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ORIGINAL ARTICLE

Vascular effects of a single high salt meal

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KEYWORDS

Endothelial function;
Resistive index;
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Salt intake;
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Abstract *Background:* High salt intakes are associated with a greater incidence of strokes and cardiovascular events. Increased dietary salt for a long time impairs endothelial function. However, the immediate effect of only one high salt meal is not fully elucidated.
Aim of study: To detect vascular responses of a group of healthy adults to a single high-salt meal.
Subjects and methods: 63 volunteers (35 male and 28 female) aged 21–40 years were subjected to measurement of office blood pressure, plasma sodium, flow mediated dilatation and both resistive (RI) and pulsatility (PI) indices of renal as well as carotid arteries at baseline (fasting 8 h over night and only water is allowed) and 60 min after consumption of high sodium soup containing 4 g salt (equal to 68 mmol Na).
Results: There is significant increase in FMD as well as the resistive and pulsatility indices of both the carotid and femoral arteries after ingestion of the test meal compared to before meal ($P < 0.001$). Blood pressure is increased in the post-prandial phase but no correlation detected with these parameters ($P = 0.89$, 0.61 & 0.73 for carotid, 0.43 & 0.74 for renal). Plasma sodium increased after high salt meal (mean ± SD = 1.32 ± 0.83) and correlated with carotid PI ($P = 0.0001$).
Conclusion: High salt intake may acutely impair vascular function in different vascular beds independent of the increase of blood pressure. Plasma sodium increase may be one of the underlying mechanisms.

1. Introduction

Metabolic abnormalities in the postprandial state are known to contribute to endothelial dysfunction and atherosclerosis progression in healthy people.[1]

It has become increasingly clear that high dietary salt intakes are associated with a greater incidence of strokes and cardiovascular events[2] that is independent of its well-known ability to increase arterial pressure in some individuals.[3]

Endothelial dysfunction that is considered to be an initial step in the development of atherosclerosis[4], has been reported with chronic higher salt intakes.[5] Recently, it was proved that high salt diet impairs brachial artery FMD to a similar extent in adults with salt sensitive blood pressure and salt resistant blood pressure.[6]

Increased vascular stiffness is an early change in atherosclerosis.[7] An increase in the pulsatility index (PI) has been suggested to reflect distal vascular resistance.[8]

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The resistive index (RI) of the carotid artery which is a hemodynamic parameter that depends on the degree of vascular resistance can be assessed as a surrogate marker of generalized atherosclerosis[9] while the carotid pulsatility index (PI) was shown to be associated with several diseases such as microangiopathy in cerebral vessels[10,11] and significantly correlated with Framingham risk scores in subjects with hypertension.[11] Furthermore, systemic artery stiffness is correlated with carotid artery stiffness.[12]

Intrarenal resistance indices are a complex integration of arterial compliance, pulsatility, and peripheral resistance. They are associated with traditional cardiovascular risk factors as well as with subclinical atherosclerotic vessel damage.[13] The pulsatility index (PI) and the resistive index (RI) are used as pulsed-wave Doppler measurements of downstream renal artery resistance.[14] Recently, renal resistive index is considered as a marker of systemic vascular changes.[15]

However, the acute effect of single high salt diet is not fully elucidated.

2. Aim of the work

To detect vascular responses of a group of healthy adults to a single high-salt meal.

3. Subjects and methods

This study was carried out in cardiology department at El Minya University Hospital, Minya, Egypt, over the period between December 2013 and March 2015.

63 volunteers (35 males and 28 females) aged 21–40 years were recruited and included in this study after giving written informed consent.

Inclusion criteria were body mass index (BMI; in kg/m^2) $\geqslant 18$ and $\leqslant 25$, systolic BP (SBP) < 130 mm Hg, and diastolic BP (DBP) < 90 mm Hg.

4. Exclusion criteria

Exclusion criteria included atherosclerotic complications such as stroke and myocardial infarction; those undergoing hemodialysis, patients with peripheral vascular disease, malignancy, infections, hypertensive and diabetic patients were excluded.

All subjects were subjected to measurement of office blood pressure, plasma sodium, flow mediated dilatation and both resistive and pulsatility indices of renal as well as carotid arteries at baseline (fasting 8 h over night and only water is allowed) and 60 min after consumption of high sodium soup (prepared by member of public health and preventive medicine) containing 4 g salt (equal to 68 mmol Na).

Measurement of blood pressure (BP): seated BP was measured with an automated sphygmomanometer while fasting. Four consecutive BP measurements were taken 1 min apart. The first reading was discarded, and the mean of the next 3 consecutive readings with SBP readings within 10 mm Hg and DBP within 5 mm Hg of each other was taken as the fasting measurement. Blood pressure measurement was repeated 60 min after consumption of test meal.[16]

Endothelial function: to assess endothelial function non-invasively with B-mode ultrasound, conduit vessel endothelium-dependent vasodilatation was induced by reactive hyperemia, while endothelium-independent vasodilatation was induced by administration of sublingual nitroglycerine (glyceryl trinitrate; GTN).[17]

Measurements were made of changes in the diameter of the brachial artery using a pulsed wave Doppler with 7 MHz probe. The ultrasound examination was performed in quiet room at temperature between 21 °C and 32 °C. Subjects rested in a supine position for 15 min before examination. A B-mode scan was obtained of the right brachial artery in longitudinal section. A resting measurement was taken and called pre-flow mediated dilatation (pre-FMD), and a pneumatic cuff was then inflated to a pressure of 200 mm Hg for 5 min, then the diameter of the artery was recorded again 45–60 s after deflation (post-FMD) (Fig. 1). A period of 15 min was allowed for recovery before testing for endothelium-independent relaxation. A repeat baseline measurement of the diameter was taken before a 400 ug dose of sublingual GTN spray was administrated (pre-GTN). The brachial artery diameter was again measured 3–4 min after the GTN was given (post-GTN).[18]

FMD, GTN, and dilatation ratio were calculated as follows:

$$\text{FMD} = (\text{Post-FMD} - \text{Pre-FMD})/\text{Pre-FMD} \times 100.$$
$$\text{GTN} - \text{MD\%} = (\text{Post-GTN} - \text{Pre-GTN})/\text{Pre-GTN} \times 100.$$
$$\text{Dilatation ratio} = \text{FMD}/\text{GTN} - \text{MD\%} \times 100.$$

Duplex examination of the carotid arteries: ultrasound examination was performed while the patient was in a supine position. In all patients, routine carotid US examinations including gray-scale and color and pulsed Doppler ultrasound examinations of the left and right common carotid arteries (CCAs) and internal carotid arteries (ICAs) were conducted. All measurements were made by using angle correction. The peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI) were calculated.[19]

As regards the duplex examination of the renal arteries: duplex examination of both renal arteries with measurement

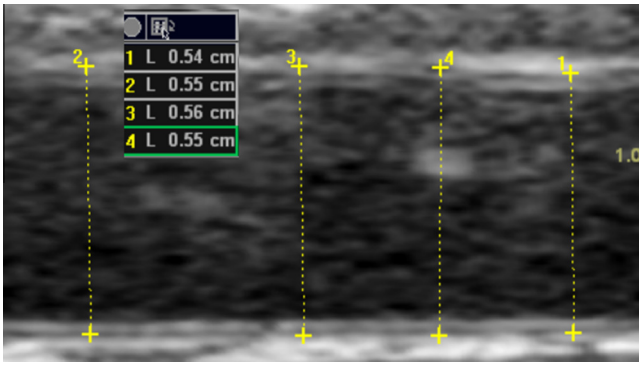

Fig. 1 Ultrasonographic measurement of brachial artery diameter.