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Vascular Calcification in CKD

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Altered mineral metabolism in chronic kidney disease (CKD) leads to vascular calcification (VC). This process is not passive, but rather highly delicate and active, involving both traditional and non-traditional factors. Dysregulated calcium and phosphorus levels, abnormal induction of osteogenesis, loss of mineralization inhibitors, and differentiation of vascular smooth muscle cells (VSMCs) into osteoblast-like cells are all reported factors contributing to the pathophysiology of VC. The osteoblastic conversion of VSMCs causes CKD-induced tunica media and atherosclerotic plaques resulting in neointimal or medial VC. KD-MBD, which involves the upregulation of calcification promoters and downregulation of calcification inhibitors, accelerates the mineralization of blood vessels, further contributing to VC. Intimal calcification in CKD involves large arteries such as coronary artery which increased risk of myocardial infarction, arrhythmia, and stroke. Medial calcification in CKD leads to stiffening of the artery wall and decreases the compliance of blood vessels. Therefore, it causes elevation of pulse wave velocity and pulse pressure, vascular remodeling, and left ventricular hypertrophy. Intimal and medial calcifications in CKD results in cardiovascular disease. In end-stage renal disease (ESRD) patients, the incidence and prevalence of cardiovascular events is significantly higher than those of the general population. So, CKD is regarded as one of the most important risk factors for cardiovascular morbidity and mortality development.

Various methods can evaluate vascular calcification, including plain radiograph, ultrasonography, and CT scan. Although plain radiography is the most cost-effective and easily accessible method, it cannot distinguish between intimal and medial calcification. In contrast, CT scan is the most precise method of assessment; however, it is associated with drawbacks such as high cost and exposure to radiation.

Unfortunately, there is no treatment that can reverse VC in CKD patients. Currently, there are several therapeutic modalities that can reduce the progression of VC. The treatment of VC is based on the modulation of the key factors of VC. Mainly, therapeutic modalities include the reduction of calcium-phosphorus complex precipitation by using calcium-free phosphate binders, calcimimetics, vitamin D, bisphosphonates, vitamin K, and magnesium. Since hyperphosphatemia is a main cause of VC, lowering the level of phosphate is one of the most important treatments. Recent research shows altering the mechanism of differentiation of VSMCs by various calcific inducers and inhibitors is a strategy to slow down the progression of VC.

VC is a known main cause of increased cardiovascular morbidity and mortality in CKD patients. However, the pathophysiology of VC in CKD is complex and still not fully understood. Currently, no definite treatment can cure VC. Therefore, future research may focus on identifying the pathogenesis of VC in CKD and developing tailored management in large-scale clinical trials.