Türkiye Çocuk Hastalıkları Dergisi
*Turkish Journal of Pediatric Disease*

# Assessment of Spot Urine Sodium to Potassium Ratio in Obese Hypertensive Children

## Hipertansif Obez Çocuklarda Spot İdrarda Sodyum/Potasyum Oranının Değerlendirilmesi

Emel ISIYEL[1], Ayşegül TAŞ[2], Kezban ÖZTÜRK[3], Elif ÇOMAK[4]

[1]Health Sciences University, Child Health and Diseases Hematology and Oncology Hospital, Pediatric Nephrology Clinic, Ankara, Turkey
[2]Kocaeli Derince Training and Research Hospital, Department of Physiology, Kocaeli, Turkey
[3]Kocaeli Derince Training and Research Hospital, Department of Child Health and Diseases, Kocaeli, Turkey
[4]Kocaeli Derince Training and Research Hospital, Department of Pediatric Nephrology, Kocaeli, Turkey

## ABSTRACT

**Objective:** To investigate the interaction between the roles of dietary excess sodium and low potassium intake in the pathogenesis of hypertension.

**Material and Methods:** The study consisted of 56 obese normotensive, 41 obese hypertensive and 29 healthy children as a control group, aged between 6 and 18 years. The ratio of urinary sodium to potassium (U Na/K) was evaluated in the afternoon urine samples in obese children. Also, the total body fat percentages were noted by using Tanita bio-impedance segmental body composition analyzer.

**Results:** Of the 41 obese hypertensive patients, 10 of them were ≤10 years old, and 31 of them were older than ten years. Insulin levels and HOMA-IR index in the obese normotensive and hypertensive groups were 16.7±8.3 and 15.9±7.9 U/ml, 4.4±3.4 and 5.3±3.4 respectively; these differences were not significant (P>0.05). The association of Na/K with blood pressure was not found statistically significant. However, it was found higher in OHT than ONT and control group.

**Conclusion:** Increased consumption of salty fat foods, high protein diets, and sedentary lifestyles predispose to obesity and related diseases.

**Key Words:** Hypertension, Obesity, Urinary Na/K, Urinary microalbumin/creatinine

## ÖZ

**Amaç:** Hipertansiyon patogenezinde diyetle aşırı sodyum ve düşük potasyum alımının ilişkisinin araştırılması.

**Gereç ve Yöntemler:** Çalışmaya 6-18 yaş arasında 56 obez normotansif, 41 obez hipertansif hasta ve kontrol grubu olarak 29 sağlıklı çocuk çalışmaya dahil edilmiştir. Öğleden sonraki idrar örneklerinde sodyum/potasyum oranı ve Tanita-biyoempedans yöntemi ile ayrıca total vücut yağ oranları değerlendirilmiştir.

**Bulgular:** Hipertansif 41 obez hastanın 10'u ≤10 yaş iken 31'i > 10 yaş olarak bulundu.

İnsülin düzeyleri ve HOMA-IR indeksleri, obez normotansif ve hipertansif gruplarda sırasıyla16.7±8.3ve 15.9±7.9 U/ml, 4.4±3.4 ve5.3±3.4 olarak bulundu, istatistiksel olarak anlamlı fark saptanmadı. İdrar Na, K, Na/K ve mikroalbumin/kreatinin oranlarında da kontrol, OHT ve ONT grupları arasında istatistiksel olarak anlamlı farklılık bulunmadı.

**Sonuç:** Artmış tuzlu ve yağlı yiyecek tüketimi, sedanter yaşam, hipertansiyon ve ilişkili hastalıklar açısından neden olabilecek faktörlerdendir.

**Anahtar Sözcükler:** Hipertansiyon, Obezite, İdrar Na/K, Mikroalbumin/kreatinin

## INTRODUCTION

Childhood obesity is a prominent metabolic disorder which extends into adulthood as a significant public health problem worldwide. Overweight and obesity in childhood lead to many disorders, one of which is hypertension (1). The International Study of Salt and Blood Pressure (INTERSALT Study) enunciated a considerable relationship between Na intake and blood pressure (2). Conforming to that, an increased intake in

Correspondence Address:
**Emel ISIYEL**
Sağlık Bilimleri Üniversitesi, Çocuk Sağlığı ve Hastalıkları Hematoloji Onkoloji Eğitim ve Araştırma Hastanesi, Çocuk Nefroloji Kliniği, Ankara, Turkey
E-mail: eisiyel@gmail.com

Received / Geliş tarihi : 25.03.2018
Accepted / Kabul tarihi : 30.05.2018
Online published : 12.07.2018
Elektronik yayın tarihi
DOI: 10.12956/tjpd.2018.370

dietary Na is associated with increased thirst and ingestion of water and/or sweetened, carbonated soft drinks that may be linked to the increased prevalence of overweight and obesity (3). Different studies and observational data suggest that there is an interaction between the roles of dietary excess sodium and low potassium intake in the pathogenesis of hypertension, such that the combined defect of diets high in Na and low in K on blood pressure seems greater than either alone (4).

The ratio of urinary Na to K (U Na/K) in the afternoon urine sample in obese children were evaluated and additionally, the total body fat percentages were noted by using Tanita bio-impedance segmental body composition analyzer. The purpose of this study was to investigate the association between urinary Na/K ratio, body fat composition, hypertension, and obesity.

## MATERIAL and METHODS

Ninety-seven patients (38 girls and 59 boys), aged between 6 and 18 years were added to the study, and 29 healthy children were added as a control group (CG) (18 girls and 11 boys). There were no acute or chronic kidney, cardiac or neurological disorder in either the patients nor in the control group. This study was approved by the Kocaeli University of Medical Sciences ethical committee, and oral informed consents were taken from all of the participants and their parents.

Height and weight samples were measured while patients were wearing light clothes and no shoes. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m2). Obesity (>95th percentile) and normal (between>5th and <85th percentile) weight definitions that were stated according to the BMI percentiles of the Turkish population based on gender and age, were used (5). Waist circumference (WC) was measured around the smallest area of the waist, approximately one inch (2.54 cm) above the umbilicus. Hip circumference (HC) measurement was taken around the largest area of the buttocks. An elastic measuring tape was used to measure WC and HC (6).

The Tanita (model MC-780MA; Tanita, Tokyo, Japan) bio-impedance segmental body composition analyzer was used to assess body composition in all subjects. This device calculates total body weight, body fat percentage (%), total body fat mass (FM) and total body water, truncal (core) FM on the basis of the data using bioelectrical impedance analysis (BIA) through the use of 8 electrodes (8-contacts; two on each hand and foot). The measurement procedures described in the pertinent studies were applied (7-9).

Insulin resistance (IR) was analyzed using the homeostasis model assessment of IR (HOMA-IR). HOMA-IR was calculated by the following formula: [fasting glucose (mg/dL) x fasting insulin (U/L)]/405 (10).

Spot urine samples were collected (12: 00-17: 00) and urinary Na, K, creatinine (Cre), microalbumin were measured.

Fasting blood samples were taken on the day of examination and blood glucose, lipids, routine laboratory parameters were recorded as well.

All patients and children in the control group were evaluated for hypertension status. Office BP measurements were performed using an aneroid sphygmomanometer with the appropriately sized cuff on the child's upper arm. Measurements were performed while the children were sitting after a rest of at least 5 minutes. Systolic blood pressure (SBP) was defined by the first Korotkoff sound and diastolic blood pressure (DBP) was identified by the disappearance of sounds. The mean of three readings was recorded as the Office BP. SBP and DBP percentiles were calculated according to the nomograms recommended by the National High Blood Pressure in Children and Adolescents Institute (11). Ambulatory blood pressure measurements (ABPM) were obtained by using a Spacelabs monitor. Cuff size was determined by measuring the circumference of the mid-arm. The patient or their parents were asked to keep a diary to record events on a 24-h basis, including the awake and asleep times; so, daytime and nighttime periods were determined according to these times recorded in their diaries. According to these records, ABPM data were registered. Hypertension was defined as BP values equal to or exceeding the 95th percentile for sex, age, and height or the 24-h SBP or DBP load at least 25% (12). According to these classifications, the obese patients were divided into two groups as obese hypertensive and normotensive. Obese hypertensive patients were evaluated for secondary hypertension reasons. Patients detected with secondary hypertension disease were excluded from the study.

### Statistical Analyses

Data were edited and analyzed by using the Statistical Program for the Social Science. Continuous clinical and biochemical variables are presented as mean (SD) or median values and quartiles as appropriate. Categorical variables are presented as frequencies and percentages. The Shapiro-Wilk test was used to assess the normality of continuous variables. To examine differences between two/more than two independent groups Student's t-test/analysis of variance (for normally distributed variables) or the Mann-Whitney/Kruskal-Wallis test (for non-normally distributed variables) was used. The post-hoc test was used to compare the differences between three groups. The chi-square analysis was used to compare the differences in nominal variables between the three groups. All statistical analyses were two-sided, and a $P < 0.05$ value was considered to be statistically significant.

## RESULTS

A total of 126 children were recruited to the study. Demographic characteristics of the study were given in Table I. Mean age at presentation was respectively 11.6±3.0 (ONT), 11.4±2.9 (OHT),

**Table I:** Demographic characteristics of the patients and control group.

| Demographic variables | ONT (n=56) | OHT (n=41) | Control (n=29) | p |
|---|---|---|---|---|
| **Males** | 33 (47.1%) | 26 (37.1%) | 11 (15.7%) | 0.085 |
| **Females** | 23 (41.0%) | 15 (26.7%) | 18 (32.1%) | |
| **Age** | | | | 0.01 |
| 6-10 yrs | 27 (50%) | 10 (18.2%) | 17 (31.2%) | |
| 11-18 yrs | 29 (40.2%) | 31 (43.0%) | 12 (16.6%) | |
| **Weight (kg)** | 61.7±22.7 | 66.2±19.0 | 41.2±14.1 | <0.001[a] |
| **Height (cm)** | 149.4±16.9 | 152.6±13.0 | 144.5±17.6 | 0.181 |
| **BMI (kg/m²)** | 26.6±4.9 | 27.7±4.7 | 19±2.5 | <0.001[a] |
| **BMI z-score** | 2.6±1.4 | 2.8±1.2 | -0.4±1.2 | <0.001[a] |
| **Percentage of body fat (%)** | 32.3±6.05 | 32.9±5.4 | 23.5±3.4 | <0.001[a] |
| **WC (cm)** | 86.8±12.2 | 87.8±10.8 | 71.2±10.3 | <0.001[a] |
| **HC (cm)** | 95.7±15.2 | 101.2±11.4 | 82.5±10.7 | <0.001[a] |
| **Waist/hip ratio** | 0.89±0.06 | 0.91±0.05 | 0.84±0.06 | <0.001[a] |
| **Waist/height ratio** | 0.54±0.05 | 0.58±0.04 | 0.48±0.05 | <0.001[a] |
| **Total body fat ratio** | 32.3±6.0 | 32.9±5.3 | 23.5±3.3 | <0.001[a] |
| **Central fat ratio** | 25.6±5.7 | 26.6±5.2 | 19.1±2.9 | <0.001[a] |
| **SBP (mmHg)** | 102.5±8.5 | 130.9±8.4 | 97.6±8.3 | <0.001[a] |
| **DBP (mmHg)** | 60.3±9.0 | 78.9±7.4 | 59.7±7.1 | <0.001[a] |

[a]*p<0.05 for controls vs. OHT and ONT groups combined.*

12.1±1.2 (CG). A statistically significant relationship was found between age and hypertension. Of the 41 obese hypertensive patients, 10 of them were 10 years old or younger than that and 31 of them were older than 11 years.

Insulin levels and HOMA-IR index in the ONT and OHT groups were 16.7±8.3 and 15.9±7.9 U/ml, 4.4±3.4 and 5.3±3.4 respectively; these differences were not significant (P>0.05). The OHT and ONT groups had significantly higher insulin, HOMA-IR, uric acid, LDL-C, VLDL-C, total cholesterol, triglyceride than the control group. Urinary Na, K, Na/K and microalbumin/creatinine were not found significantly different between the control, OHT and ONT groups. The results of laboratory findings were given in Table II.

BMI values, BMI z-scores, WC, HC, waist/hip ratio, waist/height ratio, uric acid, VLDL-C, and triglyceride values indicated a positive correlation with office SBP and DBP values and all ABPM parameters (p<0.05). A negative correlation was observed between ABPM values and HDL-C values (p<0.05). ABPM results are summarized in Table III.

Hypertension was detected with ambulatory blood pressure measurements performed after office blood measurements in 41 patients. Losartan potassium was started once daily. Echocardiography evaluation revealed left ventricular hypertrophy in 4 patients. All obese patients were recommended slimming diet and exercise by a nutritionist. Also, a no salt diet was recommended for hypertensive patients.

## DISCUSSION

Obesity has been proven to be one of the causes of hypertension in adults and children. It is estimated that at least 75% of cases of hypertension are directly related to obesity (13). Hypertension, diabetes mellitus, dyslipidemia and carotid atherosclerosis have been significant in the relationship between sodium intake and blood pressure (14, 15). An increased intake of dietary Na is associated with increased thirst and ingestion of water and/or sweetened, carbonated soft drinks that may be linked to the increased prevalence of overweight and obesity (3). Obesity results in renal sodium reabsorption by several mechanisms (16,17). As one of these, insulin has a direct effect on the kidneys, leading to increased sodium retention. Insulin also plays a role in the pathogenesis of hypertension by causing sympathetic activation in obese patients (18). We have clear findings revealing that children with obesity had significantly higher HOMA-IR compared with control group, however, there was no significant difference between obese hypertensive and normotensive groups. Uric acid, VLDL-C, and triglyceride values demonstrated a positive correlation with office SBP and DBP values and all ABPM parameters (p<0.05). A negative interdependence was observed between ABPM values and HDL-C values (p<0.05).

Salt-induced thirst and increased intake of sugar-sweetened soft-drinks or salt-promoted overeating induce high salt intake and obesity (3). A notable relationship between increased salt intake and hypertension has been proven by many studies

(14,15). The exact intake of sodium cannot be measured but can be estimated from 24-h urinary sodium excretion. However, 24-h urine collection has also some limitations because it is somehow inconvenient for children and parents. Recently, some studies have stated that spot urine Na/ K was closely correlated to 24-h urine sample Na/K. Lower potassium and higher sodium levels showed a positive correlation with BP in some adult studies (19). Although urinary Na, K, Na/K and microalbumin/ creatinine were not found significantly different between the control, OHT and ONT groups, they were found higher in OHT group than ONT and control group. Buendia et al.(20) reported that the increasing ratio of potassium and sodium intake induce

**Table II:** Laboratory findings of the study population and control group.

| Demographic variables | ONT | OHT | Control | p |
|---|---|---|---|---|
| **Fasting glucose (mg/dL)** | 86.0(80-98) | 88(83-93) | 84(81-99) | 0.041 |
| **Insulin (U/mL)** | 16.7±8.3 | 15.9±7.9 | 3.8±1.9 | <0.001[a] |
| **HOMA index** | 4.4±3.4 | 5.3±3.4 | 1.7±0.5 | <0.001[a] |
| **Uricacid (mg/dL)** | 5.5±1.3 | 5.6±1.2 | 4.4±1.5 | 0.005[a] |
| **HDL-C (mg/dL)** | 30.0 (36.0-46.0) | 44.0 (39.0-48.0) | 50.0(42.0-57.0) | 0.006[a] |
| **LDL-C (mg/dL)** | 86.3(81.0-101.0) | 92.5 (36.0-137.0) | 87.2(70-93.0) | 0.005[a] |
| **VLDL-C (mg/dL)** | 20.0 (16.0-26.0) | 24.6 (20.0-37.0) | 16.0 (12.5-19.4) | <0.001[a] |
| **Total cholesterol (mg/dL)** | 156.4±25.8 | 173.2±23.2 | 149.6±34.2 | 0.001[a] |
| **Triglyceride (mg/dL)** | 110.0(80-142.0) | 126.0(94.0-174.0) | 82.0(62.5-96.0) | 0.001[a] |
| **Urinary Na (mg/dL)** | 136±51.4 | 152±69.2 | 132±53.3 | 0.37 |
| **Urinary K (mg/dL)** | 90.0±48.5 | 72.6±30.4 | 90.3±42.3 | 0.179 |
| **Urinary Na/K** | 1.87±1.1 | 2.39±1.3 | 1.83±0.98 | 0.171 |
| **Urinary Na/Cre** | 1.18±0.68 | 1.48±0.75 | 1.2±0.72 | 0.174 |
| **Urinary microalb/Cre (mg/dL)** | 0.012±0.02 | 0.013±0.06 | 0.008±0.02 | 0.26 |

*[a] p<0.05 for controls vs. OHT and ONT groups combined.*

**Table III:** ABPM values of the three groups.

| Variables | ONT | OHT | Control | p |
|---|---|---|---|---|
| **Mean 24-h ABPM** | | | | |
| SBP(mmHg) | 114.5±4.8 | 129.4±15.1 | 96.2±7.3 | <0.001[a] |
| DBP(mmHg) | 62.4±5.2 | 76.2±5.2 | 65.2±6.1 | <0.001[a] |
| **Mean daytime ABPM** | | | | |
| SBP(mmHg) | 116.2±7.2 | 133.2±11.2 | 110.3±7.4 | <0.001[a] |
| DBP(mmHg) | 68.4±6.1 | 76.4±9.2 | 67.2±5.1 | <0.001[a] |
| **Mean nighttime ABPM** | | | | |
| SBP(mmHg) | 111.2±5.2 | 119.3±9.6 | 97.6±6.1 | <0.001[a] |
| DBP(mmHg) | 58.8±7.2 | 65.2±11.3 | 55.8±5.1 | <0.001[a] |
| **SBP load (%)** | | | | |
| Daytime | 13.5±5.1 | 48.2±19.1 | 5.4±3.1 | <0.001[a] |
| Nighttime | 14.3±3.1 | 38.1±13.1 | 2.8±3.1 | <0.001[a] |
| **DBP load (%)** | | | | |
| Daytime | 8.9±5.6 | 34.3±18.1 | 4.8±3.6 | <0.001[a] |
| Nighttime | 6.8±6.2 | 29.2±17.1 | 3.5±2.1 | <0.001[a] |
| **Nocturnal BP** | | | | |
| Dipping (%) | 35.6 | 21.8 | 17.2 | <0.001[a] |
| Non-dipping (%) | 64.4 | 78.2 | 82.8 | <0.001[a] |

*Data were presented as the mean±SD*
*[a]p<0.05 for OHT vs. control and ONT group.*

a statistically significant decrease in SBP in adolescent girls. In another study, Geleijnese et al.(4) showed that systolic blood pressure increased as the urine Na/K ratio raised. In these two studies, the patients were hypertensive, but not all of them were obese. The relationship between the urine Na/K ratio, hypertension and obesity may be demonstrated in the larger groups. This was one of the limitations of our study. Another limitation of this study was the lack of direct measurements of sodium or potassium via 24-hour urine collections.

WHR was founded significantly higher in OHT patients than the ONT and control groups in our study. Janghorbani et al.(21) also found a positive correlation between WHR and waist/height ratio and HT. WC and WHR measurements define central obesity and it is significantly related to hypertension and cardiovascular diseases; therefore, both of these measurements should be considered in evaluations of obesity.

Within our obese hypertensive patients, the age was ≤ 10 years in 10 and ≥11 years in 31 and this was found to be statistically significant. Between these two groups, there was no significant difference related to WC; HC; WHC, HOMA-IR.

In conclusion, the association of Na/K with blood pressure was not found to be statistically significant. However, it was found higher in OHT than ONT and the control group in our study. The importance of diet in obese hypertensive children may be demonstrated in further studies within larger groups by measuring the urine Na to K ratio.

## REFERENCES


1. Kotchen TA. Obesity-related hypertension: Epidemiology, pathophysiology, and clinical management. Am J Hypertens 2010;23:1170-8.
2. Lee S-G, Lee W, Kwon OH, Kim JH. Association of urinary sodium/creatinine ratio and urinary sodium/specific gravity unit ratio with blood pressure and hypertension: KHANES 2009-2010. Clinica Chimica Acta 2013;424:168-73.
3. Thuesen B, Toft U, Buhelt L, Linneber A, Friedrich N, Nauck M, et al. Estimated daily salt intake in relation to blood pressure and blood lipids: the role of obesity. European Journal of Preventive Cardiology 2015;22:1567-74.
4. Geleijnse JM, Grobbee DE, Hofman A. Sodium and potassium intake and blood pressure change in childhood. BMJ 1990;300:899-902.
5. Bundak R, Furman A, Gunoz H, Darendeliler F, Bas F, Neyzi O. Body mass index references for Turkish children. Acta Paediatr 2006;95:194-8.
6. Montague CT, O'Rahilly S. The perils of portliness: Causes and consequences of visceral adiposity. Diabetes 2000;49:883–8.
7. Reinehr T, Kiess W, Kapellen T, Andler W. Insulin sensitivity among obese children and adolescents, according to degree of weight loss. Pediatrics 2004; 114:1569-73.
8. Reinehr T, Andler W. Changes in the atherogenic risk factor profile according to degree of weight loss. Arch Dis Child 2004;89:419–22.
9. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, et al. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med 2004;350:2362-74.
10. Kurtoğlu S, Hatipoğlu N, Mazıcıoğlu M, Kendirici M, Keskin M, Kondolot M. Insulin resistance in obese children and adolescents: HOMA-IR cut-off levels in the prepubertal and pubertal periods. J Clin Res Ped Endo 2010;2:100-6.
11. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114:555-76.
12. Urbina E, Alpert B, Flynn J, Hayman L, Harshfield GA, Jacobson M, et al. American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee. Ambulatory blood pressure monitoring in children and adolescents: recommendations for standard assessment: A scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee of the council on cardiovascular disease in the young and the council for high blood pressure research. Hypertension 2008;52:433–51.
13. Strojny W, Drozdz D, Fijorek K, Korostynski M, Piechota M, Balwierz W, et al. Looking for new diagnostic tools and biomarkers of hypertension in obese pediatric patients. Blood Press Monit 2017;22:122-30.
14. Elliott P, Stamler J, Nichols R, Dyer AR, Stamler R, Kesteloot H, et al. Intersalt revisited: further analyses of 24 hour sodium excretion and blood pressure within and across populations. Intersalt Cooperative Research Group. BMJ 1996;312:1249–53.
15. Intersalt Cooperative Research Group. Intersalt: An international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. BMJ 1988;297:319–28.
16. Engeli S, Bohnke J, Gorzelniak K, Janke J, Schling P, Bader M, et al. Weight loss and the renin-angiotensin-aldosterone system. Hypertension 2005;45:356–62.
17. Bogaert YE, Linas S. The role of obesity in the pathogenesis of hypertension. Nat Clin Pract Nephrol 2009;5:101–11.
18. DeFronzo RA. Insulin and renal sodium handling: clinical implications. Int J Obes 1981;5 (Suppl 1): 93–104.
19. Adrogue´ HJ, Madias NE. Sodium and potassium in the pathogenesis of hypertension. N Engl J Med 2007;356:1966–78.
20. Buendia JR, Bradlee ML, Daniels SR, Singer MR, Moore LL. Longitudinal effects of dietary sodium and potassium on blood pressure in adolescent girls. JAMA Pediatr 2015;169:560-8.
21. Janghorbani M, Aminorroaya A, Amini M. Comparison of different obesity indices for predicting incident hypertension. High Blood Press Cardiovasc Prev 2017;24:157-66.