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Original article

The relationship between dietary salt intake and ambulatory blood pressure variability in non-diabetic hypertensive patients

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ABSTRACT

High dietary salt intake was reported to increase blood pressure by numerous studies, but no study has investigated the effect of dietary salt intake on blood pressure variability (BPV). This study aimed to determine if daily salt intake is related to ambulatory BPV. The study included 136 primary hypertensive patients (92 male, 44 female) with a mean age of 50.7 ± 11.1 years. All the patients underwent 24-h ambulatory blood pressure monitoring to determine both the 24-h systolic and 24-h diastolic BPV. 24-h urine sodium was measured. The correlation between BPV and 24-h urinary sodium was investigated. Logarithmic transformation of 24-h urinary sodium [log(24-h urinary sodium)] was positively correlated with the mean 24-h systolic ARV, and nighttime systolic ARV ($r = 0.371$ and $p = 0.001$, $r = 0.329$ and $p = 0.028$, respectively). Similarly, log(24-h urinary sodium) was positively correlated with mean 24-h diastolic ARV and nighttime diastolic ARV ($r = 0.381$ and $p = 0.001$, $r = 0.320$ and $p = 0.020$ respectively). Log(24-h urinary sodium) was an independent predictor of BPV based on multivariate regression analysis. Dietary salt intake might play a role in the pathogenesis of ambulatory BPV.

Relación entre el consumo de sal y la variabilidad de la presión arterial ambulatoria en pacientes hipertensos no diabéticos

Palabras clave:
Consumo de sal
Variabilidad de la presión arterial
Hipertensión

RESUMEN

En numerosos estudios se ha señalado que el consumo elevado de sal aumenta la presión arterial; no obstante, no se ha investigado el efecto de la ingesta alimenticia de sal sobre la variabilidad de la presión arterial (VPA). El objetivo de este estudio fue determinar si el consumo diario de sal está relacionado con la VPA ambulatoria. En el estudio se incluyeron

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136 pacientes hipertensos esenciales (92 hombres y 44 mujeres) con una edad media de 50,7 ± 11,1 años. Todos los pacientes se sometieron a una monitorización ambulatoria de la presión arterial de 24 h para determinar la VPA sistólica y diastólica de 24 h. Se midió la natriuria de 24 h y se estudió la correlación de la misma con la VPA. La transformación logarítmica de la natriuria de 24 h (log [natriuria 24 h]) se relacionó con certeza con el índice *Average Real Variability* (ARV) sistólico de 24 h y el ARV sistólico nocturno medios (r = 0,371 y p = 0,001, r = 0,329 y p = 0,028, respectivamente). De forma parecida, el log [natriuria 24 h] se relacionó con seguridad con el ARV diastólico de 24 h y el ARV diastólico nocturno medios (r = 0,381 y p = 0,001, r = 0,320 y p = 0,020, respectivamente). El log [natriuria 24 h] fue una variable independiente de la VPA, según el análisis de regresión multivariante. Es posible que el consumo de sal intervenga en la patogénesis de la VPA ambulatoria.

Introduction

Hypertension is a primary risk factor for cardiovascular and cerebrovascular diseases, and renal failure. It is well known that the quantity of dietary salt intake plays a role in the pathogenesis of primary hypertension. Increased sensitivity of blood pressure to excess sodium affects 50% of patients with primary hypertension.[1,2] Dietary salt intake causes an increase in blood pressure, and is associated with renal and cardiovascular diseases, including left ventricular hypertrophy and microalbuminuria.[3–5] Spontaneous variation in blood pressure is referred to as blood pressure variability (BPV), and is classified as short-term BPV and long-term BPV.[6,7] Fluctuation during a 24-h period is referred to as short-term BPV and is based on 24-h ambulatory blood pressure monitoring (ABPM). Variation between successive ABPM measurements is known as average real variability (ARV), which is mathematically calculated.[8,9] BPV was reported to be associated with hypertension-related target organ damage and cardiovascular mortality, independent of the blood pressure level. BPV, therefore, becomes more important day-by-day.[10–13] The pathophysiology of BPV is not fully known; however, it was reported that short-term BPV is primarily indicative of the effects of central and reflex autonomic modulation, and is associated with humoral, rheological, emotional, and behavioral factors.[9,14–18] The relationship between dietary salt intake and ambulatory BPV is not clearly known. Dietary salt intake is known to adversely affect the cardiovascular system, independent of the blood pressure level.[3,5] Excretion of sodium in 24-h urine is a measurement with proven validity that is commonly used to measure daily salt intake.[19,20] The aim of the present study was to determine if the quantity of daily salt intake is associated with ambulatory BPV.

Materials and methods

Study population

This study was performed at Ankara Numune Education and Research Hospital, Nephrology and Internal Medicine Clinic, Ankara, Turkey, and included 136 patients that presented between April 2013 and July 2013, and were diagnosed as primary hypertension. Exclusion criteria were diabetes mellitus, secondary hypertension, pregnancy, body mass index (BMI) > 30 kg/m^2, malignancy, rheumatic diseases, acute/chronic infection, liver disease, thyroid gland disease, adrenal insufficiency, syndrome of inappropriate antidiuretic hormone secretion, nephritis with salt loss, renal tubular acidosis, and a glomerular filtration rate <70 mL/min/1.73 m^2 and diuretic use. Duration of hypertension [years] was calculated based on patient self-reports of the date they were first diagnosed as hypertension to the date of inclusion in the study. The study was conducted in accordance with the Declaration of Helsinki and the study protocol was approved by the Ankara Numune Education and Research Hospital Ethics Committee. All the patients provided written informed consent to participate in the study.

Laboratory procedures

Blood samples were collected at 08.00–10.00, following overnight fasting. Laboratory evaluations included whole blood count, fasting blood glucose, urea, creatinine, sodium, potassium, total protein, and albumin. The GFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation: $GFR = 141 \times \min(Scr/\kappa,1)^{\alpha} \times \max(Scr/\kappa,1)^{-1.209} \times 0.993^{age} \times 1.018$ [if female] $\times$ 1.159.[21]

24-h urine collection

Patients were asked to collect 24-h urine; they were instructed not to save the urine from their first urination the morning they started to collect their urine, and to urinate into a collection container every time thereafter, including the first urination the following morning, and then to bring all collected urine to the laboratory. Patients were told not to make any changes to their daily dietary salt intake and to follow their normal diet during the time of urine collection. Urine sodium was measured in the patients' 24-h urine via the enzymatic colorimetric method using a Hitachi Modular P800 (Roche Diagnostic Corp. Indiana, USA) autoanalyzer. For each individual the 24-h sodium excretion value (mmol/d) was calculated