INVITED COMMENTARY

Calcium deficiency in the elderly: a factor contributing to the development of hypertension

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Several lines of evidence suggest a role for calcium deficiency in the pathogenesis of essential hypertension (1, 2). There exists a consistent inverse relationship between dietary calcium intake and blood pressure (3, 4). Urinary calcium excretion is increased in hypertensive patients, at any level of urinary sodium excretion (5). Low serum concentrations of ionized calcium have been measured in some patients with hypertension (6). Oral calcium supplementation can reduce the blood pressure of hypertensive patients (7–9). Finally, plasma parathyroid hormone (PTH) and calcitriol (1,25-dihydroxyvitamin D) levels are elevated in a fraction of patients with essential hypertension (10). Blood pressure increases with age but it is still unknown whether there exists any link between the calcium metabolism and the age-related blood pressure elevation.

The findings reported by A St John and his colleagues in this issue of the European Journal of Endocrinology describe for the first time the levels of PTH and calcitriol in elderly subjects. The individuals included in the study did not receive any antihypertensive drug, allowing us to look at relationships between the calcitrophic hormones and blood pressure. The main observation was a significant, positive correlation between both serum PTH and calcitriol concentrations and blood pressure. As anticipated, age and body mass index also were significant determinants of blood pressure. After the effects of these two confounding variables were taken into account by multivariate analysis, both calcitrophic hormones remained significant predictors of blood pressure. Unfortunately, it is not stated whether these relations are the same in men and women. It has also to be said that the clinical relevance of these statistically significant correlations remains to be established. Considering the large scatter of the individual values, it is obvious that any concentration of calcitrophic hormones can be associated with any blood pressure.

The data presented by A St John are compatible with a calcium deficit in elderly persons who exhibit the highest blood pressures. Hypocalcaemia could be responsible for an inhibition of Ca-ATPase activity, leading thereby to an increase in free intracellular calcium and a contraction of vascular smooth-muscle cells (11). Plasma renin activity tends to fall with ageing, most likely due to a progressive loss in the number of functional nephrons and an ensuing sodium retention (12). This is interesting considering the fact that ionized calcium levels are lowest and PTH and calcitriol concentrations are highest in hypertensive patients with suppressed renin secretion (10, 13). Alterations in sodium and calcium metabolism might therefore co-exist in some individuals and cause blood pressure elevations. Actually, calcium supplementation appears to reduce blood pressure only in low-renin, salt-sensitive patients with essential hypertension (10). The urinary excretion of calcium and sodium are known to change in parallel (13). The blood pressure lowering effect of calcium supplementation therefore could be mediated in part by a concurrent natriuresis. Elderly subjects may be particularly prone to develop calcium deficiency because of an insufficient calcium intake. It seems justified, therefore, to enquire about dietary habits of aged hypertensives and, if needed, to give some recommendations on not only sodium but also calcium intake, considering the relative harmlessness of increasing calcium intake.

References