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ORIGINAL ARTICLE
ORİJİNAL ARAŞTIRMA

Retrospective Evaluation of the Clinical Characteristics, Laboratory Findings, and Treatment Approaches in Pediatric and Adolescent Patients Diagnosed with Type 2 Diabetes Mellitus: A Single-Center Experience

Tip 2 Diabetes Mellitus Tanılı Çocuk ve Adölesan Hastalarda Klinik Özellikler, Laboratuvar Bulguları ve Tedavi Yaklaşımlarının Retrospektif Değerlendirilmesi: Tek Merkez Deneyimi

Gülçin Arslan[1,2], Tarık Kırkgöz[1,3], Gülin Tabanlı[1], Behzat Özkan[1]

[1]Department of Pediatric Endocrinology, İzmir Dr. Behçet Uz Training and Research Hospital, University of Health Science, Izmir, Turkiye
[2]Department of Pediatric Endocrinology, Izmir City Hospital, University of Health Science, Izmir, Turkiye
[3]Department of Pediatric Endocrinology, Kayseri City Hospital, University of Health Science, Kayseri, Turkiye

Abstract

Aim: The prevalence of type 2 diabetes mellitus (T2DM) in children has been increasing in parallel with the rise in obesity. Due to its insidious onset and slow progression, complications often manifest later in adulthood. This study aims to evaluate the clinical and laboratory findings, and complication rates in pediatric patients with T2DM.

Material and Method: We reviewed the medical records of patients diagnosed with T2DM and followed in our clinic between January 2014 and January 2023. Demographic, clinical, laboratory, and treatment data were recorded.

Results: Sixty-three patients (42 females) were included in the study. The mean age at diagnosis was 13.9±2.2 years (range 11.1–17.3), 95% were pubertal. The mean weight, height, and body mass index (BMI) standard deviation scores (SDS) were 2.3±1.5 (range -3.2 to 5.9), 0.3±1.2 (range -2.2 to 3.5), and 2.1±1.1 (range -2.5 to 3.9), respectively. Polyuria and polydipsia were reported in 22 patients (34.9%) at diagnosis. Four patients presented with ketoacidosis. A family history of T2DM was present in 34 patients (54%). Antibodies were positive in four patients (6.6%). The mean HbA1c (glycosylated hemoglobin) level was 8.7%±2.0. Treatment included metformin alone in 24 patients (38%), metformin plus insulin in 29 (46%), and insulin alone in five (8.0%). Regarding complications, neuropathy was observed in one patient (1.6%), nephropathy in two patients (3.2%).

Conclusion: Prevention strategies on T2DM must focus on reducing obesity from an early age. Community-based interventions promoting healthy eating and physical activity are essential.

Keywords: Adolescent, non–insulin-dependent diabetes mellitus, obesity

Öz

Amaç: Çocuklarda tip 2 diabetes mellitus (T2DM) prevalansı, obezite sıklığındaki artışa paralel olarak giderek yükselmektedir. Sinsi başlangıcı ve yavaş ilerleyen seyri nedeniyle komplikasyonlar çoğunlukla erişkin dönemde ortaya çıkmaktadır. Bu çalışmanın amacı, T2DM tanılı pediatrik hastaların klinik ve laboratuvar bulgularını ile komplikasyon sıklıklarını değerlendirmektir.

Gereç ve Yöntem: Ocak 2014 ile Ocak 2023 tarihleri arasında kliniğimizde T2DM tanısı ile izlenen hastaların tıbbi kayıtları retrospektif olarak incelendi. Hastalara ait demografik özellikler, klinik bulgular, laboratuvar verileri ve tedavi bilgileri kaydedildi.

Bulgular: Çalışmaya 63 hasta (42 kız) dahil edildi. Tanı anındaki ortalama yaş 13,9±2,2 yıl (11,1–17,3) olup hastaların %95'i pubertal dönemdeydi. Ortalama ağırlık, boy ve beden kitle indeksi (BKİ) standart deviasyon skoru (SDS) sırasıyla 2,3±1,5 (-3,2–5,9), 0,3±1,2 (-2,2–3,5) ve 2,1±1,1 (-2,5–3,9) olarak bulundu. Tanı sırasında 22 hastada (%34,9) poliüri ve polidipsi mevcuttu. Dört hasta diyabetik ketoasidoz ile başvurdu. Hastaların 34'ünde (%54) ailede T2DM öyküsü saptandı. Dört hastada (%6,6) diyabet otoantikorları pozitif bulundu. Ortalama HbA1c düzeyi %8,7±2,0 idi. Tedavi açısından 24 hasta (%38) yalnızca metformin, 29 hasta (%46) metformin ve insülin kombinasyonu, 5 hasta (%8,0) ise yalnızca insülin kullandı. Komplikasyonlar değerlendirildiğinde, bir hastada (%1,6) nöropati ve iki hastada (%3,2) nefropati saptandı.

Sonuç: T2DM'nin önlenmesine yönelik stratejiler, erken yaşlardan itibaren obezitenin azaltılmasına odaklanmalıdır. Sağlıklı beslenme ve düzenli fiziksel aktiviteyi teşvik eden toplum temelli müdahaleler, çocukluk çağı T2DM yükünün azaltılmasında büyük önem taşımaktadır.

Anahtar Kelimeler: Tip 2 diabetes mellitus, adölesan, obezite

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Corresponding Author: Gülçin Arslan
Address: Department of Pediatric Endocrinology, İzmir Dr. Behçet Uz Training and Research Hospital, University of Health Science, Izmir, Turkiye
E-mail: gulcinak_005@hotmail.com

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder marked by insulin resistance and inadequate insulin secretion, occurring without autoimmune-mediated β-cell destruction. (1). In recent decades, the worldwide rise in childhood obesity has paralleled a substantial increase in the occurrence of T2DM among pediatric populations. Data from the World Health Organization indicate that nearly one in five children and adolescents aged 5–19 years are classified as overweight or obese, with rates showing a persistent upward trend (2). Population-based data from the SEARCH for Diabetes in Youth study in the United States reported T2DM incidence rates of 8.1 per 100,000 among individuals aged 10–14 years and 11.8 per 100,000 among those aged 15–19 years (3). Similarly, long-term surveillance data from Japan have demonstrated a strong association between the rising prevalence of obesity and increasing rates of T2DM in school-aged children (4). In parallel with global trends, a cohort study conducted in Türkiye demonstrated a substantial rise in the proportion of T2DM among newly diagnosed pediatric diabetes cases, increasing from 1.9% during 1999–2004 to 7.9% in the 2011–2016 period (5). Additionally, a multicenter Turkish study including 105 children and adolescents with obesity and/or a family history of T2DM reported no newly diagnosed cases of T2DM on oral glucose tolerance testing; however, prediabetes (impaired fasting glucose and/or impaired glucose tolerance) was identified in 25.5% of obese participants with a positive family history of T2DM (6).

The development of T2DM is driven by a gradual decline in pancreatic β-cell function resulting from the interplay of genetic susceptibility, environmental influences, excess adiposity, and the transient insulin resistance that occurs during puberty. Established risk factors include obesity, a positive family history of diabetes, exposure to gestational diabetes, low birth weight, acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome (PCOS), and associated metabolic conditions such as nonalcoholic fatty liver disease (NAFLD) (7). These determinants play a role not only in the initiation of the disease but also in the progression and early emergence of related complications. Genetic vulnerability is considered polygenic in nature; data from the TODAY (Treatment Options for Type 2 Diabetes in Adolescents and Youth) study indicate that 50–75% of adolescents diagnosed with T2DM had at least one parent affected by the condition (8). Although the precise biological pathways connecting obesity with insulin resistance have not been fully elucidated, excess adiposity is known to induce chronic low-grade inflammation. The accumulation of lipids and fatty acid metabolites disrupts insulin signaling pathways and reduces glucose uptake, particularly in skeletal muscle. To date, approximately 25 common genetic variants reaching genome-wide significance have been linked to T2DM; however, the functional roles of many of these variants and their target genes remain poorly characterized. Collectively, these variants explain less than 10% of the total heritable risk for T2DM (9). Puberty is accompanied by an approximately 30% physiological reduction in insulin sensitivity, and this temporary state of increased insulin resistance may hasten the development of T2DM (10).

In pediatric populations, T2DM generally has a more gradual and less overt onset than type 1 diabetes mellitus (T1DM); however, disease-related complications may emerge at an earlier stage (11). T2DM with onset in childhood or adolescence is linked to an accelerated development of both microvascular and macrovascular complications, and individuals diagnosed at a young age are reported to have a life expectancy that is approximately 15 years shorter than that of their age-matched peers (12). Establishing a diagnosis in adolescents may be difficult because of overlapping clinical characteristics with type 1 diabetes mellitus, monogenic forms of diabetes, and other conditions associated with obesity-related insulin resistance. Although hyperinsulinemia is frequently observed, islet autoantibodies are detected in approximately 10–20% of affected individuals (13). The clinical spectrum at presentation ranges from overt hyperglycemic symptoms—such as polyuria, polydipsia, ketosis, or diabetic ketoacidosis—to incidentally detected, asymptomatic hyperglycemia or subtle metabolic abnormalities. At the time of diagnosis, nearly one-quarter of adolescents with T2DM present with diabetic ketoacidosis or hyperosmolar conditions, both of which are linked to a higher risk of mortality (14).

As the prevalence of T2DM continues to rise among adolescents, the burden of associated comorbidities—including hypertension, dyslipidemia, nephropathy, and retinopathy—is expected to increase accordingly. Evidence from studies in young adults suggests that T2DM with early onset is associated with a more rapid progression of complications, underscoring its growing impact as a public health concern (15). Optimal management of T2DM in adolescents requires a comprehensive approach that extends beyond glycemic control to include lifestyle interventions, management of accompanying metabolic disturbances, and psychosocial support. Nevertheless, treatment options for this age group remain limited, and maintaining long-term adherence poses a significant challenge. At present, insulin and metformin, a biguanide, are the only pharmacological therapies approved by the United States Food and Drug Administration (FDA) for the treatment of T2DM in adolescents (16).

In our country, data on T2DM in children and adolescents are limited. Clearly defining the clinical course, development of complications, and treatment approaches in this population is of considerable importance to medical literature. The aim of this study is to contribute to the current knowledge of pediatric T2DM by retrospectively analyzing the clinical characteristics, laboratory findings, complications, and treatment strategies of children and adolescents diagnosed with T2DM and followed at a single tertiary care center.

MATERIAL AND METHOD

This study was approved by Local Clinical Research Ethics Committee, Ethics Committee of Human Experimentation in our institution/country and follows the Declaration of Helsinki (Non-interventional Research Ethical Committee of İzmir Dr. Behçet Uz Training and Research Hospital; Date: 23/10/2025; Decision no: 2025/ 16-06).

A total of 63 patients (42 female) who were diagnosed with T2DM and followed regularly at our center between January 2014 and January 2023 were included in the study. Diabetes mellitus was diagnosed based on either hemoglobin A1c (HbA1c) criteria or plasma glucose measurements, including fasting plasma glucose (FPG) or 2-hour plasma glucose levels. Blood samples were obtained after an overnight fast of at least 8 hours. According to the American Diabetes Association (ADA) guidelines, an FPG level ≥126 mg/dL (7.0 mmol/L) was considered diagnostic of diabetes (17). In accordance with the ADA recommendations, abnormal glycemic results in asymptomatic patients had been confirmed by repeat testing performed on a separate day as part of routine clinical practice. For all participants, the following data were collected: age, body weight, height, body mass index (BMI), pubertal staging, blood pressure, age at diabetes onset, presenting symptoms, presence of a family history of T2DM, current treatment regimen (metformin and/or insulin), and laboratory parameters. Laboratory assessments included blood glucose, creatinine, HbA1c (glycosylated hemoglobin), insulin, C-peptide, diabetes-related autoantibodies, and lipid profiles (total cholesterol, low-density lipoprotein [LDL], high-density lipoprotein [HDL], and triglycerides), as well as serum uric acid levels. Obesity was defined according to age- and sex-specific body mass index (BMI) percentiles based on established pediatric growth references. Children with a BMI at or above the 95th percentile were classified as obese. Pubertal development was assessed using Tanner staging, which classifies sexual maturation into five stages (I–V) based on secondary sexual characteristics. Tanner stage I was considered prepubertal, while stages II–V were considered pubertal. The decision to initiate insulin therapy was based on routine clinical practice in accordance with ADA and ISPAD recommendations. Insulin treatment was initiated in patients presenting with marked hyperglycemia (HbA1c >8.5% and/or blood glucose >250 mg/dL), ketosis or diabetic ketoacidosis, or significant hyperglycemic symptoms. Patients with milder hyperglycemia were generally treated with metformin together with lifestyle modification. For complications screening, patients underwent ophthalmologic evaluation for retinopathy, urinary albumin measurements and pediatric nephrology consultation for nephropathy, and pediatric neurology evaluation for neuropathy. Diabetic neuropathy was defined based on clinical symptoms and findings on neurological examination, including reduced vibration perception, impaired sensation, or decreased ankle reflexes. Screening was performed using standardized neurological examination and, when available, validated tools for peripheral neuropathy assessment. Diabetic nephropathy was defined by the presence of persistent albuminuria, assessed as an elevated urinary albumin-to-creatinine ratio (ACR ≥30 mg/g) in at least two of three urine samples, and/or a reduced estimated glomerular filtration rate (eGFR <60 mL/min/1.73 m^2). Screening was performed using spot urine samples for ACR and serum creatinine measurements. Definitions and screening methods were based on the recommendations of the American Diabetes Association (ADA) and the International Society for Pediatric and Adolescent Diabetes (ISPAD) (18,19).

Statistical Analysis

The data obtained from the study were analyzed using the Statistical Package for the Social Sciences Statistics software, version 25.0 (IBM Corp., Armonk, NY, USA). The distribution of the data was evaluated with the Shapiro–Wilk test and the homogeneity of variance was evaluated with the Levene test. For numerical comparisons, the Student's t-test or Mann–Whitney U-tests were used to assess differences between the 2 groups according to the normal distribution of the measured parameters.

RESULTS

A total of 63 patients (42 female) were included in the study. The mean age at diagnosis was 13.9±2.2 years (range: 11.1–17.3), and 95% of the cases were in the pubertal stage. At the time of diagnosis, the mean standard deviation scores (SDS) for weight, height, and body mass index (BMI) were 2.3±1.5, 0.3±1.2, and 2.1±1.1, respectively. Fifty-seven patients (87.3%) were classified as obese. At the end of the first year, the mean SDS values for weight, height, and BMI were 2.27±0.52, 0.42±0.72, and 2.06±0.35, respectively. At the end of the second year, the values were 2.03±1.27, –0.24±0.82, and 2.05±0.66. The mean change (delta) in weight SDS over time was –0.31±0.81. Laboratory parameters of the patients with T2DM included in the study were shown in **Table 1**.

Table 1. Laboratory parameters of the patients with T2DM included in the study.

Laboratory parameters	N
Triglyceride (mg/dl)	174.13±85.5
Total cholesterol (mg/dl)	171.7±35.8
LDL (mg/dl)	107±20.5
HDL (mg/dl)	41.9±4.06
Blood glucose (mg/dl)	185.3±1.35
Creatinine (mg/dl)	0.7±0.09

N: number of participants, LDL: low-density lipoprotein, HDL: high-density lipoprotein

In 53 patients (84.1%), the diagnosis of diabetes was established based on an HbA1c level ≥6.5%, according to the American Diabetes Association (ADA) criteria. The remaining 10 patients (15.9%) had baseline HbA1c levels <6.5%. An oral glucose tolerance test (OGTT) was performed in 19 patients overall. Importantly, all 10 patients with HbA1c levels <6.5% underwent an OGTT, and each demonstrated a 2-hour plasma glucose level >200 mg/dL, confirming the diagnosis of T2DM based on the postload glucose criterion. Among the study cohort, the median HOMA-IR was 8.71 (Q1–Q3: 5.23–17.15), and the median follow-up duration was 1.50 years (Q1–Q3: 1.00–2.50).

At diagnosis, 22 patients (34.9%) presented with polyuria and polydipsia, while only 4 patients (6.6%) were diagnosed with mild diabetic ketoacidosis. Notably, 39 patients (61.9%) were diagnosed without any typical diabetes-related symptoms. A positive family history of T2DM was present in 34 patients (54%). None of the patients were diagnosed with PCOS and 10 patients (15.8%) were identified as SGA. Pharmacological treatments administered to patients with T2DM included in the study were shown in **Table 2**.

Table 2. Treatment regimens of patients with T2DM included in the study.

Type of Treatment	N (Percentage)
Metformin + insulin	29 (%46)
Metformin	24 (%38)
Insulin	5 (%8)

*N: number of participants

Anti-glutamic acid decarboxylase (anti-GAD) antibodies were found to be positive in four patients (6.6%). At the time of diagnosis, the mean HbA1c level was 8.7%±2.0, mean insulin level was 32.8±34.5 mU/L, and mean C-peptide level was 4.1±3.1 ng/mL. At the most recent follow-up, mean HbA1c had decreased to 6.8%±1.04, and mean insulin level was 20.09±1.35 mU/L. Twenty six patients (41.2%) had triglyceride levels >150 mg/dL, 27 patients (42.8%) had LDL cholesterol >100 mg/dL, and 15 patients (23.8%) had HDL cholesterol <35 mg/dL. Of the 29 patients who were initially started on insulin therapy, insulin was successfully discontinued during follow-up in 9 patients (31%). Regarding complications, diabetic neuropathy was observed in one patient (1.6%), the mean serum creatinine level was 0.7±0.09 mg/dL, and the mean albumin-to-creatinine ratio (ACR) was 0.00834±0.0047 mg/g, notably, none of the patients had microalbuminuria at diagnosis. In addition, one patient (1.6%) was being followed with a diagnosis of Cushing syndrome, and one patient (1.6%) with Prader-Willi syndrome. No cases of diabetic retinopathy were detected in our cohort.

DISCUSSION

Among the 63 pediatric and adolescent patients with T2DM, the mean age at diagnosis was 13.9 years, and most patients were female and obese, consistent with previous national and international reports. A retrospective multicenter study from Türkiye including 255 pediatric T2DM cases reported a comparable mean age at diagnosis (13.7±2.2 years) (20). Likewise, females are reported to have a 1.3- to 1.7-fold higher risk of developing T2DM than males (1), and 69% of patients in the Turkish multicenter cohort were female, similar to the 66.6% (42/63) observed in our study.

Obesity is the principal risk factor for pediatric T2DM and is considered a major contributor to its increasing incidence (21, 22). Approximately four out of five children and adolescents with T2DM have obesity, while only about 10% are overweight and a similar proportion have normal body weight (23). Consistent with these data, 87.3% (57/63) of our patients were obese, with a mean BMI SDS of 2.06±0.35.

According to the American Diabetes Association (ADA), screening for T2DM is recommended in overweight or obese children with additional risk factors, including a family history of T2DM, exposure to maternal diabetes or gestational diabetes, and clinical features of insulin resistance such as acanthosis nigricans, hypertension, dyslipidemia, PCOS, or a history of being born small for gestational age (24, 25). In our cohort, acanthosis nigricans was present in 30.1% and hypertension in 9.5% of patients. Acanthosis nigricans is a well-recognized marker of insulin resistance, with reported prevalence rates of 17%–36% among adults with newly diagnosed T2DM, increasing with obesity and cumulative metabolic risk (26).

PCOS is characterized by insulin resistance and compensatory hyperinsulinemia and is associated with an increased risk of glucose intolerance and T2DM, independent of obesity (25, 27). Li et al. demonstrated a significantly higher prevalence of T2DM among women with PCOS (28), while Cree-Green et al. showed that adolescents with PCOS develop early metabolic abnormalities, including impaired insulin sensitivity and adipose tissue dysfunction, before overt hyperglycemia (29). Although PCOS is more common in obese adolescent girls, its prevalence among adolescents with T2DM remains unclear (27). None of the patients in our cohort had a diagnosis of PCOS.

Being born small for gestational age (SGA) and fetal macrosomia secondary to maternal diabetes are recognized perinatal risk factors for pediatric T2DM (24). Infants born SGA, particularly those with rapid catch-up growth and subsequent obesity, are at increased risk of insulin resistance, T2DM, and metabolic syndrome later in life (1). In our cohort, 10 patients (15.8%) were born SGA. Although the mean HbA1c level was slightly higher in the SGA subgroup than in the overall cohort (9.03±2.16% vs. 8.7±2.0%), the difference was not statistically significant.

A positive family history is another major risk factor for pediatric T2DM. The TODAY study reported that 50%–75% of affected youth had at least one parent with T2DM, increasing to 98% when grandparents were included (8). Similar findings have been reported in other cohorts (1), including a Turkish multicenter study in which 80% of pediatric patients had a positive family history (20). In our study, 54% of patients had a family history of T2DM.

Pediatric T2DM is frequently asymptomatic at diagnosis (24,25). Consistent with previous reports from Türkiye, where only 27% of patients presented with overt diabetic symptoms and diabetic ketoacidosis was uncommon (20), 61.9% of our patients were diagnosed without classical symptoms. Polyuria and polydipsia were present in 34.9% of patients, whereas only four patients (6.6%) had mild ketoacidosis.

Approximately 90% of individuals with T2DM are negative for diabetes-related autoantibodies, although low-level positivity may occur (13). Therefore, autoantibody results should always be interpreted together with the clinical presentation and metabolic profile (17,25). In our cohort, anti-glutamic acid decarboxylase (anti-GAD) antibodies were positive in four patients (6.6%). The mean HbA1c level at diagnosis was 8.7±2.0%, comparable to that reported in the Turkish multicenter study (9.7±2.78%) (20). As expected in T2DM, fasting insulin and C-peptide levels were generally preserved, with mean values of 32.8±34.5 mU/L and 4.1±3.1 ng/mL, respectively (1).

According to the ADA, the main goals of T2DM management are weight reduction, optimal glycemic control, and treatment of associated comorbidities (18, 30). In adolescents, recommended targets include fasting plasma glucose <126 mg/dL and HbA1c <6.5% within 3–4 months (17). In our cohort, mean HbA1c decreased from 8.7±2.0% at diagnosis to 6.8±1.04% at the final follow-up, while mean insulin level was 20.09±1.35 mU/L. Among the 29 patients who initiated insulin therapy, 9 (31%) were able to discontinue insulin during follow-up.

Screening for albuminuria is recommended at diagnosis and annually thereafter (18,27). In the TODAY study, microalbuminuria increased from 6.3% at baseline to 16.6% after three years (31). In contrast, none of our patients had microalbuminuria at diagnosis, with a mean serum creatinine of 0.7±0.09 mg/dL and a mean ACR of 0.00834±0.0047 mg/g.

Hypertension and dyslipidemia are common comorbidities in youth with T2DM (8,18,31–34). In the TODAY study, hypertension increased from 11.6% to 33% and dyslipidemia from 4.5% to 11% during follow-up (31, 32), while similar findings were reported in Canadian First Nations youth (33). In our cohort, hypertension was present in 9.5% of patients. Dyslipidemia was also common, with elevated triglycerides in 41.2%, LDL cholesterol in 42.8%, and low HDL cholesterol in 23.8% of patients.

Annual retinal examinations are recommended after diagnosis (18). Consistent with the relatively short disease duration, no patients developed diabetic retinopathy. Overall, chronic complications were uncommon: diabetic neuropathy and nephropathy were observed in 1.6% and 3.2% of patients, respectively. In comparison, diabetic neuropathy has been reported in 21%–26% of youth with T2DM, and diabetic retinopathy in 2%–40%, depending on disease duration and study design (35).

Pharmacological treatment aims to improve insulin sensitivity and preserve endogenous insulin secretion (36). The TODAY study demonstrated that approximately half of adolescents maintained glycemic control with metformin monotherapy, whereas the remainder eventually required insulin because of progressive loss of glycemic control (22,37). In our cohort, 24 patients (38%) received metformin alone, while 29 (46%) were treated with combined metformin and insulin, consistent with current recommendations for patients presenting with marked hyperglycemia (22,37).

The frequency of complications in patients with diabetes increases over time. Therefore, the relatively low complication rate observed in our study may be attributed to the lack of long-term follow-up, which is one of the main limitations of our study. HbA1c levels may be influenced by factors such as anemia, hemoglobinopathies, and altered red blood cell turnover. HbA1c alone may not fully reflect glycemic status and CGM data were unavailable; therefore, glycemic variability could not be assessed. The absence of regular glucose monitoring in our patient cohort, the relatively small sample size, and the lack of objective early markers of complications, such as cystatin C, also constitute important limitations of this study. Waist circumference measurements were not available for the participants. In addition, all socioeconomic and lifestyle-related factors were not evaluated. One of the strengths of our study is the assessment of anthropometric measurements and HbA1c levels both at baseline and during follow-up, allowing for the evaluation of treatment-related changes. Additionally, the consistency of our findings with previous studies further supports the validity of our results. Prospective, multicenter studies with larger cohorts and more comprehensive metabolic, genetic, and lifestyle assessments are needed to confirm these findings.

CONCLUSION

Type 2 diabetes mellitus is a polygenic metabolic condition closely linked to obesity and marked by insulin resistance. Its occurrence has risen markedly in pediatric populations over recent years. Given that disease progression and many related complications typically become apparent later in adulthood, effective prevention across the life course largely relies on early interventions targeting obesity during childhood and adolescence. Consequently, there is a substantial need for large-scale, long-term studies to more comprehensively assess the burden and trajectory of T2DM-related complications in children and adolescents.

ETHICAL DECLARATIONS

Ethics Committee Approval: This study was approved by Local Clinical Research Ethics Committee, Ethics Committee of Human Experimentation in our institution/ country and follows the Declaration of Helsinki (Non-interventional Research Ethical Committee of İzmir Dr. Behçet Uz Training and Research Hospital; Date: 23/10/2025; Decision no: 2025/ 16-06).

Informed Consent: Written informed consent has been obtained from the patients to publish this paper.

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