Primary Hypomagnesemia with a Probable Double Magnesium Transport Defect

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Dear Sir,

Primary hypomagnesemia is a rare genetically determined condition due to a transport defect of gastrointestinal reabsorption. This disorder had recently been reviewed by Abdulrazzaq et al. [1]. The kidneys in these cases are able to reabsorb magnesium normally.

Isolated magnesium loss with an autosomal or recessive mode of inheritance [2,3] is also rare. A defect in the intestinal absorption of magnesium could be excluded in these patients. A simultaneous transport defect of the intestinal and the renal tubule would be important for the elucidation in the future of the pathogenesis of these disorders.

The data presented by Matzkin et al. [4], however, do not allow the conclusion of a double magnesium transport defect. The urinary excretion of magnesium was distinctly too high. As concerns serum magnesium, there is an impairment of tubular reabsorption of magnesium. The hypothesis of an intestinal reabsorption defect is based on the fact that large quantities of magnesium salts are required to maintain a low normal serum magnesium level. This is exactly what has to be expected when the tubular reabsorption is disturbed. In our patients [2,3] we were unable to normalize the serum magnesium by oral supplementation of magnesium. In a situation of a tubular reabsorption defect of magnesium, a magnesium loading test with 'cold' magnesium is worthless. In order

to solve the problem, an oral ²⁸Mg resorption test should be applied in this patient. Based on the absence of other reports of simultaneous occurrence of an intestinal and tubular defect of magnesium absorption, we expect the intestinal resorption to be increased [2].

References

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