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CKD-MBD related publications in the ERA-EDTA journals (NDT and CKJ)

The ERA-EDTA 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 summarise the content of several recent important papers, providing a link to their abstracts.
From January to June 2018, 17 **CKD-MBD related articles**, including several editorial comments and experimental studies, have been published; 10 in Nephrology Dialysis and Transplantation and 7 in the Clinical Kidney Journal

1. Regarding **inflammation**, **C. Cafiero et al** (Nephrol Dial Transplant 33 (1):65) explored, thourgh ex-vivo culture experiments, the potentially important cross-talk between the immune and bone systems in CKD patients. They demonstrated, in late-stage CKD and HD patients, that inflammation induces osteoclast differentiation from peripheral mononuclear cells as well as increased bone resorbing activity. **D. Zickler et al** (Nephrol Dial Transplant 33 (4):574) described that inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α) or Interleukin-6 are increased in uremic sera and that TNF-α promotes osteoblastic transition and calcification of vascular smooth muscle cells via induction of interleukin-6 expression. Additionally, **L. Hénaut et al** (Nephrol Dial Transplant 33 (4):543) reviewed and underlined the key role of inflammation and interleukin-6 (even beyond TNF-α) in vascular calcification in CKD patients. **P.C. Gregório et al** (Clin Kidney J 11 (1):89) described in vitro that sevelamer reduces endothelial inflammatory response to advanced glycation end products.
2. Focusing on **vascular calcification**, **E. Melilli et al** (Clin Kidney J 11 (3):413) reviewed the impact of arterial stiffness as a prognostic marker of cardiovascular disease in renal transplantation, and the effects of different immunosuppressive regimens on its progression, considering the potential benefits of calcineurin-inhibitor-sparing protocols. **H.Inoue et al** (Nephrol Dial Transplant 33 (4):676) explained that according to a 10-year longitudinal study, over time, chronic haemodialysis patients with a trajectory of longitudinal high or rapid accumulation of vascular calcification were at a higher risk of death. On the other hand, a systematic review by **T.T. Jansz** (Clin Kidney J 11 (3):353) concluded that there is insufficient evidence to analyse the progression of coronary artery calcification and the influence of renal replacement therapy modality.
3. **A.D. Renaghan and M.H. Rosner** (Nephrol Dial Transplant 33 (4):549) reviewed the etiology and management of hypercalcaemia; and **K. Sumida et al** (Nephrol Dial Transplant 33 (2):264) explored the independent association of higher late-stage non-dialysis dependent CKD **alkaline phosphatase** levels with higher post-ESRD mortality risk.
4. Regarding **phosphate**, **J.T. Daugirdas** (Nephrol Dial Transplant 33 (1):76) described a two-pool kinetic model predicting phosphate concentrations during and shortly after a conventional (three times weekly) haemodialysis session. **K.J. Bergsland et al** (Nephrol Dial Transplant 33 (1):65) demonstrated that hypophosphataemia in calcium stone formers results from decreased tubule phosphate reabsorption and, being associated with related changes in other proximal tubule transporters, may arise from alterations in or signaling to PDZ-containing proteins [i.e. Na+/H+ exchanger regulatory factor 1 (NHERF1)].
5. Through the Photo-Graphe3 study, **D. Fouque** et al (Clin Kidney J 11 (1):73) analysed the achievement (less than 20%) of **KDIGO** mineral and bone **targets** between 2010 and 2014 in incident dialysis patients in France, however proportion did increase slightly over time. **M. Cozzolino** (Clin Kidney J 11 (1):70) wrote an editorial entitled 'CKD-MBD KDIGO guidelines: how difficult is reaching the 'target'?.
6. Regarding CKD-MBD associated treatments, **P. Westerberg et al** (Nephrol Dial Transplant 33 (3):466) published a double-blind, randomised controlled study of high doses of **cholecalciferol** (8000 IU/day) for 12 weeks in patients with CKD Stages 3–4 and demonstrated that it halts the progression of secondary hyperparathyroidism without causing hypercalcaemia or any other side effects. No effect on grip strength, fatigue, phosphate or fibroblast growth factor 23 was observed. **L. Pereira** et al (Clin Kidney J 11 (1):80) reviewed old and new **calcimimetics** for treatment of secondary hyperparathyroidism and **C.T.S. Lim et al** (Clin Kidney J 11 (2):265) described the postoperative clinical course (morbidity and mortality) after total parathyroidectomy without autoimplantation in adults on maintenance dialysis.
7. **D. Claramunt-Taberner et al** (Nephrol Dial Transplant 33 (9):1525) showed that the poorly described bone disease in nephropathic **cystinosis** is related to cystinosin-induced osteoclastic dysfunction.

The 55th ERA-EDTA Congress 2018, held in Copenhagen, can be found in the 2018 Nephrol Dial Transplant Supplement # 1.

Abstracts from *oral presentations* on Bone Disease on hemodialysis and CKD-MBD, as well as abstracts from *posters* on Dialysis: Bone Disease, can be found by clicking the hyperlinks.

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