Low-Calcium Dialysate and Dosage of Calcium Carbonate

To the Editor:

We have read with interest the article by Fernández and associates about secondary hyperparathyroidism (SHPTH), which was published in the July issue of the Journal of the American Society of Nephrology (1). They reduced the dialysis fluid calcium to 1.25 mmol/L with a simultaneous substitution of aluminum hydroxide by calcium carbonate. The calcium carbonate dosage ranged from 3 to 6 g/day. They found that low-calcium dialysate induces a negative calcium balance during hemodialysis (HD) with a worsening of SHPTH and concluded that a change in the current policy of the treatment of renal osteodystrophy can be necessary to prevent these alterations.

For 1 yr, we studied the effect of low-calcium dialysate on the severity of SHPTH in 26 HD patients without vitamin D therapy in the previous 2 yr. The dialysate calcium concentration was lowered from 1.62 to 1.25 mmol/L. Gradually, we increased the dose of calcium carbonate and decreased the dose of aluminum hydroxide. One year after the dialysate calcium was lowered, the oral dose of calcium carbonate rose from 3.2 ± 2.6 to 9.2 ± 5.6 g/day (P < 0.001), with stable levels of serum calcium (8.8 ± 1.1 versus 9.1 ± 1.4). In 22 patients (85%), aluminum hydroxide was stopped, and in the remaining 4 patients, the dose was lowered. These variations did not produce an increase in the incidence of hypercalcemia or hyperphosphoremia. In the total group, we did not find a significant variation in the levels of intact parathyroid hormone (iPTH) (324 ± 115 versus 311 ± 256 pg/mL) or alkaline phosphatase (230 ± 115 versus 224 ± 127). We analyzed the evolution of iPTH in each case. In 15 patients (58%), the iPTH decreased; in 6 subjects (23%), it remained stable, and in only 5 (19%), it increased. However, one of these five patients had a basal iPTH that was inappropriately low (57 pg/mL) and it increased to an adequate level (187 pg/mL); two of the remaining four subjects did not take the recommended calcium dosage. The tolerance to low-calcium dialysate was good. There were no modifications in the predialysis calcium dosage. The tolerance to low-calcium dialysate basal iPTH that was Inappropriately low (57 pg/mL) and of intact parathyroid hormone (IPTH) (324 ± 115 versus 224 ± 127). Gradually, we increased the dose of calcium carbonate in these patients could prevent the net calcium loss and avoid the repetitive stimulation of PTH secretion during HD.

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Response:

The results of the literature regarding the effect of low-calcium dialysate on the parathyroid function are contradictory. In our opinion, this apparent controversy can be explained because the real calcium balance is difficult to assess in clinical studies. It is an established fact that in patients with chronic renal failure, there is a resistance to the inhibitory effect of calcium in the synthesis of parathyroid hormone (shift of the set point of calcium to the right). Therefore, if the negative calcium balance provoked by low-calcium dialysate is neutralized with a high dose of calcium compounds and/or vitamin D, the hyperparathyroidism does not worsen, as occurs in the report of Navarro et al.

On the other hand, we disagree with the authors' conclusions in their letter for the following reasons. First, at the moment, it has not been demonstrated that large doses of calcium supplements are safe. The normal serum calcium levels neither reflect the real calcium balance nor rule out the risk of soft tissue calcifications. In the second place, considering the most recent data from the literature (1–3), there is a general consensus that secondary hyperparathyroidism is best treated by "pulse" calcitriol (oral or intravenous). Finally, the hypothesis that high doses of calcium carbonate could avoid the intradialysis stimulation of the parathyroid secretion is not supported by data in the authors' study.

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