Bone Aluminum Intoxication: An Unpreventable Sequel of Parathyroidectomy?

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Dear Sir,

Recent studies have shown that parathyroidectomy (PTX) is followed by an increase in bone surface Al deposition, concomitant with a decrease in bone formation rate [1,2]. These findings imply that the indication for PTX in patients on chronic dialysis should be weighed against the risk of favoring the occurrence of Al-associated osteomalacia.

We report here our observation on a single patient suggesting that increased bone Al storage does not inevitably follow PTX. A 30-year-old woman on hemodialysis treatment for 9 years, was referred to us because of very severe clinical biochemical and radiological evidence of dialysis osteodystrophy. She had been taking Al hydroxide as phosphate-binder for several years and was dialyzed against a dialysate that had always been prepared from deionized water. Bone histomorphometry showed predominant hyperparathyroidism changes (table I). Both serum Al levels and bone Al content, measured by atomic absorption spectrophotometry, were high (table I). Aluminon staining covered only 2.8% of total trabecular surfaces, being mainly distributed on resorbing or neutral surfaces.

The patient underwent PTX with removal of four hyperplastic glands weighing altogether 4 g. Following PTX, calcium supplements as well as 25-D3 and 1,25-D3 were prescribed, while Al hydroxide was stopped; the patient continued on this regimen throughout the follow-up. After 3 years a repeated bone biopsy showed a remarkable regression of bone lesions, while serum and bone

Al concentrations fell to almost normal values (table I). Aluminon staining was similar in degree and distribution to that observed in the pre-PTX bone sample.

The increased bone surface Al deposition rate after PTX observed by Andress and de Vernejoul [1, 2], contrasting with the reduced bone Al content and no increase in bone surface Al found by us, could be explained on the basis of the different oral Al intake after PTX. In fact phosphate-binder intake in the patients of Andress et al. [1] and in the majority of those of de Vernejoul et al. [2], did not change after PTX; on the contrary, our patient

Table I. Bone histological parameters, bone and serum Al concentrations before and after PTX

stopped taking Al hydroxide. Moreover, even in the study of de Vernejoul et al. [2], if the 3 patients who stopped Al hydroxide after PTX are considered separately, stainable bone Al did not increase. It is therefore plausible that after PTX bone Al deposition rate increases only if patients continue to be exposed to Al. When the exposition to Al is stopped, as was the case with our patient, no further Al deposition occurs and the accumulated Al is slowly removed from the bone.

The changes in bone histology and in bone Al content observed in our patient cannot be ascribed to PTX alone, but represent the combined effects of PTX, Al hydroxide withdrawal and, perhaps, the consumption of vitamin D3 metabolites. Therefore, we believe that the increased bone Al deposition rate occurring after PTX can be prevented by carefully avoiding an Al burden from any source.

References

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