Calcium Diet Supplementation Increases Urinary Sodium Excretion in Essential Hypertension

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Dear Sir,

We were most interested in the article published by Kojima and Ito [1] in this journal regarding the influence of high protein intake on blood pressure, sodium metabolism and urinary kallikrein excretion in salt-loaded rats. Apart from the activation of the renal kallikrein-kinin system, which likely promotes urinary sodium excretion and consequently lowers blood pressure, the possible natriuretic effects of the high calcium load given to the rats was not taken into consideration by these authors. The high-protein diet in this study was achieved by doubling the casein intake. Casein is the chief calcium-binding protein in the milk [2]. Since the urinary calcium excretion with the various diets is not specified in the study, enhanced calcium excretion during the high-protein diet cannot be ruled out. On the other hand, it is well known that calcium and sodium have in common transporting mechanisms in the proximal tubule [3]. There are also large epidemiological studies pointing out the inverse relation between blood pressure and calcium intake [4]. The beneficial effect of oral calcium supplementation on blood pressure in humans has recently been observed [5]. No certain explanation for the blood pressure-lowering effect of oral calcium has been provided so far. Another recent study showed an increase in urinary sodium excretion in salt-loaded rats during oral calcium supplementation [6].

This connection between oral calcium intake and urinary sodium excretion was the target of an ongoing study in our Unit. Fourteen patients with mild to moderate essential hypertension, without treatment, were put on a constant diet containing 120–130 mEq sodium per day. Half of them were then randomly given 1,000 mg of ionic calcium (Mega Calcium; Sandoz) per day for 5 days. After a 2-day washout period, the patients on the high-calcium diet were switched over to the normal calcium diet and vice versa. On the last day of each period, a 2-liter isotonic saline load was infused over 4 h. Urine was collected for 4 h. The main results follow very briefly:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Ca</th>
<th>High Ca</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary volume, ml/h</td>
<td>87.9 ± 22.7</td>
<td>135.0 ± 27.9</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Urinary sodium, mEq/h</td>
<td>18.9 ± 3.9</td>
<td>34.2 ± 5.0</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Urinary sodium/urinary Cr</td>
<td>0.303 ± 0.05</td>
<td>0.646 ± 0.16</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Osmolar clearance, mOsm/min</td>
<td>3.01 ± 0.39</td>
<td>3.97 ± 0.46</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

x ± SEM, Wilcoxon’s signed ranks test.

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[3] Calcium and sodium have in common transporting mechanisms in the proximal tubule [3].
[4] Epidemiological studies pointing out the inverse relation between blood pressure and calcium intake [4].
[5] Beneficial effect of oral calcium supplementation on blood pressure in humans has recently been observed [5].
[6] Another recent study showed an increase in urinary sodium excretion in salt-loaded rats during oral calcium supplementation [6].
A small but insignificant decrease in blood pressure was observed. We presume that at least one mode of the blood pressure-lowering effect of calcium supplementation is the enhanced natriuresis.

References