PATHOPHYSIOLOGY OF EXAGGERATED NATRIURESIS OF HYPERTENSION

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Acute volume expansion in patients with hypertension results in a more prompt rise in sodium excretion than in the normotensive subject [1]. The present study was designed to evaluate the mechanism of such an exaggerated natriuresis in hypertensive man. Seven hypertensive patients (H) and 8 normotensive volunteers (N) were loaded with NaCl (4.5 mEq Na⁺/kg body wt, i.v.) during water diuresis (A) and antidiuresis (B). In 6 antidiuretic subjects (3 H and 3 N) urine volume (V) was progressively raised up to over 30ml/min, by i.v. infusion (12ml/min) of hypertonic saline (C). Clearance of Inulin (C_I), PAH (CPAH) and osmolar clearance (C_Osm) were measured before and after saline loading in A and B. C_Osm was monitored at 10-minute intervals during hypertonic infusion in C. It is assumed that in A, V approximates the volume of fluid delivered out of the proximal tubules and free-water clearance (C_{H₂O}) is an index of Na⁺ reabsorption in short Henle’s loops. In B and C free-water reabsorption (T_{cH₂O}) is proportional to Na⁺ reabsorption in long Henle’s loops [2]. As expected, the increment in urinary Na⁺ excretion rate determined by saline infusion was more rapid in H than in N both in A and in B. This exaggerated natriuresis was not accompanied by any difference in C_I nor in C_PAH. In A, C_{H₂O} was significantly lower in H than in N (15.2 ± 1.0ml/min SE compared with 20.2 ± 1.7 SEml/min, p < 0.05). In C, T_{cH₂O} reached an earlier plateau in H than in N (at about 8ml/min and 12ml/min, respectively). The reduced C_{H₂O} in H demonstrates that exaggerated natriuresis depends on defective Na⁺ reabsorption in Henle’s loops. The results suggest that this defect is accounted for by the transmission of hypertension in medullary circulation. According to this hypothesis T_{cH₂O} is not reduced in B, since the defect in Na⁺ reabsorption in long loops caused by the increase in haemodynamic pressure in vasa recta is blunted by a greater Na⁺ delivery, secondary to the increase in glomerular filtration rate, that follows the rise in filtration pressure in juxtamedullary nonautoregulating nephrons [3,4]. The fall in T_{cH₂O} becomes apparent only when the reabsorptive capacity of Henle’s loops is saturated (in C).
References

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