

April 27(Thu) - 30(Sun), 2023 Coex, Seoul, Korea

Submission No.: PG05-9191 Session Title: PG Education 5 (CKD) Date & Time, Place: April 27 (Thu), 13:00 - 14:30, Room 1+2

Current Updates in Diagnosis and Treatment of CKD-MBD

Shin Young Ahn Korea University Guro Hospital, Korea, Republic of

Chronic kidney disease mineral and bone disease (CKD-MBD) is a prevalent complication of CKD that occurs early on and is linked to significant morbidity and mortality. CKD-MBD encompasses abnormalities in mineral metabolism, bone health, and soft tissue calcification. These abnormalities may include disrupted calcium (Ca), phosphate (P), parathyroid hormone (PTH), or vitamin D metabolism, as well as abnormal bone turnover, mineralization, volume, or strength. Moreover, additional laboratory abnormalities, such as alkaline phosphatase (AP), fibroblast growth factor 23 (FGF-23), and klotho levels have been suggested to augment the original definition of CKD-MBD. Various clinical symptoms such as bone pain and fractures accompany abnormal bone metabolism disorders in CKD-MBD. As a result, it is crucial to anticipate the likelihood of fractures, and this can be accomplished through bone biopsies or bone mineral density (BMD) measurements. Dual-energy X-ray absorptiometry (DXA), quantitative computed tomography (QCT), and high-resolution peripheral quantitative computed tomography (HR-pQCT) are methods for quantitatively measuring bone mineral density.

To manage CKD-MBD, the primary focus should be on correcting or minimizing biochemical abnormalities, such as vitamin D deficiency, hyperphosphatemia, hypocalcemia, and hyperparathyroidism. Controlling hyperphosphatemia is essential, which can be achieved through dietary restrictions. Phosphorus can be further managed through dialysis treatment and the use of drugs. Recently, iron-based phosphate binders have shown promising results in reducing phosphorous levels and improving anemia, comparable to other phosphate binders. The recommended approach for managing calcium in CKD patients is to prevent calcium overload. As per the KDIGO guidelines, correcting hypovitaminosis D through nutritional vitamin D replacement is recommended to prevent secondary hyperparathyroidism. The recommended target level of vitamin D is > 30 ng/mL.

The prevalence of secondary hyperparathyroidism increases as CKD progress. For patients with stage 3b-4 CKD, it is recommended to maintain parathyroid hormone levels in the upper normal range or slightly above the upper normal level. In patients with stage 4-5 CKD before dialysis, it is permissible to allow discretely exceeding the upper limit of the normal value of PTH. In patients on dialysis (CKD stage 5D), KDIGO guidelines suggest keeping serum PTH levels within 2 to 9 times the upper limit of the normal range of the assay. Vitamin D, synthetic activated vitamin D analogues, and calcimimetic agents are used to regulate the synthesis and secretion of parathyroid hormone.

The treatment of osteoporosis or low BMD in patients with CKD typically includes calcium and vitamin D supplementation, specific medications like bisphosphonates or denosumab, and management of underlying conditions such as hyperparathyroidism.

While there is now more and better information available to understand CKD-MBD and better drugs to treat CKD patients, it has been observed that focusing solely on correcting phosphate, calcium, vitamin D, and PTH levels is not sufficient to reduce the complications associated with CKD-MBD. Therefore, the development of alternative therapeutic approaches is imperative.