Open Access
Journal of the Faculty of Medicine Baghdad
Print ISSN: 0041-9419, Online-ISSN: 2410-8057
DOI: https://doi.org/10.32007/jfacmedbaghdad.6642350
Research Article

Evaluation of Preptin and Other Biomarkers in Coronary Artery Disease Patients with and without Diabetes Mellitus

Saja T. Yassen*[1], Layla O. Farhan[1]

[1] *Department of Chemistry, College of Science for Women, University of Baghdad, Baghdad, Iraq.*

Received: March, 2024
Revised: July, 2024
Accepted: Aug. 2024
Published: Dec., 2024

Abstract

Background: Preptin is an endocrine peptide with 34 amino acids. Conjugated with insulin, it is produced by β-cells from the pro-insulin-like growth factor 2 E-peptide. However, in addition to insulin, pancreatic hormone (Preptin) is released in response to elevated blood glucose levels. Preptin's primary metabolic effect is to raise insulin synthesis, achieved through both an amplifying mechanism and a triggering route dependent on calcium signaling.

Objectives: To determine the Preptin in patients with coronary artery disease (CAD) with and without Type 2 diabetes mellitus (T2DM).

Methods: One hundred and twenty Iraqi participants between the ages of 40 and 60 years were enrolled (80 patients and 40 age-sex matched controls). The study occurred between August and December 2023 at Ibn Al-Bitar Center for Cardiac Surgery in Baghdad, Iraq. The level of Preptin in patients with CAD with and without T2DM was evaluated. The biochemical tests performed on participants included fasting blood sugar (FBS), total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), and very low-density lipoproteins (VLDL) high-density lipoproteins (HDL), blood urea, serum creatinine, and uric acid. The waist-to-hip ratio (WHR) and body mass index (BMI) were also computed. There was a significance level below 0.05 using the Mann-Whitney tests. A non-parametric method and Spearman's rank coefficient were used to determine the significance of correlation for the relationship between the two numerical variables. We determined the Preptin cut-off value by analyzing the receiver operation characteristic (ROC) curve.

Results: The CAD cases both with and without T2DM had a significantly higher serum Preptin than the control group. The levels of Preptin, HDL, and uric acid were significantly strongly correlated. The Preptin ROC curve showed a clear cut-off value (>601.71, >818.10, and >694.71) with the area under the curve (AUA) (0.973, 0.996, and 0.985) respectively when calculated in three groups: CAD without T2DM, CAD with T2DM, and both CAD groups together compared with the controls.

Conclusion: Preptin may serve as a predictive marker for the progression of declining heart function in people with T2DM. It also works well as a diagnostic tool to distinguish between patients with CAD and those without.

Keywords: Coronary artery disease; Lipid profile; Preptin; T2DM; Uric acid.

Introduction:

Coronary artery disease (CAD) is the cause of high rates of morbidity and mortality associated with cardiovascular diseases and is responsible for about 7 million deaths globally each year (1). An inadequate supply of oxygen and blood to the heart muscle is a hallmark of CAD. The obstruction of coronary arteries causes an imbalance between the supply and demand of oxygen. Plaques that block blood flow in the coronary artery lumen are often the cause of it (2). Numerous interrelated factors impact the pathogenesis of CAD, and its etiology is a very complex process. Thrombosis and stenosis, or mural atheroma, are the two primary causes of CAD when blood clots and stenosis are brought on by thin layers of fibrin and a collection of platelets building up on the lining. Next, regulation causes the intima to thicken. This platelet narrowing and arrangement may be due to factors other than the degradation of the elastic layer. Alternatively, stenosis may result from cholesterol becoming twisted or seeping beyond the endothelium in the barrier made of fibrin and platelets (3, 4). Variations in arterial tension can cause structural damage and degeneration of the artery's elastic layer. With the arteries becoming tiny and inflexible, this encourages the deposition of lipids and other materials that results in the development of mural atheroma, a lipid plaque produced by pro-athermic substance created due to degeneration and structural damage. Lipid plaque, smooth muscle development, and endothelial dysfunction cause the diameters of these blood vessels to narrow, which eventually leads to CAD (3, 5).

T2DM is a metabolic disease with a significant prevalence worldwide. It is primarily caused by a combination of two basic factors: The inability of insulin-sensitive tissues to respond to insulin (insulin resistance) and the defective synthesis of insulin by pancreatic β-cells (6). As a result, irregularities in any of the underlying systems might lead to a

* *Corresponding author:* Saja.Taher2305m@csw.uobaghdad.edu.iq

dysregulation of metabolism, which would then cause T2DM (7). Numerous risk factors, such as age, genetics, stress, hypertension, dyslipidemia, obesity, and inactivity, are linked to T2DM and CAD (8). Moreover, an increase in the incidence of diabetes increases the risk of CAD (9).

Preptin is an endocrine peptide that has 34 amino acids produced by β-cells in tandem with insulin. It is derived from the pro-insulin-like growth factor 2 E-peptide. Preptin is secreted in response to increased blood glucose levels together with insulin (10). Additionally, it can be released by the salivary gland, liver, kidney, and breast tissue, among other organs (11). The main metabolic impact of Preptin is to increase insulin production, which happens via a triggering pathway that depends on calcium signaling in addition to an amplifying mechanism (12).

Premature onset DM, impaired glucose tolerance, polycystic ovarian syndrome, and T2DM have all been positively connected with elevated Preptin levels, according to some studies (13). A previous study indicated that male patients with osteoporosis have been found to have lower bone mineral densities when there is a drop in the amount of circulating Preptin (14). Furthermore, it was shown that the peptide was osteogenic, lowering osteoblast apoptosis via MAP-kinase pathway-related mechanisms. Once endogenous proteases cleave Preptin at phenylalanine, it has a five-minute half-life in vivo (15). The pathophysiological effects of uric acid exerted on the cardiovascular system are responsible for the complex and difficult link between uric acid and CVD. During cardiac ischemia, xanthine oxidase activity affects the synthesis of uric acid by increasing the amount of uric acid through a compensatory rise (16). Increased blood uric acid levels can worsen lipid deposits and endothelial cell damage by increasing platelet aggregation and the release of more vasoactive substances (17). Meanwhile, uric acid precipitates and accumulates as crystals that are phagocytosed by leukocytes in the blood vessels, subcutaneous regions, joints, kidneys, and other tissue, causing damage to the heart and blood vessel intima (18).

The current study set out to measure the levels of Preptin, lipid profile (cholesterol, Triglycerides TG, High density lipoproteins HDL, low-density lipoproteins LDL, very low-density lipoproteins VLDL), fasting blood glucose (FBG), urea, creatinine, and uric acid in the sera of a group of Iraqi patients with CAD (with and without T2DM) and their controls.

Patients and Methods:

Case-control study an assessment of Preptin and its levels in CAD with and without T2DM patients was conducted. One hundred and twenty people, aged from 40 to 60 years participated in the study between August and December 2023. Eighty patients from Ibn Al-Bitar Center for Cardiac Surgery in Baghdad, Iraq, were compared to 40 healthy controls (matched for age and sex). Waist/hip ratio (WHR) and body mass index (BMI) were calculated for each participant.

Exclusion criteria included thyroid illness, osteoporosis, cancer, and polycystic ovaries (in women) for both the cases and the controls.

Laboratory testing included renal function, lipid profile, and fasting blood glucose. To quantify Preptin in the serum, Elabscience-USA provided an ELISA kit. With the use of the Kenza (240TX) Biolabo equipment and kit, biochemical markers such as FBS, TC, TG, HDL, urea, creatinine, and uric acid were analyzed.

Venipuncture was used to obtain 10 milliliters of blood, which was then put into a gel tube to separate the serum. The blood samples were centrifuged at 3000 revolutions / second to obtain the serum. Five aliquots of the serum were separated and stored at -20° C until testing.

Statistical Analysis:

The median, 25^{th,} and 75th percentiles were used to describe the study groups and to compare them. The justification for using these statistics is that the numerical variables were not normally distributed. The Mann-Whitney test was used for the analysis, with the level of significance being less than 0.05. The correlation between two numerical variables was ascertained by using the non-parametric approach and Spearman's rank coefficient. The receiver operation characteristic (ROC) curve was analyzed to ascertain the Preptin cut-off value.

Results:

The demographic characteristics and clinical features are shown in Table 1. The median age for CAD without T2DM, CAD with T2DM, and the controls were not significantly different (P>0.05). The median BMI and WHR were significantly different between the three groups.

Table (1): Medians and percentiles for demographic and anthropometric characteristics of the three study groups

Variable	CAD with T2DM	CAD without T2DM	Control	*P*-value
Age (year)	50.0 (57.0 - 46.0)	53.0 (56.0 - 48.0)	50.0 (52.0 - 43.	N.S
BMI (kg/m2)	32.0 (37.0 - 27.0) a	30.0 (35.0 - 26.0) b	25.0 (26.0 - 24.0)	0.00
WHR	0.93 (1.0 - 0.9) a	1.0 (1.0 - 0.92) b	0.82 (0.83 - 0.81)	0.00

The Mann-Whitney test was used to test the difference between two independent medians
a) CAD without T2DM Group and controls
b) CAD with T2DM Group and Controls.

*WHR: Waist to hip ratio.

Table 2 shows the median blood levels in the three study groups of FBG, cholesterol, TG, HDL, LDL, and VLDL. It shows a significantly higher level of FBG and lipid profile (cholesterol, TG, LDL, and VLDL) in the two CAD patient groups compared to the control group (p<0.001). It also shows a significantly lower median HDL in the two CAD patient groups compared to the control group (p<0.001).

Table (2): Medians and percentiles for the serum glucose and lipids of the three study groups

Variable	CAD without T2DM	CAD with T2DM	Control	*p*-value
FBG (mg /dL)	96.0 (102.0 - 86.0)a	189.0 (214.0 - 169.0)b	90.0 (94.0 - 80.0)	0.00
Cholesterol (mg/dL)	246.0 (271.0-226.0) a	151.0 (199.0 - 121.0) b	131.0 (150.0 -111.0)	0.00
TG (mg/dL)	247.0 (273.0 - 220.0) a	201.0 (242.0 - 157.0) b	90.0 (129.0 -83.0)	0.00
HDL (mg/dL)	32.0 (40.0 - 26.0) a	35.0 (44.0 - 32.0) b	48.0 (49.0 - 46.0)	0.00
LDL (mg/dL)	167.0 (192.0 - 152.0) a	72.0 (44.0 - 119.0)	55.0 (79.0 - 48.0)	0.00
VLDL (mg/dL)	48.0 (54.0 - 44.0) a	39.0 (49.0 - 32.0) b	18.0 (26.0 - 17.00	0.00

The Mann-Whitney test was used to test the difference between two independent medians.
a) CAD without T2DM group and controls.
b) CAD with T2DM group and Controls.

Median blood levels of urea, creatinine, and uric acid for the CAD with T2DM, CAD without T2DM) and control groups are shown in Table 3. Significantly higher values of kidney function tests (urea, creatinine, and uric acid) were seen in the two CAD groups (with and without T2DM) compared to the control group (p<0.001). The CAD with T2DM groups had significantly higher serum Preptin concentrations than those without T2DM and controls (p<0.001).

Table (3): Medians and percentiles for the blood urea, creatinine, uric acid and Preptin of the three study groups

Variable	CAD without T2DM	CAD with T2DM	Control	*p*-value
Urea (mg/ dL)	43.0 (44.0-41.0) a	30.0 (41.0-26.0)	30.0 (35.0-23.0)	0.00
Creatinine (mg/ dL)	1.0 (1.1- 0.9) a	0.9 (1.0 -0.7) b	0.5 (0.6 - 0.4)	0.00
Uric acid (mg/ dL)	6.0 (7.0- 5.0) a	5.0 (6.0 - 4.0) b	4.0 (4.3 - 3.45)	0.00
Preptin (pg/mL)	951.0 (995.0 - 849.0) a	1448.0 (1482.0-988.0) b	459.0 (528.0 - 409.0)	0.00

The Mann-Whitney test was used to test the difference between two independent medians.
a) CAD without T2DM group and controls.
b) CAD with T2DM group and controls.

Table 4 shows non-significant correlation between Preptin and the levels of HDL and uric acid in the control group and CAD without T2DM group, while a significant strong negative correlation was found between Preptin with HDL (r = -0.463**, p<0.01) and a non-significant positive correlation with uric acid (r = 0.349*, p>0.05) in those with CAD with T2DM.

Table (4): Correlation coefficient between Preptin and some studied parameters in the study groups

Parameter and Correlation coefficient		Group		
		CAD without T2DM	CAD With T2DM	Control
HDL (mg/dl)	Correlation coefficient (r)	0.020	-0.463**	0.091
	Sig. (2-tailed)	0.902	0.003	0.578
Uric acid (mg/ dl)	Correlation coefficient (r)	-0.174	0.349*	-0.117
	Sig. (2-tailed)	0.282	0.027	0.471

*Correlation is significant at the 0.05 level (2-tailed)
**Correlation is highly significant at the 0.01 level (2-tailed)

Evaluating the efficacy of serum Preptin concentration in distinguishing CAD with T2DM patients from CAD without T2DM patients and healthy individuals was conducted using ROC curve analysis, Table 5. The effectiveness of blood Preptin levels in distinguishing (CAD without T2DM patients), (CAD with T2DM), and (CAD with T2DM, and CAD without T2DM) compared to the controls was evaluated using the ROC curve analysis. The ROC curve for the (CAD with T2DM) group was much higher than the diagnostic tests, indicating greater validity (high sensitivity 100% and specificity 97.5%). As demonstrated by the area under the ROC curve for the (CAD with T2DM) diagnosis (0.996, p 0.001).

Table (5): Preptin ROC to distinguish between the three groups

Variable	Preptin		
	CAD without T2DM and control	CAD with T2DM and Control	All CAD and control
Area under the curve	0.973	0.996	0.985
p-value	0.001	0.001	0.001
Cutoff value	>601.71	>818.096	694.713
Sensitivity (%)	97.5	100.00	97.5
Specificity (%)	90	97.5	92.5
+ve predictive value	97.5	97.6	97.6
-ve predictive value	97.3	100.00	100.00

Figure 1 shows that Preptin shows the sensitivity of 100 and a specificity of 90.0) p > 0.001)*when* distinguishing between CAD without T2DM and controls.

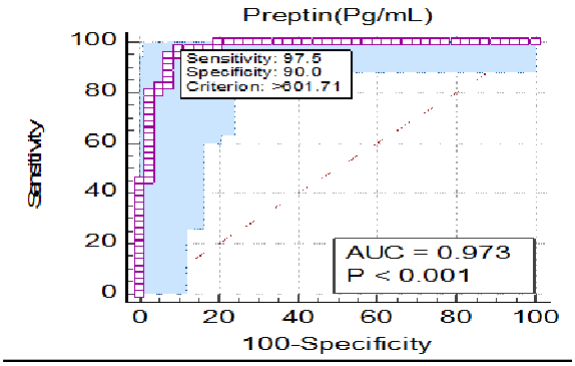

Figure (1): The ROC curve for Preptin distinguishing between CAD withoutT2DM and controls.

Figure 2 demonstrates that Preptin can differentiate between CAD with T2DM and controls with a sensitivity of 100 and a specificity of 90.0, ($p > 0.001$).

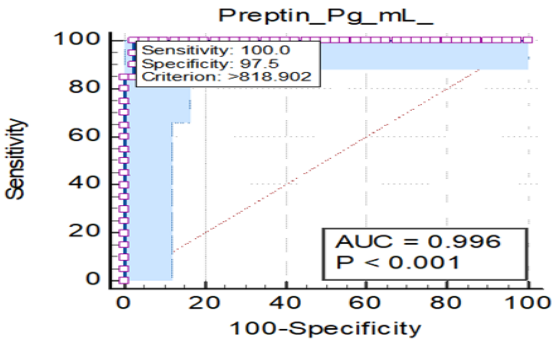

Figure (2): The ROC curve for Preptin distinguishing between CAD withT2DM and controls.

Figure 3 shows that Preptin shows a sensitivity of 97.5 and a specificity of 92.5 when distinguishing between (CAD without T2DM, and CAD with T2DM) and controls.

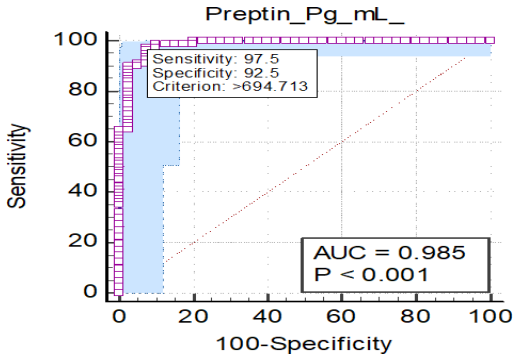

Figure (3): The ROC curve for Preptin distinguishing between all CAD cases and controls.

Discussion:

The finding of the current study that Preptin can be used as a diagnostic marker for CAD patients with T2DM is in line with the findings of Hussein et al who reported that an individual's CAD with T2DM had significantly higher Preptin levels (19). Tahir et al showed that Preptin is essential for regulating the metabolism of sugar. Hence, Preptin levels are elevated in diabetics and heart disease patients, which compromises the control over the metabolism and puts the patient at risk for several additional disease conditions (20). Hassan et al also demonstrated that Preptin levels can predict enhanced pathogenesis of CAD independently and have a significant impact on their advancement. It was linked to atherosclerosis, which is thought to be one of the primary causes of CAD (18). This may be due to Preptin functioning as a physiological amplifier of insulin secretion in response to glucose levels (15). The association between each research group and the following variables was addressed in this study: age, BMI, WHR, lipid profile, urea, creatinine, uric acid, and Preptin. A previous study reported no significant difference in BMI and WHR in Indian patients with CAD when compared to the control (21). The results of the current study that WHR was lower in CAD patients with DM than those without. The two parameters usually rise together with the rise of the prevalence of DM (22), (23). DM is frequently associated with dyslipidemia, which is defined by elevated plasma levels of (TG), (LDL), (TC), and (HDL). Dyslipidemia is a complex condition of lipoprotein metabolism that results from the interplay of hereditary and environmental variables. In individuals diagnosed with T2DM, atherosclerosis and the development of CAD are accelerated (24). Many studies have shown that serum creatinine across all study groups, significantly correlates with the severity of CAD (25).

Limitation: Number of pertinent.

Conclusions:

Preptin may serve as a predictive marker for the progression of declining heart function in people with T2DM. It also works well as a diagnostic tool to distinguish between patients with CAD and those without. In patients CAD with T2DM, the level of (FBG) has risen dramatically. On the other hand, patients' CAD with and without T2DM showed increased TC, TG, LDL, VLDL, Urea, Creatinine, and uric acid levels. HDL levels in both groups were significantly lower compared to the control group.

Authors' declaration:

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 re-publication attached to the manuscript. Authors sign on ethical consideration's Approval-Ethical Clearance: The project was approved by the local ethical committee in (Ibn Al-Bitar Center for Cardiac Surgery in Baghdad, Iraq. According to the code number (4846/22) on (31/ 8/2023)).

Conflict of Insert: None.
Funding: None.

Authors' contributions:
Study conception & design: (Saja Taha& Layla O. Farhan). Literature search: (Saja Taha& Layla O. Farhan). Data acquisition: (Saja Taha& Layla O. Farhan). Data analysis & interpretation: (Saja Taha& Layla O. Farhan). Manuscript preparation: (Saja Taha). Manuscript editing & review: (Saja Taha & Layla O. Farhan).

References:

1. *Wang H, Wang X, Cao Y, Han W, Guo Y, Yang G, et al. Association of polymorphisms of Preptin, irisin and adropin genes with susceptibility to coronary artery disease and hypertension. Medicine (Baltimore). 2020;99(10):1-7.* https://doi.org/10.1097/MD.0000000000019365
2. *Ralapanawa U, Sivakanesan R. Epidemiology and the magnitude of coronary artery disease and acute coronary syndrome: a narrative review. J Epidemiol Glob Health. 2021;11(2):169-77.* https://doi.org/10.2991/jegh.k.201217.001
3. *Ullah M, Wahab A, Khan SU, Zaman U, ur Rehman K, Hamayun S, et al. Stent as a novel technology for coronary artery disease and their clinical manifestation. Curr. Probl Cardiol. 2023;48(1):101415.* https://doi.org/10.1016/j.cpcardiol.2022.101415.
4. *Asada Y, Yamashita A, Sato Y, Hatakeyama K. Pathophysiology of atherothrombosis. Mechanisms of thrombus formation on disrupted atherosclerotic plaques. Pathol. Int. 2020;70(6):309–22.* https://doi.org/10.1111/pin.12921.
5. *Tohirova J, Shernazarov F. Atherosclerosis: causes, symptoms, diagnosis, treatment and prevention. Sci Innov. 2022;1(5):7–12.* https://doi.org/10.5281/zenodo.6988810.
6. *Banday MZ, Sameer AS, Nissar S. Pathophysiology of diabetes: An overview. Avicenna. J.Med. 2020;10(04):174.* https://doi.org/10.4103/ajm.ajm_53_20.
7. *Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, et al. Pathophysiology of type 2 diabetes mellitus. Int. J. Mol. Sci. 2020;21(17):1-34.* https://doi.org/10.3390/ijms21176275
8. *Emad S, Saeed GT. Evaluation of Brain Stem Function in Diabetics with and Without Distal Symmetrical Polyneuropathy Using the Blink Reflex. JFacMedBaghdad. 2023;65(4):318-22.* https://doi.org/10.32007/jfacmedbagdad.1987
9. *Abdulrahman AJ, Jabarah MAH, Najjar SA. Effects of liraglutide on weight control and blood pressure in type 2diabetes mellitus Iraqi patients. JFacMedBaghdad. 2022;64(4):227–32.* https://doi.org/10.32007/jfacmedbagdad.6441971.
10. *Taha BE, Majeed MJ. Estimation of Insulin Resistance in Obese Adults in Baghdad. J Fac Med Baghdad. 2023;65(4):335-40.* https://doi.org/10.32007/jfacmedbagdad.2118.
11. *Perumalsamy S, Huri HZ, Abdullah BM, Mazlan O, Wan Ahmad WA, Vethakkan SRDB. Genetic markers of insulin resistance and atherosclerosis in type 2 diabetes mellitus patients with coronary artery disease. Metabolites. 2023;13(3):1-17.* https://doi.org/10.3390/metabo13030427.
12. *Xu S, Ilyas I, Little PJ, Li H, Kamato D, Zheng X, et al. Endothelial dysfunction in atherosclerotic cardiovascular diseases and beyond: from mechanism to pharmacotherapies. Pharmacol .Rev. 2021;73(3):924–67.* https://doi.org/10.1124/pharmrev.120.000096.
13. *Kałużna M, Pawlaczyk K, Schwermer K, Hoppe K, Yusuf Ibrahim A, Czlapka-Matyasik M, et al. Is Preptin a New Bone Metabolism Parameter in Hemodialysis Patients? Life. 2021;11(4):1-10.* https://doi.org/10.3390/life11040341.
14. *Elsaeed WFM, Abdel Rahman HM, Sasi NG, Zidan HEE. Serum Level of Preptin in Children with Type-1 Diabetes Mellitus and Its Relation to Diabetic Nephropathy. Egypt J Hosp. Med. 2021;84(1):2467–73.* https://doi.org/10.21608/ejhm.2021.184670
15. *Farhan AR, Rasheed MK. Estimation of Preptin Serum level, Insulin Resistance and other biochemical parameters in Pre-diabetic and Newly Diagnosed Type 2 Diabetic Mellitus. HIV Nurs. 2023;23(2):196–201.* https://doi.org/10.31838/hiv23.02.32.
16. *Kashtl GJ, Abed BA, Farhan LO, Noori I, Salman ASD. A Comparative Study to Determine LDH Enzyme Levels in Serum Samples of Women with Breast Cancer and Women with Breast Cancer and Type 2 Diabetes Mellitus. J Med Chem Sci. 2023;6(4):883–90.* https://doi.org/10.26655/JMCHEMSCI.
17. *Abed BA, Al-AAraji SB, Salman IN. Estimation of Galanin hormone in patients with newly thyroid dysfunction in type 2 diabetes mellitus. Biochem Cell Arch. 2021; 21(1): 1317–21.* https://connectjournals.com/03896.2021.21.1317.
18. *Gao Y, Guo Y, Hao W, Meng J, Miao Z, Hou A, et al. Correlation Analysis and Diagnostic Value of Serum Homocysteine, Cystatin C and Uric Acid Levels with the Severity of Coronary Artery Stenosis in Patients with Coronary Heart Disease. Int. J. Gen. Med. 2023; 16(1): 19–31.* https://doi.org/10.2147/ijgm.s411417.2023.8119.
19. *Hussein RJA, Majid A. Evaluation the role of Preptin hormone and some biochemical parameters in Type2 diabetic patients with cardiovascular disease. Univ Thi-Qar J Sci. 2023;10(1):1-4.* https://doi.org/10.32792/utq/utjsci/v10i1.1035.
20. *Tahir NT, Al-Khateeb SMJ, Akram RS. Preptin as a Potential Marker in Iraqi Newly Diagnosed T2DM and T2DM with Cardiovascular Disease. J Contemp Med Sci. 2024; 10 (2):102-105.* https://doi.org/10.22317/jcms.v10i2.1490.
21. *Bhatia R, Bhatia A, Ganatara K. Correlation of body mass index and waist/hip ratio with severity of coronary artery disease. Int J Res Med Sci. 2022; 10(2): 388-92.* https://dx.doi.org/10.18203/2326012.ijrms20220280.
22. *Mohammed NUG, Gorial FI, Khaleel FM, Abed BA, Mutar SA, Farhan LO, et al. Role of Human β-Defensin-3 in Rheumatoid Arthritis: An Observational Single-Center Study. AJMS. 2023; 5(1S):58-62.* https://doi.org/10.54133/ajms.v5ilS.289.

23. Farhan LO, Abed BA, Dawood A. Comparison study between adipsin levels in sera of Iraqi patients with diabetes and neuropathy. Baghdad Sci J. 2023;20(3):726-33.
https://dx.doi.org/10.21123/bsj.2022.7408.
24. Lefta NA, Abed AY, Abed BA. Estimation of asprosin levels in female Iraqi patients with type 2 diabetes and hypothyroidism. J Med Chem Sci. 2023;6(2):433–9.
https://doi.org/10.26655/JMCHEMSCI.2023.2.23.
25. Qadir M, Weli SM. Prevalence of cardiovascular disease risk factors among secondary school pupils in Sulaimani city Kurdistan-Iraq. A cross-sectional study. J Fac Med Baghdad. 2023;65(2):129-34.
https://doi.org/10.32007/jfacmedbagdad.2050

How to Cite this Article
Yassen ST, Farhan LO. Evaluation of Preptin and other Biomarkers in Coronary Artery Disease Patients with and without Diabetes Mellitus. J Fac Med Baghdad. 2024; 66(4). Available from:
https://iqjmc.uobaghdad.edu.iq/index.php/19JFacMedBaghdad36/article/view/2350

تقييم البريبيتين في مرضى إعتلال الشريان التاجي الذين يعانون من مرض السكري وبدونه

سجى طه ياسين ، ليلى عثمان فرحان
فرع الكيمياء، كلية علوم البنات، جامعة بغداد، بغداد، العراق.

الخلاصة:
خلفية البحث: البريبيتين هو ببتيد الغدد الصماء مع 34 من الأحماض الأمينية. مقترنا بالإنسولين، يتم إنتاجه بواسطة خلايا β من عامل النمو الشبيه بالأنسولين E-peptide 2. ومع ذلك، بالإضافة إلى الإنسولين، يتم إفراز هرمون البنكرياس (البريبيتن) إستجابة لإرتفاع مستويات الجلوكوز في الدم. يُمثل التأثير الأيضي الأساسي للبريبيتن في زيادة تخليق الإنسولين، والذي يتحقق من خلال كل من آلية التضخيم وطريق التحفيز الذي يعتمد على إشارات الكالسيوم.
الأهداف: تحديد البريبيتين في المرضى الذين يعانون من إعتلال الشريان التاجي مع وبدون داء السكري من النوع الثاني (T2DM).
المنهجية: تم تقييم مستوى البريبيتن في المرضى الذين يعانون من إعتلال الشريان التاجي مع وبدون داء السكري من النوع الثاني (T2DM). شملت الدراسة 120 مشاركا تتراوح أعمارهم بين 40 و 60 عاما شاركوا بين آب وكانون الأول 2023. تمت المقارنة بأربعين شخصا يتمتعون بصحة جيدة (متطابقين في العمر والجنس) مع ثمانين مريضا عراقيا في مركز ابن البيطار لجراحة القلب في بغداد، العراق. لكل مجموعة بحثية، تم حساب مؤشر كتلة الجسم (BMI) ونسبة الخصر إلى الورك (WHR).
النتائج: وجدت الدراسة أن مجموعة المرضى (إعتلال الشريان التاجي مع وبدون داء السكري من النوع الثاني (T2DM)) لديها ارتفاع كبير للغاية في مصل بريبتين مقارنة بمجموعة السيطرة. كانت مستويات البريبتين والمتغيرين (البروتين الدهني عالي الكثافة (HDL) وحمض اليوريك) مرتبطة إرتباطا ذو دلالة إحصائية عالية. أظهر منحنى ROC للبريبيتين قيمة فاصلة واضحة (<601.71>818.096، و>694.713) مع المساحة الموجودة أسفل المنحنى (0.973، 0.996، و0.985) مع p<0.001 على التوالي عند حسابها في ثلاث مجموعات: إعتلال الشريان التاجي بدون داء السكري من النوع الثاني، إعتلال الشريان التاجي مع داء السكري من النوع الثاني، ومجموعة الاصحاء للمقارنة).
الإستنتاجات: قد يكون البريبتين بمثابة علامة تنبؤية لتطور إنخفاض وظائف القلب لدى الأشخاص المصابين بداء السكري من النوع الثاني. كما أنه يعمل بشكل جيد كأداة تشخيصية للتمييز بين الأشخاص الذين يعانون من إعتلال الشريان التاجي مع وبدون داء السكري من النوع الثاني والاصحاء.
مفتاح الكلمات: إعتلال الشريان التاجي، الدهون، البريبتن، السكري نوع الثاني، حمض اليوريك.