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### **ORIGINAL ARTICLE**

## Some Biochemical Parameters and Level of Preptin in Newly Diagnosed Type 2 Diabetic Women Patients in Tikrit City

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	ABSTRACT: A chronic disease categorized by high blood sugar called diabetes mellitus (DM), Its prevalence is
KEYWORDS	rapidly increasing all over the world. Peptin is a peptide produced by cells of pancreas. It plays as a physiologic
D (	enhancer of glucose-mediated insulin secretion. There, the current study aimed to note differences in the levels of
Preptin;	newly diagnosed patients with T2DM controls. This study was directed at Salah el-Deen medical Clinic in Tikrit city
Type 2 DM;	and included 80women from Salah al-Din Hospital in Tikrit in the period since Feb to Jul 2020, 40 newly diagnosed
Diabetes;	patients T2DM and 40 healthy elderly women. Preptin, Hba1c, fasting blood glucose (FBS), blood urea (BU), and
Mellitus;	patients 12DM and 40 heating elderly women. Freptin, Hoard, fasting blood glucose (FBS), blood urea (BO), and
Hba1c	serum creatinine levels will be examined. The present study discovered a significant rise in Preptin (362.460.01pg
	ml <sup>-1</sup> ), Hba1c (8.82.7 %), FBS (205±25.7mg dl <sup>-1</sup> ), BU (20.71 ±4.3 mmol l <sup>-1</sup> ), and creatinine (1.1±0.2mg dl <sup>-1</sup> ) in female
	T2DM patients compared to the control group (222.8±30.7pg ml <sup>-1</sup> , 4.6±1.3 %, 99±15. The observed 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.

#### INTRODUCTION

Diabetes mellitus (DM) is a chronic condition defined by hyperglycemia, and its incidence is quickly growing worldwide. The fundamental cause of the pathophysiology is insulin insufficiency or deficit, or the progress of insulin resistances in the context of sufficient insulin production. Based on International Diabetes Federation estimates, around 425 million diabetic globally in 2017, with this figure expected to rise to 629 million by 2045 [1-3].

Preptin is a 34- amino acid hormone with chemical structure as peptide hormone which consecrated with insulin by pancreatic  $\beta$ -cells [4, 5]. Preptin, is consist of amino acid (a.a) sequence has been mainly preserved over the evolutionary procedure in both mice and humans, resembles to Asp 69 - Leucine 102 of As an endocrinal peptide, preptin is assumed to trigger the

\*Corresponding author: firas\_tucon@tu.edu.iq (F. Faris Rija) DOI: 10.22034/jchr.2022.689290 insulin like growth factor receptor 2 (IGF2R), and by way of a result, brings calcium-dependent insulin secretion in connotation through protein C and phosphorlipase C after the sugar (glucose) level is elevated, In adding, preptin has insulin-like effect on the bone metabolism, like increasing cellular differentiation and the disturbing the function of osteoblasts and osteoclasts, is recovered from secretory granules separated from cultured β-cells with insulin and amylin. Preptin infusion into the isolated and perfused rat pancreas resulted in a considerable rise in the 2<sup>nd</sup> phase, while eliminating preptin from the experimental technique resulted in a decrease in both the 1st and 2nd phases of insulin production [4]. As definite above, initial facts concerning preptin were gotten in animals investigates. The primary medical study counting hominid subjects was showed by Yang *et al.*, who observed Insulin independent Diabetes Mellitus (DM) subjects. It was described in that research which showed the preptin concentrations in DM patient were greater than those in healthy subjects, and that preptin ranks were definitely associated with diastolic blood pressure, Tri Glycerides , total cholesterol, Glycosylated A1c, and HOMA-IR index. The research showed that males had lesser serum preptin concentations than females [6].

Preptin infusion into the isolated perfused rat pancreas stimulates glucose-mediated insulin production, but anti preptin IgG inhibits insulin secretion. Preptin appears to be a physiologic amplifier of glucose-mediated insulin production as a result [7]. Insulin independent diabetes mellitus (T2DM) and polycystic ovarian disease both increase circulation preptin levels [8, 9]. Preptin physiologically controls insulin secretion in response to glucose levels [10]. It is currently regarded to be the fourth active pancreatic hormone crucial in glucose homeostasis (together with amylin, glucagon, and insulin) [10]. The current study aims to explain how preptin hormone concentrations change throughout type 2 diabetes mellitus and to investigate if they play a function or roles in the pathophysiology of the disease.

#### MATERIALS AND METHODS

From February to July 2020, 80 females from Tikrit is Salah al-Din Hospital, comprising 40 newly diagnosed T2DM patients and 40 healthy girls, took part in the study (45-50). T2DM was diagnosed using the American Diabetes Association criteria [1].

Enzyme Linked was used to measure the amounts of preptin in patients' blood samples. Sunlong Biotech Company kits with the sandwich method [11], (RBS) is tested by an enzymatic oxidation technique in the presence of glucose oxidase, blood urea (BU) is assessed by a colorimetric and enzymatic approach (Urease), and serum creatinine is determined by a colorimetric reaction using the Jaffe method. Blood urea (BU) was quantified by a colorimetry and enzymatic approach (Urease), and serum creatinine was assessed using a colorimetric reaction using the Jaffe method.

To compare search groups, the data was analysed statistically using SPSS, utilising a one-way comparison analysis of ANOVA followed by a multi-range Duncan test at a probability (P0.05).

#### RESULTS

Table 1 and Figure 1 demonstrate that there was a significant rise in preptin, Hba1c, FBS, blood urea, and creatinine of serum levels in patients with T2DM related to the control set at the P value (P0.05).

 Table 1. Levels of study parameters including preptin, Hba1c, (FBS), blood urea (BU), and creatinine of serum in women Type 2 Diabetes Millets subjects in compared to healthy subjects.

Groups	No.	Age (Year)	Parameters				
			Preptin (pg ml <sup>-1</sup> )	Hba1c (%)	FBS (mg dl <sup>-1</sup> )	Urea(mmol l <sup>-1</sup> )	Serum creatinin
Control	40	45-50	222.8±30.7	4.6±1.3	99±15.1	12.49±2.6	0.65±0.03
Patients	40	45-50	362.4±60.01	8.8±2.7	205±25.7	20.71±4.3	1.1±0.2
P value			0.002	0.001	0.000	0.001	0.002



Figure 1. Levels of Preptin (pg ml<sup>-1</sup>), Hba1c (%), fasting blood glucose (mg dl<sup>-1</sup>), blood urea (mmol l<sup>-1</sup>), and serum creatinine (mg dl<sup>-1</sup>) concentrations in females with T2DM and the control group.

#### DISCUSSION

The finding of the current research shown that preptin concentration was greater in T2DM subjects; (T2DM) is a hereditary heterogenic syndrome characterized by insulin resistance in the periphery and insulin insufficiency [12-14]. According to Buchanan et al. [12], preptin increased glucose-stimulated insulin production from glucose-stimulated bTC6-F7 cells in а concentration-dependent and storable way. Preptin infusion into an isolated, perfused rat pancreas increased glucose (G)-mediate insulin secretion through thirty %, whereas antipreptin immunoglobulin infusion decreased insulin secretion in the 1st and 2nd phases by ninety nine % and ninety six %, correspondingly [12]. According to these findings, preptin may operate as an amplifier of glucose-mediated insulin production. Preptin levels in plasma from T2DM patients and non-diabetic controls were tested in this study.

Figure 1 and Table 1 reveal that preptin concentration in illness women was significant upper than in control women. Hba1c, fasting blood sugar, blood urea, and serum creatnine concentrations were all significantly higher in T2DM patients than in controls, according to the current study. Increased secretion and/or impaired preptin metabolism may cause elevated preptin levels in T2DM patients. To have a better understanding of this, more research is required. This study has several limitations that should be mentioned. The cross-sectional approach limits our capability to assume a causative association among higher plasma preptin levels and Type 2 Diabetes Millets (T2DM). Our research relied on a only blood preptin measurement, which might not accurately reproduce the association over time. It would be exciting to examine in order variations in preptin concentration in prediabetes and diabetic persons to better understand the role of the hormone in the aetiology of Type 2 Diabetes Millets. Finally, greater preptin concentrations in recently identified T2DM subjects recommended that preptin may performance a role in Type 2 Diabetes Millets causation. Conversely, further study is necessary to fully understand this.

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#### **Conflict of interest**

The authors declare no conflict of interest.

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