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1-Fibroblast growth factor -237 vascular calcification in relation to undercarboxylated osteocalcin

ABSTRACT

Fibroblast growth factor-23 (FGF-23) has been linked to vascular calcification,ventricular hypertrophy and mortality in chronic kidney disease (CKD), although these linksmay not be direct and independent. Similar grave outcomes are associated with inflammation and oxidative stress in CKD. Recently, accumulating evidence has linked components of phosphate homeostasis to inflammation and oxidative stress. The interaction between the triad of inflammation, FGF-23 and cardiovascular outcomes is underinvestigated

2-Serum aldosterone levels in patients with diabetic nephropathy in relation to vascular calcification

Abstract

Vascular calcification is frequently found already in early stages of chronic kidney disease (CKD) patients and is associated with high cardiovascular risk. The process of vascular calcification is not considered a passive phenomenon but involves, at least in part, phenotypical transformation of vascular

smooth muscle cells (VSMCs). Following exposure to excessive extracellular phosphate concentrations, VSMCs undergo a reprogramming into osteo-/chondroblast-like cells. Such ‘vascular osteoinduction’ is characterized by expression of osteogenic transcription factors and triggered by increased phosphate concentrations. A key role in this process is assigned to cellular phosphate transporters, most notably

the type III sodium-dependent phosphate transporter Pit1. Pit1 expression is stimulated by mineralocorticoid receptor activation. Therefore, aldosterone participates in the phenotypical transformation of VSMCs.

3- Osteoprotegrin & vascular calcification in patients with CKD

 ABSTRACT

 In patients with uremia either increased or suppressed bone turnover can be observed. However, secondary hyperparathyroidism is the most common type of renal osteodystrophy and is characterized by PTH-related bone loss. OPG serum levels are increased in patients with chronic kidney disease and it might inhibit osteoclastogenesis and bone resorption induced by PTH in patients with uremia Abnormalities of calcium–phosphate balance, with subsequent bone metabolism disorders, are among the key and earliest features of chronic kidney disease (CKD). Recently, another consequence of these abnormalities was brought to light—namely, vascular calcification. Most studies performed in patients on dialysis suggest that their vascular calcification is more advanced than that seen in the general population. Furthermore, the progression of vessel wall mineralization is much more dynamic in patients with CKD