Effect of Hemodialysis on Red Blood Cell Na\(^+\)-K\(^+\)-ATPase Activity in Terminal Renal Failure

F. Zannad, M. Kessler, R.J. Royer, J. Robert

Laboratoire de Pharmacologie, Service de Néphrologie et de Médecine Nucléaire, Vand\'uvre les Nancy, France

In chronic renal failure (CRF), intracellar 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 Na\(^+\)-K\(^+\)-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 Na\(^+\)-K\(^+\)-ATPase in CRF.

Therefore, we studied the effect of hemodialysis on RBC Na\(^+\)-K\(^+\)-ATPase activity, as measured by digoxin-sensitive 86-rubidium uptake, in patients with CRF.

30 patients with various causes of CRF, excluding hypertension, aged 23–72 has been studied. Digoxin-sensitive RBC 86Rb 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 86Rb uptake, before dialysis, was decreased in the studied patients (23.77 ± 6.23% vs. 28.65 ± 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 ± 6.5% (mean Δ % = 2.4 ± 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 9.5 ± 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].
p < 0.01 n = 30 Hemodialysis

Fig. 1. Digoxin-sensitive 86Rb 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 Rb86Rb uptake after hemodialysis in our patients.

References


