Acta Med. Okayama, 2017
Vol. 71, No. 2, pp. 143-149

Acta Medica Okayama
http://escholarship.lib.okayama-u.ac.jp/amo/

Original Article

Toe Pinch Force in Male Type 2 Diabetes Mellitus Patients

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We compared the toe pinch force in men with and without type 2 diabetes mellitus (T2DM). Sixty-eight male T2DM patients and 35 apparently healthy men matched for age, sex, and body mass index (BMI) were enrolled in this cross-sectional study. We compared the toe pinch force between the subjects with and without T2DM, and we evaluated the effect of diabetic polyneuropathy on toe pinch force in the patients. The toe pinch force of the T2DM patients was significantly lower than that of the subjects without diabetes (3.12 ± 1.22 kg vs. 4.40 ± 1.19 kg, $p < 0.001$). Multiple regression analysis showed that T2DM was a determinant of reduced toe pinch force. In addition, the toe pinch force of patients with diabetic polyneuropathy was significantly lower than that of patients without diabetic polyneuropathy (2.31 ± 0.93 kg vs. 3.70 ± 1.07 kg, $p < 0.001$). Multiple regression analysis showed that diabetic polyneuropathy was a determinant of the toe pinch force in men with T2DM, even after adjusting for age, BMI, HbA1c, and duration of diabetes. Reduced toe pinch force is a fundamental feature of motor dysfunction in men with T2DM, and diabetic polyneuropathy might be associated with toe pinch force in these patients.

Key words: type 2 diabetes mellitus, toe pinch force, diabetic polyneuropathy

Diabetic polyneuropathy, a major complication of type 2 diabetes mellitus (T2DM), develops from an early stage of diabetes, and it causes a variety of dysfunctions. Muscle weakness of the lower limbs is a well-known result of diabetic polyneuropathy. Lower-limb muscle strength was reported to be impaired in T2DM patients, and diabetic polyneuropathy was shown to be associated with the reduced muscle strength [1]. It has been thought that sensory disturbance is the main characteristic of diabetic polyneuropathy and that symptoms of motor neuropathy, such as muscle weakness, do not occur until the chronic phase [2]. However, it was clarified that not only sensory disturbance but also motor impairment is observed from an early stage of diabetes [1] and that the characteristic of the motor impairment is dominant peripheral muscle weakness (more severe in the foot than in the lower leg and the thigh). Reduced lower-limb muscle strength would make it more difficult to perform aerobic exercise, and diabetic polyneuropathy commonly affects peripheral sensation. Thus, the accurate evaluation of both lower-limb muscle strength and sensory function might be necessary in clinical practice.

Toe pinch force is a parameter of muscle strength that is closely associated with stabilization of the posture [3,4], toe lift-off in the late-stance phase of walking [5], and the ability to perform exercise. We have reported on the reproducibility of a new method for the measurement of toe pinch force [6], and we observed

Received August 31, 2016; accepted November 10, 2016.
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Conflict of Interest Disclosures: No potential conflict of interest relevant to this article was reported.

that toe pinch force showed a significant positive correlation with other muscle strength parameters in T2DM patients such as handgrip strength, knee extension force, and ankle dorsiflexion force [7]. However, the correlation was not as strong as we had expected, and toe pinch force may thus need to be evaluated in addition to other muscle strength parameters in clinical practice. There are no reports of comparisons of toe pinch force between subjects with and without T2DM, and it has not been known whether toe pinch force is reduced in individuals with T2DM compared to healthy subjects.

The present cross-sectional study is the first to compare the toe pinch force between subjects with and without T2DM. We also evaluated the relationships between toe pinch force and the presence of diabetic polyneuropathy and disease factors such as hemoglobin A1c (HbA1c), the duration of diabetes.

Subjects and Methods

T2DM patients and control subjects. Sixty-eight men with T2DM and 35 apparently healthy men matched for age, sex, and body mass index (BMI) were enrolled in this cross-sectional study. The T2DM patients were classified as either positive or negative for neuropathy according to the criteria for diabetic polyneuropathy [8]. Diabetic patients were recruited from the inpatients of KKR Takamatsu Hospital (Table 1) among 2,931 outpatients seen at the hospital's Department of Diabetes and Endocrinology from April 2012 to March 2016 who met the following criteria: (1) hospital admission was recommended (346 patients), (2) they provided written informed consent to participate in this study, (3) they were advised to increase their physical activity and perform exercise, and (4) they did not have an existing exercise habit. Patients were excluded if they had severe cardiac or lung disease, an acute or chronic musculoskeletal disorder, acute metabolic dysregulation, other neurological or endocrine disorders, a history of stroke, previous or current asymmetric proximal lower-limb weakness, toe deformity, or atrophy of foot muscles, or if they were already performing regular exercise. Control subjects matched for age, sex, and BMI were recruited from among the KKR Takamatsu Hospital staff. They had undergone an annual heath check-up and had no disorders.

The study was approved by the Ethics Committee of KKR Takamatsu Hospital and informed consent was obtained from all participants.

Clinical and laboratory measurements. We collected data on the subjects' age (years), height (cm), body weight (kg), BMI (kg/m^2), duration of T2DM (years), ankle-brachial index, cardio-ankle vascular index, medications, and laboratory test results. In all T2DM patients, the levels of fasting plasma glucose (mg/dl), HbA1c (%), total cholesterol (mg/dl), LDL-cholesterol (mg/dl), HDL-cholesterol (mg/dl), and triglycerides (mg/dl) were measured by standard methods. The ankle-brachial index and cardio-ankle vascular index were measured with a VS-1500 (Fukuda Denshi, Tokyo, Japan). The diagnosis of T2DM was based on the fasting plasma glucose level and HbA1c level, and was made according to the Japan Diabetes Society (JDS) guidelines [8].

Measurement of toe pinch force. Toe pinch force was measured by using the same dynamometer and testing procedure as in previous reports [6,7]. The dynamometer was a Checker-kun (Nisshin Sangyo, Saitama, Japan). The subject sat on a chair with arms crossed over the chest (90° hip joint flexion, 90° knee joint flexion, and 90° ankle joint flexion), and the dynamometer was attached to the foot at the interdigital space between the great toe and second toe. The test was performed twice each on the left and right sides, and the best results for the left and right feet were averaged.

Assessment of chronic complications of diabetes. The diagnoses of diabetic retinopathy and diabetic nephropathy were based on the JDS guidelines [8], while diabetic polyneuropathy was diagnosed according to the Japanese criteria [8]. As in our previous study [7], diabetic polyneuropathy was defined as being present if the patient had at least 2 of the following: (1) symptoms such as dysesthesia, pain, and/or loss of sensation, (2) decreased or absent Achilles tendon reflex, and (3) decreased vibratory sensation (≤ 10 sec).

Statistical analysis. All measured and calculated values are expressed as the mean ± standard deviation. Comparisons between 2 groups were performed using the unpaired-t test, Mann-Whitney's *U*-test and χ^2 test. Comparisons among 3 groups were done by one-way analysis of variance (ANOVA) and the Tukey test. *P*-values < 0.05 were accepted as significant. We performed a multiple regression analysis to identify factors that were determinants of the toe pinch force.

Table 1 Clinical characteristics of the patients with type 2 diabetes and control subjects

	Control subjects	T2DM	*p*-value
n	35	68	
Age (years)	58.5 ± 11.5	55.7 ± 12.4	0.258
Height (cm)	167.9 ± 5.7	168.8 ± 5.2	0.468
Weight (kg)	73.1 ± 12.4	77.4 ± 13.1	0.107
BMI (kg/m^2)	25.8 ± 3.7	27.0 ± 4.0	0.145
Smoking habit			
Current smoker (n/%)	10 / 28.6	17 / 25.0	0.086
Previous smoker (n/%)	8 / 22.9	30 / 44.1	
Non-smoker (n/%)	17 / 48.5	21 / 30.9	
Drinking habit (n/%)	15 / 42.9	25 / 36.8	0.548
HbA1c (%)		9.7 ± 2.5	
Fasting plasma glucose (mg/dl)		191.8 ± 100.0	
Total cholesterol (mg/dl)		197.8 ± 37.2	
LDL-cholesterol (mg/dl)		117.0 ± 25.2	
HDL-cholesterol (mg/dl)		46.9 ± 14.8	
Triglycerides (mg/dl)		194.0 ± 140.0	
Duration of diabetes (years)		9.9 ± 9.1	
ABI		1.1 ± 0.1	
CAVI		8.4 ± 1.4	
Polyneuropathy (n/%)		28 / 41.4	
Nephropathy			
Stage 1 (n/%)		52 / 76.4	
Stage 2 (n/%)		7 / 10.3	
Stage 3 (n/%)		8 / 11.8	
Stage 4 (n/%)		1 / 1.5	
Nephropathy			
Stage 1 (n/%)		42 / 61.8	
Stage 2 (n/%)		21 / 30.9	
Stage 3 (n/%)		3 / 4.4	
Stage 4 (n/%)		2 / 2.9	
Drug theraphy			
Insulin (n/%)		15 / 22.1	
OHA (n/%)		34 / 50.0	
Insulin and OHA (n/%)		11 / 16.2	

Value are presented as the mean ± SD.
T2DM, type 2 diabetes mellitus; BMI, body mass index; ABI, ankle-brachial index; CAVI, cardio-ankle vascular index; OHA, oral hypoglycemic agent.

The required sample size was estimated on the basis of findings in our earlier studies [6,7], *i.e.*, that the toe pinch force of healthy controls and T2DM patients was 5.8 kg and 3.2 kg, respectively. The difference to be detected was 2.6 kg, and the standard deviation was 0.5. To detect an effect size of this magnitude with 80% power assuming a two-sided test at alpha = 0.05, it was estimated that only 5 participants would be required in this study. The sample size of this study (68 T2DM patients and 35 control subjects) was thus considered to have sufficient numbers of subjects to evaluate the toe pinch force as the main outcome. In addition, the power of this study was estimated as approx. 1.0 with 0.05 alpha.

All analyses were performed using JMP 12.1.0 software (SAS, Cary, NC, USA).

Results

The clinical profiles of the subjects are summarized in Table 1. There were no significant differences in age, height, body weight, or BMI between the subjects with T2DM (n = 68) and those without T2DM (n = 35). The toe pinch force of the T2DM patients was significantly lower than that of the healthy controls (3.12 ± 1.22 kg vs. 4.40 ± 1.19 kg, $p < 0.001$) (Fig. 1), by approx. 29%.

We performed the multiple regression analysis using the toe pinch force as a dependent variable, with age, BMI, and T2DM as independent variables. The results showed that only T2DM was a determinant of the toe pinch force in all subjects (Table 2).

We next compared the toe pinch force among three groups: the controls, the T2DM patients without diabetic polyneuropathy (Non-DPN, n = 40), and the T2DM patients with diabetic polyneuropathy (DPN, n = 28). The toe pinch force of the DPN group was significantly lower than that of the Non-DPN group (2.31 ± 0.93 kg vs. 3.70 ± 1.07 kg, $p < 0.001$). Compared to the controls, the toe pinch force in the DPN and Non-DPN groups was reduced by approx. 49% and 16%, respectively (Fig. 2). In addition, the duration of T2DM was significantly longer in the DPN group than in the Non-DPN group (14.8 ± 9.2 years vs. 6.4 ± 7.2 years, $p < 0.01$) (Table 3).

We evaluated the difference of toe pinch force as classified by the sub-criterion of diabetic polyneuropathy (Table 4). The toe pinch force in patients with an abnormal (decreased or absent) Achilles tendon reflex was significantly lower than that in the subjects with normal Achilles tendon reflex: 2.83 ± 1.19 kg vs. 3.70 ± 1.09 kg, $p = 0.004$, respectively. The same was true of the subjects with abnormal vibratory sensation (2.28 ± 0.79 kg vs. 3.39 ± 1.21 kg, $p = 0.001$) and dysesthesia, pain, and/or loss of sensation symptoms (2.00 ± 0.88 kg vs. 3.45 ± 1.11 kg, $p < 0.001$).

Lastly, we performed a multiple regression analysis of the 68 patients with T2DM using toe pinch force as the dependent variable and age, BMI, duration of diabetes, HbA1c, and diabetic polyneuropathy as independent variables. We found that only diabetic polyneuropathy was a determinant of the toe pinch force (Table 5).

Discussion

Our main objective in this study was to compare the

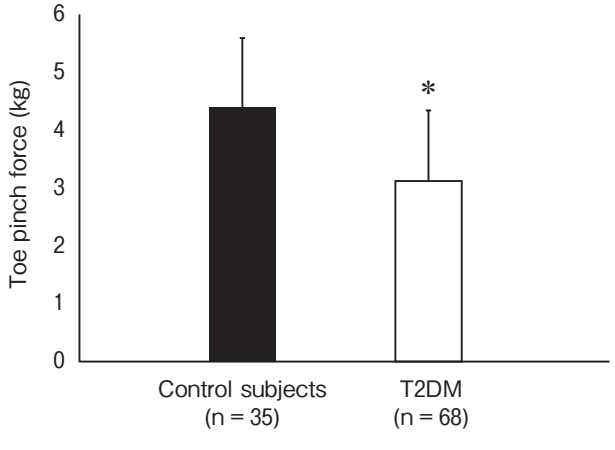

Fig. 1 Comparison of the toe pinch force between the patients with T2DM and the control subjects. T2DM: type 2 diabetes mellitus.
*$p < 0.001$ vs. control subjects.

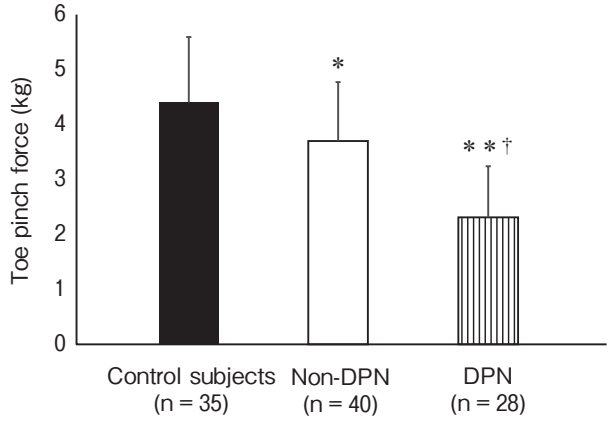

Fig. 2 Comparison of the toe pinch force among the Non-DPN, DPN, and control subjects. Non-DPN, non-diabetic polyneuropathy; DPN, diabetic polyneuropathy; *$p = 0.016$ vs. control subjects, **$p < 0.001$ vs. control subjects, †$p < 0.001$ vs. Non-DPN.

Table 2 Multiple regression analysis of the relation between toe pinch force and type 2 diabetes

	B	*β*	*p*-value	95% CI	
Constant	3.653	–	0.008	0.975	6.331
Age	−0.002	−0.015	0.886	−0.024	0.020
BMI	0.032	0.095	0.353	−0.037	0.102
T2DM	−1.314	−0.465	<0.001	−1.820	−0.808

$R^2 = 0.212$. ANOVA $p < 0.001$
B, partial regression coefficient; *β*, standardized partial regression cefficient; T2DM, type 2 diabetes mellitus; BMI, body mass index.

Table 3 Clinical characteristics of the non-DPN and DPN groups and the control subjects

	Control subjects	Non-DPN	DPN
n	35	40	28
Age (years)	58.5 ± 11.5	54.6 ± 12.9	57.3 ± 11.8
Height (cm)	167.9 ± 5.7	169.3 ± 5.4	168.0 ± 4.9
Weight (kg)	73.1 ± 12.4	78.7 ± 12.1	75.6 ± 14.4
BMI (kg/m^2)	25.8 ± 3.7	27.4 ± 3.9	26.4 ± 4.2
Smoking habit			
Current smoker (n/%)	10/28.6	10/25.0	7/25.0
Previous smoker (n/%)	8/22.8	17/42.5	13/46.4
Non-smoker (n/%)	17/48.6	13/32.5	8/28.6
Drinking habit (n/%)	15/42.9	13/32.5	12/42.9
HbA1c (%)		10.1 ± 2.8	9.2 ± 1.9
Fasting plasma glucose (mg/dl)		181.1 ± 104.7	207.2 ± 92.5
Total cholesterol (mg/dl)		202.0 ± 5.9	191.8 ± 7.0
LDL-cholesterol (mg/dl)		120.2 ± 28.9	112.4 ± 18.4
HDL-cholesterol (mg/dl)		44.8 ± 9.9	50.0 ± 19.6
Triglycerides (mg/dl)		178.3 ± 21.8	216.3 ± 2.6
Duration of diabetes (years)		6.4 ± 7.2	14.8 ± 9.2*
ABI		1.12 ± 0.1	1.09 ± 0.1
CAVI		8.3 ± 1.5	8.6 ± 1.4
Retinopathy			
none (n/%)		32/80.0	20/71.4
simple (n/%)		3/7.5	4/14.3
preproliferative (n/%)		4/10.0	4/14.3
proliferative (n/%)		1/2.5	0/0
Nephropathy			
Stage 1 (n/%)		23/57.5	19/67.9
Stage 2 (n/%)		13/32.5	8/28.6
Stage 3 (n/%)		2/5.0	1/3.5
Stage 4 (n/%)		2/5.0	0/0
Drug theraphy			
Insulin (n/%)		6/15.0	9/32.1
OHA (n/%)		20/50.0	14/50.0
Insulin and OHA (n/%)		8/20.0	3/10.7

Value are presented as the mean ± SD. BMI, body mass index; ABI, ankle-brachial index; CAVI, cardio-ankle vascular index; OHA, oral hypoglycemic agent; *$p < 0.01$ vs. Non-DPN.

Table 4 Comparison of toe pinch force as classified by sub-criterion of diabetic polyneuropathy in men with type 2 diabetes mellitus

	Achilles tendon relfex		Vibratory sensation		Symptomsa	
	Normal	Abnormal	Normal	Abnormal	Normal	Abnormal
n	23	45	52	16	53	15
Toe pinch force (kg)	3.70 ± 1.09	2.83 ± 1.19*	3.39 ± 1.21	2.28 ± 0.79*	3.45 ± 1.11	2.00 ± 0.88*

Values are the mean ± SD. Symptomsa: adysesthesia, pain, and/or loss of sensation.
*$p < 0.01$ vs. normal.

toe pinch force between men with and without T2DM. The relationship between the reduction of the toe pinch force and T2DM has not been fully established. Almurdhi *et al.* reported that increased muscle fat mass was associated with reduced lower-limb muscle strength [9]. There have also been reports that aging is associated with lower toe muscle strength [3,10,11]. In the present cross-sectional study, we evaluated the toe pinch force in patients with T2DM and compared it with that in age-matched controls. There was also no

Table 5 Multiple regression analysis of the relation between toe pinch force and diabetic polyneuropathy

	B	*β*	*p*-value	95% CI	
Constant	3.376	—	0.049	0.009	6.742
Age	0.008	0.071	0.547	0.071	1.293
BMI	0.024	0.071	0.540	0.071	1.256
Duration of diabetes	−0.024	−0.163	0.182	−0.163	1.368
HbA1c	0.066	0.122	0.264	0.122	1.100
DPN	−1.212	−0.445	<0.001	−1.848	−0.577

$R^2 = 0.340$. ANOVA $p < 0.001$

B, partial regression coefficient; *β*, standardized partial regression coefficient; BMI, body mass index; DPN, diabetic polyneuropathy.

age difference between the diabetic patients with and without diabetic polyneuropathy, and thus aging was not associated with the lower toe pinch force.

Andersen *et al.* reported that the strength of the knee flexors, ankle flexors, and ankle extensors was respectively reduced by 14%, 17%, and 14% in T2DM patients compared with age- and sex-matched controls, and they stated that diabetic polyneuropathy was closely associated with reduced muscle strength [3]. Our present findings showed that the toe pinch force was significantly lower in men with T2DM than in men without diabetes. In addition, the toe pinch force of our patients with diabetic polyneuropathy was reduced by approx. 49% compared to that of apparently healthy men, and diabetic polyneuropathy was a significant determinant of reduced toe pinch force in the T2DM patients according to the multiple regression analysis.

There are no prior studies that compared toe pinch force in patients with and without T2DM, to the best of our knowledge. Our present findings provide new evidence about reduced toe pinch force in T2DM. It is estimated that >50% of T2DM patients have diabetic polyneuropathy, and this polyneuropathy initially affects the distal parts of the limbs [12]. The onset of diabetic polyneuropathy precedes the development of diabetic retinopathy and diabetic nephropathy [13]. Some patients even have diabetic polyneuropathy before developing overt T2DM, *i.e.*, impaired glucose tolerance (IGT: 13%) and impaired fasting glucose (IFG: 11%) [14]. Thus, the evaluation of the toe pinch force could be important since reduced toe pinch force may be a fundamental feature of T2DM that should be identified in clinical practice.

With regard to the relation between foot muscle atrophy and T2DM, there have been some reports that intrinsic muscle atrophy was observed in both type 1 and type 2 DM by magnetic resonance imaging (MRI) [15,16]. Diabetic polyneuropathy is closely associated with intrinsic muscle atrophy [17]. However, we excluded T2DM patients with foot muscle atrophy from the present study, and we thus could not evaluate the link between intrinsic muscle atrophy and T2DM. In addition, the subjects of Andersen *et al.* were patients with a mean duration of illness that was ≥29 years, whereas it was much shorter (14.8 years) in our study [17].

Ijzerman *et al.* reported that muscle weakness of the feet was observed before the onset of diabetic polyneuropathy [18]. Andersen *et al.* showed that the muscle weakness that occurred in type 1 diabetic patients was not due to diabetic polyneuropathy or muscle atrophy [19]. Muscle weakness might develop earlier than muscle atrophy, and this link should be investigated in the future.

A diabetic patient's foot can require amputation, which markedly reduces the quality of life (QOL); preventive foot care is thus quite important for individuals with T2DM. Various factors are involved in the development of a diabetic foot, such as neuropathy, infection, and ulceration; toe deformity is a cause of ulceration. The characteristic deformity in diabetes is hammer/claw toe, and intrinsic muscle atrophy has been suggested to cause this structural change of the foot. Such a deformity combined with a limited range of motion of the foot joints [20] and callus formation due to elevation of the plantar pressure can lead to ulceration. Since diabetic patients have an appreciable risk of developing a diabetic foot, the evaluation of these patients' toe pinch force may provide useful information that will help prevent the development or progression of diabetic foot.

Andersen *et al.* reported that patients with T2DM had 'motor dysfunction' of the lower limbs [13]. Reduced knee and ankle muscle strength is recognized as motor dysfunction, which is related to future falls, a

bedridden state, and reduced QOL. Based on our present findings, it is apparent that (1) reduced toe pinch force is a characteristic finding in men with T2DM, and (2) impairment of toe pinch force may be an important contributor to motor dysfunction. In clinical practice, it may thus be important to prevent motor dysfunction in T2DM patients by the performance of resistance training of the toes.

This study had some limitations. First, it was cross-sectional rather than longitudinal, and we thus could not confirm that muscle weakness occurred due to diabetic polyneuropathy. Second, the definitions of diabetic polyneuropathy and muscle atrophy may not accurate. The definition of diabetic polyneuropathy used in this study was based on Japanese screening criteria and not on a nerve conduction study. In addition, muscle atrophy of the feet was assessed by an examiner, not using MRI. Third, we only evaluated men with T2DM. Fourth, we could not prove the mechanism linking reduced toe pinch force with T2DM. Nevertheless, it is noteworthy that reduced toe pinch force was a characteristic feature of these patients with T2DM, suggesting that in clinical practice the evaluations of toe pinch force could be useful for prescribing exercise and promoting physical activity. Further studies are needed to test our present findings.

In conclusion, we compared the toe pinch force between men with and without T2DM, and the results revealed that the toe pinch force in the T2DM patients was approx. 29% lower than that of the healthy controls. Our findings also showed the clinical impact of diabetic polyneuropathy on the further reduction of the toe pinch force in patients with T2DM.

Acknowledgments. This study was partly supported by a research grant from 114 Bank (Kagawa, Japan).

References

1. Andersen H, Nielsen S, Mogensen CE and Jakobsen J: Muscle strength in type 2 diabetes. Diabetes (2004) 53: 1543–1548.
2. Dobretsov M, Romanovsky Dv and Stimers JR: Early diabetic neuropathy: triggers and mechanisms. World J Gastroenterol (2007) 13: 175–191.
3. Endo M, Ashton-Miller JA and Alexander NB: Effects of age and gender on toe flexor muscle strength. J Gerontol A Biol Sci Med Sci (2002) 57: M392–397.
4. Menz HB, Morris ME and Lord SR: Foot and ankle characteristics associated with impaired balance and functional ability in older people. J Gerontol A Biol Sci Med Sci (2005) 60: 1546–1552.
5. Mann R and Inman VT: Phasic activity of intrinsic muscles of the foot. J Bone Joint Surg Am (1964) 46: 469–481.
6. Kataoka H, Ishikawa A, Hayashino S, Yamamoto K, Miyazawa Y, Tanaka S, Kurato R, Hirao T and Miyatake N: Reproducibility of toe pinch dynamometer procedure. J Dist Environ Health Welf Res (2015) 18: 39–44.
7. Kataoka H, Miyatake N, Kitayama N, Murao S, Kohi F and Tanaka S: Relationship of toe pinch force to other muscle strength parameters in men with type 2 diabetes. Environ Health Prev Med (2016) 21: 179–185.
8. Tajima N, Noda M, Origasa H, Noto H, Yabe D, Fujita Y, Goto A, Fujimoto K, Sakamoto M and Haneda M: Evidence-based practice guideline for the treatment for diabetes in Japan 2013. Diabetol Int (2015) 6: 151–187.
9. Almurdhi MM, Reeves ND, Bowling FL, Boulton AJ, Jeziorska M and Malik RA: Reduced lower-limb muscle strength and volume in patients with type 2 diabetes in relation to neuropathy, intramuscular fat, and vitamin D levels. Diabetes Care (2016) 39: 441–447.
10. Menz HB, Zammit GV, Munteanu SE and Scott G: Plantar flexion strength of the toes: age and gender differences and evaluation of a clinical screening test. Foot Ankle Int (2006) 27: 1103–1108.
11. Scott G, Menz HB and Newcombe L: Age-related differences in foot structure and function. Gait Posture (2007) 26: 68–75.
12. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM, Wilson DM, O'Brien PC, Melton LJ 3rd and Service FJ: The prevalence by staged severity of various types of diabetic neuropathy, retinopathy and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology (1993) 43: 817–824.
13. Andersen H: Motor dysfunction in diabetes: Diabetes Metab Res Rev (2012) 28 (Suppl 1): 89–92.
14. Ziegler D, Rathmann W, Dickhaus T, Meisinger C and Mielck A; KORA study Group: Prevalence of polyneuropathy in pre-diabetes and diabetes is associated with abdominal obesity and macroangiopathy: the MONICA/KORA Augsburg surveys S2 and S3. Diabetes Care (2008) 31: 464–469.
15. Bus SA, Yang QX, Wang JH, Smith MB, Wunderlich R and Cavanagh PR: Intrinsic muscle atrophy and toe deformity in the diabetic neuropathic foot: a magnetic resonance imaging study. Diabetes Care (2002) 25: 1444–1450.
16. Greenman RL, Khaodhiar L, Lima C, Dinh T, Giurini JM and Veves A: Foot small muscle atrophy is present before the detection of clinical neuropathy. Diabetes Care (2005) 28: 1425–1430.
17. Andersen H, Gjerstad MD and Jakobsen J: Atrophy of foot muscles: a measure of diabetic neuropathy. Diabetes Care (2004) 27: 2382–2385.
18. Ijzerman TH, Schaper NC, Melai T, Blijham P, Meijer K, Willems PJ and Savelberg HH: Motor nerve decline does not underlie muscle weakness in type 2 diabetic neuropathy. Muscle Nerve (2011) 44: 241–245.
19. Andersen H, Schmits O and Nielsen S: Decreased isometric muscle strength after acute hyperglycemia in type 1 diabetic patients. Diabet Med (2005) 22: 1401–1407.
20. Zimny S, Schatz H and Pfohl M: The role of limited joint mobility in diabetic patients with an at-risk foot. Diabetes Care (2004) 27: 942–946.