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HUBUNGAN ANTARA DURASI HEMODIALISIS DAN BONE MINERAL DISEASE BERDASARKAN PARAMETER BIOKIMIA PADA PASIEN PENYAKIT GINJAL KRONIS DI RSUP ADAM MALIK MEDAN

Angga Adityawan[1], Alwi Thamrin Nasution[2], Sumi Ramadhani[3]
anggaadityawan931@gmail.com[1], alwi_thamrin@yahoo.com[2], space_pirate@rocketmail.com[3]
Universitas Sumatera Utara

ABSTRAK

Pendahuluan: Pendahuluan: Penyakit ginjal kronis (PGK) ditandai dengan penurunan fungsi ginjal yang bersifat progresif. Hemodialisis umum digunakan sebagai terapi pengganti ginjal pada pasien dengan penyakit ginjal tahap akhir. Chronic kidney disease–mineral and bone disorder (CKD-MBD) sering terjadi akibat kelainan metabolisme kalsium dan fosfat. Penelitian ini bertujuan untuk mengevaluasi hubungan antara durasi hemodialisis dan bone mineral disease yang dinilai melalui kadar kalsium dan fosfat serum pada pasien PGK di Rumah Sakit Umum Pusat Adam Malik Medan. Metode: Penelitian analitik dengan desain potong lintang ini melibatkan 62 pasien PGK yang menjalani hemodialisis rutin dan direkrut menggunakan metode consecutive sampling. Data demografi dan laboratorium diperoleh dari rekam medis pasien. Kadar kalsium dan fosfat serum dianalisis sebagai indikator metabolisme mineral. Analisis statistik dilakukan menggunakan SPSS versi 24.0 dengan uji t independen atau uji Mann–Whitney U, dengan nilai $p<0,05$ dianggap bermakna secara statistik. Hasil: Dari 62 pasien, 42 adalah laki-laki (67,7%) dan 20 perempuan (32,3%) dengan rata-rata usia 48 tahun. Sebanyak 38 pasien (61,3%) menjalani hemodialisis lebih dari satu tahun dan 24 pasien (38,7%) kurang dari satu tahun. Durasi hemodialisis berhubungan dengan kadar fosfat serum (7,4 vs 6,25 mg/dL; $p=0,002$) dan kalsium (8,54 vs 8,32 mg/dL; $p=0,003$). Kesimpulan: Durasi hemodialisis berhubungan secara signifikan dengan kadar kalsium dan fosfat serum pada pasien PGK.
Kata Kunci: Penyakit Ginjal Kronis, Durasi Hemodialisis, Kalsium, Fosfat, CKD-MBD.

ABSTRACT

Introduction: Chronic kidney disease (CKD) is characterized by progressive loss of kidney function. Hemodialysis is commonly used for patients with end-stage renal disease. Chronic kidney disease–mineral and bone disorder (CKD-MBD) frequently occurs due to abnormalities in calcium and phosphate metabolism. This study evaluated the association between hemodialysis duration and bone mineral disease assessed using serum calcium and phosphate levels in CKD patients at Adam Malik General Hospital Medan. Methods: This analytical cross-sectional study included 62 CKD patients undergoing routine hemodialysis recruited using consecutive sampling. Demographic and laboratory data were obtained from medical records. Serum calcium and phosphate levels were analyzed as indicators of mineral metabolism. Statistical analysis was performed using SPSS version 24.0 with the independent t-test or Mann–Whitney U test and $p<0.05$ considered significant. Results: Of 62 patients, 42 were male (67.7%) and 20 female (32.3%) with a mean age of 48 years. Thirty-eight patients (61.3%) had hemodialysis >1 year and 24 (38.7%) <1 year. Hemodialysis duration was associated with serum phosphate (7.4 vs 6.25 mg/dL; $p=0.002$) and calcium (8.54 vs 8.32 mg/dL; $p=0.003$). Conclusion: Hemodialysis duration is significantly associated with serum calcium and phosphate levels in CKD patients.
***Keywords**: Chronic Kidney Disease, Hemodialysis Duration, Calcium, Phosphate, CKD-MBD.*

INTRODUCTION

Chronic kidney disease (CKD) is a heterogeneous group of diseases caused by multiple risk factors and comorbid conditions, with a rapidly increasing prevalence and incidence. Although CKD patients share a similar pathophysiology associated with the development of kidney disease, the course and rate of CKD progression and associated complications vary depending on the underlying cause.1 In high- and middle-income countries, the leading causes of CKD are diabetes and high blood pressure. According to the World Health Organization (WHO), in 2012, this disease was estimated to cause between 1% and 5% of deaths worldwide, and according to projections from the Global Health Observatory, it will cause 14 out of 100,000 deaths by 2030. The prevalence and incidence of CKD in high-income countries is approximately 11% and varies globally, ranging from 8–16% in the general population. Those aged 65 and over are the group with the highest prevalence, with data ranging from 23.4 to 35.8%.2 The elderly population, including centenarians, has become the fastest-growing segment of the global population. This growth has resulted in a significant social and economic burden due to chronic kidney disease (CKD), which is most common in the elderly. Therefore, prevention and treatment of CKD are crucial and urgent in elderly patients.3 Once diagnosed, clinical management of CKD requires a multifaceted and dynamic approach. Lifestyle modifications, including dietary adjustments and physical activity, are crucial in disease management. High blood pressure management, renin-angiotensin-aldosterone system (RAAS) blockade, which generally includes angiotensin-converting enzyme-1 (ACE-1) and angiotensin receptor blockers (ARBs) for high blood pressure and albuminuria, glycemic management, and reversal of metabolic acidosis are four treatments that can significantly delay the progression of CKD. Furthermore, recent advances in renal replacement therapy have substantially improved the standard of living and survival for patients with end-stage renal disease.4

The treatment of chronic kidney disease is divided into two phases: conservative management and renal replacement therapy. The most commonly administered renal replacement therapy is hemodialysis, followed by continuous ambulatory peritoneal dialysis.5 Hemodialysis is the most popular and effective treatment for patients with end-stage renal disease. There has been a 35% increase in the number of dialysis patients worldwide. In Saudi Arabia, the average annual net growth rate of hemodialysis patients is 6%. In 2021, there were more than 20,000 patients undergoing dialysis and 9,810 patients undergoing follow-up after kidney transplantation. The total prevalence of renal replacement therapy in Saudi Arabia was estimated at 294.3 per million people.6 However, hemodialysis patients currently experience an excess risk of cardiovascular mortality and morbidity that is not fully explained by more common and traditional cardiovascular risk factors. Numerous observational analyses suggest that disorders of bone and mineral metabolism, collectively known as chronic kidney disease-bone and mineral disorders (CKD-BMD), are involved in the pathogenesis of this higher risk.7 In CKD, reduced glomerular filtration rate (which limits phosphate excretion), decreased calcitriol synthesis, and various dietary restrictions affect phosphocalcic homeostasis, including secondary hyperparathyroidism and renal osteodystrophy. Mineral and bone disorders associated with chronic kidney disease should be screened for and monitored (laboratory testing, bone mineral density assessment, bone markers, and vascular calcification assessment). Correction of biological abnormalities is necessary, with dietary guidance (reduction of phosphate intake, normalization of calcium intake) and therapeutic assistance (vitamin D derivatives, phosphate chelation, calcimimetics).8 As kidney function declines, mineral metabolism disorders gradually develop. CKD-GMT is often recognized by abnormal serum biochemical parameters. As the stages of chronic kidney disease progress, sequential changes in serum biochemical

parameters appear. Briefly, circulating α-klotho levels decrease first, followed by increased serum fibroblast growth factor 23 (FGF23) levels, and decreased serum calcitriol levels in CKD stages 2 and 3. Serum parathyroid hormone (PTH) levels increase, followed by increased serum phosphate levels and decreased serum calcium levels. Bone and mineral metabolism disorders in patients with CKD were previously thought to be diseases of the bone and parathyroid glands. Abnormal serum phosphate, calcium, and PTH levels are associated with an increased risk of morbidity and mortality in hemodialysis patients. Several lines of evidence have confirmed that hyperphosphatemia is closely linked to increased cardiovascular events and mortality.4

Since its inception, hemodialysis has been continuously refined to provide better patient outcomes. The primary function of dialysis is to remove uremic toxins, maintain electrolyte balance, and restore fluid balance without compromising other vital metabolic, endocrine, and immune functions.10 A 2012 study by Daugirdas et al. confirmed previous findings that routine hemodialysis, delivered daily as 1.5- to 2.75-hour sessions or nightly as 6- to 8-hour sessions, results in better serum phosphorus control. In patients with end-stage chronic kidney disease, hemodialysis is the primary renal replacement therapy to maintain fluid and electrolyte balance. However, as hemodialysis therapy progresses, changes in mineral homeostasis occur, including an increase in serum phosphate levels. This is due to the accumulation of phosphate that is not fully eliminated during dialysis, accompanied by increased phosphate release from bone and tissue due to the impaired calcium-phosphate metabolism that accompanies chronic kidney disease. Consequently, patients on longer hemodialysis sessions tend to have higher phosphate levels than those who have recently started therapy. As hemodialysis duration increases, metabolic adaptations and changes in bone mineral homeostasis become more significant. Phosphate, which should be partially removed through dialysis, is not completely eliminated due to limited dialysis time and efficiency. Furthermore, increased bone turnover due to parathyroid hormone disorders can also contribute to the release of phosphate into the circulation. Hyperphosphatemia is common in CKD and advanced end-stage renal disease and has been associated with bone disease and increased cardiovascular morbidity and mortality. Bone involvement is also found in cases of CKD, known as renal osteodystrophy, which is characterized by histological changes in bone including abnormalities in turnover (high/low), mineralization, and volume, leading to abnormal cortical bone structure and quality, negatively impacting bone strength.12

CKD-GMT causes a decrease in the number of osteocytes and abnormal mineralization by the remaining osteocytes, leading to widespread bone hypomineralization, which increases bone fragility and leads to an increased risk of fracture. Proper bone mineralization by the osteocyte population is crucial for increasing bone mineral density. In a population with renal hyperparathyroidism treated with parathyroidectomy and concomitant adequate intake of active vitamin D (alphacalcidiol), osteocyte mineralization is greater than that of osteoblasts. However, in the absence of adequate vitamin D intake, areas of hypomineralization are observed to be higher, highlighting the crucial role of vitamin D in ensuring proper bone mineralization by osteocytes. 12 Today, these traditional methods have been complemented by new biomarkers and imaging technologies that provide deeper insights into the kidney. Innovative tools allow for the identification of CKD at its earliest stages, facilitating timely intervention and personalized treatment strategies. Advances in metabolomics, transcriptomics, and proteomics will lead to the identification of novel biomarkers in kidney disease, enabled by the emergence of new techniques.12 Detecting CKD-GMT in the early stages of CKD is crucial. Monitoring CKD-GMT-related mediators in serum parameters and taking effective measures is also crucial to prevent the progression of CKD-GMT and cardiovascular disease. In this study, researchers reviewed the biomarker parameter values in

cases of PGK-GMT in the dialysis-dependent PGK population.

RESEARCH METHODS

This study was an analytical observational study with a cross-sectional design. The study was conducted at Adam Malik General Hospital Medan. The study population consisted of patients diagnosed with chronic kidney disease who were undergoing routine hemodialysis treatment. Participants were recruited using a consecutive sampling method. Patients who met the inclusion criteria during the study period were included until the required sample size was achieved. A total of 62 patients undergoing hemodialysis were included in this study.

Data Collection: Demographic and clinical data were obtained from medical records, including age, sex, body weight, height, and laboratory results. Laboratory parameters evaluated in this study included hemoglobin, urea, creatinine, glomerular filtration rate (GFR), serum calcium, and serum phosphate levels. The duration of hemodialysis was categorized into two groups: patients who had undergone hemodialysis for less than one year and those who had undergone hemodialysis for more than one year.

Statistical Analysis: Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 24.0. Descriptive statistics were used to summarize demographic and clinical characteristics. Normality testing was conducted using the Kolmogorov–Smirnov test. Comparisons between groups were performed using the independent t-test for normally distributed data or the Mann–Whitney U test for non-normally distributed data. The Chi-square test or Fisher's exact test was used to analyze categorical variables. A p-value of less than 0.05 was considered statistically significant.

Ethical Consideration: Ethical approval for this study was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara (No. 866/KEPK/USU/2025) & Adam Malik General Hospital, Medan (No. DP.04.03/D.XXVIII.2.2.3/1436/2025).

RESULT AND DISCUSSION

The characteristics of the study subjects are presented in Table 1. A total of 62 patients with chronic kidney disease undergoing hemodialysis were included in this study. The majority of the study subjects were male, accounting for 42 individuals (67.7%), while 20 subjects (32.3%) were female. The mean age of the participants was 48 years. The average body weight was 58 kg and the mean height was 161 cm. Based on routine laboratory examinations, the average hemoglobin level was 8.18 g/dL. The median phosphate level was 6.6 mg/dL and the median calcium level was 8.4 mg/dL. Among the study population, 38 patients (61.3%) had undergone hemodialysis for more than one year, while 24 patients (38.7%) had undergone hemodialysis for less than one year. The mean urea level was 150.84 mg/dL and the mean creatinine level was 16.5 mg/dL. The average estimated glomerular filtration rate (GFR) was 5.06 mL/min/1.73 m^2.

Analysis of biochemical parameters demonstrated a statistically significant relationship between the duration of hemodialysis and serum phosphate levels. Patients who had undergone hemodialysis for more than one year had higher phosphate levels compared with those undergoing hemodialysis for less than one year (7.4 mg/dL vs. 6.25 mg/dL, $p = 0.002$). Similarly, a statistically significant association was observed between the duration of hemodialysis and serum calcium levels. Patients undergoing hemodialysis for more than one year had higher serum calcium levels compared with those undergoing hemodialysis for less than one year (8.54 mg/dL vs. 8.32 mg/dL, $p = 0.003$) (Table 2).

Table 1. Characteristics of Study Subjects

Parameter	**n = 62 (100%)**
Gender, n (%)	
Male	42 (67.7)
Female	20 (32.3)
Age (years)	48.07 ± 11.61
Body weight (kg)	58.63 ± 3.90
Height (cm)	161.17 ± 4.33
Hemoglobin (g/dL)	8.18 ± 1.66
Phosphate (mg/dL)	6.6 (3.1–11.1)
Calcium (mg/dL)	8.46 (5.54–13.2)
Urea (mg/dL)	150.84 ± 49.89
Creatinine (mg/dL)	16.5 ± 27.3
GFR (mL/min/1.73m²)	5.06 ± 3.14
Duration of HD, n (%)	
>1 year	38 (61.3)
<1 year	24 (38.7)

Table 2. Relationship Between Duration of Hemodialysis and Biochemical Parameters of Bone Mineral Disease in Chronic Kidney Disease Patients

Duration of Hemodialysis	**>1 year**	**<1 year**	**p-value**
Phosphate level (mg/dL)	7.4 (3.2–11.1)	6.25 (3.1–11.1)	0.002*
Calcium level (mg/dL)	8.54 (6.3–13.2)	8.32 (5.54–10.1)	0.003*

Mann-Whitney test, median (min–max), p-value significant if <0.05.

The results of this study analysis found a statistically significant relationship between the duration of hemodialysis and phosphate levels, where the longer the hemodialysis period (>1 year) the higher the phosphate levels. In hemodialysis patients, bone fragility fractures are a common adverse event and are associated with increased morbidity and mortality. In a study by Barrera et al. (2024) in Spain, in addition to the classic risk factors already known to cause bone fractures, high serum phosphate levels were consistently shown to be associated with an increased risk of fracture, regardless of other factors such as previous fracture history, age, gender, duration of hemodialysis, serum calcium levels, or PTH levels. It was found that patients who experienced at least one fracture during the follow-up period showed a longer hemodialysis time.13 The study concluded that elevated serum phosphate levels were independently and consistently associated with an increased risk of bone fragility fractures in hemodialysis patients, indicating that serum phosphate is a new risk factor or marker for bone fractures in this population.13,14 Another study by Abdu et al. (2019) in Nigeria, a study of 48 CKD patients undergoing maintenance hemodialysis reported that hyperphosphatemia was found in 19 (39.5%) patients, while 22 (46%) patients experienced hypocalcemia. Overall, 58% of these patients had CKD-GMT.15

CKD-GMT is generally recognized when patients exhibit abnormalities in serum mineral concentrations. In CKD patients before hemodialysis, elevated serum levels of FGF23, PTH, and phosphate, as well as decreased levels of calcitriol, calcium, and α-klotho, are often found. In patients already undergoing hemodialysis, conditions such as hypercalcemia, hyperphosphatemia, and elevated serum levels of PTH and FGF23 are also common. Observational studies have also consistently shown that abnormalities in serum CKD-GMT mediators are associated with an increased risk of morbidity and mortality, both in predialysis and hemodialysis-dependent CKD.16,17 Chronic kidney disease (CKD) is a disease with increasing incidence and prevalence, affecting more than 10% of the general population. As kidney function declines, complex disorders affecting bone, mineral metabolism, and the cardiovascular system develop, termed CKD-bone-mineral disorders (CKD-MBD). Furthermore, progressively increasing phosphate levels in advanced stages of

CKD lead to overactive parathyroid glands, termed secondary hyperparathyroidism (SHPT), which is typically characterized by hyperphosphatemia, increased fibroblast growth factor-23 (FGF-23), decreased 1,25-dihydroxyvitamin D3 [1,25(OH)2D3], and high parathyroid hormone (PTH) levels.18

Hyperphosphatemia (elevated serum phosphate levels) is a common and serious problem in patients with end-stage renal disease (ESRD) undergoing dialysis. Although various phosphate management strategies, such as dietary phosphate restriction, hemodialysis therapy, and phosphate binders, have been developed, they are still insufficient to maintain phosphate levels within the normal range. The typical phosphate intake in a modern Western diet is 1,400 mg/day. Furthermore, phosphate additives can contribute an additional 1,000 mg/day. Thus, the daily phosphate intake in a typical Western diet can approach 2,400 mg/day, more than 300% of the recommended daily intake of 700 mg. Therefore, the maximum amount of phosphate that can be removed through dialysis or bound by binders is limited. The current inability to consistently achieve recommended target phosphorus levels in most patients on dialysis highlights the need for innovation in phosphate management.19 Chronic hemodialysis as currently practiced in the United States does not remove sufficient phosphate to control serum levels according to current guidelines. Conventional hemodialysis does not remove enough phosphate to maintain phosphorus balance in most hemodialysis patients. A 4-hour hemodialysis session removes 34 mmol of phosphate (1054 mg of phosphorus), which is insufficient to meet the typical phosphorus intake of 800 to 2000 mg per day (equivalent to 25.8–64.5 mmol of phosphate) in a Western diet. In the study by Steven et al., the recommended protocol required 3-hour hemodialysis sessions, 6 times per week. This short daily hemodialysis (SDHD) regimen maximizes phosphate removal by increasing total dialysis time. A significant decrease in serum phosphorus levels was observed in patients treated with SDHD (6.3 ± 2.57 mg/dL at baseline, 4.61 ± 0.6 mg/dL at 6 months of treatment, and 4.0 ± 1.19 mg/dL at 12 months of dialysis, $P < 0.004$), which was not observed in patients undergoing conventional hemodialysis.18 Decreased extracellular phosphate concentrations play a crucial role in apoptosis in mature chondrocytes in the growth plate, as well as in the cascade of events leading to normal bone growth, such as blood vessel invasion and mineralization. Abnormal serum phosphate levels are a key component of CKD-GMT. As CKD progresses, circulating phosphate levels gradually increase and contribute, directly and indirectly, to CKD-GMT-associated bone fragility, in part through stimulation of PTH and FGF23 production. Serum FGF23 levels increase significantly in the early stages of CKD and coincide with a decrease in $1,25(OH)_2D$ levels. FGF23 is primarily produced by osteocytes and osteoblasts and performs its primary physiological function in the kidney, stimulating urinary phosphate excretion and inhibiting calcitriol synthesis after binding to a complex formed by alpha-klotho and the canonical FGF receptor.19,20 Furthermore, a study by Lee et al. (2019) in the United States of CKD patients undergoing conventional hemodialysis with dialysis treatment for at least 60 consecutive days reported that higher serum phosphorus levels were associated with a greater decline in residual kidney function (RESF) within 6 months of starting hemodialysis. Furthermore, higher levels of intact PTH and ALP, as well as lower serum calcium levels, were associated with a greater decline in REF.19 The findings regarding the relationship between hyperphosphatemia and poor renal outcomes in hemodialysis patients are also in line with previous studies in animals and in predialysis CKD patients.21

The analysis of this study also found a statistically significant relationship between the duration of hemodialysis and calcium levels, with the longer the hemodialysis period (>1 year) increasing the calcium levels. In CKD, major disturbances occur in the homeostasis of calcium, phosphate, parathormone (PTH), FGF23/klotho, and the vitamin D hormonal system (calcidiol and calcitriol). These changes significantly impact bone and vascular metabolism,

resulting in serious clinical consequences such as decreased bone mass, increased risk of fragility fractures, and progression of vascular and valvular calcification.21 Therefore, in patients with CKD stages 4–5, prior to administering specific antiosteoporotic therapy, the first step is to ensure adequate control of serum calcium levels and correct vitamin D reserve deficiencies.22-24 In CKD, major changes occur in the regulation of calcium, phosphate, parathormone (PTH), FGF23/Klotho, and the vitamin D hormonal system (calcidiol and calcitriol). These changes have a significant impact on bone and vascular metabolism, with serious clinical consequences such as decreased bone mass, increased risk of fragility fractures, and vascular and valvular calcification. Disruptions in the regulatory axis of bone and mineral metabolism affect bone modeling during growth and bone remodeling in adulthood. These progressive changes, known as CKD-GMT, begin in the early stages of CKD and peak in stages 4–5. Vascular and valvular calcification resulting from dysregulated bone-mineral metabolism accelerates the aging process of the cardiovascular system. In addition to the disease itself, CKD-GMT can also be facilitated or exacerbated by inappropriate therapy. Two important factors with significant detrimental effects are excessive calcium use and vitamin D receptor activator (VDRA). Both can disproportionately increase serum calcium levels, leading to excessive suppression of PTH and ultimately reducing bone remodeling activity.21

As kidney function progressively declines, the complex and interconnected mechanisms that regulate mineral metabolism fail to maintain balance. Despite increases in FGF23 and PTH, serum phosphate levels remain elevated, while calcitriol levels decrease. In advanced stages of CKD-GMT, this condition worsens. The decreased calcium and calcitriol levels, combined with the increased phosphate levels, induce a persistent proliferative stimulus in the parathyroid glands. Consequently, a severe form of secondary hyperparathyroidism develops with parathyroid gland hyperplasia, initially diffuse and later nodular. This process is accompanied by a significant decrease in the expression of the calcium-sensing receptor (CaSR), vitamin D receptor (VDR), and FGFR/Klotho. The decreased expression of these receptors leads to parathyroid gland resistance to calcium, vitamin D receptor activator (VDRA), and FGF23, severely limiting the response to therapy.22 Several observational studies have demonstrated an association between CKD marker abnormalities—GMT—and poor clinical outcomes, both in pre-dialysis and dialysis patients.24,25 For example, elevated phosphate, calcium, and PTH levels have been shown to be associated with increased cardiovascular-specific mortality in CKD patients. In a large, multicenter, prospective cohort study (the Netherlands Cooperative Study on the Adequacy of Dialysis) involving 1,629 hemodialysis and peritoneal dialysis patients, an increased hazard ratio (HR) of 1.57 (1.07–2.30) was reported in patients in the highest quartile of phosphate levels, based on both baseline and time-dependent values.26 Similarly, a study by Block et al. in the United States reported an increased risk of death with increasing phosphate levels, with relative risks (RR) of 1.07, 1.25, 1.43, 1.67, and 1.70, respectively. and 2.02 for serum phosphorus levels of 5.0–6.0; 6.0–7.0; 7.0–8.0; 8.0–9.0; and >9.0 mg/dL, respectively, in 40,538 maintenance hemodialysis patients. This consistent association between hyperphosphatemia and increased mortality is related to the direct calcification effect on coronary vessels and heart valves.27,28 It has also been reported that the duration (in years) of hemodialysis has a positive association with the occurrence of vascular calcification, especially in medium-caliber arteries, where it is estimated that each additional year of dialysis increases the risk of vascular calcification by approximately 15%.22

In addition, Pimentel et al. (2021) in France also reported several factors that play a role in increasing the risk of fractures related (or indirectly related) to CKD, including age, gender (with a higher risk in women), history of previous hip fracture, urine albumin levels, low body mass index, duration of dialysis (dialysis vintage), and the presence of high-turnover

bone disease (HBD) or low-turnover bone disease (LOW-TURNOVER BONE DISEASE).29 Overall, the results of this study are in line with recent research emphasizing that the duration of hemodialysis is an important factor influencing the progression of biochemical abnormalities in CKD-GMT. Research by Zhan et al. (2019) in Australia showed that patients with longer dialysis durations tended to have higher serum phosphate levels due to the limitations of hemodialysis in eliminating the total phosphate load, primarily due to the phenomenon of phosphate rebound from the intracellular space after dialysis. This condition contributes to the development of chronic hyperphosphatemia, which is a strong predictor of cardiovascular mortality and fractures in patients with end-stage CKD.30 Research by Nitta et al. (2023) in Japan also confirmed that regulation of calcium and phosphate homeostasis is increasingly disrupted with increasing duration of dialysis. Factors such as reduced residual kidney function, the use of calcium-based binders, and long-term active vitamin D therapy can lead to abnormally elevated serum calcium levels. This is associated with adynamic bone disease due to excessive PTH suppression, which is characterized by a decreased capacity of bone to store minerals, thereby exacerbating the risk of vascular calcification.31

Study Limitations: This study has several limitations. First, the cross-sectional design limits the ability to establish a causal relationship between the duration of hemodialysis and biochemical parameters. Second, this study evaluated only calcium and phosphate levels without assessing other biomarkers associated with CKD-MBD such as parathyroid hormone or vitamin D. Third, the sample size was relatively small and derived from a single center, which may limit the generalizability of the findings.

CONCLUSION

The duration of hemodialysis is significantly associated with serum phosphate and calcium levels in patients with chronic kidney disease. Longer dialysis duration (>1 year) is associated with higher phosphate and calcium levels. These findings emphasize the importance of regular monitoring of mineral metabolism in patients undergoing long-term hemodialysis.

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