Clinical Kidney Journal, 2025, vol. 18, Suppl 1, i1–i2

https://doi.org/10.1093/ckj/sfae432
Editorial Comment

EDITORIAL COMMENT

New perspectives on chronic kidney disease-mineral bone disorder

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The 2017 Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease-Mineral Bone Disorder (CKD-MBD) was a selective update of the prior CKD-MBD Guideline published in 2009 [1, 2]. While most of the 2017 recommendations appeared consistent at the KDIGO Controversies Conference on CKD-MBD held in Madrid (October 2023) [3], the classical framework of three major sections (laboratory abnormalities, bone abnormalities and vascular calcification) may perhaps no longer adequately serve current clinical needs for personalized CKD-MBD care. Meanwhile, in this *Clinical Kidney Journal* supplement, Salera *et al.* [4] review the current understanding of CKD-MBD as a consequence of declining kidney function, highlighting that compensatory mechanisms involving fibroblast growth factor-23, parathyroid hormone (PTH), klotho, calciproteins, microparticles and other factors become insufficient to maintain mineral homeostasis. This inability leads to CKD-MBD complications that extend beyond bone health, such as cardiovascular disease and vascular calcification. The authors acknowledge significant variability in the clinical phenotypic manifestations among CKD patients and review genetic, environmental factors and/or the interaction with comorbidities (such as diabetes or hypertension) that may influence the final expression and treatment of CKD-MBD. Specifically, Mazzaferro *et al.* [5] review not only the classical and most recent developments in the pathophysiology of CKD-associated secondary hyperparathyroidism but also its clinical and diagnostic features, current management strategies (targeting calcium, phosphate, PTH, vitamin D and/or the calcium-sensing receptor) and potential future therapeutic targets. In terms of vascular calcification, Hénaut *et al.* [6] discuss current knowledge of the mechanisms why which CKD and its related therapies may specifically and independently affect valvular cell activity. They also highlight the latest therapeutic targets identified in preclinical studies and clinical trials aiming at preventing the development and/or the progression of valvular calcification in CKD patients. Rodelo-Haad *et al.* [7] provide an overview of the current understanding of phosphate handling by the kidney in CKD. They emphasize the detrimental effects and mechanisms of phosphate-mediated damage on various organs, including CKD progression, and discuss tools available to identify patients at risk of an excessive phosphate load even before the clinical onset of hyperphosphatemia. Finally, Magagnoli *et al.* [8] analyse the natural sources and metabolism of vitamin D, as well as the main available pharmacological vitamin D compounds. They argue that this diversity could potentially allow for tailored and personalized approaches to CKD-MBD management. However, the authors acknowledge the ongoing debate regarding the impact of these compounds on various clinical outcomes due to the lack of direct comparative data.

In summary, the articles in this *Clinical Kidney Journal* supplement underscore the evolving understanding of CKD-MBD and emphasize the need for a more personalized approach to its management. Advances in the knowledge of the underlying mechanisms highlight the complexity and heterogeneity of CKD-MBD among patients. By integrating these insights, researchers and clinicians will be better equipped to develop individualized therapeutic approaches. Furthermore, the ongoing exploration of novel treatment strategies holds the promise of refining care which should improve patient outcomes. As personalized medicine continues to grow, the key challenge will be translating these developments into practical, evidence-based

Received: 19.12.2024; Editorial decision: 19.12.2024

recommendations that address the diverse clinical needs of CKD patients worldwide.

FUNDING

This paper was published as part of a supplement financially supported by an educational grant from CSL Vifor.

CONFLICT OF INTEREST STATEMENT

J.B. received advisory and/or lecture fees and/or congress travel expenses from Abbvie, Amgen, AstraZeneca, Bayer, CSL Vifor, GSK, Menarini, Rubió, Sanofi and Theramex.

M.C. received advisory and/or lecture fees and/or congress travel expenses from Abbvie, Amgen, AstraZeneca, Bayer, CSL Vifor, Baxter and Boehringer-Lilly.

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Received: 19.12.2024; Editorial decision: 19.12.2024