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The Relationship Between Serum Calcium-Phosphate Product Levels and Pulse Pressure Variability in Patients Undergoing Routine Hemodialysis at Adam Malik Hospital

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ABSTRACT

Background: Chronic kidney disease (CKD) is a major global health problem associated with high morbidity and mortality, primarily due to cardiovascular complications, especially in patients undergoing hemodialysis. Mineral metabolism disorders, reflected by the calcium-phosphate product (Ca × P), may contribute to vascular calcification and increased pulse pressure.

Objective: To analyze the relationship between serum calcium–phosphate product levels and pulse pressure variability in patients undergoing routine hemodialysis at Adam Malik Hospital, Medan.

Methods: This cross-sectional analytical observational study included 60 patients with chronic kidney disease receiving routine hemodialysis. Data were collected through interviews, medical record reviews, physical examinations, and laboratory tests. Serum calcium and phosphate levels were measured before dialysis, and the calcium–phosphate product was calculated by multiplying both values. Pulse pressure was obtained by subtracting diastolic from systolic blood pressure. Statistical analysis was performed using the Spearman rank correlation test, with a significance level of $p < 0.05$.

Results: The mean serum calcium level was 8.24 ± 0.83 mg/dL, and the mean serum phosphate level was 5.45 ± 1.22 mg/dL. The average calcium–phosphate product was 44.93 ± 10.62 mg^2/dL^2, while the mean pulse pressure was 61.35 ± 12.24 mmHg. A weak positive correlation was found between calcium–phosphate product and pulse pressure variability ($r = 0.216$, $p = 0.098$), indicating no statistically significant relationship.

Conclusion: The study found no significant association between serum calcium–phosphate product levels and pulse pressure variability among patients undergoing routine hemodialysis. Although a positive trend was observed, it was not statistically meaningful. These findings suggest that other factors, including hypertension, dialysis duration, and metabolic conditions, may have a greater impact on vascular stiffness. Routine monitoring of calcium, phosphate, and pulse pressure remains essential for early detection and prevention of cardiovascular complications in chronic hemodialysis patients.

Keywords: Chronic Kidney Disease, Hemodialysis, Calcium–Phosphate Product, Pulse Pressure, Vascular Stiffness

ABSTRAK

Latar Belakang: Penyakit ginjal kronik (PGK) merupakan masalah kesehatan global yang utama dan berhubungan dengan angka morbiditas serta mortalitas yang tinggi, terutama akibat komplikasi kardiovaskular. Pada pasien yang menjalani hemodialisis jangka panjang, kekakuan arteri dan kalsifikasi vaskular sering terjadi dan dapat tercermin melalui peningkatan variabilitas pulse pressure. Produk kalsium–fosfat (Ca × P) merupakan indikator penting dari gangguan metabolisme mineral yang dapat berperan dalam proses kalsifikasi vaskular dan perubahan hemodinamik pada pasien hemodialisis.

Tujuan: Menganalisis hubungan antara kadar produk kalsium–fosfat serum dengan variabilitas pulse pressure pada pasien hemodialisis rutin di Rumah Sakit Adam Malik, Medan.

Metode: Penelitian ini merupakan studi analitik observasional dengan desain potong lintang yang melibatkan 60 pasien penyakit ginjal kronik yang menjalani hemodialisis rutin. Data dikumpulkan melalui wawancara, telaah rekam medis, pemeriksaan fisik, serta pemeriksaan laboratorium. Kadar kalsium dan fosfat serum diukur sebelum hemodialisis, kemudian produk kalsium–fosfat dihitung dengan mengalikan kedua nilai tersebut. *Pulse pressure* diperoleh dari selisih tekanan darah sistolik dan diastolik. Analisis statistik dilakukan menggunakan uji korelasi Spearman rank dengan tingkat signifikansi $p < 0,05$.

Hasil: Rerata kadar kalsium serum adalah 8,24 ± 0,83 mg/dL dan rerata kadar fosfat serum adalah 5.45 ± 1.22 mg/dL. Nilai rata-rata produk kalsium–fosfat sebesar 44.93 ± 10.62 mg^2/dL^2, sedangkan rerata pulse pressure adalah 61.35 ± 12.24 mmHg. Hasil analisis menunjukkan adanya korelasi positif yang lemah antara produk kalsium–fosfat dengan variabilitas *pulse pressure* ($r = 0.224$; $p = 0.085$), namun hubungan tersebut tidak bermakna secara statistik.

Kesimpulan: Tidak terdapat hubungan yang bermakna antara kadar produk kalsium–fosfat serum dengan variabilitas *pulse pressure* pada pasien yang menjalani hemodialisis rutin. Meskipun terdapat kecenderungan korelasi positif, hubungan tersebut tidak signifikan secara statistik. Hasil ini menunjukkan bahwa faktor lain seperti hipertensi, lama menjalani hemodialisis, dan kondisi metabolik mungkin memiliki pengaruh yang lebih besar terhadap kekakuan arteri. Pemantauan rutin kadar kalsium, fosfat, dan pulse pressure tetap penting untuk deteksi dini serta pencegahan komplikasi kardiovaskular pada pasien hemodialisis kronik.

Kata kunci: Penyakit Ginjal Kronik, Hemodialisis, Produk Kalsium–Fosfat, *Pulse Pressure*, Kekakuan Arteri

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1. Introduction

Chronic kidney disease (CKD) is a major global health problem associated with increasing morbidity, mortality, and healthcare burden.[1,2] Cardiovascular disease (CVD) is a major cause of death among patients with CKD, particularly those undergoing dialysis. CKD promotes cardiovascular complications through hypertension, fluid overload, endothelial dysfunction, and disturbances in mineral metabolism, which can accelerate vascular calcification, arterial stiffness, and widened pulse pressure.[3] Greater blood pressure variability has also been associated with cardiovascular events and all-cause mortality in patients with CKD, supporting the clinical relevance of blood-pressure-related parameters in cardiovascular risk assessment.[4,5] In Indonesia, the 2018 Basic Health Research survey reported a CKD prevalence of 3.8 per 1,000 population, with the highest prevalence observed in North Kalimantan, Aceh, and West Sumatra.[6] Hemodialysis remains one of the most frequently used kidney replacement therapies for patients with end-stage kidney disease.[7–9]

Despite its role in maintaining fluid, electrolyte, and acid-base balance, hemodialysis does not eliminate the risk of cardiovascular complications.[9] Vascular calcification and arterial stiffness frequently develop in patients undergoing long-term hemodialysis and may contribute to abnormal blood pressure patterns and cardiovascular events.[10–13] Pulse pressure, defined as the difference between systolic and diastolic

blood pressure, reflects the mechanical properties of large arteries and can serve as a simple marker of arterial compliance.[14,15] Increased pulse pressure indicates reduced vascular elasticity and greater arterial stiffness, which are associated with left ventricular hypertrophy, impaired coronary perfusion, cardiovascular morbidity, and mortality in patients with end-stage kidney disease.[16–18]

Vascular calcification in CKD is closely associated with disturbances in calcium-phosphate metabolism, elevated parathyroid hormone levels, and chronic inflammation.[19,20] As kidney function declines, phosphate excretion decreases, resulting in phosphate retention, changes in serum calcium levels, and secondary hyperparathyroidism.[21,22] These abnormalities promote calcium and phosphate deposition in the arterial wall, leading to reduced vascular compliance and progressive arterial stiffness.[23–25]

The calcium-phosphate product (Ca × P) represents the interaction between serum calcium and phosphate concentrations and may reflect the severity of mineral metabolism disturbances. Persistently elevated Ca × P levels can promote mineral deposition in vascular tissues, thereby reducing arterial compliance and increasing pulse pressure.[26] Previous studies have associated elevated Ca × P levels with vascular calcification, arterial stiffness, left ventricular hypertrophy, atherosclerosis, and coronary artery calcification in patients undergoing hemodialysis.[27–30]

Although mineral metabolism abnormalities have been widely investigated in patients with CKD, the relationship between serum Ca × P levels and pulse pressure remains insufficiently studied, particularly in Indonesian hemodialysis populations.[26–30] Most previous studies have focused on CKD-mineral and bone disorder or direct assessments of coronary and vascular calcification. Pulse pressure may provide a simpler and more accessible clinical parameter for assessing vascular changes and cardiovascular risk in routine hemodialysis care.[14–18]

Evaluating the relationship between serum Ca × P levels and pulse pressure may improve the understanding of mineral metabolism abnormalities and vascular changes in patients undergoing hemodialysis. The findings may also support the clinical monitoring of calcium, phosphate, and blood pressure-related parameters. Therefore, this study aimed to analyze the relationship between serum calcium-phosphate product levels and pulse pressure in patients undergoing routine hemodialysis at Haji Adam Malik Hospital, Medan.

2. Method

This study is an analytical observational study with a cross-sectional design that examines the relationship between serum calcium-phosphate product levels and pulse pressure in patients undergoing routine hemodialysis. The study was carried out in the Hemodialysis Unit of Adam Malik Hospital, Medan, from May to July 2024, after receiving approval from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara. The study population consists of patients with chronic kidney disease undergoing regular hemodialysis at the hospital. Subjects were selected using a consecutive sampling method until the required number of participants was achieved. The inclusion criteria include patients aged 18 years or older, diagnosed with chronic kidney disease stage 5, who had been receiving regular hemodialysis for at least three months and were willing to participate by signing informed consent. The exclusion criteria include

severe cardiovascular disease, active infection, malignancy, and the use of medications that affect calcium or phosphate metabolism, such as corticosteroids or vitamin D analogues.

The minimum sample size is 60 subjects. Data were obtained through structured interviews, medical record reviews, physical examinations, and laboratory tests. Demographic and clinical characteristics such as age, sex, duration of hemodialysis, and comorbidities were collected. Blood pressure was measured on the non-fistula arm before the hemodialysis session using a calibrated sphygmomanometer after the patient had rested for at least ten minutes in a sitting position. The systolic and diastolic blood pressure values were recorded, and pulse pressure was calculated as the difference between systolic and diastolic pressures.

Blood samples were drawn before the hemodialysis session to measure serum calcium and phosphate levels. The calcium-phosphate product (Ca × P) was calculated by multiplying serum calcium (mg/dL) and phosphate (mg/dL) concentrations. All laboratory examinations were carried out in the Clinical Pathology Laboratory of Adam Malik Hospital using standardized procedures. Pulse pressure variability was categorized as normal if <60 mmHg and widened if ≥60 mmHg. Routine hemodialysis in this study was defined as hemodialysis performed two to three times per week, each lasting four to five hours per session for at least three consecutive months.

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 26. Descriptive statistics were applied to summarize the demographic and clinical characteristics of the participants. Numerical data were presented as mean ± standard deviation (SD) or median (minimum–maximum), while categorical data were expressed as frequencies and percentages. The normality of data distribution was tested using the Kolmogorov-Smirnov test. The relationship between serum calcium-phosphate product levels and pulse pressure variability was analyzed using the Spearman rank correlation test. A p-value of less than 0.05 was considered statistically significant.

3. Result

Characteristics of Subjects

A total of 60 patients undergoing routine hemodialysis were included in the analysis.

Table 1. Demographic Data of Hemodialysis Patients

Characteristics	Value (n=60)	p-value*
Sex		
Male	40 (66.7%)	<0.001*
Female	20 (33.3%)	
Duration of Hemodialysis		
< 12 Months	24 (40 %)	<0.001*
> 12 Months	36 (60 %)	
Age (Years)	48.51 ± 11.71	0.200
Systolic Blood Pressure (mmHg)	141.5 (105-200)	0.002*
Diastolic Blood Pressure (mmHg)	80 (70-111)	<0.001*

Urea (mg/dL)	151.66 ± 46.21	0.200
Creatinine (mg/dL)	13.23 ± 5.29	0.097
GFR (mL/menit/1.73 m²)	4 (1.98 – 12.90)	<0.001*

*: Statistically significant at $p < 0.05$ based on the Kolmogorov-Smirnov test.

Based on **Table 1.** above, the majority of subjects were male, totaling 40 individuals (66.7%), while females accounted for 20 individuals (33.3%). Regarding treatment duration, most of the subjects (60%) had been undergoing hemodialysis for more than one year, whereas 40% had received treatment for less than one year. The mean age of the study participants was 48 ± 11.65 years.

Clinical measurements showed that the median systolic and diastolic blood pressure values of the subjects were 140 mmHg (range 105–200 mmHg) and 80 mmHg (range 70–111 mmHg), respectively. In terms of laboratory parameters, the mean urea and creatinine levels were 153.29 ± 46.05 mg/dL and 13.23 ± 5.29 mg/dL, respectively. Finally, the median glomerular filtration rate (GFR) value among the subjects was 4 mL/min/1.73 m², with a range of 1.98–12.90 mL/min/1.73 m².

Table 2. Mineral Metabolism and Pulse Pressure Parameters among Routine Hemodialysis Patients

Characteristics	**Value**	**p-value**
Serum Calcium (mg/dL)	8.24 ± 0.83 (6.9-10.2)	<0.001*
Serum Phosphate Serum (mg/dL)	5.45 ± 1.22 (3.6-8.4)	<0.001*
Calcium-Phosphate Product (mg²/dL²)	44.93 ± 10.62 (27.6-68.2)	0.008*
Pulse Pressure (mmHg)	61.35 ± 12.24 (38-92)	0.200

*: Statistically significant at $p < 0.05$ based on the Kolmogorov-Smirnov test.

Serum Calcium Levels in Routine Hemodialysis Patients

Based on **Table 2.** above, the mean serum calcium concentration among the participants was 8.24 ± 0.83 mg/dL, with values ranging from 6.9 to 10.2 mg/dL. The distribution of calcium levels showed that the majority of patients (65%) had serum calcium levels within the normal range, while 21.7% had values below normal and 13.3% above the normal reference limit.

Serum Phosphate Levels in Routine Hemodialysis Patients

As presented in **Table 2.**, the mean serum phosphate level was 5.45 ± 1.22 mg/dL, with values ranging between 3.6 and 8.4 mg/dL. The majority of patients (56.7%) exhibited elevated phosphate levels exceeding the normal range, while 43.3% maintained phosphate concentrations within normal limits.

Calcium-Phosphate Product Values in Routine Hemodialysis Patients

Table 2. also shows that the mean calcium-phosphate product (Ca × P) value among the participants was 44.93 ± 10.62 mg²/dL², with a range of 27.6 to 68.2 mg²/dL². A total of 28 patients (46.7%) had calcium-

phosphate product values above the recommended limit of 55 mg^2/dL^2, while 32 patients (53.3%) had values below this threshold.

Pulse Pressure Values in Routine Hemodialysis Patients

Regarding pulse pressure, **Table 2.** demonstrates that the mean pulse pressure was 61.35 ± 12.24 mmHg, ranging from 38 to 92 mmHg. Based on the classification criteria, 35 patients (58.3%) exhibited widened pulse pressure (≥60 mmHg), while 25 patients (41.7%) had normal pulse pressure (<60 mmHg).

Correlation Between Serum Calcium-Phosphate Product and Pulse Pressure Variability

The relationship between serum calcium-phosphate product (Ca × P) levels and pulse pressure variability among routine hemodialysis patients was analyzed using the Spearman rank correlation test, as the data were not normally distributed. The results demonstrated a weak positive correlation between calcium-phosphate product and pulse pressure values, with a correlation coefficient (r) of 0.224 and a p-value of 0.085. Although the relationship was positive, it did not reach statistical significance ($p > 0.05$), indicating that higher calcium-phosphate product levels were not significantly associated with increased pulse pressure variability in this study population.

Further descriptive analysis showed that patients with higher calcium-phosphate product values tended to have greater pulse pressure means compared to those with lower values, but the overlap in data distribution suggested that other factors might have contributed to pulse pressure variability.

Table 3. Correlation Between Serum Calcium-Phosphate Product and Pulse Pressure

	Pulse Pressure*
Calcium-Phosphate Product	r = 0.224 p – value = 0.085 n = 60

*: Spearman Correlation Test

4. Discussion

This study analyzed the relationship between serum calcium-phosphate product levels and pulse pressure variability in patients undergoing routine hemodialysis at Adam Malik Hospital. The findings showed a weak positive correlation between calcium-phosphate product and pulse pressure, but the relationship was not statistically significant. This result suggests that while an increase in calcium-phosphate product tends to be associated with widened pulse pressure, the association is not strong enough to be considered statistically significant. These findings are in line with several previous studies that have shown inconsistent results regarding the association between calcium-phosphate metabolism and vascular stiffness in hemodialysis patients.[26–30]

The mean age of respondents in this study was 48.51 ± 11.71 years, with the majority being male. This demographic characteristic is consistent with epidemiological data indicating that chronic kidney disease and the need for dialysis therapy are more common among men than women.[31] Men are more frequently exposed

to risk factors such as hypertension, smoking, and metabolic disorders, which accelerate the decline in kidney function.[4] Similar results were reported that most hemodialysis patients were aged between 45 and 60 years, an age group at high risk for cardiovascular complications.[3,31] The predominance of middle-aged and older adults in this study also corresponds to the natural progression of CKD, where cumulative exposure to metabolic stress, oxidative damage, and comorbidities such as diabetes and hypertension leads to renal failure.[32,33]

The majority of study participants were hypertensive, which is consistent with the understanding that hypertension is both a cause and a consequence of CKD.[34] Chronic hypertension contributes to glomerular injury and accelerates kidney damage through increased intraglomerular pressure and vascular remodeling.[31,34] In turn, CKD exacerbates hypertension due to sodium and fluid retention, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system.[3,34,35] As a result, most hemodialysis patients experience persistent blood pressure elevation and increased vascular stiffness. The presence of widened pulse pressure in more than half of the participants in this study (58.3%) may reflect hemodynamic changes commonly observed in patients with CKD and those undergoing hemodialysis.[36–39]

The mean pulse pressure in this study was 61.35 ± 12.24 mmHg. Elevated pulse pressure may indicate reduced arterial compliance and increased arterial stiffness, which are commonly found in patients with CKD and those undergoing dialysis.[14,15] Pulse pressure can serve as an indirect measure of large artery stiffness and is associated with cardiovascular morbidity and mortality in dialysis patients.[16–18] Vascular calcification and endothelial dysfunction related to calcium and phosphate imbalance may also contribute to increased pulse pressure in CKD patients.[10,23–25] However, pulse pressure may be affected by age, hypertension, diabetes mellitus, cardiovascular disease, volume status, and duration of hemodialysis. Since CKD is frequently associated with cardiovascular and metabolic comorbidities, these factors may confound the observed relationship between calcium-phosphate product and pulse pressure.[31,34,36–39]

The mean serum calcium level found in this study was 8.24 ± 0.83 mg/dL, with most patients maintaining calcium values within the normal range. This finding is comparable to previous studies reporting that serum calcium concentrations in hemodialysis patients are usually maintained near normal through the regulation of dialysate calcium concentration and the administration of calcium-containing phosphate binders.[14] However, fluctuations in calcium levels are still common due to individual variations in dietary calcium intake, medication adherence, and dialysis adequacy.[19–22] Since calcium and phosphate homeostasis is closely related to secondary hyperparathyroidism, vascular calcification, and arterial stiffness in CKD, serum calcium should be interpreted together with phosphate and the calcium-phosphate product when assessing mineral metabolism status and cardiovascular risk in hemodialysis patients.[23–25]

The mean serum phosphate level among the subjects was 5.45 ± 1.22 mg/dL, indicating that more than half of the participants exhibited hyperphosphatemia. Phosphate retention is one of the earliest and most prominent abnormalities in chronic kidney disease and plays a pivotal role in the development of secondary hyperparathyroidism and vascular calcification.[19–23] High serum phosphate stimulates vascular smooth muscle cells (VSMCs) to undergo osteogenic transformation, leading to the deposition of calcium–phosphate crystals within the medial layer of arterial walls.[23] In addition, hyperphosphatemia induces oxidative stress and

inflammation, further aggravating endothelial dysfunction and arterial stiffness.[24,25] These mechanisms collectively explain the increased cardiovascular morbidity and mortality observed in dialysis patients with poor phosphate control.[22]

The mean calcium-phosphate product in this study was 44.93 ± 10.62 mg^2/dL^2, with 46.7% of participants showing values above the cutoff of 55 mg^2/dL^2. Elevated calcium-phosphate product levels may reflect disordered mineral metabolism and have been associated with vascular calcification in CKD and hemodialysis patients.[20,23–26] Previous studies by London et al. and Goodman et al. reported that high Ca × P values were associated with coronary artery and aortic calcification, which may contribute to arterial stiffness and increased pulse pressure.[12,27–30] This process may involve active vascular changes, including the differentiation of vascular smooth muscle cells into osteoblast-like phenotypes.[23–25]

Although the present study demonstrated a positive correlation between calcium–phosphate product levels and pulse pressure variability, the relationship was not statistically significant. This finding differs from several reports that showed a stronger association between Ca × P and arterial stiffness in hemodialysis patients.[27–30] The absence of a significant correlation in this study may be attributed to several factors, including the relatively small sample size, homogeneity of the study population, and the influence of confounding variables such as blood pressure control, medication use, and nutritional status.[31,36–39] Moreover, the cross-sectional nature of this study limits its ability to establish a causal relationship between calcium–phosphate product and vascular changes. Longitudinal studies are required to better understand how chronic exposure to elevated Ca × P affects the progression of arterial stiffness over time.[40]

The weak positive trend observed in this study remains physiologically plausible because mineral metabolism disorders may contribute to vascular calcification, reduced arterial compliance, and increased pulse pressure.[20,23–26] However, the weak correlation may reflect the complex interaction between mineral imbalance, inflammation, anemia, oxidative stress, and uremic toxins, which can simultaneously affect vascular integrity and hemodynamic parameters in dialysis patients.[36–39]

Previous studies have shown that the relationship between mineral metabolism markers and arterial stiffness in hemodialysis patients may vary across populations.[27–30] While disturbances in calcium and phosphate balance may contribute to vascular calcification, the degree of vascular stiffness is also influenced by other metabolic and hemodynamic factors, including hypertension, chronic inflammation, endothelial injury, dialysis duration, and volume status.[15,20,23,36–39] This multifactorial nature may explain why calcium-phosphate product alone is not consistently associated with pulse pressure across different populations.

Several potential confounding factors could have influenced the results of this study. Blood pressure control is one of the most important determinants of pulse pressure, and blood pressure variability in CKD has been associated with cardiovascular events and adverse outcomes.[4,5,41] Volume status and intradialytic hemodynamic instability may also affect blood pressure parameters in hemodialysis patients.[37–39] Furthermore, the duration and adequacy of dialysis may influence mineral balance and vascular health.[36] Differences in dialysate calcium concentration, dietary phosphate restriction, residual renal function, and adherence to phosphate binder therapy may also contribute to inter-individual variability in calcium-phosphate product and pulse pressure values.[21,22,36]

Another possible explanation for the lack of a significant relationship is the relatively narrow range of calcium-phosphate product values in this study. Most patients were maintained within moderately elevated Ca × P levels due to regular monitoring and standard therapy at the hemodialysis unit. This homogeneity could have reduced the statistical power to detect a stronger correlation between calcium-phosphate product and pulse pressure. In addition, the cross-sectional design limits the ability to capture the cumulative effects of chronic mineral imbalance and long-term vascular remodeling. It is possible that the impact of elevated Ca × P levels on arterial stiffness may become more evident in longitudinal observations over several years.[27,30,40]

Routine monitoring of calcium-phosphate product, along with noninvasive measures such as pulse pressure assessment, may help identify patients at early risk of arterial stiffness.[14–18,20,22] However, pulse pressure remains an indirect parameter and may be influenced by volume status, cardiac output, antihypertensive treatment, and dialysis-related hemodynamic changes.[37–39] Therefore, pulse pressure should be interpreted together with mineral metabolism markers and other cardiovascular risk factors.

This study has several limitations that must be considered when interpreting the findings. First, the cross-sectional design limits the ability to establish a causal relationship between calcium-phosphate product levels and pulse pressure. The data collected represent a single point in time, whereas vascular calcification and arterial stiffness are progressive processes that develop through prolonged exposure to metabolic and hemodynamic abnormalities.[23,27,30] Longitudinal studies are therefore needed to better assess temporal associations and determine whether persistent elevations in calcium–phosphate product contribute directly to worsening arterial stiffness in hemodialysis patients.

Second, the relatively small sample size and the homogeneity of the study population may have reduced the statistical power to detect significant correlations. Most subjects in this study received similar dialysis regimens and pharmacologic therapy, resulting in a narrow range of calcium–phosphate product and pulse pressure values. Including a larger and more diverse sample from multiple dialysis centers would increase variability and improve the generalizability of the findings. Third, this study did not evaluate other biochemical markers that may influence vascular stiffness, such as parathyroid hormone (PTH), fibroblast growth factor 23 (FGF-23), and vitamin D levels. These parameters play important roles in mineral metabolism and may provide additional insights into the mechanisms linking calcium–phosphate product and vascular changes.[31,34,36–39]

Fourth, arterial stiffness was assessed indirectly through pulse pressure rather than by direct measures such as pulse wave velocity (PWV) or arterial compliance index, which are considered more specific indicators of vascular function.[27–30] Although pulse pressure is a simple and practical clinical parameter, it may be affected by various factors including volume status, cardiac output, and antihypertensive treatment. Therefore, future studies incorporating more precise vascular imaging or functional assessments would be valuable for confirming the relationship between mineral metabolism and arterial stiffness.

The absence of a significant correlation between calcium-phosphate product and pulse pressure may reflect the multifactorial nature of vascular dysfunction in chronic kidney disease. Factors such as hypertension, inflammation, oxidative stress, anemia, volume status, and dialysis duration may also influence

pulse pressure and arterial stiffness.[36–39] Therefore, calcium-phosphate product should be interpreted as part of a broader cardiovascular and mineral metabolism assessment in hemodialysis patients.[19–22]

5. Conclusion

This study demonstrated that there was no statistically significant correlation between serum calcium–phosphate product levels and pulse pressure variability in patients undergoing routine hemodialysis at Adam Malik Hospital, Medan. Although a weak positive relationship was observed, it did not reach statistical significance, indicating that elevated calcium–phosphate product levels were not independently associated with increased arterial stiffness as reflected by pulse pressure.

Clinically, this study supports the need for integrated monitoring of mineral metabolism and cardiovascular risk in hemodialysis patients. Routine assessment of serum calcium, phosphate, calcium-phosphate product, and pulse pressure may help identify patients who require closer follow-up for vascular calcification and cardiovascular complications. Future studies should involve larger samples, longer follow-up periods, direct measurements of arterial stiffness, and multivariable analysis to determine whether calcium-phosphate product is independently associated with vascular changes in hemodialysis patients.

6. Conflict of Interest

All the authors declare no conflict of interest.

7. Conflict of Interest

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