Fibroblast Growth Factor-23 Levels Are Associated with Vascular Calcifications in Peritoneal Dialysis Patients

E. Asicioglu, A. Kahveci, H. Arikan, M. Koc, S. Tuglular, C.I. Ozener

DOI:10.1159/000355859

Commentary
By Professor Richard Glassock

Fibroblast Growth Factor-23 (FGF-23), a phosphaturic hormone produced by osteocytes having important effects on calcitriol and parathyroid hormone, has burst on the scene as a potential new player in complications, including mortal events, seen in patients with CKD and ESRD treated by dialysis. The ready availability of a sensitive and specific assay for the intact FGF-23 molecule has allowed numerous cross-sectional studies describing associations between FGF-23 levels in the circulation and specific outcomes or complications. One such study is exemplified by Asicioglu and colleagues, who examined in a small cross-sectional study the relationship between FGF-23 levels and vascular calcification in patients undergoing chronic peritoneal dialysis (n = 55) and age/gender matched controls (n = 40). With multi-variate logistical analysis, FGF-23 levels, age, dialysis duration and residual renal function were all independently associated with calcification of blood vessels in the pelvis (assessed by conventional X-ray methods). Over 90% of the patients were using calcium based phosphate binders and 50% were on active Vitamin D analogues. About 56% of the patients had diabetes. Klotho levels were not measured. It was not possible to unambiguously assess the independent contribution of calcium intake. Interestingly, FGF-23 levels were lower in diabetic subjects. The design of the study precluded any conclusion regarding a possible causal effect of elevated FGF-23 levels on vascular calcification. Further studies involving subjects on non-calcium containing phosphate binders are needed to help clarify the role of FGF-23 on vascular calcification in CKD and ESRD. The lack of a safe, effective and specific inhibitor of FGF-23 impedes clinical research in this arena. Until human studies involving selective inhibition of FGF-23 become feasible, the status of FGF-23 as a maker or marker of vascular calcification in CKD and ESRD will remain unresolved.