Does Long-Term Calcitriol Therapy in Dialyzed Patients 'Turn Hyperparathyroid' into 'Osteomalacic' Problem?

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Dear Sirs,

Dialysis patients who are long-term treated by means of calcitriol for secondary hyperparathyroidism frequently need posology reduction or treatment discontinuation because of persistent hypercalcemia, mostly as alkaline phosphatases reach normality [1]. These patients also often show a fall in iPTH levels and either a healing or an improvement of radiological and hystological findings of hyperparathyroidism [2]; on the contrary, osteo-malacic patterns sometimes improve [3], but generally they do not parallel the treatment [2, 4].

These findings have been confirmed by our experience with more than 55 dialyzed patients, treated with calcitriol up to 30 months [5].

But another group of dialyzed patients exhibits, when not treated, peculiarities which seem very similar, at least in our opinion, to those shown by patients who were long-term treated with calcitriol: these are patients with so-called vitamin-D-resistant osteomalacia, showing low iPTH levels, low or only slightly increased alkaline phosphatases, no radiological and hystological findings of hyperparathyroidism, tendency to develop hypercalcemia with no or small doses of vitamin D and no healing of osteomalacia under any kind of treatment [6, 7].

This puzzling resemblance raises several questions and hypotheses. It may be possible that long-term treatment with calcitriol discloses in the first group of patients an underlying osteomalacic lesion already evident in the second group. This hypothesis could be supported by the observation that parathyroid hyperactivity seems to be necessary to allow vitamin D to improve osteomalacia [8], an by the worsening of osteomalacia after parathyroidec-tomy [9].

Moreover, an osteoblast depression has been found in vitamin-D-resistant osteomalacia [10], while there is evi-

dence that calcitriol can reduce the number of osteoblasts in dialyzed patients [8].

The possible implication of aluminium in vitamin-D-resistant osteomalacia does not contradict our hypothesis, because aluminium bone deposits are probably much more frequent than we think in dialyzed patients and may be present, at least at lower levels, also in hyperparathy-roid patients with no evidence of overt osteomalacia.

Does long-term calcitriol therapy reveal aluminium bone intoxication in these patients?

We think this hypothesis to deserve attention and further investigation.

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