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The effect of diabetes mellitus on the growth of children and adolescents

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Abstract

Background: Children and teenagers with chronic illnesses often undergo different levels of developmental setbacks, affecting their eventual growth. It's worth mentioning that certain chronic conditions such as type 1 diabetes, celiac disease, and asthma have a peculiar tendency to appear during infancy and adolescence. This underscores the importance for healthcare strategists and providers to detect and proactively address growth issues in these at-risk groups.

Methods: A cross-sectional descriptive study was conducted from 1st of February to 1st of June 2023. This descriptive study included 392 patients, who were divided into two groups. The first group is uncontrolled diabetes whose HbA1C was above 8, and the second group is controlled diabetes whose HbA1C is below 8.

Objectives: The primary objective was to compare these two groups on the basis of growth and height. Our goal was to discover and describe how hyperglycemia variability affects linear growth in children with T1D.

Results: Out of the 392 participants, their ages ranged from 5 to 18 years, with a mean of 11.27 years. The highest percentage (41.6%) of them were between 10 and 13 years old.

Recommendations: In order to minimize complications and guarantee proper anthropometric growth during childhood, strong metabolic management is a fundamental treatment objective.

Keywords: Chronic illnesses, developmental setbacks, growth issues

1. Introduction

Type 1 diabetes mellitus (T1DM) is a diverse condition resulting from the immune system's attack on beta cells within the endocrine pancreas. Individuals with a genetic predisposition to this autoimmune process experience it when one or more environmental factors are present. It usually advances over several months to years, during which patients show no symptoms, maintain normal blood sugar levels, but test positive for specific autoantibodies [1]

It is well established that childhood chronic disorders like Type 1 diabetes mellitus (T1DM) have a negative impact on linear growth and pubertal development ^[2].

Growth impairment in diabetic individuals is correlated with aberrations of the growth hormone-insulin-like Growth-I (GH-IGF-I) axis and is dependent on abnormalities in physiological bone growth. These changes appear to be connected to healthy insulin levels and, in turn, to glycemic management as determined by hemoglobin HbA1C levels [2].

T1DM affects the growth hormone (GH)/insulin-like growth factor-1 (IGF-1) axis, which controls growth. Studies demonstrate that children with T1DM have higher GH and lower IGF-1 levels. Data on the ability of adolescents with TIDM to grow to their genetically predetermined adult height is inconsistent. Furthermore, although the etiology of lower peak BMD in adolescents with T1DM is not fully understood, studies support this finding. Increased sclerostin, an inhibitor of bone anabolic pathways; low osteocalcin levels, which reflect decreased bone formation; and increased leptin, an adipocytokine that affects bone metabolism through central and peripheral mechanisms, are a few of the mechanisms that have been suggested for the decline in BMD. Upregulation of the osteoprotegerin receptor activator is one of the other causes of T1DM's enhanced bone resorption [3].

T1DM, a wasting condition caused by a severe insulin shortage or absence. Prevents the body from producing adequate energy for healthy growth. By maintaining glucose uptake into cells. The anabolic hormone insulin plays a significant protective function in preventing

Corresponding Author: Rusul Sami Wasfi Basra Teaching Hospital, Basra Health Department, Ministry of Health, Basra, Iraq muscle and fat catabolism. Without this hormone, blood sugar levels will rise and eventually be eliminated in the urine. Consequently, uncontrolled or poorly controlled diabetics will experience glycosuria and accidental weight loss [17].

For normal linear growth to continue, sufficient insulin secretion is required. Hormone (GH) plays a major regulatory role in the complex process of longitudinal bone development. IGF-I (insulin-like growth factor-I) binds to various IGFBPs (insulin-like growth factor binding proteins). IGFBP-3 is one of them. And research has revealed that GH affects how much of it is circulating. IGFs have a variety of metabolic effects, but their main function is to promote the development of long bones. Insulin is one of this system's most crucial regulators. In order to maintain normal blood concentrations of IGFs and IGFBPs and indirectly stimulate growth. Numerous studies have demonstrated the necessity of appropriate insulin secretion. Low portal insulin concentrations in T1DM children will therefore result in L OW GH [18].

T1DM stands as a prevalent chronic endocrine condition in the pediatric and adolescent population. And its complications, notably compromised growth during childhood, continue to pose significant challenges. This research seeks to investigate the connection between metabolic management and the growth status of children diagnosed with T1DM.

Multiple research studies have shown that children with T1DM usually display certain irregularities in their GH/IGF-1 axis in comparison to their healthy peers. The information regarding the growth patterns of children and teenagers with T1DM still lacks uniformity. Several investigations have indicated that young individuals with T1DM who are either before or during puberty undergo inhibited growth. This growth hindrance seems to be linked to ineffective glycemic management and the duration of the illness. However, the latest research has indicated that children receiving contemporary, stringent insulin treatment achieves a standard adult height [4].

GH secretion is pulsatile and varies in concentration with age. In actuality, GH levels typically rise during puberty, rise again during maturity, and then fall again during the prepubertal phase. The primary means through which growth-enhancing impacts of GH are conveyed involve insulin-like growth factors I and II. These factors are chiefly discharged by the liver. Several IGFBPs, also known as insulin-like growth factor binding proteins, carry IGFs in their circulation. The most widely distributed form of each of them is IGFBP-3, and it has been proven that GH influences its concentrations. These binding proteins play critical roles in the control of the GH/IGFs axis by prolonging the half-life of IGFs and delivering IGFs to the target tissues by forming a ternary complex with the acid-labile subunit (ALS) [4].

Insulin-like growth factor 1 (IGF-1), also called somatomedin C, is a hormone similar in molecular structure to insulin which plays an important role in childhood growth, and has anabolic effects in adults. IGF-1 is produced primarily by the liver. Production is stimulated by growth hormone (GH). Most of IGF-1 is bound to one of 6 binding proteins (IGF-BP). IGFBP-1 is regulated by insulin. IGF-1 is produced throughout life; the highest rates of IGF-1 production occur during the pubertal growth spurt. The lowest levels occur in infancy and old age. IGF-1 is a

primary mediator of the effects of growth hormone (GH). Growth hormone is made in the anterior pituitary gland, is released into the blood stream, and then stimulates the liver to produce IGF-1. IGF-1 then stimulates systemic body growth, and has growth-promoting effects on almost every cell in the body, especially skeletal muscle, cartilage, bone, liver, kidney, nerve, skin, hematopoietic, and lung cells. In addition to the insulin-like effects, IGF-1 can also regulate cellular DNA synthesis [27].

While IGFs have diverse metabolic impacts, their primary role revolves around enhancing the growth of elongated bones. They achieve this by governing the proliferation, maturation, and enlargement of chondrocytes within the growth plate, as well as by stimulating the synthesis and degradation of the matrix. One of the most significant regulators of this system is insulin. According to a number of studies, proper portal insulin concentrations and enough insulin production are required to maintain normal IGF and IGFBP blood concentrations and, indirectly, to support growth [4].

Numerous studies have conclusively shown that insulin plays a crucial function as one of the primary regulators of the GH/IGFs axis. Insulin controls GH receptor expression in the liver and influences the synthesis of IGFs and IGFBPs by modifying GH postreceptor processes. ^[5]

Short stature is defined as a height more than two standard deviations below the mean for age (less than the 3rd percentile). Children over age 2, or teens whose BMI is: Less than the 5th percentile are considered underweight. Between the 5th percentile and less than the 85th percentile are at a healthy weight. In the 85th percentile to less than the 95th percentile are considered overweight [6].

Thorough physical examination, a review of medical history, accurate serial measurements, and calculations of growth rate, midparental height, and bone age should all be part of the initial diagnosis of short stature. Idiopathic short stature, constitutional growth and puberty delay, and familial low stature are typical types of short stature. Short stature has pathologic causes, such as persistent illnesses and a lack of growth hormone [7].

Despite advancements in treatment, complications arising from diabetes remain a substantial issue. One enduring outcome of T1DM is impaired growth [8]. Growth rate that falls below the expected pace considering age and gender [9]. Variables such as genetics, age at diagnosis, duration of the disease, blood sugar regulation, stage of puberty, and levels of growth hormone contribute to the diversity in growth patterns among individuals with diabetes ^[10].

It is still uncertain whether children with diabetes experience hindered linear growth. Several studies have demonstrated that effective metabolic management plays a crucial role in determining adult height among individuals with T1DM [11, 12]. Conversely, in many other studies, it has been observed that the growth of such children is unaffected by their hemoglobin A1c (HbA1c) levels [13, 14].

Nonetheless, several studies have suggested that diminished height growth is more influenced by the duration of the condition rather than the extent of metabolic control [15].

1.1 Objectives

The objective of this study was to determine how fluctuations in blood sugar levels impact the linear growth of children diagnosed with T1D.

2. Material and Method

2.1 Study design, place and time

This registry based descriptive study aim to evaluate the growth of patients with type 1 diabetes mellitus at Faiha Specialized Diabetes, Endocrine and Metabolism Centre (FDEMC). The study focuses on individuals who have had diabetes for a minimum of one year and attended the center from early July 2021 to the end of June 2023.

2.2. Participants(sampling and population)

This study included 392 patients, who were divided into two groups. The first group is those with uncontrolled diabetes whose HbA1C was above 8. The second group is those with controlled diabetes whose HbA1C was below 8 ^[16].

2.3 Inclusion criteria

Patients with type 1 diabetes who were. Diagnosed for at least 1 year or more.

Exclusion criteria

- Patients with type 2 diabetes
- New onset type 1 diabetes or less than one-year history of diabetes mellitus
- infants and young kids.
- Patients with chronic kidney disease, chronic liver disease, celiac and hypothyroidism, on steroids or other medication that may affect the glycemic state.

2.4 Metabolic Control Assessment

The average HbA1c measurements over the past half-year

were collected using the electronic Phoenix system at KAUH.

2.5 Anthropometric measurements

All patients underwent measurements for height and weight, and the body mass index (BMI) was computed using the formula BMI = weight / height 2.

2.6 Statistical analysis

All data obtained in the study were organized and analyzed using Statistical Package for the Social Sciences (SPSS) software version 23.

A comparative assessment was conducted among the various groups, and statistical significance was established with a p-value lower than 0.05. Results with a p-value below this threshold were considered statistically significant, indicating a significant distinction or correlation.

3. Results

This study involved 392 patients who visit the center with T1DM.

Table 1 shows gender distribution of the participants. Females formed 60.7% of them while males formed 39.3%.

Table 1: The distribution of participants according to gender

Gender	No.	%
Male	154	39.3
Female	238	60.7
Total	392	100.0

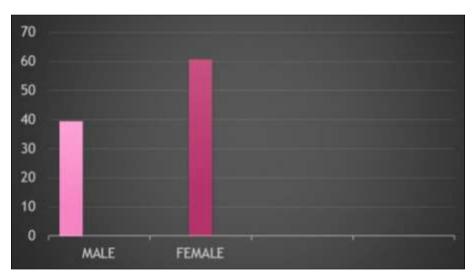


Fig 1: The distribution of participants according to gender

Table 2 shows age distribution of the participants. Their ages ranged from 6 to 18 years, with a mean of 11.27 years.

The highest percentage (41.6%) of them were between 10 and 13 years old.

Table 2: The distribution of participants according to age

Age	No.	%
Mean ±SD	11.27± 3.22 (6-18)	
5-9	130	33.2
10-13	163	41.6
14-18	99	25.3
Total	392	100.0

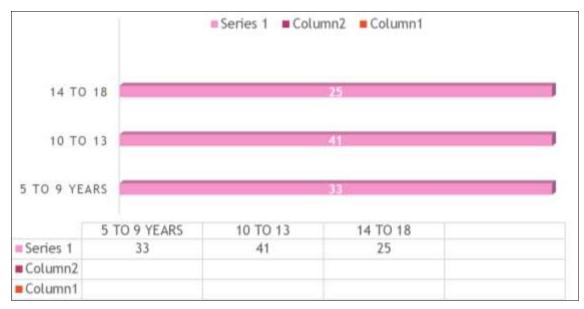


Fig 2: The distribution of participants according to age

Table 3 shows distribution of participants according to their anthropometric measures. The weight ranged from 11 to 100 kg, with a mean of 38.2 kg, while the height mean was 142.8 cm.

Height for age was done, and participants were classified into those with short stature, normal height, and very tall. 76.3% of the participants had a normal weight, while only 18.6% were short.

Table 3: The distribution of participants according to their anthropometric measures

Variable		No.	%	
Weight in kg Mean ±SD		38.2 ± 17.43 (11-100)		
Height in cm	Mean ±SD	$142.8 \pm 17.0 (92-186)$		
Height for age				
Short s	tatur	73	18.6	
Normal		299	76.3	
Very tall		20	5.1	

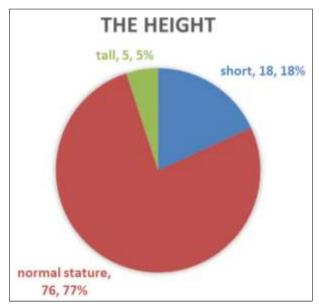


Fig 3: The distribution of participants according to their anthropometric measures

The distribution of participants according to the HbA1c

level was shown in Table 4. Only 13.3% of participants had well-controlled diabetes. While 86.7% were poorly controlled.

Table 4: The distribution of participants according to the HbA1c level

HbA1c	No.	%
Mean ±SD	10.1	± 4.6 (5-18)
Good control	52	13.3
Poor control	340	86.7
Total	392	100%

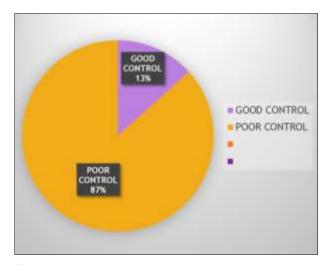


Fig 4: The distribution of participants according to the HbA1c level

Table 5 shows the association between diabetes control and gender. There's no significant statistical difference between male and female participants. p-value =0.632. Still a higher percentage of good control diabetes among males (14.3%) in comparison to females.

Table 5: The association between diabetes control and gender

Gender	Good control	Poor control	p-value
Male	22(14.3)	132 (85.7)	
Female	30 (12.6)	208 (87.4)	0.632
Total	52 (13.3)	340 (86.7)	

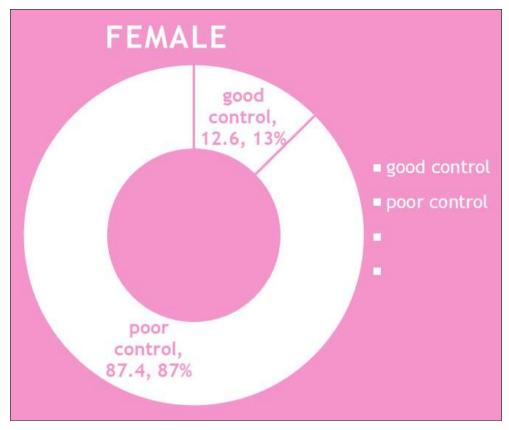


Fig 5: The association between diabetes control and gender

Table 6 shows the association between diabetes control and age. There is no significant statistical difference between different age groups concerning diabetes control. p-value=0.477.

The highest percentage of good control was among those less than 9 years (16.2%).

Table 6: The association between diabetes control and age

	Good control	Poor control	P-value
5-9	21 (16.2)	109 (83.8)	
10-13	20 (12.3)	143 (87.7)	0.477
14-18	11 (11.1)	88 (88.9)	0.477
Total	52 (13.3)	340 (86.7)	

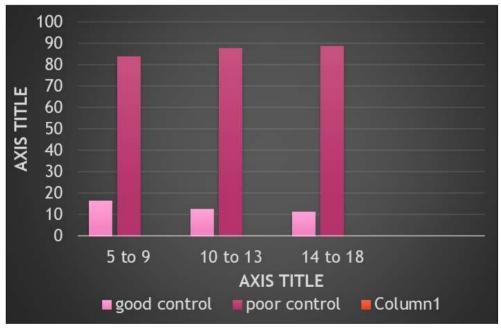


Fig 6: The association between diabetes control and age

Table 7 shows the association between anthropometric measures and gender. There are no significant differences in weight and height among both gender p-value >0.05. The height for age shows no significant differences between

males and females (P value = 0.898).

Similarly, for the BMI there is no significant difference between males and females. P-value=0.822.

Table 7: The association between anthropometric measures and gender

Measures	Male	Female	P-value		
Weight	38.7 ±17.5	37.9± 17.4	0.698		
Height	144.3 ±17.9	141.9 ±16.4	0.194		
	Height for age				
Short stature	28 (18.2)	45 (18.9)			
Normal	119 (77.3)	180(75.6)	0.898		
Very tall	7 (4.5)	13 (5.5)			



Fig 7: The association between anthropometric measures and gender

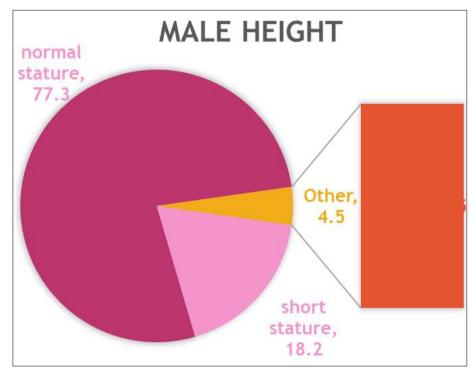


Fig 8: Male hight

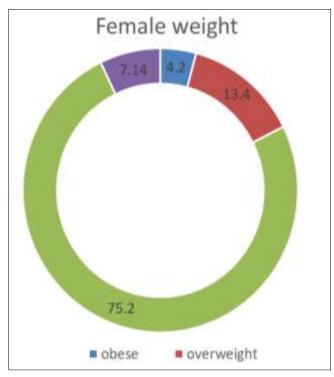


Fig 9: Female weight

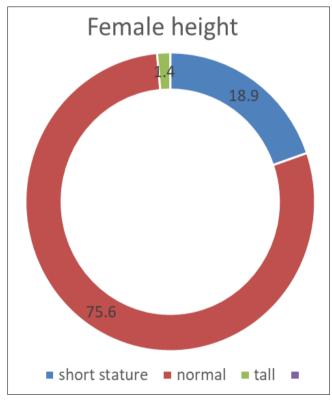


Fig 10: Female height

Table 8 shows the association between anthropometric measures and age groups. There are significant differences in anthropometric measures among different age groups. P-value =0.001.

Regarding height and age, there is a significant difference between different age groups. Participants younger than 10 years have the lowest percentage of short stature; the percentage of those with short stature increases with aging. While BMI for age shows no significant difference between different age groups, P value=0.871.

Table 8: The association between anthropometric measures and age groups

Measures	5-9 years	10-13 years	14-18 years	p-value	
Weight	23.7 ± 6.2	39.5± 13.2	54.9± 17.0	0.001	
Height	125.5 ± 9.9	146.7 ± 10.8	159.3±10.9	0.001	
BMI	13.4 ± 4.7	15.4 ±7.6	19.8 ± 7.5	0.001	
	Height for age				
Short stature	13 (10.0)	34 (20.9)	26 (26.3)		
Normal	106 (81.5)	122 (74.8)	71 (71.7)	0.006	
Very tall	11 (8.5)	7 (4.3)	2 (2.0)		

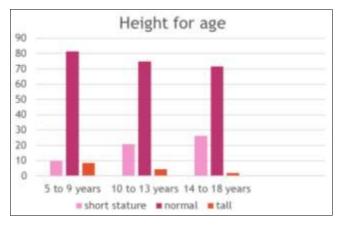


Fig 11: The association between anthropometric measures and age groups

Table 9 shows the association between anthropometric measures and diabetes control. The patients with good control showed higher measures than those with poor control. There are no significant differences in weight and height with diabetes control. P-value >0.05.

Regarding height for age, the percentage of shorter stature was 7.7% among those with good control of diabetes and 20.3% among the poor-controlled diabetes group. This difference was statistically significant since the P value was 0.036.

On the other hand, BMI shows a significant difference in diabetes control. p-value=0.05.

Table 9: The association between anthropometric measures and diabetes control

Measures	Good control	Poor control	P-value	
Weight	39.1 ±16.2	37.9 ± 17.8	0.825	
Height	143.5± 16.2	141.6 ± 17.3	0.469	
BMI	17.6 ±5.7	15.6 ± 7.4	0.05	
H	leight for age			
Short statur	4 (7.7)	69 (20.3)		
Normal	47 (90.4)	252(74.1)	0.036	
Very tall	1 (1.9)	19 (5.6)		
Above 95 th =Obese	2 (3.9)	14 (4.1)		
Above 85^{th} = overweight	10 (19.2)	40 (11.8)	0.040	
Above 5 th =normal	39 (75.0)	260 (76.5)	0.049	
Below 5th =wasted	1 (1.9)	26 (7.6)		
Total	52 (100.0)	340 (100.0)		

Table 9 shows the correlation between HbA1c level and other variables.

There is a positive correlation between age and HbA1c level as it increases with getting older, but this correlation is still not significant. P-value=0.066.

The anthropometric measures negatively correlate with HbA1c level. This correlation is not significant since the p-value >0.05.

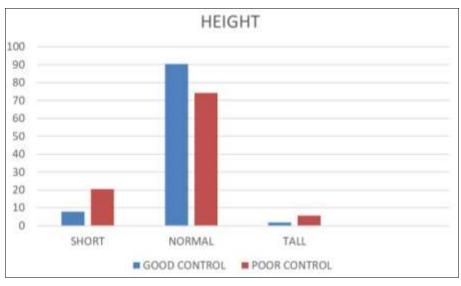


Fig 12: The association between anthropometric measures and diabetes control

4. Discussion

In the present study; approximately 41.6% of the children were diagnosed with the disease at the age of 10 years or older, while only 33.2% belonged to the younger age bracket. A remarkably similar pattern was observed in a cross-sectional study conducted at The Children's Welfare Teaching Hospital Medical City Complex in Baghdad, where children above the age of 10 constituted 47.9% of the population, while younger children comprised 30.4% [19].

A study on pediatric diabetes in Saudi Arabia, found that there were two distinct age peaks for the initiation of diabetes in children: one between 5 and 7 years old, and the other between 11 and 14 years old [20].

The slight rise in occurrence was attributed to the greater exposure of younger children to infectious agents in daycare and school environments. Due to increased gonadal steroid and pubertal growth hormone secretion, which counteract insulin action, the second peak occurs during puberty. Additionally, emotional stress associated with puberty is a contributing factor that increases the pubertal incidence of type 1 diabetes [21].

Regarding sex distribution, the current study showed a slight female excess of 60.7%; such a female preponderance was also observed in Saudi Arabia, where 53.6% of diabetic children were female [22].

On the contrary, a study conducted in Denmark showed a male preponderance with male to female ratio $1.3:1^{[23]}$.

While a balanced male-to-female ratio is commonly indicated in type 1 DM patients, several investigations have suggested a marginal prevalence in either boys or girls [24].

The current study's BMI indicated that obesity and overweight were evident in 16.8% of diabetic children. On the one hand, wasting was discovered in 6.9% of the population.

The mean age of participants was 11.27±3.22, 13.3% of them had good metabolic control with a HbA1c level greater than 8, while 86.7% had poor metabolic control greater than 8.

The mean HbA1c level in patients was 10.1±4.6, ranging from 5 to 18. There was no statistical difference in the level of HbA1c between males and females. The difference in mean HbA1c levels based on age was not statistically significant.

In the current study, HAZ showed that 76.3% of the studied

children were of normal height for their age; stunting was found in 18.6% of diabetic children, more among females (18.9%) than males (18.2%).

Five percent of children were very tall (5.5% in females, 4.5% in males), and a cross-sectional study conducted in Baghdad in 2013 found that 79.9% of the studied children were of normal height for their age. Stunting was found in 15% of diabetic children, more among females (18.9%) than males (18.2%).

And 5.1% of children were very tall (5.7 in males, 4.6% in females), which is similar to our study.

In the current study, height for age shows that the percentage of short stature was 7.7% among those with good control of diabetes and 20.3% among those with poor control of diabetes. This difference was statistically significant since the P value was 0.036.

On the other hand, BMI shows a significant difference in diabetes control. P-value=0.05.

Similarly, a multicenter trial involving 206 patients concluded that a significant impairment in growth during puberty in young people with T1D, poor glycemic control, as well as other genetic or environmental factors, could explain these associations [25].

Another multicenter study found that linear growth is impaired in children with type 1 diabetes (T1D) and poor metabolic control. Good metabolic control is a key therapeutic goal to prevent vascular complications and also ensure appropriate anthropometric development during childhood ^[26].

5. Conclusion and Recommendation

Linear growth is impaired in children with type 1 diabetes (T1D) and poor metabolic control.

6. Recommendation

In order to minimize the complication and guarantee proper anthropometric growth during childhood, strong metabolic management is a fundamental treatment objective. Our goal was to discover and describe how hyperglycemia variability affects linear growth in children with T1D.

7. References

 Paschou SA, Petsiou A, Chatzigianni K, Tsatsoulis A, Papadopoulos GK. Type 1 diabetes as an autoimmune

- Diabetologia. 2014 disease: the evidence. Jul:57(7):1500-1501.
- DOI: 10.1007/s00125-014-3229-5. Epub 2014 Apr 6. PMID: 24705607.
- Chiarelli F, Giannini C, Mohn A. Growth, growth factors and diabetes. European Journal Endocrinology, 2004 Nov, 151(3). DOI: 10.1530/eje.0.151u109. PMID: 15554895.
- Raisingani M, Preneet B, Kohn B, Yakar S. Skeletal growth and bone mineral acquisition in type 1 diabetic children: abnormalities of the GH/IGF-1 axis. Growth Hormone & IGF Research. 2017 Jun;34:13-21. DOI: 10.1016/j.ghir.2017.04.003. Epub 2017 Apr 28. PMID: 28482269; PMCID: PMC5516798.
- Santi E, Tascini G, Toni G, Berioli MG, Esposito S. Linear growth in children and adolescents with type 1 diabetes mellitus. International Journal Environmental Research and Public Health. 2019 Sep 30;16(19):3677. DOI: 10.3390/ijerph16193677. PMID: 31574933; PMCID: PMC6801810.
- Giannini C, Mohn A, Chiarelli F. Growth abnormalities in children with type 1 diabetes, juvenile chronic arthritis, and asthma. International Journal Endocrinology. 2014;2014:265954. DOI: 10.1155/2014/265954. Epub 2014 Feb 4. PMID:
 - 24648838; PMCID: PMC3932221. Maes M, Underwood LE, Ketelslegers JM. Low serum
- somatomedin-C in insulin-dependent diabetes: evidence for a postreceptor mechanism. Endocrinology. 1986 Jan;118(1):377-382.
 - DOI: 10.1210/endo-118-1-377. PMID: 3000746.
- Barstow C, Rerucha C. Evaluation of short and tall stature in children. American Family Physician. 2015 Jul 1;92(1):43-50. PMID: 26132126.
- Kim MS, Quintos JB. Mauriac syndrome: growth failure and type 1 diabetes mellitus. Pediatric Endocrinology Reviews. 2008 Aug;5(4):989-993. PMID: 18806715.
- Patel R, Bajpai A. Evaluation of short stature in children and adolescents. Indian Journal of Pediatrics. 2021 Dec;88(12):1196-1202.
 - DOI: 10.1007/s12098-021-03880-9. Epub 2021 Aug 16. PMID: 34398416.
- 10. Virmani A. Growth disorders in type 1 diabetes: an Indian experience. Indian Journal of Endocrinology and Metabolism. 2015 Apr;19(Suppl 1). DOI: 10.4103/2230-8210.155405.
 - PMID: 25941656; PMCID: PMC4413395.
- 11. Marcovecchio ML, Heywood JJ, Dalton RN, Dunger DB. The contribution of glycemic control to impaired growth during puberty in young people with type 1 diabetes and microalbuminuria. Pediatric Diabetes. 2014 Jun; 15(4): 303-308.
 - DOI: 10.1111/pedi.12090. Epub 2013 Dec 9. PMID: 24320564.
- 12. Elamin A, Hussein O, Tuvemo T. Growth, puberty, and final height in children with type 1 diabetes. Journal of and Complications. 2006 Diabetes its Jul-Aug;20(4):252-256.
 - DOI: 10.1016/j.jdiacomp.2005.07.001. PMID: 16798477.
- 13. Lebl J, Schober E, Zidek T, Baldis S, Rami B, Pruhova S, et al. Growth data in large series of 587 children and adolescents with type 1 diabetes mellitus. Endocrine

- Regulations. 2003 Sep;37(3):153-61. PMID: 14986721.
- 14. Salerno M, Argenziano A, Di Maio S, Gasparini N, Formicola S, De Filippo G, et al. Pubertal growth, sexual maturation, and final height in children with IDDM. Effects of age at onset and metabolic control. Diabetes Care. 1997 May;20(5):721-724. DOI: 10.2337/diacare.20.5.721. PMID: 9135933.
- 15. Grgic A, Rosenbloom AL, Weber FT, Giordano B, Malone JI, Shuster JJ. Joint contracture—common manifestation of childhood diabetes mellitus. Journal of Pediatrics, 1976 Apr:88(4 Pt 1):584-588. DOI: 10.1016/s0022-3476(76)80011-x. PMID: 1255316.
- 16. Giannini C, Mohn A, Chiarelli F. Growth abnormalities in children with type 1 diabetes, juvenile chronic arthritis, and asthma. International Journal Endocrinology. 2014;2014:265954. DOI: 10.1155/2014/265954. Epub 2014 Feb 4. PMID: 24648838; PMCID: PMC3932221.
- 17. Bonfig W, Kapellen T, Dost A, Fritsch M, Rohrer T, Wolf J, et al. Diabetes Patienten dokumentations system Initiative of the German Working Group for Pediatric Diabetology and the German Bundesministerium für Bildung und Forschung Competence Net for Diabetes Mellitus. Growth in children and adolescents with type 1 diabetes. Journal of Pediatrics. 2012 Jun;160(6):900-903.e2. DOI: 10.1016/j.jpeds.2011.12.007. Epub 2012 Jan 11.
- PMID: 22244464. 18. Abd-Alrazak OM, Ghalib AA, Abduljabbar HA. Growth indices among children and adolescents with type 1 diabetes - Baghdad - Iraq, 2013. Journal of the
- 19. Al-Agha A, Ocheltree A, Hakeem A. Outpatient management of childhood diabetes clinic King Abdulaziz University Hospital Jeddah, Saudi Arabia. Med. 1989;9:365-370.

Faculty of Medicine-Baghdad. 2014;56:258-263.

- 20. Borchers AT, Uibo R, Gershwin ME. geoepidemiology of type 1 diabetes. Autoimmunity Reviews, 2010 Mar, 9(5). DOI: 10.1016/j.autrev.2009.12.003. Epub 2009 Dec 5. PMID: 19969107.
- 21. Abdullah MA. Outpatient management of childhood diabetes: experience of a pediatric diabetes clinic at King Khalid University Hospital, Riyadh. Medical Journal. 1989;9(4):365-370.
- 22. Green A, Patterson C, on behalf of the EURODIAB TIGER Study Group. Trends in the incidence of childhood-onset diabetes in Europe 1989-1998. Diabetologia. 2001;44 Suppl:B3-B8.
- 23. Karvonen M. Pitkäniemi M. Pitkäniemi J. Kohtamäki K, Tajima N, Tuomilehto J, et al. Sex difference in the incidence of insulin-dependent diabetes mellitus: an analysis of the recent epidemiological data. World Health Organization DIAMOND Project Group. Diabetes/Metabolism Research and Reviews. 1997 Dec;13(4):275-291.
 - DOI: 10.1002/(sici)1099-0895(199712)13:4<275::aiddmr197>3.0.co;2-v. PMID: 9509279.
- 24. Marcovecchio ML, Heywood JJ, Dalton RN, Dunger DB. The contribution of glycemic control to impaired growth during puberty in young people with type 1 diabetes and microalbuminuria. Pediatric Diabetes. 2014 Jun;15(4):303-308.

DOI: 10.1111/pedi.12090. Epub 2013 Dec 9. PMID: 24320564.

- 25. Blasetti A, Castorani V, Polidori N, Mascioli I, Chiarelli F, Giannini C, *et al.* Role of glucose variability on linear growth in children with type 1 diabetes. Endocrine Connections, 2023 Mar 28, 12(4). DOI: 10.1530/EC-22-0370. PMID: 36799250; PMCID: PMC10083674.
- Insulin-like growth factor 1. Wikipedia. Available from:

https://en.wikipedia.org/wiki/Insulin-like_growth_factor_1.

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