Optimal dialysate calcium and vascular calcification

Sir,

Yamada et al. [1] report that the rate of progression of aortic calcification is related to the increase of serum calcium during the haemodialysis session (ΔCa), using a stepwise multivariate regression analysis. The only other serum parameter that exhibited an association with the progression of aortic calcification was CRP. The authors hypothesized that the excess calcium transferred into patients on intermittent haemodialysis treatment when using a dialysate calcium concentration of 3.0 mEq/l was responsible for this association.

We would like to ask why the authors did not correct for post-dialysis haemoconcentration, when calculating ΔCa? They could have proceeded to this correction easily, since they measured serum albumin and presumably also blood haemoglobin, although this is not indicated in the article.

We respectfully question the hypothesis that the peridialytic ΔCa is the major culprit. An alternative hypothesis is that higher serum calcium levels simply reflect higher haemoconcentration at the end of the dialysis session, and that this in turn is the expression of a greater removal of sodium and water in patients with larger interdialytic body weight gain. Although the authors failed to observe a correlation between ΔCa and ultrafiltration rate in univariate analysis (Table 2), there was a correlation between these two parameters in step-wise multivariate regression analysis (Table 3). Our hypothesis is supported by the latter positive association and also by the negative association of ΔCa with the serum calcium concentration before the dialysis session.

We therefore believe, in disagreement with the 2003 K/DOQI guidelines [2], that the dialysate calcium concentration should generally not be lowered to <3.0 mEq/l, unless patients are receiving high doses of active vitamin D derivatives and/or calcium-containing phosphate binders. Low calcium concentrations in the dialysis fluid may be hazardous in chronic haemodialysis patients with cardiovascular disease, who are hypotension-prone [3] and in those who are receiving calcimetics. The latter have the capacity to lower serum calcium [4] and to delay the progression of vascular calcification [5,6].

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Reply

Sir,

We thank Dr Joki for his comments on our recent paper [1]. As he summarizes, we consider the excess calcium transferred into patients from the dialysate as one of the causes of vascular calcification in maintenance haemodialysis (HD) patients. However, we need further studies to determine whether using a dialysate with the lower calcium concentration (for examples, 2.5 mEq/l of dialysate calcium) might reduce the acute changes of serum Ca associated with HD and prevent the progression of vascular calcification.

As Dr Joki points out, changes in serum Ca should be expressed as changes in ionized or albumin-corrected Ca because dialysis ultrafiltration induces haemoconcentration and increases albumin levels. Unfortunately, we did not preserve blood samples used in this study, and therefore we cannot measure serum ionized Ca or albumin-corrected Ca just after HD session. However, we revised the value of albumin just after HD (corrected post-HD Alb) by using a concentration rate of dialysis ultrafiltration and calculated albumin-corrected Ca just after HD (corrected post-HD Ca).

Using these corrected pre-HD and post-HD Ca values, we again performed a step-wise multivariate regression analysis and confirmed that the result was nearly the same as the previous one, analysed by non-corrected Ca values.

In our subjects, step-wise multiple regression analysis has revealed that acute changes of the serum calcium concentration before and after HD (ΔCa) was negatively and positively associated with pre-HD Ca and ultrafiltration, respectively. Therefore, we agree with Dr Joki that a low pre-HD serum Ca and an excessive ultrafiltration should be avoided in HD, in order to prevent the loading of calcium.