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The onset of puberty in adolescents with type 1 diabetes mellitus in comparison to a group of non-diabetic teenagers in Erbil City: A cross-sectional comparative study

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Abstract

Background and objective: Puberty and type 1 diabetes (T1DM) have a complex relationship. This study aims to determine the prevalence of delayed puberty among a sample of type 1 diabetes adolescents and compare it to a non-diabetic group.

Methods: A cross-sectional comparative study design was used to compare the data between January 1st, 2024, and July 31, 2024, included 380 participants, a convenient sample of 134 type 1 diabetic adolescents aged 9-16 years who were enrolled in Galyawa Specialized Diabetic Center and outpatient endocrinology at Rapareen Teaching Hospital in Erbil City, and 246 healthy adolescents in the same age range who attended Brayaty and Shady primary health care. Pubertal stage was determined by Tanner criteria. Anthropometry, BMI Z-score, and HbA1c were recorded.

Results: Mean age was 13.3 ± 1.7 years in both groups. Delayed puberty (Tanner stage < II at ≥13 y in girls or ≥14 y in boys) occurred in 53.7 % of diabetics versus 11.8 % of non-diabetics (P <0.001). The prevalence of delayed puberty among diabetic girls and boys (65.7% and 40.6%) was significantly higher than among the non-diabetic group (13.4% and 9.3%). Diabetic adolescents had significantly lower height-for-age Z-scores (< –2 SD) than non-diabetics (P <0.001). HbA1c level was not associated with pubertal delay.

Conclusion: more than half of the diabetics had delayed puberty, but there was no significant relationship between HbA1c and delayed puberty. Individuals with type 1 diabetes are shorter than non-diabetics. This study raised the importance of that the health practitioners, adolescents with type 1 diabetes and their caregivers should be informed about the possible outcomes of type 1 diabetes in adolescents.

Keywords: Delayed puberty, Puberty, Type 1 diabetes mellitus.

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Introduction

Diabetes is a chronic illness that develops when the body either cannot use the insulin that the pancreas makes properly or does not create enough of it. Hyperglycemia is a frequent consequence of uncontrolled diabetes mellitus that eventually causes major harm to numerous bodily systems, particularly the blood vessels and neurons (1).

Type 1 diabetes mellitus (T1DM) is an autoimmune disease that causes complete insulin insufficiency, due to the autoimmune destruction of pancreatic beta cells, which causes hyperglycemia and the symptoms that accompany it. It is the most prevalent kind of diabetes in children and is characterized by the four Ts: tired, thirsty, thinner, and going to the toilet (2, 3). Although genetics play an important role in developing T1DM, environmental factors trigger the developing of T1DM in genetically susceptible individuals (4). Children with T1DM may exhibit the following signs and symptoms: elevated blood sugar, glycosuria, polyuria, polydipsia, unexpected weight reduction, general malaise, and acute ketoacidosis symptoms (5).

Puberty is a delicate stage of human development, a stage in behavioral and physical development that occurs between childhood and maturity, it is a unique stage of sexual development that takes place in adolescence (6).

The release of sex hormones, testosterone in boys and estrogen in girls, marks the beginning of puberty, the child's blood sugar rises because of these hormones, cortisol and other stress hormones might also rise during this period, these substances can change a child's bodily cells, resulting in a decreased ability to utilize insulin (7).

As the hormones generated by the hypothalamus, pituitary, and sex glands, regulate pubertal development, blood glucose levels in individuals with T1DM can influence these hormones' synthesis, which in turn can impact pubertal development and cause pubertal delay (8). Detection and management of such delays in development are relevant to clinical care and long-term outcomes (1).

This study addresses the impact of type 1 diabetes mellitus (T1DM) on pubertal development, a critical yet underexplored area. It finds important risk factors including lifestyle and glycemic management by contrasting T1DM patients with their healthy counterparts. To avoid developmental delays, the results can direct early treatments. This study offers important new information about adolescent health and the treatment of childhood diabetes. The objectives of this study are to find out the prevalence of delayed puberty among T1DM children and compare it with the prevalence among non-diabetic children and to find the factors that are affecting delayed

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puberty among type 1 diabetic adolescents.

PATIENTS AND METHODS

Study setting

The cross sectional comparative study was conducted in Erbil City, Iraq. A total of 380 adolescents aged 9 to 16 years participated: 134 with type 1 diabetes mellitus (T1DM) and 246 participants of 9 to 16 year of non-diabetic adolescents that were asked about any signs and symptoms of T1DM who were healthy visited primary health centers for other reasons.

Study design and duration

Across-sectional comparative study design was carried out from a period of January 1, 2024, to July 31, 2024.

Study population and sampling

A total of 380 participants were included in the study, consisting of 134 diabetics and 246 healthy adolescents. A convenience sampling method was employed to recruit both groups based on their accessibility and willingness to participate during the study period.

Cases of 134 Patients with a confirmed diagnosis of type 1 diabetes, aged between 9 and 16 years, were taken from the outpatient clinics of Galyawa Diabetic Center and the endocrinology clinic at Rapareen Teaching Hospital. These institutions serve as specialized referral centers for pediatric diabetes in Erbil, ensuring access to a broad population of type 1 diabetes patients.

A group of 246 adolescent individuals without T1DM were recruited from the Brayati and Shady Primary Health Care Centers in Erbil. These participants visited the centers for reasons unrelated to diabetes, such as routine checkups, minor illnesses, or immunization services. All controls were screened to exclude diabetes and other chronic or endocrine conditions that could affect pubertal development.

This sampling approach ensured a representative comparison between diabetic and non-diabetic adolescents

Inclusion criteria

1- Any patients withT1DM, aged 9 to 16 years, minimum of 1 year since diagnosis to allow for stable assessment of glycemic control, who visited Galyawa Specialized Diabetic Center and Rapareen Teaching Hospital.

2- Comparative group of any person without T1DM of the same age group (9-16 years), who visited Brayaty and Shady primary health center for other reasons.

Exclusion criteria

Those with other forms of diabetes, endocrine disorders or any chronic diseases impacting puberty, history of congenital disorders or developmental delay. Current use of medications affecting growth or puberty.

Ethical consideration

A formal consent letter from the director of Health in Erbil governorate

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was obtained, and the protocol was accepted by the research ethics committee of the executive office of the Arab Board of Health Specializations just before the study's commencement. Before allowing them to participate in the study and before asking them to complete the questionnaire, the researcher acquired consent from each patient and assured them about the privacy and confidentiality of the data collected, each patient received an explanation of the purpose and scope of the study.

Data collection and study tools

The patients' information was gathered through in-person interviews. During the interview, after taking consent, the researcher used both Arabic and Kurdish languages to communicate with the patient. Without directly specifying the participant's name, the replies were written in English on the questionnaire paper using a code number. The official consent form, structured closed-ended questionnaires, and information gathering through face-to-face interviews, asked each participant patient face-to-face questions about socio-demographics and puberty in the samples.

Following data collection and questionnaire completion, all patients were asked to determine their BMI, if they were diabetics, and to provide a fasting blood sample under a septic technique to determine their HbA1c and fasting blood sugar.

T1DM diagnosis was confirmed per WHO diagnostic criteria, including raised fasting blood glucose (≥126 mg/dL) or HbA1c (≥6.5%) (9).

Puberty was evaluated using Tanner staging (10). (Sexual Maturity Rating), in a private room setting after getting their consent, which measured breast development in girls and testicular size in boys. Delayed puberty was defined as the absence of secondary sexual characteristics by the age of 13 years in girls and no testicular enlargement >2.5 cm at 14 years in boys, according to traditional endocrinological criteria (11).

Height and weight were measured, and the WHO body mass index (BMI) for age was calculated (12). Glycemic control was assessed using HbA1c and was categorized as <7% (well-controlled), 7–8.9% (moderately controlled), and ≥9% (poorly controlled) (13).

Statistical analysis

Statistical analysis was done using SPSS version 26. Descriptive statistics confirmed demographic variables. Logistic regression analysis was used where the dependent variable was binary categorical (delayed puberty). Variables found (by univariate analysis) to be significantly associated with the dependent variable were entered into the regression model as independent variables.

Chi-square and Fisher's exact tests checked associations of delayed puberty

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with risk factors. A P-value of <0.05 was considered statistically significant.

Results

The total population of the study was 380. Their mean age (SD) was 13.3 (1.7) years, the median was 13, and the age range was 9–16 years. The study population were two groups, a group of diabetic children (n = 134), and a non-diabetic group of 246 non-diabetic children.

More than half (57.6%) of the sample were females, but the difference was insignificant between the groups (P = 0.116). More than three-quarters (76.7%) of the non-diabetic group were in secondary schools, compared with 54.5% of the diabetics (P <0.001). The majority (77.1%) of children were living in urban areas, and the difference was insignificant between the groups (P = 0.106). The prevalence of obesity was 7.9%, but the difference was not significant (P = 0.556), and 17.4% were of high SES, but the difference was insignificant between the groups (P = 0.608), as presented in Table 1.

Table 1. Basic characteristics of the study groups

	Diabetics No. (%)	Non-diabetics No. (%)	Total No. (%)	P-value
Age (years)				
9-11	38 (28.4)	39 (15.9)	77 (20.3)	
12-14	46 (34.3)	144 (58.5)	190 (50.0)	
15-16	50 (37.3)	63 (25.6)	113 (29.7)	<0.001*
Mean (SD)	13.3 (2.2)	13.4 (1.7)	13.3 (1.7)	0.565**
Sex				
Male	64 (47.8)	97 (39.4)	161 (42.4)	
Female	70 (52.2)	149 (60.6)	219 (57.6)	0.116*
School grade				
Primary	61 (45.5)	57 (23.3)	118 (31.1)	
Secondary	73 (54.5)	188 (76.7)	261 (68.9)	<0.001*
Residency				
Rural	37 (27.6)	50 (20.3)	87 (22.9)	
Urban	97 (72.4)	196 (79.7)	293 (77.1)	0.106*
BMI Z scores				
Thinness (<-2 SD)	6 (4.5)	9 (3.7)	15 (3.9)	
Normal (-2 to 1 SD)	92 (68.7)	160 (65.0)	252 (66.3)	
Overweight (>1 SD)	24 (17.9)	59 (24.0)	83 (21.8)	
Obesity (>2 SD)	12 (9.0)	18 (7.3)	30 (7.9)	0.556*
Socio-economic status (SES)				
Low	54 (40.3)	91 (37.0)	145 (38.2)	
Medium	55 (41.0)	114 (46.3)	169 (44.5)	
High	25 (18.7)	41 (16.7)	66 (17.4)	0.608*
Total	134 (100.0)	246 (100.0)	380 (100.0)	

*Calculated by Chi-square test. **Calculated by unpaired t-test.

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More than half (53.7%) of the diabetics had delayed puberty, compared with 11.8% of the non-diabetic group (P <0.001). The prevalence of delayed puberty among diabetic girls (65.7%) was significantly (P <0.001) higher than the prevalence in the non-diabetic group (13.4%). The prevalence of delayed puberty among diabetic boys (40.6%) was significantly (P <0.001) higher than the prevalence among non-diabetics (9.3%), as presented in Table 2.

The rate of delayed puberty among female diabetics (65.7%) was significantly (P = 0.004) higher than that among male diabetics (40.6%). In the non-diabetic group, the rate among females was 13.4%, and among males was 9.3%, but the difference was insignificant (P = 0.325), as presented in Figure 1.

Table 2. Puberty state of the diabetic patients and of the controls

	Diabetics No. (%)	Non-diabetics No. (%)	Total No. (%)	P-value*
Puberty state (boys and girls)				
Compatible with age	62 (46.3)	217 (88.2)	279 (73.4)	
Delayed	72 (53.7)	29 (11.8)	101 (26.6)	<0.001
Total	134 (100.0)	246 (100.0)	380 (100.0)	
Girls' puberty state				
Compatible with age	24 (34.3)	129 (86.6)	153 (69.9)	
Delayed	46 (65.7)	20 (13.4)	66 (30.1)	<0.001
Total	70 (100.0)	149 (100.0)	219 (100.0)	
Boys' puberty state				
Compatible with age	38 (59.4)	88 (90.7)	126 (78.3)	
Delayed	26 (40.6)	9 (9.3)	35 (21.7)	<0.001
Total	64 (100.0)	97 (100.0)	161 (100.0)	

Calculated by the Chi-square test.

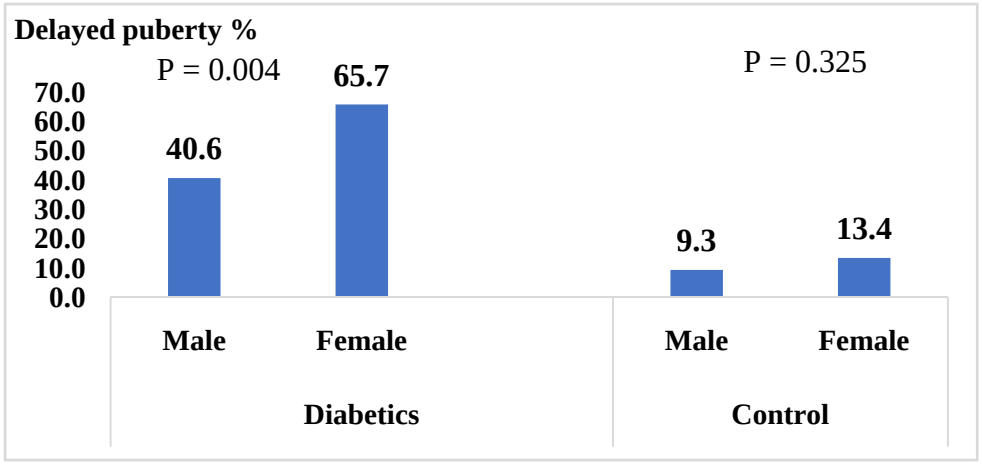

Figure 1. Delayed puberty rate by sex, in each of the study groups

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Table 3 showed no significant association between delayed puberty with the last HbA1c (P = 0.329), average HbA1c levels over the last year (P = 0.714), socio-economic status (P = 0.441), and time of going to bed (P = 0.887).

Table 3. Factors associated with delayed puberty among diabetics

	Puberty of boys and girls			
	Compatible with age No. (%)	**Delayed No. (%)**	**Total No. (%)**	**P-value**
Last HbA1c				
< 7	5 (62.5)	3 (37.5)	8 (100.0)	
7-8.9	35 (50.0)	35 (50.0)	70 (100.0)	
≥ 9	22 (39.3)	34 (60.7)	56 (100.0)	0.329**
Average HbA1c levels over the last year				
7-10	49 (47.1)	55 (52.9)	104 (100.0)	
>10	13 (43.3)	17 (56.7)	30 (100.0)	0.714*
BMI Z-Score				
Thin	5 (83.3)	1 (16.7)	6 (100.0)	
Normal	36 (39.1)	56 (60.9)	92 (100.0)	
Overweight	12 (50.0)	12 (50.0)	24 (100.0)	
Obese	9 (75.0)	3 (25.0)	12 (100.0)	0.023**
Socio-economic status				
Low	22 (40.7)	32 (59.3)	54 (100.0)	
Medium	26 (47.3)	29 (52.7)	55 (100.0)	
High	14 (56.0)	11 (44.0)	25 (100.0)	0.441*
Do you have a regular sleep pattern?				
Yes	36 (57.1)	27 (42.9)	63 (100.0)	
No	26 (36.6)	45 (63.4)	71 (100.0)	0.017*
At what time do you usually go to bed?				
9-10 PM	1 (33.3)	2 (66.7)	3 (100.0)	
10-12 PM	39 (48.1)	42 (51.9)	81 (100.0)	
>12 AM	22 (44.0)	28 (56.0)	50 (100.0)	0.887**
Total	62 (46.3)	72 (53.7)	134 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test.

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BMI was significantly associated with delayed puberty among diabetics (P = 0.023), but this association was not consistent throughout the BMI categories, the rate of delayed puberty was low among thin children (16.7%) and obese children (25%), and it was 60.9% and 50% among normal weight and overweight children respectively, as presented in Table 4.

The only factor that was significantly associated with delayed puberty in diabetic girls was irregular sleeping patterns where 80.6% had delayed puberty compared with 50% of those with regular sleeping patterns (P = 0.007), no significant association was detected with the other factors mentioned in Table 5.

A significant association was detected between BMI and delayed puberty in diabetic boys (P = 0.006), but this association was inconsistent throughout the BMI categories, (Table 6).

Table 4. Factors associated with delayed puberty among the non-diabetic group children

	Puberty of boys and girls			
	Compatible with age No. (%)	**Delayed No. (%)**	**Total No. (%)**	**P-value**
BMI Z-Score				
Thin	7 (77.8)	2 (22.2)	9 (100.0)	
Normal	137 (85.6)	23 (14.4)	160 (100.0)	
Overweight	57 (96.6)	2 (11.1)	59 (100.0)	
Obese	16 (88.9)	2 (11.8)	18 (100.0)	0.060**
Total	217 (88.2)	29 (11.8)	246 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test.

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Table 5. Factors associated with delayed puberty of diabetic girls

	Girls' puberty status			
	Compatible with age No. (%)	Delayed No. (%)	Total No. (%)	P-value
Last HbA1c				
< 7	4 (66.7)	2 (33.3)	6 (100.0)	
7-8.9	10 (32.3)	21 (67.7)	31 (100.0)	
≥ 9	10 (30.3)	23 (69.7)	33 (100.0)	0.302**
Average HbA1c levels over the last year				
7-10	17 (32.1)	36 (67.9)	53 (100.0)	
>10	7 (41.2)	10 (58.8)	17 (100.0)	0.492*
BMI Z-Score				
Thin	4 (80.0)	1 (20.0)	5 (100.0)	
Normal	18 (34.6)	34 (65.4)	52 (100.0)	
Overweight	1 (11.1)	8 (88.9)	9 (100.0)	
Obese	1 (25.0)	3 (75.0)	4 (100.0)	0.070**
Socio-economic status				
Low	9 (30.0)	21 (70.0)	30 (100.0)	
Medium	11 (35.5)	20 (64.5)	31 (100.0)	
High	4 (44.4)	5 (55.6)	9 (100.0)	0.713*
Do you have a regular sleep pattern?				
Yes	17 (50.0)	17 (50.0)	34 (100.0)	
No	7 (19.4)	29 (80.6)	36 (100.0)	0.007*
At what time do you usually go to bed?				
9-10 PM	1 (33.3)	2 (66.7)	3 (100.0)	
10-12 PM	15 (34.9)	28 (65.1)	43 (100.0)	
>12 AM	8 (33.3)	16 (66.7)	24 (100.0)	1.000**
Total	24 (34.3)	46 (65.7)	70 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact te

Table 6. Factors associated with delayed puberty of diabetic boys

	Boys' puberty status			
	Compatible with age No. (%)	Delayed No. (%)	Total No. (%)	p-value
Last HbA1c				
< 7	1 (50.0)	1 (50.0)	2 (100.0)	
7-8.9	25 (64.1)	14 (35.9)	39 (100.0)	
≥ 9	12 (52.2)	11 (47.8)	23 (100.0)	0.578**
Average HbA1c levels over the last year				
7-10	32 (62.7)	19 (37.3)	51 (100.0)	
>10	6 (46.2)	7 (53.8)	13 (100.0)	0.277*
BMI Z-Score				
Thin	1 (100.0)	0 (0.0)	1 (100.0)	
Normal	18 (45.0)	22 (55.0)	40 (100.0)	
Overweight	11 (73.3)	4 (26.7)	15 (100.0)	
Obese	8 (100.0)	0 (0.0)	8 (100.0)	0.006**
Total	38 (59.4)	26 (40.6)	64 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test.

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No significant association was detected between the puberty status (of the whole sample) with the following factors: challenges facing diabetes that are mentioned in Table 7 (P = 0.709), doctor's dietary instructions (P = 0.531), physical activity (P = 0.702), and exposure to daily stressors (P = 0.763), as presented in Table 7.

Table 7. Delayed puberty of the diabetic sample by physical and psychological stressors

	Puberty status			
	Compatible with age No. (%)	**Delayed puberty No. (%)**	**Total No. (%)**	**P-value**
Challenges facing diabetes				
Medication non-compliance	11 (50.0)	11 (50.0)	22 (100.0)	
Peer influence	2 (100.0)	0 (0.0)	2 (100.0)	
Stress	3 (33.3)	6 (66.7)	9 (100.0)	
Changes toward an unhealthy lifestyle	26 (44.8)	32 (55.2)	58 (100.0)	
Peer influence & stress	1 (100.0)	0 (0.0)	1 (100.0)	
Peer influence & changes toward an unhealthy lifestyle	8 (50.0)	8 (50.0)	16 (100.0)	
Stress & changes toward an unhealthy lifestyle	11 (42.3)	15 (57.7)	26 (100.0)	0.709**
Do you follow dietary instructions from your doctor?				
Yes	39 (44.3)	49 (55.7)	88 (100.0)	
No	23 (50.0)	23 (50.0)	46 (100.0)	0.531*
Do you engage in moderately vigorous physical activity?				
Yes	27 (48.2)	29 (51.8)	56 (100.0)	
No	35 (44.9)	43 (55.1)	78 (100.0)	0.702*
Do you have a daily stressor?				
Yes	20 (44.4)	25 (55.6)	45 (100.0)	
No	42 (47.2)	47 (52.8)	89 (100.0)	0.763*
Total	62 (46.3)	72 (53.7)	134 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test.

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In Table 8, 13.4% of the diabetics had low Z scores (< -2 SD) for height for age, compared with 2.4% of the non-diabetics (P <0.001).

In Table 9, (multivariate) Regression analysis showed that diabetes is significantly associated with delayed puberty (OR = 10.84; 95% CI = 6.1–18.9) in female sex (OR = 2.1; 95% CI = 1.1–3.6).

Table 8. Height for age Z scores of the two study groups

Height for age Z scores	Diabetic No. (%)	Non-diabetic No. (%)	Total No. (%)	P-value*
< -3 SD	0 (0.0)	2 (0.8)	2 (0.5)	
< -2 SD	18 (13.4)	6 (2.4)	24 (6.3)	
-2 to 1 SD	111 (82.8)	201 (81.7)	312 (82.1)	
> 1 SD	3 (2.2)	34 (13.8)	37 (9.7)	
> 2 SD	2 (1.5)	3 (1.2)	5 (1.3)	<0.001
Total	134 (100.0)	246 (100.0)	380 (100.0)	

*Calculated by Fisher's exact test.

Table 9. SPSS output of binary logistic regression analysis between delayed puberty as a dependent variable with several covariates

				95% CI** of OR	
	B	P	OR*	Lower	Upper
Diabetes	2.384	< 0.001	10.848	6.195	18.995
Female sex	0.742	0.010	2.101	1.195	3.693
Irregular sleep pattern	0.446	0.113	1.562	0.899	2.714
BMI Z scores		0.056			
Thin (Reference)					
Normal weight	1.089	0.143	2.970	0.691	12.762
Over-weight	0.351	0.659	1.420	0.298	6.757
Obese	0.173	0.849	1.189	0.200	7.053
Constant	-3.677	0.000	0.025		

*OR: Odds ratio. **CI: Confidence interval.

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Discussion

The present study remarkably showed that more than half (53.7%) of the diabetics had delayed puberty, compared with 11.8% of the non-diabetic group. In comparison to another study in Morocco (8), they found that 25% of children with T1DM had a delayed onset of puberty.

In the current study, the prevalence of delayed puberty among diabetic girls (65.7%) was significantly higher than the prevalence among girls in the non-diabetic group (13.4%). Study conducted in Japan revealed that patients who had menarche after being diagnosed with T1DM experienced a delayed menarche (14). However, according to a different study conducted at the University of Chile in Santiago, Chile, girls with T1DM reach pubertal development at a normal age (15).

The pathophysiologic explanation for this is that pubertal development is regulated by hormones produced by the pituitary, sex glands, and hypothalamus. In girls with T1DM, blood glucose levels may impact the synthesis of these hormones, which can therefore affect pubertal development and cause pubertal delay (8).

These discrepancies may be due to differences in glycemic control, study design, healthcare access, and population characteristics. For example, children with better-controlled diabetes are less likely to experience pubertal delay, and studies using broader or less strict definitions of delayed puberty may report lower prevalence. Additionally, genetic, ethnic, and socioeconomic differences across populations—as well as access to modern insulin therapy—can all contribute to variations in findings. Understanding these contextual factors is essential when interpreting and comparing data across different countries and healthcare settings.

Compared to controls (9.3%), males with T1DM had a considerably higher prevalence of delayed puberty (40.6%). Previous research includes a lot of contradictions. On the other hand, the Santiago, Chile study revealed that there were no instances of pubertal delay and boys reached puberty at an age that was comparable to or even earlier than the control boys (15).

The findings of the present study may be explained by the likelihood that the GnRH axis, which limits the release of LH and delays puberty, is impacted by any significant insulin deficiency in boys with T1DM (15).

Glycemic control and delayed puberty did not significantly correlate in the current study, comparably, a study conducted in Brazil revealed no correlation between the age onset of puberty and the mean pre-pubertal HbA1c (16).

There is ongoing debate on the connection between obesity and

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puberty. While longitudinal and cross-sectional studies demonstrate a shift in obese girls toward earlier puberty, the trend in obese boys is less evident. In comparison to boys of a healthy weight, overweight boys mature earlier, and obese boys mature later (17).

This study showed a significant association between BMI and delayed puberty in diabetics (P = 0.023). However, this association did not hold true for other BMI categories, as the rate of delayed puberty varied widely between thin and obese children (16.7% and 25%, respectively), and between normal weight and overweight children 60.9% and 50%, respectively.

In a study conducted in Morocco by Boukaidi LY et al, there was no discernible association found between T1DM adolescents' BMI and puberty (8).

Furthermore, there is no correlation, according to our results, between the duration of type1 diabetes and delayed puberty. However, a Brazilian study found that a longer illness course is linked to a later puberty onset (18).

One explanation for the discrepancy between the research on delayed puberty and glycemic control could be the daily accumulation of advanced glycation end-products (AGE)s, which may impact other hormones, rather than just the HbA1C.

This study, like many others carried out in Iraq and the surrounding regions, found that, in comparison to 2.4% of the non-diabetics, 13.4% of the diabetics had poor Z scores (< -2 SD) for height for age. According to a study conducted in Baghdad, Iraq in 2021, 14.3% of T1DM individuals had lower stature than their counterparts (19).

Similarly, in another study conducted in Baghdad, Iraq in 2020, researchers discovered that diabetes adolescent diabetics continued to be significantly shorter than their classmates; the percentage of short children with diabetes was 19.68% (20).

One plausible explanation could be a reduction in insulin like growth factor-I (IGF-I) and impairment of linear development even with vigorous insulin therapy (21).

If raising an adolescent is already challenging, T1DM can increase stress and strain on all members of the family. Supporting adolescents as well as family education in this stage is crucial to prevent complications of T1DM and achieve a healthy passing of this stage of their life.

Limitations of the study

Finally, because of cultural tradition, it was difficult to check pubertal maturity using tanner staging in this age group, particularly for males since we would examine the genital area. Furthermore, it was challenging to locate T1DM cases in different centers; therefore, we collected data over several weeks from two specialized centers only. Recall bias may also be present, particularly

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if participants or their caregivers inaccurately reported the age of pubertal milestones such as menarche or testicular enlargement. Furthermore, observer bias could have influenced the clinical assessment of pubertal staging, as physical examination and subjective judgment were involved. Finally, caution should be used when interpreting the data because of the short study duration and the wherefores described reasons.

Conclusion

The present study remarkably showed that more than half of the diabetics had delayed puberty (more than two-thirds of girls and more than one-third of boys), the relationship between T1DM and puberty has dual directionality. Although the study showed a significant association between BMI and delayed puberty in diabetics, this association did not hold for BMI sub-categories, individuals with T1DM have short stature, and the linear growth of adolescents is affected.

Recommendation

The present study raised the importance of that the health practitioners need to be informed about the possible complications of T1DM in adolescents and T1DM adolescents and their caregivers should be informed about the possible outcomes of T1DM in adolescents. Patient education programs and family assistance during this time of life are necessary to ensure an excellent outcome and a healthy transition for all involved. Finally, larger study is needed to find out the complex relationship between T1DM and puberty.

Competing interest

The authors declare that they have no competing interests.

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