Nestatin-1 Level as Potential Parameters in T2DM with and Without Osteoporosis

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Article Info	ABSTRACT
Article history:	In osteoporosis (OP) disease, the strength of bones is decreased, and

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Keywords:

Nestatin-1 Level, T2DM, Osteoporosis, Vitamin D3, Lipid profile it is mostly correlated with the loss of bone tissues and destruction of bone micro structure, making the patients highly vulnerable to bone fracture and markedly decreases their life quality. This study aimed to assess the nestatin-1 level and other biochemical parameters in Iraqi t2dm with and without osteoporosis. This study includes 90 volunteers; their information and history were divided to three groups with T2D with osteoporosis and without osteoporosis and control was taken by questionnaire. A highly significant elevation in the levels of FBS, HbA1C, TC, TG, LDL-C and vitamin D3 was found among study groups (T2D with osteoporosis, T2D without osteoporosis and controls), while HDL-C was highly significantly decreased among study groups (T2D with osteoporosis, T2D without osteoporosis and control). Also we have shown that there was a significant increase of calcium, phosphorus and alkaline phosphates among study groups (T2D with osteoporosis, T2D without osteoporosis and control). A highly significant decreased of Nestatin-1 level among study groups (T2D with osteoporosis, T2D without osteoporosis and the controls). It can be concluded that decreased Nestatin-1 level among T2DM with and without osteoporosis when compared with the controls may be a storing cause to insulin resistance in T2DM patients and may be considered as a biomarker for prediction of osteoporosis risks and as a therapeutic target for osteoporosis treatment.

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1- INTRODUCTION

The most common global disease is Diabetes mellitus (DM) which is still increasing in patient numbers and in significance, since the life style alteration results in low physical activities and obesity increase [1]. Type-2 Diabetes mellitus (T-2DM) is a chronic and progressive disorder characterized by presence of dysfunctional β -cells and increased insulin resistance (IR), defined as the insufficient response of liver, skeletal muscle, and adipose tissue to endogenous insulin secretions and few medications improve IR [2]. In osteoporosis (OP) disease, the strength of bones is decreased, and it is mostly correlated with the loss of bone tissues and destruction of bone micro structure, making the patients highly vulnerable to bone fracture and markedly decreases their life quality [3]. Pain and movement limitation are early clinical symptoms of the disease that may cause fractures and skeletal deformity in complicated cases [4]. The association between OP pathogenesis and type-2 DM is still not clear due to incomplete understanding of DM2 pathogenesis and its influence on the metabolism of bones is different in comparison withtype-1 diabetes (DM1) and could be affected by variable other factors [5]. In accordance with recent investigations, the prevalence rate of diabetic osteoporosis exceeded 50%, however, other studies revealed inconsistent findings regarding the association between patients and bone mineral densities (BMDs), which can be

low, high or normal [6,7]. The complexities of some factors brought a great interference with the study on the association between OP and DM-2 [8]. Nevertheless, only few adipokines were proven to affect the sensitivity of insulin either directly or indirectly. Insulin sensitivity is regulated by adipokine nesfatin-1 [9].

A mechanistic link between T2DM Nestatin-1 and Insulin resistance may be found. Glucose metabolism and phosphorylation of specific signaling proteins are affected by adipokine nestatin-1 through AMP-activated protein kinase, leading to increased sensitivity of liver insulin, all of which assist in hungers and body fat storage regulation [10]. These adipokines are present in brain are as which contribute to metabolic regulations and eating behaviors. Recent studies showed that Nestatin-1 has an anti-hyperglycemic influence on glucose homeostasis. Based on many studies, insulin sensitivity is regulated by nestatin-1 in the brain. In addition, nestatin-1 appeared to stimulate the release of insulin in pancreatic β -cells in hyperglycemia cases, and it is able to cross the blood-brain barrier bidirectional in a non-saturable manner [11]. This study aimed to assess Nestatin-1 levels with other variables in osteoporosis diabetic patients and without diabetic patients.

2- MATERIALS AND METHODS

During the period from November 2023 to February 2024 sample collection included 90 volunteers; whose information and history of T2D with osteoporosis and without osteoporosis were taken by questionnaire forms. The volunteers were divided into three groups, T2D with osteoporosis (n=30, 15 male and 15 female) (group 1) and patients T2D with T2D without osteoporosis (n=30, 15 male and 15 female) (group 2), their ages ranged between (35-60) years, and the healthy controls (group 3), (n=30, 15 male and 15 female). Fasting blood glucose (FBG), glycated hemoglobin (HbA1c), Lipids, calcium, phosphorus, and alkaline phosphate were measured by automated analyzer and vitamin D3 was measured by minividas (biomeruxe/ French). Nestatin-1 level was measured by ELISA kit. Mean values and standard deviations (SD) were used. The significance of the difference between the mean values of the control and cohort groups was compared using the Student T-test. P-Value 0.01 (HS) was considered as a highly significant.

3- RESULTS

Table (1) showed a significant increase in BMI among study groups (T2-D with osteoporosis, T2-D without osteoporosis and the controls), while non-significant appeared in age, weight and high among study groups (T2-D with osteoporosis, T2-D without osteoporosis and controls).

Variables	(G1)n=30	(G2) n=30	(G3)n=30	P Values
Males/Females	1 <mark>5 /15</mark>	15 /15	1 /15	-
Ages (years)	47.36 ±5.42*	47.33 ± 3.95	45.04 ± 5.62	0.835**
Height (m)	166.7± 0.077	166.07 ±0.77	169.17 ±0 .075	0.246
Weight (Kg)	78.72 ± 7.727	79.2 ± 8.5	73.16 ± 7.7	0.061
BMI (kg/m2)	29.20 ± 2.81	27.52 ± 3.43	25.28 ±1.21	0.028*

Data were expressed as (mean \pm SD) *(P \leq 0.05): significant ** (P \leq 0.01): Highly significant

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Table (2) showed a highly significant increased levels of FBS,HbA1C, TC, TG, LDL-C and vitamin D3 among study groups (T2-D with osteoporosis, T2-D without osteoporosis and controls), while highly significant decrease in HDL-C among study groups appeared (T2-D with osteoporosis, T2-D without osteoporosis and controls). Also we showed a significant increase in calcium, phosphorus and alkaline phosphate among the study groups (T2-D with osteoporosis, T2-D without osteoporosis, T2-D without osteoporosis, T2-D without osteoporosis, T2-D without osteoporosis and controls).

Variables	(G1) n=30	(G2)n=30	(G3) n=30	P Values
FBS (mg\dl)	303.54±27.35*	134.46 ± 76.02	86.9 ± 4.52	< 0.001**
HbA1C	10.470 ±1.32	7.44 ± 1.05	5.30 ± 0.03	< 0.001
TC (mg\dL)	325.15±34.1	226.85 ±7.97	180.86± 8.50	< 0.001
TG (mg/dL)	251.62 ±6.53	179.18 ± 4.69	112.32±17.66	< 0.001
HDL-C (mg/dL)	30.23 ± 3.33	35.93 ± 1.66	48.94± 2.92	< 0.01
LDL-C	147.11±34.41	119.71 ± 41.32	91.46 ± 4.23	< 0.001
Vitamin D3 (ng/ml)	8.35±2.25	15.36±2.25	30.25±4.25	< 0.001
Calcium (mg/dL)	5.06±1.25	6.00±2.02	8.75±0.72	< 0.05
Phosphorus(mg/dL)	2.07±0.79	2.19±0.22	4.09±1.12	< 0.05
ALP (mg/dL)	63.65±4.25	76.05±8.21	92.23±8.15	< 0.05

Data were expressed as (mean \pm SD);*(P \leq 0.05): significant;** (P \leq 0.01): Highly significant,G1; T2D with osteoporosis,G2; T2D without osteoporosis,G3;Control.

A highly significant d in Nestatin-1 level was shown among study groups (T2-D with osteoporosis, T2-D without osteoporosis and controls) as shown in table (3).

Variables	(G1) n=30	(G2) n=30	(G3) n=30	P Values
Nestatin (1g\ml)	26.37±14.56*	68.06 ± 15.7	116.25± 30.61	< 0.001**

Table (3): Nestatin-1 levels in the study groups.

Data were expressed as(mean \pm SD)*(P \leq 0.05): significant** (P \leq 0.01):Highly significant.

Table (4): Correlation of serum Nestatin-1 (pg/mL) with clinical and biochemical characteristics in T2-D with osteoporosis and T2-D without osteoporosis.

Variables	T2DM with	T2DM with osteoporosis		out osteoporosis	
	R	Р	R	Р	
Age (years)	-0.083	0.612	-0.020	0.902	
BMI (kg/m2)	-0.093	0.567	-0.055	0.735	
FBS (mg\dl)	0.318*	0.016	0.363*	0.021	
HbA1C%	0.343*	0.031	0.347*	0.051	
TC (mg\dl)	0.332 *	0.052	0.095*	0.035	
TG (mg/dl)	0.394*	0.012	0.341*	0.031	
HDL-C (mg/dl)	- 0.480**	0.002	-0.319*	0.045	
LDL-C	0.312**	0.015	0.343*	0.05	
Vitamin D3 (ng/ml)	0.431**	0.003	0.346*	0.029	
Calcium (mg/dl)	-0.013	0.938	0.113	0.487	
Phosphorus(mg/dl)	0.174	0.283	0.018	0.914	

** Significant correlation at (0.01) level (2-tailed),*Significant correlation at (0.05) level (2-tailed),r = Spearman correlation coefficient, P < 0.05 is significant.

Table (4) showed that there was a positive correlation between Nestatin-1 level and FBS, HbA1C, TC, and TG, also a highly positive correlation was found between nesfatin-1 level and LDL-C and vitamin D3 characteristics in T2-D with osteoporosis and T2-D without osteoporosis, while negative significant correlations were found between nestatin-1 level and HDL-C in T2-D with osteoporosis and T2-D without osteoporosis.



Figure 1: ROC analysis of Nestatin-1in T2DMW



Figure2: ROC analysis of Nestatin-1in T2DMWO

4- DISCUSSION

In diabetic patients, the osteoporosis risk may be highly increased by high blood glucose. Moreover, many pathological mechanisms are shown to be associated with this problem e.g. insulin disorder or deficiency, sexual hormone disturbances, obesity as well as diabetic complication [12, 13]. Osteoporosis can also be affected by adipokines, leading to stimulation of pro-inflammatory mediators synthesis, i.e. matrix metalloproteinases-1, matrix metalloproteinases-13, interleukin-1, interleukin-6, interleukin-8as well as monocyte chemo attractant protein-1 by the OP and osteoblastic line cell. Furthermore, pro-inflammatory mediators i.e. cyclooxygenase-2, interleukin-6, interleukin-8, and macrophage inflammatory protein-1a are stimulated by Nestatin-1 in cases of primary osteoarthritis chondrocyte [14]. However, in our study, a highly significant decrease was found in the level of vitamin D3, which is because all patients have abnormal glucose homeostasis since vitamin D regulation of glucose metabolism via inducing pancreatic B-cells to produce high quantities of insulin for glucose metabolism regulation, however, when this pathway has been due to developed of complicated diabetes as osteoporosis spatially patients were not controlled their diet or were systematically treated in order to study the serum D3 results. Although it is believed that the generation of vitamin D by skin is the main source for the vitamin, other factors like latitude and sun exposures also influence the levels of serum 25 (OH)D. Vitamin D-rich food must be included in the diet, especially oily fishes such as sardine, salmons, mackerels and herrings, which are believed to be the best sources [15]. The findings of Chen, Hailing, Jufen Li and Qian Wang's, (2018) showed a significant correlation between osteoporosis patients and controls [16]. In the present study, a significant increased level of ALP was shown in type 2 DM patients without OP when compared to other groups. Consequently, highly significant correlations were found between the controls and osteoporosis patients. A level of serum calcium is a poor indicator for the total calcium amount. In osteoporosis, our study revealed a distinct and significant association between lean mass (muscle mass) and BMD. The correlation between muscle tissues and bone mineral thickness was studied in osteoporosis cases for all ages, which appeared invariably correlated with the stimulation of less effective muscle tissues with the bones. All ages who had no bone weakening due to reduced mass were neglected (17,18).

According to the above-mentioned information, we can assume that Nestatin-1 with other adipokines contribute to pathological change development in cartilages where the pro-inflammatory factors play significant roles. Rather, these findings indicate a negative effect of Nestatin-1 on the skeletal systems, since pro-inflammatory cytokines contribute to bone turn overs and osteoporosis pathogenesis. They cause increased Op and bone resorption activities, resulting in unfavorable alterations in its properties and structure [19]. When we consider the above findings, the pro-inflammatory factor induction by Nestatin-1 might indicate its contribution in periarticular bone remodeling which accompany osteoarthritis [20, 21]. Akour et al. were the first who explored fasting plasma Nestatin-1 levels in type-2 diabetes cases and revealed that its level was significantly lower in type-2 diabetes patients in comparison with its levels in healthy individuals [22]. Also a study agreed with our results when they found a highly significant

decreased level of Nestatin-1 among their study group [23, 24].There was a negative correlation between serum lipids and Nestatin-1 in diabetic patients. Thus, Nestatin-1 may pose an anti-atherogenic impact on the lipid profile. Such relationships have been recently observed in patients with coronary artery diseases. Other researchers failed to detect any relationship; even in diabetic subjects meanwhile positive correlation of Nestatin-1 with triglyceride in diabetics [25]. Studies on animals showed that Nestatin-1 can regulate the metabolism of lipids. Nestatin-1 can stimulate fatty-acid oxidation via AMP-activated protein kinase activation in diabetic rats. The chronic subcutaneous infusions of Nestatin-1 decreased serum cholesterol and triglyceride and increased the levels of HDL-c in other animal models. The central Nestatin-1 in the brain can lower the lipogenic activities and promote fatty acid oxidations [26].Osteoporosis is a systemic skeletal disease known by low bone masses and deteriorated bone structures [27]. Several studies attempted to assess the relationship between lipid levels and osteoporosis; however, the results were confirmed to be controversial [28]. There was a clinical evidence for the development of OP in elderly T2-DM subjects. Therefore, clinicians must be aware of the significance of these rum lipid levels of the patients and take required interventional actions to control them. In T2-DM patients with dyslipidemia, particularly HDL-C and LDL-C, attention should be paid to the bone health of those patients and initiate drug interventions [29, 30].

5- CONCLUSION:

It can be concluded that decreased Nesfatin-1 level among T2DM with and without osteoporosis when compared with control may be a storing cause to insulin resistance in T2DM patients and may be considered as a biomarker for osteoporosis risk prediction and as therapeutic target for osteoporosis treatment.

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مستوى Nestatin - I كمعلمات محتملة في T2DM مع وبدون هشاشة العظام

الخلاصة

في مرض هشاشة العظام تقل قوة العظام ويرتبط ذلك في الغالب بفقدان أنسجة العظام وتدمير البنية الدقيقة للعظام مما يجعل المرضى أكثر عرضة لكسر عظامهم، ويقلل بشكل ملحوظ من جودة حياتهم.

تهدف هذه الدراسة إلى تقييم مستوى النسفاتين-١ والمعايير الكيميائية الحيوية الأخرى في مرضى السكري العراقيين من النوع الثاني المصابين بهشاشة العظام ومن غير المصابين بها. وتشمل الدراسة ٩٠ متطوعاً؛ تم تقسيم معلوماتهم وتاريخهم إلى ثلاث مجموعات مصابة بمرض السكري من النوع ٢ مع هشاشة العظام وبدون هشاشة العظام، حيث تم جمع هذه المعلومات عن طريق الإستبيان.

تم العثور على ارتفاع كبير في مستويات FBS و HbA1C و TG و TG و LDL-C وڤيتامين D3 بين مجموعات الدراسة T2D) مع هشاشة العظام و T2D بدون هشاشة العظام والضوابط) ، في حين انخفض HDL-C بشكل كبير بين مجموعات الدراسة T2D) مع هشاشة العظام و T2D بدون هشاشة العظام والضوابط).

أظهرت الدراسة زيادة كبيرة في الكالسيوم والفوسفور والفوسفات القلوية بين مجموعات الدراسة T2D) مع هشاشة العظام و T2D بدون هشاشة العظام والضوابط). كما أظهرت إنخفاض كبير في مستوى Nestatin-1 بين مجموعات الدراسة T2D)مع هشاشة العظام و T2D بدون هشاشة العظام والضوابط).

نستنتج من هذه الدراسة أن انخفاض مستوى النيتروجين بين مرضى السكري من النوع ٢ مع أو بدون هشاشة العظام عند مقارنته بالضوابط قد يكون سبباً متوقعا ل<mark>مقاومة الأنسولين لدى مرضى السكري من ا</mark>لنوع ٢ ويمكن اعتباره بمثابة علامة حيوية للتنبؤ بمخاطر هشاشة العظام وهدف علا<mark>جي في العلاج.</mark>