

Trends and clinical features of childhood diabetes subgroups: 28 years of single center experience

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ABSTRACT

Objective. This study aimed to explore the distribution, trends, and clinical characteristics of various types of childhood diabetes, including type 1 diabetes (T1DM), type 2 diabetes (T2DM), and maturity-onset diabetes of the young (MODY) in a tertiary health center.

Methods. We conducted a comprehensive review of medical records of individuals aged 0–18 years who were diagnosed with diabetes between January 1996 and December 2023. Clinical and laboratory characteristics at the time of diagnosis, along with the specific diabetes type, were meticulously documented.

Results. A total of 1219 patients were included in the study, of whom 48.4% were female, with a mean age at diagnosis of 9.1 ± 4.3 years. T1DM was diagnosed in 85.8% of patients, T2DM in 6.3%, clinical MODY in 5.2%, and rare forms of diabetes in 2.6%. An increasing trend in T2DM and MODY cases has been observed since 2007. Diabetic ketoacidosis (DKA) was most prevalent in T1DM (47.1%), followed by T2DM (5.2%) and MODY (1.6%). Mean C-peptide levels at diagnosis were 0.57 ± 0.5 ng/mL in T1DM, 3.2 ± 1.3 ng/mL in T2DM, and 1.4 ± 0.9 ng/mL in MODY. Antibody positivity was observed in 78.8% of T1DM, 6.5% of T2DM, and 15.9% of MODY cases. Among the MODY group, genetic analysis was performed in 48 (75%) patients, with GCK gene mutations identified as the most common genetic abnormality in 27 (56.2%) of these patients.

Conclusion. This study demonstrates that T1DM is still the most commonly diagnosed type of diabetes in childhood, while T2DM and MODY are less frequent. However, a temporal increase in the incidence of MODY and T2DM subtypes was observed. The incidence of DKA at diagnosis was significantly higher in T1DM patients compared with those diagnosed with MODY or T2DM.

Key words: type 1 diabetes, type 2 diabetes, monogenic diabetes, maturity-onset diabetes of the young (MODY), childhood.

Recent trends have shown that the incidence of diabetes is increasing rapidly worldwide¹, and there has also been a dramatic increase in its incidence in Turkish children.^{2,3} Type 1 diabetes mellitus (T1DM) is the most common type of diabetes in children and continues to increase in different parts of the world.^{1,4-6}

Childhood obesity, which is increasing worldwide depending on ethnicity and country of residence, has been associated with a variable increase in the prevalence of type 2 diabetes (T2DM).⁷ The prevalence of T2DM in children has been reported to be 11% in the USA⁸, compared to 1.3% in Europe.⁹ In six

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regions of the USA, the prevalence of diabetes in children and adolescents has been reported to be increasing significantly for both type 1 and type 2 diabetes.¹⁰

Childhood T2DM can be confused with maturity-onset diabetes of the young (MODY) due to family history, presentation and a possible confounding factor of obesity/overweight.¹¹ MODY can also be misclassified as T1DM, particularly due to *HNF1A* mutations.¹² Determining the type of diabetes is important for therapeutic evaluation and genetic counselling.¹³ The most common type of monogenic diabetes is MODY, a clinically and genetically heterogeneous group of endocrine disorders affecting 1-5% of patients with diabetes.¹⁴ The increasing availability of molecular diagnostics has made it possible to identify other types of monogenic diabetes in addition to type 1 and type 2 diabetes with polygenic inheritance. In this classification, the concept of monogenic diabetes includes MODY, mitochondrial diabetes, Wolfram syndrome, neonatal diabetes and specific syndromes due to genetic defects causing insulin resistance.¹⁵

Until about 15 years ago, the distinction between T1DM, T2DM and MODY was somewhat simpler. However, with the increase in genetic studies and the definition of associations, it has become clear that these distinctions are not clear-cut. It has shown us that there are intertwined forms of diabetes. In daily practice, the differential diagnosis includes clinical findings, laboratory tests and diabetes-associated autoantibodies. This study will discuss these parameters in the differential diagnosis of diabetes.

The aims of this study were: (1) to evaluate the aetiological distribution and temporal changes of childhood diabetes, (2) to compare the clinical and laboratory characteristics of different types of diabetes seen in the pediatric diabetes centre of a tertiary care hospital during the last 28 years. The aim was also to contribute to the differential diagnosis of childhood diabetes subgroups.

Materials and Methods

The study was approved by the Ethics Committee of İnönü University (approval no: 2024/5706). Data of children and adolescents aged 0-18 years who were diagnosed with diabetes mellitus at İnönü University Turgut Özal Medical Center Training and Research Hospital in Malatya, Türkiye during the 28-year period between January 1996 and December 2023 were analyzed. Patients with a follow-up period of less than one year and those with undetermined diabetes type due to insufficient data were excluded from the study. In addition, because we could not clearly determine the number of patients who developed drug-associated diabetes, these patients were also excluded from the study. The observational and retrospective study included 1219 patients.

Sex, age at diagnosis, height, weight, body mass index (BMI), glucose, insulin, HbA1c, C-peptide levels at diagnosis, presence of symptoms, presence of diabetes-associated autoantibodies (islet cell antibodies, glutamic acid decarboxylase antibodies and insulin autoantibodies), presence of ketone bodies, pH at diagnosis and type of diabetes were recorded. The islet cell antibody (ICA), insulin autoantibody (IAA), and glutamic acid decarboxylase (GAD) levels were measured based on antigen-antibody detection using enzyme-linked immunosorbent assays with the Isletest kit in the Seac Brio 410499 model instrument. Patients were classified according to the ISPAD Consensus 2022 (Table I).¹⁶ Clinical findings were queried as polyuria, polydipsia, polyphagia and weight loss, and patients with one of these four clinical findings were considered symptomatic. Standard deviation scores (SDS) for height, weight, and BMI were calculated based on reference data from healthy Turkish boys and girls.¹⁷

T1DM was diagnosed based on the presence of severe insulin deficiency, diabetes-associated autoantibodies positivity, and the absence of any clinical signs suggestive of alternative causes of diabetes. The diagnostic criteria for

T2DM included overweight or obesity, clinical features of insulin resistance (such as acanthosis nigricans, hypertension, and dyslipidemia), a family history of T2DM, and good metabolic control achieved with metformin alone, or with metformin combined with a low dose of long-acting insulin (<0.5 U/kg/day).^{7,15,16} Patients with a family history of diabetes for at least two generations on one side of the family, negative diabetes-associated autoantibodies, no evidence of insulin resistance and good metabolic control with diet, sulfonylurea or low-dose insulin were clinically classified as MODY. *HNF1A*, *HNF4A*, *GCK* and other genes have been analysed in clinically suspected cases of MODY, and a MODY panel has been studied in recent years. To differentiate between mutations and polymorphisms among the detected variants, segregation analyses were conducted within families. In silico analysis tools, including Provean, SIFT (Sorting Intolerant From Tolerant), PolyPhen, Franklin, and MutationTaster, were employed to evaluate the potential pathogenicity of the variants. In accordance with the 2015 ACMG guidelines¹⁸, variants were classified as “pathogenic”, “likely pathogenic”, “variant of uncertain significance (VUS)”, “likely benign” or “benign”. Individuals carrying variants classified as “pathogenic” or “likely pathogenic” based on in silico analyses were considered to have monogenic diabetes. Children with onset of diabetes before the age of six months were diagnosed with neonatal diabetes mellitus (NDM) and appropriate genetic testing was performed.

Statistical analysis

All statistical data were analysed using SPSS statistical software for Windows, version 21.0 (SPSS, Chicago, IL, USA). Variables were calculated using descriptive statistics. Data were presented as mean $\pm$ standard deviation (SD) or median (interquartile range, IQR, Q1-Q3). Normality was assessed using the Kolmogorov-Smirnov test. Parametric and non-

parametric tests were used for comparisons between groups. The chi-square test was used for categorical variables. Student's t-test was used for continuous variables in independent groups. Mann-Whitney U test was used for continuous variables that were not normally distributed. In statistical analyses, $p < 0.05$ is the accepted threshold for significance.

Results

The mean age at diagnosis of 1219 patients (48.4% female) was 9.1 ± 4.3 (range 0.0-18.0) years. 23.1% (n=242) of patients with T1DM were younger than 5 years. 1046 patients were diagnosed with T1DM (85.81%), 77 patients with T2DM (6.31%), 64 patients with MODY (5.25%) and 32 patients with other types of diabetes (2.62%) (Table I).

Table I. Distribution of diabetic patients according to subgroups

	n	%*
Type 1 diabetes mellitus	1046	85.81
Type 2 diabetes mellitus	77	6.31
MODY	64	5.25
Neonatal DM	7	0.57
Wolfram syndrome	5	0.41
Diseases of the exocrine pancreas	4	0.33
Alström syndrome	3	0.24
Mitochondrial DM	3	0.24
Genetic defect in insulin action	2	0.16
TRMA	2	0.16
Subtotal pancreatectomy**	2	0.16
IPEX syndrome	1	0.08
Woodhouse-Sakati syndrome	1	0.08
Prader-Willi syndrome	1	0.08
Generalized lipodystrophy	1	0.08
Total	1219	100

*Column percentages.

**The development of diabetes following surgery for hyperinsulinemic hypoglycemia.
DM: diabetes mellitus; IPEX: immune dysregulation, polyendocrinopathy, enteropathy, X-linked; MODY: maturity onset diabetes of the young, TRMA: thiamine-responsive megaloblastic anemia.

Seven of the cases were definitively diagnosed with NDM. During follow-up, four cases were classified as transient NDM, while three cases were confirmed as permanent NDM. The clinical characteristics at diagnosis of T1DM, T2DM and MODY are compared in Table II. The median age at diagnosis was 8.6 years (IQR: 5.1-12.0) for T1DM, 15 years (12.6-16.2) for T2DM, and 10.9 years (7.2-13.0) for MODY. A single case of T2DM diagnosed before the age of 10 years was identified. This patient, a 9.8-year-old girl at the time of diagnosis, also presented with

precocious puberty. Positivity for at least one diabetes-associated autoantibody was 78.8% in T1DM, 6.5% in T2DM and 15.9% in MODY. IAA positivity before the age of 5 years was significantly higher in T1DM cases than > 5 years (41.1% vs. 32.6%, $p=0.045$).

T2DM and MODY cases started to be seen in the cohort from 2007 onwards, this increasing trend over time is shown in Fig. 1. T1DM, on the other hand, seemed to increase over time, but the number of type 1 diabetes cases

Table II. A comparative analysis of metabolic and clinical characteristics of patients diagnosed with type 1 diabetes, type 2 diabetes, and maturity-onset diabetes of the young at time of diagnosis.

	n*	T1DM	T2DM	MODY	T1DM vs T2DM, p	T1DM vs MODY, p	T2DM vs MODY, p
Sex (F/M), n	1187	504/542	46/31	25/39	0.050 ^a	0.156 ^a	0.014 ^a
Age at diagnosis (year)	1187	8.6 (5.1-12.0)	15 (12.6-16.2)	10.9 (7.2-13.0)	<0.001 ^c	0.003 ^c	<0.001 ^c
Height SDS	336**	0.3 (-0.4-1.2)	0.2 (0.0-0.7)	-0.2 (-0.7-0.6)	0.890 ^c	0.001 ^c	0.001 ^c
Weight SDS	336**	-0.3 (-1.1-0.5)	2.1 (1.4-2.5)	-0.2 (-1.0-1.0)	<0.001 ^c	0.280 ^c	<0.001 ^c
BMI SDS	336**	-0.5 (-1.5-0.1)	2.0 (1.6-2.3)	-0.2 (-1.2-1.1)	<0.001 ^c	0.013 ^c	<0.001 ^c
BMI SDS ≥2	336**	4 (2.0%)	43 (58.1%)	5 (8.1%)	<0.001 ^a	0.023 ^a	<0.001 ^a
BMI SDS <2	336**	27 (13.6%)	0	6 (9.7%)	0.001 ^a	0.414 ^a	<0.001 ^a
Presence of symptoms	898	718 (93.7%)	26 (37.1%)	22 (35.4%)	<0.001 ^a	<0.001 ^a	0.811 ^a
Parental consanguinity	1040	413 (39.4%)	17 (23.9%)	25 (39.1%)	<0.001 ^a	0.289 ^a	<0.001 ^a
Age <5 years	1187	242 (23.1%)	0	7 (10.9%)	<0.001 ^a	0.073 ^a	0.001 ^a
Fasting glucose (mg/dL)	1040	479±182	184±92	185±172	<0.001 ^b	<0.001 ^b	0.992 ^b
Insulin (μU/mL)	1040	1.8 (1.1-3.4)	20.4 (12.1-31.0)	5.2 (2.8-9.3)	<0.001 ^c	<0.001 ^c	<0.001 ^c
C-peptide (ng/mL)	1040	0.57±0.5	3.2±1.3	1.4±0.9	<0.001 ^b	<0.001 ^b	<0.001 ^b
HbA1c (%)	1040	12.2±2.6	8.1±2.3	7.6±2.7	<0.001 ^b	<0.001 ^b	0.258 ^b
pH	891	7.24±0.15	7.37±0.04	7.38±0.25	<0.001 ^b	<0.001 ^b	0.684 ^b
DKA	891	354 (47.1%)	4 (5.2%)	1 (1.6%)	<0.001 ^a	<0.001 ^a	0.663 ^a
Ketosis	891	290 (38.6%)	10 (13.1%)	8 (12.5%)	<0.001 ^a	<0.001 ^a	0.876 ^a
Hyperglycemia	891	107 (14.2%)	62 (81.5%)	55 (85.9%)	<0.001 ^a	<0.001 ^a	0.506 ^a
Antibody positivity	831	546 (78.8%)	5 (6.5%)	10 (15.9%)	<0.001 ^a	<0.001 ^a	0.066 ^a
Anti-GAD positivity	831	419 (60.4%)	5 (6.7%)	10 (15.9%)	<0.001 ^a	<0.001 ^a	0.073 ^a
ICA positivity	831	309 (44.5%)	0	3 (4.8%)	<0.001 ^a	<0.001 ^a	0.093 ^a
IAA positivity	831	240 (34.6%)	0	1 (1.6%)	<0.001 ^a	<0.001 ^a	0.457 ^a

Data were presented as n (%), mean±standard deviation, or median (Q1-Q3).

*The n values represent the number of patients for whom data was available.

**T1DM: 198, T2DM: 74, MODY: 62.

^aChi-square test. ^bIndependent samples t test. ^cMann-Whitney U test. In statistical analyses, $p<0.05$ is the accepted threshold for significance.

BMI: body mass index, DKA: diabetic ketoacidosis, F: female, GAD: glutamic acid decarboxylase, HbA1c: hemoglobin A1c, IAA: insulin autoantibody, ICA: islet cell antibody, M: male, MODY: maturity onset diabetes of the young, SDS: standard deviation score, T1DM: type 1 diabetes mellitus, T2DM: type 2 diabetes mellitus.

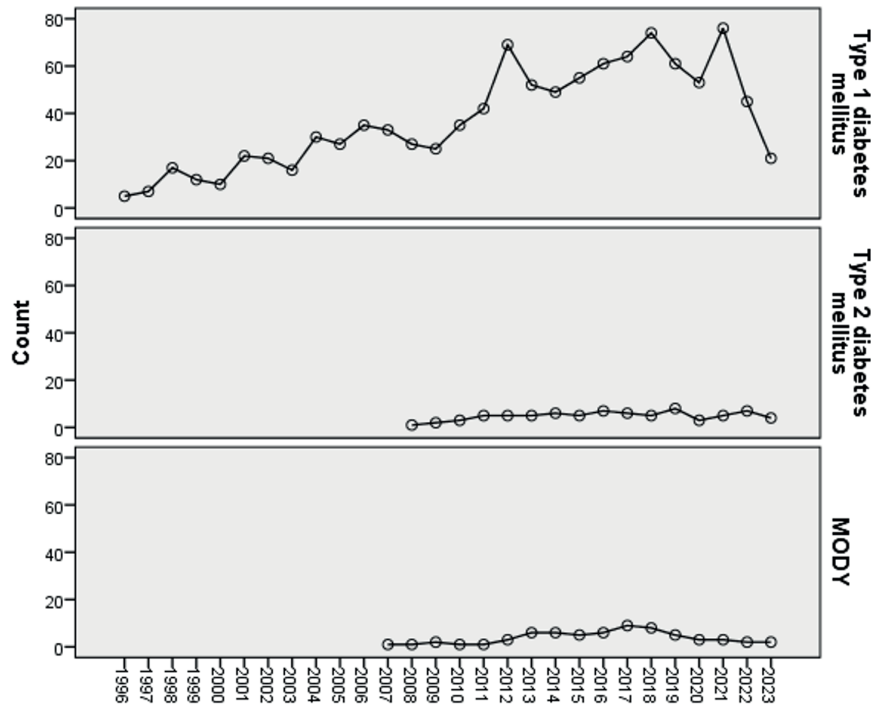


Fig. 1. Changes in the number of newly diagnosed patients with diabetes subgroups over the years. The steep decline in the number of cases diagnosed with type 1 diabetes mellitus in 2023 may be attributable to the migration of some of the population out of Malatya after the devastating twin Kahramanmaraş earthquakes in February 2023.

MODY: maturity onset diabetes of the young.

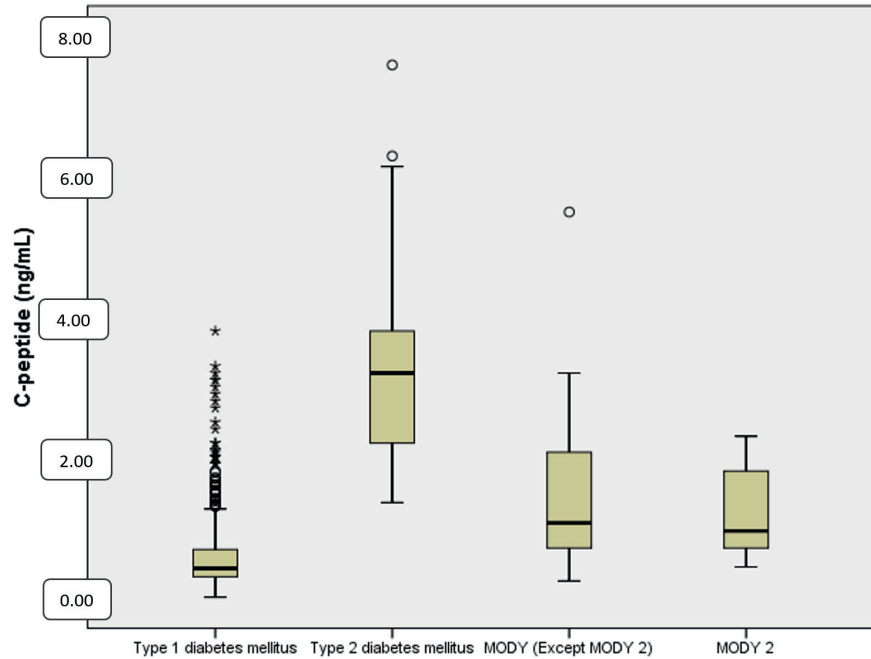


Fig. 2. C-peptide values of patients with diabetes at diagnosis.

MODY: maturity onset diabetes of the young.

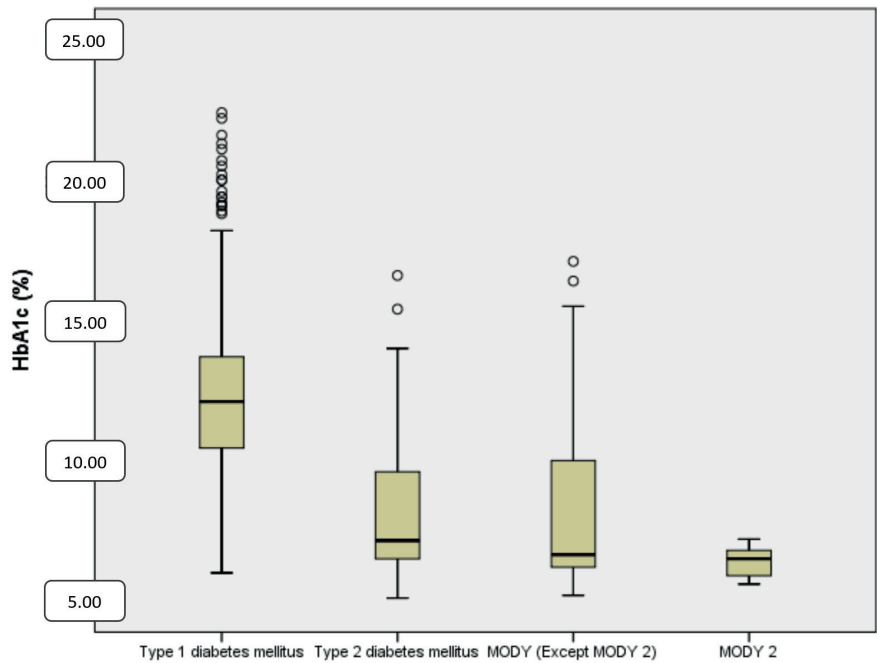


Fig. 3. HbA1c values of patients with diabetes at diagnosis.
MODY: maturity onset diabetes of the young.

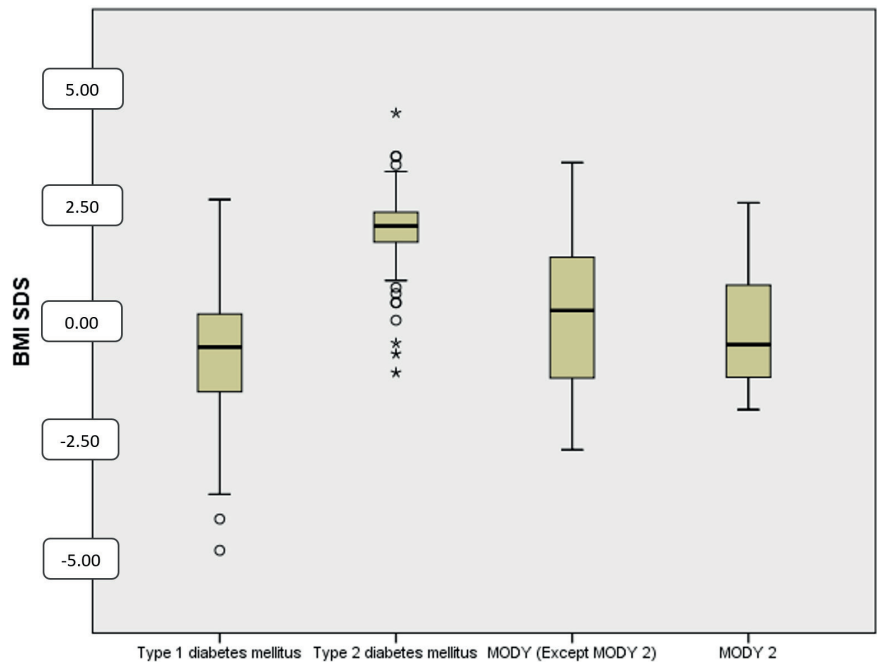


Fig. 4. BMI SDS values of patients with diabetes at diagnosis.
BMI: body mass index, MODY: maturity onset diabetes of the young, SDS: standard deviation score.

decreased in 2023. The prevalence of diabetic ketoacidosis (DKA) in T1DM at diagnosis was 47.1%, 47.8% in patients <5 years and 44.8% in patients >5 years ($p=0.486$). The prevalence of DKA decreased to 61.5% between 1996-2002, 51.0% between 2003-2009 and 32.9% between 2010-2016. However, it increased again between 2017 and 2023, reaching 41.2%. At the time of diagnosis, 62 (81.6%) patients with T2DM had hyperglycemia, ten (13.2%) had ketosis and only four (5.2%) had DKA (Table I).

Mean C-peptide levels at diagnosis were 0.57 ± 0.5 ng/mL in T1DM, 3.2 ± 1.3 ng/mL in T2DM and 1.4 ± 0.9 ng/mL in MODY patients ($p<0.001$) (Table II and Fig. 2). The mean HbA1c level at diagnosis was $12.2\% \pm 2.6\%$ in T1DM, $8.1\% \pm 2.3\%$ in T2DM and $7.6\% \pm 2.7\%$ in MODY patients. HbA1c levels were significantly higher in T1DM ($p<0.001$) (Table II and Fig. 3).

The median BMI-SDS at diagnosis was -0.5 (-1.5 - 0.1) in T1DM, 2.0 (1.6 - 2.3) in T2DM, and -0.2 (-1.2 - 1.1) in MODY patients. It was found to be significantly higher in T2DM ($p<0.001$). Obesity was present 2% of T1DM, 10.9% of MODY and 93.7% of T2DM patients. Malnutrition was found in 13.6% of T1DM patients, 9.7% of MODY patients and was absent in T2DM patients (Table II and Fig. 4). Genetic analysis was available in 48 (75%) of 64 clinical MODY patients, and GCK mutations were detected in 27 (56.2%) patients.

Discussion

In the present study, we analysed the prevalence of diabetes subgroups in our population and these data allowed us to make comparisons with the literature. The overall prevalence of T1DM, T2DM, MODY and other types of diabetes was 85.81%, 6.31%, 5.25% and 2.62%, respectively. T1DM is still the most common cause of diabetes in children and its prevalence varies from 83-95% in different parts of the World.^{5,6,8,9,12,19} This difference is due to the number of children with T2DM and MODY. In the SEARCH study (USA, multicenter), the

prevalences of T1DM, T2DM and MODY were 85.6%, 10.8% and 1.2%, respectively, whereas in the SWEET study (48 participating centers, 37 from Europe) these rates were 95.5%, 1.3% and 1.5%, respectively.^{9,20} In the SEARCH studies, the burden of T1DM was highest among non-Hispanic White youth, whereas the burden of T2DM was greatest among minority youth, particularly among American Indian and Alaska Native populations.^{8,20} The variation in frequencies may be explained by the accessibility of genetic testing and also by the prevalence of obesity in this region. In this study, the diagnoses of T2DM (6.3%) and MODY (5.2%) were more common than in SWEET. As this was not a national multicentre study, it is thought that this difference may be explained by the referral of rare types of diabetes to our tertiary care centre and easier access to genetic testing.

The country with the highest incidence of T1DM in childhood is Finland, followed by Sweden. Recent studies show an increasing trend in the Arabian Gulf countries and Türkiye.¹⁻⁶ Previously, we found an annual increase of 8.3% in the incidence of T1DM in our centre.² The incidence of T2DM, thought to be rare in children, is gradually increasing, and in parallel, the incidence of obesity in children is increasing, making it difficult to clinically differentiate between different types of diabetes.^{5,6} In studies of MODY in children, misdiagnosis of type 1 or type 2 diabetes has been observed.⁵ Recently, the number of cases diagnosed with MODY has been increasing with increasing awareness and molecular diagnostic capabilities.

The most important and accessible data in the differential diagnosis of diabetes subgroups are clinical findings (polyuria, polydipsia, polyphagia, and weight loss), glucose, insulin, C-peptide, HbA1c and the presence of diabetes-associated autoantibodies. Polyuria, polydipsia, polyphagia and weight loss are more suggestive of T1DM, but can also be seen in MODY.²¹ Clinical findings indicate the severity of insulin deficiency.²¹ In our study, the clinical findings were 93.7% in T1DM, 37.1% in T2DM and 35.4%

in MODY. In T1DM, clinical findings are the most important.

One of the most confusing clinical findings in diabetes classification is obesity. At the time of diagnosis, BMI is considered to be a less determinant feature in classification. This is because the increase in obesity has led to the emergence of obese children with T1DM and MODY.²² Although different studies have used different criteria, the prevalence of obesity at the time of diagnosis in T1DM patients varies from 3.1% to 9%.^{11,12} In this study, obesity was found in 2.0% of patients with T1DM. This may be due to the lower rates of obesity in our pediatric population compared to North America and Western Europe.²³ Consistent with previous reports, T2DM is more common in girls and in adolescents.^{14,15,23} In this study, the youngest documented case of T2DM was that of a 9.8-year-old girl who presented with precocious puberty. No additional cases of T2DM were identified in children under the age of 10 years, the mean age at diagnosis was 14.4 years, and almost all cases were in the adolescent age group. MODY can occur at any age.

While glucose and HbA1c are very effective in diagnosis, they are not very helpful in differential diagnosis. While HbA1c is generally below 7.5% in MODY 2 and 8-9% in other MODYs, this rate is above 10% in type 1 diabetes.²¹ Similar results were found in our study; in general, HbA1c is higher in T1DM. In diagnosis, C-peptide level is more valuable than insulin level in demonstrating reserve, and a C-peptide level >1.2 ng/mL in diagnosis suggests T2DM or MODY.²⁴ In our study, mean C-peptide level was 0.57 ng/mL and significantly low in T1DM, high in T2DM and moderate in MODY. In this study, there was little overlap in C-peptide levels at the time of diagnosis, especially in T1DM and T2DM, which helped us to differentiate these two types of diabetes. It also led us to distinguish T2DM from MODY, albeit weakly.

Antibody positivity in T2DM has been reported up to 15% and these antibody positive patients

tend to be younger, less overweight/obese and have higher HbA1c levels.²⁵ Therefore, different terms such as type 1.5 diabetes, double diabetes and occult autoimmune juvenile diabetes have been used. In the present study, antibody positivity in T2DM was found to be 6.5%. In T2DM patients aged 10 years and older, antibody positivity was reported in 9.8% in the TODAY study²⁶ and 21.2% in the SEARCH study.²⁷ The diagnosis of this group of seropositive patients is controversial. Studies have shown antibody positivity in 1% of people with MODY.²⁰ In the present study, 15.9% of our MODY patients had at least one antibody positivity. We were unable to explain the high antibody positivity observed in MODY patients. Therefore, in this study, antibody positivity was not used as an exclusion criterion for MODY.

In studies conducted in developed countries, the prevalence of DKA at the time of diagnosis of type 1 diabetes is reported to be 20-43%, whereas in the Arabian Peninsula these rates can be as high as 80%.^{28,29} In different studies conducted in Türkiye, the prevalence of DKA in T1DM at diagnosis varies between 44% and 65%.^{6,30-33} In our study, the rate of DKA was 47.1% and the current rate is still very high. The prevalence of DKA in pediatric T2DM at the time of diagnosis is variable, ranging from 4% to 40%.^{6,25,34} This rate was not high in our study (5.2%). The rate of DKA was lower in our MODY patients (1.6%). Ketosis without acidosis was found in 13% of our T2DM patients and 12.5% of our MODY patients. More than 80% of patients with T2DM and MODY were diagnosed after detection of hyperglycemia without ketosis or diabetic ketoacidosis. The most likely reason for this is that our centre is a tertiary care centre and we perform oral glucose tolerance test in all obese children with risk factors.

The frequency of MODY in children with diabetes varies from 1% to 6% in different studies.^{6,9,12,21} In studies reporting a higher frequency of MODY, GCK mutation is the most common cause.^{6,12,21} Similarly, we found a GCK mutation in 56.2% of genetically tested clinical MODY patients, which may be explained

by the widespread use of random glucose measurement in general pediatric clinics in Türkiye. On the other hand, since genetic analysis was not available before 2010 and not all MODY genes could be analysed, the rate of genetic analysis in clinical MODY patients in this study was relatively low (75%). A mutation in one of the known MODY genes was found in 75% of these patients. This rate varies between 27-89% in different studies.^{6,21,35} The reasons why not all patients in our study received a genetic diagnosis may be due to the inability to detect mutations, inability to perform a MODY gene panel in all patients, inclusion criteria for genetic testing, mutation in a gene not yet identified, or diagnostic overlap of different types of diabetes.

Study limitations

This study has some limitations; (1) data from a tertiary care centre, (2) specifically, prior to 2010, the diagnosis of MODY was only made clinically, (3) not all patients with MODY after 2010 could be interviewed, (4) the frequency of some specific types of diabetes such as cystic fibrosis-related diabetes (CFRD) may not be the actual frequency, (5) cases with drug-induced diabetes could not be included in the study.

Conclusion

This study examines trends in pediatric diabetes over the past 28 years in a large patient population from a large city with access to a tertiary care diabetes centre. It aimed to identify the distinguishing characteristics of different types of diabetes in children. While the prevalence of T2DM is increasing among the paediatric population in Türkiye, it remains lower than reported rates in North America and Western Europe. In recent years, the recognition of MODY has increased, largely due to the wider availability of autoantibody testing and, more importantly, genetic testing. Although overlapping clinical features—such as obesity, ketosis, and autoantibody positivity—can complicate the differential diagnosis, demographic (e.g., age, sex, pubertal status,

consanguinity) and laboratory parameters (e.g., autoantibody measurements, C-peptide levels, HbA1c) are valuable tools for accurately classifying diabetes types in the paediatric population.

Ethical approval

The study was approved by the local Ethical Committee of İnönü University (approval no: 2024/5706).

Author contribution

The authors confirm contribution to the paper as follows: Study conception and design: İD, AA; data collection: İD, EK, ZYY; analysis and interpretation of results: İD, EÇ; draft manuscript preparation: İD, AA, EÇ, EK, ZYY. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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