

Treatment for mineral bone disorder in chronic kidney disease

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Abstract

Chronic kidney disease mineral and bone disease (CKD-MBD) is a common complication of CKD that arises early in the course of the disease, and it is associated with high morbidity and mortality. CKD-MBD describe abnormalities in mineral metabolisms, skeletal health, and soft tissue calcifications. There are abnormalities in calcium (Ca), phosphate (P), parathyroid hormone (PTH), or vitamin D metabolism and bone turnover, mineralization, volume, linear growth, or strength. Among the laboratory abnormalities, alkaline phosphatase (AP), magnesium and/or fibroblast growth factor 23 (FGF-23) and klotho levels probably was added to original definition.

Management of CKD-MBD should focus on reversing or reducing the extent of biochemical abnormalities including the improvement of vitamin D deficiency, hyperphosphatemia, hypocalcemia, and hyperparathyroidism. The control of hyperphosphatemia is a key management of CKD-MBD. Dietary restriction of phosphorus is essential to control hyperphosphatemia. Phosphorus can be further managed through dialysis treatment and the use of drugs. Regarding calcium, in addition to the limitations of calcium phosphate binders, the most appropriate approach to avoid calcium overload in CKD patients. 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.

Today we have more and better information to understand the CKD-MBD and better drugs to treat the CKD patients. However, the treatment focusing on the correction of phosphate, calcium, vitamin D, and PTH was not enough to reduce the complications associated with

CKD-MBD, and the development of alternative therapeutic approaches is imperative.