

# Evaluation the role of preptin hormone and some biochemical parameters in Type2 diabetic patients with cardiovascular disease

Redaa Jaafar Abdul Hussein  
Department of Chemistry/ College of  
Science/ University of Thi-Qar,

Thi-Qar, 64001/ Iraq.

[redaa@sci.utq.edu.iq](mailto:redaa@sci.utq.edu.iq)

Alyaa Majid  
Department of Chemistry/ College of  
Science/University of Thi-Qar,

Thi-Qar, 64001/ Iraq.

[aliaa.s\\_mschem@sci.utq.edu.iq](mailto:aliaa.s_mschem@sci.utq.edu.iq)

**Abstract**— T2DM and coronary artery disease (CAD) pose significant threats worldwide, contributing to excessive morbidity and death; these co-morbidities interact synergistically with inflammatory processes. The study's goal was to look at Preptin, Lactate dehydrogenase (LDH), and Fasting Blood Glucose (FBG) levels. Serum Preptin, LDH and FBG levels were determined in 50 patients with acute myocardial infarction, 50 patients with (type 2 diabetes with myocardial infarction) and 50 healthy subjects. The results demonstrate a substantial rise in serum concentrations of (Preptin, LDH, and FBG) in the (T2DM with AMI) group as compared to the (AMI and controls) groups ( $p < 0.05$ ). However, there was no significant difference in serum Preptin and FBG concentrations between the (AMI and controls) groups ( $p < 0.05$ ). There was also no significant difference in serum LDH concentrations between the (AMI with T2DM and AMI) groups ( $p < 0.05$ ). Assessment of Preptin with lactate dehydrogenase and fasting glucose may Predict pancreatic as well as cardiac dysfunction and its helpful in stratification of severity risk.

**Keywords**— Myocardial infarction, Type 2 diabetes, Preptin, Lactate dehydrogenase Fasting Blood glucose.

## I. INTRODUCTION

Diabetes mellitus (DM) has long been recognized as a complex metabolic disorder characterized by hyperglycemia caused by insufficient insulin synthesis, ineffective insulin action, or both. Diabetes is associated with a range of long-term microvascular issues affecting the eyes, kidneys, and nerves, as well as an increased risk of cardiovascular disease (CVD) [1]. Long-term blood glucose increase has serious consequences, including vision loss, renal failure, and nerve damage, which causes abnormal gastrointestinal, urinary, and cardiovascular (CV) system function [2]. Myocardial infarction (MI) is a common sign of cardiovascular illness, and the World Health Organization (WHO) has advised that MI rates be used in epidemiological research as a proxy for cardiovascular disease rates [3,4].

A myocardial infarction (MI) is a heart attack caused by plaque formation in the interior walls of the arteries, resulting in limited blood supply to the heart and injury to heart muscles owing to a lack of oxygen distribution. [5]. heart hypertrophy is a broad phrase that refers to a greater workload and is defined by an increase in heart mass in response to an applied stimulus. Prolonging this process can lead to congestive heart failure [HF], which is described as a progressive phenomenon that develops as the last stage of the majority of cardiac illnesses. Myocardial infarction [MI] is a condition marked by decreased systolic and diastolic function [6]. MI symptoms include chest pain that goes from the left arm to the neck, difficulty of breath, vomiting, abnormal heart beating, and weakness [5]. Early cardiovascular disease, old age, cigarette smoking and high blood levels of specific lipids, diabetes, high blood pressure, lack of physical exercise, obesity, chronic renal disease, excessive alcohol consumption, and cocaine and amphetamine use are all important risk factors [7,8].

Serum LDH was among the first diagnostic biomarker established for myocardial infarction. Subsequently, LDH was also reported as a biomarker for different diseases. Even though LDH is not as effective as cardiac troponin in diagnosing acute myocardial infarction, elevated levels of LDH can be helpful in determining whether a patient has had a myocardial infarction if they come to doctors several days after an episode of chest pain [9]. LDH plays an important role in making your body's energy. It is found in almost all the body's tissues, including those in the blood, heart, kidneys, brain, and lungs. When these tissues are damaged, they release LDH into the bloodstream or other body fluids [10].

Preptin is considered a 34-amino acid peptide hormone consecrated from the  $\beta$  cells of pancreas sideways with insulin. As an endocrine peptide, Preptin is thought to activate the insulin-like growth factor receptor 2 (IGF2R) [11]. Preptin levels in serum are remarkably increased in type 2 diabetes (T2DM), gestational diabetes, and impaired



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

<https://doi.org/10.32792/utq/utjs/v10i1.1035>

glucose tolerance associated polycystic ovary syndrome [12]. The aim of this study is evaluated the Preptin, LDH, and FBG in type 2 diabetic patients with cardiovascular disease.

## II. MATERIALS AND METHODS

This study was conducted at AL- Nasiriyah Heart Center, and AL-Azher Privet hospital. It included (150) subjects, control (50) and patients (100) diagnosed with (Acute Myocardial Infarction and Acute Myocardial Infarction with DM), an age range (40-70). About (5mL) of blood samples of the patients with Acute Myocardial Infarction, acute myocardial infarction (AMI) with DM patients and controls were taken and allowed to clot at room temperature in empty disposable tubes centrifuge to separate it in the centrifuge at 3000 rotor per minute (rpm) for 10min, the serum samples were separated and stored at (-20°C) until analyzed for serum Preptin was determined using ELISA technology by spectrophotometer, serum lactate dehydrogenase and blood glucose were purchased from Biolabo (France). Patients with neuro pathy retinopathy, thyroid dysfunction and liver diseases were excluded from the study.

Table (1): Data of the studied groups

Groups		Sex (M/F)	Age (40-70 years)		
			G1 40-50 years	G2 51-60 years	G3 61-70 years
Patients (100)	AMI with T2DM	(28/22)	33	37	30
	AMI without T2DM	(27/23)			
Controls (50)		(25/25)	18	17	15
BMI (Kg/m <sup>2</sup> )					
Normal weight (18.5-24.99)		Over weight (25-29.99)	Obese (BMI ≥ 30)		
54		46	.....		
30		20	.....		

analysis, and the findings have been presented as (mean SD) with LSD. To compare parameters in various study groups, the one-way ANOVA test was performed. P-values (p<0.05) were used to determine statistical significance.

## III. RESULTS AND DISCUSSION

### A. Preptin and Fasting blood Glucose:

Table (1) shows a significant increase in the concentration of serum Preptin and FBG in (AMI with T2DM) group in comparison with the (AMI and controls) groups (p<0.05). But it was found no significant difference in the concentration of serum preptin between (AMI and controls) groups (p<0.05).

It was also discovered that the concentration of serum FBG in the AMI group was significantly higher than in the control groups (p<0.05).

Table (2): Serum Preptin and FBG levels of control and patient groups

Group	No.	Con. Preptin (pg/ml)	Con.FBG (mg/ml)
A	50	10.65±2.61 <sup>b</sup>	93.80±13.34 <sup>c</sup>
B	50	52.43±8.73 <sup>a</sup>	194.40±24.37 <sup>a</sup>
C	50	12.20±1.81 <sup>b</sup>	103.80 ± 13.99 <sup>b</sup>
LSD		1.78	5.96

\* Each value represents mean SD values with non-identical superscript (a, b or c ...etc.) were considered significantly differences (P ≤ 0.05).

A: Control Group

B: Acute myocardial infarction with type 2 diabetic patients group

C: Acute myocardial infarction without type 2 diabetic patients group.

Insulin resistance plays a key role in the pathophysiology of T2DM, causing pancreatic beta cells to secrete more insulin to compensate for higher blood glucose levels [13].

We looked examined how T2DM affects circulating levels of preptin, a new peptide released by pancreatic beta cells.

When compared to healthy controls, patients with T2DM had considerably greater levels of plasma preptin [14].

Preptin may have a role in the pathogenesis of T2DM via enhance insulin secretion. Preptin concentration was found to be high in patients with T2DM and glucose intolerance, compared to the control group in a previous research [15]. The observation on our study agreement with his fact (preptin concentration was found to be high in patients with T2DM) and with likewise other studies by, who find a link between preptin level and diabetes mellitus a suggests that this peptide may play a role in the pathogenesis of DM, which may be of particular interest to a group DM that constitutes a major public health problem [16].

who discover a relationship between preptin levels and type 2 diabetes This peptide may have a role in the development of diabetes, which may be of special concern to a group of diabetics who are a huge public health problem [12].

### B. Lactate Dehydrogenase:

Table (2) shows a significant increase in the concentration of serum LDH in (T2DM with AMI and AMI) groups in comparison with the controls group (p<0.05). But it was found no significant deference in the concentration of serum LDH between (T2DM with AMI and AMI) groups (p<0.05).

Table (3): Serum Lactate dehydrogenase levels of control and patient groups

Group	No.	LDH (IU/L)
A	50	106.62±16.95 <sup>b</sup>
B	50	170.80±10.77 <sup>a</sup>
C	50	164.82±9.45 <sup>a</sup>
LSD		4.25

The LDH is found in the heart with large quantities which are needed for providing the heart muscles with a high energy that is enough for muscle contraction [17]. However, because blood supply from the heart requires rapid movement (muscles contraction), the LDH comprises five isoenzymes; they differ in structure but operate similarly. The heart muscle contains large levels of the LDH enzyme isoenzymes LD1 and LD2 [9].

Following a myocardial infarction, LDH activities peak at 3-4 days and stay increased for up to 10 days, showing that additional serum. LDH is slowly eliminated in the blood circulation [18]. In AMI, ischemia in the myocardium persists for more than (4-6) hrs, eventually causing death for ischemic myocytes, which convert to necrotic cells that release their contents and enzymes such as (CK and LDH), due to the destruction of cell membrane by three factors formed due to the lower oxygen in cells: (1) internal free radical creation, (2) phospholipase activation, and (3) lysosome enzyme release [19].

#### IV. CONCLUSION

The following results were reached using the information considered during this work. In patients with type 2 diabetes with cardiovascular disease, we discovered that the level of lactate dehydrogenase (LDH) had risen dramatically. On the other hand, patients with diabetes and CVD alone showed increasing Preptin and FBS levels. However, neither the myocardial infarction group nor the control group had an impact on their levels.

#### REFERENCE

- [1] A. M. Egan, & S. F. Dinneen, "What is diabetes?," *Medicine*, Vol. 47, No. 1, P. 1-4, 2019.
- [2] A.J. Ahmad, A Khan, S. Khan, K. Manzoor, "Causes, complication and management of diabetes mellitus," *Chronicle Journal of food and Nutrition*, Vol. 1, No. 1, P.1-4, 2017.
- [3] S. Mendis, K. Thygesen, K. Kuulasmaa, S. Giampaoli, M. Mähönen, B. Ngu Blackett, K, ... & Writing group on behalf of the participating experts of the WHO consultation for revision of WHO definition of myocardial infarction. " World Health Organization definition of myocardial infarction: 2008–09 revision. *International journal of epidemiology*, " Vol. 40, No. 1, P. 139-146, 2011.
- [4] W. Al-Abbas," Evaluation the Role of Interleukin-8 and Interleukin-10 in Myocardial Infarction Patients in Al-Najaf City, " *University of Thi-Qar Journal of Science*, Vol. 7, No. 1, P. 84-88, 2019
- [5] L. Lu, M. Liu, R. Sun, Y. Zheng, & P. Zhang, " Myocardial infarction: symptoms and treatments," *Cell biochemistry and biophysics*, Vol. 72, P. 865-867, 2015.
- [6] A. Majid, & S. A. Sayer, "Protective effect of phenolic extract of *Cyperus rotundus* rhizomes on myocardial infarction induced by isoproterenol in female rats, " In *IOP Conference Series: Materials Science and Engineering*, Vol. 454, No. 1, P. 012005, 2018.
- [7] I. Graham, D. Atar, K. Borch-Johnsen, G. Boysen, G. Burell, R. Cifkova, ... & A. Zampelas, "European guidelines on cardiovascular disease prevention in clinical practice: executive summary," Fourth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (Constituted by representatives of nine societies and by invited experts). *European heart journal*. Vol. 28, No. 19, P. 2375-2414, 2007.
- [8] M. A. Auda, A. Majid, & K. G. Al-Fartosi, "Protective role of polyphenolic compounds extracted from *Cyperus rotundus* rhizomes and taurine on troponin-I and some oxidant/antioxidants parameters of female rats treated with isoproterenol-induced myocardial infarction," *Journal of Thi-Qar University*, Vol. 13, No. 2, P. 61-76, 2018.
- [9] A. H. Wu, F. S. Apple, W. B. Gibler, R. L. Jesse, M. M. Warshaw & Jr. R. Valdes, " National Academy of Clinical Biochemistry Standards of Laboratory Practice: recommendations for the use of cardiac markers in coronary artery diseases." *Clinical chemistry*, Vol. 45, No. 7, P. 1104-1121, 1999.
- [10] J. Ding, J. E. Karp, & A. Emadi, "Elevated lactate dehydrogenase (LDH) can be a marker of immune suppression in cancer: Interplay between hematologic and solid neoplastic clones and their microenvironments," *Cancer Biomarkers*, Vol. 19, No. 4, P. 353-363, 2017.
- [11] R. Wang, A. Xue, W. Zheng, L. Wang, F. Yan, W. Hu, ... & L. He L, "Elevated serum preptin concentrations in patients with diabetic nephropathy, " *Journal of Investigative Medicine*, Vol. 67, No. 7, P. 1048-1052, 2019.
- [12] W. T. Abed, W. E. Jassim, & E. M. Rasheed, "Detection of preptin and insulin like growth factor 2 receptor in the sera of type 2 diabetic patients in Kirkuk city, " Vol. 140, No. 1, P. 1597-1606, 2022.
- [13] J. J. Lee, S. N. Beretvas, & J. H. Freeland-Graves, "Abdominal adiposity distribution in diabetic/prediabetic and nondiabetic populations: a meta-analysis," *Journal of obesity*, Vol. 2014, P. 1-20, 2014.
- [14] M. Kalayci, I. Halifeoglu, H. Kalayci, K. Ugur, & M. Yilmaz, "Plasma preptin levels in patients with type 2 diabetes mellitus," *International Journal of Medical Biochemistry*, Vol. 2, No. 1, P. 1-5, 2019.
- [15] A. Abbas, H. A. Ali, M. M. AL-Rufaie, & R. A. Ali, "Association of serum preptin levels with insulin

resistance in Iraqi women with gestational diabetes mellitus, " *Int Res J Pharm*, Vol. 10, P. 49-55, 2019.

[16] F. Faris Rija, M. I. Sulaiman Musa, & H. T. Hamad, "Some Biochemical Parameters and Level of Preptin in Newly Diagnosed Type 2 Diabetic Women Patients in Tikrit City. *Journal of Chemical Health Risks*. " Vol. 12, No. 2, P. 179-182, 2022.

[17] S. S. Wong, "Strategic utilization of cardiac markers for the diagnosis of acute myocardial infarction. *Annals of Clinical & Laboratory Science*. " Vol. 26, No. 4, P. 301-312, 1996.

[18] Y. Wu, C. Lu, N. Pan, M. Zhang, Y. An, M. Xu, ... & L. Tan, " Serum lactate dehydrogenase activities as systems biomarkers for 48 types of human diseases," *Scientific Reports*, Vol. 11, No. 1, P. 1-8, 2021.

[19] P. R. Puleo, D. Meyer, C. Wathen, C. B. Tawa, S. Wheeler, R. J. Hamburg, ... & R. Roberts, " Use of a rapid assay of subforms of creatine kinase MB to diagnose or rule out acute myocardial infarction, " *New England Journal of Medicine*, Vol. 331, No. 9, P. 561-566, 1994.