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An Updates on Diagnostic Methods of Vascular Calcification in End Stage Renal Disease

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Cardiovascular disease is one of the main causes of mortality in end-stage renal disease (ESRD) patients, and vascular calcifications (VCs) are recognized as a strong predictor of all-cause and cardiovascular mortality in ESRD patients. The chronic kidney disease (CKD) mineral bone disorder plays a pivotal role in cardiovascular disease via the development of VC. This review presents new evidence related to molecular mechanisms involved in the process of VC, as well as to discuss screening tools and new biomarkers related to VC in ESRD patients.

A number of non-invasive imaging techniques are available to screen for the presence of VC: plain x-rays to identify macroscopic calcifications of aorta and peripheral arteries; two-dimensional ultrasound for calcification of carotid arteries, echocardiography for assessment of valvular calcification; and computer tomography technologies that constitute the gold standard for quantification of coronary artery and aorta calcification.

The transformation of vascular smooth muscle cells into osteoblast-like cells seems to be a key element in the pathogenesis of VC. Numerous studies have identified circulating biomarkers potentially responsible for VC and have evaluated their link with imbalance between inducers and inhibitors of VC. Among these, Klotho/fibroblast growth factor-23, osteoprotegerin (OPG), osteopontin (OPN), bone morphogenic protein-7 (BMP-7), and fetuin-A have emerged as potential biomarkers for VC. More recently, attention has focused on sclerostin and Dickkopf-1 (DKK1), novel candidates for this bone-vascular axis in CKD patients, which inhibit the Wnt canonical signalling pathway involved in bone formation by regulating osteoblast proliferation and differentiation. Further well-designed clinical trials are needed to test the potential value of these biomarkers as a guide for interventions targeting VC.