


Characteristics of urinary sodium excretion in patients with chronic kidney disease in Jiangsu, China

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Abstract

The current study aimed to assess the dietary salt intake in patients with CKD in Jiangsu province and investigate the relationship of urinary sodium excretion with blood pressure. A total of 800 patients with CKD stages 1–4 were recruited. All enrolled patients were asked to collect complete 24-h urine specimen. At the same time, patient's demographic and laboratory data were recorded. The mean age was 47.45 ± 15.25 years old, including 423 men and 377 women. There was no significant difference in urinary sodium excretion among different stages of CKD ($p = .748$). This study revealed that the median urinary sodium excretion of all patients was 127.20 mmol/d (IQR 91.03–172.06), corresponding to a salt intake of 7.4 g/d. Among them, only 167 (20.9%) cases had salt intake <5 g/d. Moreover, urinary sodium excretion in overweight group and obese group was higher than that in normal weight group ($p = .001$, $p < .001$). Likewise, urinary sodium excretion in men was higher than that in women ($p < .001$). Spearman correlation analysis indicated that urinary sodium excretion positively correlated with urinary protein excretion ($r = .178$, $p < .001$), SBP ($r = .109$, $p = .002$), and DBP ($r = .086$, $p = .015$). After adjusting for age, gender, BMI, eGFR, urinary protein excretion, and history of taking antihypertensive drug, multivariate linear regression demonstrated that higher level of urinary sodium excretion associated with increased level of SBP, DBP, and MAP ($\beta = 0.020$, $p = .049$; $\beta = 0.015$, $p = .040$; $\beta = 0.016$, $p = .025$, respectively). In conclusion, the dietary salt intake in CKD patients, especially in male, overweight and obese subjects, remains high in Jiangsu province. It is vital to decline salt intake to control blood pressure in Jiangsu patients with CKD.

Sun, Duan and Zuo are co-first authors of this study.

Xing and Zhang are senior authors with the equal contribution in this study.

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1 | INTRODUCTION

Chronic kidney disease (CKD) has become a major public issue related to increased risk of renal failure, cardiovascular disease, and all-cause mortality.^{1,2} Globally, in 2017, 1.2 million people died from CKD. The global prevalence of CKD was 9.1%,³ and the number of CKD surpassed that of diabetes, osteoarthritis, chronic obstructive pulmonary disease, asthma, or depressive disorders,⁴ posing huge health and economic burden. Lifestyle, in general, for instance, dietary nutrient take, is a modifiable risk factor for CKD progression.⁵ Thus, we focused on educating patients on their dietary habits, which are convertible factors in the treatment of CKD.

In the general population, approximately 90% of sodium is excreted by the kidney.⁶ In spite of patients with a decreased glomerular filtration rate (GFR), 90% of dietary salt intake remain secreted into urine.⁷ Single or preferably even multiple 24-h urinary sodium excretion is regarded as a golden standard for assessing habitual salt intake.⁸ Spot urine samples tend to overestimate or underestimate sodium intake, are susceptible to systematic error, and are not recommended by The International Consortium for Quality Research on Dietary Sodium.⁹ Numerous studies documented a high-salt intake, and urinary sodium excretion was associated with elevated levels of blood pressure, uncontrollable hypertension, frequent CKD progression, and high prevalence of cardiovascular disease and mortality in CKD.^{10–13} The Kidney Disease Improving Global Outcomes guideline, therefore, recommends a salt intake of <5 g/d and sodium <2 g/d in patients with CKD.¹⁴ A survey that investigated 6072 people among 20 provinces in China from 2009 to 2012 demonstrated that the population-weighted, mean weighed salt intake of a standard person was 9.1 g/d,¹⁵ surpassing the recommended daily maximum intake of salt.¹⁶ However, there is no research on sodium intake in patients with CKD in Jiangsu province, China.

In this paper, we assessed the current status of salt intake in CKD patients from 2017 to 2021 in Jiangsu province, China. Meanwhile, the link between urinary sodium excretion and clinical features was evaluated.

2 | MATERIALS AND METHODS

2.1 | Subjects

Eight hundred patients with CKD stages 1–4, aged over 18 years old, from May 2017 to January 2021 were recruited, and they received treatment in the Department of Nephrology, The First Affiliated Hospital of Nanjing Medical University. The exclusion criteria were as follows: (1) acute kidney injury; (2) estimated glomerular filtration rate (eGFR) < 15 ml/min/1.73 m² or undergoing renal replacement therapy, including hemodialysis, peritoneal dialysis, and renal transplantation; (3) known cirrhosis; (4) acute heart failure and advanced heart failure (the New York Heart Association (NYHA) functional classification III or IV); (5) severe edema; (6) comorbid malignancies or pregnant; and (7) history of medications affecting urinary sodium

excretion, such as diuretics, glucocorticoid, and immunosuppressive agent. Written informed consent was obtained from all patients. This study was approved by the Ethics Committee of the First Affiliated Hospital of Nanjing Medical University (approval number: No.2018-SR-250).

2.2 | Data collection

Patient's demographic data, including age, gender, comorbid disease, systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index (BMI), and current medications, were recorded. Also, patient's intravenous blood was collected under fasting condition for laboratory test, containing hemoglobin, serum creatinine, serum albumin, fasting blood glucose (FBG), serum sodium, serum potassium, triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C). Meanwhile, all enrolled patients were asked to collect 24-h urine specimen for evaluating urinary sodium, potassium, protein, and creatinine excretion. The 24-h urine collection was initiated after the first morning urine was discarded in the patient's toilet. Thereafter, the entire volume of urine was collected in a disposable 3-L container. To avoid the possibility of inadequate urine collection, we trained all enrolled patients to properly collect their urine samples and reinforced that 24-h urine collection must be initiated at a specific time and then completed at the same time the next day. eGFR was calculated by CKD-EPI²⁰⁰⁹ equation.¹⁷ Hypertension was defined as SBP \geq 140 mmHg and/or DBP \geq 90 mmHg, or history of taking antihypertensive medications. Normal weight was defined as BMI \geq 18.5 to <24 kg/m², overweight was defined as BMI \geq 24 to <28 kg/m², and obese was defined as BMI \geq 28 kg/m². On the basis of the results of laboratory examinations, the dietary salt intake was calculated using the following equation: salt intake = urinary sodium concentration (mmol/L) \times 24-h urine volume (L) \times 0.05842 (g/mmol). Low-salt intake was defined as salt intake <6 g/d (equivalent to urinary sodium excretion <100 mmol/d), medium-salt intake was defined as salt intake \geq 6 to <12 g/d (equivalent to urinary sodium excretion \geq 100 to <200 mmol/d), and high-salt intake was defined as salt intake \geq 12 g/d (equivalent to urinary sodium excretion \geq 200 mmol/d).¹⁸

2.3 | Statistical methods

Continuous variables were presented as either the mean \pm standard deviation (SD) or the median and interquartile range (IQR). Categorical variables were presented as counts (*n*) and percentages (%). Patients were divided into three groups according to urinary sodium excretion (<100, 100–200, and \geq 200 mmol/d), age (<45, 45–65, and \geq 65 years old), and BMI (<24, 24–28, and \geq 28 kg/m²), respectively. The difference in urinary sodium excretion was compared among three groups using Kruskal–Wallis test. Then, the difference in clinical parameters was compared between male and female

TABLE 1 Clinical features of patients with CKD according to gender

	All (n = 800)	Male (n = 423)	Female (n = 377)	t/ χ^2 /Z	p-Value
Clinical parameter					
Age (years)	47.45 ± 15.25	47.39 ± 16.18	47.51 ± 14.15	-0.105	.917
BMI (kg/m ²)	24.80 ± 3.75	25.68 ± 3.63	23.82 ± 3.64	7.243	<.001
SBP (mmHg)	132.50 ± 19.32	134.99 ± 19.42	129.71 ± 18.83	3.898	<.001
DBP (mmHg)	81.94 ± 13.46	84.35 ± 13.47	79.24 ± 12.95	5.459	<.001
MAP (mmHg)	98.79 ± 14.03	101.23 ± 13.93	96.06 ± 13.64	5.292	<.001
Comorbid disease					
Hypertension (%)	407 (50.9)	254 (60.0)	153 (40.6)	30.216	<.001
Diabetes (%)	156 (19.5)	101 (23.9)	55 (14.6)	10.955	.001
Cardiovascular diseases (%)	95 (11.9)	61 (14.4)	34 (9.0)	5.559	.018
CKD stage (1/2/3a/3b/4)	400/177/79/85/59	175/121/42/51/34	225/56/37/34/25	32.672	<.001
Laboratory parameter					
eGFR (ml/min/1.73 m ²)	89.87 (55.50, 108.65)	82.43 (53.21, 104.71)	97.12 (59.28, 111.99)	4.374	<.001
Scr (μmol/L)	81.35 (62.23, 114.73)	95.70 (76.50, 131.70)	62.90 (52.60, 94.25)	-12.632	<.001
Serum albumin (g/L)	37.90 (33.20, 41.10)	38.40 (33.20, 41.90)	37.40 (33.05, 40.10)	-2.437	.015
FBG (mmol/L)	4.74 (4.31, 5.33)	4.77 (4.34, 5.47)	4.67 (4.30, 5.21)	-1.825	.068
TG (mmol/L)	1.37 (1.00, 1.98)	1.51 (1.07, 2.14)	1.26 (0.89, 1.86)	-4.225	<.001
TC (mmol/L)	4.73 (4.01, 5.73)	4.63 (3.88, 5.55)	4.92 (4.16, 5.84)	3.037	.002
LDL-C (mmol/L)	3.02 (2.46, 3.64)	3.00 (2.42, 3.58)	3.11 (2.49, 3.70)	1.583	.113
HDL-C (mmol/L)	1.07 (0.92, 1.31)	0.98 (0.85, 1.14)	1.22 (1.04, 1.46)	11.295	<.001
Hemoglobin (g/L)	130 (115, 144)	140 (125, 152)	122 (110, 131)	-12.063	<.001
Serum sodium (mmol/L)	140.0 (138.6, 141.9)	140.0 (138.7, 141.5)	140.1 (138.5, 142.1)	0.807	.420
Serum potassium (mmol/L)	3.85 (3.60, 4.11)	3.85 (3.60, 4.12)	3.85 (3.60, 4.10)	-0.321	.748
Urinary protein (g/d)	0.85 (0.29, 2.34)	0.93 (0.34, 2.92)	0.77 (0.27, 1.98)	-3.030	.002
Urinary sodium (mmol/d)	127.20 (91.03, 172.06)	140.40 (102.70, 192.80)	115.96 (80.25, 151.45)	-6.791	<.001
Urinary potassium (mmol/d)	30.80 (23.23, 39.25)	32.40 (24.50, 41.60)	28.30 (22.20, 36.50)	-3.819	<.001
Urinary creatinine (g/d)	1.27 (0.99, 1.66)	1.60 (1.26, 1.94)	1.06 (0.87, 1.25)	-16.653	<.001
Medications					
ACEI/ARB (%)	268 (33.5)	160 (37.8)	108 (28.6)	7.537	.006
CCB (%)	243 (30.4)	149 (35.2)	94 (24.9)	9.982	.002
β-Blocker (%)	81 (10.1)	56 (13.2)	25 (6.6)	9.564	.002

Note: Data were presented as the mean ± standard, the median with interquartile range or counts and percentages. A two-tailed $p < .05$ was considered statistically significant.

The use of bold in the table means that P value was less than .05.

Abbreviations: ACEI/ARB, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; BMI, body mass index; CCB, calcium channel blockers; CKD, chronic kidney disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FBG, fasting blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MAP, mean arterial pressure; SBP, systolic blood pressure; Scr, serum creatinine; TC, total cholesterol; TG, triglyceride.

groups using Student's t -test, Mann-Whitney U test, or chi-square test as appropriate. Spearman correlations were calculated in order to characterize the associations between urinary sodium excretion and clinical parameters. Multivariate linear regression was used to determine the relationship of SBP, DBP, and mean arterial pressure (MAP) with urinary sodium excretion, and multiple covariables were adjusted for age, gender, BMI, eGFR, urinary protein excretion, and history of taking antihypertensive drug. A two-tailed p -value $< .05$ was considered statistically significant. All data were done in IBM SPSS v.20.0 and GraphPad Prism v.8.0.1 software.

3 | RESULTS

3.1 | The distribution of urinary sodium excretion in CKD patients

As shown in Table 1, a total of 800 patients with CKD were recruited. Their mean age was 47.45 ± 15.25 years old, including 423 men and 377 women. Hypertension was present in 50.9% of the patients; 19.5% had diabetes, and 11.9% had cardiovascular disease. The median eGFR was 89.87 ml/min/1.73 m² (IQR 55.50–108.65),

urinary protein was 0.85 g/d (IQR 0.29–2.34), and urinary sodium excretion was 127.20 mmol/d (IQR 91.03–172.06) (equivalent to salt intake 7.4 g/d). Among them, 240 (30.0%) cases had dietary salt intake <6 g/d, and only 167 (20.9%) cases had salt intake <5 g/d according to the guideline of KDIGO.¹⁴ Also, urinary sodium excretion did not differ significantly among CKD stages ($p = .748$). Next, enrolled patients were divided into low-salt intake, medium-salt intake and high-salt intake. As shown in Figures 1 and 2A, 240 (30.0%) cases had low-salt intake, their median urinary sodium excretion was 73.85 mmol/d (IQR 59.58–86.75) (equivalent to salt intake 4.3 g/d), 445 (55.6%) cases had medium-salt intake, and their median urinary sodium excretion was 137.64 mmol/d (IQR 118.03–164.02), which was equivalent to salt intake 8.0 g/d. The patients with high-salt intake accounted for 14.4%, of which median urinary sodium excretion was 232.00 mmol/d that was equivalent to salt intake 13.6 g/d. In addition, patients were classified into three groups according to different level of age and BMI respectively. Our study demonstrated that urinary sodium excretion was higher in overweight group and obese group than that in normal weight group (overweight vs. normal weight, salt intake 7.7 g/d vs. 6.9 g/d, $p = .001$; obese vs. normal weight, salt intake 8.1 g/d vs. 6.9 g/d, $p < .001$; Figure 2B). However, there was no significant difference in urinary sodium excretion among three age subgroups ($p = .131$; Figure 2C). Moreover, we found that urinary sodium excretion of men was higher than that of women in age <45 years old, 45–65 years old, and ≥ 65 years old groups, respectively ($p < .001$, $p < .001$, $p = .021$; Figure 2D). Also, SBP and DBP in high-salt intake group were higher than that in low- and medium-salt intake groups ($p = .002$, $p = .024$ in SBP; Figure 2E; $p = .019$, $p = .012$ in DBP; Figure 2F).

3.2 | Comparison of clinical parameters between male and female patients with CKD

As shown in Table 1, 423 (52.9%) cases were male, their median urinary sodium excretion was 140.40 mmol/d (IQR 102.70–192.80),

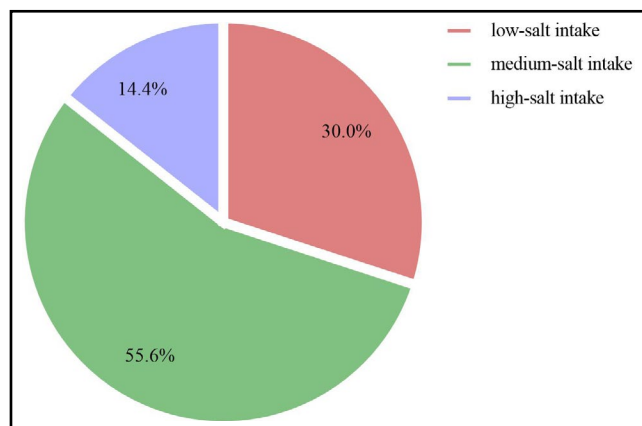


FIGURE 1 Proportion of different level of dietary salt intake in CKD patients. CKD, chronic kidney disease

corresponding to a salt intake of 8.2 g/d, which was higher than that of women ($p < .001$). Besides, compared to female patients with CKD, significantly higher levels of BMI, SBP, DBP, MAP, proportion of hypertension, diabetes and cardiovascular diseases, serum creatinine, serum albumin, TG, hemoglobin, urinary protein excretion, urinary sodium excretion, urinary potassium excretion, and urinary creatinine excretion were observed in male patients with CKD, along with lower levels of eGFR, TC, and HDL-C (all $p < .05$). Moreover, the use of medications including ACEI/ARB, CCB, and β -blocker was significantly more prevalent among male patients (all $p < .05$).

3.3 | Correlations between urinary sodium excretion and clinical parameters in CKD patients

As shown in Table 2, urinary sodium excretion positively correlated with BMI ($r = .198$, $p < .001$), urinary protein excretion ($r = .178$, $p < .001$), TG ($r = .109$, $p = .002$), TC ($r = .078$, $p = .028$), LDL-C ($r = .088$, $p = .013$), hemoglobin ($r = .098$, $p = .005$), urinary potassium excretion ($r = .444$, $p < .001$), and urinary creatinine excretion ($r = .322$, $p < .001$), whereas it was negatively correlated with HDL-C ($r = -.076$, $p = .032$). Nonetheless, the correlations between other laboratory parameters, including age, eGFR, serum creatinine and serum albumin, and urinary sodium excretion, showed no significant differences (all $p > .05$).

3.4 | Association between urinary sodium excretion and blood pressure in CKD patients

As shown in Figure 3, the correlations between urinary sodium excretion and blood pressure were investigated by calculating Spearman correlation coefficients, and urinary sodium excretion showed a significant positive correlation with SBP ($r = .109$, $p = .002$) and DBP ($r = .086$, $p = .015$). Further, we used univariate linear regression and multivariate linear regression to assess the relationships of SBP, DBP, and MAP with urinary sodium excretion (Table 3). In model 1, univariate linear regression showed that higher level of urinary sodium excretion associated with increased level of SBP ($\beta = 0.046$, $p < .001$), DBP ($\beta = 0.029$, $p < .001$), and MAP ($\beta = 0.035$, $p < .001$), respectively. After adjusting for age, gender, BMI, eGFR, urinary protein excretion, and history of taking antihypertensive drug, multivariate linear regression revealed that urinary sodium excretion remained correlated with SBP, DBP, and MAP ($\beta = 0.020$, $p = .049$; $\beta = 0.015$, $p = .040$; $\beta = 0.016$, $p = .025$, respectively, model 2).

4 | DISCUSSION

In this study, the results demonstrated that the median urinary sodium excretion was 127.20 mmol/d in patients with CKD stages 1–4 in Jiangsu province from 2017 to 2021, corresponding to a salt intake

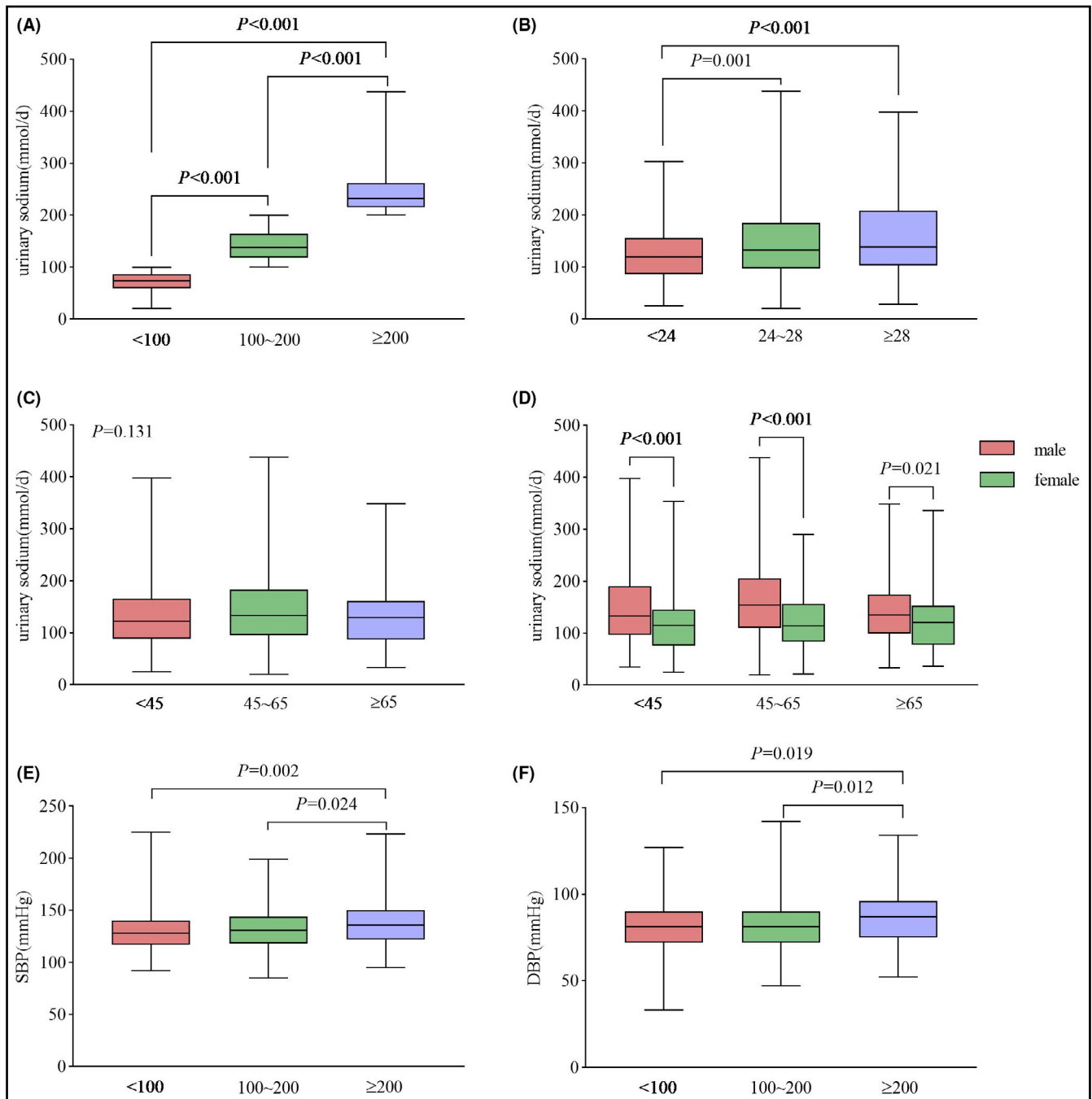


FIGURE 2 24-h urinary sodium excretion and blood pressure stratified by subgroups about age, BMI, and salt intake. (A) Comparison of urinary sodium excretion among low-, medium-, and high-salt intake subgroups; (B) comparison of urinary sodium excretion among normal weight, overweight, and obese subgroups; (C) comparison of urinary sodium excretion among young, middle-aged, and old subgroups; (D) comparison of urinary sodium excretion between men and women subgroups; (E) comparison of SBP among low-, medium-, and high-salt intake subgroups; (F) comparison of DBP among low-, medium-, and high-salt intake subgroups. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure

of 7.4 g/d. Among them, only 20.9% of cases had salt intake <5 g/d.¹⁴ These findings reflected there was still a huge gap in the amount of dietary salt intake between actual intake and recommended intake. To better manage and prevent the complication of CKD, patients still need to reduce the dietary salt intake. Nonetheless, a survey assessing salt intake in China from 2009 to 2012 indicated that the

population-weighted, mean weighed salt intake of a standard person was 9.8 g/d in Jiangsu province.¹⁵ Compared to the above study, the amount of sodium consumption decreased in Jiangsu province from 2017 to 2021. Interestingly, not merely among all subjects, but also in young, middle-aged, and old subgroups, estimated salt intake in men was higher than that in women. The reason for this

TABLE 2 Correlations between urinary sodium excretion and clinical parameters in CKD patients

Parameters	Urinary sodium (mmol/d)	
	<i>r</i>	<i>p</i>
Age(years)	.068	.055
BMI (kg/m ²)	.198	<.001
Urinary protein (g/d)	.178	<.001
eGFR (ml/min/1.73 m ²)	.012	.734
Scr (μmol/L)	.034	.341
Serum albumin (g/L)	−.061	.084
TG (mmol/L)	.109	.002
TC (mmol/L)	.078	.028
LDL-C (mmol/L)	.088	.013
HDL-C (mmol/L)	−0.076	.032
Hemoglobin (g/L)	.098	.005
Urinary potassium (mmol/d)	.444	<.001
Urinary creatinine (g/d)	.322	<.001

Note: A two-tailed $p < .05$ was considered statistically significant.

Abbreviations: BMI, body mass index; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Scr, serum creatinine; TC, total cholesterol; TG, triglyceride.

discrepancy might be the differences in food pattern between men and women, and the men seemed to have a higher whole food intake.¹⁹ Therefore, male patients with CKD should pay more attention to control dietary salt intake.

Although there was no difference in urinary sodium excretion regarding the age status, the urinary sodium excretion was higher in overweight and obese subgroups than that in normal weight subgroup. Moreover, urinary sodium excretion positively correlated with BMI, TG, TC, and LDL-C and negatively correlated with HDL-C. Previous observational and trial studies have similar discovery. A randomized crossover trial of dietary sodium restriction in patients with CKD stages 3–4 revealed that patient's body weight significantly declined after limiting salt intake.²⁰ Fan and colleagues,²¹ Kang and colleagues,²² and He and colleagues¹¹ observed that CKD patients with elevated urinary sodium excretion expressed increased BMI, LDL-C, and decreased HDL-C. The above studies indicate intimate connection between undue sodium intake and obesity. However, the underlying mechanisms have not been elucidated. Song and colleagues²³ came up with a hypothesis that excessive salt intake generated adipocyte hypertrophy and fat accumulation. Interestingly, our study found that urinary sodium excretion was similar among CKD stages 1–4, which was different from previous study. Kang and colleagues²⁴ found that 24-h urinary sodium excretion was significantly lower in patients with more advanced CKD, especially in stage 5. In our study, we excluded the patients with CKD stage 5, which aimed to avoid the inclusion of patients with impaired sodium excretion. In our experience, CKD5 patients usually have severe tubulointerstitial injury, while these

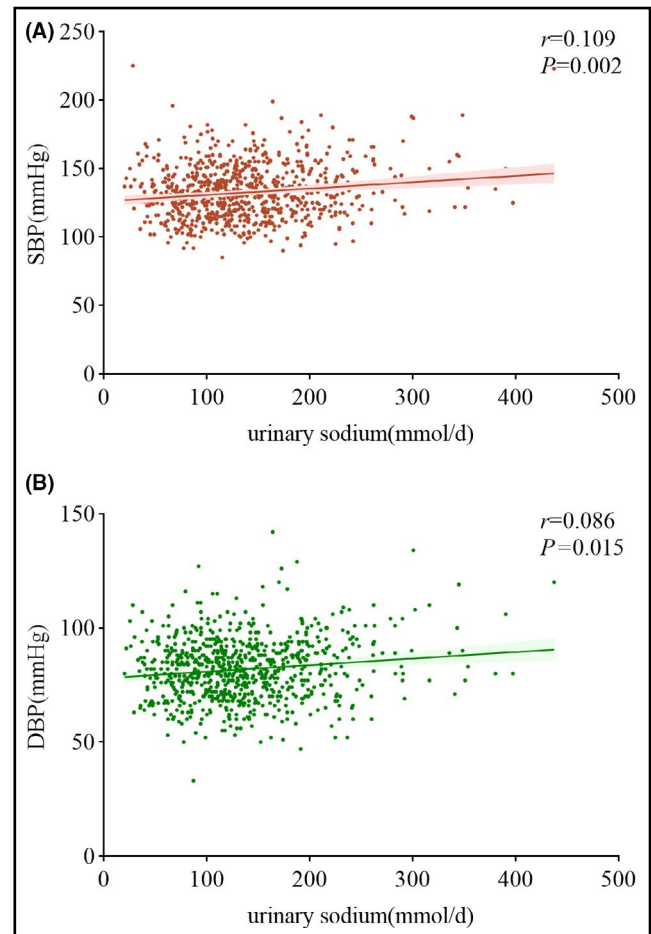


FIGURE 3 Association of urinary sodium excretion with blood pressure in CKD patients. (A) Correlation between urinary sodium excretion and SBP; (B) correlation between urinary sodium excretion and DBP. SBP, systolic blood pressure; DBP, diastolic blood pressure; CKD, chronic kidney disease

lesions may affect the excretion of sodium. Hence, the difference in CKD stage among enrolled subjects between our study and other research might account for the discrepancy in results.

Sodium overload plays a crucial role in salt-sensitive hypertension in CKD patients. Rossi and colleagues²⁵ found that distal nephron enhanced sodium reabsorption in CKD. In addition, the phenomenon that high dietary salt intake, glomerulotubular disturbance, immoderate secretion of aldosterone, activation of renal RAS system, and sodium channel at renal tubular epithelial cell activated by inflammation and urinary protein with plasmin properties contributes to sodium retention. Further, a series of variations, including extracellular fluid expansion, arteriosclerosis, and peripheral vasoconstriction, result in salt-sensitive hypertension in CKD.^{26,27} Mills and colleagues¹⁰ perceived that increased urinary sodium excretion was associated with elevated systolic and diastolic blood pressure in CKD patients. Another clinical trial observed that dietary salt restriction overtly decreased 24-h SBP and DBP in CKD stages 3–4 patients.²⁰ The alike consequence was discovered in our study. After adjusting for vital confounding factors, multivariate linear regression demonstrated

	Model 1			Model 2		
	β	95% CI	<i>p</i>	β	95% CI	<i>p</i>
SBP	0.046	0.025–0.067	<.001	0.020	0.000–0.039	.049
DBP	0.029	0.014–0.043	<.001	0.015	0.001–0.029	.040
MAP	0.035	0.019–0.050	<.001	0.016	0.002–0.031	.025

Note: Model 1: unadjusted.

Model 2: adjusted for age, gender, BMI, eGFR, urinary protein excretion, history of antihypertensive drug.

A two-tailed $p < .05$ was considered statistically significant.

Abbreviations: BMI, body mass index; CKD, chronic kidney disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate.; MAP, mean arterial pressure; SBP, systolic blood pressure.

that a high level of urinary sodium excretion related to increased SBP, DBP, and MAP in CKD patients. Other than undue salt intake, persistent refractory blood pressure give rise to the increased prevalence of cardiovascular disease and rapid progressing to end-stage renal disease.²⁸ Therefore, it is vital for CKD patients, especially complicated with hypertension, to limit dietary sodium consumption.

In addition, our study observed that urinary sodium excretion positively correlated with urinary protein excretion in CKD patients, which was consistent with previous studies.^{10,11,22,29} Park and colleagues³⁰ found that restricting salt intake improved renal inflammation and fibrosis via relieving the reactive oxygen species-mediated NF- κ B activation in a rat model of adriamycin-induced nephrotic syndrome. In spontaneously hypertensive rats, Matavelli and colleagues³¹ discovered that restricting dietary salt loading obviously deteriorated renal function, renal hemodynamics, and glomerular dynamics independent of a minimal further increase in arterial pressure. The above findings supported the concept of a strong independent causal relationship between salt excess and renal injury.

Several limitations of our study should be considered. First, it was an observational study that cannot make a causal inference. Moreover, while we controlled for many factors in the analyses, residual confounding may still be a potential limitation. Second, all enrolled patients were lack of 24-h blood pressure. Hence, single blood pressure was used to evaluate the link between urinary sodium excretion and blood pressure, which might partly influence the results. Finally, because we excluded the patients with CKD stage 5, the dietary salt intake in that stage cannot be assessed. Further, the survey on dietary salt intake in all stages of CKD, probably throughout the country, needs to be put into action.

5 | CONCLUSIONS

In summary, we found high amounts of salt intake in CKD patients in Jiangsu province, especially in male and abnormal weight patients. Only 20.9% of all subjects had salt intake <5 g/d. Moreover, our study demonstrated that urinary sodium excretion was associated with kidney injury and blood pressure, reinforcing the point limiting sodium consumption is crucial for CKD patients.

TABLE 3 Univariate linear regression and multivariate linear regression for the association between urinary sodium excretion and blood pressure in CKD patients

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

AUTHOR CONTRIBUTIONS

LS designed the study, collected and analyzed the statistics, and wrote the manuscript. SD and CZ made contribution to the writing and performed statistical analysis. ZS, GN, CZ, MZ, BS, YY, NW, and HM reviewed the manuscript. CX conceived, coordinated the study, and were responsible for the final substance. BZ is the guarantor and supervision of the work who had full and final responsibility for methodology, integrity and curation of data analysis, and editing the manuscript. All authors have revised the manuscript and approved the final version of the paper to be published.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this article.

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