

Sonographic Evaluation of the Achilles Tendon and Plantar Fascia of Type 2 Diabetics in Nigeria

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Abstract

Background: The aim of this study is to compare the Achilles tendon (AT) thickness (ATT) and plantar fascia (PF) thickness (PFT) of diabetics with and without peripheral neuropathy (PN) to that of a control population. **Materials and Methods:** B-mode sonography of the AT and PF was done. Correlation analysis was used to determine the relationship between ATT and PFT with demographic data such as body mass index, duration of diabetes, and presence of PN. A multivariate regression was used to construct models for determining the thicknesses. **Results:** Eighty type 2 diabetics were recruited and categorized into groups based on the presence or absence of PN (Groups A and B, respectively). Group A constituted 57 participants while there were 23 in Group B. Eighty controls constituted Group C. Mean values of 6.08 ± 0.65 , 5.08 ± 0.48 , and 4.57 ± 0.57 mm ($P < 0.001$) of the right ATT were obtained in Groups A to C while values of 1.95 ± 0.35 , 1.88 ± 0.39 , and 1.44 ± 0.20 mm ($P < 0.001$) were obtained for the right PFT. **Conclusion:** The presence of PN and factors such as diabetes duration can affect the thickness of AT and PF.

Keywords: Achilles tendon, diabetes type 2, peripheral neuropathy, plantar fascia, ultrasonography

INTRODUCTION

Diabetes mellitus (DM) is an endocrine disease with a prevalence of 5.1% worldwide,^[1] and Nigeria has the highest number of people living with DM in Africa.^[2-4] Diabetic neuropathy occurs in 50% of individuals with long-standing DM, manifesting as autonomic neuropathy.^[5] Plantar ulcers characterized by the triad of neuropathy, infection, and ischemia^[6] are also a known complication of DM. The global incidence of DM foot is estimated to be 15%, and 12%–24% of individuals with diabetic foot ulcers require amputation.^[7] Amputation risks in diabetic patients are about 15–40 times higher than in nondiabetic patients,^[8,9] and the risk of lower extremity amputation increases by a factor of 8 once an ulcer develops.^[10-14]

DM predisposes to plantar fasciopathy, a disorder characterized by thickened plantar fascia (PF), and loss of the normal organized PF architecture.^[15] PF is one of those tissues that may change their physiology and biomechanical function in the presence of chronic hyperglycemia. Studies by Sharkey

et al.^[16] revealed that the PF actively contributes to influence the pressure acting on the metatarsal heads.

The pathophysiology defining affection of the PF and Achilles tendon (AT) in DM can be summarized as running the following course: sustained hyperglycemia promotes increased glycosylation of proteins, resulting in accumulation of “advanced glycosylation end products” in patient’s soft tissue and in thickening and vascularization of the AT and PF. The increased AT thickness (ATT) and PF thickness (PFT) in DM have been considered an expression of soft-tissue damages.^[17] The superficial anatomical positions of the AT and PF allow high-resolution imaging with ultrasonography.^[18-20]

This study was done to evaluate the ATT and PFT in Nigerian diabetics. Our research hypotheses were that there is increased thickness of AT and PF in diabetics compared with controls

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and that the thickness of AT and PF increases with the duration of DM.

MATERIALS AND METHODS

This was a descriptive, cross-sectional, nonrandomized study carried out from February 2015 to January 2016 in a tertiary health-care facility, on 80 participants with type 2 DM (T2DM) aged 40 and 80 years consecutively recruited from the diabetes clinic of the institution along with 80 age- and sex-matched apparently healthy controls. The sample size was calculated by Leslie Fisher's formula (with 10% attrition rate factored in) using a reported prevalence rate of 4.7% in T2DM in a previous study.^[21]

The healthy controls were required to have fasting blood glucose (FBG) of <6.1 mmol/l and with no known history of DM. Diagnostic criteria for DM include fasting plasma glucose of 126 mg/dl (7.0 mmol/l) or higher on two separate tests.^[5] Also, symptoms of diabetes together with a random blood glucose of ≥ 200 mg/dl (11.1 mmol/l) suggests diabetes. Two-hour plasma glucose ≥ 200 mg/dl (11.1 mmol/l) during an oral glucose tolerance test is an indication of DM.^[5]

Participants with renal failure, dyslipidemia, history of smoking, congenital ankle deformities, chronic use of steroids, Charcot neuroarthropathy and hallux rigidus as a result of previous traumas, history of previous plantar fasciitis or injury, chronic heel pain, amputation involving the lower limbs, history of peripheral vascular disease, neurological (other than those of diabetic etiology), musculoskeletal, or rheumatoid disease along with athletes, bodybuilders, and smokers were excluded from the study.

All participants had their weight (kg) and height (m) measured and their body mass index (BMI) calculated by dividing their weight in kilograms by the square of their height in meters. Approval for the study was obtained from the ethics and research committee of the institution. A written informed consent was obtained from all participants.

A short clinical history was obtained from participants to know the duration of diagnosis of diabetes and to inquire of any previous foot ulcers. Physical examination was done for the diabetic participants, and the presence or absence of peripheral neuropathy (PN) was assessed using 10 g Semmes-Weinstein monofilament. The following parts of the foot were tested: the dorsal surface between the base of the first and second toes, the plantar surface of the first, third, and fifth toes, plantar surface of the first, third, and fifth metatarsal heads, the plantar surface of the medial and lateral midfoot, and the heel in a random order.^[22] The 10 g monofilament was pressed at perpendicular to the part examined in order to bend the monofilament for 1 s. Participants only responded with "yes" when they felt the press of the monofilament. The presence of PN was documented when participants did not feel/respond at more than 4 out of 10 sites tested.^[22]

Biochemical blood analysis

FBG concentration was measured using spectrophotometer (a chemistry analyzer), and glycated hemoglobin (HbA1c)

was assessed by chromatography (using Siemens glycosylated hemoglobin machine) to determine the level of glycemic control. HbA1c was done for the diabetic participants only while FBG was done for all the study participants. Controls with FBG of ≥ 6.1 mmol/l or a known history of diabetes were excluded from the study among the controls.

Ultrasound examination

Ultrasound evaluation of the AT and PF was done using the MINDRAY® real-time ultrasound scanner model DC-7 (Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China) equipped with a 7.5–12.0 MHz high-frequency linear array transducer. Sonography was performed by the first author who was blinded to the participants' clinical details.

To examine the AT, the participants were requested to lie down in a prone position on an examination couch with their feet extending beyond the edge of the couch. The participants were adequately exposed by rolling up the clothing to the knee level. With the ankle in a relaxed neutral position at an angle of 90° and the feet pointing downward, a layer of acoustic gel was applied over the area of the AT.^[23] The ultrasound probe was then applied as perpendicular to the tendon as possible so as to prevent anisotropy. Depth and gain settings were adjusted to achieve acceptable images.

Scanning of the tendon was done in longitudinal and transverse planes from the myotendinous junction of the AT to its insertion on the calcaneus. The thickness was measured on transverse plane at the level of the medial malleolus [Figure 1a and b], similar to previous studies.^[19,23,24] Both the right and left AT were evaluated, and their thicknesses were measured three consecutive times to minimize intraobserver variability and the mean value for each side was recorded.

To examine the PF, participants were positioned prone on the examination table with feet overhanging the edge and toes pointing away from the body. The PF was examined from its calcaneal insertion to the region of the forefoot under the metatarsophalangeal joints. The thickness of the fascia was measured at a distance of 3 cm from its calcaneal insertion [Figure 2a and b]. This site was chosen for its high reproducibility.^[25] For each participant, three measurements

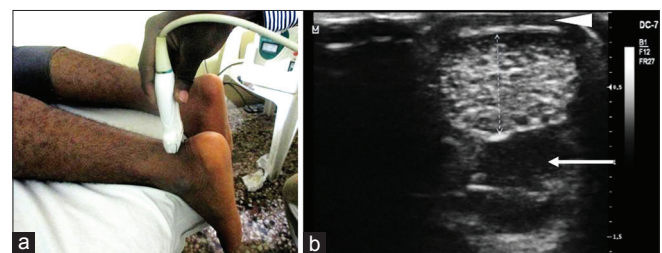


Figure 1: (a) Patient and probe positioning for measuring the Achilles tendon thickness; (b) corresponding sonographic image of the Achilles tendon (short-axis view) showing the skin and subcutaneous tissue (arrowhead), Achilles tendon (between cursors) and pre-Achilles fat pad (arrow)

each of the left and right PF were obtained to minimize intraobserver variability and the mean PFT was calculated for each foot.

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) software version 20 for Windows (SPSS Inc., Chicago, Illinois, USA). Descriptive analysis was performed using the mean and standard deviation to summarize quantitative continuous variables while frequencies and percentages, n (%), were used for categorical variables. Inferential bivariate analysis was carried out using the Pearson Chi-square test to detect any association between the categorical participant's characteristics in both the T2DM participants and the control groups while the independent sample t -test was used to compare means of quantitative continuous variables among them. Bivariate linear relationship between numeric participant parameters, ATT, and PFT was determined using the Pearson correlation analysis for normally distributed variables while Spearman correlation was used for the relationship with duration of DM. The level of statistical significance was set at $P \leq 0.05$.

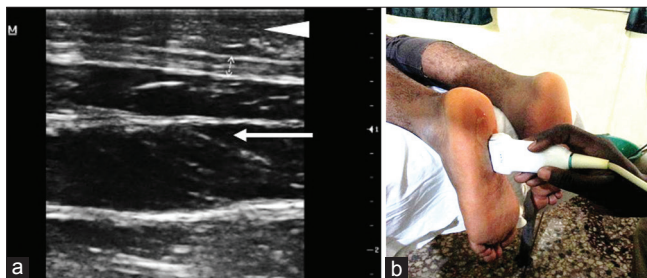


Figure 2: (a) Patient and probe positioning for measuring the plantar fascia thickness; (b) Corresponding sonographic image of the plantar fascia (long-axis view) showing the plantar fat tissue (arrowhead), plantar fascia (between cursors), and plantar muscles (arrow)

RESULTS

A total of 160 participants were recruited comprising 80 T2DM and 80 non-diabetic controls matched for age and sex. The mean age for the T2DM participants and apparently healthy controls was 60.9 ± 10.3 and 61.0 years, respectively ($P = 0.963$). There were 30 male and 50 female T2DM participants as well as 34 male and 46 female controls recruited for this study. The age group with the highest frequency was the seventh decade (60–69 years) with 36.3% of T2DM participants and 35% of controls while participants in their forties (40–49 years) constituted the age group with the least frequency having 18.8% of T2DM participants and 20.0% of controls [Table 1]. The Pearson's correlation with the age only showed statistically significant value for the right ATT [Table 2]. There was a significant correlation with r value of 0.314 ($P < 0.05$) between the age and the duration of diabetes.

Among the diabetics, 46.3% had normal BMI, 32.4% were overweight, and 21.3% were obese [Table 1]. No significant correlation was found between the BMI and tendon thicknesses using Pearson's correlation [Table 2]. Among the controls, 50.0%, 37.5%, and 12.5% had BMI that was normal, overweight, and obese, respectively [Table 1]. Contrary to the findings in the diabetic group, statistically significant ($P < 0.05$) results were obtained among controls in the correlation of ATT and PFT with BMI. $R = 0.34, 0.356, 0.248$, and 0.255 were obtained in the right ATT, left ATT, right PFT, and left PFT, respectively.

The median duration of DM was 42.0 months with a range of 1–456 months. Majority (53.8%) of the diabetic participants were diagnosed <5 years ago while 30% and 16.2% had been diagnosed of T2DM for 5–10 and >10 years, respectively. A correlation done to assess the relationship between duration of diabetes and thickness of AT and PF showed significant

Table 1: Participants' demographic characteristics and clinical parameters across the two groups

Variables	Study groups		χ^2	df	P
	Diabetics ($n=80$), n (%)	Controls ($n=80$), n (%)			
Age (years)					
Mean \pm SD	60.9 \pm 10.3	61.0 \pm 10.3	–0.046	158	0.963*
Range	41.0–79.0	40.0–79.0			
40–49	15 (18.8)	16 (20.0)	0.106	3	0.991
50–59	16 (20.0)	15 (18.8)			
60–69	29 (36.3)	28 (35.0)			
70–79	20 (25.0)	21 (26.3)			
Gender					
Male	30 (37.5)	34 (42.5)	0.417	1	0.519
Female	50 (62.5)	46 (57.5)			
Weight (kg)	70.0 \pm 12.1	67.0 \pm 10.6	1.635	158	0.104*
Height (m)	1.6 \pm 0.1	1.6 \pm 0.1	0.111	158	0.912*
BMI (kg/m ²)	25.92 \pm 4.50	25.15 \pm 3.80	1.167	158	0.245*
Normal	37 (46.3)	40 (50.0)	2.217	2	0.330
Overweight	26 (32.4)	30 (37.5)			
Obese	17 (21.3)	10 (12.5)			

*Independent samples t -test. df: Degree of freedom, BMI: Body mass index, SD: Standard deviation

differences for both ATT alone [Table 2]. Only the ATT showed significant values based on this correlation with the highest correlation value noted in the left AT.

Thirty-nine (48.8%) of the diabetic participants had poor FBG levels (FBG >7.0 mmol/L), 25 (31.2%) had impaired levels (FBG = 5.6–6.9 mmol/L), while 16 (20.0%) had good levels. Pearson's correlation was significant between the FBG levels and the ATT, but no relationship was found with PFT [Table 2].

Fifty-one (63.8%) diabetics had HbA1c values of >7.0% indicating poor glycemic control while the remaining 29 (36.2%) had good glycemic control. No significant correlation was noted between HbA1c values and ATTs [Table 2].

Fifty-seven (71.3%) of the diabetics had PN while the remaining 23 (28.8%) did not have. Three groups constituting diabetics with PN (Group A), diabetics without PN (Group B), and control participants (Group C) had the mean thickness of the AT and PF of both feet compared [Table 3]. The highest mean thicknesses of the AT and PF were both noted on the left in Group A with values of 6.15 ± 0.66 and 1.97 ± 0.4 mm, respectively. Significant differences ($P < 0.001$) were noted between mean thicknesses of the AT and PF between the three groups on both the right and left.

A multivariate regression analysis (stepwise method) using ATT as the outcome variable while age, duration of DM, and presence or absence of PN as predictor variables was done in the diabetics. Preliminary analysis was done to ensure that

there was no violation of the assumption of normality, linearity, and multicollinearity. The best model revealed that age and presence of PN were statistically significant for predicting right ATT, with PN recording a higher beta value than age [Table 4]. Only the FBG and presence of PN were found to be statistically significant for predicting left ATT. The R^2 and R values of the model built to predict the right ATT were 0.485 and 0.696, respectively, while the values obtained for the left ATT were 0.436 and 0.66, respectively.

DISCUSSION

The increased ATT and PFT in DM have been considered an expression of soft-tissue damages.^[17] The ensuing biomechanical alterations may predispose patients with DM to a greater risk of foot ulcers.^[26] Ultrasound gives allowance to assess the muscle and tendon echogenicity and thickness, as well as assisting the interventions into various musculoskeletal disorders.^[27,28]

In this study, the ATT of both limbs of T2DM was significantly thicker than corresponding age- and sex-matched controls. This is comparable with what was observed by Abate *et al.*^[18] and other researchers.^[19] Furthermore, the PFT of both limbs of T2DM participants was significantly higher than that of controls ($P < 0.001$). This is also in keeping with the findings of D'Ambrogi *et al.*^[26] and Duffin *et al.*^[25] Similarly, the significant difference in both ATT and PFT between DM and control participants is corroborated by the findings of Abate *et al.*^[18,29,30]

Table 2: Correlation of Achilles tendon thickness and plantar fascia thickness with different continuous variables

	Age (P)	BMI (P)	DM duration (P)	FBG (P)	HbA1c (P)
Diabetics					
Right ATT	0.173 (<0.05)	-0.084 (>0.05)	0.268 (<0.05)	0.246 (<0.05)	0.061 (>0.05)
Left ATT	0.129 (>0.05)	-0.113 (>0.05)	0.337 (<0.05)	0.234 (<0.05)	0.068 (>0.05)
Right PFT	0.006 (>0.05)	0.067 (>0.05)	0.057 (>0.05)	0.168 (>0.05)	-0.009 (>0.05)
Left PFT	0.09 (>0.05)	0.22 (>0.05)	0.031 (>0.05)	0.055 (>0.05)	0.0 (>0.05)
Controls					
Right ATT	0.019 (>0.05)	0.340 (<0.05)			
Left ATT	-0.032 (>0.05)	0.356 (<0.05)			
Right PFT	-0.052 (>0.05)	0.248 (<0.05)			
Left PFT	-0.066 (>0.05)	0.255 (<0.05)			

ATT: Achilles tendon thickness, PFT: Plantar fascia thickness, BMI: Body mass index, DM: Diabetes mellitus, HbA1c: Glycated hemoglobin, FBG: Fasting blood glucose

Table 3: The differences in Achilles tendon and plantar fascia thicknesses between diabetes mellitus participants with and without peripheral neuropathy and controls

Variables	Group A (n=57)	Group B (n=23)	Group C (n=80)	F	P
Right foot PN					
Right AT diameter (mm)	6.08±0.65	5.08±0.48	4.57±0.57	108.487	<0.001
Right PF thickness (mm)	1.95±0.35	1.88±0.39	1.44±0.20	55.217	<0.001
Left foot PN					
Left AT diameter (mm)	6.15±0.66	5.00±0.65	4.54±0.56	107.528	<0.001
Left PF thickness (mm)	1.97±0.40	1.81±0.43	1.45±0.20	43.808	<0.001

AT: Achilles tendon, PF: Plantar fascia, PN: Peripheral neuropathy, DM: Diabetes mellitus

Table 4: Multivariate regression model for predictors of Achilles tendon thickness

Variables	B	SE	P
Right Achilles tendon			
Age (years)	0.018	0.006	0.006
FBG	0.041	0.014	0.004
Peripheral neuropathy*	0.885	0.145	0.000
Constant	3.713	0.397	0.000
Left Achilles tendon			
FBG	0.042	0.016	0.012
Peripheral neuropathy*	1.162	0.161	0.000
Constant	4.543	0.189	0.000

*Peripheral neuropathy: absent and present coded as 0 and 1, respectively.

B: Unstandardized regression coefficient, SE: Standard error, FBG: Fasting blood glucose

With increasing duration of diabetes, it would be expected that the soft-tissue changes associated with diabetes could be exaggerated. However, we found a significant but very weak positive correlation between duration of diabetes and both ATT and PFT.

Among the diabetics, a trend of increase in ATT and PFT with increasing BMI was observed, but the difference was not statistically significant (all $P > 0.05$). This is in agreement with the findings of Duffin *et al.*^[25] but at variance with the findings of Abate *et al.*^[18] who found a significant correlation between the BMI and ATT as well as PFT. This perhaps could be because Abate *et al.*^[18] evaluated only recently diagnosed diabetics of <1-year duration. Akturk *et al.*^[19] also reported a significant correlation between BMI and ATT but only among female diabetic population.

Severe damages to both locomotor structures and functions of diabetic patients come from the onset of PN, which is responsible for sensory deficiencies of the most distal parts of the body.^[31] In this study, the observed increased thickening in the AT and PF of diabetics was more evident among T2DM with PN. A significant difference was seen between the ATT of DM with PN and that of DM without PN ($P < 0.001$). This supports the fact that PN has effects on soft tissues of the feet. However, aside diabetics with PN, those without PN also had significantly thicker AT and PF than the controls ($P < 0.05$). This possibly suggests that soft-tissue changes, such as thickening of AT and PF, which through various biomechanical mechanisms predispose to plantar ulcers, are even present before the onset of PN just as opined by Giacomozzi *et al.*^[32] The findings on AT with respect to PN in this study corroborate the observations of D'Ambrogi *et al.*^[26] Furthermore, Abate *et al.*^[18] having excluded T2DM with PN, found a significant difference between the ATT of T2DM without PN and control participants just as in this study. However, a different perspective was shared by Akturk *et al.*^[19] who reported a significant difference between DM with and without PN only among male participants but provided no direct explanation for their finding. Our study had these limitations: first, we were unable to measure the cross-sectional areas of the AT and

PF due to scanner limitations. Second, majority of our study participants were retirees and their level of physical activities was not considered.

CONCLUSION

The ATT and PFT of diabetics are significantly thicker than those of age- and sex-matched controls in our environment, which is broadly similar to previous reports from elsewhere. Duration of diabetes did not correlate strongly with ATT and PFT. The presence of PN worsens the thickness of AT and PF among diabetics and is a significant predictor of ATT. BMI is significantly related to thickening of the AT and PF among apparently healthy controls but not in diabetics. FBG was found to have a significant positive correlation with ATT, but HBA1c did not. A future research will try to explore the clinical usefulness of these parameters (ATT and PFT) for predicting the development of diabetic foot ulcers.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ramchurn N, Mashamba C, Leitch E, Arutchelvam V, Narayanan K, Weaver J, *et al.* Upper limb musculoskeletal abnormalities and poor metabolic control in diabetes. *Eur J Intern Med* 2009;20:718-21.
2. International Working Group on the Diabetic Foot. International Diabetes Federation. Diabetes Fact Sheet. Available from: http://www.idf.org/webdata/docs/background_info_AFR.pdf. [Last accessed on 2015 Jun 24].
3. Adefemi K. What you Need to Know About Diabetes. Available from: <http://www.ezine-articles.com/?What-You-Need-To-Know-About-Diabetes&id=22656>. [Last accessed on 2015 Jun 24; Last updated on 2005 Mar 24].
4. Bakare B. Reducing the Prevalence of Diabetes. Available from: <http://www.pmnewsnigeria.com/2012/11/19/>. [Last accessed on 2015 Jun 24; Last updated on 2012 Nov 19].
5. Alvin CP. Diabetes Mellitus. In: Krasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. *Harrison's Principle of Internal Medicine*. 16th ed. NY: McGraw-Hill Comp Inc.; 2005. p. 2152-79.
6. Pendsey SP. Understanding diabetic foot. *Int J Diabetes Dev Ctries* 2010;30:75-9.
7. Bronze MS. Diabetic Foot Infection. Available from: <http://www.emedicine.medscape.com/article/237378>. [Last accessed on 2014 Jul 08; Last updated on 2014 May 30].
8. Batista F. The multidisciplinary approach of diabetic foot. *Andreoli* 2010;1:368.
9. Richardson EG. Diabetic Foot. *Orth Surg Campbell* 2006;10:4111-27.
10. Adler AI, Boyko EJ, Ahroni JH, Smith DG. Lower-extremity amputation in diabetes. The independent effects of peripheral vascular disease, sensory neuropathy, and foot ulcers. *Diabetes Care* 1999;22:1029-35.
11. Batista F, Magalhães AA, Nery C, Baumfeld D, Monteiro AC. Minimally invasive surgery for diabetic plantar foot ulcerations. *Diabet Foot Ankle* 2011;2:1-4.
12. Ogbera AO, Fasanmade O, Ohwovoriole AE. High costs, low awareness and a lack of care – The diabetic foot in Nigeria. *Diabetes Voice* 2006;51:30-2.
13. Chijioke A, Adamu AN, Makusidi AM. Mortality pattern among type 2 diabetes patients in Ilorin, Nigeria. *JEMDSA* 2010;15:1-4.
14. Pendsey S, Abbas ZG. The step-by-step program for reducing diabetic

- foot problems: A model for the developing world. *Curr Diab Rep* 2007;7:425-8.
15. Kwon OY, Minor SD, Maluf KS, Mueller MJ. Comparison of muscle activity during walking in subjects with and without diabetic neuropathy. *Gait Posture* 2003;18:105-13.
16. Sharkey NA, Donahue SW, Ferris L. Biomechanical consequences of plantar fascial release or rupture during gait. Part II: Alterations in forefoot loading. *Foot Ankle Int* 1999;20:86-96.
17. Grant WP, Sullivan R, Sonenshine DE, Adam M, Slusser JH, Carson KA, *et al.* Electron microscopic investigation of the effects of diabetes mellitus on the Achilles tendon. *J Foot Ankle Surg* 1997;36:272-8.
18. Abate M, Schiavone C, Di Carlo L, Salini V. Achilles tendon and plantar fascia in recently diagnosed type II diabetes: Role of body mass index. *Clin Rheumatol* 2012;31:1109-13.
19. Akturk M, Ozdemir A, Maral I, Yetkin I, Arslan M. Evaluation of achilles tendon thickening in type 2 diabetes mellitus. *Exp Clin Endocrinol Diabetes* 2007;115:92-6.
20. Batista F, Nery C, Pinzur M, Monteiro AC, de Souza EF, Felipe FH, *et al.* Achilles tendinopathy in diabetes mellitus. *Foot Ankle Int* 2008;29:498-501.
21. Ojewole LY, Adejumo PO. Type 2 diabetes mellitus and impaired fasting blood glucose in urban South Western Nigeria. *Int J Diabet Metab* 2012;21:9-12.
22. Lee S, Kim H, Choi S, Park Y, Kim Y, Cho B. Clinical usefulness of the two-site semmes-weinstein monofilament test for detecting diabetic peripheral neuropathy. *J Korean Med Sci* 2003;18:103-7.
23. Pang BS, Ying M. Sonographic measurement of achilles tendons in asymptomatic subjects: Variation with age, body height, and dominance of ankle. *J Ultrasound Med* 2006;25:1291-6.
24. De Mello RA, Marchiori E, Dos Santos AA, Neto GT. Morphometric evaluation of achilles tendon by ultrasound. *Radiol Bras* 2006;39:161-5.
25. Duffin AC, Lam A, Kidd R, Chan AK, Donaghue KC. Ultrasonography of plantar soft tissues thickness in young people with diabetes. *Diabet Med* 2002;19:1009-13.
26. D'Ambrogi E, Giacomozzi C, Macellari V, Uccioli L. Abnormal foot function in diabetic patients: The altered onset of Windlass mechanism. *Diabet Med* 2005;22:1713-19.
27. Chang KV, Wu WT, Huang KC, Jan WH, Han DS. Limb muscle quality and quantity in elderly adults with dynapenia but not sarcopenia: An ultrasound imaging study. *Exp Gerontol* 2018;108:54-61.
28. Chang KV, Wu WT, Han DS, Özçakar L. Static and dynamic shoulder imaging to predict initial effectiveness and recurrence after ultrasound-guided subacromial corticosteroid injections. *Arch Phys Med Rehabil* 2017;98:1984-94.
29. Abate M, Salini V, Antinolfi P, Schiavone C. Ultrasound morphology of the achilles in asymptomatic patients with and without diabetes. *Foot Ankle Int* 2014;35:44-9.
30. Min-Seob K, Se-Jung Y, Yun-Hyeong C. The correlation between achilles tendon thickness and cardiovascular risk factors. *J Lipid Atheroscler* 2013;2:77-83.
31. Boulton AJ. Clinical presentation and management of diabetic neuropathy and foot ulceration. *Diabet Med* 1991;8:S52-7.
32. Giacomozzi C, Caselli A, Macellari V, Giurato L, Lardieri L, Uccioli L, *et al.* Walking strategy in diabetic patients with peripheral neuropathy. *Diabetes Care* 2002;25:1451-7.