Reviewer’s report

Title: Serum FGF-23 associated with the progression of coronary artery calcification in hemodialysis patients

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Reviewer: Marcel Roos

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In this manuscript by Ozkok et al the authors investigated the possible effects of serum FGF-23 on the progression of coronary artery calcification in HD patients. The authors were able to show that serum FGF-23 levels were related to a progression of CACS (elevated by computed) tomography independent of serum phosphorus levels. Thus they concluded that FGF-23 may play a major role in progression of vascular calcification and hypothesized that especially at early stages of calcification processes.

Although the article is well clearly written, and which is very much appreciated not long, I fear that the scientific impact of the study is poor. Unlucky a recent publication in KI “Fibroblast growth factor 23 is not associated with and does not induce arterial calcification” by Scialla JJ et al could not demonstrate an association of FGF23 with arterial calcification. Moreover the couldnot find evidence that FGF23 does promote calcification. Unlike in the study by Ozkok et al, they performed CT angiography in 1501 patients. Another study “Relationship of fibroblast growth factor 23 with left ventricle mass index and coronary calcificaton in chronic renal disease” by Unsal A could not find any statistically significant relationship between FGF23 and coronary artery calcifications.

Another weakness is the fact that authors have not paid attention to other very important markers of calcium phosphate homeostasis like fetuin-A or Klotho.

Moreover the are 6 patients in the low baseline CACS group (Figure 3a) showing extremely high CACS log values. What would happen to the statistic outcome once these patients were excluded from analysis? I also wonder why authors have chosen CACS < 30 vs > 30 as a cut off for their analysis.