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Association of Adipokines and Pro-Inflammatory Cytokines with Glycemic Control in Pediatric Type 1 DM

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Abstract

This study aimed to evaluate the association between circulating adipokines, pro-inflammatory cytokines, and glycemic management in children with type 1 diabetes mellitus (T1D).

A case-control study was carried out between April 12, 2025, and February 1, 2026, in pediatric and endocrine clinics in Thi-Qar. The research involved 150 subjects: 100 children diagnosed with TID and 50 healthy controls who were matched based on age and sex. Clinical and demographic details were recorded, and blood samples were obtained after fasting for biochemical testing. Glycemic markers, lipid levels, adipokines (adiponectin, leptin, resistin, visfatin), and inflammatory cytokines (TNF-α, IL-6, IL-1β, CRP) were evaluated using automated analyzers and ELISA methods in regulated laboratory settings.

The findings indicated that HbA1c levels were markedly higher in T1D patients versus controls ($p<0.001$), suggesting inadequate glycemic management. Children with diabetes exhibited significantly elevated fasting glucose levels and negative lipid profiles, characterized by increased cholesterol, triglycerides, and low-density lipoprotein cholesterol (LDL-C), alongside reduced high-density lipoprotein cholesterol (HDL-C). Furthermore, adiponectin levels were significantly reduced in the T1D group ($p<0.001$), whereas proinflammatory markers increased considerably, such as leptin ($p<0.001$) and TNF-α ($p<0.001$). These markers displayed notable positive correlations with HbA1c ($P<0.001$), signifying a strong link between systemic inflammation and glycemic dysregulation.

Type 1 diabetes in children is associated with an imbalance of adipokines and elevated pro-inflammatory cytokines, which correlate with poor glycemic control. Chronic elevated blood sugar may trigger inflammatory pathways and disrupt fat tissue, resulting in metabolic problems and immune system activation that exacerbate disease advancement.

Keywords: Type 1 diabetes; Adipokines; Pro-inflammatory cytokines; Glycemic control; Pediatric diabetes.

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Introduction

Type 1 diabetes mellitus (T1D) is an enduring autoimmune metabolic condition that is defined by the autoimmune destruction of pancreatic β-cells, causing absolute insulin deficiency and chronic hyperglycemia. It is among the most prevalent endocrine diseases among children and adolescents around the globe. Pediatric T1D is becoming a growing global disease, especially in the developing and middle-income states in the last decades. This increasing prevalence is a major issue in public health because of the metabolic comorbidities of chronic hyperglycemia, such as cardiovascular disease, nephropathy, neuropathy, and retinopathy (1,2). Poor glycemic regulation in children in the early phases of the disease has the potential to enhance the development of these complications in adulthood, and therefore it is important to detect metabolic and inflammatory biomarkers early in the disease to monitor and control it (3).

The pathogenesis of T1D is complex, involving a combination of genetic predisposition and environmental precipitation, and immune dysregulation. The disease is mainly diagnosed with the destruction of insulin producing β-cells in the pancreatic islets in a process of autoimmune destruction. This is facilitated by autoreactive T cells and inflammatory cytokines which enhance the progressive apoptosis of the β-cells and loss of insulin secretion (4,5). With the development of insulin deficiency, the intake of glucose by peripheral tissues is impaired leading to continuous hyperglycemia and metabolic disruptions. The most popular biomarker to assess the role of long-term glycemic control in diabetic patients, glycated hemoglobin (HbA1c) is the indicator of average blood glucose levels during the last two to three months (6).

Over the past years, more and more focus has been on the role of inflammatory mediators and adipose tissue-derived hormones in the pathophysiology of diabetes. Adipose tissue is no longer thought of as a passive energy storage organ but it is now being viewed as an active endocrine gland producing a broad assortment of endocrine bioactive molecules referred to as adipokines. These molecules control metabolic homeostasis, immune, and inflammatory signaling pathways. Adiponectin, leptin, Resistin, and Visfatin are among the most widely investigated adipokines that have been implicated in the glucose metabolism and insulin sensitivity (7,8). Adiponectin usually displays the anti-inflammatory and insulin-sensitizing properties, but leptin, Resistin, and Visfatin are usually related to the inflammatory processes and metabolic imbalances (9).

The changes in the secretion of adipokines can be a cause of the formation of metabolic disturbances and inflammation witnessed in diabetic patients. Lower levels of adiponectin have been linked to the inability to regulate glucose and enhanced inflammatory processes. On the contrary, a rise in leptin and Resistin has been associated with the insulin resistance, immune activation, and dysfunction of endothelium (10). These adipokines communicate with immune cells and inflammatory pathways, which makes it possible to consider that in the pathogenesis of diabetes, metabolic and immune processes are closely linked to each other (11). Besides adipokines, pro-inflammatory cytokines also have a central role to play in the autoimmune and inflammatory mechanisms of T1D. Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6)

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and interleukin-1 beta (IL-1β) are the cytokines that have been known to mediate b-cell damage and systemic inflammation. These cytokines may trigger intracellular signaling pathways which favor oxidative stress, apoptosis and immune-mediated pancreatic tissue destruction (12). Moreover, chronic hyperglycemia per se may augment the action of inflammatory responses by producing advanced glycation end products (AGEs) and reactive oxygen species, thus increasing the release of cytokines and metabolic impairment (13).

Adipokines, inflammatory cytokines and glycemic control interaction is a significant field of research in pediatric diabetes. Despite the available studies that have investigated the inflammatory nature of T1D, combined analysis of the adipokines, and cytokines in association with glycemic control has been relatively low, especially in pediatric groups (14).

Knowledge of these relationships could offer useful information on the processes that mediate the association between metabolic dysregulation and immune activation in children with diabetes. Besides, the detection of specific biomarkers related to the inadequate glycemic control can also be used to enhance disease monitoring and formulate specific therapeutic intercessions (15).

Thus, the current research proposal was to explore the relationship between circulating adipokines and pro-inflammatory cytokines and glycemic control among children with type 1 diabetes mellitus. This study aims to offer a clearer understanding of the metabolic-inflammatory interactions of pediatric T1D and their possible clinical implication by comparing the levels of biomarkers between diabetic children and healthy controls and studying their relationships with glycemic indicators.

Material and Methods:

Study design and setting:

The case-control study was carried out to examine the relationship between adipokines, pro-inflammatory cytokines and glycemic control in children with T1D. The study was carried out from April 12, 2025, to February 1, 2026, in the pediatric units and endocrine unit outpatient clinic of the selected hospitals and medical centers in Thi-Qar directorate.

Study population:

The assessment of family history of diabetes was done through parental report and both type 1 and type 2 diabetes mellitus were captured through first-degree relatives. The population to be studied was 150 which comprised of 100 children with T1D and 50 seemingly healthy children as the control population. The control group was chosen compared with the patient group in relation to their age and sex to reduce possible confounding factors. The diagnosis of T1D was established based on the American Diabetes Association (ADA) criteria, including clinical presentation, and the presence of hyperglycemia (fasting plasma glucose ≥126 mg/dL, random glucose ≥200 mg/dL or HbA1c ≥6.5%). All patients were confirmed as insulin-dependent by physicians and required continuous exogenous insulin therapy.

The age of children involved in the study was the pediatric age range with no previous history of other chronic metabolic, autoimmune, and inflammatory diseases. The age of children involved in the study was the pediatric age range with no previous history of other chronic metabolic, autoimmune (other than T1D), and inflammatory diseases.

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Patients who had acute infections, or endocrine disorders other than diabetes, were excluded. Moreover, those test subjects who had received within the previous 4 weeks corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs), lipid-lowering agents or immunomodulatory treatment were also excluded.

To minimize the possibility of diabetes, autoimmune disease, or other long-term medical conditions, healthy controls were screened. The structured questionnaires and medical records were used to gather demographic and clinical information after enrolment.

The documented variables were age, gender, body mass index (BMI), duration of diabetes and insulin therapy dosage. Standard formula was used in calculating the BMI (weight in kilograms/height in meters squared).

Venous blood samples (5 ml) were collected after an 8-hour overnight fast. Samples were centrifuged at 3000 rpm for 10 minutes to isolate serum, which was then stored at (-20C) until analysis.

Glycemic and Lipid Profiles: Fasting blood glucose (FBG), HbA1c, and lipid parameters (Total Cholesterol, Triglycerides, HDL-C, and LDL-C) were measured using enzymatic colorimetric methods with kits from Biolabo (France).

Adipokine Quantification: Serum Adiponectin, Leptin, Resistin, and Visfatin were quantified using Sandwich ELISA kits from Elabscience (China). These kits were selected for their high sensitivity and specificity in pediatric samples.

Inflammatory Markers: High-sensitivity ELISA kits from Elabscience were employed to measure circulating levels of TNF-α, IL-6, and IL-1β. C-Reactive Protein (CRP) was analysed using the hs-CRP immunoturbidimetric assay.

Procedure Standardization: All measurements were performed on automated clinical chemistry analyzers and microplate readers at 450 nm. Assays were conducted in duplicate to minimize intra-assay variation, following the manufacturers' strictly defined procedures.

Statistical Analysis:

Statistical Package of the Social Sciences (SPSS) version 26 was used to analyze the data collected statistically. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were given frequencies and percentages. The Kolmogorov-Smirnov test was employed to assess the normality of the data distribution. For the comparison of mean values between T1D and healthy controls, the independent samples t-test was utilized for normally distributed variables. Pearson correlation coefficient (r) was carried out to determine the relationship between the adipokines, inflammatory cytokines and the indicators of glycemic control-like hemoglobin A1c. Any p-value that was smaller than 0.05 was considered statistically significant for all tests.

Results:

In Table 1, the analysis demonstrated no statistically significant differences between children with T1D and healthy controls regarding age ($p = 0.621$), gender distribution ($p = 0.987$), or body mass index ($p = 0.573$), indicating that the groups were well matched for these baseline variables. The patients having T1D had an average of 4.6 ± 2.3 years of diabetes and the daily dose of insulin was 0.82 ± 0.21 U/kg. There was a substantial difference between T1D patients and controls in family history of diabetes (type 1 and type 2 diabetes mellitus) ($p=0.041$). Moreover, the glycemic control indexes were significantly different between the groups with fairly high levels of HbA1c among diabetic children ($p< 0.001$) indicating poor glycemic control

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Table 1: Sociodemographic and Clinical Characteristics of Pediatric Type 1 Diabetes Patients and Healthy Controls

Variable	T1D Patients (n=100) Mean±SD	Controls (n=50) Mean±SD	P-value
Age (years)	11.2 ± 3.4	10.9 ± 3.1	0.621
Gender (Male/Female)	54 / 46	27 / 23	0.987
BMI (kg/m²)	19.8 ± 3.2	20.1 ± 3.4	0.573
Duration of diabetes (years)	4.6 ± 2.3	—	—
Family history of diabetes (%)	38 (38%)	11 (22%)	**0.041**
Insulin therapy (daily dose U/kg)	0.82 ± 0.21	—	—
HbA1c (%)	9.1 ± 1.8	5.2 ± 0.6	**<0.001**

**Statistical analysis was done using the Mann-Whitney test*

Table 2: Comparison of Glycemic and Lipid Profile Parameters Between Pediatric Type 1 Diabetes Patients and Healthy Controls

Parameter	T1D Patients (n=100) Mean±SD	Controls (n=50) Mean±SD	P-value
Fasting Blood Glucose (mg/dL)	214.6 ± 52.8	88.4 ± 10.7	**<0.001**
HbA1c (%)	9.1 ± 1.8	5.2 ± 0.6	**<0.001**
Total Cholesterol (mg/dL)	185.3 ± 34.7	162.5 ± 29.4	**0.002**
Triglycerides (mg/dL)	148.2 ± 41.6	112.3 ± 30.1	**<0.001**
HDL-C (mg/dL)	42.1 ± 8.7	51.6 ± 9.3	**<0.001**
LDL-C (mg/dL)	109.4 ± 27.8	93.2 ± 22.5	**0.004**

**Statistical analysis was done using the Paired T-test test*
#Statistical analysis was done using the Wilcoxon signed rank test

in pediatric patients with T1D. These results affirm the clinical difference between the two groups and even though they shared similar baseline demographic variables.

In Table 2, the findings revealed significantly high levels of fasting blood glucose in T1D patients as compared to the control group ($p< 0.001$). HbA1c levels differed significantly between diabetic patients and healthy subjects ($p< 0.001$). Lipid profile parameters, total cholesterol concentrations were significantly increased in T1D patients compared with controls ($p= 0.002$). Triglyceride levels were also significantly elevated in the diabetic group ($p< 0.001$). In contrast, HDL-C levels were significantly lower among T1D patients compared with healthy individuals ($p<0.001$). Additionally, LDL-C levels were significantly higher in the diabetic group relative to the control group $p = 0.004$).

The comparison of the adipokines levels is demonstrated in Table 3, which illustrates the circulating levels of adipokine in pediatric patients with type 1 diabetes mellitus (T1D) and healthy controls. It was found that the level of serum adiponectin in T1D patients decreased significantly in comparison with the control group ($p < 0.001$). On the contrary, diabetic children had significantly increased pro-inflammatory and metabolically active adipokines. Serum leptin concentrations were significantly higher in the T1D group than in controls ($p < 0.001$).

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Similarly, Resistin levels were significantly increased among patients with T1D compared with healthy individuals ($p < 0.001$). There was also statistically significant increase in the diabetic group over the control group in the concentration of Visfatin ($p<0.001$).

Table 4 revealed the summary of circulating levels of some of the pro-inflammatory cytokines in pediatric patients with T1D and healthy controls. The results proved that there was a significant increase in the levels of inflammatory components in diabetic children.

Table 3: Serum Levels of Adipokines in Pediatric Type 1 Diabetes Patients and Healthy Controls

Adipokine	T1D Patients (n=100) Mean±SD	Controls (n=50) Mean±SD	P-value
Adiponectin (µg/mL)	7.4 ± 2.3	10.2 ± 2.7	**<0.001**
Leptin (ng/mL)	15.8 ± 5.1	11.4 ± 4.2	**<0.001**
Resistin (ng/mL)	8.6 ± 2.4	6.3 ± 1.9	**<0.001**
Visfatin (ng/mL)	4.9 ± 1.5	3.6 ± 1.2	**<0.001**

**Statistical analysis was done using the Paired T-test test*
#Statistical analysis was done using the Wilcoxon signed rank test

Table 4: Serum Levels of Pro-Inflammatory Cytokines in Pediatric Type 1 Diabetes Patients and Healthy Controls

Cytokine	T1D Patients (n=100) Mean±SD	Controls (n=50) Mean±SD	P-value
TNF-α (pg/mL)	18.6 ± 6.2	10.7 ± 3.5	<0.001
IL-6 (pg/mL)	9.8 ± 3.1	4.5 ± 1.7	<0.001
IL-1β (pg/mL)	6.2 ± 2.0	3.1 ± 1.2	<0.001
CRP (mg/L)	4.9 ± 1.8	1.7 ± 0.9	<0.001

**Statistical analysis was done using the Paired T-test test*
#Statistical analysis was done using the Wilcoxon signed rank test

Table 5. Correlation Between Adipokines, Inflammatory Cytokines, and Glycemic Control (HbA1c) in Pediatric Type 1 Diabetes Patients (n=100)

Biomarker	r value	P-value
Adiponectin	-0.41	<0.001
Leptin	0.36	0.002
Resistin	0.39	<0.001
Visfatin	0.33	0.004
TNF-α	0.45	<0.001
IL-6	0.42	<0.001
IL-1β	0.31	0.006
CRP	0.38	<0.001

**Statistical analysis was done using the Spearman correlation test*

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The results proved that there was a significant increase in the levels of inflammatory components in diabetic children. The level of serum tumor necrosis factor-alpha ($p< 0.001$) was significantly greater in the T1D group as compared to controls. In the same way, the interleukin-6 (IL-6) levels were also highly elevated in type 1 diabetes patients in comparison with healthy persons ($p < 0.001$). The level of interleukin-1 beta (IL-1β) also showed a significant increase in the diabetic population compared to the controls ($p < 0.001$). Besides this, C-reactive protein (CRP), a major systemic inflammatory protein, was also significantly increased in T1D patients and compared to the control group ($p < 0.001$). Together, these findings suggest a strong inflammatory phenotype in pediatric type 1 diabetes, which is in favor of the importance of chronic immune-activation and cytokine-mediated inflammation in the pathophysiology and metabolic dysregulation of the disease.

Table 5 reports the correlation study of adipokines that are in circulation, inflammation cytokines and glycemic control, measured by the level of (HbA1c) specifically in pediatric patients with type 1 diabetes mellitus group (n=100). As shown in the analysis, the correlation between the adiponectin levels and the HbA1c was found to be negative with a significant negative association, showing reduced levels of adiponectin with worse glycemic control ($r = -0.41$, $p < 0.001$). Conversely, there were positive correlations between some adipokines and HbA1c as follows, leptin ($r = 0.36$, $p = 0.002$), Resistin ($r = 0.39$, $p < 0.001$), and Visfatin ($r = 0.33$, $p = 0.004$), which indicate that they may be involved in metabolic dysregulation and insulin resistance in childhood diabetes. In addition, inflammatory cytokines were significantly and positively correlated with HbA1c levels. TNF- was the most correlated ($r = 0.45$, $p < 0.001$) then IL-6 ($r = 0.42$, $p < 0.001$), CRP ($r = 0.38$, $p < 0.001$) and finally, IL-1β ($r = 0.31$, $p = 0.006$).

Discussion:

The authors described the relationship between adipokines and pro-inflammatory cytokines and glycemic control in pediatric patients with T1D in the presented research. The sociodemographic analysis indicated that the diabetic patients and healthy controls do not have statistically significant differences in terms of age, gender distribution, and body mass index (BMI). This similarity between groups increases internal validity of the research because it reduces the potential confounding variables that are linked to demographic and anthropometric dissimilarities. This has been found in other childhood diabetes studies where age and BMI had been closely matched and hence the observed biochemical difference could be attributed to the effect of the disease-induced metabolism and immunological changes rather than demographic bias (16,17).

The present results however reported that the family history of diabetes was significantly more common among T1D patients' group (38%) as compared to controls. The fact is verified by the observation that genetic susceptibility is solid in the development of autoimmune diabetes. In children, genetic predisposition to the 2nd type of diabetes has been known to involve the immune-regulatory genes and our HLA class II alleles that predisposed genetic factors to the pancreatic destruction and immune-activation of gene triggering (18).

The evaluation of glycemic indicators showed the fasting level of blood glucose and HbA1c was significantly higher in diabetic children

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than in healthy individuals. Such results are aligned with the pathophysiology of T1D which is an autoimmune mediated damage of insulin producing β-cells in the pancreatic islets of Langerhans. With the progression of insulin deficiency, the uptake of glucose by peripheral tissues decreases, which induces chronic hyperglycemia and poor glycemic control in the long-term period, as indicated by the increase in the level of HbA1c (19,20).

The same findings were obtained in several studies involving children who showed that HbA1c continues to be the best predictor of persistent glycemic imbalance and metabolic dysregulation in children with T1D (21). Hyperglycemia in these patients may also persist and induce oxidative stress and endothelial dysfunction which leads to systemic inflammatory activation and metabolic complications (22).

The lipid profile results in the current study had shown that there was a great increase in total cholesterol, triglycerides, and LDL-cholesterol in T1D patients, but there was a significant decrease in HDL-cholesterol levels in T1D patients as compared to controls. These changes show the existence of dyslipidemia related to diabetes (23,24). The impact that the insulin deficiency has on hepatic lipid metabolism can be the reason behind this trend. Insulin normally inhibits hepatic lipolysis and regulates the activity of lipoprotein lipase and in this regard, insulin deficiency facilitates the synthesis of triglycerides and the inability to remove the circulating lipoproteins (25).

Also, chronic hyperglycemia may maximise glycation of lipoproteins, which results in decreased HDL activity and elevated LDL oxidation. Other studies have also found the same lipid abnormalities in pediatric T1D patients but others have found less severe dyslipidemia especially in patients with excellent glycemic control. This difference in studies could be attributed to variations in the glycemic control status, the duration of the disease, insulin therapy adherence and lifestyle variables like diet and physical activity (26).

They also found that there were strong changes in the circulating adipokines between diabetic children. Particularly, the level of adiponectin in T1D patients relative to their healthy controls was significantly lower, perhaps due to the high HbA1c or disease duration, whereas leptin, Resistin, and Visfatin were considerably higher. Adiponectin has been well-known as an anti-inflammatory and insulin-sensitizing adipokine that has a protective effect on glucose metabolism. The low levels of adiponectin in diabetic patients might indicate the disturbed functioning of adipose tissue and augmented graph of inflammatory signals (27).

Leptin, Resistin and Visfatin, on the other hand, are commonly linked to inflammatory and metabolic responses to stress. Increased inflammatory signaling and alterations in energy homeostasis of diabetic patients may lead to high levels of leptin. Insulin resistance, endothelial dysfunction, and inflammatory activation have been associated with Resistin and Visfatin, and therefore, the adipokines may play a role in metabolic disorders in pediatric diabetes (28). Nevertheless, other results have indicated opposite results of adiponectin levels in T1D with some reports indicating that levels were higher and not reduced. These inconsistencies can be attributed to variation in age of the patient, duration of a disease, distribution of body fats, and the compensatory metabolism responses in the early diabetes (29).

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The inflammatory character, which was noticed in the given study, also confirms the idea that type 1 diabetes is not merely a metabolic disorder but a chronic inflammatory disease. TNF-alpha, IL-6, IL-1b and CRP serum concentrations were markedly higher in diabetic children than in controls. These cytokines play a central role in the immune activation and they mediate the autoimmune destruction of pancreatic β-cells. Directly, TNF-α and IL-1β have the ability to trigger apoptosis in β-cells via nuclear factor-κB (NF-κB) and oxidative stress signaling pathways, which result in insulin release depleting progressively (30,31).

The IL-6 is also involved in the promotion of systemic inflammation as well as the modification of glucose metabolism. High CRP is a symptom of generalized inflammatory response which is common in long-term metabolic disorders. Whilst numerous studies have confirmed the existence of the association between T1D and the presence of inflammatory cytokines, other studies have found reduced or no difference between cytokines in specific groups. These discrepancies could be caused by differences in the stage of the disease, insulin treatment steps, genetics or insensitivity of the laboratory assays (32).

The correlation analysis showed that biomarkers and glycemic control have significant relationships. There was a significant negative relationship between adiponectin and positive relationship between leptin, Resistin, Visfatin and inflammatory cytokines and HbA1c levels. These results imply that decreasing glycemic control is linked to the increased inflammatory response and adipokine disproportion. Mechanistically, chronic hyperglycemia stimulates the development of advanced glycation end products (AGEs) and reactive oxygen species that trigger inflammatory responses and the production of cytokines. Meanwhile, the inflammatory cytokines may also further disrupt the insulin signaling pathways, which forms a vicious circle between the metabolic dysregulation and inflammation (33).

The high R between systemic inflammatory markers and HbA1c(specifically TNF-α($r=0.45$, $p<0.001$) and IL-6($r=0.42$, $p<0.001$), these finding imply that chronic hyperglycemia serves as the primary driver of the systemic inflammatory surge observed in these pediatric patients. The current study contributes to the notion that inflammatory markers can be used as the possible biomarkers that concentrate on the progression of the disease and metabolic adverse events of T1D in children (16).

Conclusion

The results showed that type 1 diabetes in children is connected with considerable changes in adipokines and pro-inflammatory cytokines that are closely interrelated with the lack of proper glycemic control. Lower adiponectin and increased leptin, Resistin, Visfatin and inflammatory cytokines indicate the existence of metabolic inflammatory imbalance. Sustained hyperglycemia can encourage oxidative stress and immune stimulation caused by hyperactivity of cytokines and dysfunction of adipose tissues. These interactions can possibly lead to the advancement of metabolic imbalance and inflammatory reactions, indicating the possible value of these biomarkers as the method of disease severity and glycemic control monitoring in children with T1D.

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Study Limitations

While this study provides valuable insights into the inflammatory landscape of pediatric T1D in the Thi-Qar region, several limitations must be acknowledged:

Cross-Sectional Design: Due to the cross-sectional nature of the study, we cannot establish a definitive causal relationship between adipokine dysregulation and the progression of diabetic complications. Longitudinal studies are required to determine if these biomarkers can predict long-term outcomes.

Sample Size and Diversity: Although our sample of 150 participants is statistically significant, it was drawn from a specific geographic region (Thi-Qar). Multi-center studies involving diverse Iraqi provinces would enhance the generalizability of the findings.

Pubertal Status: While age-matching was performed, we did not account for specific Tanner stages (pubertal development). Since puberty is a period of transient physiological insulin resistance and hormonal shifts, this may influence adipokine concentrations.

Nutritional Assessment: The study did not include a detailed dietary or physical activity log, which are external factors known to influence lipid profiles and adipokine secretion.

Ethical Approval:

The ethical approval of this study was taken care of by the Institutional Ethics Committee of the medical institution (Al-Habbobi Teaching Hospital-Nasiriya- Iraq) (1285, 10/04/2025) in which the research was done before the research began. Furthermore, the parents or legal guardians of the participating children gave informed consent in writing after the purpose of the study and procedures involved such as the blood sample collection, were explained to them. The research processes were also carried out in compliance with the ethical requirements of conducting research with human subjects and the protection of confidentiality and privacy of all the obtained data.

Conflict of Interest: None to declare

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