Screening of Vascular Calcification in Hemodialysis Patients

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Vascular calcification (VC) is very common in subjects with chronic kidney disease (CKD), especially in hemodialysis (HD) patients, and it is becoming more prevalent with the worsening of kidney function and CKD duration. VC is associated with many adverse clinical outcomes, including ischemic cardiac events and subsequent vascular mortality (1). VC is usually seen in aging, after vascular injury and in various clinical conditions, such as diabetes, atherosclerosis and Mönckeberg’s medial sclerosis. VC has been associated with numerous ‘traditional’ risk factors such as aging, hypertension, diabetes or dyslipidemia, as well as with ‘nontraditional’ risk factors, such as hyperphosphatemia, hyperparathyroidism, hypervitaminosis D or excess administration of calcium salts (2). The hemodynamic consequences of VC include a loss of arterial elasticity, an increase in pulse wave velocity, development of left ventricular hypertrophy, decrease in coronary artery perfusion and myocardial ischemia (3).

The pathophysiology of vascular disease in CKD is recognized to be distinct from that related to atherosclerosis in the general population. Initially, VC was found as a passive phenomenon. However, it has been subsequently recognized as an active cell-mediated process (4, 5). VC may occur in either the intimal layer or the medial layer of the vessel wall, e.g. Mönckeberg’s sclerosis, which is very common in CKD patients (6). It is not yet clear to what extent these two patterns of VC might overlap in terms of pathogenetic mechanism, but both are associated with increased mortality in CKD patients (7). Intimal calcification is focal, associated with inflammation and the development of plaques and occlusive lesions, while adjacent regions of the vessel wall may remain remarkably normal. This form of calcification represents an advanced stage of atherosclerosis and is seen in the aorta, coronary arteries and other large vessels (4). Medial calcification, characterized by diffuse mineral deposition throughout the vascular tree, can occur completely independent of atherosclerosis or alongside, and is commonly observed in muscle-type conduit arteries (5). The two forms of calcification may well co-exist in the same vessel, which could be even more detrimental.

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 (CT) technologies that constitute the gold standard for quantification of coronary artery and aorta calcification. Electron beam CT (EBCT) and the newer multi-slice CT (MSCT) are highly sensitive methods, assessing accurately and quantitatively, especially coronary artery calcification, by using an electrocardiographic trigger for heart imaging only in diastole, thus avoiding motion artifacts (8). EBCT is not readily available in many hospitals. In contrast, almost every hospital has a multipurpose MSCT with software adjustments to allow gated imaging.

However, there are conflicting results about the correlation between the severity of coronary artery calcification measured by EBCT and subsequent clinical cardiac events in dialysis patients (9, 10). This can be explained by the fact that the arterial calcification score generated by CT scanning is a composite of both medial and intimal calcification. This is a limitation of these CT-based imaging techniques, as they are unable to distinguish between the two predominant arterial calcification sites. EBCT and MSCT could also be used for the assessment of VC in the aorta (11, 12).

Conventional CT may be used to evaluate non-coronary VC, especially aortic calcifications. Measuring the proportion of aortic circumference showing calcification can generate an aortic calcification index (ACI). This method seems to be simple, relatively inexpensive and useful for an initial diagnosis of VC. Taniwaki et al (13) used this method for the quantification of VC in HD patients with diabetes mellitus. It expresses calcification in 12 sectors as a percentage, so the extent of calcification in the aortic wall circumference is assessed, but not the thickness. Ohya et al (14) have
shown the aortic calcification area index (ACAI), which is derived from the ACI, directly measures the area of calcification. From the perspective of accuracy, the ACAI has an advantage over the ACI for evaluating abdominal aortic calcification and is also useful for serial observation of changes in aortic calcification in CKD patients.

References


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