Plasma Zinc Decreases in Hemodialysis Patients Treated with Calcium Carbonate as the Phosphate Binder

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Dear Sir,

Baseline plasma zinc levels were measured by flame atomic absorption spectroscopy in 45 uncomplicated hemodialysis patients (20 males, 25 females; age range 23–67 years, mean 51.1). Peripheral venous blood for analysis was collected in the morning, after overnight fasting, at the start of the first weekly dialysis session. The method of analysis has been described in detail elsewhere [1]. Using this method, plasma zinc was detected in 40 normal adults at a mean $\pm$ SD concentration of $112.0 \pm 17.1 \mu\text{g/dl}$ [2]. The patients were maintained on long-term hemodialysis, 12–15 h per week, for 12–130 months (mean 59.1) using either cellulosic or noncellulosic membranes. The concentration of calcium in the dialysate was 3.5 mEq/l. All the patients were on an unrestricted diet and were taking aluminum hydroxide (2–5 g/day) to achieve a satisfactory control of serum phosphorus concentration. None of them received oral calcium supplements; some were on calcitriol therapy for varying periods of time. After the initial investigation was completed, the patients were randomly allocated either to continue with aluminum hydroxide at the same dosage (group 1, n = 21) or to replace it with corresponding doses of calcium carbonate (group 2, n = 24). Following 6 months of treatment, plasma zinc was checked again. Schedule of dialysis was not changed throughout.

Results are given in table 1. As shown, the mean plasma zinc concentration decreased significantly in the patients treated with calcium carbonate, although the behavior was not unequivocal (16 decrease, 2 no change, 6 increase). In contrast, no significant change was noted in those patients who were continuing to take aluminum hydroxide as the phosphate binder. Although data are not reported, there were no statistically significant differences in the mean serum calcium and phosphorus concentrations of the two groups at any time of the study. Table 1. Behavior of plasma zinc ($\mu\text{g/dl}$) in hemodialysis patients treated for 6 months with either aluminum hydroxide (group 1) or calcium carbonate (group 2) as the phosphate binder

Comment. In the presence of calcium in the intestine, trace metals form mixed salt precipitates with phytate and calcium itself [3] which are excreted by the fecal route. This observation provides a plausible explanation by which an increase in dietary calcium could reduce zinc availability. Although the plasma zinc concentration could not be an accurate indicator of the whole-body zinc status [4], our findings suggest that zinc depletion must be considered as a possible adverse effect of calcium carbonate therapy in hemodialysis patients. Since zinc

deficiency has been linked to a variety of uremic symptoms [5], this observation is, we believe, worthy of note.

Alternatively, it is possible that the calcium load decreases zinc binding to albumin and prealbumin in plasma. Albumin and prealbumin indeed are the major binding plasma proteins for the loosely bound fraction of both calcium and zinc [6]; hence, competition between these ions for plasma protein binding might be anticipated.

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References

Gilli P, Baricordi O, Fagioli F, et al: Plasma zinc concentrations in patients with different degrees of chronic renal failure: Possible role in lymphocyte function. *Trace Elements Med* 1984; 9:153–158.

Gilli P, Fagioli F, De Paoli E, et al: Is zinc status a problem in the dietary treatment of chronic renal failure? *Nephron* 1985; 40:382.

Editorial: Phytic acid: New doors open for a chelator. *Lancet* 1987;ii: 664–666.

Agget PJ: Zinc metabolism in chronic renal insufficiency with or without dialysis treatment. *Contr Nephrol* 1984;38:95–102.

Zumkely H, Zidek W, Bertram HP: Zinc substitution in renal insufficiency. *Trace Elements Med* 1984;1:43–46.

Giroux EL: Determination of zinc distribution between albumin and OC-2-macroglobulin. *Biochem Med* 1975;12:258–266.