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Relationship between Serum Magnesium and Parathyroid Hormone Levels in Non-Dialysis Dependent Chronic Kidney Disease Patients

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ABSTRACT

Background: Chronic kidney disease–mineral and bone disorder reflects disrupted mineral handling and hormonal adaptation, culminating in secondary hyperparathyroidism. The association between serum magnesium and parathyroid hormone in non-dialysis chronic kidney disease (CKD) remains incompletely defined and warrants evaluation in relation to secondary hyperparathyroidism. **Objective:** To determine the correlation between serum magnesium and intact parathyroid hormone levels in CKD stage 4 and 5 patients. **Method:** This cross-sectional study was conducted at the Nephrology Outpatient Department, Lahore General Hospital, from June to December 2024. One hundred eighty-three adults aged 18–70 years with CKD stage 4 or 5 were enrolled using consecutive sampling. Serum magnesium and intact parathyroid hormone was measured. **Results:** The mean age was 49.3 ± 11.4 years and mean disease duration was 5.4 ± 2.0 years. Mean serum magnesium was 1.86 ± 0.19 mg/dL and mean intact parathyroid hormone was 268.7 ± 142.6 pg/mL. Hypomagnesemia was present in 19.7% of patients. Serum magnesium correlated inversely with intact parathyroid hormone ($r = -0.176$, $p = 0.017$), serum calcium ($r = -0.469$, $p < 0.001$) and alkaline phosphatase ($r = -0.478$, $p < 0.001$). Intact parathyroid hormone correlated with estimated glomerular filtration rate ($r = -0.482$, $p < 0.001$) and alkaline phosphatase ($r = 0.563$, $p < 0.001$). **Conclusion:** A weak inverse correlation between serum magnesium and intact parathyroid hormone was observed in non-dialysis chronic kidney disease patients, with lower magnesium levels associated with altered mineral metabolism patterns.

INTRODUCTION

Chronic kidney disease affects almost 10% of the general population globally, which counts approximately to 800 million individuals [1]. In Pakistan, reported prevalence is much higher has accounting for 21.2% approximately, this high prevalence is due to rising trends of type 2 diabetes mellitus, hypertension and renal stone disease in local population [2].

Chronic kidney disease is associated with multiple metabolic abnormalities, including disturbed mineral metabolism. Among mineral metabolism that are affected includes calcium, phosphate, and PTH and magnesium may also affect this hormonal adjustment [3]. Parathyroid hormone regulates calcium and phosphate balance, and its secretion and activity can be modified by the concentration of magnesium in the extracellular fluid; increased magnesium levels have been shown to inhibit PTH release through activation of the calcium-sensing receptor. As renal function deteriorates, magnesium homeostasis becomes less predictable: the body's magnesium balance depends on how much magnesium is absorbed from the diet, while diminished glomerular filtration and reduced tubular reabsorption limit the ability of the kidneys to excrete excess magnesium, and patients with impaired kidney function often experience decreased intestinal magnesium absorption.

Magnesium has a concentration-dependent relationship with parathyroid hormone. Mild magnesium deficiency is often associated with higher PTH, whereas high magnesium tends to reduce PTH release. In contrast, when magnesium falls very low, it tends to reduce PTH secretion and produce a functional hypoparathyroid picture [4,5]. PTH may also modify magnesium balance via tubular reabsorption and exchange with bone stores, although the mechanism by which PTH controls magnesium metabolism in bone is less understood [4,6]. A study reported no correlation between serum magnesium and PTH [7]. While another study demonstrated a weak inverse relationship between magnesium and PTH [8]. In another report, higher serum magnesium levels were linked with higher parathyroid hormone concentrations, indicating a positive correlation between magnesium and PTH [9].

The mixed findings across studies suggest that any link between serum magnesium and parathyroid hormone (PTH) is not well studied and may be influenced by several

factors that are not fully defined. Although magnesium can influence PTH biology, evidence in non-dialysis chronic kidney disease (CKD) remains limited. Understanding this relationship is important because interpretation of PTH in CKD is influenced by multiple concurrent biochemical disturbances, and magnesium abnormalities may be one of them. This study evaluated the association between serum magnesium and PTH among non-dialysis CKD patients to understand whether magnesium status contributes meaningfully to variability in PTH levels.

MATERIAL AND METHODS

Over a period of 7 months June 2024 to December 2024, this cross-sectional study was conducted at Nephrology department of Lahore General Hospital, Lahore. Before initiating the study the approval was obtained from the institutional ethical review board (approval no. 214/LGH/AMC), All participant were enrolled after taking consent through non probability consecutive sampling. Sample size was calculated assuming an anticipated correlation between serum magnesium and intact parathyroid hormone (r = 0.28), α = 0.0, and 90% power, as follows:

$$n = \left[\frac{Z_{\{\frac{\alpha}{2}\}} + Z_{\{\beta\}}}{0.5 \times \ln(\frac{1+r}{1-r})}\right]^2 + 3.$$

This calculated a sample size of 183 participants [9].

Adult patients of either sex with age ranges between 18–70 years were eligible if they had CKD stage 4 or 5 persisting for >3 months and were not receiving dialysis. Baseline laboratory investigations obtained within the preceding 3 months were required for enrollment. Patients were excluded if a concurrent condition was present that could independently alter magnesium balance or PTH concentrations, including autoimmune disease, active infection (including urinary tract infection), malignancy, acquired immunodeficiency syndrome, thyroid disease, severe malnutrition, chronic diarrhea, or malabsorption syndromes. Exclusion criteria also included primary hyperparathyroidism, documented hypomagnesemia requiring replacement, excessive alcohol intake, use of magnesium-containing supplements, and current therapy with agents known to affect magnesium metabolism (loop diuretics, proton pump inhibitors, aminoglycosides, β-adrenergic agonists, cisplatin, cyclosporine, active vitamin D preparations, and theophylline).

Medical history was obtained from each patient, documenting the duration of chronic kidney disease, comorbidities, and current medication use. Baseline investigations included complete blood count, serum calcium, phosphate, albumin, alkaline phosphatase, and renal function parameters (serum creatinine and blood urea nitrogen). Fasting blood samples were collected for intact parathyroid hormone measurement and analyzed using a chemiluminescent immunoassay (Roche Diagnostics, Mannheim, Germany) on a Cobas e analyzer. Serum magnesium was measured by the xylidyl blue method using an automated chemistry analyzer (Beckman Coulter AU480; Beckman Coulter, Brea, CA, USA). All laboratory analyses were performed in the hospital laboratory. Data was recorded using data collection proforma.

Statistical Analysis

All analyses were performed using SPSS, version 26. Quantitative variables were presented as mean ± standard deviation; while serum creatinine as median (interquartile range). Qualitative variables were reported in the form of counts and percentages. Correlation between serum magnesium, intact parathyroid hormone, and other biochemical parameters was evaluated using Pearson's correlation coefficient. Independent predictors of intact parathyroid hormone were evaluated using multivariable linear regression using sequential models. Results were considered statistically significant at ≤ 0.05.

RESULTS

The analysis included 183 non–dialysis-dependent CKD patients. Mean age was 49.3 ± 11.4 years, with males comprising 57.4% of the sample; 56.3% were aged 46–70 years. Mean disease duration was 5.4 ± 2.0 years. CKD stage 4 and stage 5 were identified in 65.0% and 35.0% of patients, respectively. Hypertension (42.6%), diabetes mellitus (27.9%), and cardiovascular disease (19.1%) were the most frequently documented comorbidities. Renal function measures showed a mean eGFR of 17.2 ± 6.6 mL/min/1.73 m^2 and a median creatinine of 3.20 mg/dL (IQR 2.70). Biochemical variables are reported in Table 1; mean serum magnesium was 1.86 ± 0.19 mg/dL and mean intact parathyroid hormone was 268.7 ± 142.6 pg/mL.

Table 1

Baseline Demographic, Clinical Characteristics, and Laboratory Parameters of Study Population (N=183)

Variable	Frequency (%) / Mean ± SD / Median (IQR)
Age (years)	49.3 ± 11.4
Age Groups	
• 18-45 years	80 (43.7%)
• 46-70 years	103 (56.3%)
Gender	
Male	105 (57.4%)
Female	78 (42.6%)
Duration of CKD (years)	5.4 ± 2.0
CKD Stage Distribution	
Stage 4	119 (65.0%)
Stage 5	64 (35.0%)
Co-morbidities	
Hypertension	78 (42.6%)
Diabetes Mellitus	51 (27.9%)
Cardiovascular Disease	35 (19.1%)
Renal Function Tests	
Estimated GFR (ml/min/1.73m^2)	17.2 ± 6.6
Serum Creatinine (mg/dL)*	3.20 (2.70)
Blood Urea Nitrogen (mg/dL)	89.7 ± 32.9
Serum Chemistry	
Calcium (mg/dL)	8.4 ± 0.5
Phosphate (mg/dL)	5.3 ± 1.2
Albumin (g/dL)	3.3 ± 0.4
Alkaline Phosphatase (U/L)	179.0 ± 63.4
Primary Study Variables	
Serum Magnesium (mg/dL)	1.86 ± 0.19

Intact Parathyroid Hormone (pg/mL)	268.7 ± 142.6

Participants were categorized as hypomagnesemia (serum magnesium <1.7 mg/dL) and Participants were stratified by serum magnesium into <1.7 mg/dL and 1.7–2.4 mg/dL; the 1.7–2.4 mg/dL category included 147 (80.3%) and the <1.7 mg/dL category included 36 (19.7%). Intact parathyroid hormone did not differ between categories (243.3 ± 119.5 pg/mL vs 274.9 ± 147.4 pg/mL; t = −1.194, df = 181, p = 0.234), with mean difference −31.6 pg/mL (95% CI −83.9 to 20.6).

Table 2

Correlation Analysis between Serum Magnesium, Intact Parathyroid Hormone, and Biochemical Parameters (N=183).

Variables	Pearson Correlation Coefficient (r)	P-value
Serum Magnesium Correlations		
Serum Magnesium vs Intact PTH	-0.176	0.017
Serum Magnesium vs Estimated GFR	0.162	0.028
Serum Magnesium vs Serum Calcium	-0.469	<0.001
Serum Magnesium vs Serum Phosphate	-0.017	0.823
Serum Magnesium vs Alkaline Phosphatase	-0.478	<0.001
Intact PTH Correlations		
Intact PTH vs Estimated GFR	-0.482	<0.001
Intact PTH vs Serum Calcium	0.112	0.131
Intact PTH vs Serum Phosphate	0.749	<0.001
Intact PTH vs Alkaline Phosphatase	0.563	<0.001

Pearson correlation reported a small inverse association between serum magnesium and intact parathyroid hormone, r value -0.176 with p value 0.017. Serum magnesium showed a weak positive association with estimated glomerular filtration rate, r value 0.162 with p value 0.028. Serum magnesium was inversely related to serum calcium, r value -0.469 with p value less than 0.001, and to alkaline phosphatase, r value -0.478 with p value less than 0.001; no significant association was observed with serum phosphate, r value -0.017 with p value 0.823. Intact parathyroid hormone showed a negative relationship with estimated glomerular filtration rate, r value -0.482 with p value less than 0.001, and positive relationships with serum phosphate, r value 0.749 with p value less than 0.001, and alkaline phosphatase, r value 0.563 with p value less than 0.001. The association with serum calcium was not significant, r value 0.112 with p value 0.131.

Table 3

Multivariate Linear Regression Analysis with Intact Parathyroid Hormone as Dependent Variable (N=183)

Predictor	Model 1 β (95% CI)	p value	Model 2 β (95% CI)	p value
Serum magnesium (per 1 mg/dL)	-	-	**-118 (-236 to +1)**	**0.052**
Age (per 1 year)	+3.3 (+1.2 to +5.3)	0.003	+2.5 (+0.9 to +4.1)	0.003
Male gender (vs female)	+30 (-9 to +68)	0.127	+10 (-23 to +43)	0.557
CKD duration (per 1 year)	+14 (+2.0 to +26)	0.023	-7.4 (-21 to +6.4)	0.292
Diabetes mellitus (yes vs no)	-15 (-55 to +25)	0.466	-23 (-54 to +7.8)	0.141
Hypertension (yes vs no)	+11 (-25 to +47)	0.544	+8.8 (-20 to +37)	0.541
Cardiovascular disease (yes vs no)	+1.9 (-46 to +50)	0.938	**-44 (-83 to -5.8)**	**0.025**
eGFR (per 1 mL/min/1.73 m^2)	-	-	-1.9 (-5.5 to +1.8)	0.311
Calcium (per 1 mg/dL)	-	-	+0.6 (-34 to +35)	0.971
Phosphate (per 1 mg/dL)	-	-	**+85 (+69 to +101)**	**<0.001**
Alkaline phosphatase (per 1 U/L)	-	-	+0.09 (-0.51 to +0.70)	0.764

Table 1: Outcome: intact parathyroid hormone (pg/mL); coefficients are unstandardized β. Model 1 adjusted for age, gender, chronic kidney disease duration, diabetes mellitus, hypertension, cardiovascular disease, and chronic kidney disease stage (R^2 = 0.338; adjusted R^2 = 0.311; F = 12.737; p <0.001; SE = 118.34); Model 2 further adjusted for estimated glomerular filtration rate, serum calcium, serum phosphate, alkaline phosphatase, and serum magnesium (R^2 = 0.626; adjusted R^2 = 0.602; F = 25.984; p <0.001; SE = 89.99).

Model 1 indicated higher intact parathyroid hormone with increasing age, longer chronic kidney disease duration, and stage 5 disease, while gender, diabetes mellitus, hypertension, and cardiovascular disease showed no independent association. After biochemical adjustment in Model 2, explanatory power increased and serum phosphate became the principal independent correlate of higher intact parathyroid hormone; age remained significant, cardiovascular disease showed an inverse association, and serum magnesium demonstrated a borderline inverse trend that did not reach statistical significance. Estimated glomerular filtration rate, calcium, and alkaline phosphatase were not independently associated in the fully adjusted model.

DISCUSSION

In study, the mean serum magnesium was 1.86 mg/dL and mean intact parathyroid hormone was 268.7 pg/mL. Hypomagnesemia (<1.7 mg/dL) was present in about one-fifth, while intact parathyroid hormone was similar across magnesium strata (p=0.234). Across continuous measures, lower magnesium aligned with higher intact parathyroid hormone (r=−0.176 with p=0.017) and with lower estimated glomerular filtration rate (r=0.162 with p=0.028). Magnesium also inversely related to serum calcium and alkaline phosphatase, whereas intact parathyroid hormone aligned strongly with serum phosphate and alkaline phosphatase, consistent with CKD-BMD. After adjustment, phosphate remained the principal independent correlate of intact parathyroid hormone [10].

Magnesium can influence parathyroid hormone secretion through calcium-sensing receptor signalling. Mild deficiency may facilitate hormone release, whereas marked depletion may impair secretion, indicating a non-linear response [11,12]. In chronic kidney disease progression, compensatory increases in fractional magnesium excretion maintain near-normal concentrations until kidney function declines

substantially, after which hypermagnesemia becomes frequent when clearance falls below approximately 10 mL/min [4]. This pattern was confirmed by Owiredu et al (2012), who reported progressive magnesium elevation from stage 1 to stage 5 and similar trend was observed in this study [13]. Phosphate retention and reduced calcitriol activity intensify parathyroid drive and may outweigh magnesium variation in stage 4 to 5 disease [14]. Inverse magnesium associations with intact parathyroid hormone have been reported before hemodialysis and in dialysis studies [12,15–17].Conversely, data have shown parallel rises in magnesium and parathyroid hormone as kidney function worsens, implying competing influences [13,18]. The calcium to magnesium ratio has been linked inversely with parathyroid hormone, suggesting regulation beyond magnesium. Intracellular magnesium status, diet, and medications may alter associations [19].

Non-dialysis chronic kidney disease studies have reported overall heterogeneous associations between serum magnesium and parathyroid hormone, reflecting differences in stage distribution and mineral milieu. In stages 3A–4 chronic kidney disease, serum magnesium averaged 2.0 mg/dL and parathyroid hormone 74 pg/mL, with no evidence of a linear magnesium–parathyroid hormone association [10]. In contrast, the present stage 4–5 analysis showed higher overall intact parathyroid hormone and a weak inverse correlation with serum magnesium, while comparisons between magnesium categories were not significant, suggesting that a narrow magnesium range limits discrimination when parathyroid stimulation is largely phosphate-driven, as also reported in stage 5 non-dialysis disease [18]. Across stages 1–5, both magnesium and parathyroid hormone rose progressively, with a positive regression relationship, indicating parallel magnesium retention and secondary hyperparathyroidism as filtration declines [13]. This pattern may mask inhibitory effects of magnesium on secretion, which is expected to be non-linear, with mild deficiency permitting higher secretion but severe depletion impairing release [11]. Population data have linked higher magnesium quartiles with lower estimated glomerular filtration rate, reinforcing bidirectional coupling between renal function and magnesium status [17,20].

In dialysis-adjacent settings, higher serum magnesium is more often linked with lower intact parathyroid hormone, though inconsistency persists [4]. In hemodialysis, an inverse association persisted after adjustment for calcium and phosphorus, and reviews reported strong negative correlations, especially in peritoneal dialysis [21,22]. Before first hemodialysis, serum magnesium remained independently inversely associated with intact parathyroid hormone after adjustment [23]. Similar directionality was reported in maintenance hemodialysis with an independent negative magnesium term in regression [24]. Null and positive findings have also been reported, implying dependence on dialysate magnesium, medications, and inflammation [7,9].

Multivariable modelling indicated that clinical associations with age, disease duration, and stage attenuated after mineral parameters were included, implying mediation through disordered mineral homeostasis [13]. In the fully adjusted model, serum phosphate remained the dominant independent correlate of intact parathyroid hormone, consistent with phosphate-driven secondary hyperparathyroidism and with stage 5 analyses in which phosphate retained significance while magnesium did not [14,18]. The magnesium term moved toward an inverse direction but remained borderline, suggesting limited incremental value of serum magnesium once calcium, alkaline phosphatase, and kidney function are considered [4,23]. This is concordant with non-dialysis data where serum magnesium was stable across parathyroid hormone strata [10]. Dialysis regression models similarly prioritized phosphate and alkaline phosphatase, with magnesium retaining only a smaller inverse coefficient [24].

The strengths of this study include a substantial non-dialysis chronic kidney disease sample size suitable for correlation analysis and multivariable modelling, supported internal consistency and enabled adjustment for principal biochemical covariates. Limitations were the cross-sectional design, which precluded assessment of temporal direction, single-center recruitment that may restrict external validity, and absence of vitamin D metabolites, fibroblast growth factor 23, and urinary magnesium indices. Medication use and dietary intake relevant to magnesium balance were not recorded in sufficient detail. Unmeasured confounding related to comorbidity severity cannot be excluded. Routine interpretation of serum magnesium together with phosphate, alkaline phosphatase, and intact parathyroid hormone may refine biochemical surveillance in advanced non dialysis chronic kidney disease, particularly when parathyroid hormone remains disproportionate after standard phosphate evaluation, while future work should use prospective repeated measurements to define temporal relationships, add urinary magnesium indices and vitamin D profiling to separate intake from excretion, and test magnesium correction strategies for effects on parathyroid hormone trajectory, bone turnover markers, and vascular calcification outcomes in practice.

CONCLUSION

In advanced non-dialysis chronic kidney disease, serum magnesium was inversely associated with intact parathyroid hormone, supporting a contributory role for magnesium status in secondary hyperparathyroidism. The overall biochemical pattern was compatible with chronic kidney disease mineral and bone disorder, with parathyroid hormone aligning with markers of mineral load and bone turnover. Routine interpretation of magnesium alongside mineral indices may improve clinical appraisal. Longitudinal work with urinary magnesium handling and treatment documentation is required to clarify causality and therapeutic relevance.

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