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Effect of vitamin K2 on progression of atherosclerosis and vascular calcification in nondialyzed patients with chronic kidney disease stages 3–5

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Abstract

Observational studies have shown that high dietary intake of vitamin K2 is associated with reduced risk of coronary vascular disease and vascular calcification.

We assessed the effect of vitamin K2 substitution on the progression of atherosclerosis and calcification in nondialyzed patients with CKD stages 3-5.

The study included 42 nondialyzed patients with CKD. The following measurements were taken at baseline and after 270 ±12 days of supplementation with vitamin K2 at a dose of 90 µg (menaquinone, MK-7) together with 10 µg of cholecalciferol (K+D group) or 10 µg of cholecalciferol (group D): common carotid intima-media thickness (CCA-IMT), coronary artery calcification score (CACS), basic biochemical parameters, lipids, and calcification modulators: matrix Gla protein (MGP), desphosphorylated-uncarboxylated MGP (dp-ucMGP), osteoprotegerin (OPG), fetuin A, osteocalcin (OC), and fibroblast growth factor 23.

The increase of CCA-IMT was significantly lower in the K+D group compared with the D group: from 0.95 ±0.2 mm to 1.01 ±0.3, P = 0.003 vs from 1.02 ±0.2 mm to 1.16 ±0.3, P = 0.003 (ΔCCA-IMT, 0.06 ±0.08 vs 0.136 ±0.05 mm, P = 0.005, respectively). The increase in CACS was slightly lower in the K+D group than in the D group (ΔCACS, 58.1 ±106.5 AU vs 74.4 ±127.1 AU, P = 0.7). In the K+D group, a significant decrease in the level of dp-ucMGP and total OC was observed.

A 270-day course of vitamin K2 administration in patients with CKD stages 3-5 may reduce the progression of atherosclerosis, but does not significantly affect the progression of calcification. Vitamin K2 significantly changes the levels of calcification promoters and inhibitors: dp-ucMGP, OC, and OPG.

ClinicalTrials.gov NCT01101698.

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