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Original Research Article

Comparative Study of Serum Calcium and PTH (Parathyroid Hormones) In Pre-Menopausal and Post-Menopausal Women of Madhya Pradesh

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Conflict of interest: Nil

Abstract:

Background: Apart from menopause, there are many factors that cause osteoporosis, i.e., adverse effects of drug therapy, endocrine disorders, eatingdisorders, immobilization, marrow-related disorders, GIT, biliary tract or renal disorders, and cancer; hence, the exact cause of osteoporosis has to be ruled out.

Method: 25 premenopausal and 25 postmenopausal women were studied. Apart fromtheir anthropological parameters, their biochemical profile was studied. 5 ml of venousblood from each patient was collected. Serum calcium was measured by the colorimetric method (Erba kit). calcitonin and PTH were estimated by ELISA, and results were compared.

Results: In anthropological parameters, the age group of premenstrual and postmenstrual women had a significant p value ($p<0.001$). In the comparative studyof serum calcium and serum calcitonin, PTH had a significant p value ($p<0.001$).

Conclusion: In the present pragmatic study, it is concluded that decreased calcium has to be increased by calcium supplements and a nutritious diet to prevent osteoporosis and postmenopausal syndrome.

Keywords: Calorimetry, Erba-kit, ELISA, Anthropometric, Mineralization, Osteoporosis.

Introduction

The quality, quantity, and ratio of bone mineralization and turnover depend upon several hormones. Parathyroid hormone (PTH) induces the resorption of calcium from the bone and maintains the serum calcium level. Bone strength is predicted by both bone mineral density and bone architecture. Osteoporosis is a disorder characterized by compromised bone strength, which increases the risk of fragility fractures.

Osteoporotic fractures impair mobility, independence, and quality of life. Fracture of the hip also increases mortality by up to 20% [1]. The diagnostic criteria for osteoporosis are defined in terms of bone mass, but the true clinical consequences are the fractures.

The classical osteoporosis fractures are in the wrist, the upper arm, the spine, the ribs, the pelvis, and the hip [2].

The prevalence of osteoporosis increases with age. In the decade following menopause, most women experience rapid bone loss due to estrogen deficiency. The most common etiologies of osteoporosis in women are thus estrogen deficiency and later on in life the functional changes caused by aging. However, there are numerous causes of secondary bone loss, including adverse effects of drug therapy, endocrine disorders, eating disorders, immobilization and marrow-related disorders, disorders of the gastrointestinal or biliary tract, renal disorders, and cancer [3,4].

Hence, an attempt is made to evaluate the serum calcium, serum calcitonin, and PTH levels in both premenopausal and postmenopausal women to justify the causes of osteoporosis and bone fractures.

Material and Method

50 adult women visited the Chirayu Medical College Hospital in Bhopal, MadhyaPradesh, referred by the orthopaedic, obstetrics, and gynecology department were studied.

Inclusion Criteria: Pre- and postmenopausal women who gave their consent in writing for the study were selected.

Exclusion Criteria: The patients who underwent thyroid surgery and were already on treatment and nutritional supplements were excluded from the

study.

Method:

The majority of women belonged to middle socio-economic status. 25 premenopausal and 25 post-menopausal women were classified for the comparative study. The previous history of every patient was recorded. Anthropological parameter included BMI was calculated based on height (cms) and weight (Kg).

The biochemical profile was studied by collecting 5 ml of venous blood from each patient. Serum calcium was measured by the calorimetric method (Erba kit). Serum parathyroid hormone and calcitonin were estimated by enzyme-linked immunosorbent assay (ELISA), and the results were noted.

The duration of the study was April 2023 to November 2023.

Statistical analysis: Anthropological parameters and biochemical parameters in both premenopausal and postmenopausal were compared with the t test. The statistical analysis was carried out using SPSS software.

Observation and results

Table 1: Comparison of anthropological parameters in premenopausal andpostmenopausal women

- Age: mean value: 36 (± 3.2) in premenopausal women, 53 (± 2.3) in postmenopausal women; t test: 35.5; p<0.001 (p value is highly significant).
- Height: mean value: 152.60 (± 4.90) in premenopausal women, 153.30 (± 5.10) in postmenopausal women; t test: 0.21; p > 0.83.
- Weight (kg): 67.05 (± 9.82) in premenopausal women, 64.10 (± 10.32) inpostmenopausal women; t test: 1.03; p > 0.30.
- BMI: 26.5 (± 1.5) in premenopausal women, 26.2 (± 1.6) in postmenopausal women; t test: 0.68; p > 0.49

Table 2: Comparison of serum calcium, PTH, and calcitonin levels in premenopausal and postmenopausal women

- Serum calcium (mg/dl): 9.15 (± 0.90) in premenopausal women, 8.10 (± 1.10) in postmenopausal women; t test: 3.69 and p<0.001 (p value is highlysignificant).
- Serum calcitonin (pg/ml): 6.6 (± 1.80) in premenopausal women, 5.2 (± 0.80) in postmenopausal women; t test: 3.53; p value: highly significant (p<0.001).
- Serum PTH (pg/ml): 32.25 (± 8.40) in premenopausal women, 57.15 (± 19.20)in postmenopausal women; t test: 5.94 and p<0.001 (p value is highlysignificant).

Table 1: Comparison of Anthropometric parameters in premenopausal andpostmenopausal women

Parameters	Premenopausal mean value (25)	Postmenopausalmean value (25)	t test	p value
Age years	36 (±3.2)	53 (±2.3)	35.5	P<0.001
Height	152.60 (±4.90)	152.30 (±5.11)	0.21	P<0.001
Weight (kg)	67.05 (±9.82)	64.10 (±10.32)	1.03	p>0.30
BMI	26.5 (± 1.5)	26.2 (± 1.6)	0.68	p>0.49

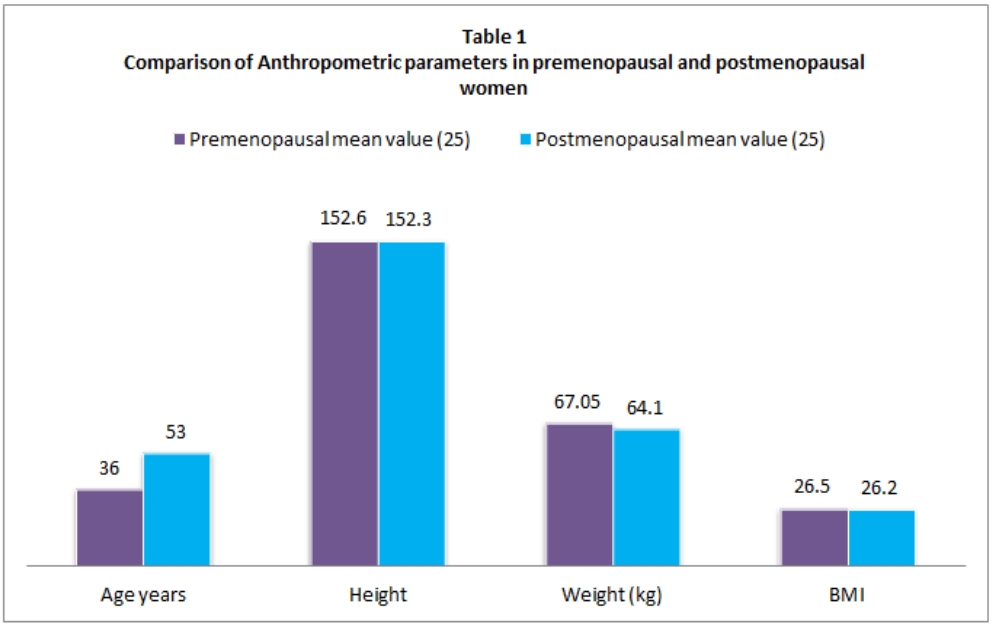

Figure 1: Comparison of Anthropometric parameters in premenopausal and postmenopausal women

Table 2: Comparison of serum calcium PTH and calcitonin in premenopausal andpostmenopausal women

Parameters	Premenopausal mean value (25)	Postmenopausal mean value (25)	t test	p value
Serum calcium (mg/dl)	9.15 (± 0.90)	8.10 (± 1.10)	3.69	P<0.001
Serum calcitonin(pg/ml)	6.6 (±1.80)	5.2 (±0.80)	3.53	P<0.001
Serum PTH pg/ml	32.25 (±8.40)	57.15 (±19.20)	5.94	P<0.001

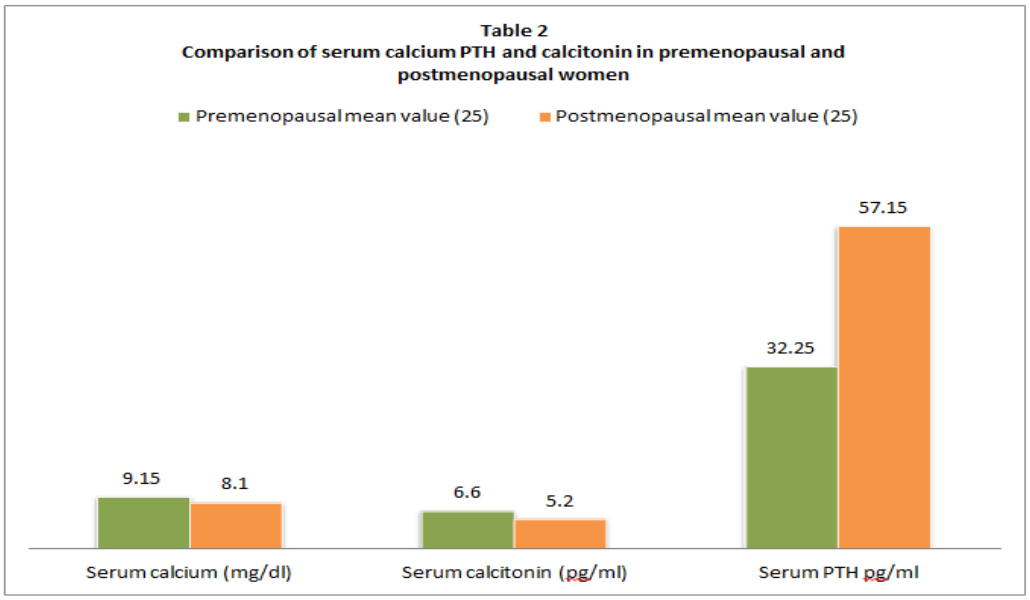

Figure 2: Comparison of serum calcium PTH and calcitonin in premenopausal andpostmenopausal women

Discussion

Present comparative study of serum calcium and parameters of thyroid hormone (PTH) in premenopausal and postmenopausal women in the Madhya Pradesh population. In an anthropological study, the age (years) of premenopausal women was 36 (± 3.2) and 53 (±2.3) in postmenopausal women, the t test was 35.5, and the p<0.001 p value was highly significant (Table 1). In comparison of serum calcium 9.15 (± 0.90) in premenopausal and 8.10 (± 1.10) in postmenopausal, the t test was 3.69 and p<0.00. Serum calcitonin was 6.6 (± 1.80) in premenopausal women and 5.2 (± 0.80) in postmenopausal women; the t test was 2.5 and p<0.001. Serum PTH was 32.05 (± 8.40) in premenopausal women and 57.15 (± 19.20) in postmenopausal women; the t test was 5.94 and p<0.001 (p value was highly significant) (Table 2). These findings are more or less in agreement with previous studies [5,6,7].

PTH is most responsible for maintaining serum ionized calcium concentration within a narrow range through its actions in the kidney to stimulate renal tubular calcium reabsorption and the conversion of 25-hydroxy vitamin D to 1,25-dihydroxy vitamin D, thereby increasing calcium absorption, and in the bone to increase bone turnover (8). PTH acts in bone to increase bone. PTH acts in bone to increase the number and activity of osteoblasts and increase bone turnover. With sustained elevations in PTH, osteoclastic activity could exceed that of osteoblasts, which results in the net release of calcium from bone and a decrease in bone marrow density (BMD) and bone strength.

In healthy postmenopausal women, there was a decrease in calcium absorption, and that decrease could be due to a decline in either the active calcium transport or diffusion components of the calcium absorption system. It was also found that urinary calcium was significantly higher in postmenopausal women, and it was due to reduced tubular resorption [9]. It is also reported that serum calcium levels were significantly reduced in postmenopausal women due to decreased estrogen levels, resulting in increased synthesis of cytokines by osteoblasts, monocytes, and T cells, which stimulated bone resorption [10]. The low estrogen levels lead to low calcium levels in the body, which in turn increase PTH levels, which further lead to the release of calcium ions from bones [11]. This leads to the weakening of bones and makes them more susceptible to diseases like osteoporosis and fractures. Parathyroid hormone is an important hormone in calcium turnover, with its main function being the maintenance of the calcium level in extracellular fluid. The secretion of parathyroid hormone is stimulated by hypocalcemia.

Decrease in hormones in females, especially estra-

diol, suppresses the intestinal absorption of calcium.

Summary and Conclusion

In the comparative study, postmenopausal women have low calcium levels and high serum PTH hormone, which leads to osteoporosis and fractures. Hence, calcium supplementation and a calcium nutrition diet are advocated for postmenopausal women. In severe hypocalcium, HRT (hormonal replacement therapy) is also recommended under gynecological supervision. But this study further demands patho- physiological, environmental, genetic, and hormonal studies because the exact pathogenesis and factors that cause osteoporosis are still unclear.

Limitation of Study:

Owing to the tertiary location of the research center, the small number of patients, and the lack of the latest technologies, we have limited findings and results.

This research paper has been approved by the ethical committee of Chirayu Medical College and Hospital, Bhopal, Madhya Pradesh (462030).

References

1. Finkelstein JS: Osteoporosis in Cecil RL Goldman L., Bennet JC, Editors Cecil Textbook of Medicine, 21st edition, Philadelphia, PA, WB Sounders Co. 2000; 1307–1373.
2. Gardner MJ, Demetrakopoulous D, Shinde MK: Osteoporosis and skeletal fractures, HSS J. 2006; 2: 62–69.
3. Garnero P, Delmas PD: Contribution of bone mineral density and bone turnovermarkets to the estimation of the risk of osteoporotic fracture in postmenopausalwomen J. Musculo-skeletal Neuronal Interaction 2004; 4 (1); 50–63.
4. Surya YP, Ashalata K: Study of bone markers (serum calcium, serum phosphorous, and serum alkaline phosphate) in postmenopausal women J. Musculo. Skeletal Neuronal Interact 2004; 4(1); 50–63.
5. Nordin BC, Need AG: Effect of age on calcium absorption in postmenopausal women. The American J. of Clinical Nutrition 2004 80 (4); 998–1002.
6. Sheweita S, Khoshal K: Calcium metabolism and oxidative stress in bone fractures: role of anti-oxidants. Curr. Drug Metab. 2007; 8; 519–25.
7. Dawson Hughers B: Effects of calcium and vitamin D supplementation on bonedensity in men and women 65 years of age or older N. Engl. J. Med. 1997; 337;670–76.
8. Khosla S., Elezabeth J.: Effects of age and estrogen status on serum parathyroid hormone levels and biochemical markers of bone turnover in women Clin. Endopcrinol. Metabol. 1997; 82; 1520–7.
9. Taboulet J, Prenkian M: Calcitonin receptor polymorphism fracture risk in postmenopausal women. Human mole Genetics 1998; 7; 2129–33.
10. Safi S, Hassikou H: Severe primary hyperparathyroidism and Nit. D deficiency Ann. Endocrinol. 2004; 65 (3); 226-32.
11. Bilez JP: Primary hypothyroidism J. Clin. Endocnnol. Metab. 2018; 103; 3993– 4004.