KSN 2017 Abstract

Clinical impact of vascular calcification in end-stage renal disease

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Vascular calcification is associated with poor prognosis in hemodialysis patients and vascular calcification in the coronary arteries and the aorta has been recognized as an important risk factor for cardiovascular disease in hemodialysis patients. The transformation of vascular smooth muscle cells into osteoblast-like cells seems to be a key element in the pathogenesis of vascular calcification in the presence of calcium and phosphate deposition. In addition to traditional risk factors including hypertension and dyslipidemia, hemodialysis patients possess a number of non-traditional cardiovascular risk factors, which may be associated with the pathogenesis of vascular calcification, such as duration of dialysis and imbalance of metabolism. Fibroblast growth factor 23, which was shown to suppress synthesis and secretion of parathyroid hormone and to be associated with vascular dysfunction, atherosclerosis and left ventricular hypertrophy in the general population and in CKD patients, may be associated with the pathogenesis of vascular calcification. Vascular calcification causes increased arterial stiffness by medial calcification, followed by left ventricular hypertrophy and decreased coronary artery perfusion, and myocardial ischemia by intimal calcification. Thus, vascular calcification is thought to be associated with increased cardiovascular morbidity and mortality. Vascular calcification in coronary artery, aortic artery and cerebral artery are especially important for cardiovascular diseases. The severity of vascular calcification can be assessed with computed tomography (CT), but a simple technique is required as a routine practice. In an attempt to evaluate the extent of vascular calcification, we have proposed a simple non-invasive technique for estimating aortic arch calcification (AoACS) in hemodialysis patients. This lecture will summarize the following aspects: (i) a method of estimating AoAC and the correlation between AoAC score estimated by chest X-ray and AoAC volume evaluated by multi-detector CT as a gold standard, (ii) Kaplan-Meier analysis in terms of cardiovasucular mortality in patients with AoACS (iii) clinical impact of left ventricular dysfunction and relation of AoAC to diastolic left ventricular dysfunction (iv) relation of oral 1alfa-hydroxy vitaminD3 to the progression of aortic arch calcification using AoACS in hemodialysis patients (v) relation of FGF23 to progression of AoAC in non-diabetic hemodialysis patients (vi) relation of the presence of cerebral arterial calcification to the prevalence of cerebrovascular disease (vii) relation of the presence of cerebrovascular disease to the presence of coronary calcification. We suggest that screening patients undergoing dialysis for the presence of AoACS or cerebral calcification

is a cost-effective, efficient way to identify those patients at the highest risk of cardiovascular events and/or cerebrovascular events.