



Physiological Evaluation of Osteopontin and Bone Alkaline Phosphatase as Metabolic Markers in Postmenopausal Women with Osteoporosis

Sabreen Malik Nayef and Faris S. Kata

Department of Biology / College of Education For Pure Sciences/ University of Basrah -Iraq

Correspondence author: faris.kataa@uobasrah.edu.iq

<https://orcid.org/0000-0003-3574-2605>

Abstract

Osteoporosis is a pathological condition that results from a disruption in the dynamic balance of bone remodeling, involving complex interactions between resorption and formation markers at the cellular level. These interactions are influenced by various molecular and hormonal factors, ultimately leading to an increased rate of bone resorption that surpasses bone formation. The assessment of bone turnover markers is considered a critical tool in the management and monitoring of patients with osteoporosis. Among the established biochemical indicators, bone-specific alkaline phosphatase (B-ALP) and osteopontin (OPN) serve as reference markers for bone formation and resorption. The present study was conducted on postmenopausal women diagnosed with osteoporosis in Basrah Province, Iraq. This study set out to examine the correlation between osteoporosis and blood concentrations of two important biomarkers, bone alkaline phosphatase (B-ALP) and osteopontin (OPN), in women who had just gone through menopause. After menopause, 80 blood samples were taken from women: 55 from those who had osteoporosis and 25 from those who were considered healthy. Patient samples were also distributed into two categories depending on age and disease severity. The results of the statistical analysis showed that compared to healthy persons, osteoporotic patients had significantly higher levels of osteopontin ($P < 0.001$). In addition, the T-test revealed that the serum B-ALP concentration was significantly lower in the osteoporotic group than in the control group. The results of this research indicate that changes in the blood levels of B-ALP and OPN are important for postmenopausal women to be able to recognise and track the course of osteoporosis early on. These findings support the potential use of B-ALP and OPN as early biochemical indicators for monitoring postmenopausal osteoporosis. **Keywords:** Osteoporosis, osteopontin, bone alkaline phosphatase, bone biomarkers.

Introduction

Osteoporosis is a chronic systemic bone disease characterized by decreased bone mass and mineral density. This causes deterioration in the microarchitecture of bone tissue. It is most common in older people, especially in postmenopausal women. It affects a large number of women worldwide after menopause [1]. Bone loss accelerates in postmenopausal women due to decreased estrogen secretion. This, in turn, stimulates the death of osteoblasts and increases the activity of osteoclasts, leading to the development of the disease [2]. Osteoporosis is a complex, silently developing disease. It is caused by the interaction of genetic, environmental, and physiological factors [3]. Osteoporosis is one of the diseases that significantly impacts an individual's health. Sources indicate that it is a risk factor for cardiovascular disease [4].

The dynamic nature of bone results in the continuous release of biomarkers, which are released from bone turnover into the bloodstream. These biochemical or cellular compounds are produced during bone resorption or formation [5]. These biomarkers provide sensitive and accurate

information for understanding bone metabolic dynamics. They are essential for early diagnosis and prognosis of disease, as well as for monitoring response to treatment. They differ from and often complement bone mineral density measurements [6;7; 8].

Bone alkaline phosphatase (B-LAP) is a glycoprotein found in bone tissue. It is outside the osteoblast cell membrane [9;10]. ALPL produces human nonspecific tissue alkaline phosphatase. The enzyme requires two Zn^{+2} ions, one Mg^{2+} ion, and one Ca^{2+} ion [11]. Osteoblasts make b-ALP. It promotes mineralization, which is essential to bone development. It binds phosphate and calcium to form hydroxyapatite crystals, which pile up between collagen fibers to mineralize bone [12]. This enzyme breaks down the bone mineral surface pyrophosphate. It reduces calcium and phosphate deposition on this surface, preventing mineral formation [13]. Keeping an eye on serum ALP levels may help find bone metabolic problems like osteoporosis early on [9].

Osteopontin is a phosphoprotein that is quite acidic. The SPP1 gene makes the protein. Thrombin cuts a part of OPN, which makes it active. [14] It has 314 amino acids in it

[15]. There are around 2% of non-collagenous proteins that are OPN [16]. Both osteoblasts and osteoclasts release OPN, and it builds up on the surfaces of bones [17]. This protein binds to and holds osteoclasts to the bone surface, which helps them do their job. When this protein binds to the integrin receptors $\alpha\beta3$ integrin and CD44 on the surface of osteoclasts, it sends a cascade of signals within the cell that make the conditions right for bone resorption to begin [18]. Osteopontin has also been proven to bind calcium ions, which stops hydroxyapatite crystals from growing. This stops the process of minerals forming in bones [19;20]. High serum OPN levels imply that bone mineral density is poor. It represents an important risk factor that is positively associated with the severity of postmenopausal osteoporosis [21;22]. The current study aimed to evaluate the physiological levels of two biomarkers associated with bone metabolism and their metabolic relationship with osteoporosis.

Materials and Methods

The study was conducted from November 2024 to February 2025. Eighty serum samples were collected from women, divided into two groups: healthy women and women with osteoporosis, at Al-Zahraa

Specialised Centre for Bone Densitometry/Ibn Al-Bitar Private Hospital/Basrah Governorate. Their bone density was determined using a Lunar Prodigy (version 16) DEXA (USA) scanner. A specialist physician diagnosed the women with osteoporosis. A questionnaire was developed to collect data related to the study sample, and diseases that may cause changes in bone metabolism were excluded.

Patient Group

This group included 55 women with osteoporosis but no other diseases. Their ages ranged from 50 to 72 years. The body mass index (BMI) of osteoporosis patients was 28.2 kg/m^2 . The patient group was divided into two age groups. The first group included 24 women aged 50-60 years. The second group included 31 women aged 61-72 years. The female patient samples were also classified into two groups based on disease severity. The first group included 34 serum samples from women who had mild osteoporosis. The T-score was between (-

2.5) and (-2.8). The second group included 21 blood samples from women with severe osteoporosis. The T-score was between (-2.9) and (-5.7).

Healthy Women Group

This group included 25 healthy women without osteoporosis or other diseases. Ages ranged from 51 to 63. The healthy group's BMI was 27.3 kg/m².

Serum Sample Preparation

Between 8:00 and 9:00 a.m., 5 ml of blood was drawn from the ulnar vein. Blood was then placed in gel tubes and left at room temperature for 30 minutes. Serum was extracted by centrifuging the samples at 3,500 rpm for 10 minutes. A micropipette removed serum, which was split into 1.5 ml Eppendorf tubes. After being stored in a deep freezer at -80°C, they were thawed before testing.

Estimating the concentration of biomarkers in the serum of women with osteoporosis and healthy women

For this immunological approach, a German-made ELISA reader was employed. The test kits, supplied by the Chinese company Elabscience, were used to determine biomarker concentrations in the

blood serum of healthy and postmenopausal women. Both osteopontin and alkaline phosphatase were indicators. The technique was based on the data sheet provided with each kit. To test each material's optical density (OD), the spectrophotometer was adjusted to 450 nm.

Statistical Analysis

The results were analyzed using SPSS version 21. To analyze the results, a t-test was conducted with a significance level of ($P \leq 0.01$). SD was used to assess data.

Results

The study found that the experimental group had significantly lower B-ALP levels than the control group ($P < 0.001$). Osteoporosis patients had serum values of 3169.84 ng/mL, compared to 4035.75 ng/mL in healthy women. There were no discernible variations in B-ALP levels among age groups or disease severity categories, as shown in Table 1.

Furthermore, the statistical analysis demonstrated a statistically significant difference at the probability level ($P < 0.00$) in the levels of osteopontin protein (OPN) in the blood serum of women with osteoporosis (6.39), compared to healthy women (4.10). Statistical examination of serum

osteoporosis patients did not reveal any significant differences in disease severity between the two age groups (Table 2).

Table 1. Estimated serum alkaline phosphatase (ALP B) concentration (pg/ml)

Variables	Number of samples	Mean ± Standard Deviation	P. Value
Healthy group	25	4035.75 ^a 379.84 ±	0.000
Osteoporosis group	55	3169.84 ^b 635.97 ±	
First age group 50-60 years	24	3396.89 ^a ±554.38	0.379
Age Group 2 61-72 years	31	3252.12 ^a ± 655.48	
Moderate intensity 2.5-to -2.8	34	3400.94 ^a ± 676.56	0.192
High intensity 2.9-to -5.7	21	3176.63 ^a ± 566.95	

Different letters indicate a significant difference at the probability level of $P \leq 0.05$.

Table 2. Estimated serum osteopontin (OPEN) protein concentration (ng/ml)

Variables	Number of samples	Mean ± Standard Deviation	P. Value
Healthy group	25	4.10 ^b 1.15 ±	0.000
Osteoporosis group	55	6.39 ^a ±1.87	
First age group 50-60 years	24	5.61 ^a 1.46 ±	0.571
Age Group 2 61-72 years	31	5.83 ^a ±1.40	
Moderate intensity 2.5-to -2.8	34	5.48 ^a 1.18±	0.102
High intensity 2.9-to -5.7	21	6.14 ^a 1.53±	

Different letters indicate a significant difference at the probability level of $P \leq 0.05$.

Discussion

The study found that osteoporosis patients had considerably lower serum alkaline phosphatase levels than healthy controls [23]. Observed that osteoporosis patients had lower b-ALP levels, supporting our results. Low vitamin D and calcium insufficiency limit enzyme activity, which lowers bone mineral density and induces osteoporosis and bone mass loss [24].

Hypocalcemia and low ALP may occur together. Calcium is essential to bone

microstructure. This ion has enzyme properties and is the main molecule in hydroxyapatite crystal formation. Chemically, enzymes have numerous Ca^{2+} , Mg^{2+} , and Zn^{2+} binding sites. Minerals regulate enzyme activity, structure, and function [25]. Reduced calcium and mineral availability reduce osteoblast activity. Poor enzyme production and reduced levels ensue. According to [26;27]. This increases bone resorption, which decreases mineralisation and bone mass and density, producing osteoporosis.

Zinc (Zn²⁺) reduces bone density and promotes bone health. Ions that cofactor the B-ALP enzyme include it. Low zinc levels may cause serum alkaline phosphatase levels to decline, according to [28].

Alkaline phosphatase levels may have decreased due to the accumulation of inorganic pyrophosphate (PPi) molecules, which are not being broken down, resulting in a higher concentration. PPi suppresses mineralisation and hydroxyapatite crystal formation, which are necessary for bone growth, by competing with Pi on calcium molecules. Their deposition between collagen fibers is prevented [29;30]. The main purpose of the β -ALP enzyme is to break down pyrophosphate molecules. Increased PPi levels in bone tissue surpass the enzyme β -ALP's decomposition capacity. This activates osteoclasts and inhibits osteoblasts. Reduced β -ALP production leads to osteoporosis [31;32].

As for the effect of age on alkaline phosphatase concentration, the study results showed no significant differences in enzyme concentration in the postmenopausal stage between the two age groups (the first group was 50-60 years old and the second group was 61-72 years old). This result may be explained by a decrease in sex hormone

levels in both groups, as these changes occur early after menopause. It may also be due to the similarity of lifestyles, which may be similar between individuals in both groups in terms of environmental and nutritional factors.

Regarding the effect of disease severity on serum alkaline phosphatase levels in women with osteoporosis, no significant differences were observed between moderate and high disease severity. The results indicate that changes in disease severity stages do not lead to a clear change in b-ALP enzyme activity. This indicator may be insensitive to the degree of disease severity. It cannot be used to assess the progression of the disease. The current results are consistent with the results of the study by [33].

The study results also revealed a significant increase in osteopontin protein concentrations in osteoporosis patients compared to healthy controls. This research matches. [34] Women with osteoporosis showed reduced bone mineral density and increased blood osteopontin [35;36].

OPN stimulates bone resorption in postmenopausal women; therefore, bone abnormalities and diseases may cause elevated hormone levels. Another function of this protein is to inhibit mineralisation

and hydroxyapatite crystal formation [37:22:38].

Healthy women with normal blood osteopontin protein levels may have lower levels than people living with osteoporosis because they overexpress the protein. This is due to its positive connection with the disease [39]. Found that women who create too much OPN are more likely to degenerate due to menopause-induced bone mineral density loss.

Inflammation-induced hyaluronic acid (HA) increases may produce osteopontin mRNA overexpression in OA joints. The presence of these inflammatory stimuli and the accumulation of HA molecules thus induce osteopontin overexpression in some women. This may explain the discrepancy in OPN levels between osteoporotic patients and healthy postmenopausal controls [40:41].

Elevated serum OPN levels may also be due to the interaction of gut microbes with the immune system following estrogen decline. Gut microbes regulate the inflammatory responses resulting from decreased sex hormones. This exacerbates bone loss. Sex hormone deficiency combined with microbial influences increases intestinal permeability, expands Th17 cells, and increases the production of the cytokines

TNF α , RANKL, and IL-17 in the gut and bone marrow. This stimulates osteoclast activity. These, in turn, increase the expression of osteopontin and cause osteoporosis. However, in the absence of gut microbes, decreased hormones do not lead to a similar response, meaning that bones are protected from mineral loss resulting from sex hormone deficiency. This indicates the effective role of microbes in stimulating inflammatory pathways, which are a key factor in activating bone loss [42:43:44].

Psychological stress, chronic tension, and depression in patients are also likely to affect multiple body systems, including the skeletal system and the sympathetic nervous system. These conditions result in increased nervous system stimulation, which in turn activates the OPN protein synthesis mechanism, thereby affecting the skeletal system. The relationship between these diseases and bone health can be explained through two physiological and behavioral systems. Physiological changes include endocrine changes, such as increased levels of thyroid hormone and prolactin, and increased levels of cortisol, which inhibit bone formation. Behavioral changes, such as altered lifestyle, physical inactivity, and poor nutrition, also affect skeletal health. Therefore, depression may be a cause of

osteoporosis and the stimulation of catabolic markers [45:46:47:48].

There were no statistically significant changes in protein concentration between the two patient age groups when it came to the impact of age on osteopontin levels. The fact that there is no discernible correlation between age and OPN protein content may account for this finding. Consistent with research [49]. Blood protein levels in osteoporosis patients rise over the age spectrum.

Regarding the effect of disease severity on osteopontin concentrations in patients, the current results revealed no statistically significant differences in OPN protein levels between women with moderate and severe osteoporosis. Osteopontin-producing cells may be sensitive to changes in disease severity, producing equal concentrations of OPN, which is why no significant difference was observed between the two severity levels.

Conclusion

The elevated serum concentration of the biomarker osteopontin and the markedly decreased concentration of the bone-regenerating enzyme alkaline phosphatase may indicate the causes of disturbances in

bone resorption and bone formation, resulting in osteoporosis in women. These biomarkers may serve as reliable biochemical tools for early diagnosis and effective monitoring of osteoporosis progression in postmenopausal women.

Conflict of interest statement

There are no disclosed conflicts of interest.

References

1. Zhang X, Wang Z, Zhang D, Ye D, Zhou Y, Qin J, Zhang Y. The prevalence and treatment rate trends of osteoporosis in postmenopausal women. *Plos one*. 2023; 18(9): 0290289.
2. Clarke B L, Khosla S. Physiology of bone loss. *Radiologic Clinics of North America*. 2010; 48(3): 483-495.
3. Choksi P, Jepsen K J, Clines G A. The challenges of diagnosing osteoporosis and the limitations of currently available tools. *Clinical diabetes and endocrinology*. 2018; 4: 1-13.
4. Azeez T A. Osteoporosis and cardiovascular disease: a review. *Molecular biology reports*. 2023; 50(2): 1753-1763.
5. Bauer D C, Clinical use of bone turnover markers. *Jama*. 2019; 322(6): 569-570.

6. Lee J, Vasikaran S. Current recommendations for laboratory testing and use of bone turnover markers in management of osteoporosis. *Annals of laboratory medicine*. 2012; 32(2): 105-112.
7. Parveen B, Parveen A, Vohora D. Biomarkers of osteoporosis: an update. *Endocrine, Metabolic & Immune Disorders-Drug Targets (Formerly Current Drug Targets-Immune, Endocrine and Metabolic Disorders)*. 2019; 19(7): 895-912.
8. Adlan MM, Kata FS, Alsulaiti SW. Estimation of concentration of some biomarkers in serum of women with rheumatoid arthritis. *Rheumatology*. 2022; XXX(3):59-66.
9. Cheng X, Zhao C. The correlation between serum levels of alkaline phosphatase and bone mineral density in adults aged 20 to 59 years. *Medicine*. 2023; 102(32):1-7.
10. YuY, Rong K, Yao D, Zhang Q, Cao X, Rao B, Qin A. The structural pathology for hypophosphatasia caused by malfunctional tissue non-specific alkaline phosphatase. *Nature Communications*. 2023; 14(1): 4048.
11. Mornet E, Stura E, Lia-Baldini A S, Stigbrand T, Ménez A, Le Du M H. Structural evidence for a functional role of human tissue nonspecific alkaline phosphatase in bone mineralization. *Journal of biological chemistry*. 2001; 276(33): 31171-31178.
12. Vimalraj S, Alkaline phosphatase: Structure, expression and its function in bone mineralization. *Gene*. 2020; 754: 144855.
13. Imam I, Rautureau G J P, Violot S, Mulard E D, Magne D, Ballut L. Structural and Functional Integration of Tissue-Nonspecific Alkaline Phosphatase Within the Alkaline Phosphatase Superfamily: Evolutionary Insights and Functional Implications. *Metabolites*. 2024; 14(12): 659.
14. Yamaga M, Tsuji K, Miyatake K, Yamada J, Abula K, Ju Y J, Muneta T. Osteopontin level in synovial fluid is associated with the severity of joint pain and cartilage degradation after anterior cruciate ligament rupture. *PLoS One*. 2012; 7(11): 49014.
15. Peraramelli S, Zhou Q, Zhou Q, Wanko B, Zhao L, Nishimura T, Leung LL. Thrombin cleavage of osteopontin

initiates osteopontin's tumor-promoting activity. *Journal of Thrombosis and Haemostasis*. 2022; 20(5): 1256-1270.

16. Si J, Wang C, Zhang D, Wang B, Hou W, Zhou Y. Osteopontin in bone metabolism and bone diseases. *Medical science monitor: international medical journal of experimental and clinical research*. 2020; 26:1-9.

17. Bastos A C S D F, Gomes A V P, Silva G R, Emerenciano M, Ferreira L B, Gimba E R P. The intracellular and secreted sides of osteopontin and their putative physiopathological roles. *International Journal of Molecular Sciences*. 2023; 24(3): 2942.

18. Kumari A, Kashyap D, Garg V K. Osteopontin in cancer. *Advances in. Clinical Chemistry*. 2024; 118: 87-110.

19. Chang I C, Chiang T I, Yeh K T, Lee H, Cheng Y W. Increased serum osteopontin is a risk factor for osteoporosis in menopausal women. *Osteoporosis international*. 2010; 21: 1401-1409.

20. Abdulsamad RK, Sawad AA, Kata FS. The Effect of Warfarin on Gene Expression of Biomarker Osteopontin in Fetuses of Rats. *Egyptian Journal of Veterinary Sciences*. 2023; 54(4) : 609-620.

21. Chiang T I, Chang I C, Lee H S, Lee H, Huang C H , Cheng Y W. Osteopontin regulates anabolic effect in human menopausal osteoporosis with intermittent parathyroid hormone treatment. *Osteoporosis international*. 2011; 22: 577-585.

22. Vancea A, Serban O, Fodor D. Relationship between osteopontin and bone mineral density. *Acta Endocrinologica (Bucharest)*. 2021; 17(4): 509-516.

23. Ng E, Ashkar C, Seeman E, Schneider H G, Nguyen H, Ebeling P R, Sztal-Mazer S. A low serum alkaline phosphatase may signal hypophosphatasia in osteoporosis clinic patients. *Osteoporosis International*. 2023; 34(2): 327-337.

24. Tang G, Feng L, Pei Y, Gu Z, Chen T, Feng Z. Low BMI, blood calcium and vitamin D, kyphosis time, and outdoor activity time are independent risk factors for osteoporosis in postmenopausal women. *Frontiers in Endocrinology*. 2023; 14: 1154927.

25. Hoylaerts MF, Van Kerckhoven S, Kiffer-Moreira T, Sheen C, Narisawa S, Millán JL. Functional significance of calcium binding to tissue-nonspecific alkaline phosphatase. *PloS one*. 2015;10(3):e0119874.
26. Bhattarai T, Bhattacharya K, Chaudhuri P, Sengupta P. Correlation of common biochemical markers for bone turnover, serum calcium, and alkaline phosphatase in post-menopausal women. *The Malaysian journal of medical sciences*. 2014;21(1):58.
27. Winter W E, Pelletier J P R, Harris N S. Calcium biology and disorders. In *Contemporary Practice in Clinical Chemistry*. 2020; pp. 795-824.
28. Ceylan M N, Akdas S, Yazihan N. Is zinc an important trace element on bone-related diseases and complications? A meta-analysis and systematic review from serum level, dietary intake, and supplementation aspects. *Biological trace element research*. 2021; 199(2): 535-549.
29. Orriss, I. R.; Arnett, T. R. and Russell, R. G. G. (2016). Pyrophosphate: a key inhibitor of mineralisation. *Current opinion in pharmacology*. 28: 57-68.
30. Ibsen C J S, Birkedal H. Pyrophosphate-inhibition of apatite formation studied by in situ X-ray diffraction. *Minerals*. 2018; 8(2): 65.
31. Orimo H. The mechanism of mineralization and the role of alkaline phosphatase in health and disease. *Journal of Nippon Medical School*. 2010; 77(1): 4-12.
32. Guibert C, Landoulsi J. Enzymatic approach in calcium phosphate biomineralization: a contribution to reconcile the physicochemical with the physiological view. *International Journal of Molecular Sciences*. 2021; 22(23): 12957.
33. Lumachi F, Ermani M, Camozzi V, Tombolan V, Luisetto G. Changes of bone formation markers osteocalcin and bone-specific alkaline phosphatase in postmenopausal women with osteoporosis. *Annals of the New York Academy of Sciences*. 2009; 1173(1): 60-63.
34. Reza S, Shaukat A, Mahmud M, Arain T M, Reza U, Waheed F. Evaluation of Osteopontin in Combination with Bone Turnover Markers for the Assessment of Osteoporosis in Postmenopausal Women. *Br J Med Med Res*. 2016; 14(6): 1-11.
35. Wei Q S, Huang L, Tan X, Chen Z Q, Chen S M, Deng W M. Serum osteopontin

levels in relation to bone mineral density and bone turnover markers in postmenopausal women. *Scandinavian Journal of Clinical and Laboratory Investigation*. 2016; 76(1): 33-39.

36. Lin J C, Liu Z G, Liu R R, Xie L W, Xie H L, Cai H G. The increase of osteopontin and β -carboxy-terminal cross-linking telopeptide of type I collagen enhances the risk of hip fracture in the elderly. *Journal of Clinical Laboratory Analysis*. 2020; 34(5): 23204.

37. Bilha SC, Branisteanu D, Buzduga C, Constantinescu D, Cianga P, Anisie E, Ungureanu M C, Modifications in the spectrum of bone mass predictive factors with menopausal status. *Endocrine Research*. 2018; 43(3): 176-185.

38. Holzinger J, Kotisch H, Richter K W, Konrat R. Binding mode characterization of osteopontin on hydroxyapatite by solution NMR spectroscopy. *ChemBioChem*. 2021; 22(13): 2300-2305.

39. Fodor D, Bondor C, Albu A, Simon S P, Craciun A, Muntean L. The value of osteopontin in the assessment of bone mineral density status in postmenopausal women. *Journal of Investigative Medicine*. 2013; 61(1): 15-21.

40. Zhang F J, Gao S G, Cheng L, Tian J, Xu W S, Luo W, Lei G H. The effect of hyaluronic acid on osteopontin and CD44 mRNA of fibroblast-like synoviocytes in patients with osteoarthritis of the knee. *Rheumatology international*. 2013; 33: 79-83.

41. Bai R J, Li Y S, Zhang F J. Osteopontin, a bridge links osteoarthritis and osteoporosis. *Frontiers in Endocrinology*. 2022; 13: 1012508.

42. Khosla S, Pacifici, R. Estrogen deficiency, postmenopausal osteoporosis, and age-related bone loss. In *Osteoporosis*. 2013; 1113-1136.

43. Li J Y, Chassaing B, Tyagi A M, Vaccaro C, Luo T, Adams J, Pacifici R. Sex steroid deficiency-associated bone loss is microbiota dependent and prevented by probiotics. *The Journal of clinical investigation*. 2016; 126(6): 2049-2063.

44. Lorenzo J. From the gut to bone: connecting the gut microbiota with Th17 T lymphocytes and postmenopausal osteoporosis. *The Journal of Clinical Investigation*. 2021; 131(5).

45. Foertsch S, Haffner-Luntzer M, Kroner J, Gross F, Kaiser K, Erber M, Ignatius A. Chronic psychosocial stress

disturbs long-bone growth in adolescent mice. Disease models and mechanisms. 2017; 10(12): 1399-1409.

46. Catalano A, Martino G, Bellone F, Gaudio A, Lasco C, Langher V, Morabito N. Anxiety levels predict fracture risk in postmenopausal women assessed for osteoporosis. *Menopause*. 2018; 25(10): 1110-1115.

47. Mazziotti G, Frara S, Giustina A. Pituitary diseases and bone. *Endocrine reviews*. 2018; 39(4): 440-488.

48. Ng J S, Chin K Y. Potential mechanisms linking psychological stress to bone health. *International Journal of Medical Sciences*. 2021; 18(3): 604.

49. Cho E H, Cho K H, Lee H A, Kim S W. High serum osteopontin levels are associated with low bone mineral density in postmenopausal women. *Journal of Korean medical science*. 2013; 28(10): 1496-1499.

التقييم الفسيولوجي للاوستيوبونتين والفسفاتيز القلوي العظمي كعلامات استقلابية لدى النساء بعد سن اليأس المصابات بهشاشة العظام

صابرين مالك نايف¹, فارس شاكر كاطع¹

¹ قسم علوم الحياة /كلية التربية للعلوم الصرفة/ جامعة البصرة- العراق

الخلاصة:

هشاشة العظام حالة مرضية تنتج عن خلل في التوازن الديناميكي لإعادة تشكيل العظام ، وتتضمن تفاعلات معقدة بين مؤشرات الامتصاص ومؤشرات التكوين على المستوى الخلوي تتأثر هذه التفاعلات بعدة عوامل جزيئية وهرمونية مختلفة، مما يؤدي في النهاية إلى زيادة معدل ارتشاف العظام بما يتجاوز معدل تكوينها. يُعد تقييم علامات دوران العظام أداة حاسمة في ادارة ومراقبة مرضى هشاشة العظام. ومن بين المؤشرات الكيميائية الحيوية التي تم اعتمادها، يُعد انزيم الفوسفاتيز القلوي العظمي (B-ALP) والأوستيوبونتين (OPN) بمثابة علامات مرجعية لتكوين العظام وامتصاصها. أُجريت هذه الدراسة على النساء بعد انقطاع الطمث مُشخصات بهشاشة العظام، في محافظة البصرة-العراق. وقد هدفت هذه الدراسة الى فحص العلاقة بين هشاشة العظام وتراكيز المؤشرين الحيويين انزيم الفوسفاتيز القلوي العظمي (B-ALP) و Osteopontin (OPN) في مصل النساء بعد انقاع الطمث. جمعت 80 عينة دم من النساء بعد سن اليأس ، أذ قُسمت الى 55 عينة مصل لنساء يعانين من هشاشة العظام و25 عينة مصل لنساء سليمات، كما وتم توزيع عينات المرضى الى فئتين اعتماداً على العمر والشدة المرض . أظهرت نتائج التحليل الاحصائي حصول ارتفاع معنوي في تركيز بروتين الأوستيوبونتين عند مستوى احتمالية ($P<0.001$) في مجموعة النساء المرضى بهشاشة العظام مقارنةً بمجموعة النساء الأصحاء. كما لوحظ حدوث انخفاض معنوي ملحوظ في تركيز انزيم B-ALP في مصل النساء المرضى بهشاشة مقارنةً بالنساء الاصحاء وفقاً لاختبار T-test . نستنتج من الدراسة الحالية ان الاضطراب في مستوى المؤشرين الحيويين في مصل الدم لدى النساء اللاتي يعانين من هشاشة العظام وفي مرحلة سن اليأس دوراً هاماً في الكشف عن مرض هشاشة ومتابعة تطوره وله ارتباط ايضي وثيق بين المرض والمؤشرات. تشير نتائج هذا البحث إلى أن التغيرات في مستويات الدم من OPN و B-ALP مهمة للنساء بعد انقطاع الطمث لتكون قادرة على التعرف على مسار هشاشة العظام وتتبعه في وقت مبكر وتدعم هذه النتائج الاستخدام المحتمل لـ OPN و B-ALP كمؤشرات كيميائية حيوية مبكرة لمراقبة مرض هشاشة العظام بعد انقطاع الطمث.

الكلمات المفتاحية: هشاشة العظام ، الأوستيوبونتين ، الفوسفاتيز القلوي العظمي، المؤشرات العظمية الحيوية.