Red Blood Cell Na⁺-K⁺ Pump Activity and Arterial Hypertension in Autosomal Dominant Polycystic Kidney Disease

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Table 1. Intraerythrocyte Na concentration, fluxes, and selected clinical and hormonal parameters in normotensive and hypertensive ADPKD patients

Normotensive

Hypertensive

* p < 0.003 versus normotensive ADPKD patients by Mann-Whitney U test.

Dear Sir,

We have read with interest the Editorial by Zeier et al. [1] published in a recent issue of your journal.

We have obtained evidence for a role of altered Na transport through the Na⁺-K⁺ pump in the pathogenesis of arterial hypertension in autosomal dominant polycystic kidney disease (ADPKD).

In a study on 15 patients with ADPKD (5 males and 10 females, median age 33 years, range 16-51 years) and normal or near-normal renal function (median glomerular filtration rate measured as the EDTA 51Cr clearance 83 ml/min/1.7 m², range 63-131 ml/min/1.7 m²), we evaluated the intracellular Na concentration and the rate constant for ouabain-sensitive Na efflux in fresh red blood cells, according to previously reported methods [2]. Six patients were hypertensive, defined as having a sitting blood pressure > 140/90 mm Hg or when taking antihypertensive drugs which were withdrawn at least 1 week prior to the study.

The results (table 1) show that hypertensive patients have a significantly higher in-tracellular Na concentration and a lower rate constant for ouabain-sensitive Na efflux than normotensives.

Moreover, the plasma renin activity tended to be lower and the atrial natriuretic peptide level higher in hypertensive ADPKD patients, possibly in keeping with the existence of an expanded extracellular fluid volume.

Significant correlations (calculated using Spearman's rank correlation) were found in the entire group of patients between mean arterial pressure and both intracellular Na concentration (rho = 0.66; p = 0.01) and rate constant for ouabain-sensitive Na efflux (rho = -0.69; p = 0.01).

Moreover, the rate con-

stant for ouabain-sensitive Na efflux was directly correlated with the plasma renin activity ($\rho = 0.61$; $p = 0.02$).

In the 6 hypertensive patients, the rate constant for ouabain-sensitive Na efflux was strongly inversely related to the systemic vascular resistance, calculated as the ratio of cardiac output, measured by transthoracic bioimpedance, to mean arterial pressure ($\rho = -0.87$; $p < 0.05$). Taken together, these observations suggest that in ADPKD patients the abnormal renal Na handling [3-5] could lead to hypertension through volume expansion, secretion of an ouabain-like factor, and inhibition of the Na⁺-K⁺ pump with an increase in vascular reactivity.

References

- Zeier M, Ritz E, Geberth S, Gonzalo A: Genesis and significance of hypertension in autosomal dominant polycystic kidney disease. *Nephron* 1994;68:155-158.
- Quarello F, Boero R, Guarena C, Rosati C, Giraudo G, Giacchino F, Piccoli G: Acute effects of hemodialysis on erythrocyte sodium fluxes in uremic patients. *Nephron* 1985;41: 22-25.
- Torres VE, Wilson DM, Offord KP, Burnett JC, Romero JC: Natriuretic response to volume expansion in polycystic kidney disease. *Mayo Clin Proc* 1989;64:509-515.
- Schmid M, Mann JFE, Stein G, Herter M, Nussberger J, Klingbeil A, et al: Natriuresis-pressure relationship in polycystic kidney disease. *J Hypertens* 1990;8:277-283.
- Nash DA Jr: Hypertension in polycystic kidney disease without renal failure. *Arch Intern Med* 1977;137:1571-1575.

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