Section: Miscellaneous

Original Research Article

OBSERVATIONAL CORRELATION BETWEEN SERUM CALCIUM AND PARATHYROID HORMONE LEVELS IN POSTMENOPAUSAL WOMEN

Kiran Malik[1], Devesh Kumar[2], Jay Ballabh[3]

[1]*Associate Professor, Department of Biochemistry, Autonomous State Medical College, Shahjahanpur, Uttar Pradesh, India.*
[2]*Professor, Department of Physiology, Autonomous State Medical College, Shahjahanpur, Uttar Pradesh, India.*
[3]*Professor, Department of Physiology, Teerthanker Mahaveer Medical College & Research Centre, Moradabad, Uttar Pradesh, India.*

Received *: 04/04/2025*
Received in revised form *: 16/05/2025*
Accepted *: 09/06/2025*

Corresponding Author:
Dr. Kiran Malik,
Associate Professor, Department of Biochemistry, Autonomous state medical college, Shahjahanpur, Uttar Pradesh, India.
Email: devkiran.2016@gmail.com

DOI: 10.70034/ijmedph.2025.3.251

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 1363-1367

ABSTRACT

Background: Postmenopausal women are at increased risk of disturbances in calcium homeostasis due to estrogen deficiency, reduced intestinal calcium absorption, and age-related changes in vitamin D metabolism. These changes may lead to hypocalcemia, which in turn stimulates parathyroid hormone (PTH) secretion. Understanding the relationship between serum calcium and PTH levels is vital for early identification of metabolic bone disorders in this vulnerable population. The aim is to evaluate the correlation between serum calcium and parathyroid hormone levels in postmenopausal women and to assess the prevalence of hypocalcemia and secondary hyperparathyroidism.

Materials and Methods: This observational, cross-sectional study was conducted in the Department of Biochemistry at a tertiary care teaching hospital and included 120 postmenopausal women aged ≥45 years. Participants were recruited consecutively based on predefined inclusion and exclusion criteria. Demographic and clinical data were collected, and fasting venous blood samples were analyzed for serum total calcium, albumin, and intact PTH levels. Corrected calcium was calculated using standard formulae.

Results: The mean age of the participants was 58.42 ± 6.17 years, with a mean BMI of 26.04 ± 3.75 kg/m². Hypocalcemia (corrected calcium <8.5 mg/dL) was observed in 25.83% of participants, and elevated PTH (>65 pg/mL) was found in 44.17%. Among hypocalcemic women, 77.42% exhibited elevated PTH levels, indicating secondary hyperparathyroidism. A significant inverse correlation ($r = -0.43$, $p < 0.001$) was noted between corrected serum calcium and PTH levels.

Conclusion: There exists a significant negative correlation between serum calcium and PTH levels in postmenopausal women. A high prevalence of hypocalcemia and secondary hyperparathyroidism highlights the importance of early screening and management of calcium and vitamin D deficiencies to preserve skeletal health in this population.

Keywords: Postmenopausal women, Serum calcium, Parathyroid hormone, Hypocalcemia, Secondary hyperparathyroidism

INTRODUCTION

Menopause marks a significant physiological transition in a woman's life, characterized by the cessation of ovarian function and a subsequent decline in estrogen production. This hormonal shift influences multiple metabolic pathways, particularly those involved in bone and mineral homeostasis. One of the key areas affected is calcium-phosphorus metabolism, which is intricately regulated by the interplay of vitamin D, parathyroid hormone (PTH), and serum calcium levels. Postmenopausal women are particularly vulnerable to disturbances in this regulatory axis due to age-related hormonal changes and decreased intestinal calcium absorption efficiency.[1]

The reduction in circulating estrogen during menopause accelerates bone resorption, decreases

calcium retention, and contributes to a negative calcium balance. Estrogen has a protective effect on bone by promoting osteoblastic activity and suppressing osteoclast-mediated bone resorption. Its deficiency leads to a cascade of metabolic alterations, including a potential decline in serum calcium levels, which can trigger compensatory mechanisms such as increased PTH secretion. PTH plays a pivotal role in maintaining calcium homeostasis by promoting calcium reabsorption in the kidneys, increasing intestinal calcium absorption indirectly via activation of vitamin D, and mobilizing calcium from bones.[2]

In postmenopausal women, the physiological demand for calcium increases, yet the efficiency of its absorption often diminishes with age. This phenomenon is largely attributed to reduced sensitivity of intestinal calcium transport mechanisms to 1,25-dihydroxyvitamin D, the active form of vitamin D.[3] Additionally, advancing age is associated with decreased renal function and alterations in vitamin D metabolism, further impairing calcium absorption and contributing to secondary hyperparathyroidism. The persistence of elevated PTH levels in this context may initially serve a compensatory role but can eventually exacerbate bone turnover and increase the risk of osteoporosis and fractures.[4]

Several studies have documented elevated PTH levels in postmenopausal women with low calcium intake or decreased serum calcium levels, even in the absence of overt parathyroid pathology. These findings underscore the importance of evaluating both calcium and PTH levels concurrently to assess the functional status of bone and mineral metabolism.

It is also essential to consider that serum calcium concentrations may not always reflect the true calcium status due to influences such as albumin levels, dietary intake, and renal handling. Corrected serum calcium, which adjusts for albumin variations, offers a more accurate assessment, particularly in older adults.[5]

The prevalence of hypocalcemia and elevated PTH among postmenopausal women is influenced by several factors including dietary habits, sun exposure, physical activity, and geographic location. In resource-limited settings and rural populations, poor nutritional intake and lack of awareness further contribute to the high incidence of calcium deficiency and its sequelae.[6] In such populations, serum PTH levels often reflect chronic compensatory responses rather than primary endocrine dysfunction. Understanding these patterns is crucial in formulating preventive strategies for osteoporosis and other metabolic bone diseases that disproportionately affect postmenopausal women.

It is also important to recognize the limitations of relying solely on serum calcium or PTH levels as isolated markers. Their interrelationship offers a more comprehensive insight into calcium balance and hormonal regulation. Elevated PTH in the setting of normocalcemia may suggest early stages of calcium deficiency or impaired vitamin D metabolism, while hypocalcemia with normal PTH may indicate a blunted parathyroid response, possibly due to nutritional deficits or chronic disease states. Hence, a combined evaluation of these markers can aid in the early identification of at-risk individuals and prompt appropriate nutritional or therapeutic interventions.[7]

Given the increasing life expectancy and the growing postmenopausal population worldwide, there is a pressing need to evaluate the biochemical profiles of these women in various regions. Early detection of alterations in calcium-PTH homeostasis can help mitigate the long-term consequences of bone loss and associated morbidity. This is particularly relevant in populations where routine screening for osteoporosis or metabolic bone disorders is not standard practice. An understanding of the serum calcium and PTH relationship in postmenopausal women can serve as a basis for initiating timely calcium and vitamin D supplementation, lifestyle modifications, and if necessary, pharmacological management.

MATERIALS AND METHODS

This observational, cross-sectional study was conducted in the Department of Biochemistry at a tertiary care teaching hospital. Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study. Written informed consent was obtained from all participants. A total of 120 postmenopausal women were recruited consecutively from the outpatient and inpatient departments based on the following inclusion and exclusion criteria.

Inclusion Criteria

- Women aged ≥45 years who had attained natural menopause (defined as cessation of menstruation for at least 12 consecutive months).
- Willingness to participate and comply with study procedures.
- Not currently on calcium, vitamin D, bisphosphonates, or hormone replacement therapy.

Exclusion Criteria

- Known history of chronic kidney disease, chronic liver disease, or parathyroid disorders.
- History of malignancy, gastrointestinal malabsorption syndromes, or medications affecting calcium metabolism (e.g., glucocorticoids, antiepileptics).
- Patients with surgical menopause or premature ovarian insufficiency.

Data Collection and Laboratory Analysis

Detailed demographic and clinical data were recorded for each participant, including age, duration since menopause, dietary calcium intake, and levels of physical activity. Anthropometric measurements such as height, weight, and body mass index (BMI) were also obtained using standardized procedures. Venous blood samples were collected from all enrolled women after an overnight fasting period of 8

to 10 hours. The collected serum samples were analyzed to measure total serum calcium using the Arsenazo III method on an automated biochemistry analyzer. Serum intact parathyroid hormone (PTH) levels were estimated using a chemiluminescent immunoassay technique. Additionally, serum albumin levels were measured to calculate corrected serum calcium, using the formula: Corrected Calcium = Measured Calcium + 0.8 × (4.0 − serum albumin in g/dL). All laboratory investigations were conducted in the central clinical biochemistry laboratory of the institution, adhering strictly to internal and external quality control protocols.

Statistical Analysis: Data were entered in Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Pearson's correlation coefficient was used to determine the association between serum calcium and PTH levels. A p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics [Table 1]

The mean age of the participants was 58.42 ± 6.17 years, indicating a relatively older postmenopausal cohort. The average duration since menopause was 9.63 ± 5.11 years, reflecting a wide range of postmenopausal periods among the study subjects. The mean height and weight were 154.76 ± 6.93 cm and 62.38 ± 8.27 kg respectively, with a mean body mass index (BMI) of 26.04 ± 3.75 kg/m², categorizing the average participant as slightly overweight. A significant proportion of the women (67.50%) reported low levels of physical activity, while only 32.50% engaged in moderate to high physical activity. Additionally, 75.83% of the participants had low dietary calcium intake, whereas only 24.17% met the recommended calcium intake, suggesting widespread nutritional inadequacy that may influence bone metabolism.

Serum Calcium Levels [Table 2]

Serum total calcium levels were suboptimal in a considerable proportion of the participants. Specifically, 49 women (40.83%) had total calcium levels below 8.5 mg/dL, indicating biochemical hypocalcemia, while the remaining 71 women (59.17%) had values within the normal range (8.5–10.5 mg/dL). When corrected for albumin, 31 participants (25.83%) still had corrected calcium levels below 8.5 mg/dL, confirming true hypocalcemia in about one-fourth of the cohort. The majority (74.17%) had corrected calcium within the normal physiological range. These results reinforce the need to assess albumin-adjusted calcium for accurate biochemical interpretation in postmenopausal women.

Serum Parathyroid Hormone Distribution [Table 3]

Serum PTH levels were elevated (>65 pg/mL) in 53 participants, accounting for 44.17% of the study population, whereas 67 women (55.83%) had PTH levels within the normal reference range. This high proportion of PTH elevation, despite a smaller number of women with hypocalcemia, suggests possible compensatory hyperparathyroidism or secondary hyperparathyroidism due to calcium or vitamin D deficiency. It reflects the body's homeostatic effort to restore calcium balance in response to hypocalcemia or related metabolic derangements.

Correlation Analysis Between Corrected Calcium and PTH [Table 4]

The mean corrected calcium in the entire study group was 9.12 ± 0.74 mg/dL, while the mean serum PTH level was 72.86 ± 29.15 pg/mL. A statistically significant negative correlation was observed between corrected serum calcium and PTH levels, with a Pearson's correlation coefficient (r) of –0.43 and a p-value <0.001. This inverse relationship indicates that as calcium levels decrease, PTH levels tend to rise, which is consistent with physiological feedback regulation governed by the calcium-sensing receptor in the parathyroid glands.

Secondary Hyperparathyroidism in Hypocalcemic Patients [Table 5]

Among the 31 women who were hypocalcemic based on corrected calcium levels, 24 (77.42%) had elevated serum PTH levels, indicating a high prevalence of secondary hyperparathyroidism in this subgroup. Only 7 hypocalcemic women (22.58%) had normal PTH levels. This observation reinforces the role of parathyroid hormone as a compensatory mechanism in the context of low serum calcium, attempting to restore calcium homeostasis. The high proportion of secondary hyperparathyroidism among hypocalcemic women further emphasizes the potential impact of calcium or vitamin D deficiency in this population.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 120).

Parameter	Mean ± SD / Frequency (%)
Age (years)	58.42 ± 6.17
Duration of Menopause (years)	9.63 ± 5.11
Height (cm)	154.76 ± 6.93
Weight (kg)	62.38 ± 8.27
BMI (kg/m²)	26.04 ± 3.75
Low Physical Activity	81 (67.50%)
Moderate/High Activity	39 (32.50%)
Low Dietary Calcium Intake	91 (75.83%)
Adequate Calcium Intake	29 (24.17%)

Table 2: Distribution of Serum Total Calcium and Corrected Calcium Levels

Calcium Level Category	Frequency (n)	Percentage (%)
Total Calcium <8.5 mg/dL	49	40.83%
Total Calcium 8.5–10.5 mg/dL	71	59.17%
Corrected Calcium <8.5 mg/dL	31	25.83%
Corrected Calcium 8.5–10.5 mg/dL	89	74.17%

Table 3: Distribution of Serum PTH Levels

PTH Level Category	Frequency (n)	Percentage (%)
PTH ≤ 65 pg/mL (Normal)	67	55.83%
PTH > 65 pg/mL (Elevated)	53	44.17%

Table 4: Correlation Between Corrected Calcium and Serum PTH Levels

Parameter	Mean ± SD	Correlation Coefficient (r)	p-value
Corrected Calcium (mg/dL)	9.12 ± 0.74	–0.43	<0.001
Serum PTH (pg/mL)	72.86 ± 29.15		

Table 5: Prevalence of Secondary Hyperparathyroidism Among Hypocalcemic Participants

Group	Frequency (n)	Percentage (%)
Hypocalcemic (Corrected Ca <8.5 mg/dL)	31	25.83%
Among them with PTH >65 pg/mL	24	77.42%
Among them with PTH ≤65 pg/mL	7	22.58%

DISCUSSION

The current study analyzed the correlation between serum calcium and parathyroid hormone (PTH) levels in 120 postmenopausal women. The findings revealed a considerable prevalence of hypocalcemia, elevated PTH levels, and a significant inverse relationship between the two parameters, highlighting an important biochemical pattern characteristic of postmenopausal endocrinological shifts.

In our cohort, the mean age was 58.42 ± 6.17 years, and the average duration since menopause was 9.63 ± 5.11 years. The mean BMI of 26.04 ± 3.75 kg/m² indicated a predominance of overweight individuals. A significant proportion of participants reported low physical activity (67.50%) and poor dietary calcium intake (75.83%). These findings are consistent with the observations made by Suchetha Kumari et al. (2010),[8] in a study conducted in Mangalore, where low physical activity and inadequate calcium intake were significantly associated with altered calcium homeostasis in postmenopausal women. Similarly, Varma et al,[9] (2005) reported that 70% of their postmenopausal cohort had insufficient calcium consumption, contributing to low bone mineralization.

Regarding calcium status, 49 women (40.83%) in the present study had total serum calcium levels below 8.5 mg/dL, while 31 women (25.83%) had corrected calcium levels below this threshold. Patwa et al,[10] (2017) in a study involving 100 postmenopausal women reported 38% had total serum calcium levels <8.5 mg/dL, which closely parallels our findings. Zaman et al,[11] (2022) also noted hypocalcemia in 28% of postmenopausal subjects, emphasizing that this is a common but often unrecognized issue. Importantly, we found that correction for albumin refined the true hypocalcemia rate, underscoring the need to interpret calcium biochemistry accurately.

Elevated PTH levels were found in 44.17% of our participants, with 53 women having serum PTH >65 pg/mL. This proportion is in line with findings from Zaman et al,[11] (2022) who reported elevated PTH in 41% of postmenopausal women, and Wihlborg et al,[12] (2022) who observed PTH elevation in 36% of women with recent fractures. The elevated PTH despite normocalcemia in many cases points to subclinical secondary hyperparathyroidism, likely due to chronic low calcium intake or vitamin D deficiency. A similar mechanism was proposed by Sarmidi et al,[13] (2008) who observed secondary hyperparathyroidism in 49% of vitamin D-deficient postmenopausal women, even when serum calcium was marginally low or borderline.

In our study, a statistically significant inverse correlation ($r = –0.43$, $p < 0.001$) was noted between corrected calcium and serum PTH. The mean corrected calcium was 9.12 ± 0.74 mg/dL, and the mean PTH was 72.86 ± 29.15 pg/mL. This physiological relationship mirrors findings reported by Felsenfeld and Rodriguez,[14] (1999) who described a tightly regulated feedback mechanism between serum calcium and PTH through calcium-sensing receptors. Vučeljić et al,[15] (2012) also demonstrated a significant inverse correlation ($r = –0.47$, $p <0.01$) between calcium and PTH in a Serbian cohort, reaffirming that such a trend is consistent across different populations.

Among the 31 participants with corrected hypocalcemia in our study, 24 (77.42%) had elevated PTH levels, indicating secondary hyperparathyroidism. These findings strongly align with the results from Neer et al,[16] (2001) where compensatory PTH elevation was found in 72% of women with biochemical hypocalcemia. Zaman et al,[11] (2022) similarly reported that 80% of

hypocalcemic postmenopausal women exhibited elevated PTH levels, suggesting that this compensatory mechanism is nearly universal.

CONCLUSION

This study demonstrates a significant inverse correlation between serum calcium and parathyroid hormone (PTH) levels in postmenopausal women, with a notable prevalence of hypocalcemia and secondary hyperparathyroidism. The findings highlight the impact of inadequate calcium intake and physical inactivity on mineral metabolism in this population. Regular biochemical monitoring, nutritional counseling, and timely intervention are essential to prevent long-term skeletal complications in postmenopausal women.

REFERENCES

1. EI-Shabrawy EM, Gabal KMA, Nassar SF. Hormonal role in osteoporosis among post-menopausal uremic women. J Am Scien. 2010;6(10):284–91.
2. Cerdà D, Peris P, Monegal A, Albaladejo C, Martínez de Osaba MJ, Surís X, et al. Increase of PTH in postmenopausal osteoporosis. Rev Clin Esp. 2011;211(7):338–43.
3. Eastell R, Yergey AL, Vieira NE, Cedel SL, Kumar R, Riggs BL. Interrelationship among vitamin D metabolism, true calcium absorption, parathyroid function, and age in women: Evidence of an age- related intestinal resistance to 1,25-dihydroxyvitamin D action. J Bone Miner Res. 1991;6(2):125–32.
4. Need AG, O'Loughlin PD, Morris HA, Horowitz M, Nordin BC. The effects of age and other variables on serum parathyroid hormone in postmenopausal women attending an osteoporosis center. J Clin Endocrinol Metab. 2004;89(4):1646–9.
5. Nordin BC, Need AG, Morris HA, O'Loughlin PD, Horowitz M. Effect of age on calcium absorption in postmenopausal women. Am J Clin Nutr. 2004;80(4):998–1002.
6. Qureshi HJ, Hussain G, Jafary ZA, Bashir MU, Latif N, Riaz Z. Calcium status in premenopausal and postmenopausal women. J Ayub Med Coll Abbottabad. 2010;22(2):143–5.
7. Achie LN, Olorunshola KV, Igashi J, Toryila JE. Assessment of the levels of serum parathyroid hormone in rural postmenopausal women in Zuturung district, Zangon Kataf Local Government Area, Kaduna State, Nigeria. J Afr Assoc Physiol Sci. 2021;9(2):130–7.
8. Suchetha Kumari N, Rosario BS, Gowda KM. Altered liver function and the status of calcium in postmenopausal women in and around Mangalore. Al Ameen J Med Sci. 2010;3(2):115–9.
9. Varma M, Paneri S, Badi P. Correlative study of bone related biochemical parameters in normal postmenopausal women and hyperglycemic postmenopausal women. Biomed Res. 2005;16(2):129–32.
10. Patwa CK, Jindani NI, Afroz S. Study of serum calcium levels in premenopausal women and postmenopausal women. MedPulse Int J Physiol. 2017;4(2):14–6.
11. Zaman SI, Tabassum H, Khan A, Shaila U. Correlation of Serum Parathyroid Hormone and Calcium Level among Pre- and Post-menopausal Women. J Curr Adv Med Res. 2022;9(1):16–20.
12. Wihlborg A, Bergström K, Gerdhem P, Bergström I. Parathyroid hormone disturbances in postmenopausal women with distal forearm fracture. World J Surg. 2022;46(1):128–35. doi:10.1007/s00268-021-06331-w. PMID: 34647149; PMCID: PMC8677684.
13. Sarmidi S, Setiyohadi B, Anggoro S. Vitamin D status and hyperparathyroidism in postmenopausal osteoporotic patients in Cipto Mangunkusumo Hospital Jakarta. Indones J Intern Med. 2008;5:35–42.
14. Felsenfeld AJ, Rodriguez M. Phosphorus, regulation of plasma calcium, and secondary hyperparathyroidism: a hypothesis to integrate a historical and modern perspective. J Am Soc Nephrol. 1999;10(4):878–90.
15. Vučeljić M, Ilić-Stojanović O, Lazović M, Grajić M. Vitamin D and parathyroid hormone in relation to bone mineral density in postmenopausal women. Vojnosanit Pregl. 2012;69(3):243–8.
16. Neer RM, Arnaud CD, Zanchetta JR, Prince R, Gaich GA, Reginster JY, et al. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med. 2001;344(19):1434–41.