DOI: 10.4274/jcrpe.galenos.2026.2025-9-8

Original Article

Effects on Muscle Mass and Strength in Children with Newly Diagnosed Type 1 Diabetes Mellitus

Hazal Canbaz Özdemir[1], Mesut Parlak[2]
[1]University of Health Sciences Türkiye, Antalya Training and Research Hospital, Pediatric Endocrinology, Antalya, Türkiye
[2]Akdeniz University Faculty of Medicine Hospital, Pediatric Endocrinology, Antalya, Türkiye

What is already known on this topic?
Children with newly diagnosed type 1 diabetes mellitus (T1DM) often present with reduced muscle strength and altered body composition at diagnosis. Insulin therapy improves metabolic control and may have anabolic effects on muscle tissue. Previous studies have evaluated metabolic and anthropometric changes after insulin initiation, but data on longitudinal changes in both muscle mass and strength in pediatric T1DM remain limited.

What this study adds?
This study is the longitudinally evaluate both muscle mass and muscle strength in children with newly diagnosed T1DM after the initiation of insulin therapy. We demonstrate significant improvements in muscle strength and body composition parameters within six months of treatment, providing novel evidence for the anabolic effects of insulin beyond metabolic control. Our findings highlight the importance of early intervention and comprehensive follow-up for musculoskeletal health in pediatric T1DM patients.

Abstract
Objective: Children with type 1 diabetes mellitus (T1DM) are at risk for reduced muscle mass and strength, which may be influenced by insulin deficiency. Although insulin is known to regulate muscle metabolism, data on its effects in newly diagnosed pediatric patients are limited. The aim was to describe changes in muscle mass and muscle strength after insulin treatment in children newly diagnosed with T1DM.
Methods: This was a prospective analysis of 36 hospitalized children with newly diagnosed T1DM and 43 age, sex-matched outpatient healthy controls at Akdeniz University Faculty of Medicine Hospital Pediatric Endocrinology Clinic between 2020 and 2021. The primary outcome was muscle strength, muscle mass measured at diagnosis, 3 months and 6 months after insulin initiation in patients with type 1 diabetes, compared with age- and sex-matched healthy controls. Total body muscle mass were assessed using bioelectrical impedance analysis and muscle strength was measured by handgrip dynamometry.
Results: Baseline muscle mass did not differ significantly between T1DM patients and controls ($p = 0.73$), but muscle strength was significantly lower in the T1DM group ($p = 0.001$). Following insulin therapy, both muscle mass and strength significantly increased in the T1DM group ($p < 0.001$ for both). No significant correlations were found between muscle parameters and biochemical markers.
Conclusion: Insulin treatment in children with newly diagnosed T1DM is associated with improvements in muscle mass and strength during early follow-up. Regular glycemic control and insulin therapy may **contribute to** delaying or mitigating complications related to impaired muscle development. Longitudinal studies are warranted to explore the long-term musculoskeletal outcomes of insulin therapy in pediatric T1DM.
Keywords: Type 1 diabetes mellitus; muscle strength; muscle mass

Prof. Mesut Parlak, Akdeniz University Faculty of Medicine Hospital, Pediatric Endocrinology, Antalya, Türkiye
mesutparlak@akdeniz.edu.tr
https://orcid.org/0000-0002-3550-1425

18.09.2025
25.01.2026

Epub: 29.01.2026

Introduction

Type 1 diabetes mellitus (T1DM) is an autoimmune disease characterized by insulin deficiency due to destruction of pancreatic β-cells (1). Insulin plays a key role in glucose uptake, glycogenesis, glucose oxidation, and protein synthesis in skeletal muscle, primarily through the protein kinase B (Akt/PKB) and mitogen-activated protein kinase (MAPK)/extracellular signal-regulated kinase (ERK1/2) pathways (2). Insulin deficiency leads to hyperglycemia and increases the risk of various complications (3). In individuals with T1DM, muscle mass, muscle strength, and bone mass are adversely affected due to impaired insulin action (4). Notably, muscle mass and strength have been shown to be negatively correlated with glycemic control, as reflected by hemoglobin A1c (HbA1c) levels (5).
The musculoskeletal system comprises approximately 40% of total body weight and 50–75% of total body protein content. Techniques such as dual-energy X-ray absorptiometry (DXA), bioelectrical impedance analysis (BIA), and magnetic resonance imaging (MRI) are commonly used to evaluate adiposity and muscle mass distribution in pediatric and adolescent populations. Among these, BIA is widely utilized owing to its safety, non-invasiveness, affordability, reproducibility, and rapid output.
Muscle function in children can be efficiently assessed using a hand dynamometer, which measures maximal isometric grip strength of the forearm (6). Grip strength serves as a useful indicator of general health status, protein reserves, and nutritional status (7).
Although reduced muscle mass and strength have been linked to cardiovascular and metabolic diseases, impaired bone health, sarcopenia, and osteoporosis in children with T1DM, there is limited evidence on changes in these parameters following insulin therapy. To our knowledge, no previous study has prospectively evaluated alterations in muscle mass and strength after the initiation of insulin therapy in newly diagnosed pediatric T1DM patients. This study aimed to assess the effects of insulin treatment on muscle strength, fat mass, and muscle mass in children and adolescents with T1DM and to explore the relationships between these outcomes and various biochemical parameters.

Methods

A total of 36 newly diagnosed T1DM patients and 43 age- and sex-matched healthy controls, aged 5–18 years, were enrolled. Diagnosis of T1DM was based on the presence of anti-glutamic acid decarboxylase (anti-GAD), anti-insulin, or anti-islet cell antibodies. Exclusion

criteria included any history of neuropathic or orthopedic disorders, current medication use that could affect muscle function, or prior upper extremity surgery. The healthy control group was evaluated only once at baseline due to logistical limitations and because repeated assessments were not considered ethically appropriate in healthy children. “Newly diagnosed diabetes” referred to children who presented with diabetic ketoacidosis and were subsequently diagnosed with type 1 diabetes mellitus at first presentation, with no prior diabetes treatment (glucose >200 mg/dL, ketonemia/ketonuria, venous pH <7.3 or bicarbonate <15 mmol/L). Laboratory evaluations were performed on the day of hospital admission. In patients, anthropometric measurements, bioelectrical impedance analysis (BIA), and handgrip dynamometry were performed after metabolic stabilization, during the diabetes education period, specifically on the fifth day of hospitalization. Total body muscle mass were assessed using bioelectrical impedance analysis (Tanita MC-780, Tanita Corp., Tokyo, Japan) and muscle strength was measured by isometric handgrip dynamometry using a GRIP-D dynamometer (Takei, Tokyo, Japan). Ethics Approval: This study was approved by the Akdeniz University Faculty of Medicine Ethics Committee (Decision No: KAEK-157, Date: February 19, 2020). Written informed consent, in accordance with the Declaration of Helsinki, was obtained from all participants and/or their legal guardians before inclusion in the study. We have included a sample consent form as a supplementary file for editorial review.

Clinical Evaluation

Height, weight, and body mass index (BMI) were recorded, and standard deviation scores (SDS) were calculated using age- and sex-specific reference values for Turkish children (8). BMI was calculated as weight (kg) divided by height squared (m²). Pubertal status was assessed according to the Marshall and Tanner staging system and categorized as prepubertal (stage 1) or pubertal (stages 2–5) (9).

Body Composition Assessment

Total body muscle mass and fat mass were evaluated using bioelectrical impedance analysis (BIA) with the Tanita MC-780 analyzer (Tanita Corp., Tokyo, Japan). All measurements were performed by a single trained clinician to ensure consistency. Participants were instructed to fast for at least one hour prior to the test, void their bladder, and wear lightweight clothing. During the measurement, individuals stood barefoot on the device platform while grasping the hand electrodes with both hands.

Muscle Strength Assessment

Muscle strength was measured using a GRIP-D hand dynamometer (Takei, Tokyo, Japan), which evaluates isometric grip strength. Three consecutive measurements were obtained from the dominant hand, positioned with the thumb over the other fingers, and the average value was used for analysis. In the T1DM group, both BIA and dynamometer assessments were conducted at baseline, 3 months, and 6 months following diagnosis. In the control group, these measurements were performed only at baseline.

Biochemical Assessment

In the T1DM group, venous blood samples were collected at diagnosis, and at 3 and 6 months of follow-up. The following parameters were analyzed: serum calcium, phosphorus, aspartate aminotransferase (AST), alanine aminotransferase (ALT), hemoglobin A1c (HbA1c), thyroid-stimulating hormone (TSH), free thyroxine (FT4), fasting blood glucose, and hemoglobin levels. In the control group, these parameters were measured once at baseline. Comparisons between groups were performed for all relevant variables.

Statistical Analysis

Statistical analyses were performed using SPSS version 23.0 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as numbers and percentages. Comparisons between groups were performed using the chi-square or Fisher’s exact test for categorical variables, and the Student’s t-test or Mann–Whitney U test for continuous variables, as appropriate based on data distribution. The Friedman test was used to compare repeated measures that did not follow a normal distribution. Pearson’s or Spearman’s correlation analyses were used to assess relationships between continuous variables depending on their distribution. Normality of continuous variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests, supported by visual inspection of histograms and Q–Q plots. Parametric tests were applied to normally distributed variables, whereas non-parametric tests were used when normality assumptions were not met. Sex-based and pubertal subgroup analyses were considered exploratory; therefore, no formal correction for multiple comparisons was applied, and these results were interpreted with caution. A two-sided p-value < 0.05 was considered statistically significant.

Results

A total of 36 children with newly diagnosed Type 1 Diabetes Mellitus (T1DM) (mean age: 9.99 ± 3.02 years) and 43 age- and sex-matched healthy controls (mean age: 10.32 ± 2.70 years) were enrolled. There were no significant differences between the two groups in terms of age, sex distribution, pubertal stage, height, weight, BMI, BMI SDS, fat mass, or muscle mass (Table 1).

At baseline, muscle strength was significantly lower in the T1DM group [median 10.4 N (5–25.5)] compared with controls [median 15.8 N (6.2–31.9); $p = 0.001$]**.** Fasting blood glucose levels were significantly higher in the T1DM group. Serum calcium and phosphorus levels were also significantly lower in patients with T1DM (both $p < 0.001$), while other laboratory parameters did not differ between groups (Table 1). During follow-up, significant improvements were observed in anthropometric, metabolic, and musculoskeletal parameters in the T1DM group. Fat mass increased from 6.65 kg (2.0–20.2) at baseline to 8.25 kg (2.2–22.3) at 3 months and 8.25 kg (2.3–28.3) at 6 months ($p < 0.001$)**.** BMI increased from 17.5 ± 3.28 kg/m² to 18.57 ± 3.28 kg/m² at 6 months ($p = 0.004$)**.** Glycemic control improved significantly following insulin initiation, with HbA1c and fasting blood glucose levels decreasing at both 3 and 6 months ($p < 0.001$ for all)**.** Muscle strength increased significantly from baseline to 6 months, representing an approximately 40–50% improvement. Similarly, muscle mass increased from a median of 23.8 kg (10.9–55.5) at baseline to 25.6 kg (11.7–61.5) at 3 months and remained stable at 6 months [24.9 kg (1.5–58.8); $p < 0.001$] (Table 2). Sex-based analyses showed that boys had higher muscle strength at baseline ($p = 0.030$); however, no significant sex differences were observed at 3 or 6 months. Both sexes demonstrated significant within-group improvements in muscle strength and muscle mass over time ($p < 0.001$) (Table 3). At 6 months, pubertal children had higher muscle strength (21.15 ± 6.39 N vs 11.9 ± 3.72 N; $p < 0.001$) and muscle mass (36.55 ± 10.25 kg vs 20.76 ± 6.18 kg; $p < 0.001$) compared with prepubertal children. From baseline to 6 months, muscle strength increased by +5.72 N in pubertal patients and +3.49 N in prepubertal patients ($p = 0.014$)**,** while muscle mass increased by +3.61 kg vs +1.81 kg, respectively ($p = 0.038$) (Table 4). Finally, muscle strength showed strong positive correlations with muscle mass at all time points ($r = 0.867–0.932$; $p < 0.001$)**,** supporting a close relationship between structural and functional muscle parameters.

Discussion

The novelty of the present study lies in the simultaneous longitudinal assessment of both muscle mass and muscle strength in children with newly diagnosed T1DM, providing functional insight beyond body composition alone. At diagnosis, muscle mass and fat mass were comparable between the T1DM and control groups; whereas, muscle strength was already significantly reduced in T1DM group. In contrast to previous pediatric studies that primarily focused on body composition, our study demonstrates that muscle strength is already reduced at diagnosis and improves longitudinally in parallel with muscle mass following insulin therapy (5,10,11,12). These findings extend existing pediatric literature by providing functional evidence supporting the anabolic role of insulin beyond changes in body composition. Although percentage-based muscle and fat mass measures could allow a more precise interpretation of compositional changes independent of weight gain, these data were not systematically recorded during the initial data collection period; therefore, analyses were limited to prospectively collected absolute values to avoid methodological bias**.** Importantly, the concurrent and significant improvement in muscle strength suggests a true functional recovery rather than a passive increase secondary to weight gain, supporting a functional anabolic contribution of insulin therapy to skeletal muscle. The absence of longitudinal follow-up in the healthy control group represents an important limitation when interpreting these findings. Because controls were evaluated only at baseline, age- and growth-related physiological increases in muscle mass and strength over the 6-month period could not be directly accounted for. Accordingly, the observed longitudinal improvements should be

interpreted as within-patient changes following insulin initiation rather than direct comparisons with normal growth **trajectories.** Age-related and sex-related differences in body composition are well documented. Boys typically have higher muscle mass and lower fat mass than girls (13). Consistent with this, boys in our cohort had higher muscle mass; however, both sexes showed significant increases in muscle and fat mass following insulin therapy**.** Notably, a significant increase in BMI and BMI SDS was observed only in girls at 6 months, a finding that parallels previous reports of greater weight gain in adolescent girls with T1DM (11,14,15). Unlike prior pediatric studies that primarily focused on weight or fat mass changes, our findings highlight sex-specific differences in the pattern of musculoskeletal response to insulin therapy. When analyzed by pubertal status, both muscle and fat mass were significantly higher in pubertal children at all time points. Furthermore, increases in these parameters were more pronounced in pubertal patients compared to prepubertal peers, supporting the known anabolic effects of insulin and the additional contribution of pubertal hormones such as androgens (16-18). Although BMI was higher in pubertal children, BMI SDS did not significantly differ, suggesting alignment with age- and sex-specific normative data. This may reflect increased bone mass and epiphyseal closure during puberty. Few studies have assessed muscle strength in children with T1DM. In our cohort, muscle strength was significantly lower at diagnosis compared to controls, corroborating previous reports (19-21), though some studies have shown no difference (22). Following insulin therapy, muscle strength improved significantly in both sexes. While boys had higher baseline strength, this difference was not sustained after treatment, possibly due to greater relative gains in girls or pubertal effects (23). Pubertal patients demonstrated greater improvements in both muscle mass and strength compared to prepubertal patients, supporting the role of pubertal hormones and insulin in promoting musculoskeletal development (24). We found strong positive correlations between muscle strength and both muscle and fat mass across all time points. Impaired muscle function in T1DM is multifactorial, involving metabolic, hormonal, and neuromuscular mechanisms (20,25,26,27,28,29). Although calcium and phosphorus levels were lower in our T1DM patients, they remained within normal ranges and were not correlated with muscle strength, suggesting that other mechanisms may contribute to the observed improvements. We identified a moderate negative correlation between HbA1c and both muscle mass and strength at the 3rd month of treatment, highlighting the impact of glycemic control on muscle health. The slight increase in HbA1c observed at 6 months may reflect adolescent insulin resistance or challenges in adherence. These findings suggest that improved glycemic control may contribute to favorable musculoskeletal outcomes in pediatric T1DM. While adult studies have similarly linked better glycemic control with increased skeletal muscle mass (30), conflicting results have also been reported (31), highlighting the complexity of this relationship.

Conclusion

Children and adolescents with newly diagnosed Type 1 Diabetes Mellitus (T1DM) exhibit reduced muscle strength at diagnosis, despite similar muscle and fat mass compared to healthy controls. Our findings suggest that insulin therapy, by restoring glycemic control, may contribute to anabolic processes associated with improvements in both muscle mass and muscle strength , particularly during early treatment. These results highlight the potential importance of optimizing insulin therapy not only for metabolic control but also for musculoskeletal health. Longitudinal monitoring of muscle function and body composition in pediatric T1DM may help identify patients at risk for sarcopenia, osteoporosis, and metabolic complications, and may support early preventive strategies.

Study Limitations

This study has several limitations. First, the control group was evaluated only at baseline, which precluded longitudinal comparisons of muscle strength and muscle mass trajectories between patients and healthy peers. As a result, normal age- and growth-related increases in musculoskeletal parameters among healthy children could not be accounted for, and the observed longitudinal improvements in the T1DM group may partly reflect physiological growth in addition to treatment-related effects, potentially leading to an overestimation of the insulin effect. Second, bone mass, an important contributor to total body weight and body mass index (BMI) was not assessed, which may limit the interpretation of body composition changes. The absence of bone-related measurements restricts the ability to fully differentiate between lean tissue accretion and skeletal growth, particularly during puberty. Additionally, serum calcium levels were measured only once in the control group, preventing comparative analysis of dynamic changes over time. Third, physical activity levels, nutritional status, and detailed insulin regimen characteristics (such as insulin dose adjustments) were not included in the analyses. These factors are known to influence muscle mass, fat mass, and muscle strength and may have affected the magnitude and interpretation of the observed relationships. Finally, the relatively small sample size and short follow-up duration may limit the generalizability of our findings. Future studies involving larger cohorts with extended follow-up and comprehensive skeletal and lifestyle assessments are warranted to validate and expand upon these results.

References

1. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2014;37(Suppl 1):S81-S90.
2. Rhoads RP, Baumgard LH, El-Kadi SW, Zhao LD. Roles for insulin-supported skeletal muscle growth. J Anim Sci. 2016;94(5):1791-1802.
3. Papatheodorou K, Banach M, Bekiari E, Rizzo M, Edmonds M. Complications of diabetes 2017. J Diabetes Res. 2018;2018:3086167.
4. Hatun Ş, Gökçe T, Can E, Eviz E, Karakuş KE, Smart C. Current Management of Type 1 Diabetes in Children: Guideline-based Expert Opinions and Recommendations. J Clin Res Pediatr Endocrinol. 2024 Sep 4;16(3):245-255
5. Wiech P, Bazalinski D, Sulacinska I, et al. Decreased bioelectrical impedance phase angle in hospitalized children and adolescents with newly diagnosed type 1 diabetes: a case-control study. J Clin Med. 2018;7(12):516.
6. de Souza MA, Benedicto MMB, Pizzato TM, Mattiello-Sverzut AC. Normative data for hand grip strength in healthy children measured with a bulb dynamometer: a cross-sectional study. Physiotherapy. 2014;100(4):313-318.
7. Gatt I, Smith-Moore S, Steggles C, Loosemore M. The Takei handheld dynamometer: an effective clinical outcome measure tool for hand and wrist function in boxing. Hand (N Y). 2018;13(3):319-324.
8. Neyzi O, Bundak R, Gökçay G, et al. Reference values for weight, height, head circumference, and body mass index in Turkish children. J Clin Res Pediatr Endocrinol. 2015;7(4):280-293.
9. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969;44(235):291-303.
10. Davis NL, Bursell JD, Evans WD, Warner JT, Gregory JW. Body composition in children with type 1 diabetes in the first year after diagnosis: relationship to glycaemic control and cardiovascular risk. Arch Dis Child. 2012;97(4):312-315.
11. Zheng Y, Rostami Haji Abadi M, Gough J, et al. Higher body fat in children and adolescents with type 1 diabetes: a systematic review and meta-analysis. Front Pediatr. 2022;10:911061.
12. Rosenfalck AM, Almdal T, Hilsted J, Madsbad S. Body composition in adults with type 1 diabetes at onset and during the first year of insulin therapy. Diabet Med. 2002;19(5):417-423.
13. Weber DR, Moore RH, Leonard MB, Zemel BS. Fat and lean BMI reference curves in children and adolescents and their utility in identifying excess adiposity compared with BMI and percentage body fat. Am J Clin Nutr. 2013;98(1):49-56.
14. De Keukelaere M, Fieuws S, Reynaert N, et al. Evolution of body mass index in children with type 1 diabetes mellitus. Eur J Pediatr. 2018;177(11):1661-1666.
15. Bartz J, Sulzbach U, Heinze E, Teller W, Holl R. Körperzusammensetzung bei Typ-I-Diabetes mellitus: Bioimpedanzmessungen bei 274 diabetischen Kindern, Jugendlichen und jungen Erwachsenen. Dtsch Med Wochenschr. 1997;122(25/26):815-819.
16. Maynard LM, Wisemandle W, Roche AF, et al. Childhood body composition in relation to body mass index. Pediatrics. 2001;107(2):344-350.

17. He Q, Karlberg J. BMI in childhood and its association with height gain, timing of puberty, and final height. Pediatr Res. 2001;49(2):244-251.
18. Hou WW, Tse MA, Lam TH, Leung GM, Schooling CM. Adolescent testosterone, muscle mass and glucose metabolism: evidence from the 'Children of 1997' birth cohort in Hong Kong. Diabet Med. 2015;32(4):505-512.
19. Fricke O, Seewi O, Semler O, et al. The influence of auxology and long-term glycemic control on muscle function in children and adolescents with type 1 diabetes mellitus. J Musculoskelet Neuronal Interact. 2008;8(2):188-195.
20. Maratova K, Soucek O, Matyskova J, et al. Muscle functions and bone strength are impaired in adolescents with type 1 diabetes. Bone. 2018;106:22-27.
21. Tan S, Gunendi Z, Meray J, Yetkin I. Evaluation of muscle strength and architecture in type 1 diabetes mellitus: a cross-sectional study. BMC Endocr Disord. 2022;22(1):153.
22. Bechtold S, Dirlenbach I, Raile K, et al. Early manifestation of type 1 diabetes in children is a risk factor for changed bone geometry: data using peripheral quantitative computed tomography. Pediatrics. 2006;118(3):e627-e634.
23. Amo-Setién FJ, Leal-Costa C, Abajas-Bustillo R, González-Lamuño D, Redondo-Figuero C; Group ER. Factors associated with grip strength among adolescents: an observational study. J Hand Ther. 2020;33(1):96-102.
24. Manzano-Carrasco S, Garcia-Unanue J, Haapala EA, et al. Relationships of BMI, muscle-to-fat ratio, and handgrip strength-to-BMI ratio to physical fitness in Spanish children and adolescents. Eur J Pediatr. 2023;182(5):2345-2357.
25. Sala D, Zorzano A. Differential control of muscle mass in type 1 and type 2 diabetes mellitus. Cell Mol Life Sci. 2015;72(20):3803-3817.
26. Monaco CM, Gingrich MA, Hawke TJ. Considering type 1 diabetes as a form of accelerated muscle aging. Exerc Sport Sci Rev. 2019;47(2):98-107.
27. Liamis G, Liberopoulos E, Barkas F, Elisaf M. Diabetes mellitus and electrolyte disorders. World J Clin Cases. 2014;2(10):488-496.
28. Ersoy B, Darcan S, Goksen D, Coker M, Mavi E. Hypercalciuria and hyperphosphaturia in insulin dependent diabetes mellitus. Turk J Endocrinol Metab. 1999;4:177-180.
29. Schubert L, DeLuca HF. Hypophosphatemia is responsible for skeletal muscle weakness of vitamin D deficiency. Arch Biochem Biophys. 2010;500(2):157-161.
30. Tansey MJ, Tsalikian E, Beck RW, et al; Diabetes Research in Children Network (DirecNet) Study Group. The effects of aerobic exercise on glucose and counterregulatory hormone concentrations in children with type 1 diabetes. Diabetes Care. 2006;29(1):20-25.
31. Kaza M, Tsentidis C, Vlachopapadopoulou E, et al. The effect of metabolic profile on leptin, adiponectin, and hs-CRP in children and adolescents with type 1 diabetes. Children (Basel). 2022;9(8):1162.

Table 1. Baseline characteristics, laboratory findings, muscle mass, and body composition levels in T1DM and control groups

Parameters	T1DM group (n=36)	Control group (n=43)	P
Age (years)	9.99 ± 3.02	10.32 ± 2.7	0.613
Gender (M/F) n (%)	19/17 (52.8/47.2)	21/20 (48.8/51.2)	0.727
Pre-pubertal/ Pubertal	19/17	20/23	0.579
Height (cm)	141.69±20.43	140.03 ± 17.1	0.695
Weight (kg)	35.85 (14- 82.9)	34.1 (18.2-78.7)	0.821
BMI (kg/m²)	16.75 (13.2-25.4)	17.3 (13.5-27.7)	0.705
BMI sds	-0.38 (-1.92-1.96)	-0.52 (-1.92-1.91)	0.894
Muscle Mass (kg)	23.8 (10.9-55.5)	22.3 (13.4-51.6)	0.738
Fat Mass (kg)	6.65 (2-20.2)	7.1 (3-26.1)	0.327
Muscle strength (Newton)	10.4 (5-25.5)	15.8 (6.2-31.9)	**0.001**
Fasting blood glucose (mmol/L)	14.85 (11.27-16.60)	5.00 (2.94-6.22)	**<0.001**
Calcium (mmol/L)	2.33 ± 0.14	2.48 ± 0.10	**<0.001**
Phosphorus (mmol/L)	1.22 ± 0.30	1.59 ± 0.22	**<0.001**
ALT (U/L)	13 (7-52)	15 (9-51)	0.085
Hemoglobin (g/L)	135.8 ± 12.4	131.3 ± 10.1	0.080
TSH (Uıu/ml)	2.16 (0.71-4.62)	2.24 (0.65-4.93)	0.867
FT4 (ng/dl)	1.14 (0.8-3.7)	1.22 (0.82-1.4)	0.668

P<0.05
Abbreviations: T1DM: Type 1 Diabetes Mellitus, BMI: Body mass index, sds: standard deviation score, ALT: Alanine aminotransferase, TSH: Thyroid stimulating hormone, FT4: Free thyroxine
Statistical tests: Mann-Whitney U and Pearson chi- square tests were used, median values were given.

Table 2. Parameters in the T1DM patient group by months				
Parameters	0. month	3. month	6. month	p
Muscle strength (N)	10.4 (5-25.5)[a,b]	13 (5.3-31.5)[c]	14.8 (7.4-34.2)	**<0.001**
Fat mass (kg)	6.65 (2-20.2)[a,b]	8.25 (2.2-22.3)	8.25 (2.3-28.3)	**<0.001**
Muscle mass (kg)	23.8 (10.9-55.5)[a,b]	25.6 (11.7-61.5)	24.9 (1.5-58.8)	**<0.001**
BMI (kg/m²)	(17.5 ± 3.28)[a,b]	18.06 ± 3.2	18.57 ± 3.28	**0.004**
BMI sds	(-0.2 ± 1.1)[a,b]	0.05 ± 1.03	0.15 ± 0.99	**0.021**
HbA1c	(13.68 ± 2.59)[a,b]	6.94 ± 1.31	7.15 ± 0.97	**<0.001**
Fasting blood glucose (mmol/L)	14.85 (11.27-16.59)[a,b]	6.11 (3.55-16.48)	7.41 (3.61-15.21)	**<0.001**
Height (cm)	(141.69 ± 20.43)[a,b]	(142.89 ± 20.05)[c]	144.28 ± 19.94	**<0.001**
Weight (kg)	35.85 (14-82.9)[a,b]	36.25 (15.6-81.7)[c]	37.4 (17.3-81.3)	**<0.001**

P<0.05, a= (0-3 month), b= (0-6 month), c= (3-6 month)
Abbreviations: T1DM: Type 1 Diabetes Mellitus, N: Newton, BMI: Body mass index, sds: standard deviation score, HbA1c: glycosylated haemoglobin
Statistical tests: ANOVA, Friedman tests were used and median values were given

Table 3. Parameters in the T1DM girls and boys by months			
Parameters	Girl (n=17)	Boy (n=19)	p
Muscle strength (N) 0.month	8.4 (5-18.5)[a,b]	12.8 (5.7-25.5)[a,b]	**0.030**
3.month	11.2 (5.3-20.1)[c]	14.5 (6.5-31.5)[c]	0.087
6.month	13.8 (7.4-23.4)	15.9 (7.6-34.2)	0.114
P	**<0.001**	**<0.001**	
Fat mass (kg) 0.month	6.5 (2.5-16.2) [a,b]	7.3 (2-20.2) [a,b]	0.684
3.month	7.4 (3.8-17.4)	8.7 (2.2-22.3)	0.707
6.month	8 (4.2-24.8)	8.3 (2.3-28.3)	0.851
P	**<0.001**	**<0.001**	
Muscle mass (kg) 0.month	19 (10.9-36.1) [a,b]	25.6 (11.9-55.5) [a,b]	0.061
3.month	21.9 (11.7-41.3)	29.9 (13.5-61.5)	0.087
6.month	22.1 (11.5-38.6)	31.1 (13-58.8)	0.100
P	**<0.001**	**<0.001**	
BMI (kg/m²) 0.month	(16.79 ± 2.71) [b]	18.25 ± 3.65	0.187
3.month	17.34 ± 2.65	18.71 ± 3.57	0.204
6.month	17.68 ± 2.59	19.35 ± 3.69	0.129
P	**0.024**	0.060	
BMI sds 0.month	(-0.32 ± 1.03) [b]	-0.1 ± 1.18	0.559
3.month	0.04 ± 0.88	0.07 ± 1.18	0.937
6.month	0.08 ± 0.7	0.2 ± 1.2	0.706
P	**0.042**	0.233	
HbA1c 0.month	(14.26 ± 2.69) [a,b]	(13.15 ± 2.45) [a,b]	0.205
3.month	7.05 ± 1.17	6.85 ± 1.45	0.644
6.month	7.11 ± 0.97	7.18 ± 1.01	0.828
P	**<0.001**	**<0.001**	

Table 4. Parameters in the T1DM pre-pubertal and pubertal stages by months			
Parameters	Pre-pubertal (n=19)	Pubertal (n=17)	P
Muscle strength (N) 0.month	(8.51 ± 2.72) [a,b]	(15.44 ± 5.07)[a,b]	**<0.001**
3.month	(10.14 ± 3.67)[c]	(18.46 ± 5.62)[c]	**<0.001**
6.month	11.9 ± 3.72	21.15 ± 6.39	**<0.001**
P	**<0.001**	**<0.001**	
Fat mass (kg) 0.month	4.8 (2-14.1) [a,b]	8.5 (5.1-20.2) [b]	**0.001**
3.month	6.1 (2.2-18.7)	10 (6-22.3)	**0.003**
6.month	6 (2.3-18.2)	10.9 (6.3-28.3)	**0.002**
P	**<0.001**	**0.010**	
Muscle mass (kg) 0.month	(18.95 ± 5.8) [a,b]	(32.95 ± 9.43) [a,b]	**<0.001**
3.month	20.72 ± 6.32	36.29 ± 10.84	**<0.001**
6.month	20.76 ± 6.18	36.55 ± 10.25	**<0.001**
P	**<0.001**	**<0.001**	
BMI (kg/m²) 0.month	16.46 ± 3.01	18.79 ± 3.21 a,b	**0.031**
3.month	16.88 ± 2.86	19.38 ± 3.12	**0.017**
6.month	17.32 ± 2.9	19.96 ± 3.2	**0.014**
P	0.124	**0.015**	
BMI sds 0.month	-0.1 ± 1.19	-0.32 ± 1.03	0.559
3.month	0.24 ± 1.06	-0.15 ± 1	0.268
6.month	0.26 ± 1.05	0.01 ± 0.093	0.455
P	0.155	0.059	
HbA1c 0.month	(13.67 ± 2.3) [a,b]	(13.68 ± 2.95) [a,b]	0.987
3.month	7.33 ± 1.26	6.52 ± 1.26	0.063
6.month	7.42 ± 1.01	6.85 ± 0.87	0.084
P	**<0.001**	**<0.001**	

P<0.05, a=(0-3 month), b= (0-6 month), c= (3-6 month)
Abbreviations: BMI: Body mass index, BMI sds: Body mass index standard deviation score, HbA1c: glycosylated haemoglobin
Statistical tests: Mann-Whitney U, ANOVA and Friedman tests were used.