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CIRCULATING FGF-23 AS AN INDEPENDENT CORRELATE OF ATHEROSCLEROSIS IN EARLY STAGES OF CKD

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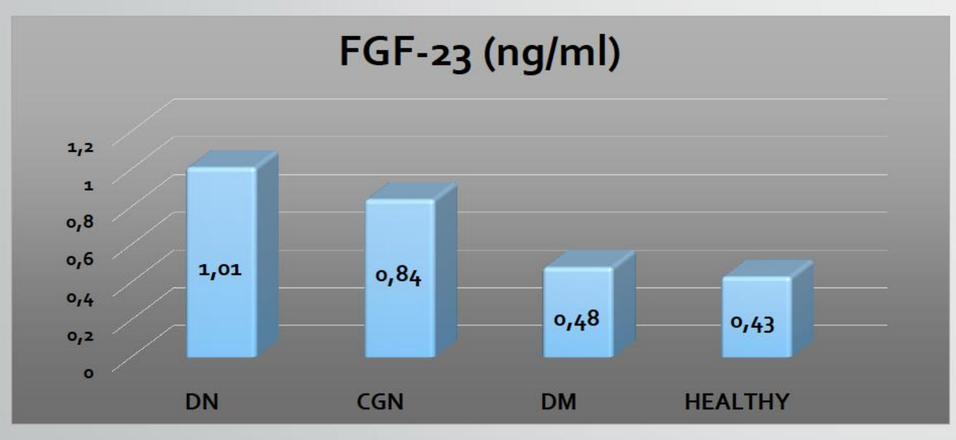
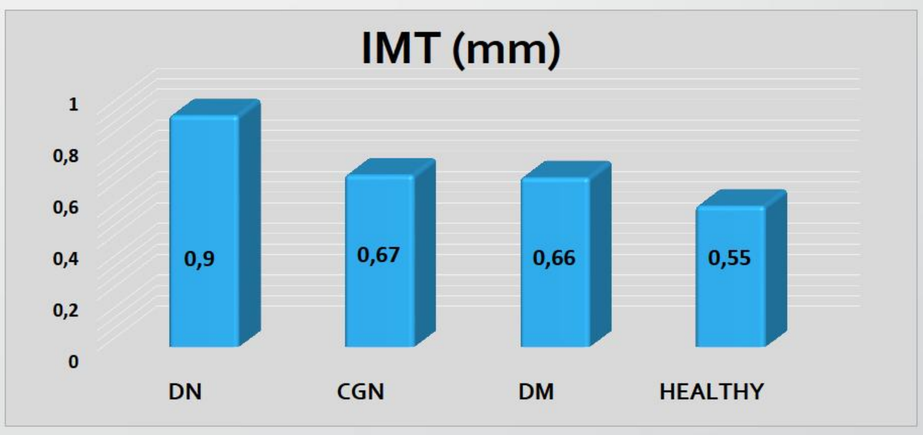
Background/Aim: Clinical and experimental evidence support a role for fibroblast growth factor (FGF-23) in promoting osteoclastic bone resorption, but the precise molecular mechanisms are not yet fully understood. FGF-23 has been implicated in chronic kidney disease (CKD) and is important in humans for osteogenesis [1,2]. However, to date the possible role of FGF-23 in secondary hyperparathyroidism (SHP) is still unclear. The aim of this study was to investigate the serum levels of FGF-23 and its potential correlation with the atherosclerotic markers and albuminuria in patients with early stages of CKD.

Methods: CKD patients (n=50) of stages 1 and 2 with type 2 diabetic nephropathy (DN, n=25) and chronic glomerulonephritis, (CG, n=25) were included. As controls, there were two groups, patients with diabetes type 2 without CKD (n=40) and healthy individuals (n=40). FGF-23 levels were measured by an ELISA method. Intima media thickness (IMT) of carotid arteries as a sub-atherosclerotic marker and presence of atherosclerotic plaque were evaluated by a high resolution ultrasonography. Statistical analysis was performed with the use of a SPSS system.

Results: The levels of FGF-23 were significantly higher in patients than in the control groups ($p<0.004$). IMT was also significantly higher in patients than in the control groups ($p<0.001$). There was negative strong correlation between FGF-23 and GFR ($r= -0.8$, $p<0.005$) and between FGF-23 and IMT ($r= 0.85$, $p<0.0001$). Further, FGF-23 levels were independent correlates of IMT ($p<0.0001$), albuminuria ($p<0.004$) and atherosclerotic plaque ($p< 0.0001$).





Discussion: Recent accumulated evidence suggests a role of FGF-23 in SHP, hyperphosphatemia and CKD. However a role in cardiovascular disease (CVD) is also suggested in CKD patients [1,2,3]. Cardiovascular events in CKD are the most common cause of morbidity and mortality whereas just 1% of CKD patients reached to end stage renal disease to haemodialysis. The observation that atherosclerosis begins in DN before in comparison with CKD from other causes leaded us to investigate the hypothesis that FGF-23 is higher in DN and responsible for preclinical atherosclerosis from the stages of micro-albuminuria. Our study suggests the FGF-23 levels as potential pathway of atherosclerosis in CKD, especially in DN.

Conclusions: This study suggests that serum levels of FGF-23 were strongly correlated with IMT, atheromatic plaque as well as with albuminuria, attributing a role for FGF-23 in atherosclerosis of CKD patients. FGF-23 might present an independent correlate of atherosclerosis in early stages of CKD.

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