Original article

Association between serum sclerostin levels and chronic kidney disease–mineral and bone disorders in hemodialysis patients

Vo Thi Hoai Huong*, Nguyen Hoang Thanh Van, Phan Thi Minh Phuong

University of Medicine and Pharmacy, Hue University

***Corresponding Author:** Vo Thi Hoai Huong; Email: vthhuong@huemed-univ.edu.vn
Received: 15/10/2025; **Accepted:** 04/04/2026; **Published:** 30/04/2026
DOI: 10.34071/jmp.2026.2.742

Abstract

Objective: 1. To evaluate the mineral and bone disorders, bone mineral density, and serum sclerostin levels in patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis, compared with a healthy control group with normal renal function; 2. To evaluate the relationship between serum sclerostin concentration and CKD – Mineral and Bone disorders (CKD-MBD) in hemodialysis patients.

Patients and methods: This is a cross-sectional descriptive study on 51 CKD hemodialysis patients at theNephrology-Musculoskeletal Department and Hemodialysis Department, Hue Central Hospital and 60 healthy people with normal kidney function regularly came for health check-ups at the Internal Medicine Clinic of Hue University of Medicine and Pharmacy from April 2023 to 2025.

Results: The study included 60 controls (mean age 49.27 ± 10.98) and 51 G5HD hemodialysis patients (mean age 47.92 ± 14.88), with no significant difference between groups ($p > 0.05$). CKD-MBD was prevalent in the hemodialysis group, with 86.27% having hyperphosphatemia, over 90% having elevated PTH, and mean BMD at the femoral neck, total femur, and lumbar spine of 0.820 ± 0.221, 0.840 ± 0.194, and 1.003 ± 0.194 g/cm², respectively. Serum sclerostin levels were significantly elevated in G5HD patients (937.152 ± 881.105 pg/mL) compared to controls (359.52 ± 168.21 pg/mL) ($p < 0.05$). Importantly, serum sclerostin correlated negatively with PTH and BMD at both the femoral neck and total hip ($p < 0.05$), suggesting that elevated sclerostin may reflect impaired bone metabolism in hemodialysis patients.

Conclusion: In conclusion, according to our study, CKD-MBD are common in patients with maintenance hemodialysis. Serum sclerostin levels were significantly higher in the hemodialysis group compared to healthy controls. Regarding the association between sclerostin and mineral-bone disorders as well as bone mineral density in hemodialysis patients, our findings demonstrate that serum sclerostin levels were significantly and inversely correlated with PTH levels and bone mineral density at the femoral neck and total hip.

Keywords: *Sclerostin, chronic kidney disease, CKD, CKD-MBD.*

1. BACKGROUND

Chronic kidney disease (CKD) represents a major global health burden, characterized by a progressive and irreversible decline in renal function that leads to multiple systemic complications. Hemodialysis (HD) serves as a life-sustaining renal replacement therapy for patients with end-stage chronic kidney disease, performing the essential functions of waste removal, fluid balance, and electrolyte regulation that the kidneys can no longer adequately maintain. Among these complications of CKD, chronic kidney disease–mineral and bone disorder (CKD-MBD) is a significant and complex manifestation, involving disturbances in calcium, phosphate, parathyroid hormone (PTH), and bone metabolism [1]. CKD-MBD contributes not only to impaired bone quality and increased fracture risk but also to vascular and soft tissue calcification, which substantially elevate cardiovascular morbidity and mortality among CKD patients, especially in HD patients.

Sclerostin, a glycoprotein produced primarily by osteocytes, plays a key role in bone metabolism by inhibiting the Wnt/β-catenin signaling pathway and suppressing bone formation [2]. Elevated serum sclerostin levels have been reported in patients with CKD, particularly in those undergoing dialysis, suggesting a potential relationship between sclerostin and CKD-MBD [3-6]. However, the exact relationship between sclerostin concentrations, bone turnover, and mineral metabolism remains incompletely understood. Understanding the interplay between sclerostin and mineral-bone metabolism is therefore crucial for improving diagnostic and therapeutic strategies in this population. Therefore, investigating serum sclerostin concentrations in hemodialysis patients is essential for improving the management of bone and mineral disorders in CKD. Our study, entitled "Serum Sclerostin Concentrations and Chronic Kidney

Disease–Mineral and Bone Disorders in Hemodialysis Patients," was conducted with the objective of:

1. Evaluating the mineral and bone disorders, bone mineral density, and serum sclerostin levels in patients with chronic kidney disease undergoing maintenance hemodialysis, compared with a healthy control group with normal renal function.

2. Evaluating the relationship between serum sclerostin concentration and CKD–Mineral and Bone disorders (CKD-MBD) in hemodialysis patients.

2. MATERIALS AND METHODS

2.1. Study subjects

- Study period and location: Our study was conducted at the Nephrology-Musculoskeletal Department and Hemodialysis Department,, Hue Central Hospital, and the Internal Medicine Clinic of Hue University of Medicine and Pharmacy Hospital from April 2023 to 2025.

- Inclusion criteria:

+ Hemodialysis group (G5HD): 51 patients aged ≥ 16 years who were diagnosed with end- stage renal disease (ESRD) and undergoing hemodialysis (HD) at least 3 months, consent to participate in the study [7].

+ Control group: 60 healthy adult individuals with normal kidney function undergoing routine health check-ups at the Internal Medicine Clinic of Hue University of Medicine and Pharmacy Hospital.

- Exclusion criteria: Patients who do not consent to participate in the study. Patients with acute kidney injury or acute-on-chronic kidney disease. Patients in critical, life-threatening conditions who are unable to participate in the study. Using of any medication known to affect bone turnover - including calcium, vitamin D, anti-osteoporotic agents (bisphosphonates, raloxifene, etc.), corticosteroids, or calcimimetics - within at least one month prior to enrollment. Patients with severe active infection, prior bone surgery or joint replacement (which may affect BMD results), neuropsychiatric disorders affecting cognitive function and pregnancy or breastfeeding.

2.2. Research method

- Study design: Cross-sectional descriptive study.

- Sample size and sampling method: Convenience sampling was used to enroll all eligible patients diagnosed with ESRD and undergone hemodialysis (G5HD group). The study included 51 HD patients and the control group consisted of 60 healthy individuals with normal kidney function undergoing routine health check-ups.

- Clinical variables: Sex, age, and blood pressure. For hemodialysis patients: Blood pressure was measured in the supine position. Measurements were taken on the arm without an arteriovenous fistula (AVF) used for hemodialysis. Blood pressure was measured twice at 2-minute intervals, and the mean of the two readings was recorded. If the difference between the two measurements exceeded 10 mmHg, a third measurement was taken, and the average of the second and third readings was used. Systolic blood pressure (SBP) was determined at Korotkoff phase I, and diastolic blood pressure (DBP) at Korotkoff phase V. Patients were diagnosed with hypertension when SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg.

- Paraclinical variables in the CKD patient group:

+ Fasting blood samples were collected in tubes from the patients and control groups after proper disinfection.

+ Biochemical parameters: Urea, creatinine, eGFR (Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI 2009 equation), calcium, phosphorus, parathyroid hormone (PTH).

The formula for calculating corrected calcium is as follows:

Corrected calcium (mg/dL) = Total calcium + [0.8 × (4 – serum albumin)]

This formula adjusts the total serum calcium concentration based on the serum albumin level to more accurately reflect the physiologically active (ionized) calcium concentration.

Diagnosis of Mineral and Bone Disorders according to KDIGO criteria:

Corrected serum calcium:

+ Normal: 2.1 - 2.5 mmol/l
+ Increased: > 2.5 mmol/l
+ Decreased: < 2.1 mmol/l

Serum phosphate:

+ Decreased: < 0.8 mmol/
+ Normal: 0.8 - 1.5 mmol/l
+ Increased: > 1.5 mmol/l

Serum parathyroid hormone (PTH): Reference range (normal value): 15 - 65 pg/mL.

+ Serum sclerostin levels were measured at the Department of Biochemistry and Immunology, Hue University of Medicine and Pharmacy Hospital, using the Enzyme-Linked Immunosorbent Assay (ELISA) method performed on a TECAN SUNRISE™ microplate reader (Switzerland), with data calculated using Magellan V7.5 software. The assay kits were manufactured by Abbexa, United Kingdom.

+ BMD measurements (g/cm^2) were determined for the anteroposterior lumbar spine (L1–L4) and mean of proximal right and left femur (total and subregions) by dual-energy X-ray absorptiometry,

according to standard protocol.

2.3. Statistical analysis

Using SPSS version 27.0 software. Data were presented as mean ± SD or number of cases and percentages. Comparisons between variables in the study groups were performed using unpaired two-tailed Student's t-tests. Correlation between sclerostin levels and various variables was done using Spearman correlation coefficient analysis. The data showed that serum sclerostin was skewed and should be logarithmically transformed before analysis. $p < 0.05$ was considered statistically significant.

2.4. Ethical Considerations in Medical Research

The study was approved by the Ethics Committee of Hue University of Medicine and Pharmacy prior to implementation. Participants were clearly informed about the study objectives and provided with detailed explanations regarding their voluntary participation.

3. RESULTS

Table 1. General characteristics of the study population

Characteristics	Control group[1] (n = 60)	G5HD[2] (n = 51)	p
Gender (Male/Female)	25/35	31/20	
Age	49.27 ± 10.98	47.92 ± 14.88	p = 0.547
SBP (mmHg)	115.42 ± 10.35	144.61 ± 17.57	**p < 0.001**
DBP (mmHg)	68.58 ± 6.83	80.78 ± 7.44	**p < 0.001**
Hypertension (%)	0%	74.51%	
Urea (mmol/l)	4.64 ± 1.09	25.26 ± 9.61	**p < 0.001**
Creatinine (µmol/l)	67.62 ± 12.19	973.06 ± 296.62	**p < 0.001**
eGFR (ml/min/1,73m^2)	99.72 ± 10.68	5.06 ± 2.40	**p < 0.001**
Duration of HD (months)		57.29 ± 51.50	

The study population in our research consisted of two groups: a control group comprising 60 individuals, including 25 males and 35 females, with a mean age of 49.27 ± 10.98; and a G5HD group including 31 males and 20 females, with a mean age of 47.92 ± 14.88. The difference in age between the two groups was not statistically significant ($p > 0.05$). The group of CKD patients with an average dialysis duration of 57.29 ± 51.50 months has higher SBP, DBP, urea, creatinine and lower eGFR than the control group with statistical significance ($p < 0.05$). The rate of hypertension in the G5HD was 74.51%.

Table 2. Charateristics of mineral and bone disorders on CKD patients

Characteristics	G5HD (n=51)	
Calcium (mmol/l)	**2.33 ± 0.22**	
	< 2.1	11.76%
	2.1 - 2.5	68.63%
	> 2.5	19.61%
Phosphorus (mmol/l)	**2.03 ± 0.59**	
	< 0.8	0.00%
	0.8 - 1.5	13.73%
	> 1.5	86.27%
PTH (pg/mL)	**711.32 ± 905.62**	
	≤ 65	9.8%
	> 65	90.2%
BMD (g/cm^2)	Femoral neck	0.820 ± 0.221
	Total neck	0.840 ± 0.194
	Lumbar Spine (L1-L4)	1.003 ± 0.194

The mean serum calcium (adjusted for serum albumin), phosphorus, and PTH concentrations in 52 patients undergoing hemodialysis in the study were 2,33 ± 0.22 mmol/l; 2,03 ± 0.59 mmol/l, and 711.32 ± 905.62 pg/dL, respectively. In terms of blood calcium disorders, 11.76% of patients had hypocalcemia and about 1/5 had hypercalcemia. Our study found that 86.27% had hyperphosphatemia and more than 90% of G5HD patients had increased serum PTH levels. Regarding bone density in the G5HD group, the mean BMD at the femoral neck, total femur, and lumbar spine were 0.820 ± 0.221; 0.840 ± 0.194 and 1.003 ± 0.194 g/cm^2, respectively.

Table 3. Serum Sclerostin levels in CKD patients and control group

Group	Control group [1] (n = 60)	G5HD[2] (n = 51)
Sclerostin (pg/ml)	359.52 ± 168.21	937.152 ± 881.105
p < 0.001		

The mean serum sclerostin concentration in the control group of our study was 359.52 ± 168.21 pg/mL. The G5HD groups exhibited significantly higher serum sclerostin level compared to the control group (p < 0.05); the mean concentrations was 937.152 ± 881.105 pg/ml.

Table 4. Correlation between sclerostin concentration and some factors related to CKD-MBD on HD patients

The data showed that serum sclerostin was skewed and should be logarithmically transformed before analysis

Characteristics		Log_Sclerostin
Calcium	**r**	-0.059
	p	0.679
Phosphorus	**r**	-0.112
	p	0.394
PTH	**r**	**-0.444**
	p	**0.001**
BMD femoral neck	**r**	**-0.375**
	p	**0.007**
BMD total hip	**r**	**-0.304**
	p	**0.030**
BMD lumbar spine (L1-L4)	**r**	-0.261
	p	0.064

Serum sclerostin concentrations in hemodialysis patients were significantly negatively correlated with serum PTH and bone mineral density at the femoral neck and total hip (p < 0.05).

4. DICUSSION

Our study included a total of 111 participants, consisting of 51 patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis for more than three months and 60 healthy controls with normal renal function. The control group comprised 25 females and 35 males, with a mean age of 49.27 ± 10.98, while the G5HD patient group had a mean age of 47.92 ± 14.88. There was no significant difference in age between the control and CKD groups (p > 0.05). The control group demonstrated significantly different characteristics in terms of systolic blood pressure (SBP), diastolic blood pressure (DBP), prevalence of hypertension, urea level, creatinine concentration, and estimated glomerular filtration rate (eGFR) compared with the hemodialysis CKD group (p < 0.05).

In our study, calcium–phosphate–PTH disorders were common complications among patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis. Specifically, regarding calcium abnormalities, 11.76% of HD patients presented with hypocalcemia, while nearly one-fifth had hypercalcemia. Disorders of calcium metabolism represent one of the major features of CKD-related mineral and bone disorder (CKD-MBD). In CKD, abnormal serum calcium levels may manifest as either hypocalcemia or hypercalcemia, both of which have adverse clinical implications. In our cohort of dialysis patients, the mean serum calcium level was 2.33 ± 0.22 mmol/L, which is higher than the mean calcium level reported by Emad in hemodialysis patients (8.8 ± 0.9 mg/dL, equivalent to 2.2 ± 0.225 mmol/L) and that reported by Jing-Wun Lu (8.96 ± 0.73 mg/dL, equivalent to 2.24 ± 0.183 mmol/L) [3], [5]. This difference may be attributable to variations in study design, patient characteristics (such as disease stage, dietary intake, treatment adherence, and duration of dialysis), and measurement standards among studies. Regarding serum phosphate, 86.27% of patients exhibited hyperphosphatemia. The mechanism underlying phosphate imbalance in CKD involves impaired intestinal absorption and reduced renal phosphate excretion. As glomerular filtration rate declines, phosphate accumulation occurs, triggering a cascade of direct and indirect effects. Clinically, hyperphosphatemia in CKD is often accompanied by abnormalities in serum calcium and parathyroid hormone (PTH) levels, both of which are routinely measured to monitor mineral and bone disorders (MBD) in CKD. In our study, the mean serum phosphate level in dialysis patients was 2.03

± 0.59 mmol/L, similar to the finding of Nguyen Huu Vu Quang's domestic study (2.03 ± 0.543 mmol/L) [8]. Emad's study reported a mean phosphate concentration of 5.7 ± 1.1 mg/dL (equivalent to 1.839 ± 0.355 mmol/L), while Jing-Wun Lu's study found an average of 4.49 ± 1.23 mg/dL (equivalent to 1.448 ± 0.397 mmol/L), which was lower than the level observed in our study [3], [5]. In our study, the mean serum PTH concentration among patients undergoing maintenance hemodialysis was 711.32 ± 905.62 pg/mL, with more than 90% of patients presenting with elevated PTH levels. Compared with both domestic and international studies above, the PTH concentration in our cohort was higher [3], [5], [8]. This difference may be explained by variations in dialysis duration, treatment approaches, and the degree of control of mineral and bone disorders among hemodialysis patients.

Regarding bone mineral density in the G5HD group, the mean BMD at the femoral neck, total femur, and lumbar spine were 0.820 ± 0.221; 0.840 ± 0.194 and 1.003 ± 0.194 g/cm^2, respectively. In patients undergoing maintenance hemodialysis, evidence indicates a higher prevalence of CKD-MBD, characterized by disturbances in mineral metabolism and abnormalities in bone quantity and quality. These alterations contribute to an increased risk of fractures and reduced long-term survival [9]. Therefore, the 2017 KDIGO guidelines recommend the assessment of bone mineral density (BMD) using dual-energy X-ray absorptiometry (DEXA) to evaluate fracture risk in this population [1].

In our study, the mean serum sclerostin concentration in the control group of our study was 359.52 ± 168.21 pg/mL. The G5HD groups exhibited significantly higher serum sclerostin level compared to the control group ($p < 0.05$); the mean concentrations was 937.152 ± 881.105 pg/ml. Cejka et al. were the first authors to report elevated serum sclerostin levels in patients undergoing maintenance hemodialysis. The study was conducted on 76 hemodialysis patients and 45 healthy individuals as controls. The results demonstrated that serum sclerostin levels were significantly higher in hemodialysis patients compared with the control group (1257 pg/mL vs. 415 pg/mL, $p < 0.001$) [10]. The study by Emad Abdallah reported that the mean serum sclerostin level in 70 hemodialysis patients was 156.8 ± 121 pmol/L, which was significantly higher than that of the control group of 25 healthy individuals, whose mean serum sclerostin level was 29.38 ± 0.84 pmol/L ($p = 0.0001$) [3]. Similarly, the study by Jing-Wun Lu reported that the serum sclerostin level in 75 hemodialysis patients was 153.32 (92.11 - 207.50) pmol/L, which was significantly higher than that of the control group consisting of 65 individuals, whose serum sclerostin level was 63.37 (40.52 - 83.27) pmol/L ($p < 0.001$) [5]. Pierre Delanaye reported that the mean serum sclerostin level in 164 hemodialysis patients was 1375 pg/mL, which was significantly higher than that of the control group, with a mean level of 565 pg/mL ($p < 0.0001$) [4]. Several other studies investigating serum sclerostin levels in hemodialysis patients did not include a control group. For instance, the study by Liesbeth Viaene et al. conducted on 100 hemodialysis patients reported a median serum sclerostin level of 110 (82 - 151) pmol/L [11]. The study by Eiji Ishimura conducted on 181 HD patients reported a mean serum sclerostin level of 125 ± 53 pmol/L [12].

About the correlation between sclerostin concentration and some factors related to CKD-MBD on HD patients, our study demonstrated that serum sclerostin concentrations were significantly negatively correlated with serum PTH and bone mineral density at the femoral neck and total femur ($p < 0.05$). Firstly, in our study, serum sclerostin levels in patients undergoing maintenance hemodialysis showed a negative correlation with serum PTH levels ($r = –0.444$, $p = 0.001$). PTH and sclerostin are tightly interconnected in the regulation of bone metabolism, and their relationship is especially important in CKD, including patients on HD. SOST was demonstrated to be a target gene for PTH in bone [13]. Data from several international studies have demonstrated similar findings with our study. The study by Emad Abdallah demonstrated a statistically significant negative correlation between serum sclerostin levels and iPTH ($r = –0.362$, 95% CI: –0.550 to –0.139, $p = 0.0021$). Likewise, studies by Delanaye and Y Asamiya also reported an inverse correlation between serum sclerostin and PTH levels in patients undergoing maintenance hemodialysis [4], [14]. In Yosuke Nakagawa's research, higher intact PTH was associated with lower sclerostin ($\beta = −0.08$; 95% CI, −0.11 to −0.06; $P < 0.001$) [6]. This association was qualitatively unchanged after multivariate adjustment ($\beta = −0.09$; 95% CI, −0.11 to −0.06; $P < 0.001$) [6].

Besides, our study showed that there was a significantly negative correlation between serum sclerostin levels and BMD at femoral neck and total hip ($p < 0.05$). A similar inverse correlation was also observed between serum sclerostin levels and lumbar spine BMD; however, this association was

not statistically significant (p>0.05). These findings are consistent with the hypothesis that, as would be expected of a negative regulator of bone formation, higher serum sclerostin levels promote low bone turnover, which leads to loss of bone mass over time. Our study showed results consistent with several international studies conducted on patients undergoing maintenance hemodialysis. The study by Emad Abdallah reported a femoral neck BMD of 0.839 ± 0.086 g/cm², which was negatively correlated with serum sclerostin levels (r = -0.469, 95% CI: -0.508 to -0.0298, p = 0.0278) [3]. Similarly, according to Manal Abd Elsalam et al., in a study investigating the correlation between serum sclerostin levels and bone mineral density (BMD) in children undergoing maintenance hemodialysis, the results showed a significant increase in serum sclerostin levels in the patient group with low BMD (2.38 ± 0.85 ng/mL) compared with those with normal BMD (1.4 ± 0.98 ng/mL) (p = 0.001) [15]. This study also concluded that sclerostin was 100% specific and sensitive in predicting CKD–MBD. Elevated serum sclerostin levels were associated with low BMD and appeared to be an independent predictor of reduced BMD in children on regular HD [15]. However, some studies have shown that sclerostin concentrations are positively related to BMD in HD patients. For example, in Yosuke Nakagawa's study, among 217 patients with metacarpal BMD measured by radiographic absorptiometry, higher sclerostin was strongly associated with higher metacarpal BMD [6]. Similarly, from Ishimura E's study, in multiple regression analysis, serum sclerostin was associated significantly and independently with BMD of both parts of the radius (β = 0.200, p < 0.001; β = 0.218, p < 0.05), after adjustment for age, hemodialysis duration, and bone metabolism markers [12]. These findings appear contradictory to the fact that sclerostin inhibits bone formation [16]. These authors also have no clear explanation for this, but given that sclerostin is secreted by osteocytes, there is a possibility that elevated serum levels of sclerostin may reflect the total number of osteocytes in bone and thus bone mass [6].

The limitations of this study were, first, that it was cross-sectional with a limited number of HD patients. Second, we did not collect the data of physical activities of HD patients, which could influence BMD, calcium-phosphorus disorders as well as the production of Sclerostin. Thirdly, we could not examine the expession of Sclerostin in bone. These limitations may limit the generalizability of our findings.

5. CONCLUSIONS

In conclusion, according to our study, CKD-MBD are common in patients with end-stage renal disease undergoing maintenance hemodialysis. Serum sclerostin levels were significantly higher in the hemodialysis group compared to healthy controls. Regarding the association between sclerostin and mineral-bone disorders as well as bone mineral density in hemodialysis patients, our findings demonstrate that serum sclerostin levels were significantly and inversely correlated with PTH levels and bone mineral density at the femoral neck and total hip.

REFERENCES

1. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD). Kidney international supplements. 2017;7(1):1-59.

2. ten Dijke P, Krause C, de Gorter DJ, Löwik CW, van Bezooijen RL. Osteocyte-derived sclerostin inhibits bone formation: its role in bone morphogenetic protein and Wnt signaling. The Journal of bone and joint surgery American volume. 2008;90 Suppl 1:31-5.

3. Abdallah E, Sherif N, Mosbah O, Metwally A, Abd ElAzim I, Mahmoud O, et al. The Relationship between Serum Sclerostin Levels and Bone Mineral Disorders and Vascular Calcification in Hemodialysis Patients. Open Access Macedonian Journal of Medical Sciences. 2021;9(B):1664-71.

4. Delanaye P, Krzesinski JM, Warling X, Moonen M, Smelten N, Médart L, et al. Clinical and biological determinants of sclerostin plasma concentration in hemodialysis patients. Nephron Clinical practice. 2014;128(1-2):127-34.

5. Lu JW, Syu RJ, Wang CH, Hsu BG, Tsai JP. Serum Sclerostin Level Is Negatively Associated with Bone Mineral Density in Hemodialysis Patients. Medicina (Kaunas, Lithuania). 2022;58(3).

6. Nakagawa Y, Komaba H, Wada T, Takahashi H, Takahashi Y, Hyodo T, et al. Serum Sclerostin Levels and Mortality in Hemodialysis Patients: An 8-Year Prospective Study. American journal of nephrology. 2022;53(11-12):767-74.

7. Stevens PE, Levin A. Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline. Annals of internal medicine. 2013;158(11):825-30.

8. Nguyễn Hữu Vũ Quang. Nghiên cứu nồng độ FGF-23 huyết thanh và mối liên quan với một số rối loạn khoáng xương ở bệnh nhân bệnh thận mạn [Luận án tiến sĩ y học]: Đại học Y-Dược, Đại học Huế; 2020.

9. Slouma M, Sahli H, Bahlous A, Laadhar L, Smaoui W, Rekik S, et al. Mineral bone disorder and osteoporosis in hemodialysis patients. Advances in rheumatology (London, England). 2020;60(1):15.

10. Cejka D, Jäger-Lansky A, Kieweg H, Weber M,

Bieglmayer C, Haider DG, et al. Sclerostin serum levels correlate positively with bone mineral density and microarchitecture in haemodialysis patients. Nephrology Dialysis Transplantation. 2011;27(1):226-30.

11. Viaene L, Behets GJ, Claes K, Meijers B, Blocki F, Brandenburg V, et al. Sclerostin: another bone-related protein related to all-cause mortality in haemodialysis? Nephrology Dialysis Transplantation. 2013;28(12):3024-30.

12. Ishimura E, Okuno S, Ichii M, Norimine K, Yamakawa T, Shoji S, et al. Relationship Between Serum Sclerostin, Bone Metabolism Markers, and Bone Mineral Density in Maintenance Hemodialysis Patients. The Journal of Clinical Endocrinology & Metabolism. 2014;99(11):4315-20.

13. Keller H, Kneissel M. SOST is a target gene for PTH in bone. Bone. 2005;37(2):148-58.

14. Asamiya Y, Yajima A, Shimizu S, Otsubo S, Tsuchiya K, Nitta K. Associations between the levels of sclerostin, phosphate, and fibroblast growth factor-23 and treatment with vitamin D in hemodialysis patients with low intact PTH level. Osteoporosis International. 2014;26.

15. Elsalam MA, El-Abden MZ, Mahmoud E, Zahab ZA, Ahmed H. Correlation between serum sclerostin level and bone density status in children on regular hemodialysis. Saudi journal of kidney diseases and transplantation : an official publication of the Saudi Center for Organ Transplantation, Saudi Arabia. 2019;30(5):1022-31.

16. Bellido T, Ali AA, Gubrij I, Plotkin LI, Fu Q, O'Brien CA, et al. Chronic elevation of parathyroid hormone in mice reduces expression of sclerostin by osteocytes: a novel mechanism for hormonal control of osteoblastogenesis. Endocrinology. 2005;146(11):4577-83.