glucose (mg/dL) | 0.075 | 0.031 | 0.119 | 0.001 |
| | BMI (kg/m$^2$) | 0.527 | 0.093 | 0.961 | 0.017 |
| | 24HU protein (mg/day) | 0.001 | 0.000 | 0.001 | 0.007 |
| | Age (yr) | 0.131 | 0.026 | 0.237 | 0.015 |
| | ALT (U/L) | 0.114 | 0.004 | 0.224 | 0.043 |
| For DBP† | Glucose (mg/dL) | 0.049 | 0.018 | 0.081 | 0.002 |
| | CKD (presence) | 2.942 | 0.617 | 5.267 | 0.013 |
| | 24HUNA group‡ | 1.965 | 0.264 | 3.665 | 0.024 |

*For SBP: adjusted with age, BMI, glucose, cholesterol, 24-hr urinary protein, ALT, GFR, uric acid, history of CVD and cancer, and 24-hr urinary sodium group, which were related factors to SBP by correlation coefficient; †For DBP: adjusted with BMI, glucose, cholesterol, 24-hr urinary protein, ALT, presence of CKD, and 24-hr urinary sodium group, which were related factors to SBP by correlation coefficient; ‡24HUNA group: 24-hr urinary sodium grouped by the criteria of 100 and 200 mEq/day.

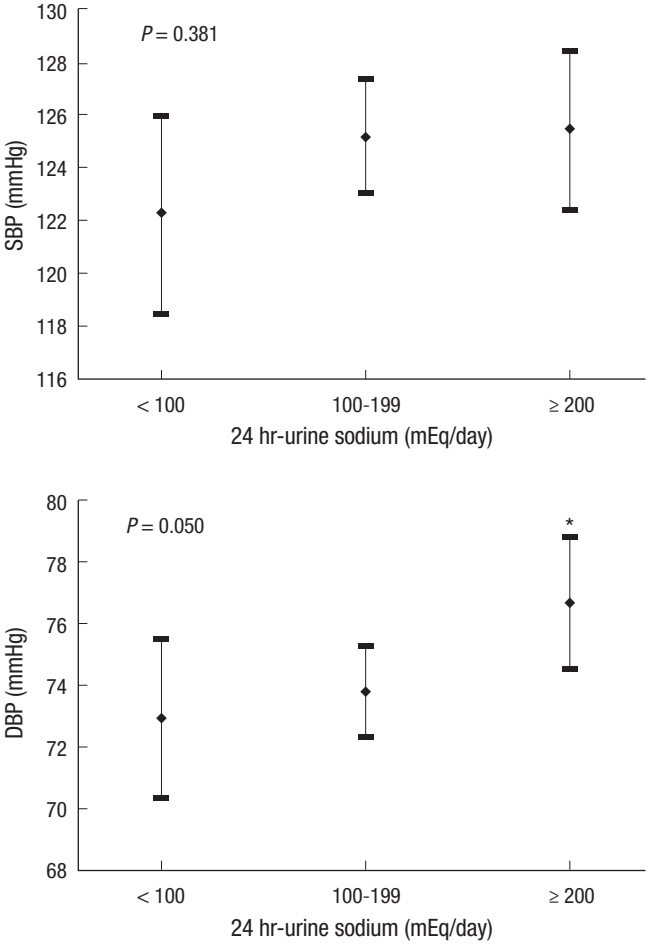


**Fig. 1.** The estimated levels of blood pressures in 24UNA group adjusted with related factors (see Table 2) by ANCOVA test. *$P$ value = 0.030 compared to group with 24 hr-urine sodium < 100 mEq/day. The bar means the 95% confidence interval of estimated value in each group.

groups divided by the amount of 24UNA 80 mEq/day or 90 mEq/day were associated with achievement of BP target (Table 3). The higher excretion of 24UNA was, the higher risk of failure to achieve BP target. The odds to fail achievement of BP target in subjects with 24UNA ≥ 90 mEq/day was 2.441 (1.249-4.772) ($P$ = 0.009) higher than those of 24UNA < 90mEq/day. The odds to fail achievement of BP target in subjects with 24UNA ≥ 80 mEq/day was 2.894 (1.310-6.395) ($P$ = 0.009) higher than those of 24UNA < 90 mEq/day.

*For patients without proteinuria; the degree of blood pressure control (the target BP < 140/90 mmHg)*

The frequency of B$P$ < 140/90 mmHg was 87.3% (62/71) in 24UNA group 1, 81.7% (178/218) in group 2 and 75.0% (78/104) in group 3 ($P$ = 0.115) (Fig. 2). We analyzed the risk factors that resulted in failure to achieve the target BP and revealed that the factors of DM, cholesterol, albumin, glucose, ALT, AST, GFR, and 24-hr urinary protein, were associated. In multiple logistic regression analysis adjusted with these variables, the group di-

**Table 3.** Correlation between BP targets and 24-hr urinary sodium parameters by multiple logistic regression model in all participants

| | 24-hr urinary sodium | B | Wald | OR | 95% CI for B | | *P* value |
|---|---|---|---|---|---|---|---|
| For SBP ≥ 130 or DBP ≥ 80* | 24UNa ≥ 80 mEq/day | 1.063 | 6.902 | 2.894 | 1.310 | 6.395 | 0.009 |
| | 24UNa ≥ 90 mEq/day | 0.893 | 6.812 | 2.441 | 1.249 | 4.772 | 0.009 |
| | 24UNa ≥ 100 mEq/day | - | - | - | - | - | 0.108 |
| | 24UNa (in mEq/day) | - | - | - | - | - | 0.364 |
| | 24UNa group | - | - | - | - | - | 0.213 |
| | 100-199 mEq/day | - | - | - | - | - | 0.771 |
| | ≥ 200 mEq/day | - | - | - | - | - | 0.282 |
| For SBP ≥ 140 or DBP ≥ 90† | 24UNa ≥ 80 mEq/day | 1.188 | 4.216 | 3.282 | 1.055 | 10.203 | 0.040 |
| | 24UNa ≥ 90 mEq/day | - | - | - | - | - | 0.086 |
| | 24UNa ≥ 100 mEq/day | - | - | - | - | - | 0.116 |
| | 24UNa (in mEq/day) | - | - | - | - | - | 0.234 |
| | 24UNa group | - | - | - | - | - | 0.224 |
| | 100-199 mEq/day | - | - | - | - | - | 0.788 |
| | ≥ 200 mEq/day | - | - | - | - | - | 0.265 |

*For BP target, 130/80 mmHg, adjusted with history of cerebrovascular disease and cardiovascular disease, DM, BMI, serum glucose, ALT, AST, and 24-hr urinary protein; †For BP target, 140/90 mmHg, adjusted with DM, cholesterol, albumin, glucose, ALT, AST, GFR, and 24-hr urinary protein.

**Table 4.** Descriptive statistics of 24-hr urinary sodium in CKD group

| Variables | Number | 24-hr urinary Na (mEq/day) | | | |
|---|---|---|---|---|---|
| | | Mean | SD | Range | |
| Non-CKD | 141 | 174 | 70 | 23 | 444 |
| CKD stage 1 | 109 | 166 | 77 | 5 | 376 |
| CKD stage 2 | 49 | 170 | 78 | 14 | 396 |
| CKD stage 3 | 53 | 163 | 77 | 29 | 373 |
| CKD stage 4 | 22 | 148 | 77 | 16 | 269 |
| CKD stage 5 | 18 | 132 | 52 | 38 | 227 |

*$P$ value = 0.235 by ANOVA test.

vided by the amount of 24UNA 80 mEq/day was associated with achievement of BP target (Table 3). The more excretion of 24UNA was, the higher risk of failure to achieve BP target. The odds to fail achievement of BP target in subjects with 24UNA ≥ 80 mEq/day was 2.441 (1.249-4.772) ($P$ = 0.040) higher than those of 24UNA < 80 mEq/day (Table 3).

**The amount of 24-hr urine sodium according to CKD stages**

There was no difference of the amount of 24UNA according to CKD stages (Table 4, $P$ = 0.235). The proportion of subjects with 24UNa < 100 mEq/day differed when we compared non-CKD with CKD (12.8% vs. 20.7%, $P$ = 0.049). However, this difference disappeared in the comparison between non-CKD group with each stage-group of CKD (Fig. 3). We estimated the amount of 24UNA within CKD stages by the ANCOCA adjusted with meaningful variables such as BMI, DBP, serum albumin, uric acid, glucose and 24-hr urinary creatinine and found that there was no difference in the amount of 24UNA between non-CKD and each CKD stage ($P$ = 0.677) (Fig. 4).

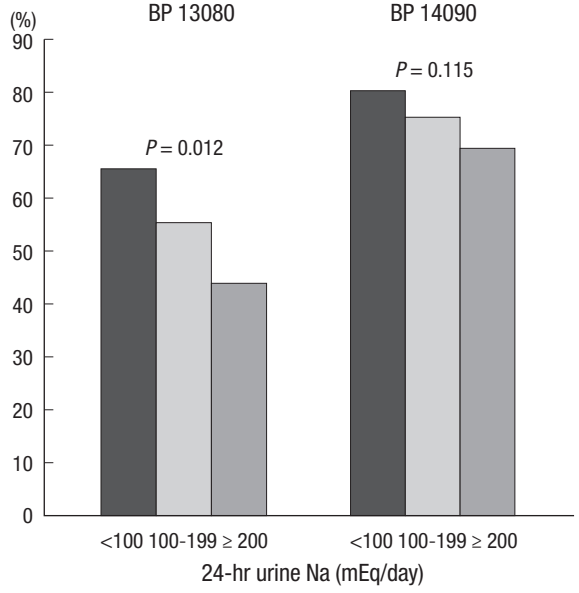


**Fig. 2.** Frequency of BP<130/80 mmHg or<140/90 mmHg among all participants according to levels of 24-hr urine sodium.

## DISCUSSION

The feasible methods to estimate the sodium intake are dietary survey and 24-hr urine collection. Dietary survey tends to underestimate sodium intake due to the difficulty of quantifying sodium intake (10). The 24-hr urine collection method is now accepted as the gold standard to estimate the sodium intake (11). However, it can be biased if the participant collects urine incompletely. To overcome incomplete sampling, urinary creatinine is also measured with sodium (12). In the present study, we judged the completeness by comparing the measured and calculated creatinine. The ratio of those values was 1.01 ± 0.13 and the mean 24UNA value of 166 ± 76 mEq per day was similar with the mean value of 166.4 ± 68.1 mEq per day for 24-hr urinary sodium in residents of a randomly selected city (13). Therefore although the data were extracted in the hospital, they had epidemiological value.

In patients with CKD, hypertension is both a cause and com-


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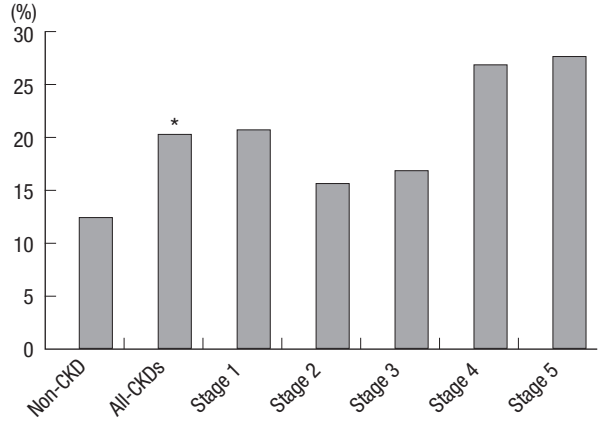


**Fig. 3.** The prevalence of 24-hr urine sodium < 100 mEq in each group of chronic kidney disease (CKD). *$P$ < 0.05 compared to Non-CKD group.

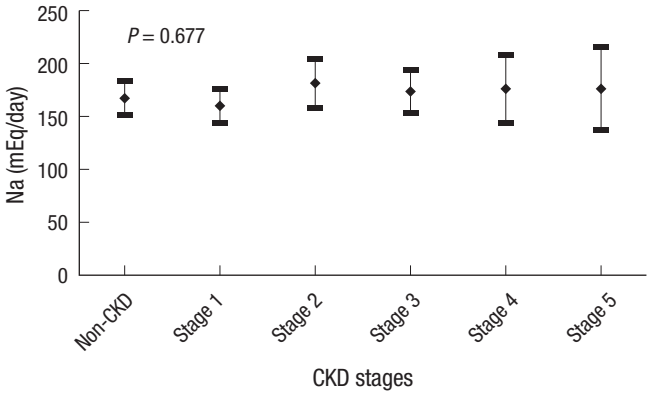


**Fig. 4.** The estimated value of 24-hr urine sodium in CKD groups adjusted with related factors, such as history of cancer, DBP, BMI, glucose, uric acid, serum albumin stratified with 3.0 g/dL, and 24-hr urine protein, by ANCOVA test. The bar means the 95% confidence interval of estimated value in each group.

plication of CKD. It is the most important risk factor for the progression of CKD and the occurrence of cardiovascular disease (14). In addition, proteinuria is evidence of kidney injury, CKD progression and a risk factor of cardiovascular disease (15). Hence many studies on CKD progression have used hypertension and proteinuria as surrogate markers for renal outcome of end-stage renal disease. High dietary salt intake increases the risk of high BP and worsens existing hypertension (16) and proteinuria (3). Many studies have investigated the associations with salt intake estimated by 24-hr urine collection and BP in both the general population and in patients with hypertension (16). However, not many studies have examined patients with other diseases. Especially, no study has investigated the associations with salt intake estimated by the 24-hr urine collection and surrogate markers in CKD and in nonCKD. Therefore, we observed such associations in CKD. We found an association of salt intake estimated by the 24-hr urine collection, with DBP.

When humans consume salt, the volume of extracellular fluid expands. In turn, BP is increased by the increase of cardiac output (17). So if patients with CKD have a high salt intake, hypertension may occur and the kidney function can be degraded. One meta-analysis of this result has been conducted (2). Following much evidence such as this study, a recommendation of low salt intake was made in the KDIGO 2012 CKD management guideline (4). This guideline recommends that patients with CKD have a daily salt intake below 5 g. Unlike other minerals, there is no recommendation for estimated average requirement for beneficial effects and an upper tolerance level for adverse effects. Sodium has only a standard for requirement of adequate intake. Therefore, we could not determine the level of salt intake that has a bad effect on hypertension and proteinuria. How much salt should humans consume to avoid the bad effects of BP and proteinuria? No study has examined salt intake with adverse effects on BP and proteinuria in CKD patients. Recommended guidelines of salt intake from an evidence-based study cannot be extended to patients with CKD. In the present study, we observed an association between salt intake estimated by 24UNA and the target of BP (130/80,140/90 mmHg). We found that a 24-hr urinary sodium level below 100 mEq/L resulted in a proportion of target BP up to 70%. When 24UNA was over 80 mEq/L, the odds of BP over 130/90 mmHg was 2.4 times in patients with CKD and nonCKD. In this context, we suggest that patients with CKD should limit their sodium intake below 100 mEq/L (2.5 gram Salt), and preferably below 80 mEq/L, to control for target BP.

The fraction of sodium excretion is increased to maintain the sodium balance because of the loss of functioning nephrons. Therefore, as kidney function deteriorates, sodium excretion is known to be increased (1). Hence the 24 hr urine sodium cannot be a good indicator of the sodium intake in patients with CKD. In the present study, however, there was difference in sodium excretion fraction between non-CKD and CKD except CKD stages. Therefore, 24UNA is thought to be a useful guide for salt intake in patients with CKD.

The present study was limited by its retrospective design. The information from the 3rd referral hospital may have produced a selection bias.

In conclusion, salt intake estimated by 24-hr urine sodium excretion is a risk factor to achieve appropriate BP control.

## DISCLOSURE

The authors declare that they have no conflicts of interest to disclose.

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