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Vascular Calcification in Chronic Renal Disease: The Pathogenesis

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Introduction

Popular among people with chronic renal disease is vascular calcification. Patients with chronic kidney disease (CKD) and end-stage renal disease (ESRD) are more likely to have it because it is linked to a reduced estimated glomerular filtration rate (eGFR) and an abnormal bone mineral metabolism (ESRD). The pattern of arterial calcification in patients with advanced CKD comprises both intimal and medial calcification, as opposed to the predominately intimal calcification found in the absence of renal disease.

Description

It is worth noting that the severity and progression of vascular calcification is in general, much more pronounced in patients with CKD than people of comparable age, gender, and ethnicity as well as ethnicity in the general population while Coronary artery calcification (CAC) has been identified. Strongly associated with cardiovascular morbidity and general population mortality, the vascular calcification's predictive value in patients with CKD has not been studied as thoroughly.

Furthermore, it is unknown whether recovery of kidney function, such as after a successful kidney transplant, leads to regression of vascular calcification. Chen and colleagues examined the independent predictive value of CAC in adults in a recent study published in JAMA Cardiology with an eGFR ranging from 20 to 70 ml/min/1.73m. The study included 1541 participants from the Chronic Renal Insufficiency Cohort (CRIC) study who had coronary computed tomography at year 1 and did not have kidney disease a history of cardiovascular disease or peripheral vascular disease CAC score was independently associated with the composite cardiovascular disease outcome after adjusting for novel and traditional cardiovascular risk factors such as baseline eGFR and proteinuria over a mean follow-up of nearly 6 years [1].

The addition of the CAC score resulted in a marginal improvement over existing prediction models, which included traditional risk factors such as eGFR, proteinuria, and serum phosphorus, as well as novel biomarkers such as C-reactive protein and fibroblast growth factor 23 [2-4]. The findings of the CRIC study are consistent with the findings of a 2015 study from the MultiEthnic Study of Atherosclerosis (MESA) cohort, which discovered that CAC score was predictive of cardiovascular events in participants with CKD, defined as a single eGFR of 60 ml/min/1.73m2 or the presence of albuminuria.

Both studies included only people with pre-dialysis CKD, with the vast majority of them being men. Both had mild to moderate CKD, and both discovered that adding the CAC score provided only a marginal improvement over traditional risk factors for predicting cardiovascular outcomes. Nonetheless, these findings highlight the clinical importance of vascular calcification, which is common in the setting of CKD [5].

Is vascular calcification caused by CKD? Is reversible has been the subject of several discussions. Small studies describing CAC changes following a kidney transplant although several small longitudinal studies have suggested that CAC Early on after a successful outcome, scores stabilise or improve more recent and larger kidney transplantation; there have been studies on kidney transplant recipients shown no change or progression of longer CAC and aortic calcification scores 3.5 to 4.4 years of follow-up.

Over 4.4 years of follow-up in the largest study to date, significant regression was observed in only 2.1% of transplant recipients. These studies lacked interval assessments of vascular calcification, and the increase in traditional cardiovascular risk factors were associated with vascular calcification scores. As a result, it is possible that early improvements in vascular calcification associated with kidney function restoration were later offset by metabolic derangements associated with the persistence or recurrence of some degree of CKD, or with chronic immunosuppressive therapy [6]. Lomashvili and colleagues used an animal model of adenine-induced advanced CKD in a recent study published in the American Journal of Pathology to investigate the evolution of vascular calcification after the restoration of kidney function that is normal Instead of using a kidney transplantation model, the researchers implanted calcified aortic segments from mice with advanced CKD into littermates with normal kidney function. In their research, model of CKD, vascular calcification was further promoted by a high-phosphate diet and the administration of calcitriol 3 times weekly. A calcified aortic segment was then dissected and transplanted into an untreated littermate, replacing the corresponding aortic segment end-to-end.

To allow for comparison, neighbouring donor aortic segments were harvested before and after transplantation of calcium content. The authors discovered a moderate decrease in calcium content of the transplanted aortic segment in 34 of the 36 successful transplants. The reduction occurred within the first 2 to 3 weeks, and no further improvements were observed after up to 35 weeks of follow-up. The authors hypothesised that the initial rapid decrease in aortic calcium content was caused by the loss of relatively soluble calcium-phosphate precipitates, noting a calcium-to-phosphorus ratio in the persistent calcifications.

These findings imply that vascular calcification that develops as a result of CKD and disordered bone mineral metabolism frequently persists and can even worsen after normal or subnormal kidney function is restored [6]. Although injury and inflammation caused by the transplant could potentially contribute to the persistence of vascular calcifications, no inflammatory cells were found using hematoxylin and eosin or CD11b staining, and the authors concluded that no calcifications were previously observed in normal aortic segments transplanted into recipients with preserved kidney function [7]. Importantly, because vascular calcification in this animal model only affects the vessel media, it is possible that restoring kidney function has a different effect on intimal calcifications [8].

It is worth noting that osteoclast-like cells were not observed in the allografts in the experimental model, and no macrophage markers were expressed. Osteoblast-like cells are found in calcified human arteries. Osteoclast-like cells are frequently observed, and they are mostly found

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around plaques in the intima. Because vascular calcification is a dynamic process with many similarities to bone formation, it is reasonable to believe that macrophage-derived osteoclast-like cells play a role in the evolution of vascular calcification after kidney transplantation. However, in accordance with a recent study, macrophages in the vicinity of calcium deposits in human atherosclerotic plaques are phenotypically defective and unable to resorb the calcium deposits.

Conclusion

The significance of preventing arterial calcification in the context of CKD and altered bone mineral metabolism has been highlighted by recent study. Vascular calcification is a significant and independent predictor of poor cardiovascular outcomes once it has developed, and it is likely to endure even after a successful kidney transplant returns the GFR and bone mineral metabolism to normal or nearly normal.

Acknowledgement

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Conflict of Interest

There are no conflicts of interest by author.

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