Is Zinc Status a Problem in the Dietary Treatment of Chronic Renal Failure?

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Dear Sir,

It is now generally accepted that dietary manipulations, such as reductions in protein and/or phosphate intake, can attenuate the progression of chronic renal insufficiency [1]. Thus, it is clear that more information is required to determine safe limits for protein restriction so as to avoid malnutrition.

A particular problem could be represented by zinc deficiency, a common abnormality in protein-energy malnutrition [2,3], which is also described in renal failure [3–5]. Zinc is an essential trace element, whose dietary availability is largely associated with animal protein intake. Its deficiency has been linked to many symptoms: skin lesions, reduced sexual function, taste and smell dysfunction, abnormal dark adaptation, impaired T-lym-phocyte function [4].

To evaluate zinc behavior in patients with renal failure, plasma zinc concentrations were determined by flame atomic spectroscopy in 46 patients (27 men, 19 women), aged 37–69 years (mean 50.9 ± 7.7), with different degrees of chronic renal failure (serum creatinine concentration from 1.6 to 10 mg%).

Plasma zinc levels in patients with chronic renal failure (mean value $100.6 \pm 20.5 \mu\text{g}\%$) were significantly different ($p < 0.001$) from those found in 63 hemodialysis patients ($82.5 \pm 14.6 \mu\text{g}\%$) and in 40 normal subjects living in the study area ($112.0 \pm 17.1 \mu\text{g}\%$).

In the patients with different degrees of chronic renal failure, a statistically significant negative relationship was found between plasma zinc and serum creatinine concentrations (fig. 1). These data confirm that plasma zinc levels are reduced in patients with chronic renal failure and suggest that zinc deficiency may be related to the degree of renal insufficiency. Many factors could contribute to the decrease of plasma zinc levels in these patients. Not only does the use of aluminum hydroxide or cation exchange resins or supplements of inorganic ions [3] as well as the diminished enteral zinc absorption due to marked small bowel mucous membrane alterations [4] cause a decrease in plasma zinc levels, but the reduction in protein intake prescribed for renal failure patients does so as well.

$$y = 123.3 - 4.5 (x) \quad r = 0.54 \quad p < 0.001$$

$\mu\text{g}\%$

150²⁷ 140–130 120–

110
 100 90 80 70–60–50–
 -1 1 1
 8 9 10 mg %
 -1 1 1 1 γ
 2 3 4 5 6

Fig. 1. Relationship between serum creatinine (x) and plasma zinc level (y) in 46 patients with different degrees of chronic renal failure.

Although the plasma zinc levels could not be an accurate indicator of whole-body zinc status [3, 5], one can conclude that in patients with chronic renal failure possible zinc deficiency must be considered as a harmful effect of dietary protein restriction.

References

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