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# Adropin, Insulin, Insulin Resistance and Lipid Profile Levels in Diabetes Mellitus Type2 Patients

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**Abstract**

**Background:** Type 2 diabetes mellitus (T2DM) is non-insulin-dependent diabetes or diabetes that affects adults, and it represents 90% of diabetics and it occurs as a result of insulin resistance and a defect in insulin production. We aimed to evaluate levels of adropin, insulin, insulin resistance and lipid profile levels in diabetes mellitus type2 patients.

**Aim:** The current study aimed to investigate the relationship between type 2 diabetes mellitus and liver and adipose tissue by measuring the level of secreted Hepatokines and a number of hormonal and biochemical parameters in men.

**Materials and Methods:** The study group include 60 men with type 2 diabetes mellitus and a control group of 30 healthy men. Proteins and biochemistry assays include Adropin levels, insulin, Fasting blood sugar, Insulin resistance indicators(HOMA-IR,HOMA-B,QUICKI),Total cholesterol, Triglycerides, High-density lipoprotein for cholesterol, Low-density lipoprotein for cholesterol, Very high-density lipoprotein for cholesterol, Phospholipid and Atherogenic index.

**Results:** There was a significant increase at (P≥0.05) in Adropin levels, Fasting blood sugar, Total cholesterol, triglycerides, High-density lipoprotein for cholesterol, Low-density lipoprotein for cholesterol, Very high-density lipoprotein for cholesteroland Phospholipid in type 2 diabetes mellitus men compared to control group. while insulin and HOMA-B show significant decrease at (P≥0.05) in type 2 diabetes mellitus men compared to control group. Atherogenic index, HOMA-IR and QUICKI didn't show any significant difference between both groups.

**Conclusions:** We conclude from the results of the current study that there is a relationship between type 2 diabetes mellitus and liver dysfunctions through a secretion imbalance of Adropin that indicates an association between liver disease and type 2 diabetes mellitus. The metabolic imbalances or risks of insulin resistance can lead to hyperglycemia, dyslipidemia, and obesity.

**Key words: T2DM, Adropin, Insulin hormone, Insulin resistance, Lipid profile.**

## Introduction

Type 2 diabetes is one of the most common metabolic disorders worldwide, and its development is mainly caused by a combination of two main factors: defective insulin secretion by pancreatic cells and the inability of insulin-sensitive tissues to respond to insulin [1]. Insulin resistance has been identified as an impaired biological response to insulin stimulation of target tissues, particularly liver, muscle, and adipose tissue. Insulin resistance impairs glucose disposal, leading to a compensatory increase in insulin production in beta cells and hyperinsulinaemia [2]. The metabolic imbalances or risks of insulin resistance can lead to hyperglycemia, hypertension, dyslipidemia, visceral obesity, and elevated inflammatory markers. The development of insulin resistance can lead to metabolic syndrome, non-alcoholic fatty liver disease (NAFLD), and T2DM [3].Several surrogate indices have been developed that are widely used in measures of insulin resistance in clinical research, including Homeostatic model assessment of insulin resistance(HOMA-IR), , Homeostatic model assessment of β-cell function(HOMA-B), and Quantitative Insulin Sensitivity Index (QUICKI) [4].Dyslipidemia is commonly seen in diabetes. Type 2 DM is one of the most common secondary causes of hyperlipidemia. The relationship between hyperlipidemia and vascular complication of diabetes has long been of interest because both tend to occur with greater frequency in Type 2 DM. Insulin resistance and obesity combine to cause dyslipidemia and hyperglycemia and hyperlipidemia have additive cardiovascular risk[5]. Adropin is a peptide hormone produced by the liver and hypothalamus in the brain, which improves dyslipidemia and hyperglycemia, plays important roles in regulating metabolism and insulin sensitivity [6].

## Materials and Methods

### Samples:

The samples was collected from men with type 2 diabetes mellitus who attended Samarra General Hospital and reviewers of external clinics and laboratories in the city of Samarra. The total members of T2DM menwas 60 as a study group and 30 healthy men were consider as control group.

### Blood collection:

Blood samples was collected from the cuboidal vein. The samples left for 15 min at room temperature. Then centrifuged at 5000 rpm for 10 min. The serum transported to test tubes until examination.

### Hormonal analysis:

The concentrations of Adropin and Insulin hormone were measured via enzyme linked immune sorbent assay (ELISA) by using the commercial kits (ELISA kit, Mybiosource-USA) and procedures was followed as given in the kits catalogs.

### Biochemical analysis:

Fasting blood sugar concentration quantified by followed the procedure that given with kit(Randox-England) insulin resistance was stimulated by using homeostatic model assessment of insulin resistance which equal: HOMA (IR)= fasting insulin (µl/ml)* fasting blood sugar (mg/dl)/450

HOMA-B = 360*Fasting Insulin (µIU/ml) / Fasting blood sugar (mg/dl)-63

QUICKI = $1/[\log(I_0)+\log(G_0)]$ [7].

Total cholesterol, triglycerides and high-density lipoprotein for cholesterol quantified by followed the procedure that given with kit (BIOLABO-France). The concentration of LDL-C in the blood serum of the experimental samples was estimated according to the following equation:

LDL - C concentration (mg/dl) = Total cholesterol – (HDL-C) – VLDL-C [8].

The concentration of (VLDL-C) in the blood serum of the experimental samples was determined according to the following equation:

VLDL-C concentration (mg/dl) = Triglycerides / 5 [9].
Calculate the concentration of phospholipids using the equation:
Phospholipids(mg/dl) = Concentration(Total cholesterol × 0.89)+ 68 [9].
The atherosclerotic content of patients and controls was calculated using the formula:
log TG/HDL –C

**Results:**

The results shows in Table (1) significant increase in Adropin levels, Fasting blood sugar, Total cholesterol, Triglycerides, High-density lipoprotein for cholesterol, Low-density lipoprotein for cholesterol, and Very high-density lipoprotein for cholesterol, Phospholipid. While Insulin and HOMA-B show significant decrease in type 2 diabetes mellitus men compared to control group. Atherogenic index, HOMA-IR and QUICKI didn't show any significant difference between both groups.

**Table(1):Adropin, Insulin, , Insulin resistance indicators, Fasting blood sugar, and lipid profile in T2DM patients and control groups**

| Groups | Control group Mean ± SD | Patients group Mean ± SD |
|---|---|---|
| **Adropin** | **104.43 ±20.34** | **177.66 ±29.71*** |
| **Insulin** | **1.99 ±0.33** | **1.02 ±0.26*** |
| **Fasting blood sugar** | **95.94 ± 6.25** | **229.54 ± 64.28*** |
| **Cholesterol** | **96.04 ± 12.87** | **134.93 ± 23.45*** |
| **Triglycerides** | **122.70 ± 29.36** | **173.84 ± 48.08*** |
| **HDL** | **44.37 ± 10.06** | **55.48 ± 14.42*** |
| **LDL** | **27.13 ± 10.97** | **44.68 ± 17.01*** |
| **VLDL** | **24.68 ± 5.98** | **34.77 ± 9.62*** |
| **Phospholipid** | **153.47 ± 11.46** | **188.08 ± 20.87*** |
| **Atherogenic index** | **0.44 ± 0.17** | **0.49 ± 0.15 N.S** |
| **HOMA-IR** | **0.47 ± 0.08** | **0.57 ± 0.19 N.S** |
| **HOMA-B** | **23.16 ± 8.36** | **2.56 ± 1.17*** |
| **QUICKI** | **0.44 ± 0.01** | **0.43 ± 0.03 N.S** |

- **(*) represents a significant difference**
- **n. s (non-significant) represents the absence of significant difference.**

**Discussion**

High levels of Adropin result from elevated glucose levels or in response to anti-diabetic medications [10]. Adropin is required for metabolic homeostasis and is involved in preventing dyslipidemia as it was negatively associated with triglyceride and low-density lipoprotein levels and was positively associated with high-density lipoprotein [11]. The decrease in Insulin hormone is caused by the failure of pancreatic beta cells and the main pathophysiologic event that contributes to the development of T2DM is the resistance of the target tissues to insulin, which is usually associated with abnormal insulin secretion[12]. High blood glucose results from an imbalance between insulin sensitivity and insulin processing due to the failure of beta cells to secrete insulin [13].

Elevated levels of total cholesterol were closely associated with the risk of T2DMand that cholesterol is strongly influenced by physical activity and food intake[14]. High triglycerides occur because in diabetes, glucose is not used in body tissues due to insulin resistance, which leads to high blood sugar. Therefore, fatty acids in fatty tissues are decomposed to obtain energy, and thus excess fat accumulates in the liver and turns into triglycerides [15]. HDL-Cis significantly higher in patients with a normal BMI and HDL-C can be increased by changing lifestyle factors such as physical activity, diet and smoking cessation [16]. Elevated LDL-C may be due to increased VLDL-C synthesis or impaired clearance of VLDL-C residues, and may be due to a deficiency or defect in the LDL-C receptor [17]. Hepatic VLDL-C production is stimulated by insulin resistance, which is associated with increased free fatty acid influx and thus triglyceride synthesis. This stimulates production of VLDL-C, which is necessary for the transport of these triglycerides[18]. Increased phospholipids have been associated with the risk of T2DM and have been considered as biomarkers of T2DM[19]. In type 2 diabetes, there is a decrease in the mass of pancreatic beta cells, and thus a decrease in their activity, and thus a decrease in the value of HOMA-B, which is an indicator of the activity of pancreatic beta cells [20].

**Conclusion:**

The results of the current study indicated that there is a relationship between type 2 diabetes mellitus and liver dysfunctions through a secretion imbalance of Adropin that indicates an association between liver disease and type 2 diabetesmellitus.The metabolic imbalances or risks of insulin resistance can lead to hyperglycemia, dyslipidemia, and obesity.

**Recommendations:**

1. Study of the relationship between Adropin and obesity.
2.Studying the relationship between insulin resistance and non-alcoholic fatty liver disease.

**Ethical approval:**

The research protocol approved by the Ethical Committee of College of Applied Sciences, Samarra University.

**Conflict of Interest:**

The authors declare that they have no conflict of interest.

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