Hyperparathyroidism after Aluminum Depletion

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Dear Sir,

Recently, Anelli et al. [1] have reported the development of hyperparathyroidism after aluminum depletion in a group of patients on chronic hemodialysis. In this letter we want to present our experience and make some comments.

In a similar study, 18 patients on chronic hemodialysis were followed-up during 1 year after an intensive aluminum depletion regimen. The patients were divided in two groups: group A consisted of patients (n = 7) with aluminum intoxication (basal aluminum > 80 ng/ml and de-feroxamine (DFO) test > 180 ng/ml); the mean time on hemodialysis was 12 ± 4 years; group B comprised patients (n = 11) without aluminum intoxication (basal aluminum < 80 ng/ml), with a mean time on hemodialysis of 3.4 ± 2.6 years ($p < 0.001$). All patients were directed to discontinue $\text{Al}(\text{OH})_3$ and to start taking CaCo_3 as phosphate binder. Calcium concentration in the dialysate was diminished to 1.49 mmol/l. Simultaneously, the patients of group A received DFO chelating treatment (1.5–3 g/week).

As Anelli et al. [1] we observed that serum aluminum levels dropped quickly after $\text{Al}(\text{OH})_3$ withdrawal. Eight weeks there after, serum aluminum levels were 157 ± 63 and 44 ± 17 ng/ml in group A and B, respectively ($p < 0.05$ compared to basal levels; fig. 1). The follow-up of parathyroid hormone-N (PTH-N) levels showed that patients of group A clearly tended to develop hyperparathyroidism, especially after the 4 months. Basal PTH-N levels increased from 21 ± 4 to 54 ± 20 ng/ml ($p < 0.001$) after 4 months and to 62 ± 26 ng/ml ($p < 0.008$) after 12 months. PTH-N levels in group B were not statistically different after 12 months of follow-up (basal 22 ± 9 , at 12 months 26 ± 24 ng/ml; p. n.s. fig. 2). Calcium and phosphorus levels were similar during the follow-up and in both groups, rather calcium levels in patients of group A were higher than in group B, debarring hypocalcemia as the cause of hyperparathyroidism.

Basal

2 months

6 months

12 months

Fig. 1. Evolution of serum aluminum levels in patients with (#) or without (O) aluminum intoxication.

Basal

6 months

12 months

Fig. 2. Follow-up of serum PTH-N levels in patients with (■) or without (□) aluminum intoxication.

There are several lines of evidence that suggest an inhibitory effect of aluminum on parathyroid gland function [2–4]. Recent studies have suggested that aluminum somehow impairs the secretion of PTH, acting on the secretion process itself [2]. This explains that aluminum-related bone disease courses with low levels of PTH [5],

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and that parathyroidectomy is followed by an increase in 4 bone surface aluminum deposition concomitant with a decrease in bone formation rate. In clinical experience, few studies have evaluated in the longterm the effect of aluminum depletion on parathyroid activity in patients with aluminum intoxication. Some reports [1, 6–8], including Anelli et al. and our's, have demonstrated a rise in parathyroid activity on removal of patients from high-aluminum exposure. This could be an important factor to take in account after aluminum depletion, because some PTH osteodystrophic lesions may develop.

8 References

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