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Role of Serum Omentin-1 and Bone Metabolism Markersin Osteoporosis among Postmenopausal Women

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Abstract.
menopause is the most common cause of accelerated bone loss in women. Omentin-1 is secreted by the visceral adipose tissue and it isdonated in the regulation of bone metabolism in postmenopausal women which is related with decreased bone mineral density.
The current study aims to investigate the role of serum omentin-1and some bone metabolism markers in osteoporosis among postmenopausal Iraqi women.
This study was performedin the Rheumatology and Rehabilitation Unit, Baghdad Teaching Hospital/ Baghdad medical city, during the period from November 2019 to July 2020 with a total of 60 postmenopausal women(30 with osteoporosis and 30 without osteoporosis), their ages ranged from 52 to 62 years. They were compared with 30 healthy premenopausal women as control group, their ages ranged from 32-42 years.The postmenopausal status was determined as termination of menses for at least 1 year. All clinical and biochemical factors were measured in these individuals.
There were significant decreases (p= 0.0001) in serum omentin-1 and growth differentiation factor11 levels in postmenopausal groups as compared to premenopausal. While a remarkable increase (p= 0.0001) was found in serum sclerostin level in postmenopausal group with osteoporosis as paralleled to those without osteoporosis and premenopausal group (OR 3.07, 95%CI 2.19-4.63). The significant alterations in serum omentin-1, sclerostinand growth differentiation factor11levels among postmenopausal women as compared to premenopausal group and they +were suggested thepossible role of these adipokines in bone metabolism. Moreover, in osteoporotic postmenopausal, estrogens deficiency enhance bone resorption. So, assessment of these adipokines levels could be beneficial in early detection and prevention of its unfavorable consequences of osteoporosis.

Keywords: Postmenopause, Osteoporosis, Omentin-1, Bone mineral density, Bone markers.

1.Introduction:

Menopause is the period of life when menstruation ceases due to decreased production of the ovarian hormones, estrogen and progesterone. Menopause is also one of the most important periods that has favor weight gain and lead to obesity [1].
The bone mineral density (BMD)is decreased when the estrogen level decreases, beginning in perimenopause. From the age of 40 until the menopause, the annual cortical bone loss is approximately 0.3–0.5%. After the menopause, the process accelerates to around 3%, and only 8 years after the menopause does it return to levels observed initially. This due to the inhibition of calcium (Ca^{+2}) absorption from the digestive tract. The Ca^{+2} and vitamin D intake are associated with the BMD. The loss of bone tissue results in the disease called osteoporosis [2].

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Osteoporosis is a systemic structural disease manifested by reduced bone mass and micro-architectural degradation of bone tissue with the consequent increase in osteoporosis, vulnerability to fractures, and decreased the quality of life [3].

Cytokines derived from adipose tissue also appear to play an important role in bone metabolism, which can modulate bone cell metabolism *in vitro* and *in vivo*, and omentin-1 in particular may play an important role in the dynamic balance of bone formation and bone resorption [4].

Omentin is a novel hydrophilic adipokine of 313 amino acids (35 kDa), which has a secretory signal sequence and a fibrinogen-related domain, and appears as a glycolized trimer with a molecular weight of 120 kDain its negative form [5].Omentin-1 isdescribed to inhibit osteoblast differentiation *in vitro* [6].

Various candidate genes associated with BMD or osteoporosis have been recognized. The sclerostin gene (*SOST*) located on chromosome 17q12–q21 encodes a secreted protein, known as sclerostin, which is a 21 kDa glycoprotein secreted almost exclusively by osteocytes and to a reduced extent other cell types, i.e., kidney and vascular [7]. Sclerostin indirectly promotes bone resorption via control of the expression of osteoclast regulators by osteoblasts to enhancing osteoclast differentiation [8].

Growth differentiation factor 11 (GDF11) is also identified as bone morphogenetic protein 11 (BMP-11) is a protein that in humans is encoded by the GDF11 gene. The GDF11 is a member of the transforming growth factor-beta (TGF-β) superfamily and many of its members were interested in organizing bone remodeling. As such, TGF-stimulates osteoclast and oppressive formation in regulating bone remodeling. As, TGF-β induces osteoclast formation and represses[9].

This study aims to investigate the role of serum omentin-1 and some bone metabolism markers in osteoporosis among postmenopausal Iraqi women.

2.Subjects and Methods:

The current study was performedin the Rheumatology and Rehabilitation Unit, Baghdad Teaching Hospital/ Baghdad medical city during the period from November 2019 to July 2020 with a total of 60 postmenopausal women(30 with osteoporosis and 30 without osteoporosis), their ages ranged from 52-62 years.They were compared with 30 healthy premenopausal women as control group,their ages ranged from 32 to 42 years.The postmenopausal status was defined as termination of menses for at least 1 year. All individuals were examined and diagnosed by physicians in a Rheumatology and Rehabilitation Outpatient Clinic.Osteoporotic women were diagnosed thethedual energy x-ray absorptiometry (DEXA) and the diagnostic criteria of osteoporosis was recommended by the WHO [10]. The DEXA test results are presented as a T-score and a Z- score. The results are normalized to age- and gender-matched members of the general population, generating a Z-score. Normalization against a population of young healthy adults givesa T-score,Table1.

Table 1. The WHO classification criteria for T-scores [10]

T-Score	Indication of BMD
≥ -1	Normal
< -1 to > -2.5	Osteopenia

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≤ -2.5	Osteoporosis
≤ -4	Sever osteoporosis

2.1.Exclusion Criteria:

Women with diabetes, any rheumatic disease, high blood pressure, malignant tumors, urinary tract stones, gout, smoking, abnormal liver, kidney or thyroid function are not included. In addition, topics related to drugs that can affect bone metabolism such as bisphosphonates, steroids, β-blockers, hormone replacement therapy, vitamin D, or other anti-osteoporotic drugs are not included. Flow diagram of the present study is presented in'Figure 1'.

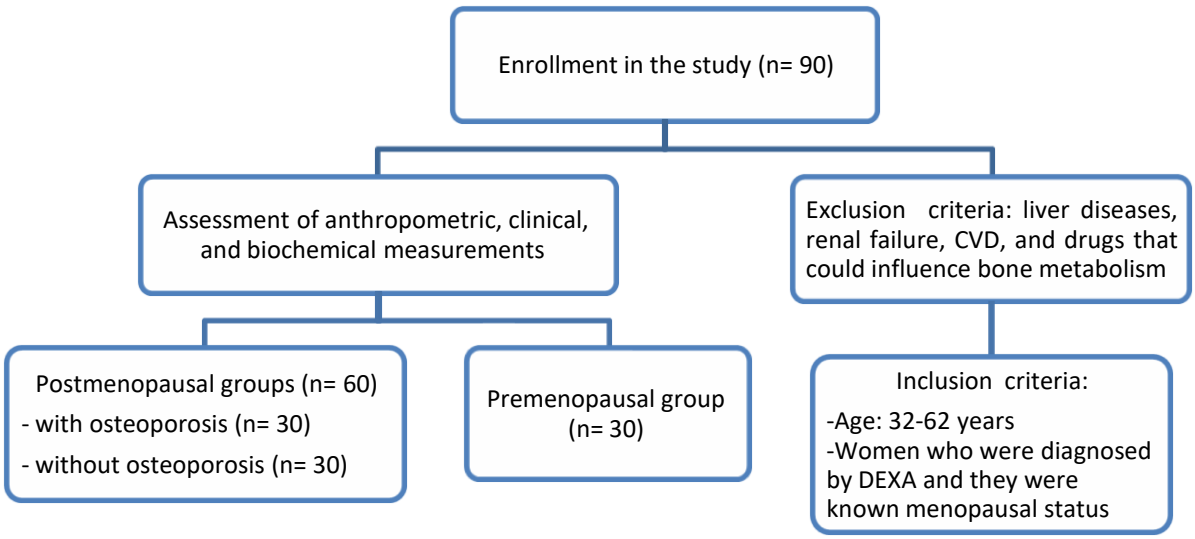

Figure 1. Flow diagram of the present study

2.2.Informed Consent:

Informed written approval was acquired from all subjectsformer to their contributing in this study.

2.3.Blood Samples:

Ten milliliters of blood were taken from each patient and control after 12 hours of fasting. The sample was transferred to clean tube, left for coagulation, then centrifuged and the separated serum was kept in clean tube in the refrigerator at -20°C until the time of the assay.

2.4.Anthropometric and Clinical Measurements:

The height, weight, and waist circumference (WC) were measured. Also, waist to hip ratio (W/H)ratio was calculated. Systolic-and diastolic blood pressure (SBP, DBP) measurements

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were done after subjectand rested for at least 5 minutes [11]. Body mass index is calculated usingthe following formula: BMI= mass (Kg)/ (height (m))^2[12].
The percentage of body fat (BF%) can be estimated by the following formula:
BF%= (1.2×BMI) +(0.23× age) - 5.4- (10.8 × gender). Where gender is 0 if female and 1 if male[13].

2.4.Laboratory Measurements:

Fasting serum glucose (FSG) and lipid profile encompassing: [total cholesterol (TC), triacylglycerol (TAG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein (VLDL)] were determined using standard biochemical techniquesby Siemens kits, Germany. Serum non HDL-C was calculated using the formula: Serum non HDL-C= (TC–HDL-C) [14].
Serum Ca^{+2}, alkaline phosphatase (ALP), and inorganic phosphorus were measured byBiomerieux kits, France usingKenza 240TX, Italian. Serum vitamin D3 was measured according to the assay principle combined an enzyme immunoassay competition method with a final fluorescent detection (ELFA)method byBiomerieux kit, France.Hypovitaminosis is defined by most experts as a serum vitamin D level < 20 ng/mL, whereas a serum vitamin D level of > 30 ng/mL is deliberated to be 20–30 ng/mL describes vitamin D insufficiency [15].Serum omentin-1, sclerostin, and GDF11 were measured using enzyme linked immuno sorbent assay (ELISA) method by Melsin kits, China[16, 17, 18] respectively.

2.5.Statistical Analysis:

All statistical designs were carried using computer programs SPSS (Statistical Package of Social Science) program, version 17 software.
Student t-test was used for comparison of numerical variables between the study groups and the comparison of variables between more than two groups was performed using ANOVA test. Chi-square test was done to compare the significance between percentage 0.05 and 0.01 probability. Also, the odd ratio (OR) and confidence interval (CI) in serum omentin-1, sclerostin, and GDF11 levels for postmenopausal groups were calculated to confirm the significance. Data were expressed as mean ± standard deviations (mean ± SD) which are comparable by Duncan's multiple range tests; statistical meaning was set at $p< 0.05$.

3.Results:

Table 1 shows the basic anthropometric and clinical characteristicsvalues ofthe studied post- and premenopausal women. There were significant increases ($p= 0.0001$) in age, BMI, and BF% in postmenopausal groups as compared to premenopausal. Also, there were significant increases ($p= 0.0001$) in WC, W/H ratio, SBP, and DBP in osteoporotic postmenopausal women as paralleled to those without osteoporosisand premenopausal group. Moreover, osteoporotic postmenopausal women showed longer duration of menopause ($p= 0.0026$) than those without osteoporosis. These women havea number of children greater than those without osteoporosis and premenopausal group.

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Table 1. Anthropometric and clinical features of the study groups

Parameters	Postmenopausal Women		Premenopausal Women (n=30)	*p* value
	with Osteoporosis (n= 30)	without Osteoporosis (n= 30)		
Age (Years)	57.63 ± 5.14[a]	56.13 ± 4.37[a]	36.53 ± 4.56[b]	0.0001
WC (cm)	98.36 ± 9.52[a]	92.53 ± 11.11[b]	71.70 ± 3.61[c]	0.0001
W/H ratio	0.92 ± 0.06[a]	0.85 ± 0.02[b]	0.72 ± 0.04[c]	0.0001
BMI (kg/m^2)	31.01 ± 4.84[a]	29.62 ± 4.98[a]	22.37 ± 1.63[b]	0.0001
BF%	45.38 ± 5.75[a]	43.35 ± 6.64[a]	30.07 ± 3.01[b]	0.0001
SBP (mmHg)	141.93 ± 10.28[a]	129.73 ± 7.57[b]	119.60 ± 2.44[c]	0.0001
DBP (mmHg)	90.93 ± 5.86[a]	84.13 ± 4.16[b]	78.76 ± 2.36[c]	0.0001
Menopause Age (Years)	50.82 ± 2.10	51.50 ± 2.27	-	0.060
Menopause Duration (Years)	8.16 ± 4.28	4.67 ± 2.31	-	0.0026
Number of Children	3.13 ± 2.11[a]	2.85 ± 1.52[a]	2.77 ± 3.40[a]	0.07

Data are expressed as mean±SDSimilar letters indicate that there are no significant variances and different letters
indicate significant variances, p < 0.05: Significant, p < 0.001: Highly significant.

Table 2 revealed the metabolic profile of the study groups. There were significant differences (*p*= 0.0001) in glycemic and lipid factors among osteoporoticpostmenopausal women as paralleled to those without osteoporosis and premenopausal group.

Table 3 lists the DEXA results in postmenopausal women groups. There were significant decreases (*p*= 0.0001) in BMD, bone mineral content (BMC), T-score, and Z-score in osteoporotic postmenopausal women as paralleled to those without osteoporosis.

Table 2. Metabolic profile of the study groups

Parameters	Postmenopausal Women		Premenopausal Women (n= 30)	*p* value
	with Osteoporosis (n= 30)	without Osteoporosis (n= 30)		
FSG (mg/dL)	118.23 ± 10.40[a]	109.36 ± 11.90[b]	86.26 ± 5.95[c]	0.0001
TC (mg/dL)	282.83 ± 13.24[a]	206.16 ± 9.84[b]	134.86 ± 6.58[c]	0.0001
TAG (mg/dL)	216.96 ± 14.79[a]	163.36 ± 12.02[b]	97.37 ± 8.46[c]	0.0001

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HDL-C (mg/dL)	42.70 ± 7.20[b]	44.33 ± 5.54[b]	68.09 ± 5.75[a]	0.0001
LDL-C (mg/dL)	196.74 ± 13.54[a]	127.40 ± 8.70[b]	47.28 ± 6.89[c]	0.0001
VLDL (mg/dL)	43.39 ± 14.95[a]	33.51 ± 13.71[b]	19.47 ± 3.49[c]	0.0001
Non HDL-C (mg/dL)	240.13 ± 14.30[a]	161.83 ± 10.45[b]	66.76 ± 8.13[c]	0.0001

Data are expressed as mean±SDSimilar letters indicate that there are no significant variances and different
Letters indicate significant variances,p < 0.05: Significant, p < 0. 001: Highly significant.

Table 3. Characteristics of DEXA measurements in postmenopausal women

Parameters	Postmenopausal Women		*p* value
	with Osteoporosis (n= 30)	without Osteoporosis (n= 30)	
L1-L4 BMD (g/cm^2)	0.77 ± 0.08	1.00 ± 0.15	0.0001
L1-L4 BMC (g)	41.43 ± 0.65	51.93 ± 10.35	0.0001
L1-L4 T-Score	-2.62 ± 0.69	-0.63 ± 1.30	0.0001
L1-L4 Z-Score	-1.29 ± 0.56	0.47 ± 1.41	0.0001
Normal	-	22 (73.33%)	-
Osteopenia	10 (33.33%)	8 (26.67%)	0.62
Osteoporosis	2 (6.67%)	-	-
Sever Osteoporosis	18 (60%)	-	-

p< 0.05: Significant, p < 0. 001: Highly significant.

In addition, bone metabolism factors of the study groups are demonstrated in Table 4. There were significant decreases (*p*= 0.0001) in serum Ca^{+2} and vitamin D3 in postmenopausal groups as paralleled to premenopausal. While, there was a significant rise (*p*= 0.0001) in serum ALP level in osteoporotic postmenopausal women as paralleled to those without osteoporosisand premenopausal group. However, there is no important significant was found for elevation of phosphorus level in osteoporotic postmenopausal women.

Table 4. Bone metabolism factors of the study groups

Parameters	Postmenopausal Women	Premenopausal	*p* value

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	with Osteoporosis (n= 30)	without Osteoporosis (n= 30)	Women (n= 30)	
Ca^{+2} (mg/dL)	4.09 ± 1.12[b]	4.50 ± 1.27[b]	9.80 ± 0.40[a]	0.0001
ALP (U/L)	77.20 ± 11.64[a]	65.40 ± 13.14[b]	59.13 ± 0.71[c]	0.0001
Phosphorus (mg/dL)	4.35 ± 0.33[a]	3.13 ± 0.64[a]	2.89 ± 0.71[a]	0.06
Vitamin D3 (ng/mL)	10.0 3 ± 2.67[c]	14.01 ± 3.94[b]	43.06 ± 4.03[a]	0.0001

Data are expressed as mean±SDSimilar letters indicate that there are no significant variances and different
letters indicate significant variances, p < 0.05: Significant, p < 0.001: Highly significant.

There were significant decreases (*p*= 0.0001) in serum omentin-1 and GDF11 levels in postmenopausal groups as compared to premenopausal. While, a significant rise(*p*= 0.0001) was found in serum sclerostin level in osteoporotic postmenopausal groups as paralleled to those without osteoporosis and premenopausal group (OR 3.07, 95%CI 2.19-4.63), Table 5.

Table 5. Serum omentin-1, sclerostin, and GDF11 levels in the study groups

Parameters	Postmenopausal Women		Premenopausal Women(n= 30)	OR	95% CI	*p* value
	with Osteoporosis (n= 30)	without Osteoporosis (n= 30)				
Omentin-1 (ng/mL)	19.40 ±4.64[b]	21.16 ± 5.05[b]	61.10 ± 7.20[a]	2.48	1.03-4.10	0.0001
Sclerostin (ρg/mL)	379.93 ± 6.13[a]	342.16 ± 5.44[b]	334.70 ± 3.71[c]	3.07	2.19-4.63	0.0001
GDF11 (ng/mL)	30.63 ± 4.47[b]	31.33 ± 3.23[b]	56.23 ± 5.72[a]	2.16	0.97-3.26	0.0001

Data are expressed as mean±SD. Similar letters indicate that there are no significant differences and different letters
indicatesignificant differences,p < 0.05: Significant, p < 0. 001: Highly significant.

5.Discussion:

Women's life undergoes different phases and menopause is one of them. There are many factors affecting the timing and course of this change in her life. There are not only medication that affect the same but studies reflect geographic and international differences in the age at menopause which indicate a genetic, socioeconomic, environmental, racial/ethnic, or lifestyle [19]. Also, geographical differences and their socioeconomic differences play a role in the age of

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attaining menopause. In a study conducted by Akahosi*et al.*,over 1136 natural menopause women, the age of menopause ranged from from 45 to 49 years [20].

The WC was used to measure the abdominal obesity which is an important influence in health, even regardless of BMI. It is an alternative body fat measure because it measures excess weight, not excess fat. Similarto the WC, the W/H ratio is also used to measure the abdominal obesity. Higher body fat is also a source of reproductive hormones thus interfering with the physiology of menopause[21]. Paralleldata were achieved in the current study where higher BMI was associated with a higher age at menopause.The SBP and DBP were significantly greater in postmenopausal women than that in the premenopausal. These data are in harmony with a former study [22].

Endogenous estrogen has a suppressive effect on lipase activity in the liver. Decreased estrogen levels during peri- and postmenopause were associated with elevated lipase activity in the liver.Furthermore, estrogen also donated to regulation of lipoprotein lipase (LPL), which is accountable for hydrolyzing TAG to chylomicrons and VLDL [23], Therefore, reduced estrogen in menopause period might cause dysregulation of LPL. Sahmani*et al.*'s, study on postmenopausal women showed an inverse correlation between cholesterol level and BMD [24].Non HDL-C is anextent of the amount of cholesterol carried by the atherogenic B containing lipoproteins. The treatment goal for non HDL-C is set at 30 mg/dL higher than that for LDL-C [25].

In postmenopause, estrogen deficiency is related with a rise in bone turnover and loss of the balance between bone resorption and bone formation, resulting in excessive bone resorption [26]. The outcomes of this study showed that the osteoporotic postmenopausal group has lower BMD and T-score, which is revealed that there was an association between T-score and age groups. Similarly, there was a relationship between BMD and age of postmenopausal groups, which suggests that age has a significantinfluence in the decline of BMD and accordingly the occurrence of osteoporosis.

The statistical analysis of the results showed an importantassociation between the levels of serum phosphorous and ALP with the occurrence of osteoporosis, while there were no significant difference in serum Ca^{+2} between postmenopausal women with and without osteoporosis.This is due to the markers of bone formation are produced directly or indirectly at every stage of osteoblast differentiation, although, serum ALP level was within normal range. These indicate that osteoporotic women included in this study did not have metabolic bone disease other than osteoporosis [27].

The vitamin D promotes Ca^{+2} absorption in the gastrointestinal tract and assist in maintaining adequate serum Ca^{+2}levels to enable proper mineralization of the bone. Vitamin D is indispensable for bone growth and bone remodeling by osteoblasts and osteoclasts. The levels of vitamin D showed a decreasing trend with age. Advanced age leads to multiple alterations in the metabolism of vitamin D and its precursors which ultimately lead to its deficiency. This could attributed to the fact that synthesis of vitamin D declines with age due to decline in synthesis by skin, decrease in absorption from food or by decline in renal function [28]. Reduced estrogen levels contribute to lower vitamin D binding protein (VDBP), and subsequently lower vitamin D levels in blood. This deficiency of vitamin D can adversely affect bodily functions, resulting in numerousconditionsparticularly in postmenopausal women. As designated by Kuhn, postmenopausal women haslesser levels of this vitamin than their premenopausal counterparts [29].Genetic, physiological, environmental, and modifiable lifestyle factors can also play a significant role in bone mass. Vitamin D deficiency leads to a lessening in the intestinal absorption of Ca^{+2}, decreasing its status and causing the release of parathyroid hormone (PTH)

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levels of which are contrary proportionate to the levels of vitamin D[30]. In menopause period, women will have thinner skin and a lesser capacity for vitamin D production. In addition, the reduction of intestinal absorption of vitamin D and hydroxylation of vitamin D in the liver and kidneys [46].These metabolic disorders will be attended by a tendency towards limited outdoor activity and a lesser dietary intake of vitamin D [31].

It has been proposed that omentin-1 plays a key role in bone homeostasis, inhibiting the anti-osteoblastic and pro-osteoclastic influence of triggered macrophages. It was shown that omentin-1 can inhibit osteoblast differentiation in vitro. The present data exhibitedlow concentrations of serum omentin-1 were adverselyrelated with BMI values. Omentin-1 has shown to be decreased in obese subjects. This might be indicative the role of adiposity in regulating omentin-1 expression[32].

Tohidi*et al*., have found a remarkable negative association between serum omentin-1 and BMD [33] and the study of Zhang *et al*., supports this conversecorrelation. However, this correlation was not statistically important [34]. In contrary, Wang *et al*., study in women before and after menopause revealed that the effect of omentin-1 on bone metabolism depends on the state of menopause, and they discovered that there was an inverse association between serum omentin-1 and BMD in the premenopausal. However, no postmenopausal women. *In vitro* and *in vivo*data have indicated that omentin-1 may play a protecting role in both bone remodeling and BMD [35].

Exhaustive analysis indicated that serum sclerostinelevated beyond the 45 years. The present dataindicates that the serum sclerostin levels raisedconsiderably with age in women. Hence, serum sclerostin levels were significantly increased in postmenopausal women as compared to premenopausal. This proposes that raisedsclerostin production by osteocytes may be complicated in the age-related decline of bone formation[36]. The difference in mean reference for serum sclerostin in the studies is possible due to environmental or lifestyle and/or genetic effects on bone mass. In the currentwork, the pattern of age-related variations in serum sclerostin among women is revealedthrough increase bone turnover at menopause. The higher serum sclerostinconcentrations in postmenopausal women may be a reason, an influence, or both of the increased bone turnover increasingat the postmenopausal state. These interpretations are reliable with formerly data, which indicate that highersclerostinlevels could result in reduced bone turnover via some compensatory mechanism [37, 38].

The main outcomes of the current study are thatserum concentrations of GDF11 peaks at 41-50 years of age and gradually declining thereafter with significant reductionsdetected after 52 years age. Aging is characterized by a progressive rise in body adiposity that may be concurrently accompanied by a lessening in muscle mass related with an elevated risk of morbidity and mortality. Also, there was significant variance in serum GDF11 levels in osteoporotic postmenopausal women who were obese as compared to those without osteoporosis. This lead to a strong association of GDF11 levels with BMI or body adiposity, which is in agreement with previous study [39]. In contrast,Schafer*et al*., revealed that GDF11 levels do not decline throughout aging and there was no variance between sexes in healthy adults [40].

6.Conclusions:

Postmenopausal women in this study especially those with osteoporosis, experienced metabolic disorders. So, primary detection of such parameters might be of potential benefit for applying protective measures to reduce the progress of osteoporosis. The significant alterations in serum omentin-1, sclerostin, and GDF11 levels among postmenopausal women as compared to premenopausal group and were suggested the possible role of these adipokines in bone

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metabolism. Moreover, Inosteoporotic postmenopausal, estrogens deficiency enhance bone resorption. So, assessment of these adipokines levels could be beneficial in early detection and prevention of its unfavorable consequences of osteoporosis.

Ethical Considerations:

This study was approved by the Ethics Committee of University of Baghdad/ College of Education for Pure Science (Ibn Al-Haitham)/ Department of Chemistry (approval number: 5863 at 16/12/2019).

Conflicts of interest:

There are no conflicts of interest.

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