Growth assessment of children and adolescents with type 1 diabetes mellitus at Steve Biko Academic Hospital in Pretoria, South Africa: A cross-sectional study of factors contributing to stunting

C J Badenhorst,[1] MB ChB; **P Rheeder**,[2] PhD; **M Karsas**,[1] FC Cert Paed Endocrinology & Metabolism (SA)

[1] *Department of Paediatrics, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa*
[2] *Department of Internal Medicine, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa*

Corresponding author: *C J Badenhorst (c.badenhorst18@gmail.com)*

Background. The continuous rise in the prevalence of type 1 diabetes mellitus (T1DM) places pressure on countries to increase the resources needed to manage patients adequately. Studies have documented an association between poor glycaemic control and stunting, which poses a significant health concern. Minimal data are available on the prevalence of stunting in T1DM in South Africa.
Objective. To establish the prevalence of stunting in a population of paediatric T1DM patients and determine contributing factors.
Methods. A descriptive, cross-sectional study was conducted at the Paediatric Diabetes Clinic of the Steve Biko Academic Hospital, Pretoria. Data were collected from patient files, a questionnaire and the National Health Laboratory Service database. Stunting was defined as height-for-age *Z-score* (HAZ)<−2.
Results. Of the 169 recruited patients, 115 were included (56.5% female). The prevalence of stunting was 10.4%. The median haemoglobin A1c (HbA1c) was 11.8%. Stunting was significantly associated with poor glycaemic control (p=0.008), older age (p=0.039), presence of comorbidities (p=0.026), underweight (p=<0.001) and food insecurity (p=0.021). Genetic factors were also associated with stunting, specifically lower paternal height (p=0.006) and decreased mid-parental height *Z-score* (p=0.035).
Conclusion. Stunting in children and adolescents with T1DM was associated with poor glycaemic control, nutritional and socioeconomic factors, comorbidities, older age and genetic factors. The results point to a multifactorial contribution to impaired growth. To promote growth in children with T1DM, a multidisciplinary approach is essential, with a focus on optimising glycaemic control, addressing nutritional status and food insecurity, managing comorbidities and monitoring growth against genetic potential regularly.

Keywords: Type 1 diabetes mellitus; stunting; growth; glycaemic control; genetic growth potential.

S Afr J Child Health 2026;20(1):e3234. https://doi.org/10.7196/SAJCH.2025.v20i1.3234

Type 1 diabetes mellitus (T1DM) is the most common type of diabetes in children and adolescents.[1] Globally, 1.1 million children and adolescents are patients living with T1DM, expected to increase by 3 - 5% annually.[2] This continuous rise in prevalence places pressure on countries to increase resources to manage diabetic patients adequately. In 2019, the International Diabetes Federation estimated that 25 000 children in Africa had T1DM,[3] with an incidence ranging from 1.5 to 10.1 per 100 000.[4] This number is thought to be underestimated owing to a lack of data from many African countries. In South Africa (SA), little data on the prevalence and complications in patients diagnosed with T1DM is available.

T1DM is an autoimmune disease in which the pancreas produces inadequate amounts of insulin, resulting in disrupted glucose metabolism.[5] Numerous acute complications (e.g. hypoglycaemia, diabetic ketoacidosis, DKA) and chronic complications relating to microvascular pathology (e.g. retinopathy, nephropathy and neuropathy) and macrovascular conditions (cardiovascular and cerebrovascular diseases) are associated with poorly controlled T1DM.[5] T1DM is irreversible and is managed through self-management, which involves administering exogenous insulin, monitoring blood glucose levels and considering dietary, exercise-related, psychological and socioeconomic factors.[6]

A long-term consequence of poorly controlled T1DM is its negative effect on longitudinal growth.[2,7,8] Stunting is defined as a height-for-age Z-score (HAZ) below −2 SD.[6] Factors known to affect growth include gender, genetic environment, age at diagnosis, diabetes duration, puberty, glycaemic control and the regulation of the growth hormone–insulin-like growth factor (GH-IGF) axis.[9] Dysregulation of this axis in T1DM is well described and is characterised by decreases in circulating IGF-1, IGF binding protein-3 (IGFBP-3) and GH binding protein (GHBP), along with an increase in GH and IGFBP-1.[9] With adequate glycaemic control, patients are expected to attain normal growth owing to the restoration of GHBP.[9] Several studies have documented a decline in height standard deviation (SD) scores relative to population norms from diagnosis to puberty, indicating reduced growth velocity compared with healthy peers.[2,7-10] In a study from Germany, the entire cohort declined by 0.41 SD in length over a disease period of 9.1 years.[10] Patients with poorer glycaemic control (mean haemoglobin A1c [HbA1c] levels >8.0%) had a decreased adult height of −0.31 SD.[8]

Stunting is a significant health concern owing to the associated health and economic consequences for the individual, the household and the community.[11-14] Children who are stunted typically perform poorly in school and may be unable to gain access to further

education, thus continuing the vicious cycle of poverty and food insecurity.[15] Food insecurity is of concern because adequate and appropriate nutrition is vital for managing diabetes mellitus.[2,6,8] Stunting is associated not only with increased morbidity and mortality but also with reduced mental development,[16,17] resulting in lower productivity as well as physical impairments.[5,11] Countries with a high burden of stunting are therefore at a greater risk of having an underdeveloped human capital and society.[11,16]

Understanding the long-term dynamic of stunting in T1DM-affected children and adolescents in SA is important to efficiently orient public health interventions needed to improve glycaemic control and limit the consequences of stunting. Given the impact of stunting on the health of the individual and economic development, data are required to formulate appropriate policies and programmes to address stunting in T1DM in SA.

Methods

A descriptive, cross-sectional analysis was conducted among children with T1DM who attended the paediatric diabetes clinic at Steve Biko Academic Hospital (SBAH) in Pretoria, Gauteng. Patients have access to a paediatric endocrinologist, paediatricians and nurses at a weekly paediatric outpatient clinic. At follow-up visits, routine measurements (blood pressure, heart rate, glucose level, weight, height) are recorded. Patients receive the required medical supplies and laboratory investigations are performed, if indicated. Patients are re-evaluated every 2 weeks to 6 months, depending on how well their condition is controlled.

Data collection

All children with T1DM seen at the SBAH Paediatric Diabetes Clinic from March 2022 to June 2023 were included, except those without consent from their guardians. Data were collected using a data collection tool and also obtained from the National Health Laboratory Service (NHLS) database. A questionnaire was used to assess socioeconomic and -demographic factors such as: the patient's age and gender; hospital classification category (based on household income); parents' level of education; number of meals per day; self-evaluation of nutritional adequacy; parental height; the presence of comorbidities; and the affordability of extra supplies. Data about the patient's treatment options, disease duration, required insulin dose, pubertal status and anthropometric measurements (height and weight) were obtained from patient files.

Stunting was determined according to the gold standard – mean height-for-age *Z*-score (HAZ) – which was calculated using AnthroCalc 2.7.1, a child growth assessment app that bases its calculations on established growth references such as the World Health Organization 2007 standards. Stunting was defined as height for age being >2 SD below the mean (gender matched).[18-21]

The mid-parental height (MPH) was also calculated to rule out standard variations of growth due to underlying genetic factors. MPH was calculated based on the height of the parents, using the following formulae:[20]

- Boys: (father's height in centimetres + mother's height in centimetres + 13 cm)/2
- Girls: (father's height in centimetres + mother's height in centimetres –13 cm)/2.

Parental heights were recorded either by direct measurement during clinic visits or, if parents were absent, from the height listed on their official identification documents. AnthroCalc 2.7.1 was also used to calculate the MPH and subsequent target-height *Z*-score.

The most recent HbA1c was obtained from the NHLS data system. Poor glycaemic control classification was based on an HbA1c level of 7.5% or higher.

Statistical analysis

The statistical analysis was conducted on de-identified electronic records. Data were captured and analysed using Microsoft Excel and R statistical software (2024).[22] Summary statistics were generated and a comparative analysis was performed between stunted and non-stunted children to identify contributory factors. These included: glycaemic control (HbA1c, frequency of admissions); age; gender; duration of illness; pubertal status; nutritional status; socioeconomic factors (parents' level of education, level of income, food insecurity); comorbid conditions; insulin regimen; and genetic growth potential (parents' individual heights and MPH). Associations between stunting and T1DM, socioeconomic and clinical variables were assessed using χ^2 or Fisher's exact tests for categorical data, and the Wilcoxon rank sum test for continuous data that were not normally distributed. Group means were compared using the independent-samples *t*-test. A significance level of $p<0.05$ was used.

Ethical considerations

Ethics approval was obtained from the University of Pretoria Research Ethics Committee (ref. no. 68/2022) and the National Health Research Database.

Results

A total of 169 children were recruited for this study, of whom 53 were excluded due to a lack of consent for participation from their caregivers. In addition, one child was excluded owing to incomplete data on file. Of the final 115 children included, 65 (56.5%) were female. Overall, 10.4% (n=12) were found to be stunted based on the gold standard (HAZ<–2). The age of patients seen at the clinic ranged from 1.6 to 17.9 years; median (IQR) age was 11 (8.1 - 13.4) years. The median (IQR) duration of T1DM was 3 (1.7 - 5.9) years; most patients (68.7%) had diabetes for <5 years. The stunted and non-stunted groups were not significantly different with regard to gender (p=0.3), age at diagnosis (p=0.6) or duration of T1DM (p=0.3). However, a significant difference in the current age of patients was found between the stunted and non-stunted groups: mean (SD) age = 12.1 (5.6) years v. 10.3 (3.3) years (p=0.039) (Table 1).

Anthropometry

Mean (SD) body mass index was not significantly different between the stunted (18.9 (3.6) kg/m^2) and the non-stunted groups (19 (5.3) kg/m^2) (p=0.7) (Table 2). However, significant differences with regard to weight for age were noted between those classified as underweight (*Z*-score<–2) in the stunted and non-stunted groups: mean (SD) *Z*-score = –1.96 (1.26) v. 0.23 (1.36) (p<0.001). The father's height was significantly different between the stunted and the non-stunted groups (mean (SD) height = 165 (3) cm v. 169 (7) cm; p=0.006), as were the MPH *Z*-scores (–0.46 (0.37) v. –0.20 (0.43); p=0.035). No significant differences were noted with regard to the mother's height or MPH *Z*-scores (p=0.6 and p=0.8, respectively).

Complications and comorbidities

No significant differences were noted between the stunted and non-stunted groups with regard to previous hospital admissions related to T1DM (p=0.8) or recurrent admissions for DKA (p=0.5) (Supplementary Table 1). Close to half of the patients (n=54, 47%) reported no previous hospital admissions. About a quarter (n=31, 27%) reported an admission more than a year ago, 10.4% (n=12)

Table 1. Patient demographics (*N*=115)

Characteristic	All (*N*=115), *n* (%)	Non-stunted (*N*=103), *n* (%)	Stunted (*N*=12), *n* (%)	*p*-value*
Male	50 (43.5)	43 (41.7)	7 (58.3)	0.3
Age of diagnosis (years), mean (SD)	6.5 (3.4)	6.4 (3.4)	7.1 (4.2)	0.6
Current age (years), mean (SD)	11.2 (3.2)	10.3 (3.3)	12.1 (5.6)	0.039
Duration of T1DM (years), median (IQR)	3 (1.7 - 5.9)	3 (1.7 - 5.5)	4.8 (2.2 - 7.3)	0.3
Tanner stage				
Pre-pubertal (Tanner I)	58 (50.4)	54 (52.4)	4 (33.3)	0.2
Pubertal (Tanner II - V)	57 (49.6)	49 (47.6)	8 (66.7)	

SD = standard deviation; T1DM = type 1 diabetes mellitus; IQR = interquartile range

* Determined using χ^2 test or Fisher's exact test for categorical variables, the independent-samples t-test for normally distributed continuous variables and the Wilcoxon rank-sum test for non-normally distributed continuous variables.

Table 2. Anthropometric measurements

Characteristic	All (*N*=115), mean (SD)	Non-stunted (*N*=103), mean (SD)	Stunted (*N*=12), mean (SD)	*p*-value*
Height (cm)	137 (20)	139 (18)	128 (30)	0.4
Weight (kg)	38 (18)	38 (18)	34 (18)	0.7
BMI (kg/m^2)	21.1 (9.5)	19.0 (5.3)	18.9 (3.6)	0.7
Height for age (*Z*-score)	–0.49 (1.44)	–0.17 (1.10)	–3.22 (1.16)	<0.001
Weight for age (*Z*-score)	–0.003 (1.5)	0.23 (1.36)	–1.96 (1.26)	<0.001
BMI for age (*Z*-score)	0.36 (1.5)	0.42 (1.54)	–0.10 (1.13)	0.3
Father's height (cm)	168 (6)	169 (7)	165 (3)	0.006
Mother's height (cm)	167 (6)	167 (6)	165 (6)	0.6
MPH (cm)	167 (8)	167 (8)	167 (9)	0.8
MPH (*Z*-score)	–0.26 (0.43)	–0.20 (0.43)	–0.46 (0.37)	0.035

SD = standard deviation; BMI = body mass index; MPH = mid-parental height

* Determined using χ^2 test or Fisher's exact test for categorical variables, the independent-samples t-test for normally distributed continuous variables and the Wilcoxon rank-sum test for non-normally distributed continuous variables.

were admitted 6 - 12 months ago and 15.7% (*n*=18) were admitted 1 - 6 months ago. Thirty-five patients (30.4%) were found to have comorbidities, with a significant difference noted between the stunted and non-stunted groups (*p*=0.026). Comorbidities were mostly related to cardiovascular conditions (*n*=13, 11.3%). Six patients (5.2%) had comorbidities of endocrine origin, five (4.3%) of neurological origin and four patients (3.5%) had musculoskeletal or skin-related comorbidities. Coeliac disease was seen in three patients (2.6%). Two patients (1.7%) had other conditions (asthma, gastroesophageal reflux disease). Microalbuminuria was noted in four patients (3.5%). Two patients (1.7%) had genetic syndromes (Down syndrome, Waardenburg syndrome) and one patient had Mauriac syndrome. (Supplementary Fig. 1) No single comorbidity was associated with a risk for stunting in this cohort, despite the presence of comorbidities as a whole being found to be a significant risk factor. Some patients had more than one of the above comorbidities, but this was of no statistical significance on further analysis (Supplementary Table 1).

Treatment types

Insulin regimens were as follows: 78 patients (67.8%) received basal-bolus insulin therapy consisting of neutral protamine Hagedorn(NPH) insulin as basal insulin with regular insulin as boluses; 35 (30.4%) received premixed insulin (Actraphane) twice daily; and two (1.7%) were on continuous subcutaneous insulin infusion (CSII) (Supplementary Table 2). The average total daily dose of insulin used was 1.4 units/kg (range: 0.1 - 3 units/kg). The majority of patients (66.1%, *n*=76) received a daily insulin dose of 1 - 1.9 units/kg, with 23.5% (*n*=27) receiving doses of <1 unit/kg. Twelve patients (10.4%) received doses of 2 - 3 units/kg. No significant differences were noted between the stunted and non-stunted groups regarding treatment type (*p*=0.8) or total daily dose of insulin (*p*=0.7). The mean (SD) HbA1c was 12.3% (3.04%) (range: 5.1 - 17.5%). A significant difference in HbA1c level was noted between the stunted and non-stunted groups (*p*=0.008). Further analysis showed that very poor glycaemic control (HbA1c>9%) was a significant risk factor for developing stunting compared with better control (HbA1c≤9%) (*p*=0.021), whereas simply poor control (HbA1c=7.5 - 9%) compared with adequate control (HbA1c<7.5%) was not a significant risk factor (*p*=0.327) (Supplementary Table 2).

Management and socioeconomic factors

Approximately a third of patients (n=40, 34.8%) reported that they were able to afford managing their condition. There was no statistically significant difference between the stunted and non-stunted groups in the proportion of patients reporting cost-related barriers to diabetes management (*p*=0.5) (Table 3). Cost-related factors included: the need to purchase additional strips (*n*=74, 64.3%); lack of money to purchase nutritious meals (*n*=39, 33.9%); transportation costs (*n*=17, 14.8%); and purchasing additional batteries for their glucometer (*n*=8, 7%). Further analysis showed that not having enough money for buying nutritious meals was a significant risk factor for stunting (*p*=0.021). Adequate meal intake (at least three meals and two snacks) was reported by 66 patients

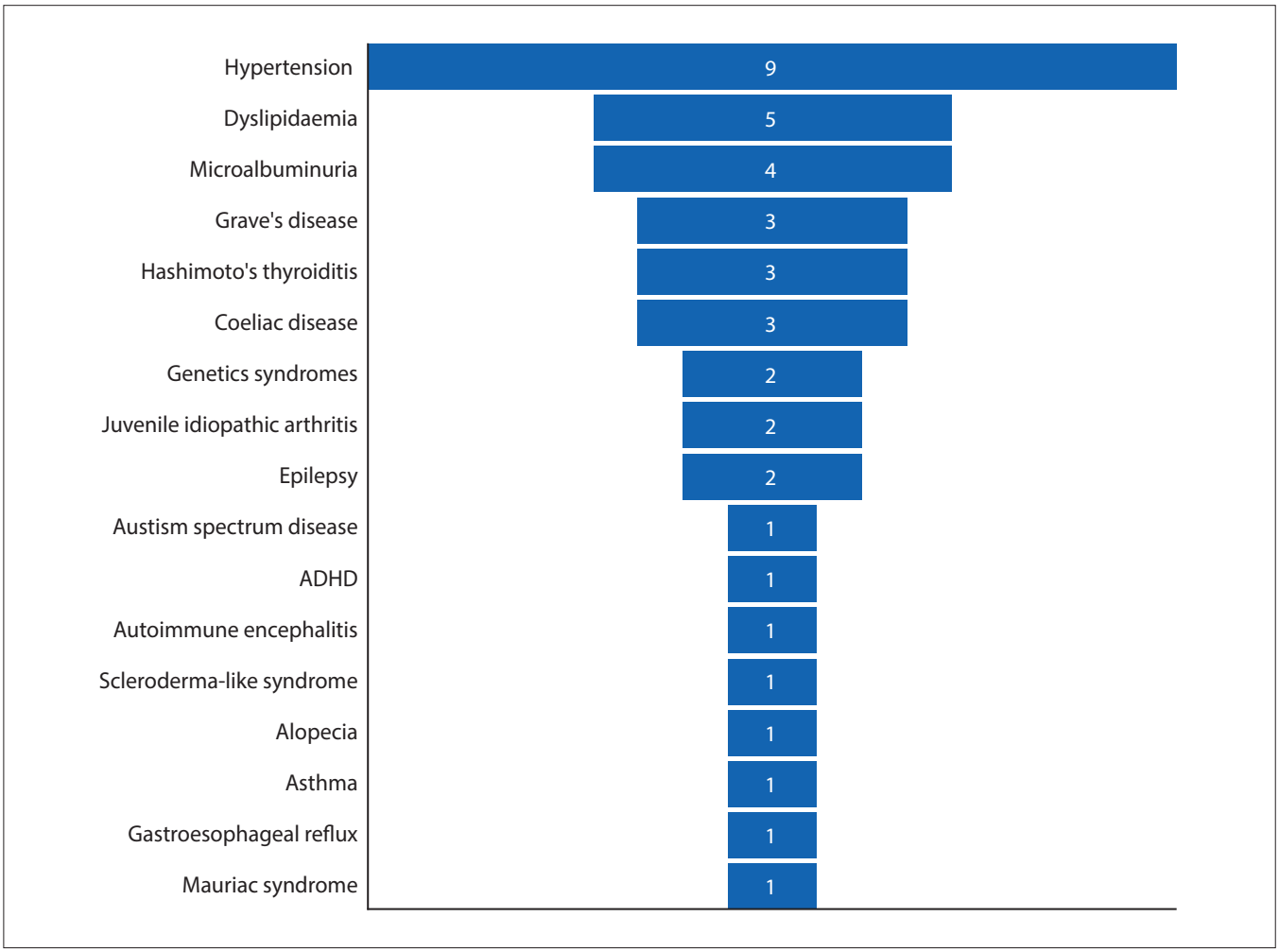

Fig. 1. Comorbidities/complications associated with T1DM.

(57.4%); 36.5% (*n*=42) had access to two or three meals per day and 6.1% (*n*=7) had at most one meal per day (Table 3). No significant differences were noted between the stunted and non-stunted groups with regard to the effect of adequate meal intake on stunting (*p*=0.3).

According to income-based categories for hospital care in the public sector, the majority of patients (*n*=55, 47.8%) were from families or households classified as H0, meaning they were eligible for full subsidisation (grant holders, pensioners and the unemployed), whereas a tenth (*n*=12, 10.4%) were from the full-paying patient (PF) category (including foreigners, private patients and those on medical aid). All remaining patients were classified as H1, H2 or H3, meaning they were eligible for partial subsidisation to some extent (Table 3 for a description of income categories). Household income was not significantly different between the stunted and non-stunted groups (*p*=0.4). Most parents reported their highest level of education to be secondary education (*n*=79, 68.7%), whereas 28.7% (*n*=33) reported having a tertiary level of education and 2.6% (*n*=3) only primary education. No significant differences were noted between the stunted and non-stunted groups with regard to parents' level of education (*p*=0.1) (Table 3).

Discussion

T1DM is the most common endocrine disease seen in children and adolescents.[1] With proper nutrition and treatment, normal growth should be attainable.[7] Owing to increased morbidity and mortality and reduced cognitive development associated with stunting, it is a major concern with regard to health, social and economic impact.[11–14] Stunting can also reflect poor general health and diabetes control in a population.[11,17] The prevalence of stunting among children under five in SA has shown a slight decline from 25% in 2008 to 23% in 2017.[23]

Although previous reports suggest greater male vulnerability to stunting in T1DM patients in sub-Saharan Africa,[7,24] our study found no gender association (*p*=0.3) (Table 1). Instead, lower paternal height and low MPH *Z*-scores were significant risk factors (*p*=0.006 and *p*=0.035, respectively), highlighting genetic contributions; interestingly, maternal height does not seem to be a risk factor (*p*=0.6). Further research is needed on paternal and genetic factors to clarify their role in male vulnerability to stunting in sub-Saharan Africa, both for T1DM patients and in general. Four of the 12 stunted patients (33.3%) had low MPH *Z*-scores, suggesting a contribution from genetic short stature rather than pathological stunting. This strengthens the idea of multimodal reasons for stunting and the importance of MPH calculations as an added parameter in growth assessment in children.

Poor glycaemic control was found to be strongly associated with stunting (*p*=0.008), with a mean HbA1c of 13.96% in the stunted group versus 12.3% overall (Supplementary Table 2). Similar findings in children with T1DM were reported in Kenya, where the mean HbA1c was 11.1%.[7] On further analysis, only very poor glycaemic control (HbA1C>9%) significantly increased stunting risk. (*p*=0.021).

Stunting prevalence in this cohort was lower than the 30.9%

Table 3. Socioeconomic factors with regard to T1DM management

Characteristic	All (*N*=115), *n* (%)	Non-stunted (*N*=103), *n* (%)	Stunted (*N*=12), *n* (%)	*p*-value*
Availability of daily meals				
1 meal	7 (6.1)	7 (6.8)	0 (0)	0.3
2 - 3 meals	42 (36.5)	35 (34)	7 (58.3)	
>3 meals	66 (57.4)	61 (59.2)	5 (41.7)	
Treatment affordability	40 (34.8)	37 (35.9)	3 (25)	0.5
Cost-related factors contributing to the unaffordability of overall diabetic management				
Lack of strips	74 (64.3)	65 (63.1)	9 (75)	0.5
Lack of meal availability	39 (33.9)	31 (30.1)	8 (66.7)	0.021
Lack of funds for transport	17 (14.8)	14 (13.6)	3 (25)	0.4
Lack of batteries	8 (7.0)	8 (7.8)	0 ()	>0.9
Parents' highest level of education				
Primary	3 (2.6)	2 (1.9)	1 (8.3)	0.1
Secondary	79 (68.7)	69 (67)	10 (83.3)	
Tertiary	33 (28.7)	32 (31.1)	1 (8.3)	
Hospital classification†				
H0	55 (47.8)	47 (45.6)	8 (66.7)	0.4
H1	40 (34.8)	38 (36.9)	2 (16.7)	
H2	7 (6.1)	7 (6.8)	0 (0)	
H3	1 (0.9)	1 (1.0)	0 (0)	
PF	12 (10)	10 (9.7)	2 (16.7)	

* Determined using χ^2 test or Fisher's exact test for categorical variables and independent-samples t-tests for continuous variables.

†Income categories for hospital care: H0 = full subsidisation (Grant holders, pensioners and the unemployed); H1 = partial subsidisation (<R70 000 single income or R100 000 family income per year); H2 = partial subsidisation (R70 000 - R100 000 single income or R250 000 - R350 000 family income per year); H3 = partial subsidisation (>R250 000 single income or >R350 000 family income per year); PF = full-paying patient (foreigners, private patients with medical aid).[22]

reported in children with T1DM in Rwanda,[7] likely owing to the younger average age of participants in our study (11.2 years v. 14.3 years in the Rwandan study). Although other studies have found associations between stunting and a longer T1DM duration as well as older age at diagnosis,[7,9] no such links were seen here. In the study from Rwanda,[7] the mean duration of T1DM was 2 years, compared with 4.1 years in our cohort (median 3 years). The mean age at diagnosis (6.5 years) aligns with a known T1DM peak (4 - 7 years).[25]

However, current age was found to be a risk factor in our study, with older children more likely to be stunted (the mean age at diagnosis in the stunted group was 12.1 years compared with 10.3 years in the non-stunted group; p=0.039) (Table 1). Although older age is expected to be associated with advanced pubertal stage, advanced pubertal status was not a significant risk factor in this cohort (p=0.2). This suggests that, despite older age being associated with increased stunting risk, the short stature observed may reflect a combination of delayed pubertal development and cumulative effects of chronic poor glycaemic control rather than pubertal stage alone, highlighting the complex interplay between age, growth and metabolic control in T1DM.

In the case of systemic disorders, growth may be further impaired owing to increased energy needs or inadequate nutrition.[26] Two patients had known genetic syndromes impacting growth, namely Down syndrome and Waardenburg syndrome, although neither patient met stunting criteria. One patient was found to have Mauriac syndrome, which is the most extreme form of growth failure in T1DM. It is characterised by short stature, an enlarged liver, limited joint mobility, tight, waxy skin, growth maturation delay, moon facies, protuberant abdomen and proximal muscle wasting, and complications of retinopathy and nephropathy are frequently evident.[17] Although it is observed in patients with poor glycaemic control, only a small portion of patients develop Mauriac syndrome.[17] It is postulated that other risk factors are associated with the development of this syndrome.

Although poor glycaemic control is linked with complications or comorbidities and recurrent hospital admissions,[9] no association was found between stunting and prior T1DM complications or recurrent DKA in this cohort (p=0.8 and p=0.5, respectively) (Supplementary Table 1). Nearly a third of the patients (30.4%) had T1DM-related comorbidities or complications, most commonly cardiovascular disease (9.6%, n=11). The prevalence was similar to what has been reported from a study in India in a similar cohort (8 - 15%).[9] The presence of comorbidities as a group was a significant risk factor for developing stunting (p=0.026), although no individual condition was associated with increased risk.

In T1DM, administering insulin daily is essential, with doses varying according to age, puberty, stress and individual factors.[5,6] Standard doses range between 0.5 and 1 units/kg/day,[6] but in this cohort higher doses (on average 1.4 units/kg/day) were seen. In contrast, Wiegand *et al.*[27] reported lower requirements (0.67 units/kg/day in prepuberty; 0.93units/kg/day in adolescence; 0.7 units/kg/day in adults) in a large study in a 222 paediatric diabetes centres in Germany and Austria.[27] Studies on insulin dosage requirements in low- or middle-income countries have not been done; however, no evidence suggests systematic differences in biological insulin needs according to income level.

Patients with T1DM are known to experience a reduction in insulin sensitivity. As insulin is an important regulator of growth, this reduction can further influence growth velocity.[2,9] Insulin is available in various forms.[6] Santi *et al.*[8] reported that basal-bolus therapy during puberty showed improved growth velocity compared with other regimens, although the use of CSII did not

seem to influence linear growth. Most patients in this cohort received basal-bolus regimens (67.8%), whereas few used CSII pumps (owing to resource constraints). Neither insulin dose nor treatment regimen was associated with stunting (p=0.7 and p=0.8, respectively) (Supplementary Table 2). We cannot draw a definitive conclusion regarding a possible protective effect of CSII on linear growth; larger studies are warranted.

Poor socioeconomic conditions contribute to stunting through food insecurity,[7,11,13,15] which is especially concerning as adequate nutrition is vital in T1DM.[2,6,7] In this cohort, only 57.4% reported adequate daily meal intake (three meals and two snacks). Patients were classified according to their socioeconomic status using the income-based categories for hospital care. In this cohort, no significant association was found between income category and stunting (p=0.4) (Table 3).

Nearly half (47.8%) of patients were in the lowest income category,[28] highlighting widespread poor socioeconomic conditions. These children with T1DM face greater challenges. Despite free provision of insulin and supplies, 65.2% reported that overall diabetes care was unaffordable. In a study in India, Rohilla *et al.*[29] showed a significant negative correlation between socioeconomic status and diabetes expenditure, with the most underprivileged families facing the highest financial burden. In almost a third (30.3%) of their cohort, more than 50% of the family income was spent on diabetes care, emphasising the financial burden of affordable comprehensive diabetes care. Although no link was found between treatment affordability and stunting (p=0.5), food insecurity was a significant risk factor (p=0.021). Identifying cost-related barriers, particularly nutritional support, is crucial to guide policy and resource allocation. Further studies are required to evaluate the actual impact of T1DM on disadvantaged families in SA.

The management of T1DM is multifaceted and is a challenge for both the patients and their families.[5] Children often require support from caregivers[6,7] and a secondary level of education (or higher) is known to be a protective factor.[7] In this cohort, most parents (97.4%) reported the highest level of education to be secondary education or higher. Although we did not find significant associations between stunting and the level of income (p=0.4) or education (p=0.1), these factors are important in identifying strategies to assist these patients.

Study limitations

Limitations of this study include the lack of documentation of a full puberty assessment and bone age, which also impact longitudinal growth. Neither point-of-care HbA1c levels nor treatment compliance was longitudinally assessed. Most patients had diabetes for less than 5 years, which could be a further limitation, as their underlying condition may not yet have had time to impact their growth, indicating the need for assessing stunting over long terms. The rate of stunting in the background population was unknown and would have been helpful as a comparison. The lack of baseline height at diagnosis limited the ability to assess longitudinal growth patterns; change in height SD over time would have provided a more accurate measure of growth trajectory. The small number of stunted individuals also impacted the extent of the statistical analysis that was possible in this study; multivariate logistic regressions would have added further value had the group been larger.

Conclusion

There is a lack of data on the prevalence and complications in patients diagnosed with T1DM in SA. This study aimed to identify the prevalence of stunting in T1DM patients and its associated factors. Despite very poor glycaemic control being seen in a large proportion (75.7%) of patients, a relatively low incidence of stunting (10.4%) was found. Multiple risk factors were found to have a statistically significant role, emphasising the need for holistic management of patients, including assessment of their genetic target heights. Using MPH as a screening tool in SA may not always be feasible (both parents aren't always contactable/traceable and when they are there may be inaccuracy with regard to home measurements and calculations), but it may be useful to determine the patients' genetic potential and allow for earlier identification of inappropriate growth or an alternative diagnosis. Stunting in children and adolescents with T1DM was found to be associated with poor glycaemic control, undernutrition, food insecurity, comorbidities, older age and genetic factors, highlighting its multifactorial nature. Promoting growth requires a multidisciplinary approach, including optimising glycaemic control, addressing nutritional status and food insecurity, managing comorbidities and monitoring growth relative to genetic potential. Glycaemic control, influenced by insulin therapy, remains a key modifiable factor affecting growth, emphasising treatment optimisation. Greater education, awareness and access to healthcare resources, supported by government and private stakeholders, are essential to improve outcomes for children with T1DM in SA.

Declaration. None.

Acknowledgements. None.

Author contributions. CJB was responsible for study conceptualisation and design, data collection, analysis and interpretation, assisted by PR and MK. CJB wrote the manuscript.

Funding. None.

Conflicts of interest. None.

1. Haller MJ, Atkinson MA, Schatz D. Type 1 diabetes mellitus: Etiology, presentation, and management. Pediatr Clin North Am 2005;52(6):1553-1578. https://doi.org/10.1016/j.pcl.2005.07.006.
2. Giannini C, Mohn A, Chiarelli F. Growth abnormalities in children with type 1 diabetes, juvenile chronic arthritis, and asthma. Int J Endocrinol 2014;2014:265954. https://doi.org/10.1155/2014/265954.
3. International Diabetes Federation. Type 1 diabetes. IDF (accessed 16 August 2024).
4. Katte JC, McDonald TJ, Sobngwi E, et al. The phenotype of type 1 diabetes in sub-Saharan Africa. Front Public Health 2023;11:1014626. https://doi.org/10.3389/fpubh.2023.1014626.
5. Benton M, Cleal B, Prina M, et al. Prevalence of mental disorders in people living with type 1 diabetes: A systematic literature review and meta-analysis. Gen Hosp Psychiatry 2023;80:1-16. https://doi.org/10.1016/j.genhosppsych.2022.11.004.
6. South African National Department of Health. Standard Treatment Guidelines and Essential Medicines List, Paediatric Hospital Level. Pretoria: NDoH, 2023.
7. Kayirangwa A, Rutagarama F, Stafford D, et al. Assessment of growth among children with type 1 diabetes mellitus: A cross-sectional study of factors contributing to stunting. J Diabetes Metab 2018;9(4):1-8. https://doi.org/10.4172/2155-6156.1000793.
8. Santi E, Tascini G, Toni G, et al. Linear growth in children and adolescents with type 1 diabetes mellitus. Int J Environ Res Public Health 2019;16(19):3677. https://doi.org/10.3390/ijerph16193677.
9. Virmani A. Growth disorders in type 1 diabetes: An Indian experience. Indian J Endocrinol Metab 2015;19(Suppl 1):S64-S67. https://doi.org/10.4103/2230-8210.155405.
10. Bonfig W, Kapellen T, Dost A, et al. Growth in children and adolescents with type 1 diabetes. J Pediatr Clin Pract 2012;160(6):900-903.e2. https://doi.org/10.1016/j.jpeds.2011.12.007.
11. Said-Mohamed R, Micklesfield LK, Pettifor JM, et al. Has the prevalence of stunting in South African children changed in 40 years? A systematic review. BMC Public Health 2015;15:534. https://doi.org/10.1186/s12889-015-1844-9
12. Victora C, Adair L, Fall C, et al. Maternal and child undernutrition: Consequences for adult health and human capital. Lancet 2008;371:340-357. http://doi.org/10.1016/S0140- 6736(07)61692-4.
13. Le Roux M, Nel M, Walsh C. Determinants of stunting at 6 weeks in the Northern Cape province, South Africa. Front Public Health 2020;8:166. https://doi.org/10.3389/fpubh.2020.00166

14. Kimani-Murage EW. Exploring the paradox: Double burden of malnutrition in rural South Africa. Glob Health Action 2013;6(19249):193-205. https://doi.org/10.3402/gha.v6i0.19249
15. Govender L, Pillay K, Siwela M, et al. Food and nutrition insecurity in selected rural communities of KwaZulu-Natal, South Africa: Linking human nutrition and agriculture. Int J Environ Res Public Health 2016;14(1):17. https://doi.org/10.3390/ijerph14010017.
16. Sanders D, Reynolds L. Ending stunting: Transforming the health system so children can thrive. In: Jamieson L, Berry L, Lake L, eds. South African Child Gauge 2017. Cape Town: Children's Institute, University of Cape Town, 2017:68-76. https://ci.uct.ac.za/child-gauge/cg-2017-survive-thrive-transform.
17. Mitchell DM. Growth in patients with type 1 diabetes. Curr Opin Endocrinol Diabetes Obes 2017;24(1):67-72. https://doi.org/10.1097/MED.0000000000000310
18. Centers for Disease Control and Prevention. Clinical growth charts. CDC. https://www.cdc.gov/growthcharts/clinical_charts.htm (accessed 12 August 2024).
19. World Health Organization. Length/height-for-age. WHO. https://www.who.int/tools/child-growth-standards/standards/length-height-for-age (accessed 12 August 2024).
20. Nwosu BU, Lee MM. Evaluation of short and tall stature in children. Am Fam Physician 2008;78(5):597-604.
21. Chanyarungrojn PA, Lelijveld N, Crampin A, et al. Tools for assessing child and adolescent stunting: Lookup tables, growth charts and a novel appropriate-technology "MEIRU" wallchart – a diagnostic accuracy study. PLOS Glob Public Health 2023;3(7):e0001592.
22. R Foundation for Statistical Computing. A Language and Environment for Statistical Computing. R Core Team. https://www.R-project.org (accessed 12 August 2024).
23. Wand H, Naidoo S, Govender V, et al. Preventing stunting in South African children under 5: Evaluating the combined impacts of maternal characteristics and low socioeconomic conditions. J Prev (2022) 2024;45(3):339-355. https://doi.org/10.1007/s10935-024-00766-2.
24. Thurstans S, Opondo C, Seal A, et al. Boys are more likely to be undernourished than girls: A systematic review and meta-analysis of sex differences in undernutrition. BMJ Glob Health 2020;5(12):e004030. https://doi.org/10.1136/bmjgh-2020-004030.
25. Karet B. Early diagnosis of type 1 diabetes in children. Diabetes Prim Care 2011;13(4):207.
26. Almutairi, R. Short stature in children. Int J Med Dev Countries 2018;2(1):9-15. https://doi.org/10.24911/IJMDC.2.1.3.
27. Wiegand S, Raile K, Reinehr T, et al. Daily insulin requirement of children and adolescents with type 1 diabetes: Effect of age, gender, body mass index and mode of therapy. Eur J Endocrinol 2008;158(4):543-549. https://doi.org/10.1530/EJE-07-0904.
28. Western Cape Government. Subsidised Patients. Western Cape Government. 2022. https://www.westerncape.gov.za/general-publication/western-cape-government-hospital-tariffs-overview?toc_page=3 (accessed 10 September 2024).
29. Rohilla L, Gujjar N, Kaur G, et al. Financial burden for families of children with type 1 diabetes: A cross-sectional survey from North India. Diabetol Int 2022;13(4):665-671. https://doi.org/10.1007/s13340-022-00589-8.

Received 8 March 2025. Accepted 18 September 2025