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ORIGINAL RESEARCH

Impact of Serum Magnesium Levels on Mineral-Bone Metabolism in Non-Dialysis-Dependent Chronic Kidney Disease

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Background: Magnesium is one of the important minerals in the human body. The metabolic imbalance of magnesium has been found in patients with chronic kidney disease (CKD), but the research on the serum magnesium level of patients with non-dialysis-dependent CKD (NDD-CKD) and its relationship with mineral-skeletal metabolic disorder (CKD-MBD) is still relatively limited. This study aims to explore the changes in serum magnesium levels in patients with non-dialysis-dependent CKD at different stages and evaluate its relationship with mineral metabolism markers (calcium, phosphorus, iPTH and alkaline phosphatase).

Methods: A retrospective analysis was conducted on data from 156 patients with stage 3–5 CKD (G3–G5) admitted to Handan First Hospital between March 2017 and December 2019. Serum magnesium, calcium, phosphorus, intact parathyroid hormone (iPTH), and alkaline phosphatase (AKP) levels were analyzed. The patients were categorized into hypomagnesemia, normal magnesium, and hypermagnesemia groups based on serum magnesium levels. Correlations between magnesium levels and phosphorus, iPTH, and AKP levels were analyzed.

Results: Serum magnesium levels displayed an upward trend across CKD stages G3 to G5, though this trend was not statistically significant. The prevalence of hypomagnesemia was 0%, 6.82%, and 15.07%, while the incidence of hypermagnesemia was 5.13%, 13.64%, and 30.14% in G3, G4, and G5 stages, respectively. Serum magnesium levels demonstrated a significant positive correlation with serum calcium and the calcium-phosphorus product, whereas no correlation was observed with serum phosphorus or iPTH levels.

Conclusion: Magnesium metabolic disorders progressively increase with CKD severity, with the highest prevalence observed in stage G5. These findings indicate that changes in serum magnesium levels may influence the development and progression of CKD-MBDs.

Keywords: non-dialysis-dependent chronic kidney disease, NDD-CKD, mineral metabolism disorders, secondary hyperparathyroidism, magnesium imbalance

Introduction

Chronic kidney disease (CKD) has emerged as a significant global health concern, with projections indicating that it will impose substantial economic burdens on society in the future.[1] CKD is associated with various complications, such as electrolyte imbalances, mineral metabolism disorders, and cardiovascular diseases. In clinical practice, potassium, sodium, chloride, calcium, and phosphorus are the electrolytes most frequently assessed. However, magnesium, despite being the second most abundant intracellular cation, is often overlooked.[2] Recent studies have shown that higher serum magnesium concentrations are associated with a decreased risk of death from heart failure, coronary heart disease, and stroke in non-dialysis-dependent CKD patients, particularly those in stages 4–5.[3] Magnesium plays critical physiological roles, including functioning as a biological competitor to calcium, serving as a cofactor in over 3,000 enzymatic reactions, and regulating ion channels responsible for transmembrane transport of potassium and calcium.[4,5] Consequently, magnesium is sometimes referred to as the "neglected cation" in clinical settings.

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The kidneys play a key role in maintaining magnesium balance, reabsorbing approximately 95% of circulating magnesium ions through the renal tubules. The ascending limb of the loop of Henle is responsible for approximately 60% of this reabsorption. Impaired renal function, particularly in patients with end-stage renal disease (ESRD), disrupts magnesium homeostasis, leading to variable increases in serum magnesium levels.[6] Clinical and experimental evidence indicates that magnesium deficiency contributes to complications associated with CKD, such as dyslipidemia and carotid intima-media thickening.[7] Furthermore, low serum magnesium levels have been associated with an increased incidence of cardiovascular events, particularly in patients with type II diabetes and hypertension, thereby negatively impacting prognosis and survival outcomes in CKD.[8,9]

Conversely, elevated serum magnesium levels have been linked to adverse outcomes, including abnormalities in bone metabolism, parathyroid dysfunction, and exacerbation of pruritus, and other conditions.[10] Magnesium ions play a key role in the pathophysiology of chronic kidney disease-mineral and bone disorder (CKD-MBD), renal osteodystrophy (ROD), and vascular calcification (VC).[6–10] Balanced regulation of serum magnesium levels may offer therapeutic benefits, particularly for patients with ESRD. However, reference ranges for serum magnesium levels vary across institutions, and there is limited clinical evidence to determine if current reference ranges are appropriate for patients with CKD. The clinical significance of magnesium homeostasis in this population remains poorly understood.

In this study, we retrospectively analyzed serum magnesium, calcium, phosphorus, and intact parathyroid hormone (iPTH) levels in patients with stage 3–5 non-dialysis-dependent CKD (NDD-CKD) (G3–G5) who were admitted to our hospital. Variations in calcium-phosphorus metabolism and parathyroid function were examined among patients with varying serum magnesium levels. The study aimed to elucidate the relationship between magnesium metabolism disturbances and the development of CKD-MBD, providing a theoretical basis for optimizing serum magnesium regulation in patients with NDD-CKD, particularly in the context of CKD-MBD diagnosis and treatment.

Methods

Participants

The study group included 156 patients with CKD in a stable condition who were admitted to Handan First Hospital between March 2017 and December 2019. Eligibility criteria included a confirmed diagnosis of stage 3–5 CKD based on the US definition criteria and complete clinical data. The optimized Modification of Diet in Renal Disease (MDRD) formula, validated for the Chinese population [$175 \times (\text{Scr, mg/dL})^{-1.234} \times \text{age}^{-0.179} \times (0.79 \text{ female})$], was used to calculate the glomerular filtration rate (GFR). Patients with an estimated GFR (eGFR) of < 60 mL/(min·1.73 m^2) who had not undergone dialysis were included in the study. The study was approved by Ethics Committee of the Handan First Hospital (No.HDYY-LL-KY2019-H07) and all participants provided informed consent.

Exclusion criteria were defined as follows: 1) history of parathyroid surgery; 2) severe infections; 3) severe malnutrition (albumin < 30 g/L); 4) severe anemia (hemoglobin < 60 g/L); 5) presence of malignant tumors; 6) severe cardiovascular conditions, including grade 3 chronic heart failure, history of coronary artery bypass grafting, coronary stent implantation, or malignant arrhythmias; and 7) diagnoses of multiple myeloma, primary hyperparathyroidism, or bone disease associated with glucocorticoid use.

Clinicopathological Data

General patient data, including sex, age, blood pressure, medical history, primary disease, and other clinical data, was retrieved from existing records. Patients were categorized into three groups based on their Kidney Disease Outcomes Quality Initiative (K/DOQI) CKD stage, categorized according to estimated eGFR values: CKD G3 (eGFR 30–59 mL/min/1.73 m²), CKD G4 (eGFR 15–29 mL/min/1.73 m²), and CKD G5 (eGFR < 15 mL/min/1.73 m²).

Serum magnesium levels were classified based on the hospital's normal reference range (0.75–1.02 mmol/L). Patients were further stratified into three groups based on serum magnesium levels: low magnesium (< 0.75 mmol/L), normal magnesium (0.75–1.02 mmol/L), and high magnesium (> 1.02 mmol/L). Serum magnesium levels were directly measured during the research process, and the grouping was based on these measurement results.

Diagnostic criteria for secondary hyperparathyroidism (SHPT) were applied according to the K/DOQI recommendations, with thresholds defined as follows: for CKD G3, iPTH > 70 pg/mL; for CKD G4, iPTH > 110 pg/mL; and for CKD G5, iPTH > 300 pg/mL.

Research Methodology

Biochemical data were obtained from fasting venous blood samples collected in the morning following patient admission. Routine blood analyses were conducted using a SYSMEX XN9000 automatic blood cell analyzer with a Beckman automatic biochemical analyzer (Micon Hishori, Japan). The determination of blood creatinine, urea nitrogen, calcium (Ca), phosphorus, magnesium, alkaline phosphatase, and albumin levels was conducted using a Beckman AU5821 automatic biochemical analyzer. iPTH levels were measured using a Roche cobas e411 electrochemiluminescence automatic immunoassay system. The albumin-corrected calcium level was calculated using the following formula:

$$\text{corrected calcium (mmol/L)} = \text{measured calcium (mmol/L)} + 0.02 \times [40 - \text{serum albumin (g/L)}].$$

Statistical Analyses

All patient data were compiled into a database and analyzed using the Statistical Product and Service Solutions (SPSS) software, version 23.0. Continuous variables with a normal distribution are presented as mean ± standard deviation (SD), while those with a non-normal distribution data were reported as the median and interquartile range (IQR). Categorical data are expressed as frequencies and percentages (%). Nonparametric tests were used for continuous data with unequal variances or non-normal distributions.

Correlation analyses were conducted using Pearson's or Spearman correlation coefficients, depending on the data distribution. Chi-squared tests were utilized to compare proportions, and a binary logistic regression model was applied to identify independent risk factors for VC. A threshold of $p < 0.05$ was considered statistically significant.

Results

General Characteristics of Patients with CKD G3–G5 Before Dialysis

Among the 156 patients included in the study, the male-to-female ratio was 1.48:1. Patient ages ranged from 23 to 90 years, with a mean age of 57.33 ± 1.19 years. Based on GFR levels, the distribution of patients across CKD stages was as follows: 39 patients (25%) in stage 3 (CKD G3), 44 patients (28.21%) in stage 4 (CKD G4), and 73 patients (46.79%) in stage 5 (CKD G5) (Table 1).

Serum Magnesium Levels and Incidence of Magnesium Metabolic Abnormalities in NDD-CKD G3–G5

Serum magnesium levels demonstrated a gradual increase with advancing CKD stages; however, these differences were not statistically significant (Table 2). The incidences of both hypomagnesemia and hypermagnesemia were observed to rise with disease progression, with significantly higher rates of these abnormalities in patients with NDD-CKD G5 compared to those in CKD G3 and CKD G4 ($p < 0.05$). Additionally, patients with CKD G4 exhibited a higher incidence

Table 1 Clinicopathological Characteristics

Characteristic	Number of Patients	Ratio %
Gender		
Male	93	59.62
Female	63	40.38

(*Continued*)

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Table 1 (Continued).

Characteristic	Number of Patients	Ratio %
Protopathy		
Chronic glomerulonephritis	78	50.00
Diabetic nephropathy	52	33.33
Hypertensive benign arteriolar glomerulosclerosis	3	1.92
Primary small vessel vasculitis	2	1.29
Polycystic kidney	7	4.49
Lupus nephritis	1	0.64
Others	13	8.33
Hypertension		
Yes	132	84.62
No	24	15.38
Diabetes		
Yes	79	50.64
No	77	49.36
CKD stage		
G3	39	25.00
G4	44	28.21
G5	73	46.79

Abbreviation: CKD, chronic kidney disease.

Table 2 Proportion of Patients with High and Low Magnesium Levels in Different Stages of CKD

Stage	Number of Patients	Serum Magnesium (mmol/L)	Normal Blood Magnesium (%) (SD)	Hypermagnesemia (%)	Hypomagnesemia (%)
CKD G3	39	0.89 (0.88, 0.91)	37 (94.87)	2 (5.13)	0 (0)
CKD G4	44	0.90 ± 0.03	35 (79.54)*	6 (13.64)	3 (6.82)*
CKD G5	73	0.92 (0.80, 1.07)	40 (54.79)*$^{\Delta}$	22 (30.14)*$^{\Delta}$	11 (15.07)$^{\Delta}$

Note: *Compared with CKD G3, P < 0.05; $^{\Delta}$Compared with CKD G4, P < 0.05. As there are two different expressions of values, the details have not been included. The line numbers of serum magnesium levels are distinguished by whether they conform to the normal distribution. If the serum magnesium level does not conform to the normal distribution, it is represented by the median (minimum, maximum), similar to stage 3 (G3). If the serum magnesium level conforms to a normal distribution, it is represented by the mean plus/minus standard deviation, similar to stage 4 (G4).
Abbreviation: CKD, chronic kidney disease.

of abnormal magnesium metabolism compared to those with CKD G3, with a statistically significant difference in hypomagnesemia, though no significant difference was observed for hypermagnesemia between these groups.

CKD-MBD in Patients with NDD-CKD G3–G5

Serum calcium levels progressively declined as CKD advanced, accompanied by a significant increase in iPTH levels (both $p < 0.01$). Serum phosphorus levels and calcium-phosphorus product levels were significantly elevated in patients with NDD-CKD G5 compared to those in CKD G3-G4 ($p < 0.01$).

Incidence of Low Calcium, High Phosphorus, and High iPTH Levels in Patients with NDD-CKD G3–G5

The incidence of hypocalcemia and hyperphosphatemia progressively increased from CKD G3 to G5 ($p < 0.01$). However, the incidence of secondary hyperparathyroidism (SHPT) peaked in CKD G4, with a significant decrease in CKD G5 pre-dialysis compared to CKD G3 and G4 ($p < 0.01$).

CKD-MBD and Correlation Analysis Among Different Serum Magnesium Groups

The serum calcium level was highest in the normal magnesium group, while serum phosphorus and iPTH levels were lowest in this group (Table 3). Significant differences were observed in these parameters when compared to the hypomagnesemia group. Compared to the hypermagnesemia group, the normal magnesium group exhibited significant differences in serum phosphorus and iPTH levels, though the difference in serum calcium levels between these groups was not significant. Serum phosphorus and iPTH levels in the hypermagnesemia group were significantly lower than those in the hypomagnesemia group, with no significant differences in serum calcium levels between these two groups.

Correlation analysis indicated a significant positive correlation between serum magnesium levels and both serum calcium levels ($r = 0.264$, $p < 0.001$) and calcium-phosphorus product levels ($r = 0.273$, $p < 0.001$). However, no correlation was identified between serum magnesium levels and either serum phosphorus or iPTH levels (Table 4).

Table 3 Comparison of Clinical Indices Between the Hypomagnesemia, Normal Magnesium, and Hypermagnesemia Groups

Clinical Index	Hypomagnesemia Group (n = 14)	Normal Magnesium Group (n = 112)	Hypermagnesemia Group (n = 30)	X^2	P
Gender					
Male	11	69	13	5.578	0.061
Female	3	43	17		
Age	59.5 ± 3.07	57.56 ± 1.45	55.47 ± 2.72	0.618	0.734
eGFR	10.75 ± 0.97	17.99 (9.55, 38.51)**	8.44 (6.08, 15.37)$^{\Delta}$	22.212	0.000
Serum calcium	1.86 ± 0.08	2.26 (2.15, 2.34)**	2.16 ± 0.03*	22.165	0.000
Serum phosphorus	2.15 ± 0.13	1.49 (1.17, 1.90)*	1.94 ± 0.08$^{\Delta}$	23.145	0.000
Serum iPTH	305.21 ± 27.84	161.00 (80.53, 283.58)*	275.85 (141.48, 497.00)$^{\Delta}$	15.835	0.000

Note: Compared with the hypomagnesemia group, *P < 0.05, **P < 0.001; compared with the normal magnesium group, $^{\Delta}$P < 0.05.
Abbreviations: iPTH, intact parathyroid hormone; eGFR, estimated glomerular filtration rate.

Table 4 Correlation Analysis of Serum Magnesium with Various Indices

Index	Correlation Coefficient	P
Serum calcium	0.175	0.029
Serum phosphorus	0.042	0.604
Calcium-phosphorus product	0.142	0.077
AKP	0.095	0.241
iPTH	0.034	0.674

Abbreviations: iPTH, intact parathyroid hormone; AKP, alkaline phosphatase.

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Discussion

Magnesium, the second most abundant divalent cation in serum, is a critical component of numerous physiological processes.[2,5,7] The role of magnesium in CKD has been increasingly recognized, particularly its involvement in mineral metabolism and CKD-MBD.[11] Despite its importance, it has received limited attention in clinical research, often being referred to as the "neglected cation." CKD is clinically associated with fluctuations in serum magnesium levels. Previous studies have highlighted the significant role of magnesium in CKD-MBD, ROD, and VC. However, further investigation is necessary to better understand the clinical implications of magnesium homeostasis disorders.

This study aimed to establish a theoretical framework for the management of serum magnesium levels in patients with NDD-CKD and to support advancements in the diagnosis and treatment of CKD-MBD. A retrospective analysis was conducted to assess serum levels of magnesium, calcium, phosphorus, and iPTH in patients with stage 3–5 CKD (G3–G5) (NDD-CKD).

Studies have shown that disturbances in magnesium levels are common in CKD, particularly in the later stages, where renal function decline impairs magnesium excretion.[3,4,6,11] Recent work at the Hemodialysis Center of Peking University People's Hospital also demonstrated a significant prevalence of hypermagnesemia in patients undergoing dialysis, with adverse outcomes observed in patients with elevated magnesium levels.[12] Correlation analysis demonstrated a significant positive association between serum magnesium levels and both serum calcium levels and the calcium-phosphorus product. No significant correlation was identified with serum phosphorus or iPTH levels. However, the role of serum magnesium in bone metabolism disorders was evident, underscoring the necessity of routine monitoring and targeted interventions for magnesium imbalances. Such measures may offer a novel therapeutic approach for managing these disorders. Furthermore, both hypermagnesemia and hypomagnesemia have been linked to severe complications such as arrhythmias, cardiovascular mortality, and worsening of CKD-MBD. The clinical implications of these disorders are crucial in understanding the management strategies for CKD patients, underscoring the need for close monitoring and targeted interventions.[6,13]

Magnesium is the second most prevalent bivalent intracellular cation. In healthy individuals, magnesium balance is maintained through dietary absorption and renal excretion. Homeostasis relies on dietary intake, gastrointestinal absorption, and renal regulation.[14] The kidneys manage circulating magnesium levels through glomerular filtration and tubular reabsorption, with extracellular magnesium balance determined by these processes along with dietary intake and renal excretion.

In patients with CKD, magnesium metabolism becomes dysregulated due to impaired renal excretion. As renal function declines, particularly in patients with ESRD, magnesium homeostasis is increasingly disrupted, resulting in variable serum magnesium levels depending on the severity of renal impairment. Studies indicate that hypermagnesemia is more prevalent in patients undergoing hemodialysis or peritoneal dialysis.[15,16] A study conducted at the Hemodialysis Center of Peking University People's Hospital reported that up to 81% of patients with hypermagnesemia were undergoing hemodialysis. However, none of these patients exhibited serum magnesium levels exceeding 2 mmol/L, and no cases of hypomagnesemia were reported.

The findings of this study indicated that, in pre-dialysis patients with CKD stages G3–G5, serum magnesium levels demonstrated a gradual increase, with hypermagnesemia observed as early as CKD G3. The prevalence of both hypomagnesemia and hypermagnesemia increased with CKD stage progression, reflecting a higher likelihood of magnesium metabolic disturbances in advanced CKD stages. Notably, pre-dialysis patients with CKD G5 exhibited significantly higher rates of both hypomagnesemia and hypermagnesemia compared to those with CKD G3 and G4. These results underscore the importance of close monitoring of serum magnesium levels beginning at CKD stage G3.

The association between increasing serum magnesium levels and progressive CKD stages is likely attributable to reduced renal magnesium excretion as kidney function declines. However, the concurrent increase in hypomagnesemia indicates that additional factors may influence magnesium homeostasis. Research indicates that as CKD progresses, urinary magnesium excretion may become insufficient to counteract gastrointestinal magnesium absorption, making dietary magnesium intake a key determinant of serum magnesium levels.[10] Furthermore, various pharmacological agents significantly influence magnesium metabolism. For instance, studies comparing patients with ESRD to healthy controls

have demonstrated that vitamin D facilitates magnesium reabsorption in the jejunum.[16] However, the use of magnesium-containing medications, including acid inhibitors, laxatives, and dialysate with high magnesium concentrations, has been associated with magnesium overdose and, in severe cases, fatal hypermagnesemia.[17] Conversely, excessive use of diuretics or low-magnesium dialysate solutions can lead to magnesium deficits, disrupting magnesium homeostasis.[18]

Vitamin D and diuretics are frequently prescribed for patients with CKD, while gastrointestinal symptoms often necessitate the use of acid inhibitors, complicating magnesium homeostasis further. Such factors are particularly relevant for patients undergoing regular dialysis, where reduced gastrointestinal symptoms may accompany the removal of toxins.

This study analyzed data from patients presenting to the hospital for the first time, none of whom had initiated dialysis. As CKD advances, gastrointestinal toxin load increases, exacerbating complications such as acidosis, particularly in the later stages of the disease. Hypomagnesemia in these cases may experience inadequate dietary magnesium intake, gastrointestinal dysfunction, and symptoms such as nausea, vomiting, or diarrhea. The pre-dialysis period is characterized by the frequent use of acid inhibitors and diuretics, which may contribute to an elevated risk of hypomagnesemia. The clinical implications of magnesium homeostasis disorders and the associated risks of hypermagnesemia and hypomagnesemia remain topics of ongoing debate.

Epidemiological studies have indicated that elevated serum magnesium levels are associated with improved survival rates in patients with CKD, particularly those with ESRD.[19–25] Conversely, hypomagnesemia has been linked to accelerated renal function decline in patients with CKD.[13,26] Bone metabolic disorders are a common complication of CKD, with increasing evidence highlighting the key role of magnesium ions in CKD-MBD, ROD, and VC.[19,21–23] Magnesium complexes, used as alternatives to aluminum-based phosphate binders, are considered effective and cost-efficient options for managing phosphorus levels.[27,28]

The relationship between serum magnesium and iPTH levels is complex. iPTH can elevate serum magnesium levels by enhancing gastrointestinal absorption and renal reabsorption. At the same time, magnesium is essential for iPTH synthesis, release, and maintaining tissue sensitivity to iPTH. Elevated serum magnesium levels can bind to calcium-sensing receptors on parathyroid cells, promoting calcium ion entry and suppressing iPTH secretion.[16] Studies in animal models, such as goat and sheep parathyroid glands perfused with varying magnesium levels, have demonstrated that a sharp increase in magnesium inhibits iPTH secretion. Similarly, human studies have demonstrated that magnesium levels influence the balance of calcium ions, iPTH, and 1,25-dihydroxy vitamin D. A reduction in magnesium levels disrupts this balance, leading to calcium ion dysregulation and decreased iPTH and vitamin D levels.[29] Restoration of magnesium levels has been shown to normalize iPTH secretion.

Navarro et al identified a significant negative correlation between serum magnesium and iPTH levels in patients undergoing continuous ambulatory peritoneal dialysis, indicating that high-magnesium peritoneal dialysis solutions may reduce iPTH levels and potentially prevent VC.[16] However, the relationship between serum magnesium and iPTH in patients undergoing hemodialysis remains contentious. Some studies report no significant effect of magnesium on iPTH in this population, while others, such as Huang et al, have found a positive correlation, with low magnesium levels reducing iPTH secretion.[30–32] Additional studies have reported a significant negative correlation between serum magnesium and iPTH in hemodialysis patients.[33,34]

In this study, the highest serum calcium levels were observed in the normal magnesium group, while the lowest serum phosphorus and iPTH levels were noted in this group. Both serum phosphorus and iPTH levels were significantly lower in the hypermagnesemia group compared to the hypomagnesemia group, although no significant differences in serum calcium levels were identified between these groups. Correlation analysis demonstrated a positive relationship between serum magnesium levels and both serum calcium and the calcium-phosphorus product, with no significant correlation observed between serum magnesium and serum phosphorus or iPTH levels.

Although serum magnesium plays a role in bone metabolic disorders, the underlying mechanisms are complex and influenced by multiple factors. Clinical management should prioritize regular monitoring of magnesium levels and consider appropriate interventions, as these may provide novel therapeutic opportunities for managing bone metabolic disorders in patients with CKD.

A limitation of this study is its single-center, cross-sectional design, with participants selected from hospitalized patients, often presenting with advanced stages of CKD. Consequently, the findings may not fully represent the broader

spectrum of patients with CKD. Future studies should involve larger, multi-center cohorts and conduct more detailed investigations to clarify the role of serum magnesium in bone metabolic disorders and its potential therapeutic implications.

Conclusion

This study explored the relationship between serum magnesium levels and mineral-skeletal metabolic disorder (CKD-MBD) in patients with chronic kidney disease (CKD). The research results show that with the progression of CKD, hypomagnesemia and hypermagnesemia are more common in advanced patients, and the serum magnesium level changes significantly at different CKD stages. Although this study did not directly correlate magnesium levels with clinical outcomes (such as cardiovascular events and mortality), it provided basic data for the potential role of magnesium metabolism in CKD management and emphasized the importance of regular monitoring of magnesium levels. Future studies should further explore the relationship between magnesium imbalance and clinical outcomes to improve the treatment and prognosis of CKD patients.

Abbreviations

CKD, Chronic Kidney Disease; CKD G3, Chronic Kidney Disease Stage 3; CKD G4, Chronic Kidney Disease Stage 4; CKD G5, Chronic Kidney Disease Stage 5; ESRD, End-Stage Renal Disease; NDD-CKD, Non-Dialysis Dependent Chronic Kidney Disease; CKD-MBD, Chronic Kidney Disease-Mineral and Bone Disorder; ROD, Renal Osteodystrophy; MBDs, Mineral and Bone Metabolic Disorders; VC, Vascular Calcification; MDRD, Modification of Diet in Renal Disease; K/DOQI, Kidney/Disease Outcomes Quality Initiative; SHPT, Secondary Hyperparathyroidism; GFR, Glomerular Filtration Rate; eGFR, Estimated Glomerular Filtration Rate; iPTH, intact Parathyroid Hormone; G3–G5, Stage 3–5 Chronic Kidney Disease; Ca, Calcium; P, Phosphorus; Mg, Magnesium; AKP, Alkaline Phosphatase; ALB, Albumin.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki (as was revised in 2013). The study was approved by Ethics Committee of the Handan First Hospital (No.HDYY-LL-KY2019-H07) and all participants provided informed consent.

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Disclosure

The authors declare no conflicts of interest in this work.

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