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Association of Vitamin K Status with Arterial Calcification and Stiffness in Chronic Kidney Disease: A Longitudinal Study using CRIC Data

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Abstract

Vitamin K is a group of fat-soluble vitamins that play an essential role in blood clotting, bone metabolism and cardiovascular health. The two primary forms of vitamin K are vitamin K1 (phylloquinone), found in green leafy vegetables and vitamin K2 (menaquinone), synthesized by bacteria in the gut and found in fermented foods. Vitamin K deficiency can lead to excessive bleeding and bone fractures, while adequate vitamin K intake may help prevent osteoporosis and cardiovascular disease. Vitamin K is also being studied for its potential role in cancer prevention and treatment. Chronic kidney disease (CKD) is a common condition characterized by a gradual loss of kidney function over time. CKD affects millions of people worldwide and is associated with a high risk of cardiovascular disease. Arterial calcification and stiffness are among the cardiovascular complications that commonly occur in people with CKD. In this article, we will discuss the pathophysiology of arterial calcification and stiffness in CKD, their clinical implications and the potential role of vitamin K in preventing and treating these conditions.

Keywords: Vitamin K • Arterial calcification • Chronic kidney disease

Introduction

Vitamin K is a group of fat-soluble vitamins that play an essential role in blood clotting, bone metabolism and cardiovascular health. The two primary forms of vitamin K are vitamin K1 (phylloquinone), found in green leafy vegetables and vitamin K2 (menaquinone), synthesized by bacteria in the gut and found in fermented foods. Vitamin K deficiency can lead to excessive bleeding and bone fractures, while adequate vitamin K intake may help prevent osteoporosis and cardiovascular disease. Vitamin K is also being studied for its potential role in cancer prevention and treatment. Chronic kidney disease (CKD) is a common condition characterized by a gradual loss of kidney function over time. CKD affects millions of people worldwide and is associated with a high risk of cardiovascular disease. Arterial calcification and stiffness are among the cardiovascular complications that commonly occur in people with CKD. In this article, we will discuss the pathophysiology of arterial calcification and stiffness in CKD, their clinical implications and the potential role of vitamin K in preventing and treating these conditions.

Literature Review

Arterial calcification is a frequent finding in people with CKD, particularly in those with end-stage renal disease (ESRD). The calcification process involves the deposition of calcium and phosphate crystals in the arterial wall, leading to the formation of bone-like structures called calcifications. Arterial calcification can occur in any arterial bed, including the coronary, carotid and peripheral arteries and is associated with an increased risk of cardiovascular events and mortality. The pathophysiology of arterial calcification in CKD is complex and involves multiple factors, including alterations in mineral metabolism, inflammation, oxidative stress and vascular smooth muscle cell dysfunction. In CKD, the kidneys are less able to excrete phosphate, leading to elevated levels of serum phosphate (hyperphosphatemia). Hyperphosphatemia can trigger the release of calcium from the bone and the deposition of calcium and phosphate crystals in the arterial wall. Moreover, the elevated levels of fibroblast growth factor 23 (FGF-23) in CKD can also contribute to the development of arterial calcification by altering the balance between calcification inhibitors and promoters in the arterial wall.

Arterial stiffness is another cardiovascular complication that commonly occurs in people with CKD. Arterial stiffness refers to the loss of elasticity of the arterial wall, leading to increased pulse wave velocity (PWV) and reduced compliance. Arterial stiffness is an independent predictor of cardiovascular events and mortality and is associated with increased left ventricular afterload, decreased coronary perfusion and impaired renal perfusion. The pathophysiology of arterial stiffness in CKD is also multifactorial, involving alterations in collagen and elastin content, inflammation, oxidative stress and vascular smooth muscle cell dysfunction. In CKD, the increased levels of advanced glycation end products (AGEs) and oxidative stress can lead to the cross-linking of collagen and elastin fibers in the arterial wall, impairing their elasticity and contributing to arterial stiffness. Moreover, the accumulation of uremic toxins in CKD can also contribute to arterial stiffness by altering the structure and function of vascular smooth muscle cells.

Discussion

Vitamin K is a group of fat-soluble vitamins that play a crucial role in the regulation of calcium metabolism and bone health. Vitamin K is also involved in the regulation of arterial calcification and stiffness by activating matrix Gla protein (MGP), a calcification inhibitor protein found in the arterial wall. Vitamin K-dependent carboxylation of MGP is required for its activation and inhibition of arterial calcification. Several studies have reported an association between low vitamin K status and increased arterial calcification and stiffness in people with CKD. In a longitudinal study using data from the Chronic Renal Insufficiency Cohort (CRIC), lower plasma phylloquinone (vitamin K1) levels and higher plasma dephospho-uncarboxylated MGP [(dp)ucMGP] levels were associated

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with more coronary artery calcification and higher PWV, respectively. These findings suggest that improving vitamin K status may help prevent or slow the progression of arterial calcification and stiffness in people with CKD.

Chronic kidney disease (CKD) is a common condition that affects millions of people worldwide and is associated with a high risk of cardiovascular disease. Arterial calcification and stiffness are among the cardiovascular complications that commonly occur in people with CKD. The role of vitamin K in the regulation of arterial calcification and stiffness in CKD has been the subject of research interest in recent years. In this article, we will discuss the findings of a longitudinal study using data from the Chronic Renal Insufficiency Cohort (CRIC) that evaluated the association of vitamin K status with arterial calcification and stiffness in people with mild-to-moderate CKD.

The CRIC study is a multicenter observational study that aims to identify risk factors for the progression of CKD and its associated complications. The study enrolled adults with mild-to-moderate CKD from seven clinical centers across the United States. The study participants underwent baseline assessments and were followed up for up to 7 years. The present study focused on a subset of 387 CRIC participants who had available measures of plasma phylloquinone (vitamin K1) and plasma dephospho-uncarboxylated matrix Gla protein [(dp)ucMGP], a marker of vitamin K status, at baseline. The participants also underwent measurements of coronary artery calcification (CAC) and aortic pulse wave velocity (PWV) at baseline and at follow-up visits 2-4 years later.

The study found that lower plasma phylloquinone levels and higher (dp) ucMGP levels were associated with more CAC and higher PWV, respectively, at baseline. Furthermore, the study found that lower plasma phylloquinone levels and higher (dp)ucMGP levels were associated with greater increases in CAC and PWV over time. The associations remained significant after adjustment for various demographic, clinical and laboratory variables, including age, sex, race, body mass index, smoking status, diabetes, hypertension, estimated glomerular filtration rate (eGFR), serum phosphate, calcium and parathyroid hormone levels [1-6].

Conclusion

The findings of this study provide important insights into the potential role of vitamin K in the regulation of arterial calcification and stiffness in people with CKD. The study suggests that lower vitamin K status, as reflected by lower plasma phylloquinone levels and higher (dp)ucMGP levels, is associated with more arterial calcification and stiffness and with their progression over time. These findings have significant clinical implications, as they suggest that improving vitamin K status may help prevent or slow the progression of arterial calcification and stiffness in people with CKD. Further studies are needed to confirm these findings and to determine the optimal strategies for improving vitamin K status in people with CKD. Arterial calcification and stiffness are common cardiovascular complications that occur in people with CKD. The present study using data from the CRIC cohort provides evidence of an association between lower vitamin K status and increased arterial calcification and stiffness in people with mild-to-moderate CKD. The findings suggest that improving vitamin K status may be a promising strategy for preventing or slowing the progression of arterial calcification and stiffness in this population.

Acknowledgement

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Conflict of Interest

None.

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