Correlations between Serum Magnesium and PTH Levels and Vascular Calcification in Patients Undergoing Hemodialysis and Study of Associated Factors

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Keywords. Hemodialysis; Vascular calcification; Magnesium; Parathyroid hormone

Introduction. This study is an attempt to investigate the correlations between serum magnesium (Mg) and parathyroid hormone (PTH) levels and abdominal aortic calcification **(A**AC) score.
Methods. This retrospective analysis was conducted on the clinical records of 101 cases undergoing maintenance hemodialysis at Liyang People's Hospital (China) between May 2021 and May 2023. The clinical features of the calcified and non-calcified groups were compared. Cardiovascular calcification was defined as intima-media thickness (IMT) ≥1.2 mm or local thickness≥0.5 mm. Logistic regression analysis was carried out for identifying independent influencing factors of calcification. Furthermore, the correlations between these influencing factors and AAC were analysed.
Results. Calcification was diagnosed in 76 patients (75.2%). In comparison to the non-calcified group, the calcified group showed markedly elevated age, dialysis duration, and phosphorus levels of (P), alkaline phosphatase (AKP), parathyroid hormone (PTH), ferritin (FER), and low-density lipoprotein (LDL). ($P < .05$). Conversely, the calcified group had significantly lower body mass index (BMI) and magnesium (Mg) levels compared to the non-calcified group ($P < .05$). Logistic regression analysis identified age ($P = .006$), dialysis duration ($P = .002$), PTH ($P = .016$), and Mg ($P < .001$) as independent influencing factors of calcification. Furthermore, age, dialysis duration, and PTH were positively bound up with AAC score ($P < .05$), while Mg was negatively bound up with it ($P < .001$).
Conclusion. Mg may be a protective factor to inhibit vascular calcification. Age, dialysis duration and PTH affect the calcification process through various mechanisms.

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INTRODUCTION

Currently, the prevalence of chronic kidney disease (CKD) is increasing annually.[1] According to a 2012 epidemiological survey, conducted in China,[1] the incidence of CKD among adults aged 18 and above was reported to be 10.8%. CKD can be caused by various factors, including diabetes mellitus, hypertension, primary nephrotic syndrome, IgA nephropathy, polycystic kidney disease, and lupus nephritis.[1] These conditions lead to a progressive decline in kidney function, ultimately triggering end-stage kidney disease (ESKD). Individuals with ESKD typically require renal replacement therapy to sustain their lives, with maintenance hemodialysis serving as one of the treatment options.[2]

Hemodialysis utilizes a semi-permeable membrane to facilitate the exchange of substances, such as solutes and water, to remove toxins and excess water from the body, as well as to maintain water, electrolytes, and acid-base balance. This exchange is achieved through processes such as diffusion, convection, adsorption, and ultrafiltration.[3] However, patients undergoing maintenance hemodialysis often experience vascular calcification.[3] Vascular calcification serves as a significant risk factor for cardiovascular diseases,[4] thereby substantially increasing the incidence and mortality rates of cardiovascular complications in these patients. Compared with the general population, patients undergoing hemodialysis face a significantly higher risk of cardiovascular disease-related mortality, with an increased risk ranging from 10 to 20 times higher.[5] Therefore, an increasing number of researchers have shifted their focus towards studying vascular calcification in patients with ESKD.[4]

Vascular calcification, characterized by calcium phosphate deposition in vascular walls and heart valves, primarily occurs as two distinct types: intimal calcification (associated with atherosclerosis in the general population) and medial calcification (specifically linked to ESKD as well as metabolic disorders.[6] Among them, intimal calcification primarily occurs in the intima of large blood vessels and coronary arteries, contributing to the development of atherosclerosis. This process is manifested by intimal hyperplasia and involves the thickening of the intima due to the accumulation of plaques formed by macrophage infiltration and lipid build-up. As a result, the affected vascular walls become stiff and narrow, which leads to reduced blood flow, gradually blocking blood vessels, finally resulting in tissue ischemia, hypoxia, and ultimately necrosis.[7] In contrast, vascular calcification in patients with CKD predominantly affects the middle layer, which is also referred to as Monckeberg's calcification.[8] Medial calcification primarily occurs in the smooth muscle layer of the aorta and medium-sized arteries. It is characterized by the absence of inflammatory cells and lipid infiltration in the elastic layer of the arterial media. Alternatively, there is linear deposition of hydroxyapatite crystal calcium which contributes to the stiffening of blood vessels and reduced compliance, bound up with the development of hypertension.[9]

The current study focused on identifying the key factors associated with vascular calcification in patients undergoing hemodialysis. While previous studies have investigated various factors influencing vascular calcification, our study took a step further by specifically examining the relationship between serum magnesium (Mg) and parathyroid hormone (PTH) levels and the abdominal aortic calcification (AAC) score. This unique research aimed to gain a deeper understanding of the mechanisms underlying vascular calcification in patients undergoing hemodialysis and to uncover potential associations between these parameters. By employing this novel approach, we anticipated providing more targeted insights and strategies for the management and prevention of cardiovascular diseases in this high-risk population.

MATERIALS AND METHODS

Clinical data

A retrospective analysis study was conducted on 101 patients who underwent maintenance hemodialysis treatment at Liyang People's Hospital (China) between May 2021 and May 2023. This study received approval from the ethnical committee of the hospital, with ethical approval number of LW2023350.

Inclusion and exclusion criteria

The inclusion criteria were: patients (1) 18 years old or above; (2) on maintenance hemodialysis for a duration of more than three months; (3) with a dialysis frequency of two times/week or three times/week, with each session lasting four hours; (4) receiving dialysis with dialysate of consistent concentration and blood flow rate ranging from 180 to 260 mL/min through polyflux14L dialyzer manufactured by Kimball Company of Germany.

The exclusion criteria were : patients with (1) severe malnutrition or abnormal liver function; (2) connective tissue diseases; (3) tuberculosis; (4) benign or malignant tumors; patients who (5) had undergone surgery, experienced trauma, or had an infection within the past month; (6) had received immunosuppressants or hormone therapy within the past year; (7) had undergone parathyroidectomy; and (8) patients with mental illness or cognitive impairment that hindered their ability to cooperate.

Collection of clinical data

We collected clinical data and laboratory indices based on the pathological information of the patients. The clinical data included age, sex, systolic and diastolic blood pressures, body mass index (BMI), as well as dialysis duration. The laboratory indices included the following parameters: hemoglobin (HGB), calcium (Ca), phosphorus (P), calcium and phosphorus product (Ca × P), albumin (ALB), alkaline phosphatase (AKP), PTH, ferritin (FER), glucose (Glu), cholesterol (CHOL), triglycerides (TG), low-density lipoprotein (LDL), high-sensitivity C-Reactive Protein (hs-CRP), as well as Mg. Moreover, we collected the abdominal aorta calcification (AAC) scores after treatment.

Definition of vascular calcification

The carotid calcification detection method involved the patient lying on their back with their head slightly turned to one side, exposing the neck. The examiner then examined the trunk of the common carotid artery and the beginning of the internal carotid artery. The carotid intima-media thickness (IMT) and presence of plaques were recorded. The classification criteria were as follows: 1) carotid intima-media thickening was defined as an IMT greater than 0.9 mm and less than 1.2 mm; 2) plaque formation was defined as either an IMT ≥ 1.2 mm or a local thickness ≥ 0.5 mm. Carotid intima thickening or plaque formation is considered as carotid atherosclerosis, which is a form of cardiovascular calcification. The detection method for heart valve calcification involved the patient lying on their back, with the chest exposed. A probe was used to examine the heart valve area. If one or more strong echoes greater than 1 mm were observed in the aortic valve, mitral valve, or annulus, it was defined as valve calcification. The detection of AAC involved performing a lateral X-ray plain film examination of the abdomen. This examination included capturing images of the 11th and 12th thoracic vertebrae, the 1st to 5th lumbar vertebrae, and the 1st and 2nd sacral vertebrae. By analysing these images, calcification of the anterior and posterior walls of the abdominal aorta corresponding to these anatomical regions could be observed. The Kauppila scoring method for AAC was as follows:[10] 0 points: No calcification observed; 1 point: Calcification range was less than 1/3 of the arterial wall length; 2 points: Calcification range was between 1/3 and 2/3 of the arterial wall length; 3 points: Calcification range was greater than 2/3 of the arterial wall length. The total score range was 0 to 24, which was scored independently by two selected blindly radiologists, and finally averaged.

Calcification grouping

Patients who exhibited carotid atherosclerosis, heart valve calcification, and AAC were included in the calcified group (n = 76). Any presence of calcification in these areas defined a patient as having vascular calcification. Patients who did not exhibit these

symptoms or had a carotid IMT less than 0.9 mm were included in the non-calcified group (n = 25).

Result measurement

1. The clinical data and laboratory indices were compared between the calcified and non-calcified groups.
2. The measurement data were classified one the basis of the cut-off values determined by the Receiver Operating Characteristic curve (ROC).
3. Logistic regression was employed for identifying the independent risk factors linked to vascular calcification in dialysis patients.
4. The correlations between risk factors and AAC score were examined.

Statistical analyses

Data analysis was carried out by using SPSS 26.0. The mean ± standard deviation (x ± s) was adopted for describing the normally distributed measurement data. The t-test was employed to compare the measurement data from two independent samples. The t non-normally distributed measurement data were expressed by median (1/4~3/4), analysed by the rank sum test and presented by Z. Counting data were analyzed by using the$\chi 2$ test with a four-grid table, and the statistical results were represented by $\chi 2$ values. Spearman correlation analysis was employed for univariate correlation analysis, while logistic regression analysis was adopted for identifying risk factors. $P < .05$ indicates a conspicuous difference.

RESULTS

Comparison of baseline data

In the comparison of the two groups for clinical data and laboratory indices, the calcified group

had significantly higher levels of age, dialysis duration, phosphorus (P), alkaline phosphatase (AKP), parathyroid hormone (PTH), ferritin (FER), and low-density lipoprotein (LDL) ($P < .05$, Table 1). Conversely, the calcified group had significantly lower BMI and Mg levels compared to the non-calcified one ($P < .05$, Table 1)

Analysis of risk factors affecting vascular calcification in patients

The relevant clinical data were analyzed and assigned meaningful values. Prior to the assignment, the Cut-off value for each measurement data was calculated by using the ROC used as the binary threshold (Figure 1, Table 2). After assigning the data, a multivariate logistic regression analysis was performed, which revealed that age, dialysis duration, PTH, and Mg were independent risk factors for vascular calcification in dialysis patients (Figure 2). Additionally, all the assigned data can be found in Table 3.

Correlations between risk factors and AAC score

The Pearson correlation test was used to evaluate the correlations between AAC score and risk factors in this study. The results indicated that age, dialysis duration, and PTH were positively bound up with AAC score, while Mg was negatively bound up with it ($P < .05$, Figure 3).

DISCUSSION

Currently, there is no clear demarcation of chronic vascular inflammation, atherosclerosis, and arterial calcification, as these conditions often overlap and influence each other in vascular disease progression. All of these conditions can result in vascular calcification, leading to decreased elasticity and increased brittleness of affected blood vessels and consequently contributing to the development of cardiovascular diseases and death.[11,12] ESKD patients on hemodialysis are more prone to vascular and heart valve calcification.[5] Ventura *et al.*[13] included 135 patients with vascular calcification in their study and reported a calcification rate of 78% in the aortic valve and 26% in the mitral valve. Rufino *et al.*[14] found that 38.4% of 52 hemodialysis patients had valve calcification. These findings suggest that both cardiovascular calcification and cardiac valve calcification contribute to increased vascular calcification, cardiovascular events, and mortality.

Vascular calcification is a complex and regulated process similar to bone development and cartilage formation. It involves multiple

Table 1. Baseline data of patients

Marker	Calcified group (n=76)	Non-calcified group (n=25)	χ^2/t/Z	*P*
Age (years)	56.88 ± 12.41	48.92 ± 10.22	3.196	.002
Sex (male/female)	42/34	12/13	0.398	.527
Systolic blood pressure (mmHg)	143.45 ± 19.62	143.52 ± 16.12	-0.018	.985
Diastolic blood pressure (mmHg)	82.00 (77.00,89.00)	78.00 (70.00,91.00)	0.838	.404
BMI (kg/m^2)	22.16 ± 2.45	23.30 ± 1.63	-2.663	.010
Dialysis duration (month)	66.50 (42.75,89.25)	38.00 (19.00,49.00)	3.875	< .001
HGB (g/L)	107.76 ± 16.73	104.30 ± 15.53	0.949	.348
Ca (mmol/L)	2.28 ± 0.25	2.32 ± 0.22	-0.685	.497
P (mmol/L)	2.20 ± 0.66	1.92 ± 0.48	2.262	.028
Calcium-phosphorus product	62.20 ± 20.13	55.24 ± 15.47	1.804	.077
ALB (g/L)	38.37 ± 4.76	39.59 ± 4.39	-1.18	.245
AKP (U/L)	123,250 ± 46,820	96,200 ± 34,970	3.068	.003
PTH (ng/L)	533.93 ± 229.66	356.69 ± 162.44	4.238	< .001
FER (μg/L)	0.32 (0.18,0.48)	0.17 (0.13,0.33)	2.195	.028
Glu (mmol/L)	5.86 ± 1.61	5.74 ± 1.20	0.370	.713
CHOL (mmol/L)	4.75 (3.86,5.09)	4.75 (3.79,5.21)	0.090	.931
TG (mmol/L)	1.94 ± 0.89	2.10 ± 0.70	-0.919	.363
LDL (mmol/L)	2.97 ± 1.57	2.50 ± 0.65	2.088	.040
hs-CRP (mg/L)	5.86 ± 2.48	5.01 ± 3.01	1.275	.211
Mg (mmol/L)	1.03 ± 0.19	1.27 ± 0.18	-5.623	< .001

Note: BMI: body mass index; HGB: hemoglobin; Ca: calcium; P: phosphorus; ALB: Albumin; AKP: alkaline phosphatase; PTH: parathyroid hormone; FER: ferritin; Glu: glucose; CHOL: cholesterol; TG: triglycerides; LDL: low-density lipoprotein; hs-CRP: high-sensitivity C-Reactive protein; Mg: magnesium.

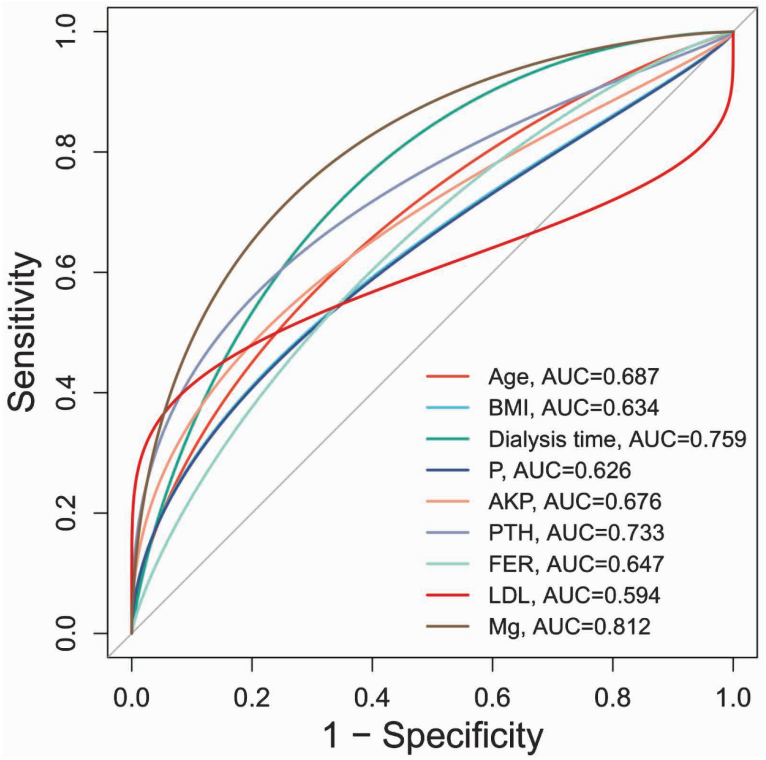

Figure 1. ROC curve of single factor index

Table 2. ROC parameters of single factor index

Marker	AUC	95%CI	Cut off	Specificity	Sensitivity	Youden index
Age (years old)	0.687	0.571 - 0.687	54.500	72.00%	59.21%	31.21%
BMI (kg/m^2)	0.634	0.516 - 0.634	23.290	64.00%	68.42%	32.42%
Dialysis duration (month)	0.759	0.649 - 0.759	58.500	88.00%	59.21%	47.21%
P (mmol/L)	0.626	0.507 - 0.626	2.205	76.00%	50.00%	26.00%
AKP (U/L)	0.676	0.565 - 0.676	139.525	92.00%	40.79%	32.79%
PTH (ng/L)	0.733	0.631 - 0.733	526.195	88.00%	50.00%	38.00%
FER (μg/L)	0.647	0.523 - 0.647	171.06	52.00%	77.63%	29.63%
LDL (mmol/L)	0.594	0.488 - 0.594	3.400	96.00%	43.42%	39.42%
Mg (mmol/L)	0.812	0.722 - 0.812	1.165	76.00%	72.37%	48.37%

Note: BMI: body mass index; P: phosphorus; AKP: alkaline phosphatase; PTH: parathyroid hormone; FER: ferritin; TG: triglycerides; Mg: magnesium.

factors and shares similarities with physiological bone mineralization.[15] This study analysed the correlations between serum biomarkers and AAC scores in 101 maintenance hemodialysis patients. The incidence rate of vascular calcification was found to be 75.24%, which is consistent with previous study reports.[7,16] Subsequently, we conducted logistic regression analysis for identifying the independent risk factors linked to vascular calcification in dialysis patients. Age, dialysis duration, and PTH were identified as independent risk factors contributing to calcification, while Mg was identified as a protective factor against vascular calcification in hemodialysis patients. Long-term dialysis reflects a prolonged duration of kidney failure, which can lead to electrolyte imbalance, particularly calcium and phosphorus.[17] Advancing age is associated with reduced vascular function and impaired tissue repair ability, making individuals more susceptible to vascular calcification.[17] Furthermore, dialysis can induce increased secretion of PTH, potentially resulting in hypercalcemia.[18] Therefore, as dialysis duration and age increase, the process of calcification can be exacerbated through various mechanisms.[19] Previous research by Roumeliotis *et al*.[20]

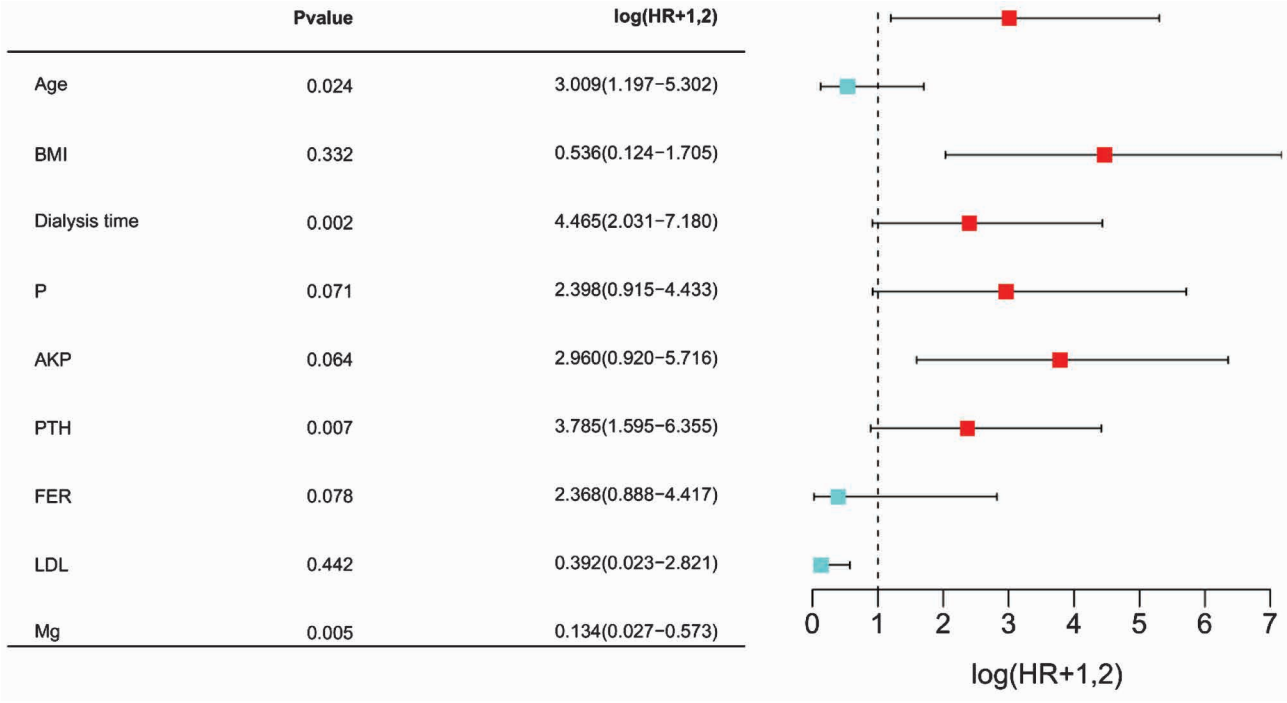

Figure 2. Multivariate logistics regression analysis
Notes: BMI: body mass index; P: phosphorus; AKP: alkaline phosphatase; PTH: parathyroid hormone; FER: ferritin: TG: triglycerides; Mg: magnesium.

Table 3. Assignment of data

Marker	Assignment content
Age (years old)	< 54.5 = 0, ≥ 54.5 = 1
BMI (kg/m^2)	< 23.29 = 0, ≥ 23.29 = 1
Dialysis duration (month)	< 58.5 = 0, ≥ 58.5 = 1
P (mmol/L)	< 2.205 = 0, ≥ 2.205 = 1
AKP (U/L)	< 139.525 = 0, ≥ 139.525 = 1
PTH (ng/L)	< 526.195 = 0, ≥ 526.195 = 1
FER (μg/L)	< 171.06 = 0, ≥ 171.06 = 1
LDL (mmol/L)	< 3.4 = 0, ≥ 3.4 = 1
Mg (mmol/L)	< 1.165 = 0, ≥ 1.165 = 1
Calcification	Calcified group = 1, non-calcified group = 0

Notes: BMI: body mass index; P: phosphorus; AKP: alkaline phosphatase; PTH: parathyroid hormone; FER: ferritin: TG: triglycerides; Mg: magnesium.

demonstrated the significance of the activation state of vitamin K in the activity of matrix Gla protein (MGP). Insufficient levels of vitamin K can result in the inactivation of MGP, promoting arterial calcification or sclerosis. According to another study,[21] vascular calcification progresses rapidly in patients undergoing maintenance dialysis, and the incidence and progression rate vary depending on the type of vascular calcification. By consistently achieving target levels of serum calcium, P, and PTH, the risk of coronary artery calcification can be reduced.[16] The relationship between bone mineral metabolism and vascular calcification in ESKD patients has been verified in one study,[7] shedding light on the intricate interaction between these two processes. Among elderly hemodialysis patients, risk factors for mortality include diabetes mellitus, central venous catheter, early initiation of dialysis, frailty, functional impairment, and cognitive impairment. These factors may also be associated with vascular calcification.[22]

Mg is an essential cation in the human body, playing a crucial role in various biological functions. It is involved in regulating heart rhythm, participating in DNA synthesis, and influencing platelet activation and atherosclerosis.[23] A decrease in Mg levels has been observed to be associated with vascular calcification in patients with CKD.[23] Studies have indicated that Mg supplementation may help in preventing the progression of vascular calcification.[17,24] Additionally, serum Mg levels are related to PTH levels.[25] PTH is a crucial hormone produced by the parathyroid gland in the neck, and it takes a central part in maintaining the metabolic balance of calcium and P.[26] In patients with CKD requiring dialysis, the body's calcium and P metabolism is significantly affected due to

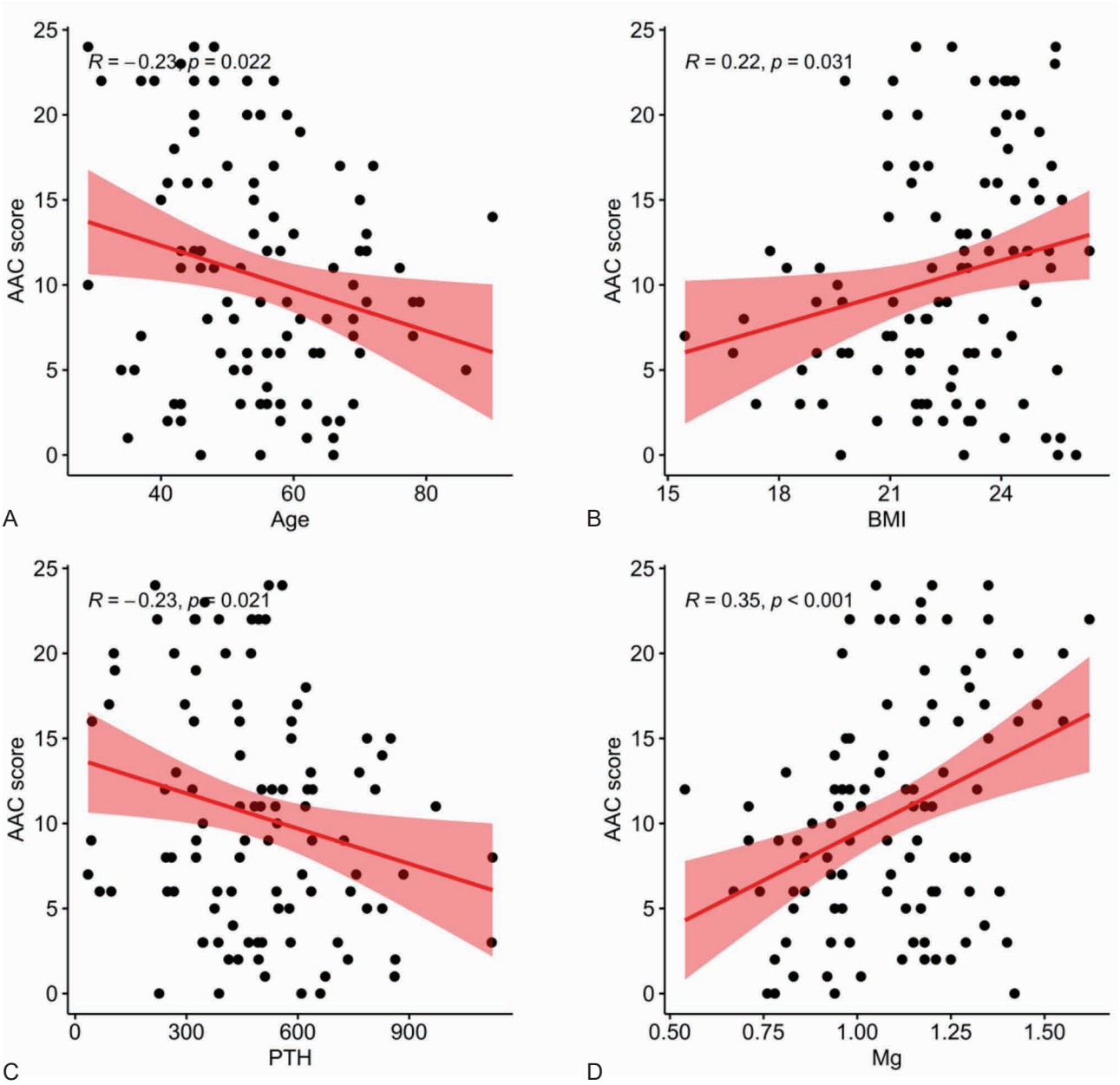

Figure 3. Correlation analysis between risk factors and patient's AAC score. (A) Correlation analysis between age and patient's AAC score; (B) Correlation analysis between BMI and patient's AAC score; (C) Correlation analysis between PTH and patient's AAC score; (D) Correlation analysis between Mg and patient's AAC score.

impaired renal function.[23] A high PTH level can directly and indirectly promote calcium deposition in tissues.[25] Simultaneously, the decrease Mg level, as a physiological antagonist of calcium, may weaken the tolerance of tissues and cells to calcium, which can activate proinflammatory factors and exacerbate hyperphosphatemia, further accelerating the process of vascular calcification.[11] Therefore, abnormal levels of PTH and Mg can independently increase the risk of calcification through multiple mechanisms. These findings underscore the significance of monitoring and regulating Mg and PTH levels in dialysis patients to prevent vascular calcification.

AAC score is a quantitative index for evaluating the degree of calcification in the abdominal aorta.[27] Higher AAC scores are associated with an increased risk of cardiovascular events and mortality in CKD patients, particularly those undergoing dialysis treatment.[10] In the current study, we applied the Pearson correlation test to examine the relationship

between various risk factors and AAC score. The results indicated that age, dialysis duration, and PTH levels were positively correlated with AAC score. These results demonstrate that AAC score tends to increase with advancing age, prolonged dialysis treatment duration, and elevated PTH levels in patients. On the contrary, the Mg level was negatively correlated with AAC score, indicating that a higher Mg level may be associated with a lower level of aortic calcification. The positive correlation between age and AAC score may be attributed to the long-term exposure to cardiovascular risk factors throughout one's life, resulting in progressive arteriosclerosis.[10] Likewise, the prolonged duration of dialysis treatment reflects the extended period of CKD. CKD disrupts the balance of calcium and P, which can contribute to vascular calcification development.[28] Elevated PTH levels resulting from secondary hyperparathyroidism commonly occur in patients with CKD, and can contribute to bone mineral metabolism disorders and vascular calcification.[28] Conversely, Mg is believed to have a protective effect against vascular calcification. Mg acts as a natural calcium antagonist, competing with calcium to bind to vascular smooth muscle cells and inhibiting calcium deposition.[29] Moreover, Mg can also exert anti-inflammatory effects and inhibit the transformation of vascular smooth muscle cells into osteoblast-like cells, which is a crucial process in vascular calcification development.[29] Our research underscores the significance of monitoring and managing these factors in patients with CKD, as it may help reduce the risk of AAC and its associated adverse health outcomes.

CONCLUSION

This study demonstrates that vascular calcification is prevalent among hemodialysis patients, with age, dialysis duration, and elevated PTH levels identified as independent risk factors, while higher serum Mg levels exhibit a protective effect.

LIMITATIONS

However, there are limitations to this study. Firstly, the sample size is small, consisting of only 101 patients receiving maintenance hemodialysis. This limited sample size may introduce bias into the results. Expanding the sample size is necessary to mitigate potential biases. Additionally, our study was conducted retrospectively and lacked prospective follow-up, making it impossible to evaluate the relationship between vascular calcification and patient prognosis. Therefore, a prospective cohort study with long-term follow-up is needed to observe the relationship between calcification and the risk of cardiovascular events and mortality. Lastly, our study focused solely on the analysis of traditional risk factors and did not include intervention research. In the future, it would be valuable to evaluate the impact of different treatments targeting calcification inhibition on patient prognosis through randomized controlled trials.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was performed with approval from the Medical Ethics Committee of Liyang People's Hospital (ethical approval number: LW2023350) and in accordance with the Declaration of Helsinki.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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