Re: The Use of Magnesium-Containing Phosphate Binders in Patients with End-Stage Renal Disease on Maintenance Haemodialysis

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Sir,

Guillot et al. [Nephron 30: 114–117, 1982] propose to use Mg-containing phosphate binders for patients on maintenance dialysis after having performed a short-term study which showed good control of serum phosphorus concentrations. In contrast to the USA, most phosphate binders available in Switzerland up to about 1980 contained Mg salts. Thus, the majority of the Basle as well as the Swiss dialysis population was receiving Mg-containing phosphate binders up to 1980. At this time we became aware of the possibility that Mg, a potent inhibitor of biological apatite crystal formation [1], could cause osteomalacia. Indeed, many of our long-standing dialysis patients had developed progressive bone disease with multiple rib and other fractures.

Histologically, wide unmineralised osteoid seams were prominent whilst signs of fibroosteoclasia were scarce or absent. Treatment with dihydrotachysterol or vitamin D caused hypercalcaemia but failed to improve the bony lesions. Aluminium in the dialysate could not be the culprit as aluminium is not used in water preparation in most parts of Switzerland and dialysate aluminium levels have always been far below the concentrations which are known to cause dialysis encephalopathy and fracturing bone disease.

In order to correct Mg overload in our patients, we substituted pure aluminium hydroxide for the previously used Mg-containing phosphate binders and entirely removed Mg (hitherto 0.5–0.7 mmol/l) from the dialysate for 6 months. Thereafter the bath Mg concentration has been kept at 0.25 mmol/l as recommended by Parsons and Davison [2]. All our patients now have Mg levels which rarely exceed the accepted normal range. The impact on bone disease was striking. Old osteomalacic fractures have healed in all our patients and complaints about bone pain have diminished rapidly. The only new fracture occurred in the femoral neck of a patient who had had the worst incapacitating bone disease with fractures all over her body and who -after years of barely having been able to walk without or even with the aid of crutches – recovered to the extent of regularly walking for hours upon correction of hyper-magnesaemia. The change to Mg-free phosphate binders caused few problems with constipation which were easily solved with bran or by substituting calcium lactate gluco-nate for calcium carbonate as supplemental calcium.

In view of our experience we should like to warn against the long-term use of Mg-containing phosphate binders. However, hyperphosphataemia with predominant secondary hyperparathyroidism might be easier to control with Mg administration which then should be combined with dialysis against a bath fluid devoid of Mg.

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