



Original Article

Correlation Between Serum Parathyroid Hormone Levels and Relevant Biochemical Parameters in Different Stages of Chronic Kidney Disease: A Period Cross-Sectional Study in A Tertiary Care Hospital, Chattogram, Bangladesh

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Abstract

Background: Distinguishing between acute kidney injury (AKI) and chronic kidney disease (CKD) in the emergency setting can be challenging, particularly when a patient's prior medical history and records are unavailable. Parathyroid hormone (PTH) has proven to be a valuable biomarker of CKD and is frequently measured because of its central role in the regulation of calcium and phosphate metabolism. However, reliance on PTH as a sole marker of CKD may be misleading under certain clinical conditions. Therefore, accurate interpretation of PTH values is essential when making initial management decisions. In this context, the present study was conducted to evaluate the relationship between PTH and other biochemical parameters in CKD and ESRD patients and to find correlation between serum PTH and biochemical parameters in the study group. **Materials and Methods:** A cross-sectional observational study was carried out in the department of Biochemistry in collaboration with the department of Nephrology, Marine City Medical College for a period of one year from January 2023 to December 2023. Two hundred and twenty-four (224) subjects aged more than 18 years were included in this study by purposive sampling method. The study group was divided into three different groups according to the different stages of CKD as stage III (eGFR 30-59 ml/min/1.73m²), stage IV (eGFR 15-29 ml/min/1.73m²) and stage V (eGFR <15 ml/min/1.73m²). **Results:** The study population demonstrated a predominance of male participants. The mean age of participants was 56.22 years, with an age range of 18 to 85 years. Mean serum creatinine, serum phosphate and serum PTH levels were markedly elevated, whereas mean serum calcium levels and estimated glomerular filtration rate (eGFR) were decreased. Serum PTH levels showed a progressive increase with advancing stages of CKD, rising from 94.5±54.7 pg/mL in Stage III to 160.3±128.6 pg/mL in Stage IV and 228.4±184.4 pg/mL in Stage V (p=0.0001). No significant difference in serum PTH levels was observed between male and female participants. A significant inverse correlation was noted between serum PTH and eGFR as well as between serum PTH and serum calcium levels. Conversely, serum PTH demonstrated a significant positive correlation with serum phosphate levels. **Conclusion:** The present study demonstrated a significant correlation between PTH levels and key biochemical parameters, including eGFR, serum calcium, and serum phosphate, among patients with CKD.

Keywords: Acute kidney injury (AKI), Chronic kidney disease (CKD), End stage renal disease (ESRD), Parathyroid hormone (PTH).

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Introduction

Hyperparathyroidism is a recognized contributor to kidney failure, and elevated parathyroid hormone (PTH) levels are commonly used as a marker of renal impairment¹⁻³. Kidney dysfunction is a frequent cause of emergency department presentations and acute medical admissions. In the emergency setting, a key diagnostic challenge is differentiating acute kidney injury (AKI) from chronic kidney disease (CKD), particularly in patients without prior medical records for comparison. Establishing the acuity of renal impairment is critical, as it directly influences initial management decisions, including the intensity of

fluid resuscitation and other therapeutic interventions.

PTH is routinely measured in the assessment of kidney disease, with elevated levels most often associated with CKD³. However, rare cases of markedly elevated PTH have been reported in patients with AKI in the absence of intrinsic parathyroid pathology⁴⁻⁵. PTH is a polypeptide hormone secreted by the parathyroid glands and plays a central role in calcium and phosphate homeostasis through its actions on bone, renal tubules, and gastrointestinal tract⁶.

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In CKD, impaired renal synthesis of vitamin D leads to reduced activation of the enzyme 1α -hydroxylase, a process in which PTH normally plays a regulatory role, resulting in decreased conversion of 25-hydroxyvitamin D to its active form, $1,25$ -dihydroxyvitamin D⁷. Progressive vitamin D deficiency subsequently reduces intestinal calcium absorption, stimulating compensatory parathyroid gland hyperplasia and the development of secondary hyperparathyroidism⁷. In advanced CKD, PTH is no longer able to maintain adequate renal phosphate excretion, leading to the characteristic biochemical profile of hypocalcemia, hyperphosphatemia, and low vitamin D levels⁸⁻¹⁰. As a result, calcium and vitamin D supplementation, along with phosphate binders, are commonly prescribed in patients with advanced CKD and those receiving dialysis¹¹.

Chronic kidney disease (CKD) is a pathophysiological process with diverse etiologies that leads to the progressive and irreversible loss of nephron number and function, frequently culminating in end-stage renal disease (ESRD)¹². ESRD represents a clinical condition characterized by the permanent loss of endogenous renal function^{12,13}. Diabetic nephropathy and hypertensive nephron sclerosis are the leading causes of both CKD and ESRD¹⁴.

Chronic kidney disease is associated with multiple disturbances in calcium, magnesium, and phosphorus metabolism. Secondary hyperparathyroidism is an early complication of chronic renal insufficiency and may occur at glomerular filtration rates (GFR) as high as 50-80 mL/min/1.73 m²¹⁵. This condition is generally attributed to hypocalcemia resulting from phosphate retention and impaired renal synthesis of vitamin D. Thus, an increase in serum phosphorus suppresses vitamin D production and stimulates parathyroid hormone (PTH) secretion. Elevated PTH, in turn, promotes urinary phosphate excretion in an effort to maintain normal serum calcium and phosphorus concentrations. Consequently, PTH plays a pivotal role in the regulation of calcium, magnesium, and phosphorus homeostasis. The primary target organs of Parathormone action are the kidneys and the skeleton¹⁶. Intact parathyroid hormone (iPTH) is a polypeptide with a molecular weight of approximately 9,425 kDa and consists of 84 amino acids. The biologically active region of the molecule resides in the amino-terminal (1-34) segment, whereas the middle and carboxyl-terminal (35-84) regions are biologically inactive but retain immunological activity¹⁷.

In light of these considerations, the present study was undertaken to estimate PTH levels and to assess their correlation with mineral metabolism in patients with CKD.

Materials and methods:

Study design: A cross-sectional observational study was conducted in the Department of Biochemistry from January 2023 to December 2023, in collaboration with the Department of Nephrology, at Marine City Medical College (MCM), Chattogram. Using purposive sampling, a total of 224 participants aged 18 years and above were recruited after meeting the inclusion criteria and obtaining approval from the Institutional Ethics Review Board (IERB) (memo no: MCMCH/IRB/2022/01). The study population was categorized according to chronic kidney disease (CKD) stages as follows: Stage III (eGFR 30-59 mL/min/1.73 m²), Stage IV (eGFR 15-29 mL/min/1.73 m²), and Stage V (eGFR <15 mL/min/1.73 m²). **Inclusion criteria:** Adults aged ≥ 18 years diagnosed with chronic kidney disease (CKD) stages III, IV, and V were included in the study. **Exclusion criteria:** Individuals who were HIV-positive or HBsAg-positive, bedridden patients, pregnant women, and subjects with a diagnosed malignancy were excluded from the study.

Data collection and Procedure: Data was collected using a pre-tested structured questionnaire that captured all relevant variables. Participants were recruited from the department of Nephrology, MCMCH, after obtaining informed consent, followed by brief history-taking and an explanation of the study objectives. Subjects were instructed to report to the Biochemistry Laboratory, MCMC, between 8:00 and 9:00 am. Venous blood (5 mL) was drawn from the median cubital vein under strict aseptic conditions. Blood samples were collected in red-top tubes. After clotting of blood, serum was separated, within an hour; by centrifugation at 3000-5000 cycles for 5 min. The samples were promptly transported to the Biochemistry Laboratory for the estimation of serum creatinine, serum parathyroid hormone (PTH), serum calcium, and serum phosphate. eGFR was calculated by Cockcroft and Gault equation $eGFR = (140 - \text{Age in years}) \times \text{Weight (Kg)} \times [0.85 \text{ if female}] / 72 \times [\text{Serum Creatinine (mg/dL)}]$.

Data Analysis: Data was analyzed using Microsoft Excel on Windows 10 and IBM SPSS (Statistical Package for the Social Sciences), version 23. Quantitative variables were expressed as mean $\pm$ standard deviation. Statistical significance across different stages of chronic kidney disease (CKD) was assessed using Student's *t*-test, Chi-square and ANOVA test, as appropriate. Data were presented in the form of tables, charts, and correlation graphs.

Results

A total of 224 participants aged 18 years and above were recruited using a purposive sampling method and categorized according to the stages of chronic

kidney disease (CKD): Stage III (eGFR 30-59 mL/min/1.73 m²), Stage IV (eGFR 15-29 mL/min/1.73 m²), and Stage V (eGFR <15 mL/min/1.73 m²). The study population showed a male predominance, with males constituting 54% and females 46% of the participants (Figure-1). The mean age was 56.22 ± 12.78 years, with an age range of 18 to 85 years. Mean serum creatinine (6.69 ± 4.46 mg/dL), serum phosphate (5.64 ± 1.98 mg/dL), and serum parathyroid hormone (PTH) levels (198.6 ± 170.3 pg/mL) were markedly elevated, whereas mean serum calcium levels (7.88 ± 0.96 mg/dL) and estimated glomerular filtration rate (eGFR) (13.35 ± 10.81 mL/min/1.73 m²) were reduced (Table-I). No significant difference in serum PTH levels was observed between male and female participants (Table-II). Serum PTH levels increased progressively with advancing CKD stages, measuring 94.5 ± 54.7 pg/mL in Stage III, 160.3 ± 128.6 pg/mL in Stage IV, and 228.4 ± 184.4 pg/mL in Stage V ($p = 0.0001$) (Table III). A significant inverse correlation was established between serum PTH and eGFR ($r = -0.31$, $p = 0.0001$) as well as serum calcium levels ($r = -0.25$, $p = 0.0001$) (Figure-2 & 3). Conversely, a significant positive correlation was observed between serum PTH and serum phosphate levels ($r = 0.21$, $p = 0.001$) (Figure-4).

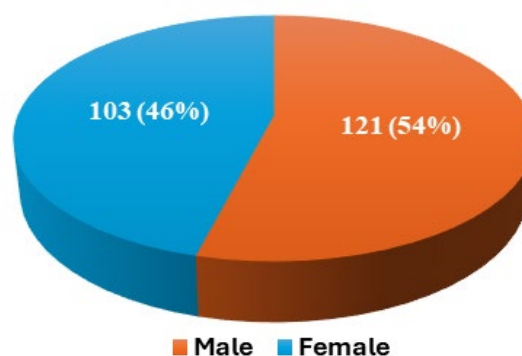


Figure-1: Pie chart showing the gender distribution in study subjects (n=224)

Table-I: Baseline characteristics of the study participants (n = 224)

Variables	Mean± SD	Range
Age (Years)	56.22 ± 12.78	18-85
S. Creatinine (mg/dl)	6.69 ± 4.46	1.42-21.5
eGFR ml/min/1.73m ²	13.35 ± 10.81	2.0-50.9
S. Calcium (mg/dl)	7.88 ± 0.96	4.7-11
S. Phosphate (mg/dl)	5.64 ± 1.98	2.9-19.9
Serum PTH (pg/ml)	198.6 ± 170.3	9.4-975

Table-II: Serum PTH concentrations between male and female cases (n = 224)

Variables	Male (n=121) Mean± SD (Range)	Female (n=103) Mean± SD (Range)	Significance
S. PTH (pg/ml)	192.7 ± 161.5 (11.6-780)	205.4 ± 179.8 (9.39-975)	$p=0.57^{ns}$

*ns=non-significant

Table-III: Distribution of serum PTH level according to CKD stages with ANOVA test significance (n=224)

Variable	CKD Stage	Number (n=224)	Mean± SD	Range	Significance
S. PTH (pg/ml)	Stage III	28	94.5 ± 54.7	17.6 - 230.6	$p=0.0001^s$
	Stage IV	43	160.3 ± 128.6	25 - 603.7	
	Stage V	153	228.4 ± 184.4	9.3 - 975	

*s=significant

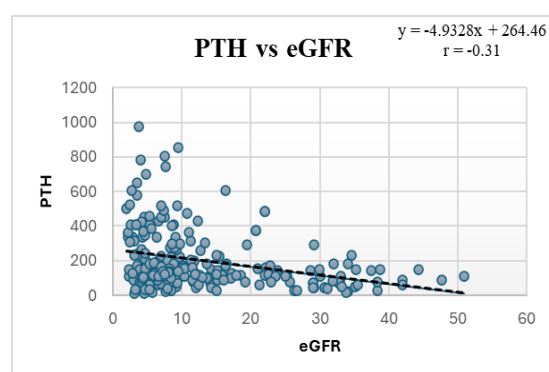


Figure-2: Scatter diagram shows inverse correlation between serum PTH & eGFR in the study cases ($r = -0.31$, $p=0.0001$)

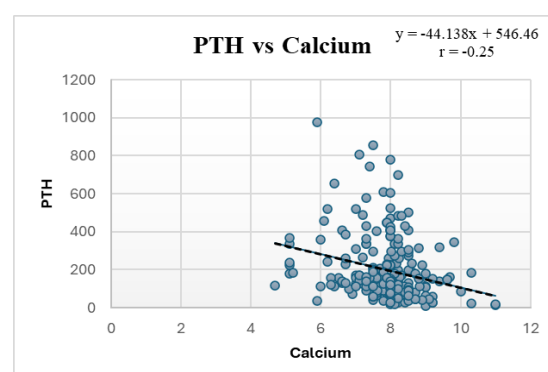


Figure-3: Scatter diagram shows inverse correlation between PTH and serum calcium in the study cases ($r = -0.25$, $p=0.0001$)

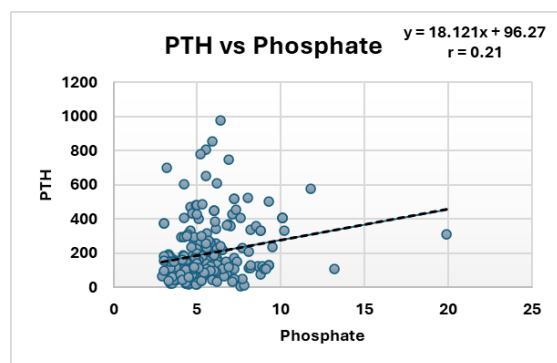


Figure-4: Scatter diagram shows a positive significant correlation between PTH and serum phosphate in the study cases ($r=0.21$, $p=0.001$)

Discussion

Chronic kidney disease (CKD) is a major global public health concern due to its strong association with multiple comorbidities, increased cardiovascular events, high mortality rates, and substantial healthcare costs¹⁸⁻¹⁹. The global prevalence of CKD ranges from 8% to 16%, depending on disease definitions, study design, and racial or ethnic groups¹⁸. In Bangladesh, the estimated national prevalence of CKD is 22.48%¹⁹. Identifying accurate risk factors associated with CKD is essential for early diagnosis and timely intervention to reduce disease burden.

In CKD, declining glomerular filtration rate (GFR) limits substrate delivery to renal 1α -hydroxylase, resulting in reduced production of 1,25-dihydroxyvitamin D. As renal function deteriorates, the tightly regulated interplay among vitamin D, fibroblast growth factor-23 (FGF-23), intact parathyroid hormone (PTH), and calcium-phosphorus-bone metabolism becomes increasingly disrupted. However, the precise relationships among intact PTH, calcium & phosphate metabolism, and CKD progression remain controversial¹⁸.

Serum PTH levels increased progressively with advancing CKD stage, measuring 94.5 ± 54.7 pg/mL in Stage III, 160.3 ± 128.6 pg/mL in Stage IV, and 228.4 ± 184.4 pg/mL in Stage V ($p = 0.0001$). A significant inverse correlation was observed between serum PTH levels and both eGFR and serum calcium levels. Conversely, a significant positive correlation was identified between serum PTH and serum phosphate levels. These findings are consistent with previously published studies^{1,12,18,19}.

The study revealed that elevated PTH levels have been associated with abnormalities in calcium-phosphate metabolism and was similar to that of the previous research works^{20,21}. Data from the United States National Health and Nutrition Examination Survey demonstrated a stepwise increase in the prevalence ratio of elevated PTH, reaching 2.30 and

4.69 among individuals with eGFR values of 45-59 and 3044 mL/min/1.73 m², respectively, compared with those with normal renal function (eGFR ≥ 60 mL/min/1.73 m²)²². Although alterations in PTH and mineral metabolism are well documented in moderate to advanced CKD and ESRD, the combined influence of PTH, calcium, and phosphate on the risk and early development of CKD in otherwise healthy individuals remains incompletely understood¹⁸⁻²².

Limitation

The study was a cross-sectional observational study of short duration with small sample size. Further, we did not do follow up of the patients, in order to evaluate time-dependent changes in PTH and eGFR precisely.

Conclusion

PTH is linked with derangements in the metabolism of calcium and phosphate and can contribute to a plethora of complications. PTH should be measured early in CKD, and appropriate interventions targeting calcium and phosphate should be implemented to prevent complications arising from PTH excess. Thus, the present study established a significant correlation between PTH and relevant biochemical parameters in CKD patients.

Recommendations

Further large-scale, multicenter studies with larger sample sizes are needed to generate generalizable results for the Bangladeshi population. Research across multiple tertiary hospitals in different regions would provide more representative data. Additionally, community-based interventions should be implemented to raise awareness of healthy lifestyle practices and promote better dietary choices.

Conflict of Interest

The authors declared that they have no conflicts of interest.

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References

1. Nwosu IF, Ibeson CE, Olawoye A, Kyaw H, Kumar K, Odigwe C, et al. Interpretation of Parathyroid Hormone Levels in Renal Impairment. *Cureus*. 2022; 14 (6): e25819. doi: 10.7759/cureus.25819.
2. Verdelli C, Corbetta S. Mechanisms in Endocrinology: Kidney involvement in patients with primary hyperparathyroidism: an update on clinical and molecular aspects. *Eur J Endocrinol*. 2017; 176 (1): R39-R52. doi: 10.1530/EJE-16-0430.

3. Xu Y, Evans M, Soro M, Barany P, Carrero JJ. Secondary hyperparathyroidism and adverse health outcomes in adults with chronic kidney disease. *Clin Kidney J.* 2021; 14 (10): 2213-20. doi: 10.1093/ckj/sfab006.
4. Pietrek J, Kokot F, Kuska J. Serum 25-hydroxyvitamin D and parathyroid hormone in patients with acute renal failure. *Kidney Int.* 1978; 13 (2): 178-85. doi: 10.1038/ki.1978.25..
5. Fuss M, Bagon J, Dupont E, Manderlier T, Brauman H, Corvilain J. Parathyroid hormone and calcium blood levels in acute renal failure. With special reference to one patient developing transient hypercalcemia. *Nephron.* 1978; 20 (4): 196-202. doi: 10.1159/000181222.
6. Habas E Sr, Eledrisi M, Khan F, Elzouki AY. Secondary Hyperparathyroidism in Chronic Kidney Disease: Pathophysiology and Management. *Cureus.* 2021; 13 (7): e16388. doi: 10.7759/cureus.16388.
7. Yuen NK, Ananthakrishnan S, Campbell MJ. Hyperparathyroidism of renal disease. *Perm J.* 2016; 20 (3): 15-127. doi: 10.7812/TPP/15-127.
8. Murray SL, Wolf M. Calcium and Phosphate Disorders: Core Curriculum 2024. *Am J Kidney Dis.* 2024; 83 (2): 241-56. doi: 10.1053/j.ajkd.2023.04.017.
9. O'Neill WC. Targeting serum calcium in chronic kidney disease and end-stage renal disease: is normal too high? *Kidney Int.* 2016; 89 (1): 40-5. doi: 10.1016/j.kint.2015.10.001.
10. Metzger M, Houillier P, Gauci C, Haymann JP, Flamant M, Thervet E, et al. Nephro Test Study Group. Relation between circulating levels of 25(OH) vitamin D and parathyroid hormone in chronic kidney disease: quest for a threshold. *J Clin Endocrinol Metab.* 2013; 98 (7): 2922-8. doi: 10.1210/jc.2013-1294.
11. Chan S, Au K, Francis RS, Mudge DW, Johnson DW, Pillans PI. Phosphate binders in patients with chronic kidney disease. *Aust Prescr.* 2017; 40 (1): 10-4. doi: 10.18773/austprescr.2017.002.
12. Arora K, Goyal G, Soin D, Kumar S, Arora H, Garg C. Correlation of Parathyroid Hormone Levels with Mineral Status in End-stage Renal Disease Patients. *Indian J Endocrinol Metab.* 2018; 22 (6): 735-9. doi: 10.4103/ijem.IJEM_279_18.
13. Bakris GL, Ritz E; World Kidney Day Steering Committee. The message for World Kidney Day 2009: hypertension and kidney disease: a marriage that should be prevented. *Clin J Am Soc Nephrol.* 2009; 4 (3): 517-9. doi: 10.2215/CJN.00080109.
14. Barsoum RS. Chronic kidney disease in the developing world. *N Engl J Med.* 2006; 354 (10): 997-9. doi: 10.1056/NEJMp058318.
15. Rahman MH, Hossain MM, Sultana S, Jamal CY, Karim MA. Correlation of serum parathormone level with biochemical parameters in chronic renal failure. *Indian Pediatr.* 2005; 42 (3): 250-4.
16. Levin A, Bakris GL, Molitch M, Smulders M, Tian J, Williams LA, et al. Prevalence of abnormal serum vitamin D, PTH, calcium, and phosphorus in patients with chronic kidney disease: results of the study to evaluate early kidney disease. *Kidney Int.* 2007; 71 (1): 31-8. doi: 10.1038/sj.ki.5002009.
17. Cunningham J, Locatelli F, Rodriguez M. Secondary hyperparathyroidism: pathogenesis, disease progression, and therapeutic options. *Clin J Am Soc Nephrol.* 2011; 6 (4): 913-21. doi: 10.2215/CJN.06040710.
18. Wang WH, Chen LW, Lee CC, Sun CY, Shyu YC, Hsu HR, et al. Association between Parathyroid Hormone, 25 (OH) Vitamin D, and Chronic Kidney Disease: A Population-Based Study. *Biomed Res Int.* 2017; 2017: 7435657. doi: 10.1155/2017/7435657.
19. Banik S, Ghosh A. Prevalence of chronic kidney disease in Bangladesh: a systematic review and meta-analysis. *Int Urol Nephrol.* 2021; 53 (4): 713-8. doi: 10.1007/s11255-020-02597-6.
20. Zhao G, Ford ES, Li C, Kris-Etherton PM, Etherton TD, Balluz LS. Independent associations of serum concentrations of 25-hydroxyvitamin D and parathyroid hormone with blood pressure among US adults. *J Hypertens.* 2010; 28 (9): 1821-8. doi: 10.1097/HJH.0b013e32833bc5b4.
21. Grønhøj MH, Gerke O, Mickley H, Steffensen FH, Lambrechtsen J, Sand NPR, et al. Associations between calcium-phosphate metabolism and coronary artery calcification; a cross-sectional study of a middle-aged general population. *Atherosclerosis.* 2016; 251: 101-8. doi: 10.1016/j.atherosclerosis.2016.06.001.
22. Muntner P, Jones TM, Hyre AD, Melamed ML, Alper A, Raggi P, et al. Association of serum intact parathyroid hormone with lower estimated glomerular filtration rate. *Clin J Am Soc Nephrol.* 2009; 4 (1): 186-94. doi: 10.2215/CJN.03050608.

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