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Bone and Cardiovascular Crosstalk in CKD

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Chronic kidney disease (CKD) mineral and bone disorder (MBD) is comprised of a triad of biochemical abnormalities (of calcium, phosphate, parathyroid hormone and vitamin D), bone abnormalities (turnover, mineralization, volume and growth) and extra-skeletal calcification. Mineral dysregulation leads to bone demineralization causing bone pain and an increased fracture risk compared to healthy peers. Vascular calcification, with hydroxyapatite deposition and arteriosclerosis is a part of the CKD-MBD spectrum and in turn causes vascular stiffness, left ventricular hypertrophy and a very high cardiovascular mortality risk. While the growing bone needs calcium, excess calcium can deposit in the vessels, such that the intake of calcium and calcium containing medications but be carefully regulated.

Normal physiological bone mineralization continues into the third decade of life, many years beyond the rapid growth associated with adolescence, implying that skeletal calcium requirements are very different at these ages. Much of the research into the link between bone (de)mineralization and vascular calcification in CKD has been performed in older adults and these data cannot be extrapolated to children or younger adults.

In this talk, I will explore the physiological changes in bone turnover and mineralization in childhood and adolescence, the pathophysiology of mineral bone disease in CKD and a potential link between bone demineralization and vascular calcification. Also, the role of non-radioactive calcium isotopes as a novel biomarker of bone mineralisation will be discussed.