with their normal work. After 3 months, these percentages changed to 58.5 and 81.8% in the cognitive-behavioral therapy (CBT) and control groups, respectively.

The relationship between pain and depression in end-stage renal disease patients is important. The impact of CBT in alleviating body pain in these patients requires future research.

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Priscila Silveira Duarte[1] and Ricardo Sesso[1]
[1]*Division of Nephrology, Federal University of São Paulo, Rua Botucatu, São Paulo, Brazil*
Correspondence: *Priscila Silveira Duarte, Division of Nephrology, Federal University of São Paulo, Rua Botucatu, 740, Vila Clementina, São Paulo 04023900, Brazil. E-mail: psduarte@nefro.epm.br*

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Sudden cardiac death and mineral metabolism in chronic kidney disease

Pun *et al*. showed that reductions in the estimated glomerular filtration rate (eGFR) were associated with an increase in the risk of sudden cardiac death in a graded fashion in patients with coronary artery disease. The authors claimed that decreased eGFR induces many metabolic and physiological changes that might be responsible for increased sudden cardiac death in chronic renal failure patients.[1] Although the study is informative, we are especially concerned about the relationship between calcium, phosphorus, Ca × P product, parathyroid hormone levels, and sudden cardiac death in the study population. It is well established that abnormalities in mineral metabolism are apparent early in the course of chronic kidney disease (CKD). Beginning in CKD stage 3, the ability of the kidneys to appropriately excrete a phosphate load is diminished, leading to hyperphosphatemia, elevated parathyroid hormone, and decreased vitamin D levels. Furthermore, there is emerging evidence linking some of these abnormalities (for example, hyperphosphatemia and hypercalcemia) to the high cardiovascular morbidity and mortality experienced by nondialyzed patients with CKD. One of the mechanisms for deranged mineral metabolism to induce cardiovascular disorders is thought to be the calcification of the vascular tree that result in arterial stiffness. Arterial stiffness of the large arteries has important clinical consequences: raised systolic blood pressure, increased pulse pressure, left ventricular hypertrophy, and reduced coronary perfusion.[2,3]

Experimental evidence showed that high levels of phosphate and/or calcium directly activated genes related to an osteoblastic phenotype in the smooth muscle cells.[4] In addition, elevated phosphorus and calcium stimulated the transformation of vascular smooth muscle cells into osteoblast-like cells *in vitro* using cell-culture techniques.[5] Besides, clinical evidence also suggests that high pre-dialysis serum phosphate is a powerful predictor of sudden cardiac death.[6]

Since the regular control of calcium, phosphorus, and parathyroid hormone in chronic renal failure patients is strongly recommended, if available, the presently informative results by Pun *et al*. would have been much more valuable with the addition of parameters of mineral metabolism in the adjusted analyses.

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Baris Afsar[1] and Rengin Elsurer[1]
[1]*Department of Nephrology, Zonguldak Atatürk State Hospital, Zonguldak, Turkey*
Correspondence: *Baris Afsar, Department of Nephrology, Zonguldak Atatürk State Hospital, Zonguldak, Turkey. E-mail: afsarbrs@yahoo.com*

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The Authors Reply: We appreciate Drs Afsar and Elsurer for their interest and comments about our study.[1] We agree that disordered mineral metabolism has been associated with cardiac risk in hemodialysis patients,[2] in those with less severe chronic kidney disease,[3] and in some patients who lack overt kidney disease.[4] We now report available laboratory data on serum calcium, phosphorus, and parathyroid hormone (PTH) concentrations obtained within 3 months prior to cardiac catheterization in the study cohort. Concurrent PTH data were unavailable on the majority of patients, but calcium and phosphorus data were available in 46% of patients with glomerular filtration rate (GFR) <15 and in 18% of patients with GFR $\geqslant 15$ (Table 1). Calcium and calcium–phosphorus product had no significant relationship with the composite outcome, but phosphorus had a significant relationship (hazard ratio 1.27, 95% confidence interval 1.04–1.55) in univariate analysis. However, this relationship was abolished after accounting for baseline GFR. Accounting for serum phosphorus did not alter the relationship between GFR and outcome in an adjusted model. Therefore, our study findings could not be explained by measured abnormalities in mineral metabolism, although the analysis was limited by missing data.