CKD-MBD
Chronic Kidney Disease Mineral and Bone Disorder

CKD-MBD related publications in the ERA journals
From January to July 2022

The ERA acknowledges the high clinical and scientific relevance of the CKD-MBD syndrome, reflected by several key publications in the journals of our society. We hereby summarize the content of several recent important papers, providing a link to their abstracts.

From January to July 2022, 21 CKD-MBD related articles, including editorial comments and experimental studies, have been published; 11 in Nephrology Dialysis and Transplantation and 10 in the Clinical Kidney Journal (some were previously commented on after publication in advanced form).

1) Again, many reports dealt with **vascular calcification (VC) and/or calciphylaxis**. **N. Seyahi et al** (Clin Kidney J 15 (1): 101-108) examined coronary artery calcification (CAC) progression and long-term cardiovascular outcomes in kidney transplant (KT) recipients. They found that CAC at baseline and CAC progression robustly predicted the risk of death and cardiovascular events in these patients. **B. Van Berkel et al** (Clin Kidney J 15 (2): 295-302) aimed to define the prevalence, progression and implications of breast artery calcification (BAC) in female patients with CKD across various stages [CKD G2–5D-5T). The role of magnesium to prevent CKD–associated vascular calcification was reviewed by **A.D. ter Braake et al** (Nephrol Dial Transplant 37 (3):421-429), and experimental studies performed by **NHJ Leenders et al** (Nephrol Dial Transplant 37 (6):1049-1058) demonstrated that increased dietary magnesium only inhibited abdominal vascular calcification in an experimental animal model of CKD *in vivo*. **G. Wasilewski et al** (Nephrol Dial Transplant 37 (4):652-662) described that only the combination of high vitamin K_2 with phosphate binder treatment significantly attenuated VC in an experimental model of kidney failure with vitamin K deficiency,

E. Guillén-Olmos et al (Clin Kidney 15 (4):663-671) analyzed their local series of **calciphylaxis** in KT recipients (largest to date), emphasizing that calciphylaxis can still occur after KT, in many cases during the first year and in patients with a good renal function. An associated editorial on the epidemiology and pathogenesis of calciphylaxis in different patient populations, also discussing recent findings for its therapeutic management, was reported by **M.A. Podestà et al** (Clin Kidney J 15 (4):611-614). A successful treatment of calciphylaxis with vitamin K in a patient on haemodialysis was reported by **Z. Wajih and R. Singer** (Clin Kidney J 15 (2):354-356). **S. Sinha et al** (Clin Kidney J 15 (1):136-144) described the design of Phase 3 CALCIPHYX clinical trial which will examine the efficacy and safety of SNF472 in patients who have ulcerated calciphylaxis lesions.

2) Regarding **cardiovascular** (CV) events, **M. Epstein and M. Freundlich** (Nephrol Dial Transplant 37 (2): 211-222) reviewed the intersection between mineralcorticoid receptor activation and the FGF23-Klotho cascade, potentially promoting both renal and CV injury. **H. Kim et al** (Clin Kidney J 15 (1):119-127) reported that total hip- and femur neck-low bone mineral density was associated with coronary arterial calcification progression and incident major adverse CV events (after adjusting for total hip T-score) in patients with CKD G1-5. **D.A. Jaques et al al** (Clin Kidney J 15 (6):1188-1195) described the association between femoral neck-bone mineral density and both mortality and fracture risk in patients receiving renal replacement therapy. Smoking was also shown as a significant risk factor for any fracture requiring hospitalization in Japanese haemodialysis patients by **M. Wakasugi et al** (Nephrol Dial Transplant 37 (5):950-959). **Wu P-S et al** (Nephrol Dial Transplant 37 (6):1162-1170) showed that **osteoprotegerin** predicted CV events in haemodialysis patients, independently of cytokine activity, as opposed to other bone-associated proteins. Finally, osteocrin, another bone-derived humoral factor, was reported to exert a renoprotective role in ischemia-reperfusion injury in

an experimental mice model conducted by **Y. Nishiguchi et al** (Nephrol Dial Transplant 37 (3):444-453).

3) **P. Ureña-Torres et al** (Nephrol Dial Transplant 37 (4):613-616) commented on a report by **M. Bozic et al** (Nephrol Dial Transplant 37 (4):663-672) regarding the independent effects of **hyperphosphatemia** and **secondary hyperparathyroidism** (SHPT) on CKD progression and CV events. Clustering **phosphate** and iron-related markers and prognosis in dialysis patients were studied by **H. Morooka et al** (Clin Kidney J 15 (2): 328-337) using unsupervised machine learning methods. Authors found that the use of phosphate binders was associated with a lower risk of all-cause death in two clusters which were characterized by older age and a higher prevalence of diabetes mellitus, among other factors. The pharmacogenetic role of vitamin D-binding protein and vitamin D receptor polymorphisms in the treatment response of dialysis patients with SHPT was reviewed in a research letter by **R. de Alarcón et al** (Nephrol Dial Transplant 37 (4):792-795). **A. Iyengar et al** (Nephrol Dial Transplant 37 (2):326-334) reported that intensive treatment with oral cholecalciferol in children with CKD as daily, weekly or monthly regimens achieved similar calcidiol concentrations without toxicity. Both FGF-23 and klotho concentrations significantly increased. Children with glomerular disease required higher doses of colecalciferol. Finally, **A.S. Shankar et al** (Nephrol Dial Transplant 37 (1):190-193) described vitamin D metabolism in human kidney organoids.

4) Finally, abstracts and contents of the 59th ERA-EDTA Congress 2022 can be found in the 2022 Nephrol Dial Transplant Supplement # 3

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