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Commentary

See the original article by Mahmoodnia et al (J Parathyr Dis. 2020;8:e9154)

Sclerostin in hemodialysis patients

Azar Baradaran*

Implication for health policy/practice/research/medical education

Sclerostin is a glycoprotein with 190-amino-acid, which is mainly released by osteocytes. Sclerostin reduces bone construction following preventing the final differentiation of osteoblasts and accelerating their apoptosis. The exact role of sclerostin in dialysis patients requires further studies since some authors believe that this biomarker has a relationship with bone-vascular axis and accordingly with parathyroid function. It is possible that, sclerostin related to the all-cause mortality, coronary artery calcifications and the prevalence of heart and vascular events.

Keywords: Sclerostin, Hemodialysis, Parathyroid hormone, Bone-vascular axis

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In the descriptive-analytic investigation, to find the association of plasma sclerostin concentration with demographic and biochemical factors, in hemodialysis, Mahmoodnia et al showed the level of parathormone was greater in non-diabetic hemodialysis patients versus hemodialysis diabetic population. They also found the serum level of sclerostin had a positive correlation with dialysis adequacy detected by urea reduction ratio and Kt/V. Their study was conducted on 89 hemodialysis patients consisting diabetic and non-diabetics. In diabetic patients, sclerostin had a negative correlation with plasma hemoglobin (r=-0.343, P=0.021). Moreover, in diabetic cases, sclerostin had a positive relationship with urea reduction ratio (r=0.463, P=0.001) (1). Sclerostin is a glycoprotein with 190-amino-acid, which is mainly released by osteocytes. Sclerostin reduces bone construction following preventing the final differentiation of osteoblasts and accelerating their apoptosis (2). The regulating effect of sclerostin in bone formation is conducted by inhibiting the signaling pathway of Wnt–β-catenin (3). It is possible that, this substance modulates mineral bone metabolism in diabetic individuals with chronic kidney disease (4). Additionally, Sclerostin was detected as an anti-anabolic bone parameter triggering the calcification of soft tissues (5). Study regarding the impact of sclerostin on chronic kidney disease -mineral and bone disorder (*CKD-MBD*) is scares and requires investigations. In a previous study Asamiya et al showed, serum sclerostin levels were related to serum phosphate and fibroblast growth factor-23 levels in patients with low intact parathyroid hormone (iPTH) levels (3). In another study Abdallah et al showed, higher levels of sclerostin in individuals with end-stage renal disease on hemodialysis were correlated with higher risk of vascular calcification (6). The study by Kuo et al on eighty-nine peritoneal dialysis patients, found sclerostin was associated negatively with intact parathyroid hormone ($r=-0.357$, $P<0.001$) after adjustments for age and sex (7), since such a correlation was not detected in the study by Mahmoodnia et al in hemodialysis individuals (1). Meanwhile, Zou et al, in a study on 165 peritoneal dialysis and hemodialysis patients found a low plasma concentration of sclerostin was correlated with better overall survival and lower prevalence of cardiovascular events in patients with peritoneal dialysis. However, this relationship was not detected in hemodialysis. They also showed, sclerostin concentration was not associated with cardiovascular events in either patients with peritoneal dialysis or hemodialysis (8). The above studies showed that, to find the exact role of sclerostin in dialysis still requires much investigation. Since this hormone is a novel biomarker which has relationship with bone-vascular axis and accordingly with parathyroid glands function. It is possible that, sclerostin related to the all-cause mortality, coronary artery calcifications and the prevalence of heart and vascular events (9,10). Findings of the study by Mahmoodnia et al are interesting and requires further evaluation by larger studies in hemodialysis population

Author's contribution

AB is the single author of the manuscript.

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Department of Pathology, Isfahan University of Medical Sciences, Isfahan, Iran.
***Corresponding author:** Prof. Azar Baradaran, Email: azarbaradaran@yahoo.com, azarbaradaran@med.mui.ac.ir

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