Letter to the Editor

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Plasma 25(OH)D Levels in Children on Long-Term Hemofiltration

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Alberto Bettinelli, MD, Clinica Pediatrica De Marchi, Dialisi Infantile, Via Commenda 9, I-20122 Milano (Italy) Dear Sir,

The available studies on plasma levels of 25(OH)D during hemofiltration treatment show quite different results. Recently Sebert et al. [1] noted that, in adult patients on hemofiltration, the plasma concentrations of 25(OH)D are slightly lower than in hemodialyzed subjects. Schneider et al. [2] found that the plasma levels of 25(OH)D decreased during hemofiltration treatment. However, in a study on 5 patients by Schaefer et al. [3], the levels of 25(OH)D remained unchanged during hemofiltration. In an acute observation that we made on 6 children (aged 6–14 years) on periodic hemofiltration, the plasma levels of 25(OH)D did not change after a hemofiltration session [4]. There were only small losses in the ultrafiltrate.

To better clarify this aspect we evaluated plasma levels of 25(OH)D twice, with a 12-month interval, in 8 children (aged 6–12 years) on periodic hemofiltration. Both measurements were done in winter, in order to avoid the proven seasonal fluctuations [5]. Plasma 25(OH) levels were determined by a radiocompetition method (protein binding) (normal range 20–60 ng/ml).

The age of our patients, the mode and duration of substitution treatment and plasma 25(OH)D levels are shown in table I. Five children had been on hemofiltration treatment for 26–46 months (28.4 ± 16.7). The remaining 3 children were on hemodialysis at the time of the first determination, but after 1–3 months were put on hemofiltration. Three of the children (patients 1–3) were completely anuric, whereas the other 5 had a poor residual renal function at the first determination ($3.2 \pm 2.6 \text{ ml/min/1.73 m2}$). These 5 children showed a decrease of their already low creatinine clearance during the study ($1.7 \pm 2.8 \text{ ml/min/1.73 m2}$). No child received 25(OH)D therapy, but all were taking oral 1,25(OH)2 D3 at the dose of 0.25–0.50 µg/day. Only 1 child (patient 5) had hepatic dysfunction. None of them required anticonvulsant therapy. In all the children, except patient 5, 25(OH)D levels decreased after 1 year of hemofiltration. We cannot explain the increase of plasma 25(OH)D in the patient with

 T_0^{\bullet} = First observation; I_1^{\bullet} = second observation (after 1 year); HF = hemofiltration; HD = hemodialysis. 328

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hepatic dysfunction. In the other children it seems that long-term hemofiltration per se was the only reasonable cause of a fall in 25(OH)D. In some studies [2,3] as well as in our previous acute study [4], only trace amounts of 25(OH)D were found in the ultrafiltrate. Therefore, a possible explanation of the decrease in plasma 25(OH)D was low but continuous losses of 25(OH)D in the ultrafiltrate. In the children who showed a reduction in residual renal creatinine clearance we cannot exclude that the further although small decrease in residual parenchymal renal function may have at least partly influenced vitamin D metabolism, but this change would affect 1,25(OH)2 D3 and not 25(OH)D levels.

Therefore, in agreement with Sebert et al. [1], we believe that 25(OH)D supplementation is advisable during hemofiltration, especially in children, in order to avoid vitamin D depletion. References

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