Dialysate calcium use in hemodialysis patients

To the Editor: A recent *Kidney International* article [1] demonstrates an impressive decrease in cardiac calcification with the use of sevelamer as phosphate binder when compared to calcium-based binders. However, there is a striking omission in the data. Dialysis calcium is not presented at all. Dialysate calcium doubtlessly has a strong impact on most, if not all, of the study outcomes, including serum calcium, phosphorus, and parathyroid hormone (PTH) [2], and most probably, soft tissue calcification. Dialysate calcium is an important variable that should have been measured and presented to aid in the interpretation of the study.

Another omission in the data is the mean PTH level. The median of PTH levels that is given is an extremely limited description of this important parameter in the study. Why was the mean ± SD not presented?

The omission of easy-to-obtain, pertinent variables, obscures the clinical implications of this study.

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REFERENCES


RENAL DISEASE MEDICATIONS

Renal disease medications and evidence-biased medicine

To the Editor: In a recent article in *Kidney International* [1] I read about a comparison between sevelamer and calcium-containing substances. Although the paper seemed quite scientific, I feel the need to state some objections. The paper compares three different medications in hemodialysis patients but considers two of them to be the same: calcium carbonate and calcium acetate. The truth is that these two calcium-containing substances are quite different, both in their effectiveness and their side effects. The mixed data this paper presents from the patients in the United States and Europe who used calcium acetate and calcium carbonate, respectively, is biased. In many European countries calcium carbonate is the only calcium-containing phosphate binder that exists. In the United States, calcium acetate has been used for several years, in many cases superseding calcium carbonate. The trouble with this paper is that it does not say how many patients used calcium carbonate and how many used calcium acetate. It only says that half the

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**Table 1.** Adjusted odds ratios for cardiovascular diseases associated with an increase of one quintile in plasma sFas and CRP

<table>
<thead>
<tr>
<th>Markers</th>
<th>Odds ratio (95% CI)*</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>sFas</td>
<td>1.74 (1.21–2.89)</td>
<td>0.01</td>
</tr>
<tr>
<td>CRP</td>
<td>2.08 (1.11–3.24)</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Abbreviations are: CRP, C-reactive protein; sFas, soluble Fas.

*Odds ratios are adjusted for the following additional risk factors: age, sex, traditional risk factors for atherosclerosis (obesity, hypertension, hyperlipidemia, smoking, and diabetes), serum albumin level, nutritional status (nPCR), and dialysis adequacy (eKt/V).

mL/min) were enrolled. Clinical data included duration of ESRD, traditional risk factors for CV disease (CVD), normalized protein catabolic rate (nPCR) in g/kg/d, and dialysis adequacy (eKt/V). Cardiovascular morbidity and mortality criteria were myocardial infarction, angina pectoris, coronary artery revascularization, a positive stress test, or cardiac imaging procedure. Hemodialysis and non-HD ESRD patients had evidence of CVD compared with control patients (P = 0.02) with 11%, 6%, and 0% CV mortality, respectively (P = 0.01). Levels of sFas were significantly higher in HD patients compared with non-HD ESRD patients (P = 0.02) and control patients (P = 0.01). After adjustment for traditional CV risk factors, sFas and C-reactive protein (CRP) remained independent markers of CVD (Table 1). The correlation between sFas and CRP was R² = 0.67 (P = 0.005). These results suggest that sFas may represent a novel and independent predictor of CVD morbidity and mortality in ESRD patients. The addition of sFas to the inflammatory factor CRP allows better determination of CVD in ESRD patients.