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Current concepts of CKD-MBD

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In chronic kidney disease (CKD), hyperphosphatemia induces fibroblast growth factor-23 (FGF-23) expression that disturbs renal 1,25-dihydroxy vitamin D (1,25D) synthesis; thereby increasing parathyroid hormone (PTH) production. FGF-23 acts on the parathyroid gland (PTG) to increase 1α-hydroxylase activity and results in increase intra-gland 1,25D production that attenuates PTH secretion efficiently if sufficient 25D are available. Interesting, calcimimetics can further increase PTG 1α-hydroxylase activity that emphasizes the demand for nutritional vitamin D (NVD) under high PTH status. In addition, the changes in hydroxylase enzyme activity highlight the greater parathyroid 25-hydroxyvitmain D (25D) requirement in secondary hyperparathyroidism (SHPT); the higher proportion of oxyphil cells as hyperplastic parathyroid progression; lower cytosolic vitamin D binding protein (DBP) content in the oxyphil cell; and calcitriol promote vitamin D degradation are all possible reasons supports nutritional vitamin D supplement is crucial in SHPT. Clinically, NVD can effectively restore serum 25D concentration and prevent the further increase in PTH level.

Accumulation of IS or PCS enhance cytokine expression and inflammation, and promotes the degeneration of renal tubular epithelial cells and renal interstitial cells, ultimately resulting in renal interstitial fibrosis and renal glomerulosclerosis in CKD. The inflammatory response induced by IS and PCS results in secretion of many cytokines that cause arteriosclerosis and other cardiovascular diseases. Further, IS or PCS not only promote the apoptosis of osteoblasts, inhibit the differentiation and proliferation capacity of bone cells, affect bone turnover rate, and lower bone mineralization density, but also affect the arrangement of colloids, fibers, and crystals in the bone, and destroy the consistency of BAP orientation, resulting in reduced bone quality, and rendering the bone fracture-prone.

Vascular calcification is a critical complication in patients with chronic kidney disease (CKD) because it is predictive of cardiovascular events and mortality. In addition to the traditional mechanisms associated with endothelial dysfunction and the osteoblastic transformation of vascular smooth muscle cells (VSMCs), the regulation of calcification inhibitors, such as calciprotein particles (CPPs) and matrix vesicles plays a vital role in uremic vascular calcification in CKD patients because of the high prevalence of vitamin K deficiency. Vitamin K governs the gamma-carboxylation of matrix Gla protein (MGP) for inhibiting vascular calcification, and the vitamin D binding protein receptor is related to vitamin K gene expression. For patients with chronic kidney disease, adequate use of vitamin D supplements may play a role in vascular calcification through modulation of the calciprotein particles and matrix vesicles (MVs).