مجلة كلية التربية الاساسية
كلية التربية الاساسية – الجامعة المستنصرية
Journal of the College of Basic Education Vol.31 (NO. 132) 2025, pp. 57-68

Comparison Of Osteoprotegrin And Estradiol Levels In Iraqi Women With Osteoporosis And Women With Osteopenia

Mohammed Amer Jihad
Ministry Of Health, Al-Rusafa Health Department, Alawiya Maternity Teaching Hospital
Moh.Jooooj868@Uomustansiriyah.Edu.Iq
07733365229

Amer Hasan Abdullah
Mustansiriyah University, College Of Science, Department Of Chemistry
Amerhasan1965@Uomustansiriyah.Edu.Iq
07711137955

Khitam Jaber Nabhan
Mustansiriyah University, College Of Science, Department Of Chemistry
Khitam.Jaber2@Uomustansiriyah.Edu.Iq
07730014752

Mohammed Amer Jihad — moh.jooooj868@uomustansiriyah.edu.iq

Author Contribution

Mr Mohammed Amer Jihad has carried out the work in laboratories of Chemistry Department and biochemistry; Dr Amer Hassan Abdullah is the supervisor of M.Sc student (1) and he competed the calculation and checked the results; Dr Khitam Jaber Nabhan is the 2nd supervision for the student and she has done the statical data according to analytical branch of clinical field.

ملاحظة : البحث مستل من رسالة ماجستير

Abstract:

Osteoporosis is a common bone disease, especially in women after menopause and with aging, where bones become more fragile due to decreased density and structural changes. This condition is closely linked to a decline in estrogen levels, leading to accelerated bone loss due to an imbalance between bone-forming cells (osteoblasts) and bone resorbing cells (osteoclasts).With aging and the decline in estrogen levels after menopause, caused by the cessation of egg production in the ovaries, the production of inflammatory factors such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor (TNF) increases, while the level of the regulatory protein OPG decreases. This imbalance leads to higher bone turnover and osteoporosis. Additionally, it contributes to vascular calcification due to the increased activity of RANKL on bone and vascular cells.Objectives: Evaluate the differences in Osteoprotegerin (OPG) and Estradiol (E2) levels between Iraqi women with osteoporosis and those with osteopenia to determine the impact of these biomarkers on bone health.Method and patients: Three group in this study of population divided into patient group(n=39) seemingly healthy volunteers as control group ,Additionally, 33 women suffered from osteopenia and 34 women suffered from osteoporosis All participants underwent bone mineral density assessment using dual-energy X-ray absorptiometry.Medical examinations, including laboratory tests for women, were conducted at Al-Alwiya Hospital in Baghdad Province, in addition to other tests performed at Al-Wasiti teaching Hospital. Results: The DEXA

outcomes revealed osteopenia patients had a higher mean age (49.6 ± 14.17 years) than the control group (42.25 ± 13.13 years), while osteoporosis patients were the oldest (57.47 ± 11.28 years, p = 0.015). Osteoprotegerin levels were highest in the control group (6.47 ± 1.69 ng/ml) and significantly lower in osteopenia (5.56 ± 1.91 ng/ml, p = 0.036) and osteoporosis patients (4.85 ± 1.46 ng/ml, p < 0.001). The decline in osteoprotegerin was more pronounced between healthy individuals and osteopenia patients, while the difference between osteopenia and osteoporosis patients was not significant (p = 0.089). Conclusion: Aging is associated with decreased OPG and estradiol levels, leading to bone imbalance and increased loss. The significant decline from osteopenia to osteoporosis highlights the crucial role of hormonal disturbances in disease progression.

Keywords: Osteoprotegerin , Estradiol, DEXA, Osteopenia, Osteoporosis.

Introduction

Osteoporosis is a prevalent and serious bone disease, particularly affecting postmenopausal women. It is characterized by a decrease in bone mineral density and alterations in bone structure, leading to increased bone fragility and a higher risk of fractures. Women over the age of 50 are particularly susceptible to osteoporosis, primarily due to a decline in estrogen levels after menopause [9]. This hormonal deficiency accelerates bone loss by disrupting the balance between osteoclast-mediated bone resorption and osteoblast-mediated bone formation [18]. As a result, the imbalance between these two processes contributes to the progression of osteoporosis and the increased risk of fractures [6].

Osteoprotegerin (OPG) is a soluble glycoprotein, also known as Tumor Necrosis Factor Receptor Superfamily Member 11B (TNFRSF11B) or Osteoclastogenesis Inhibitory Factor (OCIF). It functions as a decoy receptor for the Receptor Activator of Nuclear Factor Kappa-B (RANK), playing a vital role in regulating bone resorption by inhibiting osteoclast differentiation and activity [5,8]. OPG is expressed in various tissues, including bone cells, vascular cells, lungs, and skin. It is considered an anti-resorptive factor because it neutralizes RANKL, a cytokine that strongly suppresses osteoclast activity, thereby inhibiting bone resorption [11].

The OPG/RANKL system is crucial in regulating mineral metabolism in both bone and vascular tissues. While RANKL promotes bone resorption and vascular calcification, OPG inhibits bone resorption and has a protective effect on bone integrity. This regulatory mechanism is essential for maintaining bone health and preventing excessive bone loss [8,13].

Estrogen, a key hormone in women's health, plays a significant role in regulating OPG expression. By increasing OPG production in osteoblasts, estrogen suppresses osteoclastogenesis and enhances bone formation [3,18]. Additionally, estrogen contributes to calcium absorption, further supporting bone health [14]. Given that estrogen receptors are highly expressed in bone cells, estrogen binding to these receptors stimulates the expression of interleukin-1 (IL-1), insulin-like growth factor (IGF-1), and transforming growth factor (TGF-β), while simultaneously suppressing RANKL activity. This cascade of events results in the inhibition of osteoclast formation and bone resorption [8,19].

Experimental Section

Provide sufficient detail to allow the work to be reproduced, including Materials, Instrumentation, and procedures.

Materials

An analytical study was carried out among Iraqi female participants selected through successive sampling from Alawiya Educational and AL-wasiti teaching Hospitals in Baghdad . 106 individuals participated in this research , three group included in this study consisting of 39 apparently healthy patients were considered to represent control group , 33 female suffered from osteopenia and 34 suffered from osteoporosis .Patient demographic ,including age, weight and height was collected from self-reported entries in questionnaire filled out at the time of DEXA machine .

Instrumentation

The patient group had been previously diagnosed with osteoporosis using a DEXA machine to measure bone mineral density. All blood samples were tested for estradiol (E2) and osteoprotegerin (OPG) levels, with OPG levels determined using the ELISA technique.

Procedure

A 5 ml blood sample was collected from women, and venous blood from each individual was stored at -20°C for analysis. All data were statistically analyzed using SPSS version 19.

Results And Discussion

It found that the value of mean age of osteopenia patients (49.6 ± 14.17 years) was higher than that of the control group (42.25 ± 13.13 years).Also found osteoporosis patients had higher mean age (57.47 ± 11.28 years) compared to the control group. Additionally, osteoporosis patients were significantly older than osteopenia patients (p = 0.015).As shown in table (1) and fig (1).

Table (1): Comparison of age a mong control, osteopenia, and osteoporosis groups.

Groups	Parameter	*p*-value		
	Age(year) (Mean SD)	**Control**, N=39	**Osteopenia patients**, N=33	**Osteoporosis patients**, N=34
Control	42.25±13.13		0.024*	< 0.001**
Osteopenia Patients	49.6± 14.17	0.024*		0.015*
Osteoporosis Patients	57.47±11.8	<0.001**	0.015*	
**Significant at P≤ 0.05, **Highly significant P< 0.001, NS: Non-Significant.*				

The control group exhibited the highest level of osteoprotegerin, with a mean of 6.47 ± 1.69 ng/ml, indicating a well-balanced bone remodeling process in healthy individuals. In contrast, the mean osteoprotegerin level in osteopenia patients was 5.56 ± 1.91 ng/ml, which was significantly lower than in the control group ($p = 0.036$). This suggests a disruption in the balance between bone resorption and formation in this group of patients.For osteoporosis patients, osteoprotegerin levels were the lowest, with a mean of 4.85 ± 1.46 ng/ml. When compared to the control group, this difference was highly significant ($p < 0.001$), confirming a marked decline in the body's ability to regulate bone remodeling as the disease progresses. However, when comparing osteoprotegerin levels between osteopenia and osteoporosis patients, the difference was not statistically significant ($p = 0.089$). This indicates that the decline in osteoprotegerin levels is more pronounced between healthy individuals and those with osteopenia, whereas the reduction is less sharp as the disease advances from osteopenia to osteoporosis.shown at table(2) and figure (2).

مجلة كلية التربية الاساسية
كلية التربية الاساسية – الجامعة المستنصرية
Journal of the College of Basic Education Vol.31 (NO. 132) 2025, pp. 57-68

Table (2): "Osteoprotegerin Levels in Different Patient Groups and Statistical Significance Values

Groups	Parameter	*p*-value		
	Ostoprotogren (ng/ml) Mean ± SD)	**Control**, N=39	**Osteopena patients**, N=33	**Osteoporosis patients**, N=34
Control	6.473 ± 1.69		0.036*	< 0.001**
Osteopenia Patients	5.56 ± 1.912	0.036*		0.089
Osteoporosis Patients	4.85 ± 1.46	<0.001**	0.089	

**Significant at P≤ 0.05, **Highly significant P< 0.001, NS: Non-Significant.*

Table(3) and figure (3) demonstrate significant differences in estradiol levels among the three study groups: the control group (healthy individuals), osteopenia patients, and osteoporosis patients.The control group exhibited an average estradiol level of 73.24 ± 68.8 pg/ml, which was considerably higher compared to the patient groups. In contrast, osteopenia patients had a significantly lower mean estradiol level of 11.26 ± 18.01 pg/ml, showing a highly significant difference ($p < 0.001$) compared to the control group.

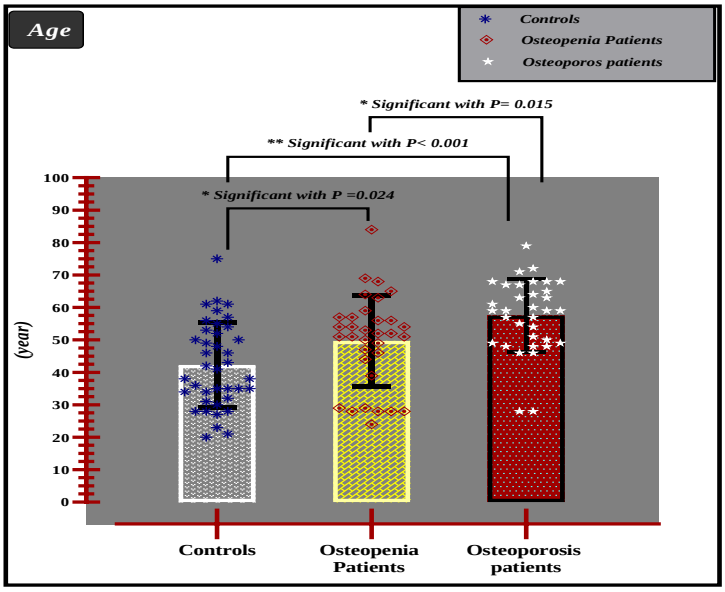
Table (3):The Impact of Declining Estradiol Levels on the Progression of Osteopenia and Osteoporosis

Groups	Parameter	*p*-value		
	Estradiol (pg/ml) (Mean ± SD)	**Control**, N=39	**Osteopenia patients**, N=33	**Osteoporosis patients**, N=34
Control	73.245 ± 68.8		< 0.001**	< 0.001**
OsteopeniaPatients	11.26 ± 18.01	< 0.001**		0.029*
Osteoporosis Patients	4.31 ± 2.20	< 0.001**	0.029*	

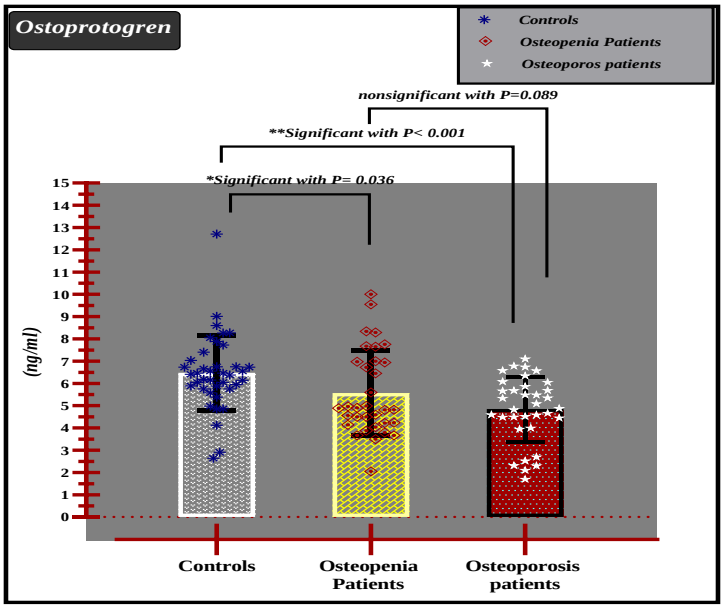
**Significant at P≤ 0.05, **Highly significant P< 0.001, NS: Non-Significant.*

For osteoporosis patients, estradiol levels were even lower, with a mean of 4.31 ± 2.20 pg/ml. When compared to the control group, this difference was highly significant ($p < 0.001$), confirming a sharp decline in estradiol levels among osteoporosis patients.Furthermore, the comparison between osteopenia and osteoporosis patients revealed a statistically significant difference ($p = 0.029$), indicating that estradiol levels continue to decrease as the condition progresses from osteopenia to osteoporosis.

مجلة كلية التربية الاساسية
كلية التربية الاساسية – الجامعة المستنصرية
Journal of the College of Basic Education Vol.31 (NO. 132) 2025, pp. 57-68



Fig(1): Statistical comparison of age across study groups (control, osteoporosis, and osteopenia).



Fig(2): "Comparison of Osteoprotegerin Levels Among Control, Osteopenia, and

مجلة كلية التربية الاساسية
كلية التربية الاساسية – الجامعة المستنصرية
Journal of the College of Basic Education Vol.31 (NO. 132) 2025, pp. 57-68

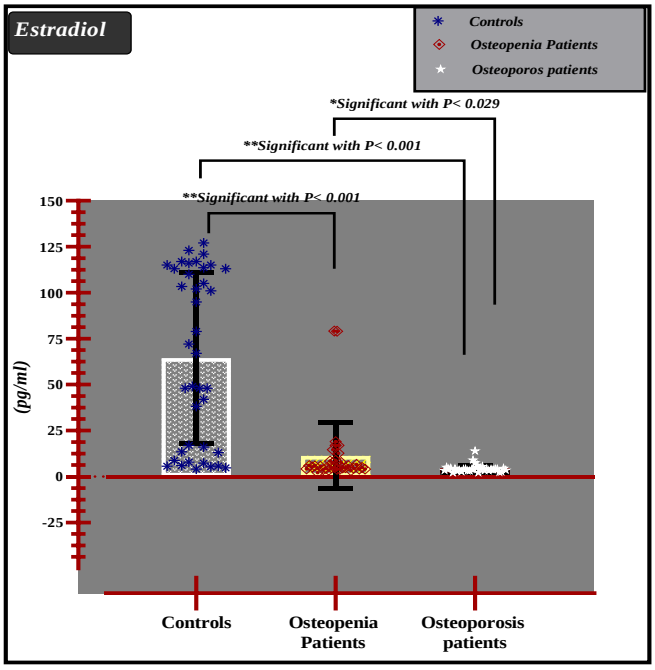


Fig (3): The Role of Estradiol Decline in the Development and Progression of Osteopenia and Osteoporosis.

Discussion

According to table (1) and figure(1) the mean age of osteopenia patients was significantly higher than that of the control group ($p = 0.024$) and osteoporosis patients had a significantly higher mean age compared to the control group, with a highly significant difference ($p < 0.001$),While osteoporosis patients were significantly older than osteopenia patients ($p = 0.015$).These results indicate a progressive increase in age across the groups, with osteoporosis patients being the oldest, followed by osteopenia patients, and then the control group because of decreased bone formation rate with aging, the activity of osteoblasts declines, leading to bone loss occurring at a faster rate than bone formation[6].The findings of this study highlight a progressive decline in osteoprotegerin (OPG) levels as bone health deteriorates from a normal state to osteopenia and eventually to osteoporosis. Osteoprotegerin is a key regulator of bone remodeling, acting as a decoy receptor that inhibits receptor activator of nuclear factor kappa-B ligand (RANKL), thereby preventing excessive bone resorption[5,11]. The observed reduction in OPG levels suggests a disruption in this regulatory mechanism, contributing to increased bone loss[8].The control group exhibited the highest mean OPG levels (6.47 ± 1.69 ng/ml), indicating a well-maintained balance between bone formation and resorption. This supports the role of OPG in preventing excessive osteoclast activation, thereby preserving

bone density and structural integrity [19]. While in osteopenia patients, OPG levels were significantly lower (5.56 ± 1.91 ng/ml) compared to the control group ($p = 0.036$). This suggests the early disruption of bone homeostasis, where the inhibition of osteoclast activity by OPG becomes less effective, leading to gradual bone loss [11]. The significant difference between these two groups implies that changes in OPG levels could serve as an early biomarker for bone deterioration before osteoporosis develops. But Osteoporosis patients had the lowest OPG levels (4.85 ± 1.46 ng/ml), with a highly significant difference compared to the control group ($p < 0.001$). This indicates a severe imbalance favoring bone resorption over bone formation, accelerating bone loss and increasing fracture risk [9,14]. The drastic reduction in OPG suggests that osteoclastic activity is no longer effectively regulated, leading to the structural weakening of bones. However, when comparing OPG levels between osteopenia and osteoporosis patients, the difference was not statistically significant ($p = 0.089$). This may indicate that while OPG levels drop significantly during the early stages of bone loss, other mechanisms, such as hormonal changes (e.g., estrogen deficiency) and inflammatory cytokines, may play a more dominant role in the progression from osteopenia to osteoporosis[8,22]. Based on table (3) reveal a marked decline in estradiol levels among individuals with osteopenia and osteoporosis compared to the healthy control group. Estradiol plays a critical role in preserving bone density by inhibiting bone resorption and promoting bone formation [18]. Consequently, a substantial reduction in its levels may serve as a key contributor to the pathogenesis of bone disorders[17].These results are consistent with previous research indicating that estrogen deficiency, particularly in postmenopausal women, is a major determinant of bone mass loss and an increased risk of fractures[9]. Estrogen regulates osteoclast activity, and its decline leads to an acceleration of bone resorption, ultimately compromising bone integrity [14,18]. Moreover, the significantly lower estradiol levels observed in osteoporosis patients compared to those with osteopenia ($p = 0.029$) suggest a progressive hormonal decline as the disease advances. This supports the hypothesis that estradiol deficiency is not only implicated in the development of osteopenia but also plays a pivotal role in its progression to osteoporosis [3,17].

Conclusion

Aging is associated with decreased OPG and estradiol levels, leading to bone imbalance and increased loss. The significant decline from osteopenia to osteoporosis highlights the crucial role of hormonal disturbances in disease progression.

Patient Consent

Informed consent was obtained for all the patients involved in this study.

Funding

The study was self-funded.

Conflicts Of Interest

The authors declare no conflicts of interest related to this study.

Data Availability

The data supporting the findings of this study is available on reasonable request from the corresponding author. The data is not publicly available due to privacy and ethical considerations.

References

[1] A. J. . Jumaa and W. Y. . Kassim, "The Influence of Age, Gender and Weight on Sex Hormone Levels and Histological Development of Reproductive Organs in Arabi Sheep", Al-Mustansiriyah J. Sci., vol. 33, no. 5, pp. 27–32, Feb. 2023, doi: 10.23851/mjs.v33i5.1309.

[2] Abdullah A, Eltayef E, Khamis W. Comparison of Levels of Vitamin D, Vitamin D Receptor and Vitamin D Binding Protein in Women with Osteoporosis and Osteopenia. Iran J War Public Health 2022; 14 (2) :211-216

[3] Almeida M., Iyer S., Martin-Millan M., Bartell S.M., Han L., Ambrogini E., Onal M., Xiong J., Weinstein R.S., Jilka R.L., et al. Estrogen Receptor-α Signaling in Osteoblast Progenitors Stimulates Cortical Bone Accrual. J. Clin. Investig. 2013;123:394–404. doi: 10.1172/JCI65910.

[4] AL-TEMIMI RB, ABDULLA AH, TAHIR NT, Serum soluble alpha klotho and periostin as a biomarker for detection of early diabetic nephropathy in Iraqi male with Type 2 Diabetes. J.Res.Pharm.2024;28(5):16651673

[5] **Armada L, Marotta Pdos S, Pires FR, Siqueira JF Jr**. Expression and distribution of receptor activator of nuclear factor kappa B, receptor activator of nuclear factor kappa B ligand, and osteoprotegerin in periradicular cysts. J Endod. 2015;41:1281–1287.

[6] Boskey, A. L., & Coleman, R. Aging and bone. Journal of dentalresearch. 2010;89:12–13331348.

[7] Chaki O. [Osteoporosis]. Nihon Rinsho. 2006 Apr;64 Suppl 4:370-4. Japanese. PMID: 16689336.

[8]Collin-Osdoby P. Regulation of vascular calcification by osteoclast regulatory factors RANKL and osteoprotegerin. Circ Res. 2004 Nov 26;95(11):1046-57. doi: 10.1161/01.RES.0000149165.99974.12. PMID: 15564564.

[9]Eastell R., O'Neill T.W., Hofbauer L.C., Langdahl B., Reid I.R., Gold D.T., Cummings S.R. Postmenopausal Osteoporosis. Nat. Rev. Dis. Primers. 2016;2:16069. doi: 10.1038/nrdp.2016.69.

[10] F. F. Hasan and H. L. Mohammed, "Comparison between Benign and Malignant Primary Bone Tumors-A Histopathological Study of 119 Cases", Al-Mustansiriyah J. Sci., vol. 29, no. 2, pp. 74–82, Nov. 2018, doi: 10.23851/mjs.v29i2.182.

[11] Ferrer Cañabate J, Tovar I, Martínez P. Osteoprotegerina y Sistema RANKL/RANK: el futuro del metabolismo óseo? [Osteoprotegrin and RANKL/RANK system: is it the future of bone metabolism?]. An Med Interna. 2002 Aug;19(8):385-88. Spanish. PMID: 12244784.
Immunity, Bone, and Beyond. Front. Immunol. 2014;5:511.

[12] J ubair, S., Nsaif, A. S., Abdullah, A. H. and Dhefer, I. H. (2021) 'Vitamin D deficiency is associated with thyroid diseases', Journal of Physics: Conference Series,2021; 1853(1), p. 012036.

[13]Kuroda Y., Matsuo K. Molecular mechanisms of triggering, amplifying and targeting RANK signaling in osteoclasts. World J. Orthop. 2012;3:167–174. doi: 10.5312/wjo.v3.i11.167.

[14] **Liu S., Li J., Sheng Z., Wu X., Liao E.** Relationship between Body Composition and Age, Menopause and Its Effects on Bone Mineral Density at Segmental Regions in Central Southern Chinese Postmenopausal Elderly Women with and without Osteoporosis. Arch. Gerontol. Geriatr. 2011;53:e192–e197. doi: 10.1016/j.archger.2010.09.002.

[15] Migliorini F, Giorgino R, Hildebrand F, Spiezia F, Peretti GM, Alessandri-Bonetti M, Eschweiler J, Maffulli N. Fragility fractures: risk factors and management in the elderly. Medicina. 2021;57(10):1119.

[16] Prada G, Fita I, Nacu R, Ignat I, Petrescu R, Jugravu O. Risk factors for complications of osteoporosis in older people. Eur Geriatr Med. 2013;4:S25

[17] Reinhardt RA, Payne JB, Maze CA, Patil KD, Gallagher SJ, Mattson JS. Influence of estrogen and osteopenia/osteoporosis on clinical periodontitis in postmenopausal women. J Periodontol. 1999;70(8):823-828. doi:10.1902/jop.1999.70.8.823

[18]Riggs B.L. The Mechanisms of Estrogen Regulation of BoneResorption. J. Clin. Investig. 2000;106:1203–1204.doi: 10.1172/JCI11468

[19] Rochette L., Meloux A., Rigal E., Zeller M., Cottin Y., Vergely C. The role of osteoprotegerin in the cross talk between vessels and bone: Its potential utility as a marker of cardiometabolicdiseases.Pharm.her.2018;182:115–132.doi: 10.1016/j.pharmthera.2017.08.015.

[20] S. M. . Oudah, I. H. . Hamzah, B. F. Matti, and R. Alkasir, "The Role of Immune Check Point Gene Expression of Pd-1 and Tim-3 in Patients with Acute Myeloid Leukemia", Al-Mustansiriyah J. Sci., vol. 35, no. 1, pp. 27–32, Mar. 2024, doi: 10.23851/mjs.v35i1.1418.

[21] Wasilewska A, Rybi-Szuminska AA, Zoch-Zwierz W. Serum osteoprotegrin (OPG) and receptor activator of nuclear factor kappaB (RANKL) in healthy children and adolescents. J Pediatr EndocrinolMetab.2009;22(12):1099-1104.

[22] Yeung RS. The osteoprotegerin/osteoprotegerin ligand family: role in inflammation and bone loss. J Rheumatol. 2004 May;31(5):844-6. PMID: 15124240.

مقارنة مستويات أوستيوبروتيغرين وإستراديول لدى النساء العراقيات المصابات بهشاشة العظام والنساء المصابات بنقص كثافة العظام

الكلمات المفتاحية: نقص أوستيوبيروجيرين، إستراديول، قياس كثافة العظام(*DEXA*)، الكتلة العظمية، هشاشة العظام

ختام جابر نبهان
الجامعة المستنصرية،
كلية العلوم، قسم الكيمياء
khitam.jaber2@uomustansiriyah.edu.iq
07730014752

عامر حسن عبدالله
الجامعة المستنصرية،
كلية العلوم، قسم الكيمياء
amerhasan1965@uomustansiriyah.edu.iq
07711137955

محمد عامر جهاد
وزارة الصحة، دائرة صحة الرصافة،
مستشفى العلوية التعليمي للولادة
moh.jooooj868@uomustansiriyah.edu.iq
07733365229

المؤلف المسؤول عن المراسلات:
محمد عامر جهاد moh.jooooj868@uomustansiriyah.edu.iq

مستخلص البحث:

تُعد هشاشة العظام من الأمراض الشائعة التي تصيب العظام، خاصة لدى النساء بعد انقطاع الطمث ومع التقدم في العمر، حيث تصبح العظام أكثر هشاشة نتيجة لانخفاض الكثافة والتغيرات البنيوية. وترتبط هذه الحالة ارتباطًا وثيقًا بانخفاض مستويات هرمون الإستروجين، مما يؤدي إلى تسارع فقدان العظام نتيجة لخلل في التوازن بين الخلايا البانية للعظم (Osteoblasts)والخلايا الهادمة للعظم (Osteoclasts). ومع التقدم في السن وانخفاض مستويات الإستروجين بعد انقطاع الطمث، الناتج عن توقف إنتاج البويضات في المبايض، يزداد إنتاج العوامل الالتهابية مثل إنترلوكين-1 (IL-1) وإنترلوكين-6 (IL-6)وعامل نخر الورم(TNF) ، بينما ينخفض مستوى بروتين التنظيم OPG. يؤدي هذا الخلل إلى زيادة معدل تبدل العظام وحدوث هشاشة العظام، كما يُسهم أيضًا في تكلس الأوعية الدموية نتيجة زيادة نشاط RANKL على خلايا العظم والأوعية الدموية.

مجلة كلية التربية الاساسية
كلية التربية الاساسية – الجامعة المستنصرية
Journal of the College of Basic Education Vol.31 (NO. 132) 2025, pp. 57-68

الأهداف:
تقييم الفروقات في مستويات أوستيوبروتوجيرين (OPG) وإستراديول (E2) بين النساء العراقيات المصابات بهشاشة العظام والمصابات بنقص الكتلة العظمية (Osteopenia) لتحديد تأثير هذه العلامات الحيوية على صحة العظام.
الطريقة والمرضى:
تضمنت الدراسة ثلاث مجموعات من السكان، شملت مجموعة المرضى(n=39) ، بالإضافة إلى متطوعات سليمات ظاهريًا كمجموعة ضابطة. كما شملت 33 امرأة مصابة بنقص الكتلة العظمية و34 امرأة مصابة بهشاشة العظام. خضع جميع المشاركين لتقييم كثافة المعادن في العظام باستخدام تقنية الأشعة السينية ثنائية الطاقة (DEXA).
أُجريت الفحوصات الطبية، بما في ذلك التحاليل المختبرية للنساء، في مستشفى العلوية بمحافظة بغداد، بالإضافة إلى اختبارات أخرى أُجريت في مستشفى الوسيط التعليمي.
النتائج:
أظهرت نتائج فحص DEXA أن متوسط العمر لدى مرضى نقص الكتلة العظمية كان أعلى (49.6 ± 14.17سنة) مقارنةً بالمجموعة الضابطة (42.25 ± 13.13 سنة)، في حين كان مرضى هشاشة العظام الأكبر سنًا (57.47 ± 11.28 سنة، p = 0.015). كانت مستويات أوستيوبروتوجيرين الأعلى في المجموعة الضابطة (6.47 ± 1.69 نانوغرام/مل) وأقل بشكل ملحوظ لدى مرضى نقص الكتلة العظمية (5.56 ± 1.91نانوغرام/مل، p = 0.036) ومرضى هشاشة العظام (4.85 ± 1.46 نانوغرام/مل، p < 0.001). كان الانخفاض في أوستيوبروتوجيرين أكثر وضوحًا بين الأفراد الأصحاء ومرضى نقص الكتلة العظمية، بينما لم يكن الفرق بين مرضى نقص الكتلة العظمية وهشاشة العظام ذا دلالة إحصائية (p = 0.089).
الاستنتاج:
يرتبط التقدم في السن بانخفاض مستويات OPG والإستراديول، مما يؤدي إلى اختلال التوازن العظمي وزيادة الفقد العظمي. يشير الانخفاض الملحوظ من نقص الكتلة العظمية إلى هشاشة العظام إلى الدور الحاسم للاضطرابات الهرمونية في تطور المرض.