

Hyperprolactinemia-Associated Changes in Vitamin D and Electrolyte Levels in Osteoporotic Women

Shahad Saad Khudhair^{1*}, Wifaq Jabori Al-Bazii¹, and Mustafa Waleed Yahya²

¹Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Karbala, Iraq

²College of Medicine, University of Karbala, Karbala, Iraq

*Corresponding author

Shahad Saad Khudhair: shahd.s@s.uokerbala.edu.iq

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Abstract

Background: Hyperprolactinemia is a known endocrine disorder that adversely affects bone metabolism. Its relationship with vitamin D status and serum electrolytes in osteoporotic women remains poorly characterized.

Methodology: A cross-sectional comparative study was conducted on 81 women with confirmed osteoporosis: 30 with normal prolactin levels and 51 with hyperprolactinemia. Serum levels of prolactin, vitamin D, calcium, sodium, potassium, and chloride were measured and compared using independent t –tests.

Results: Hyperprolactinemic women showed significantly lower vitamin D (18.66 vs 25.97 ng /mL, $p<0.0001$) and markedly reduced levels of all measured electrolytes ($p<0.001$) compared to normoprolactinemic women.

Conclusion: Hyperprolactinemia is associated with significant reductions in vitamin D and electrolyte concentration in osteoporotic women, suggesting a multifactorial mechanism in prolactin – induced bone loss that warrants clinical attention.

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التغيرات المرتبطة بفرط بروتين الدم في مستويات فيتامين د والكهارل لدى النساء

المصابات بهشاشة العظام.

شهد سعد خضير المعموري، وفاق جبوري البازي ومصطفى وليد يحيى

الخلاصة

المقدمة: يُعد فرط بروتين الدم اضطراباً غذائياً صمّوياً معروفاً يؤثر سلباً في استقلاب العظم. ومع ذلك، لا تزال العلاقة بينه وبين حالة فيتامين د ومستويات الكهارل في المصل لدى النساء المصابات بهشاشة العظام غير مفهومة بشكل كافٍ.

طرق البحث: أُجريت دراسة مقطعية مقارنة على 81 امرأة مصابة بهشاشة العظام المؤكدة، شملت 30 منهن بمستويات طبيعية للبروتين و 51 منهن بمستويات مرتفعة (فرط بروتين الدم). تم قياس مستويات البروتين، فيتامين د، الكالسيوم، الصوديوم، البوتاسيوم، والكلوريد في المصل، ثم قورنت باستخدام اختبار t-test للعينات المستقلة.

النتائج: أظهرت النساء ذوات فرط بروتين الدم انخفاضاً ملحوظاً في مستويات فيتامين د (18.66 مقابل 25.97 نانوغرام/مل، قيمة احتمالية < 0.0001)، وكذلك انخفاضاً واضحاً في مستويات جميع الكهارل المقاسة (قيمة احتمالية < 0.001) مقارنة بالنساء ذوات المستوى الطبيعي للبروتين.

الاستنتاج: يرتبط فرط بروتين الدم بانخفاضات جوهرية في مستويات فيتامين د والكهارل لدى النساء المصابات بهشاشة العظام، مما يشير إلى وجود آلية متعددة العوامل في فقدان العظم الناجم عن البروتين، وهو ما يستدعي اهتماماً سريرياً.

1. Introduction

Osteoporosis is a systemic disease characterized by the loss of bone mass and microarchitectural impairment, which leads to a higher susceptibility of bone to fracture (Kanis JA et al. 2019; Eastell R et al. 2019). Osteoporosis is now considered a major public health problem globally, especially in post-menopausal women; bone loss is due to a reduction in estrogen level but may be aggravated or can occur independently due to endocrine abnormalities in this population (Cosman F., Et al. 2014), (LeBoff MS., Et al. 2022). Hyperprolactinemia is a condition defined as elevated serum prolactin levels (>25 ng/ml) in women and it is the most common endocrine disorder with a prevalence in the general population of approximately 0.4% but significantly higher in women of reproductive age (Melmed et al. 2011) (Klibanski 2010) Prolactin plays multiple roles in the body including the regulation of milk production, immune responses and skeletal health (Seriwatanachai D., et al. 2008) (Bernard V., Et al. 2019). Elevated prolactin suppresses GnRH at the level of the hypothalamus, leading to suppressed gonadal function and lower estrogen level (Greenspan SL., Et al. 1989), (De Rosa M., Et al. 2004). This hypoestrogenic condition is known to accelerate bone resorption and suppress bone formation. Moreover, recent studies demonstrate that prolactin receptors can be found directly on osteoblasts and osteoclasts, hence prolactin directly acts on bone tissue independently of hypogonadotropic effect (Abe E., Et al. 2003) (Bole-Feysot C., Et al. 1998). Vitamin D: Vitamin D is a fat-soluble vitamin and one of the key regulators of calcium homeostasis and bone mineralization. Hypovitaminosis D is highly prevalent in the presence of endocrinopathies and is itself associated with increased fracture rate and lowered bone mineral density (Holick MF., Et al. 2011; Reid IR et al. 2020). Electrolytes: the essential role of electrolytes, namely, sodium, potassium, chloride, and calcium, to the structural integrity of bone and in neuromuscular function cannot be overemphasized (Cianferotti L., Et al. 2014), (Fuleihan GE., Et al. 2015). The role of vitamin D deficiency is specifically interesting in the context of the Middle Eastern and Iraqi women; full body coverage, limited outdoor exposure, and particular habits may lead to significantly lower cutaneous synthesis of vitamin D despite ample sunshine. Multiple studies carried out in Iraq and neighboring countries revealed a high prevalence of severe vitamin D deficiency in women of all ages, which further augments the risk of skeletal disorders in women with comorbid endocrine diseases such as hyperprolactinemia (Mansour MM et al. 2012) (AL-Turki HA et al. 2008). AL-Turki et al. Reports over 80% of Saudi women with severe vitamin D deficiency, and very similar observations have been noted in Iraq (Hussain AN et al. 2014). Based on these observations, the current study was designed to investigate the relationship between hyperprolactinemia, serum vitamin D, and selected electrolytes in a group of osteoporotic women using a cross-sectional design and, in an attempt, to shed lighter on the pathogenesis of prolactin-induced bone loss.

2. Materials and Methods

2.1. Study Design and Population

A cross-sectional study was conducted at [Marjan Teaching Hospital] [Imam AL-Hussain Medical City], Babylon& Kerbala, Iraq, between [December 2025] and [March 2026]. Laboratory analyses were performed at the AL-Ameen Center for Scientific Research, Najaf, Iraq. Ethical approval was obtained from the Institutional Review Board, and written informed consent was obtained from all participants before enrollment. A total of 81 women with osteoporosis were enrolled. Participants were divided into two groups based on serum prolactin levels. Group A (Normal Prolactin Group, $n=30$) serum prolactin ≤ 25 ng/mL; Group B (Hyperprolactinemia Group, $n=51$) , serum prolactin >25 ng/ml.

Osteoporosis was confirmed by dual-energy X-ray absorptiometry (DEXA) with a t-score of -2.5 or lower at the lumbar spine or femoral neck.

2.2. Inclusion and Exclusion Criteria

Inclusion criteria: Female patients aged 50-70 years. confirmed diagnosis of osteoporosis (T-score ≤ -2.5).

Exclusion criteria: pituitary tumors, uncontrolled thyroid or kidney diseases, and other causes.

2.3 Laboratory Measurements

Fasting venous blood samples (5 ml) were collected from all participants in the morning between 8:00 and 10:00 AM to minimize diurnal hormonal variation. Serum was separated by centrifuging at 3000rpm for 10 minutes and stored at -20 °C until analysis. Serum prolactin levels were measured using a commercial enzyme –linked immunosorbent assay (ELISA) kit (E0999Hu, BT-Lab, China). The kit had a standard curve range of 20-4500 μ IU/mL and a sensitivity of 9.57 μ IU/mL. Serum levels of vitamin D (25-hydroxyvitamin D) were measured using commercially available ELISA kits according to the manufacturers ' instructions. Serum electrolytes (calcium, sodium, potassium, chloride) were measured using an automated ion-selective electrode (ISE) analyzer.

2.4 Statistical Analysis

Statistical analysis was performed using GraphPad Prism version 8.0 (GraphPad software, San Diego, USA). Continuous variables are expressed as Mean $\pm$ Standard Error (SE). Comparisons between the two groups were made using the independent samples t-test. A p-value of <0.05 was considered statistically significant. Pearson correlation coefficients were calculated to assess relationships between prolactin and other biochemical parameters.

3. Results

3.1. Demographic Characteristics

The two study groups were demographically comparable. The mean age of the normal prolactin group was 57.70 ± 1.70 years, compared to 56.24 ± 1.65 years in the hyperprolactinemia group. There was no statistically significant difference in age between the two groups ($p = 0.3926$), confirming that related confounding was adequately controlled.

Table1: Comparison of Age Between Study Groups

Parameter	Normal Prolactin Group (n=30)	Hyperprolactinemia Group (n=51)	P-value
	Mean $\pm$ SE	Mean $\pm$ SE	
Age (years)	57.70 ± 1.70	56.24 ± 1.65	0.3926
Values expressed as Mean $\pm$ SE. Independent t-test; NS = Not Significant.			

3.2 Serum Prolactin and Vitamin D Levels

Serum prolactin levels were significantly higher in the hyperprolactinemia group (41.00 ± 0.87 ng/mL) compared to the normal prolactin group (21.46 ± 0.62 ng/mL), confirming adequate group stratification ($p < 0.0001$). Importantly, serum vitamin D levels were significantly lower in hyperprolactinemic women (18.66 ± 0.63 ng/mL vs. 25.97 ± 0.62 ng/mL, $p < 0.0001$), indicating a clinically meaningful vitamin D insufficiency in this group Table2.

Table2: Comparative Analysis of Prolactin and Vitamin D Levels

Parameter	Normal Prolactin Group (n=30) Mean $\pm$ SE	Hyperprolactinemia Group (n=51) Mean $\pm$ SE	P-value
Prolactin (ng/mL)	21.46 $\pm$ 0.62	41.00 $\pm$ 0.87	<0.0001
Vitamin D (ng/mL)	25.97 $\pm$ 0.62	18.66 $\pm$ 0.63	<0.0001

Values expressed as Mean $\pm$ SE. *** p<0.0001 (independent t-test).

3.3 Serum Electrolyte Levels

All four measured electrolytes were significantly reduced in hyperprolactinemic osteoporotic women compared to normoprolactinemic women. Serum calcium (8.30 $\pm$ 0.07 vs. 9.05 $\pm$ 0.05 mg/dL), sodium (131.6 $\pm$ 0.49 vs. 136.8 $\pm$ 0.46 mEq/L), potassium (3.60 $\pm$ 0.05 vs. 4.05 $\pm$ 0.04 mEq/L), and chloride (96.7 $\pm$ 0.35 vs. 101.5 $\pm$ 0.27 mEq/L) were all significantly lower (all p<0.001), as summarized in Table3. Fig.1. A-D.

Table3: Comparison of Serum Electrolyte Levels Between Study Groups

Variable	Normal PRL (n=30) Mean $\pm$ SE	Hyperprolactinemia (n=51) Mean $\pm$ SE	P-value
Calcium (mg/dL)	9.05 $\pm$ 0.05	8.30 $\pm$ 0.07	<0.001
Sodium/Na (mEq/L)	136.8 $\pm$ 0.46	131.6 $\pm$ 0.49	<0.001
Potassium/K (mEq/L)	4.05 $\pm$ 0.04	3.60 $\pm$ 0.05	<0.001
Chloride/Cl (mEq/L)	101.5 $\pm$ 0.27	96.7 $\pm$ 0.35	<0.001

Values expressed as Mean $\pm$ SE. ** p<0.001 (independent t-test).

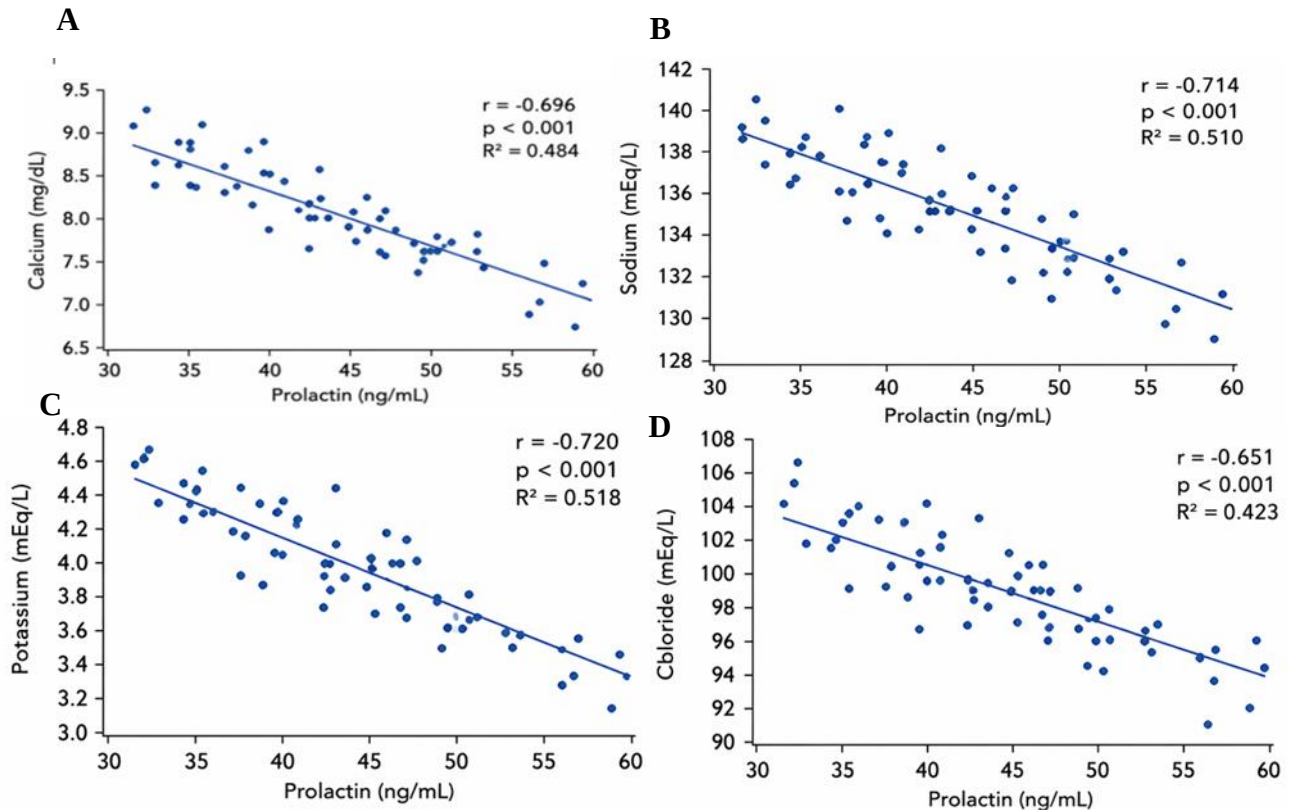


Figure1: Correlation of Serum Prolactin Levels with Serum Electrolytes and Calcium. Scatter Plots Showing Significant Inverse Correlations Between Prolactin Concentration And (A) calcium, (B) sodium, (C) potassium, and (D) chloride. Pearson correlation coefficients (r), coefficients of determination (R^2), and corresponding P values are indicated on each graph.

4. Discussion

These data may reflect a possible relationship between hyperprolactinemia and vitamin D insufficiency. As has been shown, it is established that there were suggestions before that prolactin was able to control the renal 1-hydroxylase (it has not been tested here), which catalyzes the conversion of 25(OH)D to calcitriol, the biologically active form. Reduced calcitriol levels could impair calcium intestinal absorption and renal calcium reabsorption, causing hypocalcaemia and demineralization of the bone. Our study is in accordance with studies published previously by Yavropoulou et al. (2017) and Cortet et al. (2020), who reported lower vitamin D in women with hyperprolactinemia-associated bone disease. More recently, Aboelnaga MM et al (2017) confirmed that prolactin excess is an independent risk factor for lower vitamin D, even after correcting the risk factors, and more recently, Krysiak et al. (2023) also confirmed that prolactin excess is related to lower vitamin D levels and that vitamin D status independently modifies the metabolic impact of hyperprolactinemia in women. The mechanism by which serum calcium levels were reduced in hyperprolactinemic women may be attributed to the reduced levels of vitamin D found in this group, as well as a probable direct effect of prolactin on parathyroid hormone (PTH) release. Lower serum calcium can stimulate PTH secretion, thereby acting to normalize serum calcium but consequently accelerating bone demineralization by the resultant increases in osteoclastic activity. This chronic imbalance in calcium metabolism has been widely recognized as an important contributor to secondary osteoporosis in hyperprolactinemic subjects (Klibanski 1999, Seriwatanachai et al 2008). With respect to the correlation between serum prolactin levels and serum vitamin D levels. In our study, serum prolactin and serum vitamin D showed a significant inverse correlation, consistent with previously reported findings by Diri et al. (2016) and AL-Musawi et al (2021) indicated a negative correlation between prolactin and vitamin D in women with hyperprolactinemia. However, other studies, like that of Naliato et al (2008), did not observe any significant correlation between these hormones in treated patients with prolactinoma, possibly due to normalization by dopamine agonist therapy, which influences the metabolism of prolactin and vitamin D as well. The differences between the previous studies indicate a need to perform further longitudinal studies to elucidate the exact relationship. Hyponatremia and hypochloremia in hyperprolactinemic women could be related with indirectly changes in fluid balance or metabolic alterations in hyperprolactinemia. However, it is difficult to explain it in terms of a direct relation between pituitary deficiency or SIADH. As stated, it is not known exactly how those electrolyte imbalances occur. Hypokalemia could be related to nutritional factors or a secondary increase in aldosteronism. Hyponatremia and hypochloremia could be associated with a shift in fluid balance or pituitary disorders, although this has not been clearly demonstrated. Pituitary tumor subjects were excluded from this study so that any compression mechanism on the pituitary gland is unlikely. These two imbalances have not been found previously to correlate with hyperprolactinemia or any other parameter and need further investigation. The main strength of this study is the exclusion of most confounding factors affecting vitamin D status and metabolic parameters. Simultaneous examination of all these markers has been a strength of the comprehensive metabolic profile. A limitation of the present study is its cross-sectional nature; thus, causal inferences can't be made from its results. Large-scale studies including BMD, clinical manifestations, and the impact of treatment on metabolic and hormonal profiles are needed to establish causality and guide therapy. The results of this study demonstrate a significant and consistent pattern of metabolic changes in hyperprolactinemic osteoporotic women as compared to their normal-prolactinemic

counterparts. This most importantly involves marked changes in serum vitamin D and the four measured electrolytes – serum calcium, sodium, potassium, and chloride. In conclusion, the present study provides further evidence suggesting that hyperprolactinemia not only contributes to osteoporosis through estrogen deficiency but may also lead to systemic metabolic dysregulation, which includes vitamin D insufficiency, secondary hyperparathyroidism, and disturbances of fluid balance with consecutive imbalances in serum calcium, sodium, potassium, and chloride levels, which eventually potentiate osteoclast-mediated bone resorption. Routine laboratory testing for serum vitamin D and the four electrolytes is thus advocated in the management of hyperprolactinemic women with osteoporosis. In conclusion, the data from this study collectively support the concept that hyperprolactinemia in osteoporotic women is not only mediated through estrogen deficiency but also through a broader metabolic disruption encompassing vitamin D insufficiency, secondary hyperparathyroidism, and systemic electrolyte imbalance, all of which converge to accelerate osteoclastic bone resorption. Routine biochemical profiling, including vitamin D and electrolytes, is therefore recommended as part of the clinical evaluation of hyperprolactinemic women with osteoporosis.

Conclusion

Hyperprolactinemia in osteoporotic women is significantly associated with reduced serum vitamin D, hypocalcemia, and generalized electrolyte imbalance (sodium, potassium, and chloride), indicating a multifactorial prolactin-mediated bone disease mechanism.

Ethical considerations

This study was approved by the Ethical Committee at the College of Applied Medical Science/ University of Kerbala. All subjects involved in this work were informed, and agreement was obtained verbally from each one before the collection of samples.

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