

The Predictive Role of Osteopontin Level in Patients with Type 2 Diabetes Mellitus without Fatty Liver Disease

Doi: <https://doi.org/10.32007/jfacmedbagdad.6612182>

Qassim K. Kadhum ^{1*}  Manal K Rasheed ¹  Khalid AJ AlKazraj ² 

¹ Department of Biochemistry, College of Medicine, University of Baghdad, Baghdad, Iraq

² Baghdad's Teaching Hospital, Medical City, Baghdad, Iraq



This work is licensed under a [Creative Commons Attribution-Noncommercial 4.0 International License](https://creativecommons.org/licenses/by-nc/4.0/)

Abstract

Background: Type 2 diabetes mellitus is a condition in which the body is unable to use insulin effectively. This condition was previously known as non-insulin-dependent or adult-onset diabetes. Osteopontin (OPN) is a phosphorylated glycoprotein initially found in a secreted form in bones. Later, it was discovered that it also exists as an intracellular protein.

Objectives: The study aimed to predict Osteopontin levels in type 2 diabetes mellitus patients without fatty liver disease.

Methods: The research involved 80 participants from Iraq, aged between 45 to 73 years old. Out of these participants, 45 were males, and 35 were females. Group A included 40 patients with Type 2 Diabetes Mellitus without nonalcoholic fatty liver, while group B consisted of 40 healthy individuals.

In all participants the following parameters were measured: FBS, HbA1C, BMI, Insulin, HOMA-IR, Lipid profile, and Osteopontin.

Results: The study found that group A had significantly higher levels of FBS, HbA1c, insulin, HOMA-IR, triglycerides, and VLDL compared to group B ($P \leq 0.05$). Additionally, group A had significantly lower levels of HDL than group B. However, there were no significant differences in the levels of cholesterol, LDL, and ALP between the two groups ($P \geq 0.05$). The mean \pm SD levels of Osteopontin were 23.66 for group A (DM with NAFLD), 15.65 for group B (DM without NAFLD), and 5.43 for group C (control group). This indicates a statistically significant difference ($P \leq 0.05$) in the mean \pm SD levels of Osteopontin among the studied groups.

Conclusion: All parameters are increased in patients with type 2 DM without nonalcoholic fatty liver disease compared to the control group. The recommended threshold for Osteopontin in predicting type 2 diabetes mellitus without non-alcoholic fatty acid liver disease is 9.31 ng/ml.

Keywords: Diabetes mellitus II; Fatty acids; Fatty liver disease; Non-alcoholic; Osteopontin.

J Fac Med Baghdad
2024; Vol.66, No. 1
Received: Aug., 2023
Accepted: Dec., 2023
Published: April, 2024

Introduction

Type 2 diabetes mellitus (T2DM), also known as non-insulin-dependent or adult-onset diabetes, is a medical condition marked by the body's incapacity to utilize insulin, according to the World Health Organization (WHO 2019). Nearly ninety-five percent of people with diabetes have type II diabetes. This particular form of diabetes is hereditary and frequently linked to obesity and a sedentary lifestyle (1). According to the American Diabetes Association (ADA), type 2 diabetes is a condition where the insulin hormone fails to effectively stimulate the body's cells. (2). Insulin resistance occurs in the liver, skeletal muscle, and adipose tissues in type 2 diabetes, which has a defect in the pancreatic beta-cells' ability to secrete insulin. (3) Approximately 30 million Americans between the ages of 18 and 65 years have been identified as having type II Diabetes. Type 2 diabetes is more common in the Middle East and North Africa region, where there are a startling 39 million cases, including (4). The emergence of this condition is influenced by several variables, including genetics, genetic predisposition, ethnicity, and unhealthy eating.

patterns, sedentary lifestyle, obesity, and dyslipidemia (5). More than 100 genetic variations have also been linked to type II diabetes. The extracellular matrix contains the protein Osteopontin (OPN), which is phosphorylated and glycosylated. It can be produced by various cell types and is involved in many normal and abnormal processes, including bone remodeling, the growth of new blood vessels, wound healing, and the accumulation of cells that cause inflammation. (6,7) Bone fractures are more common in people who are overweight or have (T2DM), but it is unclear how obesity affects the bone deficiencies brought on by diabetes. (8) More than 100 genetic variations have also been linked to T2DM. "Insulin resistance" describes a decline in the way cells react to insulin, which results in a diminished capacity to lower blood glucose levels. This condition prevents the use of glucose for energy and metabolism in the cells of tissues like muscle, liver, and fat. (9,10) Diabetes, hyperlipidemia, hypertension, and cardiovascular diseases are all known to be caused by obesity and insulin resistance, glycated hemoglobin HbA1c a frequently employed indicator of long-term glycemic control. The morbidity and mortality linked to metabolic syndrome and type 2 diabetes

*Corresponding author: Qassim K. Khalaf
qasem.khalif1209f@comed.uobaghdad.edu.iq

mellitus (T2DM) can get worse. (11) One marker for predicting the metabolic syndrome is the fasting triglycerides-glucose index (T.G. index). (12) The purpose of this study is to evaluate the clinical relevance of Osteopontin levels in patients with type 2 diabetes mellitus and without nonalcoholic fatty liver disease. (13)

Subjects, Materials and Methods

A case-control study which included (80) Iraqi subjects with age ranged from (45-73) years ((45)male, and(35) female), (40) patients Diabetes mellitus type 2 without fatty liver group A, and (40) healthy control group B. Samples were obtained from Baghdad Teaching Hospital in Medical City/ Baghdad-Iraq, from October 2022 to February 2023. The permission to do the research was obtained from the Department of Biochemistry/ College of Medicine University of Baghdad, Baghdad Teaching Hospital and Al Zahraa Teaching Hospital in Wassitt Governorate each participant, about 5ml of blood samples were obtained from the veins of subjects (control and patients) after fasting (8-12 hours). Each blood sample was divided into two parts.

A - The first part is 2 ml of whole blood retained in EDTA tubes for measuring glycated hemoglobin (HbA1C) by using NYCOCARD™ reader II.

B- In the second step, 3 ml of blood was separated by spinning it in a centrifuge at 3000 rpm for 10 minutes. The resulting liquid was drawn out and then divided into two smaller portions in Eppendorf tubes. These portions were immediately tested for FBS (fasting blood sugar) and lipid profile using an automated system called Abbott Architect 4000.

Measurements of Osteopontin and Insulin

Using enzyme-linked immune sorbent assay (ELISA) Then insulin resistance (IR) values for each sample have been calculated by equation and measure of BMI. The formula for the HOMA model is:

$$\text{HOMA-IR} = \frac{[\text{fasting insulin } (\mu\text{U/ml})] \times [\text{fasting glucose}(\text{mmol/L})]}{22.5} \text{ OR } \text{HOMA-IR} = \frac{[\text{fasting insulin } (\mu\text{U/ml})] \times [\text{fasting glucose}(\text{mg/ml})]}{405} \text{ (14).}$$

Statistical analysis:

The SPSS version 25 software was used to analyze the data. The results were presented as the average, standard deviation, and ranges. Pearson correlation was used to determine the correlation between two quantitative variables, with a t-test to assess the significance if $p \leq 0.05$. The ability of Osteopontin levels to predict type 2 diabetes without non-alcoholic fatty acid liver disease was evaluated using Receiver Operating Characteristic analysis (ROC). The cutoff values were determined using the Youden index. Specificity, sensitivity, negative predictive value, and positive predictive value were calculated. The diagnostic accuracy of Osteopontin levels was also evaluated based on the area under the curve. A correlation coefficient value (r) less than 0.3

indicated no correlation, 0.3-0.5 indicated weak correlation, 0.5-0.7 indicated moderate strength, and above 0.7 indicated strong correlation. A p-value below 0.05 was considered significant.

Results:

There was no significant statistical difference ($P \geq 0.05$) observed between the two groups (group A consisting of patients with type 2 Diabetes mellitus without nonalcoholic fatty liver and group B consisting of healthy controls) in terms of age, gender, and BMI, as shown in (Table 1).

Table 1: Comparison of Age, Gender, and BMI “between” the Study Group

	Group A n= 40	Control Group B n= 40	P-Value
35 – 44	10 (25.0)	11 (27.5)	0.734
45 – 54	11 (27.5)	21 (52.5)	
≥ 55	19 (47.5)	8 (20.0)	
Gender			
Male	24 (60.0)	21 (52.5)	0.640
Female	16 (40.0)	19 (47.5)	
BMI			
Normal	8 (20.0)	11(27.5)	0.636
Overweight	8 (20.0)	11(27.5)	
Obese	18 (45.0)	20 (50.0)	
Normal	14 (35.0)	9 (22.5)	

Non a Significant difference between the two independent means using Student-test at 0.05 level ($p \geq 0.05$).

Comparison of Biochemical parameters between group A and group B

In a comparison of biochemical parameters between the group A and control group (B), the mean levels of FBS, HbA1c, insulin, HOMA-IR, triglycerides, and VLDL were significantly higher ($P \leq 0.05$) in the group A than the controls group B. The mean level of HDL was significantly lower in group A than in the controls group B. No significant difference ($P \geq 0.05$) was found in the mean levels of cholesterol and LDL between the two groups (Table 2).

Table2 Comparison in mean levels of biochemical parameters between study groups .

Parameters	Group A Mean ± SD	Control Group B Mean ± SD	P-value
FBS (mg/dl)	146.1 ± 22.4	89.4 ± 7.54	0.001
HbA1c (mmol/mol)	8.07 ± 1.28	5.03 ± 0.48	0.001
Insulin (ng/ml)	23.58 ± 2.01	9.68 ± 3.80	0.001
HOMA-IR	8.58 ± 1.57	1.79 ± 0.31	0.001
Cholesterol (mg/dl)	154.6 ± 38.3	155.8 ± 34.9	0.107
Triglyceride (mg/dl)	183.7 ± 61.4	118.5 ± 31.7	0.001
HDL (mg/dl)	42.28 ± 9.89	47.93 ± 9.72	0.001
LDL (mg/dl)	82.64 ± 32.65	75.57 ± 30.82	0.323
VLDL (mg/dl)	36.74 ± 12.28	23.38 ± 6.27	0.001

* Significant difference between two independent means using Student-test at 0.05 level, $p \leq 0.05$

Osteopontin level:

This study found a statistically significant difference ($P \leq 0.05$) in the mean level of Osteopontin between the studied groups. Osteopontin of group A higher than group B because group A diagnostic Diabetes Mellitus type2 without non-alcoholic fatty livers disease, and OPN Markers to DMT2 and OPN are significantly increased in (DMT2) (Table

Table 3: Comparison of Osteopontin level between the study groups:

Variable	Study Groups		P Value
	Group A Mean ± SD	Control Group Mean ± SD	
Osteopontin (ng/ml)	15.65 ± 4.93	5.43 ± 1.67	0.001

*Significant difference between two independent means using Student-test at 0.05 levels, $p \leq 0.05$

Post hoc tests (LSD) were run to confirm the differences in the mean Osteopontin level between the studied groups. Group A and group B had a significantly higher Osteopontin levels when compared with control group (15.65 ng/ml and 5.43 ng/ml, $P \leq 0.001$) (Table 4).

Cut-off value of Osteopontin

A ROC curve analysis was conducted to determine the effectiveness of Osteopontin levels in diagnosing type 2 diabetes mellitus without non-alcoholic fatty liver disease. The study identified the optimal Osteopontin value that can be used as a cut-off point for predicting this condition. If the level of Osteopontin is greater than 9.31 ng/ml, it is an indicator of type 2 diabetes mellitus without non-alcoholic fatty acid liver disease. This is supported by a large area under the curve ($AUC=99.7\%$), suggesting a significant association between higher levels of Osteopontin and the presence of type 2 diabetes mellitus without non-alcoholic fatty liver disease. This cut-off value has a sensitivity of 95% and specificity of 100%, with an accuracy of 97.5%. The positive predictive value of Osteopontin is 100%, and the negative predictive value is 95.2% (Figure 1) and (Table 4).

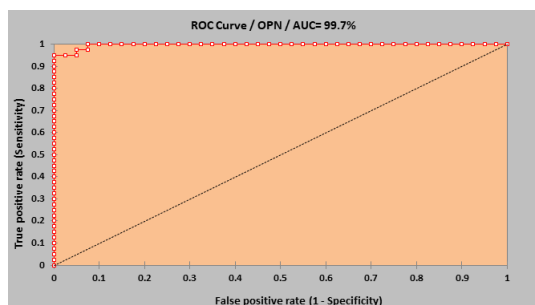


Figure 1: ROC curve of Osteopontin in the diagnosis of type 2 diabetes mellitus without non-alcoholic fatty acid liver disease.

Table 4: Diagnostic accuracy of Osteopontin levels in prediction of type 2 diabetes mellitus without non-alcoholic fatty acid liver disease.

Clinical Parameter	Cut-off value	SN %	SP %	PPV %	NPV %	Accuracy
Osteopontin (ng/ml)	9.31	95 %	100 %	100 %	95.2 %	97.9%

Correlation between Osteopontin level and biochemical parameters of study groups

In the Pearson correlation analysis, there was a significant positive correlation between Osteopontin levels and BMI ($r= 0.397, P \leq 0.001$), FBS ($r= 0.701, P \leq 0.001$), HbA1c ($r= 0.679, P \leq 0.001$), insulin ($r= 0.675, P \leq 0.001$), HOMA-IR ($r= 0.784, P \leq 0.001$), triglycerides ($r= 0.580, P \leq 0.001$), and VLDL ($r= 0.588, P \leq 0.001$). On the other hand, Osteopontin level was negatively correlated with HDL ($r= -0.578, P \leq 0.001$), while it was not significantly correlated ($P \geq 0.05$) with cholesterol, ALP and LDL (Table 5).

Table 5: Correlation of Osteopontin levels with biochemical parameters.

Parameters	Osteopontin (ng/ml)	
	r	P - Value*
BMI (kg/m ²)	0.397	0.001
FBS (mg/dl)	0.701	0.001
HbA1c (mmol/mol)	0.679	0.001
Insulin (ng/ml)	0.675	0.001
HOMA-IR	0.784	0.001
Cholesterol (mg/dl)	0.096	0.297
Triglyceride (mg/dl)	0.580	0.001
HDL (mg/dl)	- 0.578	0.001
LDL (mg/dl)	0.149	0.105
VLDL (mg/dl)	0.588	0.001

*Correlation is significant at the 0.05 level.

Discussion:

The investigated variables—Osteopontin, insulin, T.G., and HOMA-IR—exhibited a favorable age-related correlation. This suggests that the risk of developing diabetes mellitus rises with age. People who are older have insulin-resistant muscle, fat, and liver cells, which prevents them from absorbing enough sugar. Due to dysfunction in the pancreatic beta cells and resistance to insulin in the organs that the hormone targets, type 2 diabetes is primarily brought on by this resistance. (15) According to this study, there is a significant positive correlation between Osteopontin levels and a number of variables, such as BMI, FBS, HbA1c, insulin, HOMA-IR, triglycerides, and VLDL. This suggests that Osteopontin and inflammation brought on by metabolism may be related. The impact of Osteopontin on IRS-2(16) phosphorylation, as well as its inhibition of the transcription factor Forkhead box O1 and its target genes involved in gluconeogenesis, are some of the mechanisms by which it influences glucose regulation and insulin sensitivity. Furthermore, it has been demonstrated that Osteopontin inhibits hepatic signal transducer and activator of transcription 3. (17) Additionally,

data point to a connection between Osteopontin levels and the buildup of intrahepatic lipids, more specifically liver triglyceride levels. Lastly, (16) The fact that Osteopontin can be found in the extracellular matrix and in secreted forms in bodily fluids like plasma has led to its identification as a potential tumor marker. (18)

An HbA1c test was used to determine the degree of glycemic control. According to ADA recommendations, a level greater than 6.5 denoted uncontrolled diabetes (19).

Insulin's main function is to reduce blood glucose levels. Adipose tissue and muscle can use glucose as their main source of energy thanks to insulin's stimulation of glucose uptake. In the end, this process results in a reduction in the level of glucose in the blood (20).

The equilibrium between the liver's production of glucose and the pancreas' release of insulin is shown by the relationship between glucose and insulin in the body at rest. This idea is based on the hypothesis that the liver and cells form a feedback system. (20) Men had higher levels of insulin resistance than women, according to the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). The study found that men were more physically fit than women, who were classified as morbidly obese. (21) Additionally, while females had more peripheral or subcutaneous adipose tissue, males showed larger quantities of visceral and hepatic adipose tissue. These discrepancies, along with variations in sex hormones and adipokines, are probably responsible for women's greater sensitivity to insulin than men's. 2009's Geer and Shen (22)

People with abnormally high lipid levels have diabetic dyslipidemia. These patients typically have increased levels of small dense LDL particles, decreased levels of high-density lipoprotein cholesterol, and elevated levels of total cholesterol and triglyceride levels. In contrast, low-density lipoprotein cholesterol levels could be slightly elevated or within normal limits. People with type 2 diabetes and pre-diabetes frequently have abnormal lipid levels in their blood.

Additional tests (LSD) were conducted to confirm the difference in average Osteopontin levels between the study groups. Osteopontin levels in Group A were significantly higher than those in the control group (measurements were 15.65 ng/ml and 5.43 ng/ml, respectively), according to the findings, with a p-value of less than or equal to 0.005 (23).

Recommendation

In the future, it will be necessary to use Osteopontin as a biomarker for predicting non-alcoholic fatty liver disease in individuals with type 2 diabetes mellitus.

1. Increasing the patients' sample size for more evaluation of the role of Osteopontin I in disease activity and functional severity

Conclusion:

The levels of several variables, including FBS, HbA1c, insulin, HOMA-IR, cholesterol, triglycerides, LDL-c, and VLDL-c, have increased in people with Type 2 diabetes mellitus who do not have nonalcoholic fatty liver disease when compared to the control group. This implies that these variables may be used as markers for disease diagnosis.

The ideal Osteopontin value for predicting type 2 diabetes mellitus without nonalcoholic fatty liver disease was 9.31 ng/ml. Thus, an Osteopontin level greater than 9.31 ng/mL is an indicator of type 2 diabetes mellitus without nonalcoholic fatty liver disease. There is a positive correlation between Osteopontin, insulin, and insulin resistance with age, which leads to the conclusion that the higher the risk of developing diabetes accompanies advancing age, the older the age, the greater the risk of developing diabetes.

Authors' declaration:

Conflicts of Interest: The authors declare no conflict of interest.

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for republication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee in the Iraqi Ministry of Health, Medical City Department, Baghdad Teaching Hospital in Medicine City, Iraq according to the code number (1223) on (9/ 9/ 2022).

Author contributions:

Study conception & design: (*Manal Kamal Rasheed*). Literature search: (*Qassim K. Kadhum*). Data acquisition: (*Qassim K. Kadhum*). Data analysis & interpretation: (*Qassim K. Kadhum, Manal Kamal Rasheed, Khalid AJ AlKazraj*). Manuscript preparation: (*Qassim K. Kadhum*). Manuscript editing & review: (*Manal Kamal Rasheed, Khalid AJ AlKazraj*)

References:

1. World Health Organization. Classification of diabetes mellitus: WHO; 2019 Available from: <https://www.who.int/publications/i/item/classification-of-diabetesmellitus>
2. Davies MJ, Aroda VR, Collins BS, Gabbay RA, Green J, Maruthur NM, Rosas SE, Del Prato S, Mathieu C, Mingrone G, Rossing P. Management of hyperglycaemia in type 2 diabetes, 2022. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetologia*. 2022;65(12):1925-66. <https://doi.org/10.1007/s00125-022-05787-2>
3. American Diabetes Association. Standards of Medical Care in Diabetes-2021. *Diabetes Care*.

- 2021 Jan; 44(Supplement 1): S1-S2 <https://doi.org/10.2337/dc21-Sint>
3. DeFronzo RA. Banting lecture. From the triumvirate to the ominous Octet. A new paradigm for the treatment of type 2DM Diabetes 2009; 58(4):773-95. <https://doi.org/10.2337/db09-9028>
4. American diabetes association .Classification and diagnosis of diabetes sec2 in standards of medical care in diabetes 2016. Diabetes care 2016; 39(suppl1.1):s13-s22 <https://doi.org/10.2337/dc16-S005>
5. CDC. Centers for disease control and prevention: National Diabetes control and prevention, US: Dept of health and human services 2017.
6. International Diabetes federation. Diabetes Atlas, Eight edition 2017. <https://diabetesatlas.org/>
7. Sangahera DK, Blackett PR. Type2 diabetes genetics beyond GWAS J Diabetes Metab 2012 ;(198). <https://doi.org/10.4172/2155-6156.1000198>.
8. Florez JC. Leveraging genetics to advance type2 DM prevention PLOS medicine 2016; 13(7) e1002102. <https://doi.org/10.1371/journal.pmed.1002102>
9. O'Regan A, Berman JS. Osteopontin: a key cytokine in cell-mediated and granulomatous inflammation. Int J Exp Pathol. 2000; 81:373-390. <https://doi.org/10.1046/j.1365-2613.2000.00163.x>
10. Sodek J, Ganss B, McKee MD. Osteopontin. Crit Rev Oral Biol Med. 2000;11:279-303 <https://doi.org/10.1177/10454411000110030101>
11. Denhardt DT, Noda M, O'Regan AW, Pavlin D, Berman JS. Osteopontin as a means to cope with environmental insults: regulation of inflammation, tissue remodeling, and cell survival. J Clin Invest. 2001;107:1055-1061. <https://doi.org/10.1172/JCI12980>
12. Abed E, Manal K Rasheed, Khalaf G. Hussein. Assessment of Total Procollagen Type 1 Intact N-terminal Propeptide, C-telopeptide of type 1 collagen, Bone Mineral Density and its Relationship to Body Mass Index in Men with Type 2 Diabetes. JFacMedBagdad [Internet]. 2022. 24 ;64(2):81-5. <https://doi.org/10.32007/jfacmedbagdad.6421942>
13. Petersen MC, Shulman GI. Mechanisms of Insulin Action and Insulin Resistance. Physiol Rev. 2018;98(4):2133-2223. <https://doi.org/10.1152/physrev.00063.2017>
14. Bermudez V, Salazar J, Martínez MS, Chávez-Castillo M, Olivar LC, Calvo MJ, Palmar J, Bautista J, Ramos E, Cabrera M, Pachano F, Rojas J. Prevalence and Associated Factors of Insulin Resistance in Adults from Maracaibo City, Venezuela. Adv Prev Med. 2016; <https://doi.org/10.1155/2016/9405105>
15. Al-Shamma ZA, Al-Yassin HD, Hashim HM. Resistin , Insulin resistance and BMI in type 2 diabetes mellitus and healthy subjects. JFacMedBagdad. 2008; 50(3):377-82. <https://doi.org/10.32007/jfacmedbagdad.5031262>.
16. Hamed IK, Abed BA, Rashid NF. Glycated haemoglobin as a dual biomarker Association between HbA1c and dyslipidemia in type 2 diabetic patients. JFacMedBagdad. 2012 54(1):88-92. <https://doi.org/10.32007/jfacmedbagdad.541778>
17. Hameed EK, Al-Ameri LT, Hasan HS, Abdulqahar ZH. The Cut-off Values of Triglycerides-Glucose Index for Metabolic Syndrome Associated with Type 2 Diabetes Mellitus. Baghdad Sci. Jour. 2022; 19(2):0340.- <https://doi.org/10.21123/bsj.2022.19.2.0340>
18. Okita K, Iwahashi H, Kozawa J, Okauchi Y, Funahashi T, Imagawa A, Shimomura I. Homeostasis model assessment of insulin resistance for evaluating insulin sensitivity in patients with type 2 diabetes on insulin therapy. Endocr J. 2013; 60(3):283-90. <https://doi.org/10.1507/endocrj.EJ12-0320>
19. Daryabor G, Atashzar MR, Kabelitz D, Meri S, Kalantar K. The Effects of Type 2 Diabetes Mellitus on Organ Metabolism and the Immune System . Front Immunol. 2020 22; 11:1582 <https://doi.org/10.3389/fimmu.2020.01582>
20. Fouad SA, Mohamed NA, Fawzy MW, Moustafa DA. Plasma osteopontin level in chronic liver disease and hepatocellular carcinoma. Hepatitis monthly. 2015;15(9). <https://doi.org/10.5812/hepatmon.30753>
21. FW, Zeyda M, Gollinger K, Pfau B, Neuhofer A, Weichhart T, Säemann MD, Geyerregger R, Schleder M, Kenner L, Stulnig TM. Neutralization of osteopontin inhibits obesity-induced inflammation and insulin resistance. Diabetes. 2010; 1; 59(4):935-46. <https://doi.org/10.2337/db09-0404>
22. Ramaiah SK, Rittling S. Pathophysiological role of osteopontin in hepatic inflammation, toxicity, and cancer. Toxicological sciences. 2008 May 1; 103(1):4-13. <https://doi.org/10.1093/toxsci/kfm246>
23. American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of medical care in diabetes-2018. Diabetes care. 2018; 41(Supplement 1):S13 <https://doi.org/10.2337/dc18-S002>
24. Newsholme P, Cruzat V, Arfuso F, Keane K. Nutrient regulation of insulin secretion and action. Journal of Endocrinology. 2014 Jun 1; 221(3):R105-20. <https://doi.org/10.1530/JOE-13-06>
25. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, and Turner RC, et al. Homeostasis model assessment: insulin resistance and beta cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia, 1985; 28:412-419. <https://doi.org/10.1007/BF00280883>
26. Geer EB, Shen W. Gender differences in insulin resistance, body composition, and energy balance. Gender medicine. 2009 Jan 1;6:60-75. <https://doi.org/10.1016/j.genm.2009.02.002>.
27. Santos-Gallego CG, Rosenson RS. Role of HDL in those with diabetes. Current cardiology reports. 2014 Sep;16:1-4. <https://doi.org/10.1007/s11886-014-0512-5>

How to Cite this Article

Qassim K. Kadhum, Manal Kamal Rasheed, Khalid AJ AlKazraj. The Predictive Role of Osteopontin Level in Patients with Type 2 Diabetes Mellitus without Fatty Liver Disease. JFacMedBagdad [Internet]. 66(1). Available from: <https://iqjmc.uobaghdad.edu.iq/index.php/19JFacMedBaghdad36/article/view/2182>

الدور التنبؤي لمستوى الأوستيوبوننتين في المرضى المصابين بداء السكري من النوع الثاني غير المصابين بمرض الكبد الدهني

قاسم خلف كاظم¹, منال كامل رشيد¹, خالد الخزرجي
قسم الكيمياء السريرية كلية الطب جامعة بغداد, بغداد, العراق
² مستشفى بغداد التعليمي, مدينة الطب, بغداد, العراق

الخلفية: النوع الثاني من مرض الداء السكري عندما يكون الجسد غير قادرا على استخدام الأنسولين بشكل مناسب. ولقد تمت إليه الإشارة مسبقا على أنه السكري الغير معتمد على الأنسولين او مرض السكري الذي يصيب البالغين. أما بالنسبة للاوستيوبوننتين OPN فهو البروتين الفسفوري والذي تم كشفه أصلا في مكان مخفي في العظم. وفيما بعد تم اكتشاف وجوده كبروتين داخل الخلايا.
الهدف: من الدراسة الحالية هو التحقق من تأثير مستوى الأوستيوبوننتين على مرضى النوع الثاني من الداء السكري المصابين بأمراض دهون الكبد من غير الكحول.

المواد وطريقة البحث: جمعت 80 مشاركا من العراق وتتراوح أعمارهم ما بين 45 و 73 سنة من البالغين. وينقسم عدد المشاركين في هذا البحث إلى مجموعتين تتكون الأولى من الذكور 45 مشاركا والمجموعة الثانية من الإناث 35 مشاركا. المجموعة الأولى أ تتكون من 4 شخصا مصابين بداء السكري الثاني وبدون مرض دهون الكبد من غير الكحول بينما المجموعة الثانية ب تتكون من 40 مشاركا ذوي الصحة الجيدة. وتم إجراء الفحوصات على كافة المشاركين (FB, HbA1C, BMI, Insulin, HOMA-IR, Lipid profile and Osteopontin).
النتائج: وجد ان المجموعة (أ) كانت لديها مستويات عالية جدا لكل من FBS و HbA1c و Insuline و HOMA-IR Triglycerides و VLDL مقارنة مع المجموعة (ب) ($P \leq 0.05$) لكن المجموعة (أ) كانت اقل معنويا بمستويات ال HDL من المجموعة (ب) على الرغم من ذلك لا توجد فروق معنوية في مستويات الكوليسترول و LDLc و ALP بين المجموعتين. ($P \geq 0.05$)
الاستنتاجات: تزداد كافة المعاملات في المريض الذي يحمل مرض الداء السكري الثاني وبدون مرض دهون الكبد من غير الكحول مقارنة بالمجموعة الضابطة. ان المستوى الموصى به للاستيوبوننتين في تَقَع النوع الثاني من مرض الداء السكري وبدون مرض دهون الكبد من غير الكحول هو 9.31 ng/ml.
الكلمات المفتاحية: الاستيوبوننتين, النوع الثاني من مرض الداء السكري بدون مرض دهون الكبد من غير الكحول.