Effect of Hemodialysis on Red Blood Cell $\text{Na}^+\text{-K}^+\text{-ATPase}$ Activity in Terminal Renal Failure

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In chronic renal failure (CRF), intracellular Na^+ concentrations have been shown to be increased [1]. This increase has been related to partial inhibition of the sodium pump in red blood cells (RBC) [2, 3].

In patients with CRF, we have shown recently low RBC $\text{Na}^+\text{-K}^+\text{-ATPase}$ activity, which was totally normalized after successful renal transplantation [1, 4, 5]. Thus, we could speculate on the presence of an endogenous < uremic > toxin which could be responsible for the partial inhibition of RBC $\text{Na}^+\text{-K}^+\text{-ATPase}$ in CRF.

Therefore, we studied the effect of hemodialysis on RBC $\text{Na}^+\text{-K}^+\text{-ATPase}$ activity, as measured by digoxin-sensitive ^{86}Rb uptake, in patients with CRF.

30 patients with various causes of CRF, excluding hypertension, aged 23–72 has been studied. Digoxin-sensitive RBC ^{86}Rb uptake [4–6], blood urea nitrogen, serum creatinine and plasma potassium concentrations were measured prior to and at the termination of one of the twice weekly routine hemodialysis periods.

As compared to age-matched healthy volunteers, RBC digoxin-sensitive ^{86}Rb uptake, before dialysis, was decreased in the studied patients ($23.77 \pm 6.23\%$ vs. $28.65 \pm 5.16\%$; $p < 0.05$; Student's unpaired test), which is in agreement with our previous findings [5]. After dialysis values increased significantly to $25.7 \pm 6.5\%$ (mean $\Delta\% = 2.4 \pm 0.84\%$; $p < 0.01$; Student's paired t test, fig. 1). This increase was not related to changes in body weight (66.3 ± 14.0 to 61.2 ± 18.4 kg); blood urea nitrogen (30.7 ± 5.6 to 19.5 ± 3.6 mmol/l); serum creatinine (1.031 ± 295 to 428 ± 169 mmol/l).

The mechanism of this increase is yet to be demonstrated. Many authors however, postulated that an endogenous toxin with digitalis-like properties could be present in the plasma of patients with renal failure [7, 8].

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After

Before

$p < 0.01$ n = 30 Hemodialysis

Fig. 1. Digoxin-sensitive ^{86}Rb uptake before and after hemodialysis.

Our results do not provide any evidence of the presence of a such substance. But, if present and dialysable, partial clearance of this endogenous toxin could explain the rise of ^{86}Rb uptake after hemodialysis in our patients.

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