Pharmacological Parathyroidectomy by Oral 1,25(OH)\textsubscript{2}D\textsubscript{3} Pulse Therapy

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Dear Sir,

Renal osteodystrophy is one of the major complications of chronic renal failure. Many uremic patients, especially those who have been receiving hemodialysis for a long time, suffer from bone pain and bone fractures caused by this disorder. Avoidance of this disorder can be achieved by prevention or treatment of secondary hyper-parathyroidism (HPT). Prevention of secondary HPT can be accomplished by oral administration of 1,25-dihydroxycholecalciferol [1,25(OH)\textsubscript{2}D\textsubscript{3}] combined with phosphate binders. However, the treatment of secondary HPT cannot always succeed by a maintenance dose of 1,25(OH)\textsubscript{2}D\textsubscript{3} when the serum parathyroid hormone (PTH) level is very high. In order to treat secondary HPT without a surgical procedure, Slatopolsky et al. [1] reported that intravenous administration of high-dose 1,25(OH)\textsubscript{2}D\textsubscript{3} was effective in suppressing PTH secretion. Although their attempt was very successful, this therapy is not available for patients at the present time, because the 1,25(OH)\textsubscript{2}D\textsubscript{3} preparation for intravenous administration is no longer on the market.

We report here that periodic oral administration of high doses of 1,25(OH)\textsubscript{2}D\textsubscript{3} was also very effective in reducing the high serum PTH level. 1,25(OH)\textsubscript{2}D\textsubscript{3} was administered orally to 9 patients undergoing hemodialysis at the end of each dialysis session twice a week for a period of 20 weeks. The serum PTH (carboxy-terminal) level was higher than 7.0 ng/ml, and the average value was 17.2 ± 7.7 ng/ml (normal range in our facility 0.20–1.00 ng/ml) at the beginning of the therapy. Initially, the dose was 2.0 µg and was increased to 6 µg. The dose was determined so that the serum calcium level did not exceed 11 mg/dl. Aluminum hydroxide was administered to maintain the serum phosphorus level below 5.0 mg/dl. The results are shown in table I. One of the clinical courses of therapy is also depicted in figure 1. The serum PTH-level (c-terminal and midregion) decreased dramatically in every patient under therapy, and the average decrement was 41% of the initial level after 5 months of treatment. The serum alkaline phosphatase level (normal range 73–248IU) also decreased significantly, suggesting improvement of an excessive bone calcium turnover. In most cases, serum PTH and alkaline phosphatase levels

*p < 0.01 compared with the data before therapy, analyzed by Wilcoxon’s test for paired data.

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venous administration in suppressing the PTH secretion in uremic patients with secondary HPT, we found that this is not true. If high oral doses of 1,25(OH)2D3 are administered periodically and not daily (we call this pulse therapy), this method can be very effective in the treatment of secondary HPT in uremic patients (pharmacological parathyroidectomy).

started to fall significantly within 5 weeks of therapy, and a dose of 4–6 µg of 1,25(OH)2D3 was considered to be adequate to maintain these effects.

Although Slatopolsky et al. [1] reported that oral administration of 1,25(OH)2D3 was not as effective as intra-