

Original article

Trends and characteristics of childhood diabetes in a tertiary care center in Thailand

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Background: Reports on characteristics of pediatric diabetes in children from Southeast Asian countries are limited.

Objectives: To describe the clinical characteristics, prevalence, glycemic control, and current treatment regimens of diabetes in Thai children.

Materials and Methods: Data from 132 patients seen at our pediatric diabetes clinic at Chulalongkorn University during 2001–2013 were retrospectively reviewed.

Results: We found an increasing number of patients newly-diagnosed with type 1- (T1DM) or type 2- diabetes mellitus (T2DM). The overall proportion of T1DM was 69.7%, T2DM 23.4%, and other types 6.9%. Children with T1DM were younger at diagnosis, had higher initial glucose and glycated hemoglobin A_{1c} (HbA_{1c}), a lower body mass index z-score, lower C-peptide and insulin levels, and were more likely to have classic diabetes symptoms and ketoacidosis, compared with children with T2DM. Mixed diabetes phenotypes were found in about 12%–14% of these children. Glutamic acid decarboxylase and islet antigen-2 autoantibodies were found in 70% and 54% of T1DM patients, respectively, and not in T2DM patients. HbA_{1c} in T1DM was $9.6 \pm 2.2\%$ total hemoglobin, and in T2DM was $7.9 \pm 2.6\%$. There were no differences in HbA_{1c} levels between different insulin regimens in the T1DM group.

Conclusion: The number of children with T1DM or T2DM has been increasing and there are overlapping phenotypes in a significant proportion of these children. Correct diagnosis requires clinical evaluation and monitoring of the clinical course. Further research is needed to determine the risk factors for the poor glycemic control found in children with T1DM.

Keywords: Characteristics, children, diabetes mellitus, trend

Diabetes mellitus (DM) is a common metabolic disease in childhood [1]. Recent epidemiological studies show that the incidence of both type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM) has been increasing worldwide [2-4]. A previous study in 2006 demonstrates that T1DM is still the most common form of diabetes in Thai children and adolescents [5], although the incidence of T1DM in Thailand is relatively low compared with studies conducted of patients living in Western countries [6, 7].

The prevalence of obesity in most Asian countries has increased dramatically [8], with the epidemic

particularly affecting children and adolescents who comprise the growing part of the population [9]. A study in 2006 demonstrated that Thailand had the highest rate of obesity in Asia at 6.8% of adults [8]. A national survey in 2011 reported a prevalence of 7.6% overweight and 9% obesity among Thai children [10]. Many reports from various countries show increasing numbers and proportion of T2DM in the pediatric population, along with increasing childhood obesity [4, 11, 12].

Accurate diagnosis of the type of diabetes is crucial for long-term clinical management. There are increasing numbers of children who have mixed phenotypes of both T1DM and T2DM [13, 14]. However, to date there are limited studies describing the clinical and biochemical phenotypes associated with T1DM and T2DM in children from Southeast Asia. There have been no reports on trends and

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clinical characteristics of childhood diabetes in Thailand since 2007 [15]. Therefore, the aims of this study were to (1) evaluate trends and clinical characteristics of childhood diabetes in our center; and (2) evaluate the current treatment regimens and glycemic control among the presented patients.

Materials and methods

Patients

The study protocol was reviewed and approved by the Institutional Review Board of the Faculty of Medicine, Chulalongkorn University. We retrospectively reviewed the medical records of all diabetic patients who had been regularly followed-up at our pediatric diabetes clinic at King Chulalongkorn Memorial Hospital during 2001–2013. This study excluded patients with steroid-induced diabetes mellitus. A total of 132 patients (65 boys and 67 girls, aged from 1 month to 17 years) were included. Our study is a retrospective chart review and was conducted under the ethical principles as presented in the Belmont Report—respect for persons, beneficence, and justice. All the information was recorded by the investigator in such manner that patients cannot be identified, directly, or through identifiers linked to the patients (unlinked anonymization). The policies and regulations of the institutional review board of the Faculty of Medicine, Chulalongkorn University, do not usually require written informed consent for a retrospective chart review study and follow principles set by the Declaration of Helsinki.

Data collected from the medical charts included age, sex, onset characteristics, including clinical and biochemical variables, signs and symptoms, comorbidities, type of diabetes, and details of diabetes management. Data regarding glycemic control were obtained from the most recent visit by the patients.

Body mass index (BMI) was calculated as weight (Wt) (kg)/height² (m²). Wt-standard deviation scores (SDS) and BMI-SDS were calculated based on World Health Organization (WHO) standards.

Terms and definitions

Diagnosis and type of diabetes

The diagnosis of DM and diabetes type was based on the American Diabetes Association (ADA) 2013 classification [16]. The diabetes type was specified by the pediatric endocrinologist, and reported as T1DM, T2DM, maturity-onset diabetes of the young (MODY), neonatal diabetes, or other types.

Overweight and pubertal status

Overweight was defined as a BMI >85th percentile for age and sex based on WHO growth charts. Pubertal status was stratified into (1) prepuberty, if the patient was at Tanner stage I (2) early puberty, if the patient was at Tanner stage II–III, and (3) late puberty, if the patient was at Tanner stage IV–V, at initial diagnosis.

Goals of glycemic control for age

Goals for glycemic control for age in this study were based on the ADA 2013 guidelines [16]. For young children aged 0–5 years, the targeted glycated hemoglobin A_{1c} (HbA_{1c}) is <8.5% total hemoglobin (69.4 mmol/mol); school-aged children 6–12 years, <8% (63.9 mmol/mol); and adolescents and young adults aged 13–19 years <7.5% (58.5 mmol/mol).

Comorbidities

The patients were defined to have comorbidities by definitions described below.

1. Hypertension: blood pressure (either systolic or diastolic) >95th percentile for age, sex, and height.
2. Nephropathy: microalbuminuria (urine albumin/urine creatinine ratio >30 µg albumin/mg creatinine).
3. Dyslipidemia: serum low-density lipoprotein cholesterol ≥130 mg/dL or triglyceride levels ≥150 mg/dL.
4. Retinopathy: as examined and recorded by ophthalmological consultation.
5. Neuropathy: by performing physical examination at routine follow-up visits.

Biochemical analyses

Commercial immunoassays were used to measure insulin, C-peptide levels (Electrochemiluminescence Immunoassay (ECLIA); Diagnostic Products Corporation, Los Angeles, CA, USA), and HbA_{1c} levels (Immunoturbidity (nephelometry); Roche Diagnostics, Indianapolis, IN, USA). Glucose was measured on a Cobas Integra 400 plus instrument (Roche Diagnostics) using a hexokinase method. Liver enzymes, lipids, and uric acid levels were measured by standard enzymatic methods. Anti-glutamic acid decarboxylase 65 kDa isoform (GAD65) and anti-islet antigen-2 (IA-2) autoantibodies were measured using enzyme-linked immunosorbent assays (ELISAs) (RSR, Pentwyn, Cardiff, United Kingdom).

Statistical analyses

Data are expressed as numbers (percentage) or the mean \pm standard deviations (SD). Comparisons between two groups were analyzed by a chi-square test for nominal data, and a Student *t* test or analysis of variance (ANOVA) for continuous data. All statistical analyses were performed using SPSS version 18.0 (SPSS, Chicago, IL). $P < 0.05$ was considered significant.

Results

Trend and etiology of childhood diabetes

A total of 132 patients (65 boys, 67 girls, age range 1 month – 17 years) who had been regularly followed-up at our pediatric diabetes clinic during 2001–2013 were included in this study. Among 132 patients, there were 102 participants (78 had T1DM and 24 had T2DM) being newly-diagnosed with diabetes during this study period. Demographic data, the yearly-trend of diabetes type, and T1DM/T2DM ratio were shown in **Table 1** and **Figure 1**. Of 132 patients, 92 were diagnosed with T1DM (70%), 31 were diagnosed with T2DM (23%), 3 patients were diagnosed with MODY (2%), 3 were diagnosed with neonatal diabetes (2%), and 3 patients were diagnosed with other types (2%).

Clinical characteristics at onset

The patients with T1DM had lower mean age at diagnosis than T2DM patients (9.4 ± 4 years vs. 12.6 ± 2 years, $P < 0.001$). Children with T1DM had a lower BMI-SDS compared to children with T2DM (-0.4 ± 1.7 vs. 2.7 ± 1.8 , $P < 0.001$). Overweight at initial diagnosis was found in 27 patients in the T2DM group (87%), and 13 patients (14%) in the T1DM group ($P < 0.001$). Acanthosis nigricans was found in 20 of 30 patients with T2DM (67%), and in 8 (12%) of 67 patients with T1DM. Onset data of pubertal staging were available in only 75 patients. Most patients with T2DM (79%) had already entered puberty, whereas 44% of patients with T1DM were pubertal, but the difference was not significant.

Presenting symptoms (Table 2)

Fifty-eight patients (63%) in the T1DM group and 3 patients (10%) in the T2DM group had diabetic ketoacidosis (DKA) as an initial presentation ($P < 0.001$). Four patients with T1DM group (4%) and 14 patients with T2DM (52%) were mildly symptomatic or asymptomatic at diagnosis ($P < 0.001$). Classic symptoms such as polyphagia, polydipsia, polyuria, or weight loss were more common in children with T1DM (76%) than in those with T2DM (48.4%) ($P = 0.001$).

Table 1. Baseline demographic and onset characteristics

	T1DM	T2DM	MODY	Neonatal	Other	<i>P</i>
Total numbers of cases	92 (70%)	31 (23%)	3 (2%)	3 (2%)	3 (2%)	
Female: male (n)	43:49	18:13	2:1	1:2	3:0	0.305
Mean age at diagnosis (y)	9.4 ± 4	12.6 ± 2	12.2 ± 4	1.0 ± 0.5 months	1.4 ± 2	<0.001
Age group (y)						
<5	15 (16%)	0	0	3 (100%)	3 (100%)	
5–10	32 (35%)	3 (10%)	2 (67%)	0	0	
>10	45 (49%)	28 (90%)	1 (33%)	0	0	
Mean BMI z-score	-0.4 ± 1.7	2.7 ± 1.8	0.03 ± 2.6	-0.3 ± 0.3	-0.5 ± 0.4	<0.001
Overweight at diagnosis	13 (14%)	27 (87%)	1 (33%)	0	0	<0.001
Acanthosis nigricans*	8/67 (12%)	20/30 (67%)	0	0	0	<0.001
Pubertal staging*						0.07
Prepuberty	28/50 (56%)	4/19 (21%)	0	3 (100%)	2/2 (100%)	
Early puberty	15/50 (30%)	9/19 (47%)	1/1 (100%)	0	0	
Late puberty	7/50 (14%)	6/19 (32%)	0	0	0	

Data are expressed as mean \pm SD or n (%). *Onset data available for 106 patients (presence of acanthosis nigricans); and 75 patients (pubertal staging). BMI = body mass index, T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus, MODY = maturity-onset diabetes of the young, Neonatal = neonatal diabetes mellitus, Prepuberty = Tanner stage I, Early puberty = Tanner stage II–III, Late puberty = Tanner stage IV–V at initial diagnosis.

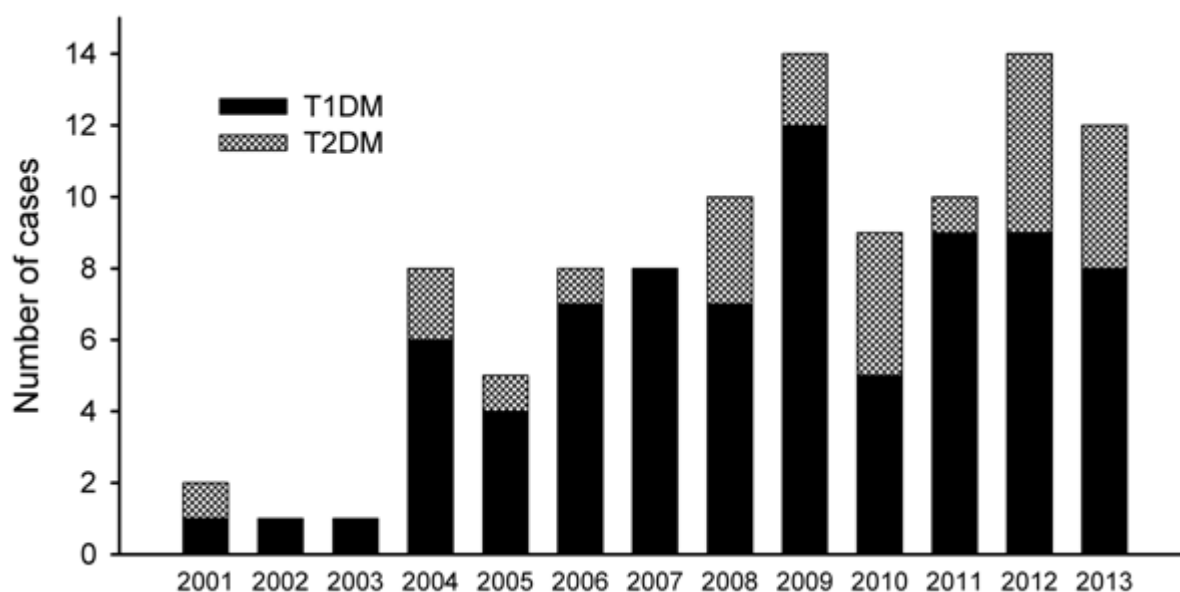


Figure 1. Trends of childhood diabetes (newly-diagnosed cases) during 2001-2013
T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus.

Table 2. Presenting symptoms by diabetes type

Presenting symptoms	T1DM n = 92	T2DM n = 31	MODY n = 3	Neonatal n = 3	Other n = 3	P
Ketoacidosis	58 (63%)	3 (10%)	0	3 (100%)	0	<0.001
Classic symptoms	70 (76%)	15 (48%)	1 (33%)	0	0	0.001
Mildly symptomatic or asymptomatic	4 (4%)	14 (52%)	2 (67%)	0	3 (100%)	<0.001

T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus, MODY = maturity-onset diabetes of the young, Neonatal = neonatal diabetes mellitus

Initial laboratory data (Table 3)

Patients with T1DM had higher mean initial blood glucose (517 ± 228 vs. 292 ± 199 mg/dL, $P < 0.001$), and higher mean initial HbA_{1c} levels than patients with T2DM ($13.1 \pm 2.6\%$ vs. $10.0 \pm 3.1\%$ total hemoglobin, $P < 0.001$). Of 57 patients with T1DM, anti-GAD65 and anti-IA-2 autoimmune antibodies were found

in 40 (70%) and 30 (53%) patients, respectively. Of 16 patients with T2DM, none had antibodies to GAD65 or IA-2. Insulin levels at diagnosis were significantly lower in patients with T1DM than in patients with T2DM (3.9 ± 5.0 vs. 15.9 ± 10.2 μ IU/mL, $P < 0.001$), and similarly lower C-peptide levels were found (1.4 ± 1.6 vs. 3.6 ± 2.9 ng/mL, $P < 0.001$).

Table 3. Initial laboratory data of type 1- and type 2-diabetes mellitus patients

Laboratory data	T1DM (n = 92)	T2DM (n = 31)	P
Initial glucose (mg/dL)	517 ± 228	292 ± 199	<0.001
Initial HbA _{1c} (% total hemoglobin)	13.1 ± 2.6	10.0 ± 3.1	<0.001
Pancreatic autoantibodies*			
Anti-GAD65 positive	40/57 (70%)	0	<0.001
Anti-IA-2 positive	30/57 (53%)	0	0.002
C-peptide levels (ng/mL)	$1.4 (\pm 1.6)$	$3.6 (\pm 2.9)$	<0.001
Insulin levels (μ IU/mL)	$3.9 (\pm 5)$	$15.9 (\pm 10.2)$	<0.001

Data are expressed as mean \pm SD or n (%) or % total hemoglobin. *Pancreatic autoantibody data available for 57 patients with T1DM, and 16 patients with T2DM. HbA_{1c} = glycated hemoglobin A_{1c}, T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus, GAD65 = glutamic acid decarboxylase 65 kDa isoform, IA-2 = islet antigen-2 (ICA512).

Glycemic control and current treatment regimens

Based on the laboratory findings during the most recent follow-up visits, T2DM patients had lower mean fasting blood glucose levels than T1DM patients (132 ± 63 vs. 202 ± 72 mg/dL, $P < 0.001$). A one-way ANOVA revealed significant differences in mean HbA_{1c} levels between the different types of diabetes ($P < 0.001$) (Figure 2). Bonferroni post hoc testing demonstrated that differences in HbA_{1c} levels existed between the groups of patients with T1DM or T2DM ($9.6 \pm 2.2\%$ vs. $7.9 \pm 2.6\%$ total hemoglobin, $P = 0.002$), but not between the other types. However, patients with MODY or neonatal diabetes tended to have more favorable glycemic control (Figure 2). The percentage of patients who achieved targeted HbA_{1c} levels according to the ADA statement was higher in the T2DM group than in the T1DM group [17 of 31 patients (55%) vs. 22 of 92 patients (24%), $P = 0.001$] (Table 4).

Treatment regimens differed between subgroups. Among patients in T1DM group, 36 patients (39%) were on basal-bolus insulin regimen with 4 or more daily injections, 25 patients (27%) were on twice-daily injections with premixed insulin, 19 patients (21%) were on twice-daily injections with split-mixed human insulin (regular insulin/neutral protamine Hagedorn insulin = RI/NPH), 5 patients (5%) were on three daily insulin injections (modified conventional regimen), and 3 patients (3%) used an insulin pump. There were

no significant differences in HbA_{1c} levels or percentage of patients who achieved targeted HbA_{1c} adjusted for age among different insulin regimens (Table 4). Among children with T1DM, we found that patients between 0–5 years of age had the best glycemic control, while patients in the older age groups had less favorable control. In addition, patients with diabetes duration >4 years appeared to have better glycemic control (Table 5).

In the T2DM group, 17 patients were treated with metformin only, 9 patients were taking insulin and metformin, 4 patients were on diet control only, and only one patient was on basal-bolus insulin therapy. Mean HbA_{1c} levels in T2DM patients who had been taking insulin were significantly higher than those who were simply on diet control or taking metformin only (Table 4).

Comorbidities

Hypertension was found in 11 out of 92 patients (12%) with T1DM, and 13 out of 31 patients (42%) with T2DM ($P < 0.001$). Dyslipidemia was detected in 18 patients (20%) with T1DM, and 13 patients with T2DM (31 total) ($P = 0.006$). Microalbuminuria seemed to be more common in T2DM than in T1DM, but it was not significantly different. Retinopathy or neuropathy was not reported in any patient (Table 6).

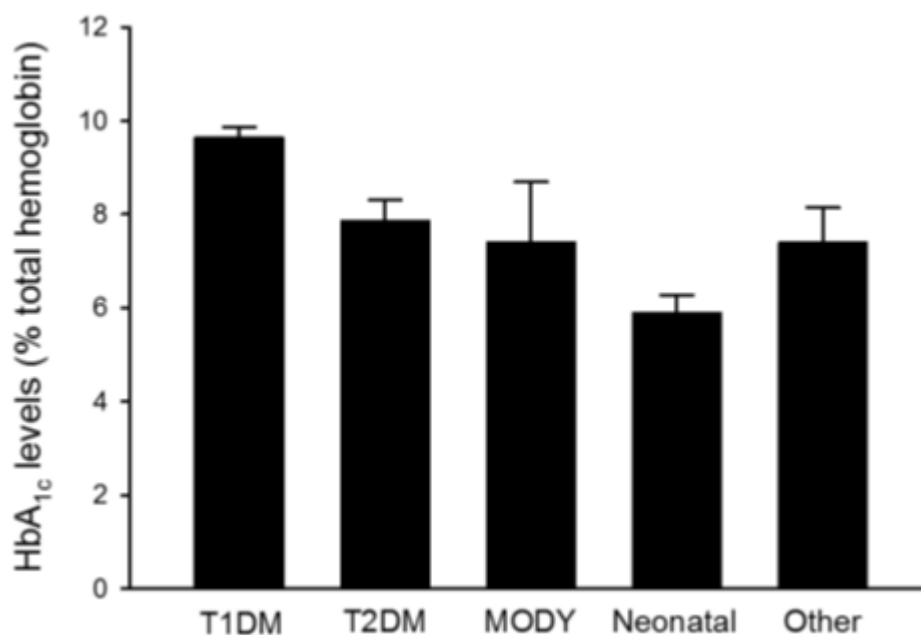


Figure 2. Mean glycated hemoglobin A_{1c} levels during recent follow-up visits by diabetes type T1DM = type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; MODY = maturity-onset diabetes of the young; Neonatal = neonatal diabetes mellitus; HbA_{1c} = glycated hemoglobin A_{1c}

Table 4. Mean glycated hemoglobin A_{1c} (HbA_{1c} % total hemoglobin) and numbers of patients achieving targeted HbA_{1c} levels by treatment regimen

	T1DM (n = 92)	
	Mean HbA _{1c} (± SD) %	Patients achieved targeted HbA _{1c}
Basal bolus insulin (n = 36)	9.4 ± 2.2	11
Conventional RI/NPH (n = 19)	10.9 ± 2.1	2
Conventional premixed (n = 25)	9.5 ± 2.2	6
Modified conventional (n = 5)	10.0 ± 3.1	1
Insulin + metformin (n = 4)	9.8 ± 3.0	1
Insulin pump (n = 3)	8.0 ± 1.1	1
	<i>P</i> = 0.189	<i>P</i> = 0.710
All patients with T1DM	9.6 ± 2.2*	22 (24%) ^α
	T2DM (n = 31)	
	Mean HbA _{1c} (± SD) %	Patients achieved targeted HbA _{1c}
Diet control (n = 4)	5.9 ± 0.7	4
Metformin (n = 17)	6.9 ± 2.0	12
Insulin + metformin (n = 9)	10.1 ± 2.5	1
Basal bolus insulin (n = 1)	10.3	0
	<i>P</i> = 0.002	<i>P</i> = 0.004
All patients with T2DM	7.9 ± 2.6*	17 (54.8%) ^α

**P* = 0.002 (post hoc Bonferroni test), ^α*P* = 0.001 (chi-square test). T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus, HbA_{1c} = glycated hemoglobin A_{1c}, RI/NPH = split-mixed human insulin (regular insulin/neutral protamine Hagedorn insulin)

Table 5. Mean glycated hemoglobin A_{1c} levels (HbA_{1c} % total hemoglobin) at recent follow-up visits by patient characteristics in the type 1 diabetes group (n = 92)

Characteristics	n (%)	Mean HbA _{1c} ± SD %	<i>P</i>
Sex			0.358
Male	49 (53%)	9.8 ± 2.2	
Female	43 (47%)	9.7 ± 2.4	
Age (y)			0.114
0–5	3 (3%)	7.7 ± 1.6	
6–12	29 (32%)	9.8 ± 1.9	
13–18	38 (41%)	10.2 ± 2.4	
>18	22 (24%)	9.1 ± 2.5	
Duration of treatment			0.023
<12 months	27 (29%)	10.3 ± 2.4	
1–4 y	24 (26%)	10.4 ± 2.4	
>4 y	41 (45%)	9.0 ± 2.0	

Table 6. Comorbidities in different types of childhood diabetes

	T1DM (n = 92)	T2DM (n = 31)	MODY (n = 3)	Neonatal DM (n = 3)	Other types (n = 3)
Hypertension	11 (12%)*	13 (42%)*	1	0	0
Microalbuminuria	10 (11%)	5 (16%)	2	0	0
Dyslipidemia	18 (20%)*	14 (45%)*	0	1	1
Retinopathy	0	0	0	0	0
Neuropathy	0	0	0	0	0

P < 0.05 (T1DM compared with T2DM), T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus, MODY = maturity-onset diabetes of the young.

Discussion

This study indicated that the number of newly-diagnosed patients with childhood diabetes in our center had been increasing for cases of T1DM or T2DM. The rising trends were similar to previous reports from various countries worldwide [3, 4, 17-19]. The overall ratio of T1DM:T2DM in this study was about 70:30, which was similar to previous findings in Thailand in 2007 [15], indicating that T1DM is still the predominate type of pediatric diabetes in Thailand.

The prevalence of childhood obesity in most Asian countries has increased rapidly [8-10]. It is often difficult to correctly identify diabetes type at the time of initial diagnosis [13, 14, 20]. Our findings demonstrated that onset characteristics more associated with T1DM included a lower age at diagnosis, the presence of classic DM symptoms or DKA, lower BMI z-score, higher initial blood glucose and HbA_{1c}, and lower C-peptide and insulin levels compared with T2DM. Interestingly, there remained a significant proportion of patients with a mixed phenotype such that 14% of patients with T1DM were also overweight, and 12% had acanthosis. Similarly, Lipton et al. found that 10% of patients had mixed diabetes phenotype, and no onset feature was completely reliable for predicting diabetes type. The authors suggested that careful monitoring of all children with diabetes is required to correctly determine eventual diabetes type [14].

We found that positivity for pancreatic autoantibodies was helpful to diagnose T1DM. However, of those of T1DM patients who received antibody testing in our study, anti-GAD65 and anti-IA2 were found in 70% and 53%, respectively, which is similar to previous findings in Thailand in 2007 [15]. The prevalence of islet-specific autoantibodies in the Thai population appears to be comparable to those found in people living in Western countries, and Japan [21-23]. By contrast, the prevalence of pancreatic antibody positivity in some Asian countries especially in Koreans or Chinese was lower than in Australians of European descent with T1DM [24, 25], suggesting that there are variations among different Asian ethnicities.

In this study, we found that the mean HbA_{1c} levels in patients with T1DM was 9.6%, and only about 24% of all children with T1DM in our center could achieve satisfactory glycemic control. The challenge in achieving targeted glycemic control in children with T1DM has been observed in many studies from

different countries [17, 26, 27]. We also found that there were no differences in HbA_{1c} levels or percentage of patients who achieved targeted HbA_{1c} between different insulin regimens, as consistent with findings in previous studies [26, 28]. Interestingly, the earlier Thai study in 2007 showed that only 8% of Thai children with T1DM were using basal-bolus regimens compared with about 39% of pediatric patients in the present study, but that the mean HbA_{1c} levels in our patients appeared to be higher (8.9% vs. 9.6%) [15]. Thus, the number of daily insulin injections and/or insulin types may not necessarily guarantee better glycemic control. Future studies to determine factors associated with glycemic control are crucial to improve the management of childhood diabetes in our population. Because this study was retrospective, some important data are missing, including the frequency of self-monitoring of blood glucose, psychosocial factors, and family support. These factors might contribute to glycemic control.

We found that patients with T2DM had better glycemic control than patients with T1DM. This finding is consistent with previous studies [15, 29]. Most children with T2DM who achieved optimal glycemic control were treated mainly with lifestyle interventions or metformin alone. By contrast, those with poorer glucose control were mostly treated with insulin, suggesting a greater degree of insulin resistance or worse β -cell function in this group.

The presence of complications and comorbidities in children with T1DM or T2DM in this series was comparable to the presence of complications and comorbidities found in other studies [17, 30]. Consistent with previous studies [17, 30, 31], children with T2DM had higher rates of hypertension and dyslipidemia than those with T1DM. Additionally, microalbuminuria appeared to be more prevalent in children with T2DM, compared with T1DM. Notably, the rate of progression to microalbuminuria seemed to be faster in children with T2DM [31], indicating early detection and intervention for this problem are crucial. Therefore, other than achieving targeted glucose control, interventions aimed at these comorbidities are essential to prevent long-term complications in children with diabetes.

In summary, we found that the number of children with T1DM or T2DM has been increasing. Although the prevalence of T2DM in children is increasing in Thailand, the overall prevalence remains low compared with T1DM. The clinical distinction between

T1DM and T2DM has become more challenging because of the overlapping clinical pictures in a significant proportion of children with diabetes. The correct diagnosis of diabetes type requires thorough clinical and laboratory evaluation, and careful monitoring of the clinical course. The overall glycemic control in children with T1DM in Thailand is considered poor. Further research is needed to determine the risk factors for poor glycemic control and prevention of acute and chronic complications, in the hope of assigning more appropriate management in children and adolescent patients with diabetes.

No authors have any conflict of interest to report.

References

1. Gortmaker S, Sappenfield W. Chronic childhood disorders: prevalence and impact. *Pediatr Clin North Am.* 1984; 31:3-18.
2. Smith TLS, Drum ML, Lipton RB. Incidence of childhood type 1 and non-type 1 diabetes in a diverse population: The Chicago Childhood Diabetes Registry, 1994 to 2003. *J Pediatr Endocrinol Metab.* 2007; 20:1093-107.
3. Patterson CC, Dahlquist GG, Gyurus E, Green A, Soltész G, and the EURODIAB study group. Incidence trends for childhood type 1 diabetes in Europe during 1989-2003 and predicted new cases 2005-2020: a multicentre prospective registration study. *Lancet.* 2009; 373:2027-33.
4. Pinhas-Hamiel O, Zeitler P. The global spread of type 2 diabetes mellitus in adolescent and children. *J Pediatr.* 2005; 146:693-700.
5. Likitmaskul S, Wacharasindhu S, Rawdaree P, Ngarmukos C, Deerochanawong C, Suwanwalaikorn S, et al. Thailand diabetes registry project: type of diabetes, glycemic control and prevalence of microvascular complications in children and adolescents with diabetes. *J Med Assoc Thai.* 2006; 89 (Suppl 1):S10-6.
6. Panamonta O, Laopaiboon M, Tuchinda C. Incidence of childhood type 1 (insulin dependent) diabetes mellitus in northeastern Thailand. *J Med Assoc Thai.* 2000; 83:821-4.
7. Tuchinda C, Likitmaskul S, Unachak K, Panamonta O, Patarakijavanich N, Chetthakul T. The epidemiology of type 1 diabetes in Thai children. *J Med Assoc Thai.* 2002; 85:648-52.
8. Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH, Ko SH, et al. Epidemic obesity and type 2 diabetes in Asia. *Lancet.* 2006; 368:1681-8.
9. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. *Int J Pediatr Obes.* 2006; 1: 11-25.
10. Jitnarin N, Kosulwat V, Rojroongwasinkul N, Boonpradern A, Haddock CK, Poston WS. Prevalence of overweight and obesity in Thai population: results of the National Thai Food Consumption Survey. *Eat Weight Disord.* 2011; 16:e242-9.
11. Likitmaskul S, Kiattisathavee P, Chaichanwatanakul K, Punnakanta L, Angsusingha K, Tuchinda C. Increasing prevalence of type 2 diabetes mellitus in Thai children and adolescents associated with increasing prevalence of obesity. *J Pediatr Endocrinol Metab.* 2003; 16:71-7.
12. Alberti G, Zimmet P, Shaw J, Bloomgarden Z, Kaufman F, Silink M. Type 2 diabetes in the young: the evolving epidemic: the international diabetes federation consensus workshop. *Diabetes Care.* 2004; 27: 1798-811.
13. Libman IM, Pietropaolo M, Arslanian SA, LaPorte RE, Becker DJ. Evidence for heterogeneous pathogenesis of insulin-treated diabetes in black and white children. *Diabetes Care.* 2003; 26:2876-82.
14. Lipton RB, Drum ML, Danielson KK, Greeley SA, Bell IG, Hogopian WA. Onset features and subsequent clinical evolution of childhood diabetes over several years. *Pediatric Diabetes.* 2011; 12:326-34.
15. Santiprabhob J, Weerakulwattana P, Nunloi S, Kiattisakthavee P, Wongarn R, Wekawanich J, et al. Etiology and glycemic control among Thai children and adolescents with diabetes mellitus. *J Med Assoc Thai.* 2007; 90:1608-15.
16. American Diabetes Association. Standards of medical care in diabetes 2013. *Diabetes Care.* 2013; 36 (Suppl 1):S11-66.
17. Liu LL, Yi JP, Beyer J, Mayer-Davis EJ, Dolan ML, Dabelea DM, et al. Type 1 and type 2 diabetes in Asian and Pacific Islander U.S. youth: the SEARCH for diabetes in youth study. *Diabetes Care.* 2009; 32 (Suppl 2):S133-40.
18. Variation and trends in incidence of childhood diabetes in Europe. EURODIAB ACE Study Group. *Lancet.* 2000; 355:873-6.
19. DIAMOND Project Group. Incidence and trends of childhood Type 1 diabetes worldwide 1990-1999. *Diabet Med.* 2006; 23:857-66.
20. Hyponen E, Virtanen SM, Kenward MG, Knip M, Akerblom HK. The Childhood Diabetes in Finland Study Group. Obesity, increased linear growth, and risk of type 1 diabetes in children. *Diabetes Care.* 2000;

- 23:1755-60.
21. Feeney SJ, Myers MA, Mackay IR, Zimmet PZ, Howard N, Verge CF, et al. Evaluation of ICA512As in combination with other islet cell autoantibodies at the onset of IDDM. *Diabetes Care*. 1997; 20:1403-7.
22. Libman IM, Pietropaolo M, Trucco M, Dorman JS, LaPorte RE, Becker D. Islet cell autoimmunity in white and black children and adolescents with IDDM. *Diabetes Care*. 1998; 21:1824-7.
23. Yokota I, Matsuda J, Naito E, Ito M, Shima K, Kuroda Y. Comparison of GAD and ICA512/IA-2 antibodies at and after the onset of IDDM. *Diabetes Care*. 1998; 21:49-52.
24. Tuomi T, Zimmet P, Rowley MJ, Min HK, Vichayanrat A, Lee HK, et al. Differing frequency of autoantibodies to glutamic acid decarboxylase among Koreans, Thais, and Australians with diabetes mellitus. *Clin Immunol Immunopathol*. 1995; 74:202-6.
25. Thai AC, Ng WY, Loke KY, Lee WR, Lui KF, Cheah JS. Anti-GAD antibodies in Chinese patients with youth and adult-onset IDDM and NIDDM. *Diabetologia*. 1997; 40:1425-30.
26. Rosilio M, Cotton JB, Wieliczko MC, Gendraul B, Carel JC, Couvaras O, et al. Factors associated with glycemic control. A cross-sectional nationwide study in 2,579 French children with type 1 diabetes. The French Pediatric Diabetes Group. *Diabetes Care*. 1998; 21:1146-53.
27. Mortensen HB, Hougaard P. Comparison of metabolic control in a cross-sectional study of 2,873 children and adolescents with IDDM from 18 countries. The Hvidøre Study Group on Childhood Diabetes. *Diabetes Care*. 1997; 20:714-20.
28. Dorchy H, Roggemans MP, Willems D. Glycated hemoglobin and related factors in diabetic children and adolescents under 18 years of age: a Belgian experience. *Diabetes Care*. 1997; 20:2-6.
29. Copeland KC, Zeitler P, Geffner M, Guandalini C, Higgins J, Hirst K, et al. TODAY Study Group. Characteristics of adolescents and youth with recent-onset type 2 diabetes: the TODAY cohort at baseline. *J Clin Endocrinol Metab*. 2011; 96:159-67.
30. Maahs DM, Snively BM, Bell RA, Dolan L, Hirsch I, Imperatore G, et al. Higher prevalence of elevated albumin excretion in youth with type 2 than type 1 diabetes: the SEARCH for Diabetes in Youth study. *Diabetes Care*. 2007; 30:2593-8.
31. Pinhas-Hamiel O, Zeitler P. Acute and chronic complications of type 2 diabetes mellitus in children and adolescents. *Lancet*. 2007; 369:1823-31.