From July to December 2022, 19 CKD-MBD related articles have been published, including editorial comments and experimental studies; 13 in Nephrology Dialysis and Transplantation and 6 in the Clinical Kidney Journal.

1) Cardiovascular risk and cardiovascular calcifications were the subject of several reports. M.A. Podestà et al (Nephrol Dial Transplant 37 (11):2063-2071) provide an overview of mediators involved in the pathogenesis of cardiovascular calcification in kidney transplantation, describe the clinical and radiological features, and discuss current evidence on preventive and novel strategies to potentially prevent their long-term deleterious effects. In a different population of hemodialysis (HD) patients, T. Saritas et al (Clin Kidney J 15 (12): 2300-2311) demonstrated in a small but randomized clinical trial that vitamin K1 is a potent, safe and cost-effective approach not only to correct vitamin K deficiency but also to potentially reduce progression of cardiovascular calcification in this population. In an experimental in vivo model of CKD, dietary magnesium supplementation was shown to inhibit abdominal vascular calcification in a report by N.H.J. Lenders et al (Nephrol Dial Transplant 37 (6):1049-1058). Interestingly, P.-H. Wu et al (Nephrol Dial Transplant 37 (6):1162-1170) described that osteoprotegerin was the most important bone biomarker related to cardiovascular events in a prospective cohort in Danish HD patients, independently of cytokine inflammatory activity. Associations of time-dependent changes in phosphate (P) levels with cardiovascular diseases in patients undergoing dialysis without secondary hyperparathyroidism were shown by E. Koshi-Ito et al (Clin Kidney J 15 (12): 2281-2291). Their results suggest the importance of maintaining stable P levels, not only in the normal range but also without fluctuations. K.J. Martin (Nephrol Dial Transplant 37 (10):1830-1832) reports that we should shift current P classical evaluations and that clinical decisions should be made with a method which takes into account serial P levels for potentially better clinical outcomes.

2) Regarding P and fibroblast growth factor-23 (FGF-23), J.T. Daugirdas (Nephrol Dial Transplant 37 (12):2522-2527) describes similar results when comparing measured vs kinetic-model predicted P removal during HD and hemodiafiltration (10% or greater P removal for postdilution hemodiafiltration). S.B. Ascher et al (Nephrol Dial Transplant 37 (9):1637-1646) reported that higher serum FGF-23 was individually associated with higher risk of the composite adverse-event outcome in multivariable-adjusted models, including kidney tubule health biomarkers, eGFR and albuminuria from SPRINT (Systolic Blood Pressure Intervention Trial). D. Verbeek and O.W. Moe (Nephrol Dial Transplant 37 (10):1800-1807) summarized strategies to lower FGF-23 bioactivity and addressed critical questions remaining to be answered. In an experimental rat model of CKD-MBD, A. Biruete et al (Nephrol Dial Transplant 37 (10):1857-1867) demonstrated that oral ferric citrate improved P homeostasis, some iron-related parameters and the production and cleavage of FGF23. On the other hand, F. Di Mario et al (Nephrol Dial Transplant 37 (12):2505-2513) reports that hypophosphatemia is a frequent complication in critically ill patients undergoing sustained low-efficiency dialysis (SLED) with standard dialysis solutions, that worsens with increasing SLED treatment intensity. Moreover, in patients undergoing daily SLED, phosphate supplementation is strongly associated with reduced
mortality.

3) Secondary hyperparathyroidism (SHPT) is frequent in patients with Bartter syndrome type I and II as reported by M.F.A. Verploegen et al (Nephrol Dial Transplant 37 (12):2474-2486) in an international cross-sectional study. Low serum P was observed in 22% of patients with Bartter and Gitelman syndrome and appeared to be associated with renal P wasting. Interestingly, L.D.Dubourg et al (Nephrol Dial Transplant 37 (11):2150-2156) described tubular maximum phosphate reabsorption per glomerular filtration rate (TmP/GFR) reference values from child to adulthood in the era of IDMS-standardized creatinine assays. SHPT treatment, defined as the use of vitamin D analogs, phosphate binders, calcimimetics or parathyroidectomy (PTX), was associated with a lower risk of incident dementia among older patients (age ≥66 years) with end-stage kidney disease as reported by A. Mathur et al (Nephrol Dial Transplant 37 (11):2111-2118). L. Magagnoli et al (Nephrol Dial Transplant 37 (11):2039-2041) wrote an editorial on these new perspectives of CKD-MBD beyond vessels and bones. Finally, G. Cianciolo et al (Clin Kidney J 15 (8): 1459-1474) propose a roadmap to PTX for kidney transplant candidates.

4) Regarding CKD-associated risk of fractures, D.A. Jaques et al (Clin Kidney J 15 (6): 1188-1474) described that bone mineral density (BMD) measured at the femoral neck is predictive of mortality in patients requiring renal replacement therapy, although low BMD might be a marker frailty rather than a direct causal factor. Femoral neck BMD was also a strong predictor of hip and any fracture risk, as well as an overall prognostic marker in these patients. Metabolic acidosis was associated with fractures, falls protein-calorie malnutrition and failure to thrive in a large cohort of patients with CKD G3-5 as reported by V. Mathur et al (Clin Kidney J 15 (6): 1379-1386). It was also shown an association between the cause of kidney failure and fracture incidence in a national US dialysis cohort study performed by S. Ziolkowski et al (Clin Kidney J 15 (12): 2245-2257); however, the study was limited by lack of data regarding numerous potential confounders beyond the cause of kidney failure. Perhaps unexpectedly, lupus nephritis was associated with a lower fracture hazard.

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