

## FGF23 and inflammation

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### Abstract

Systemic inflammation is a recognized feature in chronic kidney disease (CKD). The role of systemic inflammation in the pathogenesis of vascular calcification was recently settled. FGF23 was recently accused as a direct stimulus of systemic inflammation. This finding explains the strong association of FGF23 to vascular calcification and increased mortality among CKD.

**Key words:** Chronic kidney disease; Inflammation; Vascular calcification; FGF23; Phosphate binders

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**Core tip:** In this letter, the authors confirm the direct offending role of FGF23 in the pathogenesis of vascular calcification and increased mortality among chronic kidney disease patients.

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### TO THE EDITOR

In the September 6 issue of *World J Nephrol*, the review article titled "Vascular calcification: When should we interfere in chronic kidney disease patients and how?" showed that the exact pathogenesis of inflammation in chronic kidney disease (CKD) is not fully understood and that many of the inflammatory markers and mediators can promote vascular calcification (VC) in CKD patients. These factors include interleukin 1 (IL-1), IL-6, C-reactive protein and tumor necrosis factor alpha (TNF $\alpha$ )<sup>[1-4]</sup>. *In vivo* molecular imaging techniques have disclosed that VC is preceded by inflammation within the arterial wall<sup>[5,6]</sup>. Similar finding was confirmed by a longitudinal study using positron emission tomography (PET)/computed tomographic (CT) scan<sup>[7]</sup>.

The positive correlation between FGF23 and VC was reported by many groups<sup>[8,9]</sup>. Similar results were reported in healthy older men, irrespective of traditional risk factors<sup>[10]</sup> and in children with CKD<sup>[11]</sup>. Inflammation markers can mitigate the correlation between FGF-23 and vascular calcification<sup>[12]</sup>.

In November issue of kidney international (KI), Singh *et al*<sup>[13]</sup> demonstrated that FGF23 stimulates the

hepatic secretion of the inflammatory markers IL-6 and C-reactive protein. This finding demonstrates the contribution of FGF23 to the chronic inflammatory status of CKD. It also highlights the role of FGF23 in the vessel inflammation that precedes arterial calcification. In addition, this finding stimulates the energetic control of FGF23 starting in the very early days of stage 2 in CKD patients as we recommended in our review<sup>[14]</sup>. Lastly, this study supported the value of phosphate binders that can suppress FGF23 and the possible link between such agents and their anti-inflammatory effects and their impact on overall mortality<sup>[15-18]</sup>.

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