Zinc Deficiency and Haem Synthesis: An Additional Problem in Uraemia

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Dear Sir,

In a recent letter by Gilli et al. [1] it is emphasized that plasma zinc levels are decreased in uraemic patients and the possible clinical significance of this finding is discussed.

Zinc has also an important role in the haem biosynthesis. It is a necessary activator [2] of δ -aminolaevulinic acid (ALA) dehydrase, the enzyme involved in the condensation of two molecules of ALA to form porphobilinogen. A deficiency of this enzyme – which occurs in lead intoxication [3] and in some rare cases of acute intermittent porphyria [4] – leads to accumulation of ALA. ALA is a neurotropic agent and is believed to be responsible for the neurologic manifestations of the above conditions and, in general, of the acute porphyrias. We have found a considerable decrease of erythrocyte ALA dehydrase activity in non-dialysed and, even more, in dialysed uraemic patients as well as an increase in serum ALA concentration [Yalouris et al., submitted to Scand. J. Haematol.]. Similar findings have also been reported by other authors [5].

The decrease of ALA dehydrase activity has been attributed by Goubeaud et al. [6] and by us to retained toxic factors. Is zinc deficiency an additional mechanism? In that case restoration of normal zinc status might improve ALA dehydrase activity and possibly decrease serum ALA levels. Could that be of any benefit to the anaemia or the neurologic manifestations (to which ALA

possibly contributes) of uraemia? That is a question that has to be answered in the future.

References

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