Evaluation of Adiponectin Level and Other Clinical Variables in Iraqi Adolescence Type 1 Diabetic Patients

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ABSTRACT

Childhood and adolescence are the times when T1DM develops more frequently than adults. This study investigated the relationship between Adiponectin levels **and some clinical variables in Iraqi adolescents with T1DM. The study included 90 subjects. Two groups were created for this study based on the initial mutant cell's lineage.: 45** Adolescence **of Type I diabetes mellitus ranging in age (12-17** years**) and control group 45 Participants in this study were matched for age and sex and appeared healthy. There was a highly** substantial **increase of** rise **weight, heigh, FBS, PPBS, TG, and HOMA-IR in T1DM** adolescents **in** comparison **to groups under control.** There is a **significant increase** in **BMI, TC and insulin level IR in T1DM Adolescence in contrast to groups under control.** There **was an appositive correlation between** Adiponectin **level and weight, BMI, and Insulin level and a highly positive correlation coefficient with FBS. PPBS, TC, TG, AIC, HOMA-IR while negative correlation with LDL in** adolescent **T1DM patients. We conclude that the decrease in adiponectin in patients with type 1 diabetes and adolescent children is an indicator of the appearance of many early diabetes complications in them, and this is considered a risk indicator for many diseases and thus leads to metabolic dysfunction in diabetic patients.**

Keyword: Adeponectin, Adolescence, T1DM, c-peptide, HbA1c, Insulin resistance.

INTRODUCTION

It is known that type 1 diabetes mellitus (T1DM) is an autoimmune illness that results in complete insulin insufficiency by **using** the **immune** system's destruction of the pancreatic cells that produce insulin [1]. A combination of environmental and genetic variables ~~will~~ is believed to determine how the condition develops [2]. Unlike type II diabetes, which is linked to a relative insulin shortage, type 1 diabetes predominantly affects children and adolescents as opposed to adults [3]. Both big and small blood arteries, as well as **and,** eventually, vital organs, may sustain damage from the illness. Heart disease [4], renal and eye problems, and amputations are instances of **long-term** effects that occur while the condition is being treated. According to the Journal of Health Monitoring, T1D can reduce quality of life because of its substantial health implications. Type 1 diabetes in children and adolescents: incidence, prevalence, and treatment [5]. **High-quality care is essential for** those with T1DM to achieve ideal metabolic regulation and well-being in later years, particularly during childhood and adolescence. This is why it is **highly** significant to public health [5]. Adipocytes secrete adiponectin ADP, an endogenous bioactive molecule with a molecular weight of 30k Da. The adiponectin gene encodes 244 amino acids and has two introns and three exons. In adipose tissue, this gene is strongly expressed [6]. **Adiponectin** can increase fatty acid oxidation and carbohydrate utilization, **which** are critical **in** regulating **carbohydrate and lipid metabolism** to lower blood glucose and blood fats [7]**. One of the** leading **players in the link between obesity and insulin resistance is adiponectin, an insulin sensitizer produced from adipose tissue that is a** significant **risk** factor **for type 1 diabetes. Ten years** before **the diagnosis of diabetes, lower** levels of adiponectin were noted [8]. Higher adiponectin level was linked to a lower risk of **diabetes** across **various** demographics. This finding is consistent with a dose-response connection. Furthermore, a growing body of research has established a strong correlation between a high incidence of aberrant glycolipid metabolism and low adiponectin levels [9]. **Lipid** metabolism, glucose, Insulin sensitivity, and cardiovascular homeostasis are all regulated by adiponectin [10]. This study looked into the relationship between adiponectin levels and a few clinical traits in teenagers with Type 1 Diabetes from Iraq.

METHODOLOGY

The study has ninety participants. Two groups were created for this study based on the initial mutant cell's lineage: 45 **Adolescents** of Type I diabetes mellitus ranging in age (12-17 **years**) and a control group of 45 Participants in this study who matched in age and sex and appeared to be in good health The calculation of a subject's body mass index involved dividing their height (m2) by their weight (kg). Mass (kg) / height (m2) equals BMI [11]. Serum fasting glucose, HbA1c, and lipid profile (TC, TG, HDL-C, LDLC) were estimated in auto analyzer cobas. The DRG insulin ELISA kit ([12]) was used to measure **serum insulin amounts**. Homeostasis model of IR (HOMA-IR) = {fasting insulin ,µU/ml * fasting glucose ,mmol/l}/22.5 [13]. Using the Sunlong ELISA kit, serum **adiponectin** concentrations were determined.

Statistical Analysis

Microsoft Office Excel 2010 was the software used to conduct statistical analysis and store the data, expressing means (±standard deviation [SD]) and t-test, at $P<0.05$ significance.

RESULTS

Table (1): There was a significant increase in weight, height, FBS, PPBS, TG, and HOMA-IR in T1DM Adolescence when compared

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with control groups. A **substantial** increase in BMI, TC, and insulin level IR in T1DM **adolescents compared to** control groups. As shown in **Table** (2), there was a significant **decrease** in **adiponectin levels** in T1DM **adolescents** when compared with control groups. As shown in **Table** (3), there was **a positive** correlation between **adiponectin** level and weight, BMI, and Insulin level and a highly positive correlation coefficient with FBS.PPBS, TC, TG, AIC, HOMA-IR while negative correlation with LDL in **adolescent** T1DM patients. **Adiponectin** level increased for girl more than boy as shown in Figure (1).-

Table 1. Mean ±SD of age, BMI, and levels of blood glucose markers, lipid profile, Insulin and HOMA-IR between groups (T1DM Adolescence and control)

P-value	Control	T1DM Adolescence	Parameter
	Mean ±SD	Mean ±SD	
/	45	45	number
0.102	16.45±1.68	17.33 ± 0.942	Age (M/F)
0.01	29.88±15.17	36.74±18.40	Weight (kg)
0.01	126.6±20.56	139.85 ± 11.83	High (m)
0.05	15.02±9.09	19.98±4.41	BMI (kg/m)
0.0001	73.88±10.61	176.97±68.11	FBS (mg/dl)
0.01	4.78±0.31	9.94±2.27	HbA1C (mg/dl)
0.0001	98.57±4.57	213.51 ±75.20	PPBS (mg/dl)
0.05	145.86±21.54	158.25±29.14	TC (mg/dl)
0.0001	93±9.09	139.64±50.97	TG (mg/dl)
0.006	53.11±5.51	52.17±5.24	HDL (mg/dl)
0.05	74.15±22.13	84.11±30.54	LDL (mg/dl)
0.05	13.82±5.25	15.11±5.88	Insulin levels
0.01	2.51±1.05	6.48±2.98	HOMA-IR

N = number; Data are present as mean± SD; NS is no significant; P-value is significant P<0.05, and highly significant P<0.01 and p<0.001.

Table 2. Mean ±SD of adiponectin level between groups (T1DM **adolescent** and control).

P-value		Control	T1DM	Parameter
	T1DM	Mean ±SD	Mean ±SD	
		n = 45	n = 45	
	0.0001	25.17±4.77	11.61±5.02	Adiponectin level (ng/dl)

n=number; Data are present as mean± SD; NS is no significant; P-value is significant P<0.05, and highly significant P<0.01 and p<0.001.

Table 3. Correlation coefficient of adiponectin in (T1DM **adolescent**) with deferent parameters.

Adiponectin level (ng/dl)	Parameter
0.08	Age (M/F)
0.311*	Weight (kg)
0.118	High (m)
0.359*	BMI (kg/m)
0.621**	FBS(mg/dl)
0.502**	HbA1C (mg/dl)
0.611**	PPBS (mg/dl)
0.516**	TC (mg/dl)
0.508**	TG (mg/dl)
0.101	HDL (mg/dl)
-0.315*	LDL (mg/dl)
0.407*	Insulin levels
0.515**	HOMA-IR

**Correlation is significant at the 0.05 level.*
***Correlation is significant at the 0.01 level.*

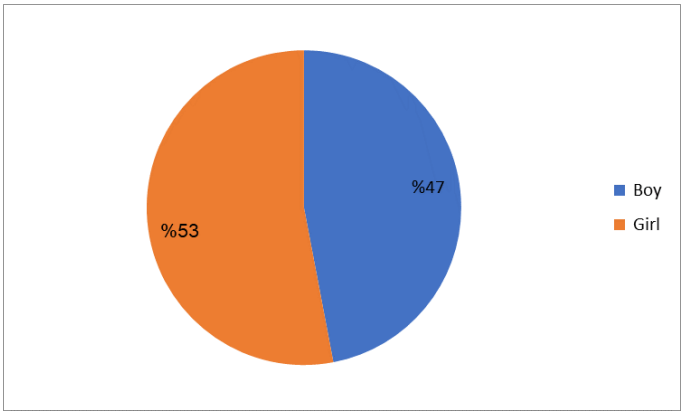

Figure 1. distribution of adiponectin level in both genders.

DISSCUSSION

Adipose tissue is a source of several adipocytokines that may contribute to vascular complications. The pathogenesis of vascular complications in T1D is poorly understood but may involve **chronic low-grade** inflammation [14]. Adiponectin lipid and glucose metabolism and it is suggested to play a role in **preventing** atherosclerosis. Diagnostic usage of adiponectin **has been** a subject of increasing interest in recent years[15],[16] .

Insulin deficiency and muscle and fat cell glucose deprivation are hallmarks of type 1 diabetes. Adiponectin must be positioned within this specific pathophysiological framework as a signal to save and **use** available energy. Since adiponectin increases insulin sensitivity, hyperglycemia and insulin reserve depletion should be the primary causes of hyperadiponectinemia [17]. Adiponectin appears to signal cellular hunger and energy depletion in multiple ways. Adiponectin levels were lower in newly diagnosed T1D patients with detectable c-peptide than in those with undetectable c-peptide and longer-standing diabetes [18]. Regardless of the type of diabetes or body weight, blood glucose decreased adiponectin levels in patients with hyperglycemia in an acute emergency department. This suggests that insulin-induced restoration of energy flow in cells also **reduced** adiponectin levels. Thus, adiponectin should be understood as a warning signal to conserve and use available energy hyperglycemia and relative or absolute insulin insufficiency. It also likely signals the skeleton to minimize energy expenditure 19. Adolescents with T1D are more likely than children to have insulin resistance because sex hormones increase throughout puberty, which counteracts the effects of insulin and aids in the evolution of IR. Adiponectin plasma concentration was negatively correlated with insulin resistance (IR) in adults who were overweight, especially visceral obesity. Additionally, as a marker of IR, metabolic syndrome (MS) was linked to hypoadiponectinemia [19,20] In TIDM children with low levels of adiponectin, metabolic control was poor,. In contrast, in children with normal levels of adiponectin, metabolic control was adequate, as reported by Karamifar et al. 2013 [20]. Adiponectin levels were lower in patients with higher BMIs and **average** in those with lower BMIs. Soliman et al. 2023 [21] demonstrated, **that** serum adiponectin levels and body mass index **correlate positively** among T1D patients. The study, however, showed that serum adiponectin levels **depended** on fat reservation. Low adiponectin levels slowed the **more significant** amounts of fatty acids, and the rate of fatty acid oxidation led to IR [22]. LDL levels and serum adiponectin showed a statistically significant inverse relationship in this study. According to Blaslov et al. (2013) [23], There is a negative correlation between LDL and serum adiponectin levels.

Additionally, children with type 1 diabetes who had low adiponectin levels also had higher LDL levels than children with normal adiponectin levels. Furthermore, it has been demonstrated that adiponectin contributes to the metabolism of lipid profiles. However, no statistically significant correlation **was** discovered between serum adiponectin and low-density lipoprotein in a study carried out by Blaslov et al. (2013) [24], which **found** no statistically meaningful correlation between LDL and serum adiponectin. Adiponectin has been reported to be significantly correlated with ~~measures of~~ insulin resistance (IR) **measures**, such as fasting serum insulin or the homeostasis model assessment of insulin resistance (HOMA-IR), in previous research, including children and adolescents. Begum et al. (2023) [25] discovered that those who satisfied more National Cholesterol Education Program Criteria for the metabolic syndrome had ~~greater~~ more significant increases in fasting insulin and decreases in adiponectin concentrations. Individuals diagnosed with type 1 diabetes have abnormal blood lipid levels over time, which might result in macrovascular dysfunction.

Studies suggest adiponectin may lower VLDL catabolism by increasing **the expression of** skeletal muscle LPL and VLDL receptors, **raising** TG plasma concentrations. Adiponectin increases fatty acid oxidation via AMP kinase activity, which lowers TG accumulation in skeletal muscle. Conversely, HMW **can** enhance TG metabolic activity [26].

The use of nutritional strategies, such as diet and weight loss and the use of healthy eating patterns, to improve adiponectin levels is advised due to the hormone's important role in preventing dyslipidemia, its inverse relationship with the concentration of TC and TG levels, and its positive relationship with HDL cholesterol levels [27]. The majority of studies on the possible relationship between low-density lipoprotein cholesterol LDL-C) and circulating adiponectin have not **been** discovered. One new Small dense LDL is a risk factor for cardiovascular diseases CVD). High TG and low levels of HDL-C have been linked to high LDL levels, which are ~~a~~ typical characteristics of type 2 diabetes and the metabolic syndrome [28,29] .

Smaller, more buoyant particles are less vulnerable to oxidation than larger, less buoyant particles, and oxidative changes in LDL are a precursor to atherosclerosis. **Adiponectin-mediated improvements in TG and HDL may decrease the atherogenic lipoprotein sd-LDL**. [9]. We conclude that adiponectin declines in type 1 diabetic patients and adolescent children are predictive of numerous early diabetes complications. This is because adiponectin is thought to be a risk factor for **multiple** diseases, including metabolic dysfunction in diabetic patients. **that have more significance with distal metastasis and circulating tumor principle.**

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