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Original Research Article

Growth Failure Factors and Stunting Prevalence in Children with Type 1 Diabetes: An Observational Study

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Abstract

Background: Children with Type-1 diabetes mellitus (T1DM) are most commonly found in India. Little is known about growth failure in children with diabetes, particularly in those with comorbidities and complications. Aim of this study is to determine the prevalence and predictors of stunting in children with T1DM.

Methods: This cross-sectional observation study was conducted at Pediatrics Department of SKMCH, Muzaffarpur, Bihar from June 2025 to November 2025. Total 125 children and adolescents aged 1–18 years with T1D were included this study. Demographic data, anthropometry, diet, sexual maturity rating, and biochemical measurements were performed using standard protocols. Short stature was defined as height for age Z-score ≤2. $p<0.05$ was considered statistically significant.

Results: 60 (48%) and 65 (52%) of the 125 children in the study were male and female, respectively. The children in the study group were 13.0±3.5 (1–18) years old on average, and they had had diabetes for 7.4±4.0 years on average. The average HbA1c for the children was 13.8±0.8%. In our group of children with T1D, we found that 20% of them had stunting. Children that were stunted had higher urine albumin creatinine ratios, poorer hemoglobin, lower midparental height Z-scores, and greater cholesterol. Stunting was significantly predicted by pre-existing comorbidities, worse renal function, prolonged disease duration, and short mid-parental height, according to binary logistic regression.

Conclusion: Of children with T1D, slightly less than one-sixth were short. Monitoring these youngsters' growth is crucial, particularly for those with short parents, long-term diabetes, pre-existing comorbidities, and declining renal function.

Keywords: Albumin-creatinine ratio, Children, Insulin-like growth factor 1, Midparental height, Stunting, Type-1 diabetes.

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Introduction

Diabetes is the most common pediatric endocrinologic disorder [1]. It is a disorder in which the pancreas is unable to produce enough insulin that is needed by different cells of the body to maintain normal glucose metabolism [1]. Be prevalence of diabetes has increased worldwide. Type 1 Diabetes mellitus (T1DM) is a global public health challenge [2]. According to the 2012 IDF atlas report, an estimated 480,000 children, aged 14 and under, are thought to have diabetes, with 77,000 new cases being diagnosed each year. T1DM has been increasing over time with a higher incidence in western countries than in Sub Saharan Africa [3]. Prevalence of diabetes has increased even faster in low resource settings, especially in urban areas [4]. Diabetes management in children is complicated, and requires good long-term care with close follow-up and compliance with treatment. Complications of diabetes include diabetic ketoacidosis (DKA), hypoglycemia, and long-term complication such as nephropathies, retinopathies, hypertension, and growth impairment

[5]. WHO reports that patients with diabetes require up to triple the healthcare resources when compared to those without diabetes [2]. Burden of diabetes is a significant issue, especially in the developing world where the cost of medication and access to laboratory tests make it heavy to bear given the underlying poverty and limited access to food [2]. Lot of improvement in diabetes management in the developed world over the last two decades, but there has been no or little improvement in developing world [6,7]. Children with diabetes are expected, with proper nutrition and care to attain normal nutritional status and growth [8,9].

One sign of poorly controlled diabetes is poor longitudinal growth and development. Growth impairment is a well-known complication of DM1 as described in several studies [5,8,10]. It is associated with poor glycemic control, along with other complications such as microalbuminuria, as described by Marcovecchio [10].

The Indian experience with paediatric diabetes is important to learn from, not only because of the high burden of diabetes (likely prevalence 5 million diabetic children and adolescents; of which 3–4 million face poverty along with diabetes) but also because of problems like death before diagnosis (due to missed diagnosis), poor management because of low awareness and high costs, limited availability of insulin and poor cold chains in rural areas, limited availability of blood glucose strips, yet greater family support, as in many developing countries of Asia and Africa.

In developing countries, medical training pays little attention to chronic disorders and long term care, since the focus is mainly on infections and other acute illnesses, vaccinations, reproductive issues, and nutrition. Other major limitations are poor infrastructure and low public expenditure on health, leading to significant out-of-pocket expenses by individuals.[11] Poor care and delayed or no prevention strategies lead to more complications. Due to these inadequacies in the medical care system, the patient and family are trapped in a vicious cycle of increased acute and chronic complications, thus further elevating costs, mortality, and absenteeism from work and hence decrease in income, employability, and quality of life, further pushing the family into indebtedness and poverty.

Material and Methods

This was an observational, cross-sectional study conducted for six months (June 2025 to November 2025), children with T1D aged 1 to 18 and their parents attended the pediatric department of Sri Medical College and Hospital in Muzaffarpur, Bihar.

The study period was when they were approached. In order to evaluate the impact of complications and comorbidities on growth failure, the study included all patients who were admitted to the hospital throughout the study period and consented to participate. Written informed consent was given to parents, and children agreed to participate in the study. The study involved one hundred and twenty-five children. Using standardized questionnaires, a pediatrician collected data on the patients' age, age at diabetes diagnosis, duration of diabetes, number of admissions for ketoacidosis, current medications, birth weight, health history, family history, type of insulin regimen, and daily total insulin dose. The parents' medical history was confirmed using hospital medical data. A pediatric endocrinologist conducted Tanner staging for sexual maturity. Three categories of pubertal staging were established: prepubertal, pubertal, and postpubertal. Using validated activity questionnaires adapted for Indian adolescents, physical activity was measured. A portable stadiometer (Leicester Height Meter, Child Growth Foundation, UK) was used to measure the patients' and parents' standing height to the nearest millimeter, and an electronic scale was used to assess their weight to the nearest 100 g. Weight in kilograms was divided by height in meters squared to calculate the body mass index, or BMI. Then, using Indian norms, the height, weight, and BMI were converted to Z scores.

The 1-day dietary recall method was used to capture dietary data on one week day, one holiday, and a mean was calculated. To provide a precise estimate of the meals ingested, trained nutritionists conducted interviews with the children and their primary caregivers. The C Diet prepared food database program was then used to calculate nutrient intakes.

For each nutrient, the percentage of the recommended dietary allowance (RDA) for Indian children was calculated in order to determine the adequacy of nutrient intakes. Glycosylated hemoglobin (HbA1c) was measured in order to assess glucose management. A pediatric phlebotomist took a 5-milliliter fasting blood sample between 7 and 9 in the morning. High performance liquid chromatography was used to measure HbA1c. The Horiba Yumizen H500 hematology analyzer was used to quantify hemoglobin using spectrophotometry at a wavelength of 555 nm. The lipid profile (total cholesterol, triglycerides, and HDL-C) of the fasting blood samples was then evaluated by the enzymatic method, and the Friedewald formula was used to determine the quantities of low-density lipoprotein cholesterol. Urine microalbumin, creatinine, and urine albumin: creatinine ratio (ACR) were measured in the first voided urine

sample, which was taken in a sterile container. The ratio of urine albumin to urine creatinine (the concentrations of urinary albumin and creatinine were determined using radioimmunoassay and Jaffe's method, respectively) was used to calculate the results. Samples were not collected after intense exercise, during menstruation, during fever, or in cases of severe hyperglycemia. If ACR was in the range between 0 and 30 ug/mg, it was considered normal.

An altered ACR was defined as a single high value of early morning urine ACR (more than 30 ug/mg). A solid-phase enzyme-linked immunosorbent assay with an intraassay coefficient of variation (CV) of 4.7% and an interassay CV of 7.2% was used to measure the quantities of serum IGF1. Next, using the reference data that was available, the IGF1 concentrations were translated into Z scores. The SPSS for Windows software, version 26 (SPSS, Chicago, IL, USA), was used for all statistical analyses. Prior to conducting statistical analysis, the normality of each outcome variable was examined. Student's t-test for parametric data, Mann-Whitney U-test for non-parametric data, and Chi-square test for categorical variables were used to test for mean differences. Binary logistic regression analysis was used to examine the connections between continuous predictors and dichotomous-dependent variables. Stunting was the model's dependent variable, and binary logistic regression analysis was used to examine the associations between dichotomous dependent variables and continuous predictors.

Stunting was the dependent variable in the model, and the independent variables were glycemic control (HbA1c), diabetes duration, tanner stage (categorized into two groups: pre-pubertal and in puberty/post-pubertal), midparental height Z scores, and comorbidities/complications like lipids, hypothyroidism, and altered ACR. The threshold for statistical significance was $p<0.05$.

Results

Sixty (48%) of the 125 children in the study were male, and sixty-five (52%) were female. The children in the study group had an average age of 13.0±3.5 (1–18) years, and they had had diabetes for an average of 7.4±4.0 years. The HbA1c of the children was 13.8±0.8% on average. We found that 20% of the children in our T1D cohort had stunting. Greater urinary albumin creatinine ratios, poorer hemoglobin, lower midparental height Z-scores, and greater cholesterol were all observed in stunted children.

Table 1: Clinical/laboratory findings of patients classified by stature

Parameter	Stunting(n=25)	No stunting(n=100)	p-value
Age (years)	13±3.5	11.6±4	0.020*
Diabetes duration (years)	7.4±4	4.5±3.1	0.000*
Height Z scores	–2.7±0.6	–0.6±0.9	0.000*
(20% stunted)			
Weight Z scores	–1.8±0.8	–0.6±0.9	0.128
BMI Z scores	–0.6±0.9	–0.4±0.9	0.000*
Midpaternal height Z scores	–1.3±0.8	–0.8±0.7	0.000*
Physical activity (min/week)	292.5±224.9	301.5±226.5	0.788
Percent RDA energy (%)	68.9±27.3	72.8±31.8	0.391
Percent RDA protein (%)	104.6±54.1	115.9±60.6	0.204
Percent RDA zinc (%)	66.9±52.3	67.9±47.4	0.884
Insulin (unit/kg/day)	1.2±0.4	1.1±0.3	0.168
Creatinine (mmol/L)	0.1±0.0	0.1±0.0	0.048*
HbA1c (mmol/L)	13.8±0.8	13.4±0.8	0.308
Vitamin D (ng/ml)	19.7±9.1	18.9±9.1	0.576
Hemoglobin (g/dl)	12.9±1.3	13.4±1.5	0.020*
Total cholesterol (mmol/L)	4.3±1.0	3.9±0.9	0.004*
LDL cholesterol (mmol/L)	1.0±1.0	1.0±0.8	0.366
Albumin Creatinine ratio (ug/mg)	242.1±698.1	29.3±76.7	0.000*

***p<0.05, all normal variables are mentioned as mean±standard deviation, all non-normal variables are expressed as median (interquartile range) (BMI: Body mass index, HbA1c: Glycated hemoglobin, LDL: Low-density lipoprotein, NS: Not significant)**

Children with T1D, whether or not they were stunted, were the dependent variable in our logistic regression analysis, and the independent variables were the duration of diabetes, physical activity and dietary intake (energy percentage of the recommended daily allowance), midparental height Z-scores, tanner stage, comorbidities, urine ACR, serum cholesterol, HbA1c, and IGF-1 Z-scores. Stunting was found to be significantly predicted by binary logistic regression in children with

comorbidities (such as hypothyroidism, vitiligo, and celiac disease), children with impaired renal function, longer disease duration, children who had achieved puberty, and children with short midparental height ($p<0.05$).IGF-1, HbA1c, cholesterol, and dietary calorie intake were not significant predictors ($p>0.05$), nor were other variables like physical activity.

Table 2: Binary logistic regression to determine the predictors of stunting

Parameter	OR	Wald	Significant	95% CI for EXP (B)	
				Lower	Upper
Duration of diabetes	1.452	20.75	0.000*	1.237	1.704
Midparental height Z-scores	3.597	12.307	0.000*	1.76	7.352
Tanner stage	0.154	8.172	0.004*	0.043	0.555
Children having comorbidities (hypothyroidism, vitiligo, and celiac disease)	4.297	3.594	0.05*	0.952	19.404
Albumin: creatinine ratio	5.751	8.216	0.004*	1.739	19.022
Cholesterol	0.652	0.271	0.603	0.13	3.269
Vitamin D	0.731	0.441	0.507	0.29	1.844
HbA1c	1.61	0.886	0.347	0.597	4.344
IGF1_zscores	0.702	2.247	0.134	0.443	1.115
Sports duration (weekly/min)	1	0.01	0.921	0.998	1.002
Percent RDA_energy	0.999	0.024	0.878	0.98	1.018
Duration of diabetes	1.452	20.75	0.000*	1.237	1.704
Constant	0.004	14.2	0		

***p<0.05. HbA1c: Glycated hemoglobin, IGF1: Insulin-like growth factor1, Percent RDA_energy: Percentage Recommended Dietary Allowance**

Discussion

One of the most prevalent chronic pediatric endocrine disorders is type-1 diabetes mellitus (T1D), a condition in which the pancreatic ß-cells are unable to produce enough insulin to sustain normal glucose metabolism. [12] According to the 10th edition of the International Diabetes Federation Atlas, 1,211,900 children and adolescents under the age of 20 worldwide are estimated to have type 1 diabetes. Populations of North European descent and a number of Middle Eastern and North African nations have the highest age-standardized occurrences of T1D. The two nations with the greatest estimated incident instances of T1D are the United States and India, followed by a number of other highly populated nations. [13]

Thus, T1D and its side effects, such as stunted growth and small stature in children, continue to be a serious concern. [14] A child is considered to have stunting or short stature if their height or length is less than two standard deviations (i.e., less than the 2.3 percentile) for their age and gender.

Low height-for-age, or stunting, is regarded as a key indicator of both chronic undernutrition and the nutritional status of children and adolescents. [15] 60 (48%) and 65 (52%) of the 125 children in the study were male and female, respectively. The children in the study group were 13.0±3.5 (1–18) years old on average, and they had had diabetes for 7.4±4.0 years on average.

The average HbA1c for the kids was 113.8±0.8%. In our group of children with T1D, we found that 20% of them had stunting. Children that were stunted had higher urine albumin creatinine ratios, poorer hemoglobin, lower midparental height Z-scores, and greater cholesterol. Children with T1D, whether or not they were stunted, were the dependent variable in our logistic regression analysis, and the independent variables were the duration of diabetes, physical activity and dietary intake (energy percentage of the recommended daily allowance), midparental height Z-scores, tanner stage, comorbidities, urine ACR, serum cholesterol, HbA1c, and IGF-1 Z-scores. Stunting was significantly predicted ($p<0.05$) by binary logistic regression for children with comorbidities (e.g., hypothyroidism, vitiligo, and celiac disease), children with impaired renal function, children with longer duration of disease, children who had reached puberty, and children with short midparental height. IGF-1, HbA1c, cholesterol, and dietary calorie intake were not significant predictors ($p>0.05$), nor were other variables like physical activity. HbA1c's pubertal peak is probably caused by behavioral reluctance to changing one's lifestyle as well as physiological changes including increased insulin resistance throughout puberty [16]. [17] We also examined the relationship between child-specific physiological traits and individual variations in pubertal height growth. The timing of the growth spurt—later diagnosis and longer duration were independently linked to larger height and later

puberty—as well as the course of the condition, namely the age at diagnosis and the period since diagnosis, influenced mean height. But there was a significant sex difference: girls were more affected in terms of size, especially with mean HbA1c, where being 2 cm shorter was linked to a 1 SD rise in HbA1c.In terms of timing and severity, boys were more affected, and this was positively correlated with both the disease's length and the age at diagnosis. Accordingly, T1DM generally has the effect of decreasing height in girls while slowing and delaying growth in boys. As stated by Dunger et al. [18], this observed difference may be significant because pubertal growth in malesand females is dependent on growth hormone and testosterone, respectively. Although they did not note any sex differences, a recent investigation by Blasetti et al. [19] likewise revealed that shorter patients had a higher HbA1c SDS.

Glycemic management did not contribute to stunting in children with type 1 diabetes, according to our research.

The Royal Children's Hospital (RCH) in Melbourne, Australia, conducted a study that found no significant correlation between the linear development of children with T1D and metabolic management. [20] Contrary to our findings, a study carried out in the Kingdom of Jeddah, Saudi Arabia, between June and August of 2017 revealed no correlation between the length of diabetes and the Z-scores for height, weight, or BMI.

Additionally, they note a relationship between height Z scores and HbA1c levels. The exclusion of children with celiac disease, hypothyroidism, and familial short stature may help to explain this. [21, 22]

Conclusion

According to our stidy, slightly less than one-sixth of kids with T1D were short. It is crucial to keep an eye on these patients' growth, particularly in those with short parents, long-term diabetes, pre-existing comorbidities, and declining renal function.

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