Evaluation of Some adipokines Levels in Pre and Postmenopausal Osteoporosis Women

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Abstract
The most frequent form of chronic metabolic bone disease is osteoporosis, which is defined by increased bone fragility. Osteoporosis can be linked to a variety of variables, including menopause and age. Because menopause and advanced age produce an imbalance between the rates of resorption and creation (resorption becomes higher than absorption), the risk of fractures increases. The participants' ages varied from 18 to 76 years old, and the research was carried out at the Dijla Hospital for Medical Rehabilitation in Tikrit. The study began in December 2021 and continued until April 2022. The overall number of subjects was 80 people, with 50 of them being pre and postmenopausal women who had osteoporosis (25 for each group), and the remaining 30 being pre and postmenopausal women who were healthy (15 for each group). According to the results of our research, premenopausal and postmenopausal women who had osteoporosis had significantly higher levels of the hormones resistin, adiponectin, and chemerin compared to the control group. The research came to the conclusion that there was an increase in adipokines, which are regarded to be diagnostic indicators for the osteoporosis condition.
Osteoporosis, Chemerin, Resistin, and Adiponectin are some keywords to keep in mind.

1. Introduction

Loss of bone mass and microstructural deterioration of bone structure are two symptoms of the systemic disease osteoporosis, which can increase bone fragility and the risk of fractures (1). The patient frequently needs to be hospitalized as a result of this asymptomatic condition being misdiagnosed until it manifests as a low-trauma fracture of the hip, spine, proximal humerus, pelvis, and/or wrist (2). Bone mineral density (BMD) testing, particularly in the hip and lumbar spine using a technology called dual-energy x-ray absorptiometry (DXA), or the development of nontraumatic hip or vertebral fractures are the two best ways to diagnose osteoporosis. The presence of osteopenia is another way to identify osteoporosis (3). The alteration in bone remodeling that results from an imbalance between bone formation and bone resorption, with bone resorption taking precedence, is the root cause of osteoporosis. This imbalance results in the loss of bone mass and the emergence of microstructural deviations known as body composition deviations, which may cause osteoporosis (4).

An 18 kDa protein chemoattractant called chemerin is released by adipocytes, which are mostly derived from fat but can also be found in other tissues (5). This substance has been given the name of a novel adipocytokine. Due to the fact that chemerin plays a dual role in both the immune system and the metabolism, it may be at the intersection of inflammation and obesity (6). The process of adipogenesis and osteogenesis can both be affected by chemerin, which belongs to the adipokines family. The function of its regulation is primarily focused on the adipogenesis side of things. During the process of adipocyte differentiation, the level of expression and secretion of chemerin significantly increased. A decrease in the expression of chemerin or chemerin-like receptor 1 (CMKLR1) could prevent adipocytes from differentiating and lead to an increase in the expression of osteoblast marker genes and mineralization in response to osteoblastic stimulation (7).

Located in inflammatory zone 3, resistin is a protein with a molecular weight of 12.5 kDa. It is also known as adipocyte-secreted factor (FIZZ3). The protein known as resistin is a member of the FIZZ family, which consists of 108 different amino acids (8). Resistin-like molecules are another name for this family. Within macrophages, PBMCs, and vascular cells, human resistin is one of the inflammatory regulators that is responsible for controlling the downstream activity of inflammation. Human macrophage cells, peripheral blood mononuclear cells (PBMCs), and hepatic stellar cells all produce TNF-a, IL-6, IL-12, and MCP-1 via an NFjB-mediated pathway in response to stimulation with recombinant human resistin (9). There is evidence that osteoblasts and osteoclasts also express the protein known as resistin. During the process of osteoclast development, there is an increase in the production of this adipokine. Recombinant mouse resistin

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stimulates the differentiation of osteoclasts as well as the proliferation of osteoblasts (10).

The current study found that the mean and standard deviation of the chemerin levels in the serum of premenopausal osteoporosis women were (121.5386.21) ng/ml and the control group was (6.165.22) ng/ml respectively. On the other hand, the chemerin levels in the serum of postmenopausal osteoporosis women were (80.7637.50) ng/ml and the control group were (4.

The resistin levels in the serum of premenopausal osteoporosis women were (16.882.29) ng/ml and the control group were (8.851.23) ng/ml respectively; however, the resistin levels in the serum of postmenopausal osteoporosis women were (17.661.70) ng/ml and the control group were (6.950.60) ng/ml respectively; this indicated a highly significant difference (P

There was a significant difference (P 0.01) in the osteoporotic groups when compared with the control groups. The mean and standard deviation of the adiponectin levels in the serum of premenopausal osteoporosis women were 1.620.32 ng/ml, while the control group had 0.630.12 ng/ml. The adiponectin levels in the serum of postmenopausal osteoporosis women were 1.47 Table 1 displayed all of the results.

Recent research has shown that adiponectin has a direct effect on osteoblast proliferation and differentiation, and that it also inhibits osteoclastogenesis caused by tumor necrosis factor alpha (TNF-alpha) and receptor activator of nuclear factor kappa B ligand (RANKL) (14). On the other hand, other research suggests that adiponectin may have negative effects on bone metabolism. These negative effects may be caused by the fact that adiponectin stimulates the RANKL pathway while also preventing the synthesis of osteoprotegerin and the decoy receptor for RANKL (15).

2. Materials and Methods

After collecting each sample of 5 ml of blood from osteoporosis patients and placing it in a plain tube, the blood was allowed to clot for twenty to thirty minutes before being centrifuged for five to fifteen minutes at four thousand revolutions per minute to obtain serum. The serum was then divided into aliquots, placed in plastic tubes, and stored at a temperature of minus twenty degrees Celsius until the time of assessment. The Dijla Hospital for Medical Rehabilitation in Tikrit was the location of the study that was carried out. The beginning of the study was in December 2021, and it lasted until April 2022. The ages of the people who participated in the study ranged from 18 to 76 years old. The overall number of subjects was 80 people, with 50 of them being pre and postmenopausal women who had osteoporosis (25 for each group), and the remaining 30 being pre and postmenopausal women who were healthy (15 for each group). Using dual-energy X-ray absorptiometry, the patient groups were given a diagnosis after having their bone mineral density (BMD) measured through the lumbar spine (L2-L4) as well as the right and left femurs (DEXA). People who had hypertension, diabetes, thyroid disease, rheumatoid arthritis, hepatic or renal insufficiency, any kind of cancer or precancerous condition, or any of those disorders were not allowed to participate in the study. Estimates for several other assays, such as chemerin, resistin, and adiponectin, were obtained by employing the sandwich approach with enzyme-linked immunosorbent assay (ELISA) kits purchased from mybiosource (16). Statistical analysis was carried out with the assistance of (SPSS, 21), and a comparison of the results obtained from the t-test was carried out across the various groups. The level of statistical significance was determined to be (P0.05), according to the calculations.

3. Results

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The mean and standard deviation of the adiponectin levels in the serum of premenopausal women with osteoporosis were 1.62 and 0.32 ng/ml respectively, while the levels in the serum of postmenopausal women with osteoporosis were 1.47 and 0.27 ng/ml respectively. There was a significant difference (P 0.01) in the osteoporotic groups when compared with the control groups. The findings are shown in table 1, as a whole.

Table (1): - Levels of Adipokines in Pre and Postmenopausal Osteoporosis Women with control groups

parameters	Mean±S.D Premenopause patients	Mean±S.D Premenopause Control	P- value	Mean±S.D Postmenopause patients	Mean±S.D Postmenopause Control	P- value
Chemerin (ng/ml)	(121.53±86.21)	(6.16±5.22)	(P≤0.01)	(80.76±37.5)	(4.56±1.89)	(P≤0.01)
Resistin (ng/ml)	(16.88±2.29)	(8.85±1.23)	(P≤0.01)	(17.66±1.70)	(6.95±0.60)	(P≤0.01)
adiponectin (ng/ml)	(1.62±0.32)	(0.63±0.12)	(P≤0.01)	(1.47±0.27)	(0.81±0.68)	(P≤0.01)

4. Discussion

Experimental evidence suggests that chemerin may stimulate adipocyte differentiation at the expense of osteoblastogenesis, which may be the cause of increased levels of chemerin in premenopausal and postmenopausal women with osteoporosis in this study when compared with the control group. This may be due to the fact that this study used a control group. In addition, chemerin is responsible for regulating the differentiation of osteoclasts; hence, an increase in chemerin levels will result in an almost total absence of osteoclastogenesis. As a result, larger levels of chemerin led to a stronger inhibition of bone growth as well as stronger support for bone resorption (17). There has not been a lot of research done on the correlation between high chemerin levels and high bone density in individuals. The association between chemerin and BMD has only been the subject of a limited number of epidemiological research, which have all pointed to a causal link in the opposite direction between the two. On the other hand, it might be challenging to locate an in-depth comparison of these research due to the fact that the study groups themselves have varying characteristics (18).

When compared with controls, the levels of resistin were higher in premenopausal and postmenopausal women with osteoporosis. Resistin levels are inversely proportional to bone mineral density in the lumbar spine in adult men and women (19). As a result of recombinant resistin's ability to stimulate osteoclast development as well as osteoblast proliferation, the production of this lipoprotein goes up during the osteoclast differentiation process (20). Even though some research have revealed that resistin is not related with bone mineral density (BMD), it nevertheless makes a contribution to bone metabolism and remodeling by promoting osteoclast differentiation and osteoblast recruitment (21). Recent research has shown that patients who suffer from osteoporosis have resistin levels that are inversely related with osteocalcin (19).

There is a possibility that the presence of adiponectin receptors, as well as the transcription, translation, and secretion of the adiponectin protein in human bone-forming osteoblasts, are responsible for the increased levels of adiponectin that were found in premenopausal and postmenopausal women with osteoporosis when compared with controls. This is because premenopausal and postmenopausal women are more likely to experience Additionally, an increase in the formation of osteoblasts and osteoclasts was brought about by the introduction of recombinant adiponectin into human osteoblasts. It is possible that an increase in the activity of the RANKL pathway is responsible for the effect that adiponectin has on bone metabolism. (22).

5. Conclusion

The study concluded that the Chemerin, Resistin and Adiponectin were increased in pre and postmenopausal osteoporosis women which considered as diagnostic markers to diagnosis the osteoporosis disease according to age, sex and BMD.

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