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■ Original Article

Evaluation of the relationship of urinary sodium excretion with metabolic syndrome, hypertension, and graft function in renal transplant patients

Renal transplant hastalarında idrar sodyum atılımının metabolik sendrom, hipertansiyon ve greft fonksiyonu ile ilişkisinin değerlendirilmesi

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ABSTRACT

Aim: To evaluate the relationship between high sodium intake determined by spot urinary sodium excretion with metabolic syndrome, hypertension, and graft function in renal transplant (RT) recipients.

Materials and Methods: 152 RT (35.5% were female) recipients were enrolled. The demographic characteristics, office blood pressure (BP) values, height, weight, body mass index, waist and hip measurements, immunosuppressive drugs, other medications, and biochemical parameters of the patients were recorded. Spot urinary sodium and protein excretions were measured in the RT recipients' first-morning urine. The patients were grouped as low sodium excretion (≤ 57 mmol/L) and high sodium excretion (≥ 58 mmol/L) based on the median value.

Results: When the groups were compared according to spot urinary sodium excretion, no difference was found in terms of creatinine values, systolic BP and diastolic BP ($p=0.21$, $p=0.18$ and $p=0.80$, respectively). In the low sodium group, creatinine values were significantly lower ($p<0.001$), and eGFR was high in female patients ($p=0.03$). The mean protein in spot urine was lower in women ($p=0.03$). In the high sodium group, BUN and creatinine levels were significantly higher in male patients than in female patients ($p=0.04$ and $p=0.02$, respectively). The ejection fraction was significantly lower in male patients than in female patients ($p=0.008$). When the spot urinary sodium excretion of patients with and without metabolic syndrome was compared, no difference was found between the two groups ($p=0.99$).

Conclusion: Spot urinary sodium excretion can be an inexpensive and relatively effective screening method that can be used to evaluate sodium intake in RT patients. It can be considered a more valuable follow-up method, especially in RT recipients with male gender, kidney dysfunction, and high BP.

Keywords: Graft function, hypertension, metabolic syndrome, renal transplant, spot urine sodium

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Öz

Amaç: Renal transplant (RT) hastalarında idrar sodyum atılımı ile metabolik sendrom, hipertansiyon, greft fonksiyonu arasındaki ilişkinin değerlendirilmesidir.

Gereç ve Yöntemler: Nakil polikliniğinde düzenli takip edilen 152 RT (%35.5 kadın cinsiyet) alıcısı çalışmaya alındı. Hastaların demografik özellikleri, ofis kan basıncı değerleri, boy, kilo, vücut kitle indeksi, bel ve kalça ölçümleri, immünyüpresif ilaçlar, diğer ilaçlar ve biyokimyasal parametreleri kaydedildi. Tüm RT alıcılarında sabah ilk idrarlarında spot sodyum ve protein atımları ölçüldü. Hastalar spot idrar sodyum medyan değerine göre düşük sodyum atılımı (≤ 57 mmol/L) ve yüksek sodyum atılımı (≥ 58 mmol/L) olarak gruplandırıldı.

Bulgular: Gruplar spot idrar sodyum atılımına göre karşılaştırıldığında kreatinin değerleri, sistolik KB ve diyastolik KB açısından fark bulunmadı (sırasıyla $p=0,21$, $p=0,18$ ve $p=0,80$). Düşük sodyum grubunda; kadınlarda kreatinin değerleri anlamlı derecede düşük ($p<0,001$) ve eGFR yüksek saptandı ($p=0,03$). Yüksek sodyum grubunda; BUN ve kreatinin düzeyleri cinsiyete göre karşılaştırıldığında erkeklerde anlamlı derecede yüksek bulundu (sırasıyla $p=0,04$ ve $p=0,02$). EF değeri erkeklerde kadınlardan anlamlı derecede düşüktü ($p=0,008$). Metabolik sendromu olan ve olmayan hastaların spot idrar sodyum atımları karşılaştırıldığında her iki grupta spot idrar sodyum atılım değerleri arasında fark saptanmadı ($p=0,99$).

Sonuç: Spot idrar sodyum atılımı, RT hastalarında sodyum alımını değerlendirmek için kullanılabilir ve nispeten etkili bir tarama yöntemi olarak görülebilir. Özellikle erkek cinsiyet, böbrek fonksiyon bozukluğu ve yüksek tansiyonu olan RT alıcılarında daha değerli bir takip yöntemi olarak kabul edilebilir.

Anahtar kelimeler: Böbrek nakli, greft fonksiyonu, hipertansiyon, metabolik sendrom, spot idrar sodyum.

Introduction

Metabolic syndrome (MS) is a fatal endocrinopathy starting with insulin resistance, followed by a series of systemic disorders such as abdominal obesity, glucose intolerance or diabetes mellitus (DM), dyslipidemia, hypertension (HT), and coronary artery disease (CAD) [1]. MS is associated with a risk of developing cardiovascular disease [2].

Hypertension, a component of metabolic syndrome, is a common cardiovascular disease and has been indicated as one of the leading causes of death [3]. Numerous observational studies have shown that cardiovascular morbidity and mortality are associated with systolic and diastolic blood pressure (BP) [4]. According to the argument starting with Guyton, the main problem of hypertensive patients is the failure of the kidney to eliminate the excess sodium load taken with a high-salt diet [5]. Beyond raising BP, a high-salt diet is an independent risk factor for target organ damage, leading to fatal cardiovascular events, including stroke, cardiac hypertrophy, diastolic dysfunction, and renal failure [6]. Retention of excess sodium in the kidneys triggers HT. This retention may occur due to congenital or acquired deficiency in nephron number or function [7]. Although the number of cardiovascular disease-related deaths is decreasing thanks to new treatments [8], it remains the leading cause of death in renal transplant (RT) patients [9].

In the non-transplant population, high sodium consumption

is generally associated with insulin resistance, metabolic syndrome, and hypertension [10, 11]. The number of studies on the relationship between increased sodium intake and excretion and metabolic syndrome in RT patients is limited. Therefore, in this study, we aimed to investigate whether there is a relationship between daily urinary sodium excretion, an indirect indicator of daily salt intake, metabolic syndrome, hypertension, and graft function in RT patients.

Material and Methods

The study was prospectively carried out on 152 patients over 18 years, who were admitted to the nephrology outpatient clinic between January 2016 and April 2016, underwent renal transplantation, had stable kidney functions, and gave informed consent forms. The patient's gender, age, body weight, height, body mass index, transverse waist circumference, medication use, BUN (blood urea nitrogen), creatinine, sodium, spot urinary sodium, spot urinary protein, estimated glomerular filtration rate (eGFR), fasting blood glucose, insulin, low-density lipoprotein (LDL)-cholesterol, high-density lipoprotein (HDL)-cholesterol, triglyceride, and albumin values were recorded. During the examinations of the patients, the BP measurements were made by a sphygmomanometer in the sitting position after at least 10 minutes of rest. They were recorded as systolic BP (mmHg) and diastolic BP (mmHg). As use of antihypertensive drug or an above BP of 130/85 mm Hg measured during the examination was considered the diagnostic criteria for hypertension.

Metabolic syndrome was diagnosed according to the Adult Treatment Panel III (ATP III) criteria updated in a report of the American Heart Association (AHA)/National Heart, Lung and Blood Institute (NHLBI) in 2005 [1].

At the same time, left ventricular hypertrophy and ejection fractions were recorded by evaluating the reports of transthoracic echocardiography performed in the last year.

The local ethics committee approved the study.

Statistical analysis

The Statistical Package for Social Sciences version 15.0 software was used to evaluate the data. Descriptive statistical data are expressed as frequency (percentage), number and mean±standard deviation, or median (min-max). Kolmogorov-Smirnov test evaluated the distribution properties of the numeric variables. Independent-samples t-test was used for intergroup comparisons of numeric variables with normal distribution, and Mann-Whitney's U test was used for variables without normal distribution. Categorical data were evaluated using the chi-square test. A p-value of <0.05 was considered statistically significant.

Results

The mean age of the patients in the study was 40.7±12.0 years. Of the patients, 64.5% (98 individuals) were male. Left ventricular hypertrophy (LVHT) was detected in 59.2% (71 individuals) of the 120 patients registered in the system and had echocardiography performed in the last year. The mean time after RT was 7.9±6.4 years. The clinical and laboratory characteristics of the patients are shown in Table 1.

The patients were grouped as low sodium excretion (57 mmol/L and below) and high sodium excretion (58 mmol/L and above) based on the median value.

Low Sodium Group: The mean creatinine level was 1.7±0.6 mg/dL in male patients, while it was 1.1±0.4 mg/dL in female patients and was significantly lower in female patients (p<0.001). The mean eGFR value was 53.5±18.5 ml/min/1.73 m² in male patients and 65.2±23.9 ml/min/1.73 m² in female patients and was significantly higher in female patients (p=0.03). The mean spot urinary protein value was 69.4±97.8 mg/dL in male patients, while it was 33.4±49.5 mg/dL in female patients, and this difference was statistically significant (p=0.03) (Table 2).

High Sodium Group: The mean BUN value was 25.1±11.9 mg/dL in male patients and 19.7±8.9 mg/dL in female patients (p=0.04). The mean creatinine was measured as 1.5±0.6 mg/dL in male patients and as 1.2±0.5 mg/dL in female patients and was found to be significantly higher in male patients (p=0.02). The mean insulin level was 17.1±6.5 µU/mL in male patients, while it was 13.2±5.2 µU/mL in female patients, and this difference

was significant (p=0.009). The mean EF measurement was significantly lower in male patients (57.5±5.4) than in female patients (61.0±3.3) (p=0.008) (Table 2).

When the groups were evaluated by sodium levels regardless of gender, there was no significant difference between the measurements (p>0.05) (Table 2).

53.1% (52 individuals) of male patients were in the low sodium group, this rate was 46.3% (25 individuals) in female patients. There was no significant difference between the groups regarding sodium excretion (p=0.42). Of the patients with and without diabetes mellitus, 51.9% (4 individuals) and 48.8% (61 individuals) were in the high sodium group, respectively, and this difference was not significant (p=0.77). Of the patients using and not using a statin, 63.6% (28 individuals) and 45.4% (49 individuals) were in the low sodium group, respectively, and this difference was statistically significant (p=0.04) (Table 3).

Table 1. Clinical and laboratory characteristics of the patients

Characteristics	Patient Group (n=152)
Age, years (Mean ± SD)	40.7±12.0
Female/Male	54/98
Diabetes Mellitus	27 (%17.8)
Use of Statin	44 (%28.9)
Metabolic Syndrome	81 (%53.3)
Tacrolimus	63 (%41.4)
Sirolimus	45 (%29.6)
Cyclosporine	39 (%25.7)
ECHO LVHT	71 (%59.2)
	median (min-max)
Post-Transplant Duration, years	7 (1-36)
BUN, mg/dL	19.5 (7.2-67.5)
Creatinine, mg/dL	1.39 (0.59-4.76)
Albumin, g/dL	4.21 (2.91-4.76)
eGFR, ml/min/1.73 m ²	58.04 (14.81-127.25)
Spot Urine Sodium, mmol/L	57 (7-195)
Spot Urine Protein, mg/dL	21.9 (0.9-544.8)
LDL-cholesterol, mg/dL	124 (22-263)
HDL-cholesterol, mg/dL	47 (17-90)
Triglyceride, mg/dL	162.5 (35-561)
Fasting blood glucose, mg/dL	93 (55-269)
Insulin, µU/mL	16 (4-32)
Systolic Blood Pressure, mmHg	130 (90-170)
Diastolic Blood Pressure, mmHg	80 (60-100)
EF, %	60 (30-71)
BMI, kg/m ²	25.53 (15.43-92)
Waist circumference, cm	74 (56-120)

BUN: Blood urea nitrogen; BMI: Body mass index; EF: Ejection fraction; ECHO LVHT: Echocardiography left ventricular hypertrophy; eGFR: Glomerular filtration rate

Table 2. Comparison of the characteristics of the patients according to sodium groups and gender

	Spot Urine Sodium								
	Low (≤ 57 mmol/L) (Mean \pm SD)				High (≥ 58 mmol/L) (Mean \pm SD)				
	Male (n=52)	Female (n=25)	Total (n=77)	p	Male (n=46)	Female (n=29)	Total (n=75)	p	p*
Age, years	41.7 \pm 11.9	39.1 \pm 10.8	40.8 \pm 1.6	0.37	42.2 \pm 12.3	38.0 \pm 12.6	40.5 \pm 12.5	0.15	0.88
Post-Transplant Duration, years	8.8 \pm 6.4	7.9 \pm 7.6	8.5 \pm 6.8	0.57	6.9 \pm 6.3	7.8 \pm 5.3	7.2 \pm 5.9	0.54	0.23
BUN, mg/dL	23.7 \pm 12.1	18.4 \pm 7.7	22.1 \pm 11.1	0.05	25.1 \pm 11.9	19.7 \pm 8.9	22.9 \pm 11.1	0.04	0.60
Creatinine, mg/dL	1.7 \pm 0.6	1.1 \pm 0.4	1.5 \pm 0.6	<0.001	1.5 \pm 0.6	1.2 \pm 0.5	1.4 \pm 0.6	0.02	0.21
Albumin, g/dL	4.2 \pm 0.3	4.1 \pm 0.3	4.2 \pm 0.3	0.69	4.1 \pm 0.3	4.1 \pm 0.3	4.1 \pm 0.3	0.35	0.20
eGFR, ml/min/1.73 m ²	53.5 \pm 18.5	65.2 \pm 23.9	57.3 \pm 21.1	0.03	59.7 \pm 20.3	65.5 \pm 27.1	61.9 \pm 23.1	0.33	0.20
Spot Urine Protein, mg/dL	69.4 \pm 97.8	33.4 \pm 49.5	57.7 \pm 86.5	0.03	63.1 \pm 80.3	41.0 \pm 90.1	54.5 \pm 84.4	0.27	0.97
LDL-cholesterol, mg/dL	130.7 \pm 46.6	122.8 \pm 42.1	128.1 \pm 45.1	0.47	121.6 \pm 35.1	137.8 \pm 42.2	127.9 \pm 38.5	0.07	0.97
HDL-cholesterol, mg/dL	43.8 \pm 11.4	51.5 \pm 16.6	46.3 \pm 13.7	0.04	42.4 \pm 11.3	50.6 \pm 10.8	45.6 \pm 11.8	0.003	0.72
Triglyceride, mg/dL	196.6 \pm 103.6	144.7 \pm 3.5	179.7 \pm 95.2	0.02	194.3 \pm 97.7	149.5 \pm 60.1	176.9 \pm 87.4	0.01	0.85
Fasting blood glucose, mg/dL	102.4 \pm 19.5	100.2 \pm 35.4	101.7 \pm 25.5	0.72	103.5 \pm 38.5	101.5 \pm 40.9	102.7 \pm 39.2	0.82	0.84
Insulin, μ U/mL	16.9 \pm 6.4	14.7 \pm 6.3	16.2 \pm 6.4	0.15	17.1 \pm 6.5	13.2 \pm 5.2	15.6 \pm 6.3	0.009	0.55
BMI, kg/m ²	26.9 \pm 9.4	28.1 \pm 14.8	27.3 \pm 11.2	0.69	25.7 \pm 4.1	27.9 \pm 13.3	26.5 \pm 8.8	0.40	0.65
Waist circumference, cm	78.5 \pm 12.2	73.6 \pm 12.5	76.9 \pm 12.4	0.10	78.7 \pm 11.5	75.3 \pm 13.2	77.4 \pm 12.2	0.25	0.80
SBP, mmHg	131.6 \pm 13.9	125.2 \pm 13.2	129.5 \pm 13.9	0.05	129.1 \pm 14.8	122.2 \pm 4.6	126.4 \pm 15.1	0.05	0.18
DBP, mmHg	80.2 \pm 9.5	76.8 \pm 9.0	79.1 \pm 9.4	0.13	80.0 \pm 7.9	76.8 \pm 9.9	78.7 \pm 8.8	0.14	0.80
EF, %	57.8 \pm 7.3	60.1 \pm 3.5	58.6 \pm 6.3	0.11	57.5 \pm 5.4	61.0 \pm 3.3	58.9 \pm 4.9	0.008	0.76

*Low vs High; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; BUN: Blood urea nitrogen; BMI: Body mass index; EF: Ejection fraction; eGFR: Glomerular filtration rate

Table 3. Comparison of sodium levels of patients.

		Spot Urine Sodium		p
		Low	High	
Gender	Male	52 (%53.1)	46 (%46.9)	0.42
	Female	25 (%46.3)	29 (%53.7)	
Diabetes	Presence	13 (%48.1)	4 (%51.9)	0.77
	Absence	64 (%51.2)	61 (%48.8)	
Use of Statin	Presence	28 (%63.6)	16 (%36.4)	0.04
	Absence	49 (%45.4)	59 (%54.6)	
MS	Presence	41 (%50.6)	40 (%49.4)	0.99
	Absence	36 (%50.7)	35 (%49.3)	
Tacrolimus	Presence	34 (%54.0)	29 (%46.0)	0.49
	Absence	43 (%48.3)	46 (%51.7)	
Sirolimus	Presence	25 (%55.6)	20 (%44.4)	0.43
	Absence	52 (%48.6)	55 (%51.4)	
Cyclosporine	Presence	16 (%41.0)	23 (%59.0)	0.16
	Absence	61 (%54.0)	52 (%46.0)	
ECHO LVHT	Presence	35 (%49.3)	36 (%50.7)	0.85
	Absence	25 (%51.0)	24 (%49.0)	

ECHO LVHT: Echocardiography left ventricular hypertrophy; MS: Metabolic syndrome

The systolic BP values of the patients using and not using tacrolimus were compared. While the median systolic BP value of the patients using tacrolimus was 120 mmHg, the median value of those who did not was found to be 130 mmHg, and this difference was statistically significant ($p=0.036$). There was no statistically significant difference when using sirolimus and cyclosporine compared to the systolic BP values. When the diastolic BP values of the patients included in the study were compared by the immunosuppressives they used, there was no statistically significant difference. No difference in sodium excretion between the group with high BP and the other group among the patients using tacrolimus suggests that the elevation of BP due to tacrolimus is independent of sodium.

When the patients with controlled and uncontrolled BP were compared for spot urinary sodium excretion, there was a statistically significant difference between the groups ($p<0.001$). The median value of the spot urinary sodium excretion level was 66 (IQR 52) mmol/L in patients with controlled BP and 43 (IQR 32) mmol/L in patients with uncontrolled BP. This difference was statistically significant ($p<0.001$).

Discussion

In this study, we evaluated the relationship between urinary sodium excretion, which is an indirect indicator of daily salt intake, and metabolic syndrome, hypertension, and graft function in RT patients; when the groups were compared according to spot urinary sodium excretion, no difference was found in terms of creatinine values, systolic BP and diastolic BP. In the low sodium group, creatinine values were significantly lower, and eGFR was higher and the mean protein in spot urine was lower in female patients. In the high sodium group, while BUN and creatinine levels were significantly higher and the ejection fraction was lower in male patients than in female patients. When the spot urinary sodium excretion of patients with and without metabolic syndrome was compared, no difference was found between the two groups.

Compared to the general population, the prevalence of MS appears to be higher in renal transplant patients (32%-44.8%) [12, 13]. This study detected MS in 53.3% of renal transplant patients. The spot urinary sodium excretions of the patients with and without metabolic syndrome were compared, and no statistically significant difference could be demonstrated. The absence of a substantial difference in these results may be because spot urinary sodium might have yet to reflect 24-hour urinary sodium fully. The gold standard method for estimating urinary sodium excretion is 24-hour urinary sodium measurement. Although spot urinary sodium yielded similar results with 24-hour urinary excretion in some studies [10, 11], spot urinary sodium may not be an appropriate indicator in RT patients, considering that kidney functions of this group are different from the general population and some immunosuppressive drugs used can differentiate spot urinary sodium excretion.

Similarly, an evaluation performed on people with normal BP (781 patients, BP < 130/85 mmHg) in Brazil showed no difference between 24-hour urinary sodium excretions in the comparison of patients with or without MS. Urinary sodium excretion was lower in those with MS than in those without MS [9]. In a study reported from South Korea, no difference was observed between urinary sodium excretion of patients with and without MS [14]. On the contrary, in a study conducted by Unal et al. [15] on 76 renal transplant patients, MS was detected in 52 patients (68.4%), and daily urinary sodium excretion was significantly higher in MS patients. Likewise, Hoffman and Cubeddu [11] showed that 24-hour urinary sodium excretion was significantly increased in patients with metabolic syndrome.

Based on previous studies, we can predict that salt intake is also elevated in the high sodium excretion group. Baudrand et al. [10] showed that high salt intake increases the risk of metabolic syndrome by two times. In addition, they showed that high salt intake was associated with HT, dyslipidemia, insulin resistance, and high glucocorticoid production [10]. Donovan et al. [16] showed that high salt intake was more insulin resistant than low salt intake in euglycemic and normotensive individuals. In this study, "insulin levels" were significantly higher in male patients in the high salt intake group. TG levels of male patients with high insulin were also considerably higher. It is thought that this is caused by increased TG synthesis due to the anabolic effect of insulin on the liver. Because TG-rich HDL cholesterol tends to break down more quickly, HDL cholesterol values are low in these male individuals with high TG levels. Again, BUN and creatinine levels are statistically significantly higher in male patients. These results suggest that male patients in the high-risk group for cardiovascular diseases consume a higher amount of salt than female patients, and spot urinary sodium excretion may be an independent risk factor.

Male patients are expected to have higher mean creatinine levels due to higher muscle mass. Accordingly, creatinine-based eGFR estimations differ similarly. In this study, creatinine levels were significantly lower, and eGFR levels were significantly higher in female patients with low sodium excretion. In this case, it can enable us to predict that low salt consumption may contribute to long-term graft function.

In this study, the mean spot urinary protein was higher in male patients than in female patients. High proteinuria levels in male transplant patients were found in the group with low sodium excretion; suggests that the reason for the low sodium excretion may be related to the protein and sodium restricted diet recommendation in patients with proteinuria. We know that high dietary sodium intake increases albuminuria [17]. Spot urinary sodium of male patients due to both high levels of sodium intake and relatively poor functioning grafts point to male RT patients as a group at higher risk for long-term cardiovascular diseases.

The rate of hypertensive patients among RT recipients appears to be 60-80% or more, and the use of antihypertensive drugs increases over time after kidney transplantation [18]. Hypertension is a major cardiovascular complication and increases graft loss in RT recipients with a systolic BP \geq 140 mmHg [19]. This shows the great significance of BP control in RT patients. It may be difficult for patients with a single functional kidney to achieve BP control due to restriction therapy, including immunosuppressive and corticosteroid drugs.

In this study, the relationship between sodium excretions and systolic and diastolic BP, though not statistically significant, was found to be quite substantial when evaluated by gender in the low and high sodium excretion groups. A study reported that 24-hour urinary sodium excretion within one year after kidney transplantation between 1997 and 2009 was associated with systolic and diastolic BP [20]. However, some authors have reported a lack of relationship between sodium excretion and BP [21, 22]. On the contrary, in their extensive study including 660 RT patients, Van den Berg et al. [23] showed that 24-hour urinary sodium excretion increased with systolic and diastolic BP. The relationship between sodium excretion and BP has always been more significant for systolic than diastolic BP in transplant and non-transplant populations [23, 24]. The data of our study show that BP is higher in patient groups with increased sodium excretion, even though it is not statistically significant. Thus, restriction of sodium intake for BP control should be considered crucial. In a cross-sectional study conducted in Japan with 889 patients, a positive gender-independent correlation was found between spot urinary sodium concentrations and systolic BP. In a cross-sectional study conducted in Japan with 889 patients, a positive gender-independent correlation was found between spot urinary sodium concentrations and systolic BP [25]. In a study from England, 23,104 male and female patients between the ages of 45-79 years were analyzed. The spot urinary sodium/creatinine ratio was positively correlated with systolic and diastolic BPs [26]. Daily urinary sodium excretion correlated with diastolic BP, serum glucose concentration, and creatinine clearance. Additionally, although no significant correlation has been found between urinary sodium excretion and systolic BP, their relationship is statistically borderline significant [15]. These data are in line with the data of our study. Here, although spot urinary sodium concentration alone is not an important risk factor, in the presence of the other risk factors that may accompany it, it may be of considerable importance in terms of hypertension and metabolic syndrome.

This study observed no significant difference between systolic and diastolic BP by sodium excretion. When the patients were grouped based on their BP values as controlled (BP<140/90 mmHg) and uncontrolled (\geq 140/90 mmHg) and compared in terms of spot urinary sodium excretion, a significant difference was found between the groups. The role of sodium in the hypertension mechanism by causing volume load in the body supports this result. Based on these results, we suggest that

it is impossible to control BP without salt restriction in renal transplant patients with unregulated BP. That follow-up of salt restriction can be performed with spot urinary sodium.

Our study compared the systolic and diastolic BP of the patients using and not using tacrolimus. The systolic BP of the patients using tacrolimus was significantly lower than those who did not. No correlation was found in the patients used cyclosporine. In a similar study, 80% of controlled HT patients and 50% of patients with uncontrolled HT used tacrolimus as an immunosuppressive drug [22]. Cyclosporine increases systemic and renal (primary afferent arteriolar effect) vascular resistance. The increased release of vasoconstrictors, especially endothelins, is thought to play a substantial role [27]. Therefore, reviewing the immunosuppressive regimen of patients with high spot urinary sodium and uncontrolled BP may be appropriate.

Our study has some limitations. First, our study included a relatively small number of groups with renal transplantation. The patients' 24-hour sodium consumption could not be recorded since reliable information could not be obtained. We did not re-studied spot urinary sodium values to support the initial results of our patients. The BP of the patients was recorded only as the measurements we performed during the follow-up. Thus, masked hypertension (normal in-office BP but elevated out-of-office BP) and white-coat hypertension (high in-office BP but normal BP at home) could not be distinguished. At the same time, the patient's smoking status was not questioned. Although no medication change was made in the last month in the patients in the study, it would be more accurate to record their antihypertensive and diuretic use.

In conclusion, spot urinary sodium excretion is an inexpensive and relatively effective screening method that can evaluate sodium intake in renal transplant patients. It can be considered a more valuable follow-up method, especially in male RT recipients with kidney dysfunction and high BP.

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The authors declare no conflicts of interest to disclose.

The corresponding author's data supporting this study's findings are available upon reasonable request.

The study was approved by the ethics committee of Baskent University Medical Faculty (Date: 15/02/2016, Approval number: KA16/17).

Authors' contributions to the article

Conception and design of the study; TID, SY, CBS
Generation, collection, assembly, analysis, and/or interpretation of data; TID, SY, CBS
Drafting or revision of the manuscript; TID, SY, CBS
Approval of the final version of the manuscript; TID, SY, CBS

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