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Response to the comment on "Magnesium and chronic kidney disease"

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To the Editor:

The authors reply to our publication on magnesium and chronic kidney disease[1] and provide data on their preliminary experience with 10 peritoneal dialysis patients who received calcium acetate/magnesium carbonate (Osvaren®). The median treatment dose during follow-up was 2 pills per day (range 1-3). In two patients, it was necessary to reintroduce another phosphate binder at low doses. During follow-up, they only found one high magnesium value (1.8mmol/l) and none for calcium higher than 10.5mg/dl.

Firstly, it is necessary to highlight that the most important factor in serum magnesium concentrations is the concentration in dialysate, which the authors did not report. In our experience in peritoneal dialysis the mean magnesium concentrations with dialysate of 0.25mmol/l and of 0.50mmo/l were 2.04±0.3mg/dl (n: 17 patients) and 2.35±0.3mg/dl (n: 56 patients), respectively.[2] We should bear in mind that, up to concentrations of <4.88mg/dl (<2.0mmol/l), hypermagnesaemia is clinically irrelevant and is associated with beneficial effects.[1] The question that we must answer is whether in peritoneal dialysis patients who receive calcium acetate/magnesium carbonate (Osvaren®) these serum magnesium values increase above these figures. In our experience with 12 peritoneal dialysis patients (11 with dialysate of 0.50mmol/l and one with 0.25mmol/l) treated exclusively with calcium acetate/magnesium carbonate (Osvaren®) for 6 months, the mean serum magnesium values increased from 2.38±0.33 to 2.63±0.64, with the highest value reached in a patient being 3.5mg/dl (Table 1).

In the Calmag study[3] on haemodialysis patients, the number of calcium acetate/magnesium carbonate pills (Osvaren®) required was 7.29±3.026/day, and as such, the dose the authors referred to in their letter (2 pills per day with a range from 1 to 3) is rather low, which may explain why in two patients, it was necessary to reintroduce another phosphate binder. In our haemodialysis studies over six months in real clinical practice (n: 52 patients), the mean CaMg dose required for the reduction of phosphorus (from 6.43±1.93 at baseline to 4.83±1.98mg/dl after six months) was 4.66±1.52 pills without the requirement for any other phosphate binder.[4]

Therefore, we agree with the authors of this letter that calcium acetate/magnesium carbonate (Osvaren®) has an important role as the first step of treatment in peritoneal dialysis and that it is important to carry out more comprehensive studies in peritoneal dialysis patients.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Table 1. Biochemical parameters in peritoneal dialysis patients treated with Ca-Mg

	Baseline	3M	6M
Mg	2.38±0.33	2.62±0.46	2.63±0.64
Ca	8.83±0.65	9.11±0.53	9.33±0.79
P	8.46±1.91	6.63±1.25	5.78±1.29
PTH	343±296	258±264	205±218

3M: 3 months; 6M: 6 months; Ca: calcium; Mg: magnesio; P: phosphorus; PTH: parathyroid hormone.

4. de Francisco A, et al. Effect of calcium acetate/magnesium carbonate in the treatment of hyperphosphatemia in dialysis patients in real clinical practice. One year follow up [Abstract].

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Comment on "Dehydration upon admission is a risk factor for incomplete recovery of renal function in children with haemolytic uremic syndrome"

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To the Editor:

We read with great interest the article by Ojeda et al. that was recently published in *Nefrología*.[1] In this study, the authors identified the presence of dehydration prior to admission as a risk factor for incomplete recovery of renal function during a follow-up on children with haemolytic uraemic syndrome associated with diarrhoea (D + HUS).

We previously reported that the presence of dehydration at diagnosis in children with D + HUS increases the likelihood that they will require dialysis in the acute stage. Additionally, we observed that the aforementioned patients had a significantly longer oligoanuric and dialysis phase than patients with normal hydration; nevertheless, our study focussed exclusively on the acute phase of the disease.[2] By contrast, the present study aimed to identify whether certain clinical variables prior to hospitalisation allowed long-term renal involvement to be predicted. Although the authors found the only predictor of non-recovery of renal function to be the presence of dehydration at diagnosis, in the study discussion they maintained that their patients with long-term renal injury had a "more severe degree of acute deterioration" than patients without long-term renal involvement. Bearing in mind that the best predictor of non-recovery in patients with D+ HUS is the length of the oligoanuric period,[3] we believe that reporting the duration of the latter in groups with and without long-term renal involvement, along with the statistical significance of this comparison, would allow for a more comprehensive explanation of the study. Likewise, the authors reported that patients with incomplete recovery of renal function required dialysis more often (12 out of 13 patients; 92.3%) than those with recovery (9 out of 23 patients; 39.1%), but the difference was not statistically significant (P=.2052). We were struck by the percentage difference between both groups (92.3% compared with 39.1%) and we re-calculated this comparison and obtained a P value of .0039 (Fisher's exact test) with an odds ratio of 18.66, 95% confidence interval (2.05-169.34), which would mean, if our estimation is correct, that patients with long-term renal involvement required dialysis significantly more often. As a result, in the event that both the oligoanuric period and the requirement for dialysis were significantly greater in patients with long-term renal damage, we must ask the question whether the real predictor of non-recovery is initial dehydration or more severe renal dysfunction in the acute phase (possibly aggravated by concomitant volume depletion in dehydrated patients).

From our point of view, although not necessarily correct, dehydration determines greater renal injury in the acute phase and its severity, which is deduced by the dialysis requirement and the duration of the oligoanuric period, ultimately increases the risk of long-term renal involvement. We do not believe it is sufficient to associate dehydration on admission with the development of long-term renal damage without considering the acute phase of the disease. Nevertheless, we agree with the authors that, in order to obtain definitive conclusions in this regard, prospective studies should be conducted on a higher number of patients; in the meantime, we would like to highlight the importance of preventing volume depletion in patients at risk of developing D+ HUS.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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