Sedentary lifestyle and body composition in type 2 diabetes

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Abstract

Background: Body composition alterations may participate in the pathophysiological processes of type 2 diabetes (T2D). A sedentary lifestyle may be responsible for alterations of body composition and adverse consequences, but on which body composition of patients with T2D and to what extent the sedentary lifestyle has an effect have been poorly investigated.

Methods: We recruited 402 patients with T2D for this cross-sectional study. All patients received questionnaires to evaluate sedentary time and were further divided into three subgroups: low sedentary time (LST, < 4 h, n = 109), middle sedentary time (MST, 4–8 h, n = 129) and high sedentary time (HST, > 8 h, n = 164). Each patient underwent a dual energy X-ray absorptiometry (DXA) scan to detect body composition, which included body fat percentage (B-FAT), trunk fat percentage (T-FAT), appendicular skeletal muscle index (ASMI), lumbar spine bone mineral density (BMD) (LS-BMD), femoral neck BMD (FN-BMD), hip BMD (H-BMD) and total BMD (T-BMD). Other relevant clinical data were also collected.

Results: With increasing sedentary time (from the LST to HST group), B-FAT and T-FAT were notably increased, while ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD were decreased (p for trend < 0.01). After adjustment for other relevant clinical factors and with the LST group as the reference, the adjusted mean changes [B (95% CI)] in B-FAT, T-FAT, ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD in the HST group were 2.011(1.014 to 3.008)%, 1.951(0.705 to 3.197)%, −0.377(−0.531 to −0.223) kg/m², −0.083(−0.124 to −0.042) g/cm², −0.051(−0.079 to −0.024) g/cm², −0.059(−0.124 to −0.031) g/cm² and −0.060(−0.088 to −0.033) g/cm², p < 0.01, respectively.

Conclusions: A sedentary lifestyle may independently account for increases in trunk and body fat percentage and decreases in appendicular skeletal muscle mass and BMD of the lumbar spine, femoral neck, hip and total body in patients with T2D.

Keywords: Sedentary lifestyle, Fat distribution, Skeletal muscle mass, Bone mineral density

Introduction

Type 2 diabetes (T2D) is a chronic metabolic disease characterized by sustained hyperglycaemia and is a leading cause of morbidity and mortality [1]. Patients with T2D present with numerous pathophysiological disorders, including alterations in body composition. The characteristics of body composition in T2D, including abnormalities in fat distribution, increases in total fat mass, and decreases in muscle mass or bone mineral density (BMD) [2–4], might lead to a wide spectrum of
adverse health outcomes, such as cardiovascular diseases (CVDs) [5], sarcopenia and osteoporosis [6–8]. At present, ongoing global research efforts are trying to identify intrinsic and external risk factors for alterations in body composition, which can help guide the development of appropriate therapeutic regimens to modify these pathophysiological disorders and the subsequent prognosis of diabetes.

With the rapid development of China’s information society, physical work has become increasingly less common, followed by a sharp increase in the number of sedentary people. Sedentary behaviour refers to any low energy consumption behaviour when a person is awake, including sitting, leaning, or lying down. Studies have shown that sedentary behaviour is inseparable from all-cause mortality, cardiovascular disease mortality and tumour mortality, as well as the incidence of cardiovascular disease, tumours and T2D [9–12]. A survey shows that in the awake state, the sedentary time accounts for at least 50% of total lifetime in T2D patients [10]. Long-term sedentary behaviour is also closely related to the prevalence of human bone and muscle-related diseases [13], leading to an increase in the proportion of lumbar disc disease and a decrease in BMD [14–16]. There is a negative correlation between mineral content [17], the ability of the spinal muscles to maintain maximum output, and muscle strength [18]. A sedentary lifestyle may be responsible for altered body composition and adverse consequences, but on which body composition of patients with T2D and to what extent the sedentary lifestyle has an effect have been poorly investigated.

Therefore, the purpose of this study was to quantify the relationship between sedentary time and body composition in patients with T2D.

**Materials and methods**

**Participants**

We recruited T2D patients in the Endocrinology Department of the Second Affiliated Hospital of Nantong University from March 2020 to May 2021. The inclusion criteria were as follows: patients who: (1) met the 1999 World Health Organization (WHO) T2D diagnosis and classification criteria [19]; (2) were 25 to 75 years old; and (3) were fully aware of the purpose and significance of this study and were willing to sign up to participate. The exclusion criteria were as follows: (1) type 1 diabetes, gestational diabetes and other special types of diabetes; (2) a history of metabolic diseases or diseases affecting nutritional status, such as hyperthyroidism, hypothyroidism, Cushing syndrome, rheumatoid arthritis, etc.; (3) complication with severe chronic diseases, severe infections, immune disorders and malignant tumours; and (4) treatment with glucocorticoids or sex hormones in the past 6 months. Finally, we included 402 eligible patients with complete data for statistical analysis. The study was reviewed and approved by the Ethics Committee of the Second Affiliated Hospital of Nantong University and was in line with the Helsinki Declaration. In addition, all participants provided informed consent when recruited for the study.

**Data collection**

A self-designed questionnaire was used to collect daily sedentary time, demographic data and medical history from all recruited patients. Patients were further divided into three subgroups: low sedentary time (LST, < 4 h), middle sedentary time (MST, 4–8 h) and high sedentary time (HST, > 8 h). Demographic data included age, sex, height, weight, waist circumference (WC), body mass index (BMI), systolic blood pressure (SBP), and diastolic blood pressure (DBP). Body mass index was calculated as follows: BMI = weight (kg)/height (m)². Medical history included diabetes duration, history of hypertension, dietary habits (seafood, milk, soft drinks, tea, coffee, etc.), smoking and drinking history, and glucose-lowering therapies. Glucose-lowering therapies were categorized as insulin secretagogues, insulin, metformin, pioglitazone, α-glucosidase inhibitors (AGIs), dipeptidyl peptidase-4 inhibitors (DPP-4Is), sodium-glucose cotransporter-2 inhibitors (SGLT-2Is) and glucagon-like peptide-1 receptor agonists (GLP-1RAs).

For blood samples collection, each patient fasted overnight for 8 h, and the nurses in the ward took blood from the antecubital vein in the early morning hours of the next day for measurement of biochemical markers. Fasting glucose, triglycerides (TGs, colorimetric method), total cholesterol (TC, cholesterol oxidase method), low-density lipoprotein cholesterol (LDLC, selective melting method) and high-density lipoprotein cholesterol (HDLc, enzyme modification method) were measured by an automatic biochemical instrument (model 7600, Hitachi). The level of HbA1c was assessed by ion exchange high-performance liquid chromatography. The levels of fasting insulin, N-terminal osteocalcin (N-MID), β-collagen special sequence (β-CTX), total type I procollagen N-terminal extension peptide (TP1NP), 25-hydroxyvitamin D (25(OH)D) and parathyroid hormone (PTH) were quantified by electrochemiluminescence immunoassay. All biochemical indices were assessed by professional doctors in the medical laboratory department of our hospital. Basal insulin resistance was assessed by homeostasis model assessment of insulin resistance (HOMA-IR), which was defined as follows:
HOMA-IR = (fasting glucose × fasting insulin)/22.5. HOMA-IR was natural log-transformed for the further data analysis (lnHOMA-IR).

All the subjects in this study underwent scanning with a dual energy X-ray absorptiometry (DXA) (Hologic DiscoveryWt, S/N86856) to assess the body composition of T2D patients, and scanning was performed by professionals in the corresponding medical and technical departments. All operations were carried out in accordance with the specifications of the instrument manual: the patient lay flat and was scanned from the head to the feet in the standard mode. The width of the scanning range was fixed at 60 cm, and the scanning time was approximately 20 min. The measured indices included lumbar L1–L4 bone mineral density (LS-BMD), femoral neck bone mineral density (FN-BMD), hip bone mineral density (H-BMD), total (whole-body) bone mineral density (T-BMD), whole-body fat mass (B-FAT), trunk fat mass (T-FAT) and limb skeletal muscle mass (ASMI).

Clinical variables of recruited patients are exhibited for all participants and the LST (< 4 h), MST (4–8 h) and HST (> 8 h) subgroups. Descriptive statistics for the statistical significance was identified based on a threshold. GraphPad Prism (Version 9.0) to analyse the data. Statistically adjusted for other relevant clinical factors. Model 0, Model 1, Model 2 and Model 3 were gradually adjusted for other relevant clinical factors.

We used IBM SPSS Statistics (Version 22.0) and GraphPad Prism (Version 9.0) to analyse the data. Statistical significance was identified based on a threshold p value < 0.05.

**Results**

**Clinical characteristics of patients**

Table 1 shows the basic clinical characteristics of 402 participants, who were divided into three groups according to the length of sedentary time: LST (< 4 h), MST (4–8 h), and HST (> 8 h). There were 109 (27.1%), 129 (32.1%) and 164 (40.8%) participants in each respective group. With the increase in sedentary time in T2D patients, the main parameters of body composition, such as LS-BMD, FN-BMD, H-BMD, T-BMD and ASMI, decreased significantly (p < 0.001). For the patients with sedentary time ≥ 4 h, B-FAT and T-FAT were higher than those in patients with sedentary time < 4 h (p = 0.009), which is shown in Fig. 1. In addition, with the increase in sedentary time, the duration of diabetes increased, Hba1c increased, the levels of N-MID and 25(OH)D decreased, and the levels of β-CTX, TP1NP and PTH increased (p < 0.001). Regarding the living habits of patients, as the duration of sedentary time increased, a higher frequency of drinking soft drinks, tea or coffee, smoking and drinking and a lower frequency of eating seafood and drinking milk were observed. However, there was no significant difference in sex, BMI, WC, HC, SBP, history of hypertension, TG, TC, HDLC or LDLC with prolonged sedentary time (p > 0.05). Among the glucose-lowering therapies, the frequency of AGIs, SGLT-2Is and GLP-1RAs use increased with the prolongation of sedentary time, while lifestyle alone and use of insulin, insulin-secretagogues, metformin, pioglitazone and DPP-4Is remained the same.

**Mean differences in body composition parameters between the three sedentary duration groups**

Tables 2 and 3 present multivariate linear regression models, taking the LST (< 4 h) group as a reference, to display the mean changes of body composition parameters in the MST (4–8 h) and HST (> 8 h) groups (B[95%CI]). The mean differences in B-FAT, T-FAT, ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD between the LST (< 4 h) group and HST (> 8 h) group were 2.289 (0.630 to 3.948)% 2.338 (0.594 to 4.082)% − 0.700 (0.971 to − 0.429) kg/m², − 0.152 (0.189 to − 0.115) g/cm², − 0.125 (0.152 to − 0.099) g/cm², − 0.121 (0.149 to − 0.093) g/cm² and − 0.112 (0.138 to − 0.086) g/cm², respectively. After adjustment for other relevant clinical factors and with the LST group as the reference, the adjusted mean changes [B (95% CI)] in B-FAT, T-FAT, ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD in the HST group were 2.011 (1.014 to 3.008)% 1.951 (0.705 to 3.197)% − 0.377 (0.531 to − 0.223) kg/m², − 0.083 (0.124 to − 0.042) g/cm², − 0.051 (0.079 to − 0.024) g/cm², − 0.059 (0.087 to − 0.031) g/cm² and − 0.060 (0.088 to − 0.033) g/cm², respectively. Sedentary time was independently and positively correlated
Table 1 Clinical characteristics of the total patients and subgroups based on the sedentary time

| Variables | Total       | Sedentary time | F/χ² value | p for trend |
|-----------|-------------|----------------|------------|-------------|
|           | LST (< 4 h) | MST (4–8 h)   | HST (> 8 h)|             |
| n         | 402         | 109(27.1)      | 129(32.1)  | 164(40.8)   | –            | –            |
| Female, n(%) | 173(43.0) | 44(40.0)       | 58(45.4)   | 71(42.7)    | 0.170        | 0.680        |
| Age(year) | 55.20 ± 10.45 | 53.06 ± 9.11  | 55.21 ± 10.96 | 56.60 ± 10.71 | 7.611        | 0.006        |
| BMI(kg/m²) | 25.4 ± 3.7 | 25.2 ± 3.2     | 26.5 ± 3.6 | 24.8 ± 4.1  | 0.900        | 0.343        |
| WC(cm)    | 90.5 ± 10.2 | 89.4 ± 8.4     | 93.2 ± 10.6 | 89.1 ± 10.5 | 0.053        | 0.891        |
| SBP(mmHg) | 133.0 ± 14.4 | 132.7 ± 14.2  | 134.7 ± 14.5 | 131.7 ± 14.3 | 0.287        | 0.593        |
| DBP(mmHg) | 81.6 ± 9.2 | 82.8 ± 8.7     | 82.7 ± 9.0 | 79.8 ± 9.4  | 6.873        | 0.009        |
| Diabetes duration(year) | 7.48 ± 5.70 | 5.26 ± 4.20 | 6.86 ± 5.41 | 9.44 ± 6.14 | < 0.001      |

Glucose-lowering therapies

- Lifestyle alone, n(%) 33(8.2) 9(8.3) 16(12.4) 8(4.9) 1.517 0.218
- Insulin treatments, n(%) 183(45.5) 47(43.1) 50(38.8) 86(52.4) 2.954 0.086
- Insulin-secretagogues, n(%) 98(24.4) 26(23.9) 33(25.6) 39(23.8) 0.003 0.954
- Metformin, n(%) 175(43.5) 45(41.3) 63(48.8) 67(40.9) 0.058 0.810
- Pioglitazone, n(%) 64(15.9) 18(16.5) 17(13.2) 5(17.7) 0.141 0.707
- AGLs, n(%) 96(23.9) 16(14.7) 35(27.1) 45(27.4) 5.203 0.023
- DPP-4s, n(%) 53(13.2) 12(11.0) 17(13.2) 24(14.6) 0.74 0.390
- SGLT-2s, n(%) 97(25.2) 23(21.1) 29(22.5) 45(27.4) 5.199 0.023
- GLP-1RAs, n(%) 46(11.4) 8(7.3) 10(7.8) 28(17.1) 6.966 0.008

Hypertension, n(%) 179(44.5) 44(40.4) 74(57.4) 61(37.2) 0.853 0.356

Smoking, n(%) 142(35.3) 25(22.9) 35(27.1) 82(50.0) 23.22  < 0.001

Alcohol consumption, n(%) 226(56.2) 44(40.4) 50(38.8) 120(52.4) 43.06  < 0.001

Seafood, n(%) 141(35.2) 49(45.0) 50(38.8) 42(25.8) 11.16 0.001

Milk, n(%) 149(37.1) 54(49.5) 46(35.7) 49(29.9) 10.38 0.001

Soft drink, n(%) 304(75.6) 68(62.4) 91(70.5) 145(88.4) 25.55  < 0.001

Tea or coffee, n(%) 209(52.3) 38(35.2) 51(39.5) 120(73.6) 43.06  < 0.001

TG(mmol/L) 2.51 ± 1.88 2.49 ± 1.65 2.99 ± 2.13 2.14 ± 1.73 2.436 0.119

TC(mmol/L) 4.36 ± 0.97 4.20 ± 0.97 4.42 ± 0.83 4.41 ± 1.05 3.191 0.075

HDLc(mmol/L) 1.15 ± 0.30 1.17 ± 0.28 1.12 ± 0.30 1.17 ± 0.30 0.016 0.900

LDLc(mmol/L) 2.82 ± 0.85 2.89 ± 0.83 2.74 ± 0.81 2.84 ± 0.89 0.206 0.650

N-MID(ng/mL) 11.7 ± 3.8 12.43 ± 4.03 11.78 ± 3.50 11.16 ± 3.83 7.372 0.007

β-CTXng/mL) 0.46 ± 0.20 0.43 ± 0.20 0.43 ± 0.18 0.49 ± 0.21 5.538 0.019

TP1NP(ng/mL) 40.22 ± 12.33 38.21 ± 11.59 39.34 ± 11.40 42.24 ± 13.24 7.058 0.008

25(OH)D3ng/mL) 17.5 ± 6.5 18.83 ± 6.11 18.12 ± 6.95 16.12 ± 6.11 11.76 0.001

PTH(pg/mL) 40.70 ± 16.91 35.86 ± 13.45 41.81 ± 20.10 43.03 ± 15.64 12.10 0.001

HbA1c(%) 9.04 ± 1.76 8.53 ± 1.68 8.89 ± 1.52 9.50 ± 1.87 21.00 0.001

HOMA-IR 3.29 ± 3.52 3.22 ± 4.63 3.55 ± 3.05 3.12 ± 2.99 0.047 0.828

InHOMA-IR 0.81 ± 1.10 0.69 ± 1.08 0.91 ± 1.00 0.82 ± 0.96 0.952 0.330

B-FAT(%) 30.76 ± 6.89 29.04 ± 6.75 31.48 ± 6.13 31.33 ± 7.38 7.357 0.007

T-FAT(%) 33.28 ± 7.27 31.29 ± 7.15 34.54 ± 6.37 33.63 ± 7.76 6.949 0.009

ASM(kg/m²) 7.09 ± 1.16 7.38 ± 1.18 7.36 ± 1.07 6.68 ± 1.11 25.81 0.001

LS-BMD(g/cm²) 0.97 ± 0.16 1.05 ± 0.15 0.99 ± 0.16 0.90 ± 0.14 66.35 0.001

FN-BMD(g/cm²) 0.78 ± 0.12 0.84 ± 0.10 0.79 ± 0.12 0.72 ± 0.11 86.43 0.001

H-BMD(g/cm²) 0.90 ± 0.12 0.97 ± 0.11 0.91 ± 0.17 0.85 ± 0.11 71.65 0.001

T-BMD(g/cm²) 1.10 ± 0.12 1.17 ± 0.10 1.12 ± 0.11 1.06 ± 0.11 70.39 0.001

ANOVA followed by a post-test for linear trend and linear-by-linear association chi-squared test were applied to detect trends in continuous data and categorical data among sedentary time, respectively. Corresponding test statistics and p values for trends are also provided.
Discussion
In this study, we systematically analyzed the relationship between sedentary time and body composition in 402 patients with T2D. First, through univariate analysis, sedentary time was found to be closely related to B-FAT, T-FAT, ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD in T2D patients; second, multiple linear regression showed that sedentary duration was independently correlated with B-FAT, T-FAT, ASMI, LS-BMD, FN-BMD and H-BMD and T-BMD in T2D patients. Compared with the LST group, the HST group showed an increase in B-FAT and T-FAT, and the corresponding adjusted mean changes were 2.011(1.014 to 3.008) and 1.951(0.705 to 3.197), respectively. Additionally, compared with the LST group, HST group exhibited a decrease in ASMI, LS-BMD, FN-BMD, H-BMD and T-BMD, and the corresponding adjusted mean changes were $-0.377(-0.531$ to $-0.223)$, $-0.083(-0.124$ to $-0.042)$, $-0.051(-0.079$ to $-0.024)$, $-0.059(-0.087$ to $-0.031)$, and $-0.060(-0.088$ to $-0.033)$, respectively. Third, with the increase in sedentary time in T2D patients, HbA1c levels were significantly increased, while there were no significant differences in TG, TC and HDL-C levels between the three subgroups with different sedentary behaviors.

Studies have shown that the biological and metabolic characteristics of fat and muscle tissue in different parts of the body are different. In general, the risk of metabolic diseases is proportional to the total amount of fat in the body. Abdominal obesity caused by the accumulation of abdominal visceral adipose tissue (VAT) is more closely related to hypertension, dyslipidaemia and insulin resistance (IR) than obesity caused by the accumulation of subcutaneous adipose tissue (SAT), which promotes glycogen heterogeny, lipid synthesis and IR enhancement, leading to the occurrence of metabolic diseases such as glucose and lipid metabolism disorders [25]. Studies have shown that lack of exercise and prolonged sitting are the main factors leading to obesity and IR [26, 27].

Bones and muscles make up the skeletal muscle system. An increasing number of studies have shown that bones and muscles share common paracrine and endocrine regulation. In ageing and chronic diseases, bone loss and muscle atrophy occur at the same time [28]. Muscle mass peaks around the age of 25, followed by loss of muscle mass after the age of 30, a slight decrease in the number of muscle fibers at the age of 50 and rapid decline after the age of 60. Similar to muscle loss, bone mass increases before age 30, peaks around age 30, and then enters a phase of bone loss, which is more pronounced in postmenopausal women. Although bone loss and muscle atrophy were corrected with aging, appendicular skeletal muscle mass (ASMI) and BMD of the hip, lumbar spine, femoral neck and total body were decreased with the increasing of sedentary time ($p$ for trend $<0.01$), even after adjusting for age. Studies have shown that an increase or decrease in muscle mass is positively correlated with an increase or decrease in BMD, respectively, and that the loss of skeletal muscle can lead to a decrease in BMD. Muscle atrophy, muscle strength decline and muscle dysfunction can accelerate thin cortical bone absorption, weaken the ability to resist shear force, torsion and bending force, reduce the number of horizontal trabeculae in cancellous bone, diminish the vertical trabeculae, and decrease BMD [29]. In addition, there has been growing clinical concern with the effect of glucose-lowering agents on bone metabolism with B-FAT and T-FAT, and was independently and negatively correlated with ASMI, LS-BMD, FN-BMD and H-BMD and T-BMD in T2D patients.

(See figure on next page.)

Fig. 1 Scatter plots of body composition indices in three subgroups with low sedentary time (LST), middle sedentary time (MST) and high sedentary time (HST). A body fat percentage (B-FAT); B trunk fat percentage (T-FAT); C appendicular skeletal muscle index (ASMI); D lumbar spine bone mineral density (LS-BMD); E hip bone mineral density (H-BMD); F femoral neck bone mineral density (FN-BMD); G total bone mineral density (T-BMD)
Fig. 1 (See legend on previous page.)
metabolism. In our study, we found the frequency of AGIs, SGLT-2Is and GLP-1RAs use increased with the prolongation of sedentary time. These glucose-lowering therapies were more frequent in the T2D patients with longer sedentary time and higher HbA1c, and may have effects on bone mass and bone mineral density in these T2D patients. A previous study has shown that liraglutide, one of the GLP-1RAs, can improve bone mineral density and bone quality while reducing blood glucose in diabetic rats [30]. SGLT-2Is can increase the reabsorption of phosphorus in renal tubules, which may affect calcium and phosphorus metabolism, increase blood phosphorus, stimulate PTH secretion and enhance bone resorption. In a multicenter RCT study, 104-week treatment with canagliflozin may result in a mild but significant decrease in bone mineral density of the total hip rather than the femoral neck, lumbar vertebrae or distal forearm in women with T2D when compared to the placebo group [31]. However, fewer studies are available in the field of AGIs with bone metabolism [32]. In our present study, we should consider the effects of glucose-lowering therapies on bone metabolism. Furthermore, after adjusted for these glucose-lowering therapies by multivariate linear regression analysis, we found that different sedentary time was responsible for body composition independent of glucose-lowering therapies.

The changes in human muscle mass are related to poor nutrition, the weakening of hormones, metabolism, and immune function, and the degeneration of motor units and muscle fibres [33]. Muscle fibre degeneration leads to decreased muscle mass, and muscle fibre atrophy is one of the causes of sarcopenia. In T2D patients, IR, ectopic lipid deposition, oxidative stress, endoplasmic reticulum stress, accumulation of advanced glycation end-products (AGEs), neuropathy and nephropathy can all cause damage. The synthesis and decomposition balance of skeletal muscle [34, 35] and IR are the main pathogenic mechanisms of sarcopenia caused by diabetes. IR reduces protein synthesis by inhibiting the activation of the skeletal muscle differentiation signalling pathway and increases muscle protein decomposition by activating the ubiquitin–proteasome pathway [36]. AGE accumulation can inhibit the expression of myogenic genes and osteocalcin in myoblasts, thus aggravating muscle and bone loss [37]. Studies have shown that approximately 50% of diabetes patients have diabetic osteoporosis (OP), and with the extension of the course of diabetes, the incidence of diabetic OP gradually increases. Second, a large number of epidemiological studies have shown that the risk of hip, foot and proximal femoral fracture in patients with diabetes is significantly higher than that in nondiabetic people. Tebé et al. [38] conducted a follow-up study of 50,000 T2D patients and 100,000 nondiabetic people for 6 to

### Table 2

| Models     | Sedentary time | t value | p for trend | Adjusted R² |
|------------|----------------|---------|-------------|-------------|
|            | LST (< 4 h)    | MST (4–8 h) | HST (> 8 h) |             |
| ASMI       |                |         |             |             |
| Model 0    | 0-reference    | -0.020 (−0.305 to 0.265) | -0.700 (−0.971 to −0.429) | -5.391 | <0.001 | 0.060 |
| Model 1    | 0-reference    | 0.042 (−0.185 to 0.270) | -0.660 (−0.876 to −0.444) | -6.395 | <0.001 | 0.395 |
| Model 2    | 0-reference    | -0.057 (−0.224 to 0.110) | -0.331 (−0.510 to −0.152) | -3.671 | <0.001 | 0.715 |
| Model 3    | 0-reference    | -0.004 (−0.237 to 0.050) | -0.377 (−0.531 to −0.223) | -4.865 | <0.001 | 0.799 |
| B-FAT      |                |         |             |             |
| Model 0    | 0-reference    | 2.439 (0.693 to 4.186) | 2.289 (0.630 to 3.948) | 2.519 | 0.012 | 0.013 |
| Model 1    | 0-reference    | 1.994 (0.754 to 3.234) | 2.005 (0.827 to 3.183) | 3.137 | 0.002 | 0.502 |
| Model 2    | 0-reference    | 0.663 (−0.314 to 1.640) | 2.005 (0.959 to 3.050) | 3.790 | <0.001 | 0.723 |
| Model 3    | 0-reference    | 0.560 (−0.369 to 1.490) | 2.011 (1.014 to 3.008) | 4.011 | <0.001 | 0.760 |
| T-FAT      |                |         |             |             |
| Model 0    | 0-reference    | 3.249 (1.407 to 5.091) | 2.338 (0.594 to 4.082) | 2.325 | 0.021 | 0.011 |
| Model 1    | 0-reference    | 2.933 (1.437 to 4.429) | 2.090 (0.675 to 3.506) | 2.548 | 0.011 | 0.345 |
| Model 2    | 0-reference    | 1.327 (0.120 to 2.534) | 2.050 (0.762 to 3.337) | 3.120 | 0.002 | 0.624 |
| Model 3    | 0-reference    | 1.047 (−0.118 to 2.213) | 1.951 (0.705 to 3.197) | 3.079 | 0.002 | 0.667 |

Model 0: unadjusted
Model 1: additionally adjusted for sex
Model 2: additionally adjusted for age, WC, SBP, DBP, diabetes duration, hypertension, smoking, alcohol and glucose-lowering therapies
Model 3: additionally adjusted for BMI, HbA1c, lnHOMA-IR and lipid profiles
8 years and showed that after excluding the confounding factor of death, the risk of hip fracture in T2D patients was still higher than that of nondiabetic people. In T2D patients, insulin deficiency leads to a decrease in renal tubular reabsorption, loss of calcium and phosphorus, bone calcium mobilization, increase in bone resorption, decrease in bone formation and decrease in BMD. At the same time, insulin deficiency inhibits osteocalcin synthesis by osteoblasts, thus weakening bone mineralization and bone cell activity and inhibiting bone formation [39].

OP increases the risk of falls, while reduced muscle mass and decreased function reduce strength, lead to limited movement, and increase the risk of falls and fractures [40].

There is evidence that sedentary behaviour has a direct effect on metabolism, bone mineral content and vascular health [41]. Excessive sedentary behaviour can aggravate chronic diseases. One of the effects of sedentary behaviour is metabolic disorder, which is characterized by an increase in TG levels, a decrease in HDLC levels,
a reversible decrease in multiple organ insulin sensitivity and cardiopulmonary adaptability, and an increase in liver fat and dyslipidaemia, resulting in metabolic disorders and changes in body composition [42]. This is because prolonged sedentary time may lead to a decrease in skeletal muscle contractile activity, thereby reducing the activity of lipoprotein lipase (LPL) in muscle [43]. In addition, rat experiments have shown that there is a relationship between LPL and limb activity [44]. On the one hand, the sedentary state decreases the physiological activity of muscle, which leads to a decrease in LPL activity and oxidation disorder, which has an adverse effect on muscle fibres. On the other hand, sedentary behaviour reduces the basal metabolism of the body, which is often accompanied by an increase in food intake and a decrease in energy output, resulting in weight gain and an increase in glucose concentration, which further promotes the occurrence of T2D [45]. Therefore, an increase in physical activity can induce an increase in LPL activity and have a positive effect on metabolic glycylsated fibres [46]. However, in our study, the TG levels in the HST group had decreased trend than MST group. In addition, the HDLC level did not change between these three groups. T2D patients in our study have received glucose-lowering therapies. Glucose-lowering agents, such as SGLT-2Is and GLP-1RAs, can regulate serum lipid metabolism [47–49], which may account for the differences of lipid profiles between the present and previous studies. At the same time, interrupting sitting for a long time through simple activities such as standing or walking can reduce the concentration of postprandial glucose and insulin [50, 51], which plays an important role in the prevention and control of T2D. Another adverse effect of sedentary behaviour is a decrease in BMD. The relationship between sedentary behaviour and osteopenia is regulated by changes in the balance between bone resorption and deposition. Sedentary behaviour will lead to a rapid increase in bone resorption but will not be accompanied by changes in bone formation, resulting in a decrease in bone mineral content and an increase in the risk of OP. In addition, sedentary time is related to decreased body function and blood flow in the legs, which may lead to individual falls, bone loss and fractures. LaMonte et al. [52] found that regular physical activity and less sedentary time reduced the risk of fracture in older women and that women who spent more than 9.5 h a day being sedentary had a 4% increased risk of fracture compared with those who had the shortest sedentary time. A meta-analysis evaluated the relationship between total sedentary time and all-cause mortality [11]. Another meta-analysis reported that adults with a sedentary time of more than 8 h a day have a higher risk of death from cardiovascular disease, while adults with a higher exercise level have a lower risk of death [12]. At the same time, there is a nonlinear dose–response relationship between sedentary time and all-cause mortality, cardiovascular mortality, cancer mortality and cardiovascular adverse events, but it varies with exercise levels [12, 53]. In general, sedentary behaviour had a stronger impact on health for people with a low exercise level but had less impact on people who performed moderate- or even high-intensity exercise for a long time [53]. HUNT Study explored the interaction between sedentary time and physical activity and found that, compared with people who were sedentary for less than 4 h a day, people with a daily sedentary time of 5–7 h and those with more than 8 h had a 26% and 30% increased risk of T2D, respectively [54]. In China, the average daily sedentary time of diabetes patients is as high as 6.1 h. The sedentary time of T2D patients can account for 70% of their daily waking time. To reduce the harmful effects of sedentary behaviour on health, sedentary time should be shortened as much as possible. For people who cannot avoid being sedentary due to the nature of their work, exercise at higher than the recommended level should be carried out every week to mediate the health hazards of being sedentary to a certain extent.

Limitations
First, the subjects selected in this study included men and women in a large age range (25–75 years old). T2D patients of a single sex and in the same age group have not been studied, but this study is closer to the clinical situation. Second, this study is only a cross-sectional study and did not perform a follow-up survey. The results of the study cannot be compared before and after, and a follow-up survey is needed to confirm the authenticity and reliability of the research results.

Conclusions
A sedentary lifestyle may account for increases in trunk and body fat percentage and decreases in appendicular skeletal muscle mass and BMD of the lumbar spine, femoral neck, hip and total body in patients with T2D. These results may indicate that sedentary time may have an effect on body composition in T2D.

Abbreviations
T2D: Type 2 diabetes; LST: Low sedentary time; MST: Middle sedentary time; HST: High sedentary time; DXA: Dual energy X-ray absorptiometry; B-FAT: Body fat percentage; T-FAT: Trunk fat percentage; ASMI: Appendicular skeletal muscle index; BMD: Bone mineral density; LS-BMD: Lumbar spine BMD; FN-BMD: Femoral neck BMD; H-BMD: Hip BMD; T-BMD: Total BMD; WC: Waist circumference; SBP/DBP: Systolic/diastolic blood pressure; BMI: Body mass index; AGIs: α-Glucosidase inhibitors; DPP-4Is: Dipeptidyl peptidase-4 inhibitors; SGLT-2Is: Sodium-glucose cotransporter-2 inhibitors; ANOVA: One-way analysis of variance; TG: Triglycerides; TC: Total cholesterol; HDLC: High-density lipoprotein
The authors declare that they have no competing interests.

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Authors' contributions

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Declarations

Ethics approval and consent to participate

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Competing interests

Author details

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