Early complicated hypertension, hypokalaemia and salt taste abnormality: a possible link?

Sir,

We report the case of a 24-year-old woman, referred in September 1998 for grade 2 hypertension since 1994, associated with hypokalaemia (range 2.7–3.1 mEq/l) documented since 1987. Her medical history included an osteosarcoma of the left humerus in 1987 and an ischaemic stroke of the right sylvian area, in July 1998. Sitting blood pressure was 150/110 mmHg. Physical examination was otherwise unremarkable. Laboratory tests confirmed hypokalaemia (2.75 mEq/l, normal range 3.5–4.5 mEq/l) with mild alkalosis (CO2 30.5 mEq/l, normal range 24–28 mEq/l) and conserved urinary potassium excretion (36 mEq/24 h). Supine and standing plasma aldosterone levels were 0.36 and 0.55 nM, respectively (normal < 0.4 nM), with corresponding aldosterone to renin ratios of 6.2 and 5.8, arguing against the diagnosis of primary aldosteronism. Plasma cortisol, urinary excretion of cortisol, aldosterone, androstenedione, DHEA–sulfate and free DHEA were normal. Assays for diuretics in plasma and urine were negative. Abdominal MRI allowed to rule out renal artery stenosis and adrenal adenoma. Family screening failed to disclose hypertension or hypokalaemia in first-degree relatives. Interestingly, the patient declared that she ate little salt and had not felt salt taste since childhood. A gustometry performed according to Nejadnik [1] confirmed the absence of salt taste perception at usual concentrations. At very high concentrations (320 and 1000 mM NaCl; usual perception threshold for NaCl 7–15 mM), salt taste was not recognized as such but described as ‘bad’. Complex abnormalities of bitter, sour and sweet tastes were also noticed, bitter taste being perceived as sour and vice versa.

We hypothesized that the association of early complicated hypertension, hypokalaemia and salt taste abnormalities might reflect a common genetic abnormality of the Epithelial Sodium Channel (ENaC), involved in both Na\(^+\) reabsorption at the distal nephron [2] and salt taste perception at the anterior part of the tongue [3]. In order to test this hypothesis, we screened the last exons of the β and γ subunits of ENaC, already involved in Liddle’s syndrome [2] and possibly other forms of hypertension [4]. Though no mutation was found, we cannot rule out an influence of ENaC variants located elsewhere. Whatever the mechanism, several studies have shown heightened thresholds for salt recognition in hypertensive patients. This phenomenon could reflect either an abnormality of Na\(^+\) transfer in papillae, kidney tubules and/or vascular smooth muscle cells, or a down-regulation of Na\(^+\) transporter(s) secondary to hypertension [5]. Though unsolved, this case report emphasizes the interest of looking for salt taste disorders in patients with early hypertension and hypokalaemia. More generally, the hypothesis of a link between salt taste abnormalities and high blood pressure might deserve to be revisited, in light of recent advances in the molecular basis of taste transduction and hypertension.

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