Original Article

Dynamic Muscle Function Parameters in Indian Children and Adolescents with Type 1 Diabetes Mellitus: A Case-Control Study

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Abstract

Introduction: Recent evidence reveals that type 1 diabetes mellitus (T1DM) impairs muscle function (MF) in adolescents. However, despite its importance in physical well-being, data on dynamic MF in Indian children and adolescents (C and Y) with T1DM are scarce. We assessed MF using Jumping Mechanography (JM, a measurement method for motion analysis and assessment of muscle power and force). (1) To assess dynamic MF by JM in C and Y with T1DM as compared to healthy controls (2) To determine predictors of MF in children with T1DM. **Methods:** A cross-sectional observational study on 266 children (133 – T1DM duration >1 year with no known comorbidities + 133 age and gender-matched healthy controls) aged 6–19 years. Anthropometry, body composition, and MF (maximum relative power Pmax/mass, maximum relative force Fmax/BW by JM) were recorded. The lean mass index (LMI) was calculated as lean mass (kg)/height (m^2). HbA1c was assessed in T1DM. Independent sample *t*-test and linear regression were performed. **Results:** MF parameters (Pmax/mass 33.5 ± 7.2 vs 38.0 ± 8.6 W/kg and Fmax/BW 10.5 ± 2.9 vs 11.4 ± 4.1 N/kg, $P < 0.05$) were significantly lower in T1DM group vs controls. Positive association of body mass index and LMI with both MF parameters and negative association of insulin requirement and HbA1c with Fmax was observed in T1DM. Predictors of MF identified were MMI (Pmax/mass:β = 1.6,95%CI = 0.6–2.6; Fmax/BW:β =2.0,95%CI = 1.6–2.4) and HbA1c (Pmax/mass:β = −2.1,95%CI = −4.5–−0.5; Fmax/BW:β = −1.1,95%CI = −2.0–−0.2) ($P < 0.05$). **Conclusion:** C and Y with T1DM exhibits compromised muscle function. Poor glycaemic control increases the risk of having decreased MF, irrespective of diabetes duration and may contribute to sarcopenia in adulthood.

Keywords: Children, Indian, jumping mechanography, muscle function, type 1 diabetes

Introduction

Poor glycaemic control in type 1 diabetes mellitus (T1DM) is associated with a plethora of acute and chronic complications. One of the complications of diabetes that is frequently overlooked is a progressive deterioration of muscle function (MF), which in turn is associated with sarcopenia and osteoporosis later in life.[1,2] Reduced muscle function and bone mass in T1DM can be attributed to an interplay of longstanding diabetes, increased advanced glycation end-products, vitamin D deficiency, poor glycaemic control causing urinary calcium loss, impaired renal function, chronic inflammation, and altered growth hormone/IGF 1 axis.[3-5] Muscle mass and strength may be adversely affected in T1DM due to diabetic polyneuropathy and deficiency of insulin leading to muscle wasting.[6] Structural and functional changes in skeletal muscles even before the onset of clinical symptoms and other complications of diabetes are seen.[7-9] Thus, early onset of compromised MF coupled with chronicity of the illness may potentially

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accelerate sarcopenia in children with T1DM even as a primary complication (in contrast to secondary complication following neuropathy).

MF has been traditionally measured by testing handgrip strength using dynamometers. This evaluates isometric force at a non-weight-bearing part of the body and hence, does not take into account movements required for activities of daily living.[10] On the contrary, Jumping Mechanography (JM), which is a measurement method for motion analysis, assesses dynamic MF and therefore, is more representative of daily activities.[10] Very few studies have evaluated dynamic MF in children with T1DM. Realizing the unmet need for early detection of compromised MF in children with T1DM, this study was planned with the following objectives: 1) To assess dynamic MF parameters using JM in Indian children and adolescents with T1DM in comparison with age and gender-matched healthy controls. 2) To assess the relationship of dynamic MF parameters with anthropometry, body composition, and parameters of diabetes control in children and adolescents with T1DM.

Materials and Methods

Study design and participants

This was a cross sectional, case-control, observational study conducted between November 2021 and April 2022. Cases were children with type 1 diabetes attending an outpatient clinic for T1DM at a tertiary care hospital in Pune, Maharashtra, India. Around 450 children routinely attend diabetes clinic at our facility. These children predominantly belong to middle/lower socioeconomic class and are provided with diabetes care, which includes free insulin, glucometers, and test strips for monitoring blood glucose as well as diagnostic tests. Insulin doses and frequency of insulin injection are prescribed by a pediatric endocrinologist as per the age and pubertal stage of the child. Children are on multiple daily injections of insulin therapy; no children are on insulin pumps.

All children (and their families) who were over 6 years of age (as children under 6 years find it hard to follow instructions for JM test) with a confirmed diagnosis of type 1 diabetes mellitus via blood tests (serum C-peptide concentration and/or GAD65 antibodies) and having disease duration of more than a year who attended diabetes clinic during the study period were offered the study (202 children). Due to fluctuating weight and metabolic instability observed at onset and during initial diagnosis and treatment for diabetes, children and adolescents with diabetes duration <1 year were not included.[11] Children on medications other than insulin for blood glucose control, with known comorbidities such as celiac disease, nephropathy, untreated hypothyroidism, eating disorders, or any other chronic disorder were excluded (n = 44). Twenty-five children did not agree to take part in the study, mainly because they could not spare time for assessments due to schooling commitments. Thus, 133 children (61 boys) with T1DM were enrolled in the study.

Additionally, 133 age and gender-matched healthy controls (61 boys), who were from the same socioeconomic class as the children with T1DM, were enrolled from nearby schools and colleges. The schools and colleges were approached for enrolling healthy children. The teachers explained the study in detail. Information pamphlets were distributed to students, and meetings with parents were arranged. The children whose parents agreed to take part in the study were examined by pediatricians to rule out chronic disease conditions and congenital abnormalities. Inclusion criteria were children and adolescents aged 6–19 years with growth parameters between 3rd and 97th percentile according to Indian reference data[12] for children and normal BMI for adolescents above 18 y. Children consuming vitamin D or any other drug known to affect bone or muscle health, and having any chronic systemic illnesses or congenital abnormalities were excluded.

A post-hoc power of more than 0.8 was achieved with a sample size of 133 in each group, with case: control ratio of 1:1 with an effect size of 1.17 using $r^2 = 0.54$.

Anthropometric and body composition parameters

Standing height was measured using a portable stadiometer (Seca 213 Portable Stadiometer, Germany). Body mass and composition (fat percentage, fat mass, fat-free mass, bone-free lean tissue mass (lean mass), and total body water) were measured using the bioelectrical impedance analysis (BIA) method (Tanita Body Composition Analyzer (Model BC-420MA) after the children were asked to empty their pockets and stand barefoot on the scale. All children were measured around similar times of the day (10 am–12 pm), after voiding, to ensure a similar hydration state. The same instruments and protocols were used for cases and controls to minimize error.

BMI was calculated by dividing weight in kilograms by height in meters squared. The lean mass index (LMI) was computed by dividing lean mass in kilograms by height in meters squared. Z-scores for height for age (HAZ), weight for age (WAZ), BMI for age (BAZ), lean percentage, and fat percentage for age were computed using Indian growth references.[12]

Biochemical measurements

Control of diabetes was evaluated by measuring glycosylated hemoglobin (HbA1C, HPLC method). A trained pediatric nurse collected blood samples. Assessment of serum 25 (OH) D was performed by ELISA technique using standard kits (DLD Diagnostika GMBH, intra-assay coefficient of variation [CV] 5.0%; inter-assay CV 7.8%) only in the T1DM group.

Dietary intakes

Dietary data were recorded using the 24-hour dietary recall method over three non-consecutive days including one holiday or a Sunday. Nutrient intakes were then computed using the cooked food database software, C-Diet (version 3.2).[13]

Muscle function

Leonardo Mechanograph Ground Reaction Force Plate (Novotec Medical, Pforzheim, Germany) was used

for assessing dynamic MF. For detection, storage, and calculation of the outcomes, software provided by the manufacturer (Leonardo Mechanography GRFP version 4.4, Novotec, Pforzheim, Germany) was used.[14]

Two types of jumps were performed by all participants: single 2-legged jump (s2LJ) which detected maximum relative power and multiple 1-legged hopping (m1LH) which detected maximum relative force. Each type of jump was repeated until 3 acceptable jumps (described below) were obtained, and the jump with the greatest peak power/force was used for analyses. Peak power and peak force were adjusted for the weight of the participants for comparison between groups. The inter-day test-retest measurements of the main outcome parameters of these tests have shown low variability ranging from 3.4% to 6.4% in healthy children.[15]

Single 2-legged jump (s2LJ)

The jump was performed as a counter movement jump (children briefly squatted before jumping) with freely moving arms. Main outcomes of interest for s2LJ are maximum power (Pmax) and maximum power relative to body mass (Pmax/mass, Watt/kg).[16] This test also gives Esslinger Fitness Index (EFI), which is Pmax/mass normalized to age and gender and standard deviation score of EFI (EFI-SDS). Force efficiency is also obtained through this test. Force efficiency, as the name suggests, tells about the force used to achieve a given power.

Multiple 1-legged hopping (m1LH)

The child was instructed to jump repeatedly (approximately fifteen jumps), as fast as was possible on the forefoot of his/her dominant leg. Any repetitions with heel contact were excluded from analysis by the manufacturer's software. Maximum voluntary force (Fmax) and maximum relative force, i.e. Fmax normalized to body weight (Fmax/BW) were considered the main outcome variables for m1LH. Fmax/BW standard deviation score (Fmax-SDS) was also used for analysis to explore the MF of these children in this study as compared to reference data provided by the manufacturer.[17]

Statistical analyses

Statistical analyses were carried out using IBM SPSS Statistics for Windows (version 26.0.0, IBM Corp, Armonk, NY, USA). Before statistical analyses, all the study parameters were tested for normality. All results have been expressed as mean ± standard deviation. Student's *t*-test was used for normal variables to test the differences between cases and controls and non-parametric tests were carried out for non-normal variables. Pearson correlations were computed to examine the correlations of age, BMI, MMI, disease duration, insulin requirement per day, HbA1c with Pmax and Fmax. The significance level was set at $P < 0.05$. Further, to assess the predictors of MF parameters in children with T1DM, a linear regression was performed. Gender and MMI were entered in the 1st block of the hierarchical regression model whereas the diabetes parameters, specifically, disease duration in years and poor diabetes control (HbA1c >9.5%) were entered in the 2nd block.

Ethical Aspect

The research protocol was approved by the Institutional Ethics Committee (Ethical Committee, Jehangir Clinical Development Centre Pvt Ltd) ECR/352/Inst/MH/2013/RR-19 dated 22nd July 2021. All the procedures performed in the study were in accordance with the ethical standards of the Ethical Committee and with the Helsinki Declaration of 1975 (revised in 2000) and its later amendments or comparable ethical standards. Parents provided written informed consent and children gave assent for participation and use of the data for research purposes.

Results

A total of 266 children and adolescents (133 children and adolescents with T1DM and 133 controls) were recruited in the study. Mean age of both the groups was similar ($P > 0.05$) and the age ranged from 6.0 years to 19.9 years for the whole group. Mean disease duration of the children with T1DM was 5.6 ± 2.8 years and the mean HbA1c was 9.9 ± 1.7%. Mean serum 25 (OH) D concentration of the T1DM group was 18.3 ± 7.3 ng/ml and 60% of the children were found to be vitamin D deficient (vitamin D <20 ng/ml).[18] All children in the T1DM group were on multiple daily injection regimens. None of the children had any other co-morbidity or microvascular complications.

Table 1 describes the anthropometric and body composition parameters of the children in the study. Children with T1DM had significantly lower Z-scores for height, weight, and BMI as compared to controls ($P < 0.05$). However, the body composition parameters, specifically, lean percentage, LMI, and fat percentage of both groups were not significantly different ($P > 0.05$).

The dietary intakes of the participants in both groups differed significantly ($P < 0.05$). The energy intake of the children in the T1DM group did not fulfill the estimated average requirements (EAR) for Indians.[19] The mean energy intake (percentage of EAR) of the T1DM group was 1217 ± 494 kcal/day (52%) while that of the healthy control group was 1776 ± 623 kcal/day (78%). The mean protein intake (percentage of EAR) of the control group was 40 ± 17 g/day (140%) while that of the T1DM group was 33 ± 15 g/day (109%).

The dynamic MF parameters were significantly different in both groups [Table 2]. The maximum power and maximum force were found to be significantly lower in children with T1DM than in the controls. The difference remained significant even after adjusting for body mass. The children with T1DM had lower maximum relative power and maximum relative force ($P < 0.05$). Controls had higher EFI and force efficiency than the children with T1DM ($P < 0.05$). The EFI Z-score was significantly higher in controls (−0.9 ± 1.0) than in the T1DM group (−1.6 ± 1.0) ($P < 0.05$). Similarly, the force efficiency Z-score had a significantly lower value in the T1DM group (−1.1 ± 1.0) than in controls (−0.3± −1.1) ($P < 0.05$).

To investigate the differences further, we categorized the C and Y in both groups based on age at the onset of puberty of girls and boys. The cut-off age for pre-puberty in girls was considered as <10.5 years and in boys it was <11.5 years.[20,21] In the prepubertal age group, maximum power, EFI, EFI Z-score, force efficiency, and force efficiency Z-score were significantly higher ($P < 0.05$) in the controls than in the T1DM group. Maximum force was comparable in both groups at pre-puberty. At post-puberty, the T1DM group was seen to have significantly lower values of all MF parameters than the controls ($P < 0.05$).

Figure 1 illustrates the relationship between maximum power and age. The association is moderately positive ($r^2 = 0.6$) in healthy controls while in the T1DM group, the association was very weak ($r^2 = 0.03$). In C and Y with T1DM, the maximum power seems to decline with age. Figure 2 shows an association between maximum voluntary force and age. Maximum voluntary force and age had a moderate positive linear relationship in both groups, however, the association was marginally stronger in healthy controls ($r^2 = 0.6$) than in the T1DM group ($r^2 = 0.5$).

To assess the relationship of MF parameters with anthropometry, body composition, and diabetes parameters (disease duration, insulin units taken per kg per day, and HbA1c,) correlation coefficients were computed for the T1DM group [Table 3]. Age showed a significant positive correlation with Fmax. BMI and LMI correlated positively with both MF parameters. However, MF parameters did not correlate with disease duration. Elevated serum HbA1c concentration correlated negatively with Fmax but not with Pmax. A significant negative correlation was found between maximum voluntary force and insulin units taken/kg/day ($P < 0.05$).

Data were then analyzed using hierarchical linear regression models to identify the predictors of dynamic MF in children with T1DM [Table 4]. Separate models run for each MF parameter. To study the predictors of effective MF, the body mass/weight adjusted parameters of MF (Pmax/mass and Fmax/BW) were entered into the regression model. A hierarchical

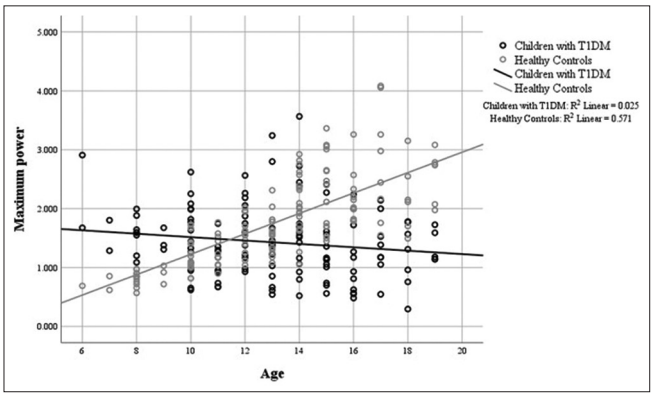

Figure 1: Scatter plot showing the association of the maximum power with age in healthy children and children with T1DM

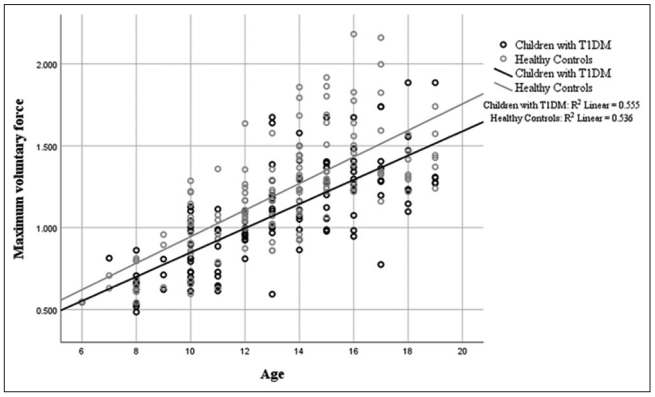

Figure 2: Scatter plot showing the association of the maximum voluntary force with age in healthy children and children with T1DM

Table 1: Comparison of anthropometric and body composition parameters between children with T1DM and healthy controls

Parameters	T1DM Boys (61)	Control Boys (61)	T1DM Girls (72)	Control Girls (72)	T1DM Total (133)	Controls Total (133)
Age (years)	13.6±3.2	13.7±3.2	13.3±3.3	13.4±3.2	13.4±3.3	13.5±3.2
Height (cm)	150.3±17.6	154.2±16.4	143.9±13.1	149.9±12.4†	146.8±15.6	152±14.6‡
Weight (kg)	39.4±13.2	43.5±14.3	38.3±12.1	43.3±11.1†	38.8±12.6	43.4±12.7‡
BMI (kg/m^2)	16.9±2.7	17.8±3.3	18.0±3.3	19.0±3.0	17.5±3.1	18.4±3.2‡
HAZ	−0.6±1.0	−0.07±1.0*	−0.6±1.1	0.3±0.8†	−0.6±1.1	0.1±1.0‡
WAZ	−0.8±0.8	−0.3±1.0*	−0.5±1.0	0.1±0.6†	−0.7±1.0	−0.1±0.9‡
BAZ	−0.6±0.7	−0.4±0.9	−0.3±0.9	−0.1±0.7†	−0.5±0.8	−0.2±0.9‡
Lean %	83.8±6.7	82.1±9.2	73.1±8.0	70.8±6.4	78.1±9.1	76.3±9.7
Lean % *Z*-score	0.6±0.8	0.4±1.0	0.2±0.9	−0.1±0.6†	0.4±0.9	0.2±0.9
LMI	14.0±1.3	14.4±1.7	12.9±1.1	13.2±1.0	13.4±1.3	13.7±1.5
Fat %	11.7±7.0	13.4±8.9	22.8±8.1	24.9±6.6	17.7±9.4	19.4±9.7
Fat % *Z*-score	−0.7±1.0	−0.5±1.0	−0.2±0.9	0.1±0.6†	−0.4±0.9	−0.2±0.9

P<0.05; All values are expressed as mean±SD; BMI: Body mass index; HAZ: Height for age *Z*-score; WAZ: Weight for age *Z*-score; BAZ: BMI for age *Z*-score; LMI: Lean Mass Index; *Significantly different than T1DM boys; †Significantly different than T1DM girls; ‡Significantly different than T1DM children

Table 2: Comparison of dynamic muscle function parameters between children with T1DM and healthy controls

Parameters	T1DM Boys (61)	Control Boys (61)	T1DM Girls (72)	Control Girls (72)	T1DM Total (133)	Controls Total (133)
Maximum power (kW)	1.6±0.7	2.0±0.9 *	1.3±0.5	1.5±0.5†	1.4±0.6	1.7±0.7‡
Maximum relative power (W/kg)	38.0±6.6	42.1±9.4 *	29.6±5.2	34.0±5.3†	33.5±7.2	38.0±8.6‡
EFI (%)	83.1±14.9	91.0±14.7 *	70.8±12.6	81.7±12.6†	76.4±15.0	86.3±14.4‡
Force efficiency (%)	88.4±13.8	97.9±16.3 *	81.8±13.2	94.4±16.3†	84.8±13.8	96.2±16.3‡
Maximum voluntary force (kN)	1.1±0.3	1.2±0.4	1.1±0.3	1.2±0.3†	1.2±0.3	1.3±0.3‡
Maximum relative force (N/kg)	10.7±3.0	11.6±4.8	10.3±2.8	11.0±3.3	10.5±2.9	11.4±4.1‡

P<0.05; All values are expressed as mean±SD; EFI: Esslinger Fitness Index;*Significantly different than T1DM boys; †Significantly different than T1DM girls; ‡Significantly different than T1DM children

Table 3: Pearson's correlation coefficients of anthropometry, body composition, diabetes, and biochemical parameters with dynamic muscle function parameters in children with T1DM

Parameters	Maximum power (kW)	Maximum voluntary force (kN)
Age (years)	− 0.2	0.75 *
BMI (kg/m^2)	0.22 *	0.76 *
LMI (kg/m^2)	0.49 *	0.77 *
Disease duration (years)	− 0.09	0.13
Insulin units/kg/day	− 0.01	− 0.33 *
HbA1c (>9.5%)	− 0.12	− 0.20 *

**P*<0.05. BMI: Body mass index; LMI: Lean mass index

regression analysis was performed, and variables were added in 2 blocks (Block 1: gender and LMI, Block 2: disease duration in years and poor diabetes control, HbA1c >9.5%). These variables explained the maximum variance in Fmax/BW (r^2 = 54.4) followed by Pmax/mass (r^2 = 35.3).

It was observed that the male gender was a positive predictor of having higher Pmax/mass (β =6.5, 95% CI = 3.8–9.3), whereas it was a negative predictor for Fmax/BW (β = −1.7, 95% CI = −2.7–−0.7) (P < 0.05, for both). LMI was found to be a positive predictor of both, Pmax/BW (β = 1.6, 95% CI = 0.6–2.6) and Fmax/BW (β = 2.0, 95% CI = 1.6–2.4) (P < 0.05, for both). Disease duration was not found to have any significant relationship with MF in these children. Poor diabetes control (HbA1c >9.5%) was a significant negative predictor of Pmax/mass (β = −2.1, 95% CI = −4.5–−0.5) as well as Fmax/BW (β = −1.1, 95% CI = −2.0–−0.2) (P < 0.05, for both).

Discussion

In our study comparing MF in children with diabetes and healthy controls, we found that although the lean and fat percentages in the two groups of children were similar, maximum power and force (even after adjusting for body mass or weight) were significantly lower in children with T1DM. Unlike for most other complications of diabetes, disease duration was not a predictor of poor MF; MF deterioration was affected by poor glycaemic control. The EFI Z-score and force efficiency Z-score were also significantly lower in the T1DM group. Lower EFI signifies having lower muscle power even after adjusting for age and gender and lower force efficiency indicates having to use more force to achieve a given power. BMI and LMI correlated positively with both MF parameters, whereas a negative correlation was observed between maximum voluntary force and insulin units/kg/day. Male gender was a positive predictor of Pmax/mass and LMI was a positive predictor of both, Pmax/mass and Fmax/BW. Disease duration was not found to have any significant relationship with MF in these children.

There is a paucity of data on the objective assessment of MF using equipment such as JM. We found only two studies published on the evaluation of MF using JM and its determinants in children and adolescents with type 1 diabetes, with none being reported from low-middle-income countries such as India.[22,23]

Similar to our study, Maratova *et al*.[22] have reported significantly lower maximum relative power and force in adolescents with T1DM. Fricke *et al*.[23] reported lower maximal isometric grip force in German children and adolescents with T1DM; however, they found no difference in the Pmax and Fmax between the cases and the reference population.

Muscle mass and strength increase from prepubertal to pubertal progression and is higher in taller individuals.[24-26] Muscle mass has been reported to influence MF, i.e., more the muscle mass, the better the MF.[24] On similar lines, we found that LMI was a positive predictor of MF in children with T1DM. However, LMI between the two groups was not significantly different, indicating that despite the lean mass percentages and lean mass adjusted for height (LMI) being comparable between the cases and controls; MF was significantly lower in T1DM. This suggests that MF was affected owing to the disease, and not merely due to body size and composition. Male gender was a positive predictor of Pmax/mass. In a study conducted among healthy children, similar results were observed.[2] Studies evaluating muscle structure in T1DM have reported a reduction in muscle volumes, area, and myofiber size even in newly diagnosed patients.[27] Monaco *et al*.[7] have postulated that T1DM alters the mitochondrial function and reduces the rate of protein synthesis and also decreases calcium retention capacity which

Table 4: Predictors of dynamic muscle function parameters in children with T1DM

	Maximum Relative Power					Maximum Relative Force				
Model R^2	35.3					54.4				
Predictors	***B***	**SE**	***t***	***P***	**95% CI**	***B***	**SE**	***t***	***P***	**95% CI**
Block 1 R^2	31.7					51.6				
Gender (Boys)	6.5	1.1	7.8	<0.001	3.8–9.3	−1.7	0.5	−3.4	0.001	−2.7–−0.7
LMI	1.6	0.5	3.3	0.001	0.6–2.6	2.0	0.2	11.1	<0.001	1.6–2.4
Block 2 R^2	3.6					2.9				
Disease duration in years	−0.1	0.2	−0.3	0.8	−0.5–0.4	−0.0	0.1	−0.3	0.8	−0.2–0.1
Poor control; (HbA1c >9.5%)	−2.1	1.3	−1.6	0.04	−4.5–−0.5	−1.1	0.5	−2.4	0.02	−2.0–−0.2

CI: Confidence interval; LMI: Lean Mass Index

results in increased cell death by apoptosis, thereby causing muscle fiber loss.

Insulin, being an anabolic hormone, deficiency leads to proteolysis of muscle and other proteins. However, a significant negative correlation between Fmax and insulin requirement per kg body weight was observed in our study. It may be postulated that the higher the dose of insulin required, the poorer the control of diabetes, which may have negatively affected Fmax. Similar findings have been observed by Hiromine *et al.*,[28] who observed that following adjustment for HbA1c and C-peptide concentrations, the association between insulin requirement and sarcopenia was no longer significant, indicating that higher prevalence of sarcopenia in insulin-treated individuals is a reflection of poor diabetes control/lower C-peptide concentrations.

Almost all complications have a temporal relation with diabetes, with more complications being witnessed as disease duration increases. Surprisingly, disease duration did not show any correlation with MF parameters. Various studies have demonstrated that MF decline begins early in the course of diabetes and is usually a primary complication, i.e., it occurs even in the absence of neuropathy.[6] Even at the onset of clinical presentation, MF may be compromised indicating insulin deficiency-mediated muscle proteolysis as the underlying cause; with chronic hyperglycemia, skeletal muscle proteins undergo glycation. Structural modification of muscle proteins and reduced myosin filament motility have been demonstrated using experimental models.[29] Polyneuropathy involving motor nerves causes axonal and myelin loss leading to sarcopenia.[8] Thus, it is possible that different mechanisms for reduced MF prevail at different times in the course of the disease.

Apart from duration, glycaemic control is a major determinant for the development of complications. Not only does it directly affect muscle structure and function, but it also causes other complications indirectly affecting skeletal muscles (neuropathy and alterations in microvascular perfusion).[28,30,31] Poor diabetes control (HbA1c >9.5%) was a significant negative predictor of Pmax/mass and Fmax/BW in this study. Glycation of hemoglobin increases affinity to oxygen and there may be a resultant disturbance in muscle blood flow.[26] In poorly controlled diabetes, decreased muscle blood volume during activity may signify microvascular dysfunction even before symptomatic microangiopathy.[31]

This is the first study from India (and one of the few studies the world over) that has reported the assessment of dynamic MF in children and adolescents with T1DM. Our finding of compromised MF and its relationship with poor control is an important contribution to optimizing the care of children with diabetes. Moreover, while most other diabetes-related complications develop and worsen as the duration of the disease increases, MF deterioration has no temporal association and a high index of suspicion needs to be maintained right from the beginning. Our study is limited by the fact that this was a single-center study and children were from lower and middle socioeconomic classes. However, all patients belonged to a similar socioeconomic and educational background. Further, we have not reported confounders such as pubertal status and physical activity. We could not perform any blood parameters on the controls due to school and parental dissent. Also, data on pubertal status would have added more value to the results. We have used age cutoffs for pubertal status as a surrogate. More studies including children from more centers and socioeconomic classes are thus required to confirm our results. We also could not measure hand grip and perform the chair rise test in this study due to logistic limitations. These test results would have strengthened the study as they are more widely used.

Conclusion

MF (power and force) was compromised in children with type 1 diabetes mellitus. Regular assessment of MF and sarcopenia thus need to be included under the umbrella of diabetes care so as to optimize MF. Longitudinal studies to assess MF are required to further understand the etiopathogenesis of reduced MF in type 1 diabetes.

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Authors' contribution

Sonal V. Kasture: Conceptualization, methodology, investigation, writing – Original draft preparation.
Shruti A. Mondkar: Investigation, Writing – Original draft preparation
Anuradha Khadilkar: Conceptualization, visualization, writing – Reviewing and editing
Ketan Gondhalekar: Formal analysis
Anshu Sethi: Reviewing and editing
Vaman Khadilkar: Supervision, Editing

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Conflicts of interest

There are no conflicts of interest.

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