Letter to the Editor

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Indication for Oral 1,25-Dihydroxy vitamin D3 Pulse Therapy in Patients with Renal Osteodystrophy at Hemodialysis Induction

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Dear Sir,

Most cases in which 1,25-dihydroxyvitamin D3 [1,25(OH)2D3] pulse therapy was performed seem to have severe secondary hyperparathyroidism [1, 2]. The set-point between Ca and parathyroid hormone (PTH) is thought to be already abnormal at the end stage of renal failure and the number of receptors of parathyroid glands to 1,25 (OH)2D3 is also already decreased [3-6]. It is not thought to be effective to administer conventional doses of 1,25(OH)2D3 to patients in this condition. Therefore, many patients develop secondary hyperparathyroidism instead of receiving the effect of 1,25(OH)2D3 therapy.

Figure 1 shows a case that had a good serum PTH level after receiving 1,25(OH)2D3 by conventional therapy following hypercalcemia due to its pulse therapy. We consider hypercalcemia as a sign of normalization of the set-point and a reduction of hungry-bone status. At this point, alkaline leukozyte phos-phatase (ALP) isoenzyme is also normalized. We noted several cases which had moderately elevated serum PTH levels (C-PTH: 7-12 ng/ml, normal range: less than 0.5 ng/ml).

We would like to propose a protocol for administering 1,25(OH)2D3. At first the set-point should be normalized by oral 1,25(OH)2D3 pulse therapy from induction of hemodialysis while testing for hypercalcemia. When hypercalcemia appears and ALP isoenzyme is normalized (%ALP2 > %ALP3), the set-point is considered to change for the better, and conventional therapy should

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1,25(OH)2D3 2.5 µg x 2/week C = 1,25(OH)2D3 2.0 µg x 2/week

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1991/1 2 3 4 5 6 7

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Fig. 1. A case (male, 39 years old, hemodialysis duration 76 months) that had a good serum PTH level after receiving 1,25(OH)2D3 by conventional therapy following hypercalcemia after oral pulse therapy.

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be decreased. As the result, phosphate bind-

References


Hyodo/Kumano/Mori/Shio/Yoshino/ Sakai