Dear Sir,

It has been found recently that secondary hyperparathyroidism develops more slowly in diabetics when compared with non-diabetic patients on long-term hemodialysis [1]. These data are in agreement with the results reported in other studies [2–4], indicating that serum parathyroid hormone (PTH) levels are significantly lower in diabetics than in non-diabetics undergoing long-term hemodialysis. Vincenti et al. [5] showed that a low rate of bone formation is a prominent finding in dialysis-treated diabetics, and Andress et al. [6] suggested that the low rate of bone formation may be due to the increased rate of accumulation of aluminum in bones of diabetics on long-term hemodialysis. Factors responsible for reduced PTH secretion in diabetics on hemodialysis remain to be clarified because insulin-dependent diabetes mellitus is associated with a low rate of bone formation (and osteopenia) in patients with normal renal function [7], and certain observations indicated that aluminum can inhibit the PTH secretion [8, 9].

We report on the investigation of secondary hyperparathyroidism and aluminum concentration in patients with insulin-dependent diabetes and non-diabetic patients undergoing a long-term hemodialysis.

Serum calcium, phosphate, PTH and aluminum levels were measured and radiological investigation of the bone status was performed in 20 insulin-dependent diabetics (12 females and
8 males) and 20 (10 females and 10 males) age-matched non-diabetics (mean age 44.4 vs. 40.1 years) on long-term hemodialysis (mean 4.5 vs. 3.5 years). Hemodialysis was performed three times a week with a dialysate containing 1.75 mmol/l calcium and less than 0.37 µmol/l aluminum in both groups. The mean dose (± SD) of calcitriol was not significantly different in diabetics when compared with the non-diabetic group (0.25 ± 0.08 vs. 0.30 ± 0.14 µg/day) as well as the

\[
\begin{align*}
8,000 & \quad 5,360 \pm 1,658 \\
6,000 & \quad 5,000 \\
4,000 & \quad 4,000 \\
3,000 & \quad 3,000 \\
1,000 & \quad 1,000 \\
0 & \quad 0
\end{align*}
\]

\[p < 0.01\]

Fig. 1. Serum aluminum levels (a) and PTH concentrations (b) in the diabetic (■) and non-diabetic (¤) groups (mean ± SD).

duration of the therapy (3.0 ± 0.4 vs. 2.86 ± 1.06 years). The oral intake of aluminum hydroxide was significantly higher in the non-diabetic group (0.45 ± 0.05 vs. 0.25 ± 0.04 kg/year, \(p < 0.01\)). None of the patients had undergone previous parathyroidectomy, and no exogenous blood was administered for 2 months prior to the study. The aluminum concentration in serum was determined by graphite-furnace atomic-absorptiometry \[10\] and PTH was measured with a commercial radioimmunoassay kit (Byk Sangtec, Dietzen-bach, FRG). The radiological investigations included conventional views of lumbosacral spine, pelvis, hands and feet.

Our findings demonstrated that PTH levels were significantly lower (1,106 ± 540 vs. 5,360 ± 1,658 pmol/l, \(p < 0.01\), fig. 1b) in insulin-dependent diabetics in comparison to non-diabetic uremics. Serum aluminum concentrations, however, were significantly higher in insulin-dependent diabetics versus non-diabetic uremics on long-term hemodialysis (1.67 ± 0.28 vs. 0.54 ± 0.31 µmol/l, fig. 1a, \(p < 0.01\)).

Aluminum and PTH Concentrations in Insulin-Dependent Diabetics on Long-Term Hemodialysis


Phalangeal subperiosteal erosions were absent in diabetics, but were found in 35% of non-diabetics. Rugger-Jersey appearance of the spine was seen in 4% of diabetics and in 20% of non-diabetic uremics.

The results of this investigation are in accordance with recent reports [1, 2] indicating that secondary hyperparathyroidism develops more slowly in diabetics than in non-diabetics undergoing long-term hemodialysis.

Data from this study demonstrated that the serum aluminum level is significantly elevated in insulin-dependent diabetic patients in comparison to non-diabetic uremics. The differences in aluminum concentration cannot be explained with different aluminum load since both groups of patients were on the same therapy regimen with the identical exposure to aluminum contaminated dialy-sate. However, the oral intake of elemental aluminum was even lower in diabetic patients. This may indicate that elevated aluminum concentrations in diabetics is eventually related to diabetic gastroenteropathy and/or other factors promoting the enhanced rate of aluminum absorption. Also, it should be emphasized that because of the low aluminum loading none of the patients have had an aluminum level compatible to that associated with a considerable risk of aluminum toxicity.