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Types and clinical features of 140 newly diagnosed cases of diabetes in childhood: a single-center experience

Çocukluk çağında yeni tanı almış 140 diyabet olgusunun tanı tipleri ve klinik özellikleri: tek merkez deneyimi

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Abstract

Purpose: Type 1 diabetes (T1DM) accounts for the majority of childhood diabetes mellitus (DM). However, in recent years, there has been an increase in the prevalence of type 2 diabetes mellitus (T2DM) and the diagnosis of monogenic diabetes (MD). The aim of this study was to evaluate the clinical and laboratory findings, as well as the types of DM, in patients diagnosed between the ages of 0 and 18.

Materials and methods: In the study, 140 patients diagnosed with DM in our clinic were evaluated retrospectively.

Results: During the 3-year period, 140 patients (n=76, 54.3% male) were diagnosed with diabetes. The mean age at diagnosis of the patients was 10±4.19 years. 93.6% of patients were diagnosed with T1DM, 2.8% of patients with T2DM and 3.6% of patients were diagnosed with MD. It was observed that the cases of T1DM peaked in the 5-9 (36.6%) and 10-14 (37.4%) age groups. the prevalence of diabetic ketoacidosis (DKA) was 61.8%. The majority of patients 64.9% with T1DM were diagnosed in the autumn/winter months. 75% of the patients with T2DM were female, and the mean age at diagnosis was 15.05±1.11years. Two of the cases of MD were neonatal DM, two were GCK-MODY and one was CEL-MODY.

Conclusion: Although the majority of childhood diabetes cases are T1DM, the frequency of T2DM tends to increase, especially in obese adolescents. It should be kept in mind that obesity may also occur in autoantibody-positive T1DM patients. It was determined that T1DM cases were more common in the winter season, in the 10-14 age group, and that DKA was higher. Genetic examination should be performed in cases with suspected MD.

Keywords: Diabetes mellitus, type 1 diabetes, type 2 diabetes, monogenic diabetes, childhood.

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Öz

Amaç: Çocukluk çağındaki diyabetes mellitusun (DM) çoğunluğunu tip 1 diyabet (T1DM) oluşturur. Ancak son yıllarda tip 2 diyabetes mellitus (T2DM) sıklığında ve monogenik diyabet (MD) tanısı koymada artış gözlenmektedir. Bu çalışmada 0-18 yaş arasında tanı konulan diyabetli hastalarda klinik ve laboratuvar bulgularının, DM tiplerinin değerlendirilmesi amaçlandı.

Gereç ve yöntem: Çalışmada kliniğimizde DM tanısı alan 140 hasta retrospektif olarak değerlendirildi.

Bulgular: Üç yıllık süreçte 140 hastaya (76 erkek, %54,3) diyabet tanısı konuldu. Hastaların ortalama tanı yaşı 10±4,19 yıldı. Hastaların %93,6'sına T1DM, %2,8'ine T2DM, %3,6'sına MD tanısı konuldu. Tip 1 diyabetes mellitus vakalarının 5-9 (%36,6) ve 10-14 (%37,4) yaş gruplarında zirve yaptığı görüldü. Diyabetik ketoasidoz (DKA) prevalansı %61,8 idi. Yine bu olguların %64,9'u sonbahar/kış aylarında tanı aldı. Tip2 diyabetli hastaların %75'i kadındı ve ortalama tanı yaşı 15,05±1,11 idi. Monogenik diyabetli olgularının ikisi neonatal DM, ikisi GCK-MODY ve biri CEL-MODY idi.

Sonuç: Çocukluk çağı diyabet vakalarının çoğunluğu T1DM olmasına rağmen, özellikle obez ergenlerde T2DM sıklığı artma eğilimindedir. Otoantikor pozitif T1DM'li hastalarda da obezitenin olabileceği akılda tutulmalıdır. Tip1 diyabet olgularının daha çok kış mevsiminde, 10-14 yaş grubunda başvurduğu ve DKA sıklığının yüksek olduğu saptandı. Monogenik diyabet şüphesi olan olgulara genetik inceleme yapılmalıdır.

Anahtar kelimeler: Diyabetes mellitus, tip 1 diyabet, tip 2 diyabet, monogenik diyabet, çocukluk çağı.

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Introduction

Diabetes mellitus is a metabolic disease characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Deficient insulin action in tissues leads to abnormalities in carbohydrate, fat, and protein metabolism. The main diagnostic criterion in all types of diabetes in children is the biochemical increase in plasma glucose level in the presence of hyperglycemia symptoms. In the presence of overt symptoms, a significant increase in fasting plasma glucose (≥126 mg/dl) or random plasma glucose (≥200 mg/dl), or an OGTT 2-hour plasma glucose level of ≥200 mg/dl and an HbA1c value of ≥6.5% confirm the diagnosis of diabetes [1].

Type 1 diabetes (T1DM) is usually characterized by the loss of endogenous insulin production as a result of damage to β cells by an autoimmune mechanism. There is the presence of one or more autoantibodies. These cases are defined by the presence of at least one of the following: islet cell autoantibodies (ICA), insulin autoantibodies (IAA), anti-glutamic acid decarboxylase 65 autoantibodies (anti-GAD), and β-cell-specific zinc transporter 8 autoantibodies (ZnT8). Some patients have autoantibody negativity and are referred to as idiopathic [1-3]. T1DM accounts for 90% of diabetes in children [1]. It occurs at a rate of 52.2 per 100.000 in Finland, where the highest incidence has been observed [1, 4]. In our country, the incidence of T1DM has been reported as 11.3 per 100.000 in the 0-14 age group and 10.8 per 100.000 in the 0-18 age group [5].

Type 2 diabetes (T2DM) results from inadequate insulin response in the presence of increased insulin resistance [2]. It is a metabolic disorder that predominantly affects adults. However, its prevalence has been on the rise in recent years, particularly among obese children and adolescents. T2DM has a multifactorial etiology consisting of genetics, physiology, a high-calorie diet, low physical activity, and a sedentary lifestyle. The possibility of microvascular complications is higher in cases of T2DM than in cases of T1DM [6]. In Canada, which is one of the countries with a high incidence of T2DM, the incidence in the 0-18 age group is 821/100.000; and in Australia, the incidence of T2DM is 670/100.000 in the under 24 age group. Low incidence rates have been reported in Europe and the United Kingdom (0.6-1.4/100.000) [7].

Monogenic diabetes (MD) is caused by a defect in a single gene involved in β-cell development or function. It constitutes 1-6% of the cases. It can occur as neonatal diabetes (NDM), maturity-onset diabetes of the young (MODY), and various syndromes. MODY is the most common type. Since the clinical features in cases of MODY are similar to T1DM and T2DM, patients may sometimes get misdiagnosed [1]. Neonatal diabetes is an MD that occurs in the first 6 months of life. Neonatal diabetes should also be considered in patients between 6-12 months of age who have no findings of autoimmunity [8]. Neonatal diabetes can be transient/permanent. Today, dysfunctions in more than 40 genes have been identified to cause MD [9]. Monogenic diabetes is identified more in childhood with the increase in genetic testing. Monogenic diabetes should be considered in cases of diabetes occurring especially in the first 12 months of life, in the presence of autosomal dominant familial hyperglycemia/diabetes, and in patients with extrapancreatic findings (elevated liver enzymes, congenital heart disease, diarrhea, brain malformations, optic atrophy, deafness, etc.) [1, 8].

In this study, it was aimed to determine the clinical and laboratory features on admission and types of diabetes in patients who were diagnosed with diabetes in Mardin Training and Research Hospital between the ages of 0-18.

Materials and methods

Study population and laboratory

In the study, 140 patients between the ages of 0-18 who were diagnosed with diabetes between 2020 and 2023 in the Pediatric Endocrinology Clinic of Mardin Training and Research Hospital were included. Records of the patients were examined retrospectively. Diabetes was diagnosed according to the criteria of the American Diabetes Association (ADA) and International Society for Pediatric and Adolescent Diabetes (ISPAD) guidelines [1, 10].

The age, gender, body weight (BW), BW standard deviation score (SDS), height, height SDS, body mass index (BMI), and BMI SDS of the patients at the time of admission were obtained. Obesity was defined as a BMI ≥95th percentile for age and gender. Calculations of oxological data were made using the growth curves of Turkish children [11]. In addition, the season of admission, consanguinity between parents, number of siblings, whether the patients were from rural or urban areas, and family history of diabetes were recorded. The plasma glucose levels, C-peptide levels, blood ketones, blood pH, glycated hemoglobin (HbA1c) values, Anti-GAD, IAA and ICA at the time of admission were obtained from the medical records of the patients and evaluated. ZnT8 autoantibodies could not be measured. The presence of one or more autoantibodies against β-cells was considered positive autoimmunity [1]. The cases were defined as ketosis or diabetic ketoacidosis (DKA) according to blood gas values and presence of ketones. Patients with a beta-hydroxybutyrate (BOHB) level of ≥3 mmol/L in the blood or a ketone level of ≥+2 in urine were considered positive for ketones, a venous blood pH of <7.30 and bicarbonate (HCO3) of <18meq/L were considered DKA. Diabetic ketoacidosis was classified according to the pH and HCO3 values of the patients (patients with a pH of <7.3 and a HCO3 of <18 mEq/L were considered to have mild DKA, patients with a pH of <7.2 and a HCO3 of <10 meq/L were considered to have moderate DKA, and patients with a pH of <7.1 and a HCO3 of <5 meq/L were considered to have severe DKA) [12].

The patients were evaluated during the diagnosis/follow-up in terms of thyroid stimulating hormone (TSH), free thyroxine (fT4), anti-thyroglobulin (anti-Tg), anti-thyroid peroxidase (Anti-TPO), celiac autoantibodies (tissue transglutaminase IgA and IgG antibodies) and accompanying autoimmune diseases. The cases of T2DM were evaluated in terms of hyperlipidemia, hypertension, and fatty liver. Genetic analysis for MD was performed in patients with negative diabetes autoantibodies, family history of autosomal dominant diabetes, patients younger than 12 months, and patients with low insulin requirement (such as 0.5U/kg/day) 1 year after diagnosis. [1].

Approval for the study was obtained from Mardin Artuklu University Faculty of Medicine Ethics Committee (approval date: 19/04/2023-94149, approval number: 2023/4-7).

Statistical analysis

SPSS 11.0 for Windows® package software was used in the analyses. Data were expressed as mean±SD (range) or median (interquartile range, IQR). In the comparison of the data of two independent groups: if the group distribution was normal, the independent samples t test, and if the distribution was not normal, the Mann-Whitney U test was used. A *p* value less than 0.05 was considered statistically significant.

Results

Of the 140 patients included in the study, 93.6% were diagnosed as T1DM, 2.8% as T2DM and 3.6% as MD. Of all patients, 64 (45.7%) were female, 76 (54.3%) were male and the mean age at diagnosis was 10±4.19 years (Table 1). A minimum of one autoantibody-positive result was observed in 69.2% of patients, while 44.2% of patients exhibited two or more autoantibody-positive results. Of the 83 patients (59.2%) who had DKA at presentation, 42.1% (n=35) had severe DKA. Six percent (n=5) of all DKA patients were younger than three years old. Cerebral edema developed clinically in 3 patients who presented with severe DKA. There was no mortality in any of the patients.

Type 1 diabetes cases

Type 1 Diabetes was diagnosed in 93.6% (n=131) of our patients. Of the T1DM cases, 58 (44.3%) were female and 73 (55.7%) were male. The prevalence of DKA was found to be 61.8% in all patients with T1DM. From the medical records of the patients, it was determined that 20.6% had a family history of T1DM. There was a female predominance in patients under the age of 10 and a male predominance in patients over the age of 10. The majority of patients (64.9%) were diagnosed in the autumn/winter months (Table 2). A total of 25.2% (n=33) of patients exhibited least one antibody positivity, while 48.9% (n=64) were positive for more than one antibody. These cases were classified as autoimmune T1DM. A total of 25.9% (n=34) of the patients were found to be autoantibody-negative, and these cases were classified as non-autoimmune T1DM (idiopathic).

Table 1. Sociodemographic and clinical characteristics of patients at the time of diagnosis.

	Total
Age at diagnosis[a] (years)	10±4.19
Gender (Female/Male)	64 (45.7%) / 76 (54.3%)
Family history of diabetes	20%
Parental consanguinity	37.8%
Living in the city center	37.9%
Mean number of children of the families	4.2
Prepubertal	49.2%
Plasma glucose[a] (mg/dL)	436.9±154.9
C-peptide[b] (ng/ml)	0.35 (0.4)
HbA1c[b] (%)	11.8% (3.38)

SDS: standard deviation score, [a]: mean± standard deviation, [b]: median (IQR), HbA1c: Glycated hemoglobin

Table 2. Characteristics of type 1 diabetes cases

Variable	**Category**	**n (%)**
Gender	Female	58 (44.3%)
	Male	73 (55.7%)
Age at diagnosis	0-4 years old	14 (10.7%)
	5-9 years old	48 (36.6%)
	10-14 years old	49 (37.4%)
	15-18 years old	20 (15.3%)
Season of diagnosis	Winter	49 (37.4%)
	Autumn	36 (27.5%)
	Spring	31 (23.7%)
	Summer	15 (11.4%)
Diabetic ketoacidosis	Present	81 (61.8%)
	Absent	50 (38.2%)

At the time of diagnosis, 8 (6.1%) patients were found to have autoantibody positivity for Hashimoto's thyroiditis, and the patients were euthyroid. In 11 (8.3%) patients, celiac serology positivity was found. In 3 (27.2%) of these patients, spontaneous normalization was detected, while celiac disease was identified by biopsy in 8 (6.1%) patients.

Of the autoantibody-negative patients, 53% presented with DKA, 23.5% with ketosis, and 23.5% with hyperglycemia. The number of obese patients diagnosed with T1DM was 4. Only one of these patients had autoantibody negativity, and the remaining three patients presented with DKA. There was no difference between autoantibody-positive and negative cases in terms of age, weight SDS, height SDS, and BMI SDS. The mean plasma glucose levels, median C-peptide and HbA1c levels of both groups at the time of admission were similar (Table 3).

Table 3. Clinical and laboratory features of T1DM cases

	Total (n=131)	Antibody+ n=97)	Antibody- (n=34)	t, z value	*p* value
Age[a]	10.2±3.94	9.88±3.89	10.44±4.12	t=-0.71	0.477
Weight SDS[a,b]	-0.53 (1.64)	-0.45±1.16	-0.40±1.33	t=-0.23	0.814
Height SDS[a]	-0.08±1.07	-0.11±1	-0.0003±1.26	t=-0.53	0.596
BMISDS[a]	-0.59±1.27	-0.54±1.25	-0.73±1.34	t=0.73	0.464
Glucose[a]	446.2±146.9	444.7±144.3	450.5±156.2	t=-0.19	0.844
C-peptide[b]	0.34 (0.34)	0.32 (0.31)	0.35 (0.59)	z=-1.31	0.190
HbA1c[a,b]	12.1 (3.2)	12.4 (3.65)	12.21±2.12	z=-0.38	0.700

SDS: standard deviation score, [a]: mean± standard deviation, [b]: median (IQR), HbA1c: Glycated hemoglobin, BMI: Body mass index
t: Independent samples t test, z: Mann-Whitney U test

Type 2 diabetes cases

Four (2.8%) patients were diagnosed with T2DM. The mean age at diagnosis of the patients was 15.05±1.11 years, the mean BMI SDS was 3.36±1.16, the mean C-peptide level was 2.63±0.39 ng/ml, and the mean HbA1c value was 6.32±1.15 %. The patients were pubertal. They were asymptomatic at the time of diagnosis. Three of the patients, who presented with obesity, were diagnosed by high fasting plasma glucose levels. In the other patient, diabetes was detected by the OGTT. Acidosis or ketosis was not observed in the patients. All patients were autoantibody-negative and had acanthosis nigricans, hyperlipidemia, and fatty liver. Hypertension or other microvascular complications were not detected. All of the patients had a family history of T2DM. Metformin was administered to three patients, and metformin+ insulin treatment was administered to one patient.

Monogenic diabetes cases

This group consisted of 5 (3.6%) patients. Two patients were diagnosed with neonatal diabetes. One of them presented with hyperglycemia in the neonatal period. A heterozygous mutation of c.692G>T (p.Trp231Leu) was detected in the ABCC8 gene in this patient. Following a brief course of treatment, the patient achieved remission at six months of age. When the same mutation was detected in the 21-year-old uncle of this patient, who was receiving insulin treatment, his treatment was changed to sulfonylurea (SU). The other patient presented with severe DKA at 5 months of age. In the genetic analysis, a homozygous mutation was detected in the lipopolysaccharide-responsive beige-like anchor (LRBA) gene. Immunodeficiency was not observed in the patient who was receiving insulin treatment and had autoantibody negativity.

A c.214G>A (p.Gly72Arg) heterozygous mutation was detected in the GCK gene in a 23-month-old patient presented with hyperglycemia, who had a family history of diabetes in his mother, grandmother, and aunts. Low-dose insulin treatment was initiated in the patient whose hyperglycemia continued despite the diet. Plasma glucose regulation was achieved with insulin treatment. In the genetic analysis of a 13-year-old girl who presented with fasting hyperglycemia, c.661G>A (p.E221K) heterozygous mutation was detected in the GCK gene. In this patient, blood sugar was regulated simply by adjusting the diet. In the genetic analysis performed on a patient who presented with hyperglycemia and obesity, and had a family history of diabetes and autoantibody negativity, c.1776dup (p.Val593Argfs*6) heterozygous mutation was detected in the CEL (carboxyl ester lipase) gene. Treatment was initiated in the patient who required insulin.

Discussion

Type 1 diabetes is the most common type of diabetes in childhood. According to 2021 data from the International Diabetes Federation (IDF), it is estimated that 108.200 children and adolescents under the age of 15 are diagnosed with T1DM every year [13]. It is stated that T1DM has two peaks, at ages 5-7 and during puberty. It is thought that the first peak occurs due to infections during the school starting period, and the second peak occurs due to the increase in insulin-counteracting hormones during puberty [14]. In our study, it was observed that T1DM peaked in the 5-9 and 10-14 age groups, and this result was consistent with previous studies conducted in Türkiye [3, 5, 15].

Some patients cannot be clearly classified at the time of diagnosis. Accurate identification of the type of diabetes can help determine treatment approaches [1]. The presence of autoantibodies against pancreatic islet cells is used as the best diagnostic tool for T1DM [1, 16]. In our T1DM series consisting of 131 cases, the rate of autoantibody positivity at the time of diagnosis was found as 74.1%. In a study conducted in China, the rate of autoantibody positivity at the time of diagnosis was reported as 61.01% in patients with T1DM [17]. With the increase in obesity in the general population, patients with autoimmune T1DM may present with overweight/obesity as well. Detection of autoimmune markers is useful in the diagnosis of T1DM, especially in patients, in whom the distinction is not clear, such as obese adolescents. However, studies show that autoantibody positivity may be present in 10-20% of the patients who have been clinically diagnosed with T2DM [7].

It has been stated that approximately 10% of children and adolescents with T1DM do not have autoimmunity [16, 18]. 25.9% of our patients with T1DM were autoantibody-negative. In a study, genes responsible for MD were identified in 10.5% of patients requiring insulin therapy and exhibiting no autoantibodies [19]. Monogenic diabetes may be one of the reasons for the high number of autoantibody-negative patients in our society, where the rate of parental consanguinity is high. However, genetic analysis could not be performed in all of these patients.

The risk of developing T1DM is higher among relatives of patients with T1DM than in the general population. The incidence rate of diabetes in the sibling of a child with T1DM is between 6-7% [1]. A total of 20.6% of patients with T1DM had a family history of T1DM, which is consistent with the findings of previous studies [20]. The aetiology of T1DM is influenced by a number of factors, including environmental factors and viral infections. In our study, similar to previous studies [21, 22], it was observed that the patients presented predominantly in the autumn/winter seasons, when viral infections were more common (64.9%). In some publications, male predominance has been reported in T1DM cases diagnosed during adolescence and later. In our T1DM cases, there was a male predominance over the age of 10 [23].

Diabetic ketoacidosis is a life-threatening complication of diabetes. Morbidity and death may occur during the follow-up of patients. It has been observed that the prevalence of DKA is lower in regions where socioeconomic status is higher and better health services are provided. In a study conducted in 13 countries, the prevalence of DKA at diagnosis was reported as 29.9% [24]. The frequency of DKA was reported at varying rates in North America and Europe (15-70%) [12]. In the SEARCH study conducted in 2021, it was found that the prevalence of DKA had increased over the years [25]. In our study, the frequency of DKA in patients with T1DM was found to be 61.8%. In a recent study conducted in the neighboring province of Diyarbakır, the frequency of DKA was found high (64.9%) [26]. The previously reported rates in our country were found to be lower [3, 27, 28]. The high rate of DKA in our region was attributed to the late presentation/diagnosis of the patients. In addition, since the starting year of the study overlapped with the COVID-19 pandemic, cases may have presented with DKA more frequently. Early diagnosis of diabetes and reducing the frequency of DKA through effective education will reduce the incidence of fatal complications such as cerebral edema.

Type 1 diabetes is frequently accompanied by other autoimmune diseases in children. Studies have reported thyroid autoantibody positivity in approximately one-quarter of patients with

TIDM [29]. In our study, autoantibody positivity was detected in 6.1% of the patients, and all of these patients were euthyroid. The incidence of celiac disease in patients with T1DM has been reported to vary between 1-10% [30]. In a study conducted in our country, positive celiac serology was found in 15.4% of the patients and spontaneous recovery occurred in 23.3% of these patients [30]. In our study, positive celiac serology was detected in 8.3% of the patients, the frequency of spontaneous normalization (27.2%) and biopsy-confirmed celiac disease was found in 6.1% of all patients with T1DM; these results were consistent with the literature [30].

The incidence of T2DM in children and adolescents has increased significantly in the last 20 years in parallel with obesity. It can be observed in all racial/ethnic groups. However, higher incidence rates have been reported in the USA, Canada, Australia, East/South Asia, and the Middle East [7]. T2DM is more common in the 10-19 age group and girls due to hormonal changes that occur during adolescence [31]. Studies have shown that β-cell function decreases by an average of 20-35% per year in cases of T2DM that develop at a young age. These data show substantially higher rates than the 7–11% annual decline rates in β-cell function that have been reported in adults with T2DM [7, 32]. In children with T2DM, β-cell function deteriorates faster than in adults, therefore complications occur earlier [33]. Similar to the literature, the majority of our patients with T2DM were female and were over the age of 10 years at the time of diagnosis [31]. Our patients had obesity, insulin resistance, acanthosis nigricans, and a family history of T2DM. No pathology was found in the screening for microvascular complications.

Two of our patients with monogenic diabetes had neonatal diabetes and the other three had MODY. The patient, who was diagnosed with diabetes in the neonatal period and had a heterozygous mutation in the ABCC8 gene, had TNDM. Oral treatments such as SU can be initiated in patients with KATP mutations. Since the genetic analysis result of our patient was delayed and the patient was in remission by the time the result was received, SU treatment could not be initiated. The follow-up of the patient in remission is continuing. SU treatment was beneficial in the uncle of the patient, who had the same mutation. Homozygous LRBA mutation was detected in the patient who presented with severe DKA at 5 months of age. LRBA deficiency can be accompanied by endocrine, hematological, autoimmune diseases and recurrent infections. No immunological abnormality has been observed in our patient, who is still receiving insulin treatment and being followed up in our clinic. However, the patient had a sibling who was diagnosed with diabetes in the neonatal period and died due to sepsis at the age of 5. The death was possibly associated with immunodeficiency, but there was no genetic diagnosis in this patient.

Heterozygous inactivating mutations in the GCK gene are a common cause of MODY. They cause slightly elevated HbA1c levels and a stable fasting hyperglycemia that does not require treatment. Patients requiring pharmacological treatment have been reported in the literature [34, 35]. Two of our patients with MODY had GCK-MODY. Plasma glucose regulation was achieved in one of the patients with diet only and with low-dose insulin treatment in the other patient. The mother of the patient who was receiving insulin treatment also had diabetes and was receiving insulin treatment, however, genetic analysis could not be performed in the mother. In patients with CEL-MODY, there is exocrine pancreatic dysfunction, which usually occurs in childhood. This is followed by diabetes in adulthood [36]. Our patient with CEL-MODY presented with hyperglycemia. Insulin treatment was initiated in the patient whose exocrine pancreas functions were normal. His mother also had diabetes, but genetic analysis could not be performed. The patient, in whom plasma glucose has been regulated with insulin treatment, is still being followed up in our clinic.

The small number of cases was a limitation of our study. Another limitation was that genetic analysis could not be performed in all cases considered to have monogenic diabetes.

In conclusion, although the majority of childhood diabetes cases are T1DM, the frequency of T2DM tends to increase, especially in obese adolescents. However, it should be kept in mind that autoantibody-positive patients with

T1DM may also have obesity. It was determined that T1DM cases were more common in the winter season, in the 10-14 age group, and the prevalence of DKA was high. Early diagnosis and treatment can reduce the risk of childhood complications such as DKA and cerebral edema. As observed in our cohort, which consisted of a small number of children and adolescents with diabetes, the presence of monogenic diabetes, the rate of which reaches up to 4%, should not be overlooked. It should always be kept in mind that the types of diabetes diagnosed in childhood and adolescence may be different. It should be taken into consideration that different types of diabetes cases may require different treatment options and therefore, different parameters may need to be scanned during follow-up.

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Authors contributions: A.A.K. and M.N.O. constructed the main idea and hypothesis of the study. A.A.K. and R.Y. developed the theory and arranged/edited the material and method section. A.A.K. and G.T. have evaluated the data in the results section. The discussion section of the article was written by A.A.K., A.A.K., and M.N.O. reviewed, corrected and approved. In addition, all authors discussed the entire study and approved the final version.

Conflict of interest: The authors declare that they have no conflict of interest.

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