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Long-Term Hyperglycemia Triggered Growth Pattern of Pediatrics with Type 1 Diabetes -A Five-Year Retrospective Follow-Up Study

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Abstract

Introduction: Children with Type I Diabetes (T1D) usually have lesser stature for age than their regular counterparts.

Objective: This study was conducted to assess the effect of long-term hyperglycemic condition on growth pattern concerning height for age of pediatric subjects with Type 1 Diabetes Mellitus.

Methods: The retrospective follow-up study was conducted among 162 pediatric subjects with T1D. Pediatric subjects with T1D registered for medical and nutritional care from 2009-2013 at King Abdullah Specialized Children's Hospital (KACSH)/NGHA, Riyadh, KSA; a tertiary care teaching hospital formed the study population. The subjects were selected randomly and followed up for the immediate post-diagnostic period up to 5 years. Data were extracted from the Hospital Information system- Best care of the hospital. The data for demography and biochemical variables were extracted at the time of diagnosis. In contrast, data of height and weight were collected not only at the time of diagnosis but also followed up for the immediate post-diagnostic period of five years. The data were analyzed by using SPSS Version 22.

Result: Majority (59.9%) of the subjects were in the age group of 7 to 10 years, and 53.1% were females. The study results indicated that there was gender variation in growth pattern with the advancement of age which leads to short height for age.

Conclusion: T1D can affect children's growth pattern with a permanent effect on the stature of the affected child. Children with T1D require periodical growth monitoring and nutrition care to prevent short stature.

Keywords: BMI; Growth Pattern; Height for Age; Pediatrics; T1D

Introduction

Type 1 diabetes (T1D) is a chronic childhood autoimmune disease characterized by insulin deficiency and hyperglycemic conditions. The onset of T1D happens typically in childhood, although it can present at any stage of life **[1]**. Recently, an increasing trend in the incidence of T1D has been reported worldwide.

In continental subgroups, the incidence of T1D among children in America (20 per 100 000) is higher than in Asia (15 per 100 000) and the global prevalence of continental subtypes of T1D in the above regions was 12.2 per 10 000, 6.9 per 10

000, respectively **[2]**. In terms of incidence rates of T1D (0-14 years), Saudi Arabia ranks the 5th country in the world with 31.4 per 100,000 population per year **[1]**.

The onset of T1D is likely the result of the interplay among various genetic and epigenetic factors. The proposed epigenetic factors involved in the pathogenesis of T1D include environmental, dietary and somatic factors. Virus infections and immunizations, pollutants, gut flora, rural versus urban residence are the prime environmental factors with pathogenicity for T1D. The dietary factors such as inadequate breastfeeding, complimentary feeding of cow's milk instead of breast milk, Vitamin D deficiency, consumption of gluten rich food, poor intake of dietary fiber rich food and polyunsaturated fatty acids. Somatic factors including low birth weight, childhood growth, childhood obesity and psychological stresses also have an influence on the incidence of T1D **[3]**.

The growth of children with T1D was reported to be affected with gender, genetic factors, age at diagnosis of T1D, duration of the disease, puberty, metabolic control, growth-promoting agents and its binding proteins such as growth hormone (GH), insulin-like growth factors (IGFs), and IGF binding proteins (IGFBPs). IGFs' most important role is to aid increase in bone length by regulating growth plate chondrocyte proliferation and maturation. Insulin is considered an essential regulator of this process as adequate insulin concentration is needed to maintain normal serum concentrations of IGFs and IGFBPs and intra-portal insulin concentrations, which leads to GH hypersecretion, low circulating IGF and IGFBPs **[4, 5]**. Reduced growth of children with T1D may be due to alterations in GH concentrations causing physiological abnormalities in bone growth **[6, 7]**.

In T1D, the intra-portal insulin concentrations, poor glycemic control, autoimmune disorders, improper renal function, and psychosocial factors and associated diseases may cause growth retardation and pubertal delay **[8]**. Besides, poor metabolic control, longer disease duration is also associated with growth abnormalities in them **[7]**.

The incidence and prevalence of T1D are high in the Saudi population. Scarcely few studies were done on the effect of T1D on growth of pediatrics with T1D. Hence, the current research investigates the pattern of change in height, BMI, and associated factors in T1D patients between 1 and 10 years over five years post diagnosis.

Methodology

This longitudinal observational study was conducted by analyzing the health charts of children registered for medical and nutritional care and management of T1D in the outpatient pediatrics clinics in King Abdullah Specialized Children's Hospital (KASCH), Ministry of National Guard Health Affairs (NGHA), Riyadh, KSA. The study was approved by the Institutional Review Board of King Abdullah International Medical Research Centre and was conducted following the Declaration of Helsinki **[9]**.

Initially, 300 T1D patients who visited outpatient pediatrics clinics between 2009 and 2013 were selected for this study. Data were collected from the electronic medical records of KASCH and NGHA.

A total of 162 patients who fulfilled the inclusion criteria were screened out for the study. The study inclusion criteria were Saudi TID patients of age between 0-10 years and followed up for five years post diagnostically. Children with psychological and physical disabilities and other chronic diseases that may affect the growth, patients who did not follow up for the five years, and patients with missing data were excluded from the study.

Anthropometric and demographic parameters such as age, gender, height and weight were collected for each subject. Body mass index (BMI) was calculated as the quotient of weight (kg) divided by height square (m2). Biochemical data such as hemoglobin A1C (HbA1c), fasting blood glucose (FBG), total cholesterol, low density lipoprotein (LDL), high density lipoprotein (HDL), triglyceride, blood urea and serum creatinine were collected. All the data were collected for a period of five years starting from the date of diagnosis. In addition, the dietary pattern followed by the subjects were recorded.

Data management and analysis

The study sample size was calculated with Rao soft online sample size calculator **[10]**. With a confidence level of 95% at margin of error of 5%. Statistical analysis of the data was done by using SPSS (version 22). Categorical variables were expressed in frequencies and percentages. Mean and standard deviation (mean ± SD) were used for continuous variables.

In order to assess the growth pattern of the subjects the data were compared to standard growth charts for the children from Saudi Arabia **[11, 12]**. From the standard growth chart, +2SD (90th percentile) and +1SD (75th percentile) were used as standard cutoff values for both height and BMI. The student's t-test was used to compare differences between male and female groups.

Results

Table 1 shows the baseline demographic, clinical, and biochemical parameters of male and female pediatrics with T1D. The selected subjects, (53.09% female) had a mean ± SD age of 6.79±2.42 years. Age (p = 0.037) and height (p = 0.029) significantly differed between both male and female T1D patients. Mean weight, BMI, HbA1c, total cholesterol, LDL, HDL, triglycerides, urea, serum creatinine were similar among male and female patients.

Data with respect to dietary pattern of the subjects indicated that 79% of them followed the diet recommended by American Diabetes Association (ADA). However, the higher HbA1c values point out that all the participants were of poor glycemic control.

Table 1. Baseline characteristics of type 1 diabetic patients at the time of diagnosis

Parameters***	Total**	Male	Female	p

(n)%	162	(76)46.91	(86)53.09	NA
Age, years (mean ± SD)	6.79±2.42	6.37±2.67	7.16±2.13	0.037*
1-3 years(n)%	(22)13.6	(16)72.7	(6)27.3	NA
4-6 years(n)%	(43)26.5	(17)39.5	(26)60.5	NA
7-10 years(n)%	(97)59.9	(43)44.3	(54)55.7	NA
Height (cm) (mean ± SD)	119±14.9	116±16.42	121±13.09	0.029*
Weight (kg) (mean ± SD)	24.29±8.71	23.29±9.18	25.17±8.23	0.17
BMI, kg/m2(mean ± SD)	16.59±2.87	16.57±2.89	16.61±2.87	0.924
HbA1c, % (mean ± SD)	10.46±2.14	10.14±2.09	10.74±2.15	0.076
Total cholesterol, mmol/L (mean ± SD)	4.27±0.49	4.64±0.13	4.23±0.49	0.272
LDL, mmol/L (mean ± SD)	2.49±0.54	2.96±0.10	2.44±0.54	0.214
HDL, mmol/L (mean ± SD)	1.39±0.29	1.07±0.57	1.43±0.24	0.094
Triglycerides, mmol/L (mean ± SD)	0.61±0.15	0.59±0.14±	0.62±0.15	0.82
Urea, mmol/L (mean ± SD)	4.42±1.49	4.57±1.38	4.31±1.58	0.384
Serum creatinine, µmol/L (mean ± SD)	59.76±15.10	58.52±15.59	60.73±14.77	0.46
Dietary pattern				
Regular diet (n)%	(34)20.99	(12)15.8	(22)25.6	NA
ADA recommended diet(n)%	(128)79.01	(64)84.2	(64)74.4	NA

* Values of $p < 0.05$ were considered significant

** Data are presented as percentage and mean ± standard deviation.

*** **Abbreviations:** - BMI; body mass index, LDL; low density lipoprotein, HDL; high-density lipoprotein, ADA; American Diabetes Association.

The height for the age distribution of male subjects was exhibited in **Table 2**. It was observed that, the height for age of the subjects who were diagnosed with diabetes at the age of 3 years was significantly ($p = 0.040$) lower than the standard cutoff, and the height showed a decreasing tendency for five years follow-up. No significant difference was found in height for age for the subjects diagnosed with diabetes at the age of 4-6 years. Further, male subjects diagnosed with diabetes at the age of 7 onwards were significantly ($p = 0.005$) lower than the standard cutoff, and the height showed a decreasing tendency for five-year follow-up.

The height for the age distribution of female subjects is given in **Table 3**. Unlike males, female subjects diagnosed with diabetes at the age of 4-10 were significantly ($p=0.049$) lower than the standard cutoff. This decreasing tendency from the standard cutoff was observed throughout the five year follow-up.

The BMI for the age distribution of both male and female subjects are shown in **Tables 4 and 5**. The BMI for age for the male subjects diagnosed with diabetes at the age of 7 was significantly ($p=0.045$) lower than the standard cutoff, and the BMI showed a decreasing tendency for five-year follow-up. The BMI for age for the female subjects diagnosed with diabetes at the age of 9 years was lower than the standard cutoff, and the BMI significantly ($p=0.009$) decreased after a two-year follow-up.

Discussion

This study aimed to evaluate the long-term effect of hyperglycemia due to T1D on the height and BMI of children. Our study observed that children with TID were shorter when compared to standard cutoff values. Reduced growth pattern as evidenced by slow rate of increase in height was exhibited by both male and female T1D patients during the five year follow up.

Those male subjects with T1D, diagnosed under the age of 7 years were similar in height. But a significantly lower height velocity was found in patients diagnosed at an age more than seven years.

Similar trend of significantly lower height velocity was exhibited by female subjects who were diagnosed T1D at an age more than four years. Our study's result was contradictory to previous findings that children with T1D aged 5-10 years at diagnosis were taller than controls of the same age, while those diagnosed at <5 years of age were shorter **[13]**.

Hypponen et al**.,** reported that children with T1D gained more weight during infancy and exhibited more remarkable linear growth than healthy controls at the time of diagnosis **[14]**.

In this study, the HbA1c values of T1D throughout the study indicate all participants were with poor glycemic control and with insufficient insulin concentration precipitated by T1D. Adequate insulin concentration is needed to maintain normal serum concentrations of IGFs, and IGFBPs. IGFs had a pivotal role in both muscle and bone turnover with subsequent increase in bone length by regulating growth plate chondrocyte proliferation and maturation **[4, 5, 15]**. Earlier studies reported that IGF-1 serve as a significant determinant of height; this has a more substantial influence than glycemic control and insulin level **[16]**. Furthermore, the insulin treatment has a direct positive relation to IGF-1 level. Hence, higher insulin dosages have been associated with increased height in children with T1D **[17]**.

Table 2. Height for age distribution of male subjects

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Age at diagnosis of T1D	Year 1			Year 2			Year 3			Year 4			Year 5		
	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p
1(n=3)	84 (6.65)	80.24	0.629	94.5 (1.26)	92.14	0.202	98.5 (2.5)	101.02	0.419	104.3 (2.33)	109.02	0.182	109.3 (1.45)	114.32	0.075
2 (n=6)	89.2 (2.66)	92.14	0.315	98.9 (2.68)	101.02	0.468	107.2 (2.28)	109.02	0.453	119.2 (1.56)	114.32	0.185	116.2 (2.00)	121.25	0.061
3 (n=7)	94.6 (2.43)	101.02	0.040*	99.5 (2.64)	109.02	0.011*	105.6 (3.23)	114.32	0.036*	110.2 (2.7)	121.25	0.006*	115 (3.08)	127.00	0.008*
4 (n=3)	109 (2.0)	109.02	0.993	116.8 (2.04)	114.32	0.345	122.1 (1.07)	121.25	0.51	127 (1.32)	127.00	1	130.7 (1.33)	132.5	0.303
5 (n=6)	113.3 (2.89)	114.32	0.738	120.7 (2.87)	121.25	0.86	126.8 (3.53)	127.00	0.953	132.3 (3.27)	132.5	0.961	136.5 (3.20	137.5	0.768
6 (n=8)	119.3 (2.36)	121.25	0.453	123.8 (2.46)	127.00	0.234	128.1 (2.13)	132.5	0.08	133.6 (2.36)	137.5	0.142	139 (2.17)	142.5	0.151
7 (n=15)	121.3 (1.73)	127	0.005*	126 (1.69)	132.5	0.002*	131.5 (1.82)	137.5	0.005*	135.9 (2.05)	142.5	0.006*	140.1 (2.080	147.5	0.003*
8(n=9)	122.4 (2.31)	132.5	0.002*	127.7 (2.36)	137.5	0.003*	132.8 (2.33)	142.5	0.003*	137.9 (2.62)	147.5	0.007*	143.4 (2.69)	154.0	0.004*
9 (n=9)	131.1 (2.58)	137.5	0.039*	135.9 (3.12)	142.5	0.067	140.2 (3.04)	147.5	0.044*	143.9 (3.42)	154.0	0.019*	149.6 (4.17)	160.0	0.037*
10 (n=10)	134 (2.27)	142.5	0.005*	140.7 (1.54)	147.5	0.002*	144.9 (1.73)	154.0	0.001*	149.4 (2.21)	160.0	0.001*	155.5 (2.78)	166.25	0.004*
* Values of p < 0.05 were considered significant															

Table 3. Height for age distribution of female subjects

Age at diagnosis of T1D	Year 1			Year 2			Year 3			Year 4			Year 5		
	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p	Actual Height mean (SD)	Standard height for age	p
2(n=1)	85.1	91.68	-	89.5	100.46	-	105	108.59	-	107	113.72	-	111.5	120	-
3 (n=5)	99.5 (2.95)	100.46	0.775	109.4 (2.67)	108.59	0.777	115.6 (4.96)	113.72	0.724	120.2 (2.4)	120	0.9	124.5 (2.88)	126.25	0.569
4 (n=7)	104.9 (1.5)	108.59	0.049*	109.6 (1.39)	113.72	0.026*	116.7 (1.92)	120	0.097	121.6 (1.45)	126.25	0.018*	130.2 (3.83)	132	0.65
5 (n=7)	108.8 (1.67)	113.72	0.026*	115.2 (1.97)	120	0.036*	120 (1.41)	126.25	0.004*	128.1 (3.51)	132	0.315	130.2 (2.07)	137.25	0.014*
6 (n=12)	116.6 (1.45)	120	0.020*	120.9 (1.27	126.25	0.001*	126.8 (1.07)	132	<0.001*	118.7 (1.05)	137.25	0.341	138.3 (1.79)	143	0.023*
7 (n=6)	125.1 (2.47)	126.25	0.661	129.7 (1.56)	132	0.191	137.1 (2.05)	137.25	0.945	142.5 (2.38)	143	0.847	148.4 (2.87)	149.5	0.721
8 (n=19)	125.7 (1.97)	132	0.005*	130.2 (1.78)	137.25	0.001*	134.7 (1.21)	143	<0.001*	140.3 (1.26)	149.5	<0.001*	145.3 (1.39)	155	<0.001*
9 (n=20)	130.9 (1.68)	137.25	0.001*	135.9 (2.01)	143	0.002*	140.3 (1.65	149.5	<0.001**	146.0 (1.68)	155	<0.001*	150.3 (1.35	158.5	<0.001*
10 (n=9)	136.1	143	0.001*	141.7	149.5	0.001*	149.0	155	0.006*	151.9	158.5	<0.001*	154.3	161.75	0.001*

	(1.41)			(1.29)			(1.63)			(1.12)			(1.38)		
* Values of p < 0.05 were considered significant															

Table 4. BMI for age distribution of male subjects

Age at diagnosis of T1D	Year 1			Year 2			Year 3			Year 4			Year 5		
	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p
1 (n=3)	16.92 (1.66)	18.81	0.188	15.33 (0.90)	17.29	0.064	14.25 (0.51)	16.60	0.016*	13.80 (0.72)	16.01	0.034*	13.82 (0.60)	15.77	0.031*
2 (n=6)	16.39 (1.56)	17.29	0.219	16.01 (1.60)	16.60	0.409	15.35 (0.98)	16.01	0.164	15.07 (1.08)	15.77	0.175	15.27 (1.26)	16.00	0.215
3 (n=7)	15.98 (0.86)	16.60	0.113	16.44 (1.37)	16.01	0.431	16.39 (1.75)	15.77	0.378	16.46 (2.32)	16.00	0.618	15.76 (2.68)	16.20	0.684
4 (n=3)	15.41 (1.81)	16.01	0.628	14.94 (1.63)	15.77	0.476	14.85 (0.55)	16.00	0.069	15.13 (0.81)	16.20	0.151	15.34 (0.92)	16.80	0.111
5 (n=6)	15.91 (2.11)	15.77	0.871	14.65 (3.82)	16.00	0.429	16.56 (2.79)	16.20	0.761	16.95 (2.78)	16.80	0.895	17.79 (3.02)	17.50	0.821
6 (n=8)	15.75 (1.70)	16.00	0.699	15.79 (1.11)	16.20	0.342	16.43 (1.71)	16.80	0.563	18.14 (3.03)	17.50	0.565	18.61 (3.40)	18.50	0.925
7 (n=15)	14.90 (2.27)	16.20	0.045*	15.48 (1.57)	16.80	0.006*	15.70 (1.95)	17.50	0.003*	16.26 (2.37)	18.50	0.003*	16.73 (2.79)	18.90	0.010*
8 (n=9)	15.73 (1.80)	16.80	0.115	16.73 (2.74)	17.50	0.428	16.79 (2.64)	18.50	0.088	17.79 (3.33)	18.90	0.351	18.94 (3.90)	20.2	0.365
9 (n=9)	18.09 (3.53)	17.50	0.627	18.91 (3.49)	18.50	0.732	19.08 (4.09)	18.90	0.898	20.81 (4.64)	18.90	0.703	21.48 (4.88)	21.0	0.777
10 (n=10)	20.27 (3.88)	18.50	0.183	19.71 (2.93)	18.90	0.403	20.52 (3.29)	18.90	0.764	20.28 (2.64)	21.0	0.409	21.48 (2.75)	21.8	0.726
* Values of p < 0.05 were considered significant															

Table 5. BMI for age distribution of female subjects.

Age at diagnosis of T1D	Year 1			Year 2			Year 3			Year 4			Year 5		
	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p	Actual BMI mean (SD)	Standard BMI for age	p
2 (n=1)	15.32	17.28	-	14.73	16.34	-	13.33	15.97	-	13.63	15.80	-	13.67	15.9	-
3 (n=5)	16.32 (2.46)	16.34	0.986	15.80 (2.24)	15.97	0.880	16.63 (1.66)	15.80	0.324	16.68 (2.10)	15.90	0.456	17.94 (2.19)	16.10	0.133
4 (n=7)	15.45 (1.97)	15.97	0.513	16.22 (1.75)	15.80	0.549	15.99 (1.28)	15.90	0.857	15.65 (2.29)	16.10	0.626	15.84 (3.88)	16.90	0.497
5 (n=7)	15.61 (1.11)	15.80	0.674	16.07 (1.46)	15.90	0.764	16.52 (2.12)	16.10	0.618	16.83 (2.22)	16.90	0.948	17.65 (2.32)	17.80	0.872
6 (n=12)	15.55 (2.99)	15.90	0.696	16.46 (2.36)	16.10	0.607	16.74 (2.74)	16.90	0.847	16.10 (5.84)	17.80	0.337	17.93 (3.21)	18.80	0.369
7(n=6)	16.20 (1.63)	16.10	0.879	16.82 (2.53)	16.90	0.946	17.02 (2.54)	17.80	0.487	17.91 (3.20)	18.80	0.529	18.69 (2.81)	19.80	0.376
8 (n=19)	17.53 (3.63)	16.90	0.455	18.27 (3.90)	17.80	0.600	18.01 (3.02)	18.80	0.276	18.99 (3.78)	19.80	0.367	21.14 (4.09)	21.0	0.880

9 (n=20)	16.63 (2.98)	17.80	0.097	17.19 (4.03)	18.80	0.092	18.03 (2.72)	19.80	0.009*	19.19 (3.59)	21.0	0.036*	21.35 (4.30)	22.0	0.507
10 (n=9)	18.33 (2.52)	18.80	0.596	18.99 (2.96)	19.80	0.438	20.84 (4.44)	21.0	0.918	22.83 (3.75)	22.0	0.526	25.28 (3.61)	23.2	0.122
* Values of $p < 0.05$ were considered significant															

In previous studies, it was observed that obesity and cardio-metabolic risk factors were highly prevalent in a pediatric cohort with T1D **[18]**. Our data on newly diagnosed type 1 diabetes indicated that BMI at diagnosis of the disease was not significantly different from standard cutoff values for both males and females with type 1 diabetes. Furthermore, BMI at diagnosis for seven years and nine years respectively for males and females showed dissimilarity.

A study on pubertal characteristics among schoolgirls in Saudi Arabia revealed that schoolgirls' mean puberty age was reported as 10 ± 0.80 years **[19]**. In our study, we observed that female T1D patients diagnosed with diabetes at the age of 9 were lower than the standard cutoff BMI, and the BMI significantly decreased after two years of the disease diagnosed at the age of 9.

A previous study among female children from Saudi Arabia noted a significant positive correlation between the onset of puberty and BMI **[20]**. BMI of female subjects of our study was lower than the standard cutoff at the pubertal age. Hence it is assumed that delayed somatic growth pattern can alter the onset of puberty. In this regard, the role of dietary pattern can be ruled out as 74.4% of the female subjects followed ADA recommended diet. The possible delay in puberty may be due to the interplay between GH and the altered metabolism.

Among male pediatrics diagnosed with T1D at the age of 9 and above shows higher BMI than the standard cutoff, but not significant. The age at onset of pubertal characteristics, based on gonadal development, among Saudi boys were reported to be between 11 and 15 years **[21]**. In Saudi boys, it was well demonstrated that BMI increased from 11 to 13 years of age, and BMI in childhood and adolescence was associated with height, sex, and pubertal status **[22, 23]**.

The study's primary limitations are related to data collection, from the electronic files from the best care system and quadramed. Due to the unavailability of data related to insulin dosage, the role of IGF in maintaining the height of the children cannot be illustrated. Furthermore, data related to the pubertal status of participants are also unavailable.

Conclusion

Based on the study results, it was concluded that T1D could cause long-term and short-term hyperglycemic irreversible effects on the affected child's growth pattern. Hence adequate medical management with precise insulin dosage and nutrition care is vital for achieving appropriate growth velocity among children with T1D. It is recommended to conduct a large sample study with more nutritional factors influencing the growth velocity. Programs to monitor food and nutrient intake, physical activity for glycemic control should be planned and implemented along with diabetic care of children.

Competing interests:

The authors wish to declare that there is no competing interest at any stage of the study and preparation of manuscript.

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