Original Article

Ultrasonographic Evaluation of Thickness and Stiffness of Achilles Tendon and Plantar Fascia in Type 2 Diabetics Patients: A Cross-sectional Observation Study

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Abstract

Background: Diabetes mellitus (DM) can contribute to the development of foot ulcers, a known complication of DM with a high financial and social burden. Achilles tendon (AT) and plantar fascia (PF) are well known to play an important role in foot biomechanics. The present study focuses on the alteration in thickness and stiffness of the AT and PF in Type 2 DM patients compared with the normal controls. **Methods:** A cross-sectional observational study was conducted with 55 DM patients and 55 healthy volunteers as controls. The thickness of the AT and PF were measured using B-mode ultrasound and stiffness was measured using shear wave elastography. Both the thickness and stiffness in the patient group and controls were compared. The values were also compared with the clinical and demographic profiles of the patients. **Results:** DM patients had considerably thicker AT and PF than controls ($P < 0.05$); mean values of AT thickness for DM patients and controls were 5.66 ± 0.54 mm and 4.61 ± 0.39 mm, respectively, and for PF were 2.53 ± 0.51 mm and 1.97 ± 0.19 mm, respectively. Furthermore, the stiffness of AT and PF was significantly ($P < 0.05$) lower in DM patients compared to controls, suggestive of softening of AT and PF in Type 2 DM patients. Mean values of shear wave velocity for DM patients and controls in AT were 5.53 ± 0.54 m/s and 7.25 ± 0.61 m/s, respectively, and for PF, 4.53 ± 0.89 m/s and 6.28 ± 0.88 m/s, respectively. **Conclusion:** We conclude that there is softening and thickening of the AT and PF in Type 2 DM patients, which can impair foot biomechanics.

Keywords: Achilles tendon, diabetes mellitus, plantar fasciitis, sonoelastography

Introduction

India is at the epicenter of diabetes mellitus (DM) globally, an endocrine illness with extensive social and economic burden.[1] Due to the high prevalence of microangiopathic and macroangiopathic consequences, including the dreaded diabetic foot with a significant social and financial burden, Type 2 DM is a significant cause of decrepitude. Diabetic foot and plantar ulcers are characterized by a triad of neuropathy, infection, and ischemia.[2]

Regarding foot biomechanics, Achilles tendon (AT) and plantar fascia (PF) are crucial components. They work in tandem with the metatarsophalangeal joint to lock the mid-tarsal bones and stabilize the arch during propulsion (PF operates like a beam) and to absorb shock and prevent the longitudinal arch of the foot from collapsing during landing (PF acts like a truss). Hicks referred to this stabilizing mechanism as the "Windlass mechanism." It should occur in a healthy control at the start of the heel rise, when the AT helps to produce talus supination and tighten the PF. In order to effectively accomplish the propulsion, the longitudinal arch must remain high and rigid, which is maintained by the plantar ligament being further tightened by deflection at the metatarsophalangeal joints.[3,4]

Patients with DM are prone to plantar fasciopathy, a condition marked by the thickening of PF and loss of normally organized

Received: 31-10-2022 **Revised:** 25-11-2022 **Accepted:** 13-12-2022 **Available Online:** 13-02-2023

Access this article online

Quick Response Code:

Website: https://journals.lww.com/jmut

DOI: 10.4103/jmu.jmu_109_22

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How to cite this article: Saroha A, Saran S, Saxena S, Kant R, Bhadoria AS. Ultrasonographic evaluation of thickness and stiffness of achilles tendon and plantar fascia in type 2 diabetics patients: A cross-sectional observation study. J Med Ultrasound 2023;31:282-6.

architecture of PF. PF is one of the tissues that may modify its physiology and biomechanical function in the presence of chronic hyperglycemia.[5] The pathophysiology leading to AT and PF affection in DM may be summed up as following: sustained hyperglycemia encouraging higher glycosylation of proteins resulting in the accumulation of "advanced glycosylation end products" in the patient's soft tissue with the thickening and increased vascularization of AT and PF. In addition to neuropathy, vasculopathy, and metabolic problems the increased thickness of AT and PF in DM have been thought to be signs of soft-tissue injury which may play a part in the onset of foot ulcer in DM patients.[6,7]

Elasticity is a tissue's ability to resist altering in size or shape in response to an external stimulus. Changes in the thickness and echo pattern of tendons are some of the indirect factors that indicate an abnormality in the elastic properties of the tendons, while sonoelastography, an ultrasound-based technology, directly assesses the stiffness of numerous tissues, including tendons. Elastography has seen a significant surge in use recently for musculoskeletal system evaluation.[8,9] This method evaluates the tissue's response to mechanical stimulation. The two main categories of elastographic techniques are shear wave imaging and strain imaging. In strain imaging, displacement of the tissue perpendicular to the direction of compression is recorded after compression is applied.[10,11] Manual compression or tissue displacement brought on by physiological processes such as cardiac and respiratory movements are utilized. One of the more contemporary elastographic techniques, shear wave elasticity imaging (SWEI), makes use of an acoustic radiation force pulse, which is a high intensity, brief duration sonic "pushing pulse" that moves the tissue and creates shear waves that go through the tissues perpendicular to the compressive force. These waves are then traced using tracking pulses, and the velocity in the region of interest (ROI) is determined. The elasticity of the tissue is reflected in the shear wave velocity (SWV) calculation. With this method, no external compression is required.[12-14]

The primary objective of this study was to compare the thickness and stiffness of AT and PF in DM patients with healthy volunteers. The secondary objective of the study was to correlate thickness and stiffness of AT and PF with the duration of diabetes, age of the patients, and hemoglobin A1c (HbA1c) levels.

Materials and Methods

This cross-sectional observational study was approved by the institutional ethics committee (126/IEC/PGM/2021) and was conducted in the department of radiology and internal medicine of our institute from January 2021 to August 2022. We recruited 55 patients with Type 2 DM and 55 healthy controls. DM Patients and healthy volunteers with a history of foot trauma, foot surgery, plantar fasciitis and Achilles tendinitis from other causes, local or systemic steroid treatments, renal failure, congenital ankle deformities, dyslipidemia, rheumatological disorders (e.g., rheumatoid arthritis and psoriatic arthritis), or acromegaly were excluded from the study. The fasting blood sugar level for the healthy controls was <100 mg/dl.

All participants provided their informed consent. Clinical information of the patients that were recorded included age, sex, the occurrence of foot ulcers, comorbidities, and sequelae from DM. HbA1c and random blood sugar values were evaluated to assign them to the patient group. A single radiologist with at least 5 years of expertise in musculoskeletal ultrasound and elastography and blinded to the subject's status conducted each examination. All ultrasound examinations were performed on Esaote Mylab 9 EXP machine with a high-frequency linear array transducer (7–14 MHz). AT thickness and PF thickness were measured with the patient lying prone on the examination table with their feet hanging freely in a neutral posture at the edge of the table. A greater amount of ultrasonic coupling gel was applied to avoid any mechanical compression with the probe since both AT and PF are superficial structures. The PF was evaluated on the medial side of the foot, whereas the AT was evaluated in its middle section (6 cm from the point of its insertion). Transverse scans were used to measure the anteroposterior diameters. Measurements were expressed in millimeters [Figures 1 and 2].

The transducer with a generous amount of coupling gel was gently placed on the PF and AT without applying any manual pressure to assess the SWEI of the PF and AT. The probe was positioned longitudinally to examine the tendon and fascia in the sagittal plane. The ROI, a rectangle, was set on the PF and AT once the virtual touch quantification mode was activated. The size of the ROI was adjusted to carefully avoid the inclusion of the nearby structures. SWEI creates a displacement by applying an acoustic radiation force pulse, which causes shear waves to move in the perpendicular direction. These shear waves are tracked by tracking pulses, and the SWV is measured and expressed in meters/second (m/s). SWV is more in stiffer tissues. At least four such values were taken for PF and AT, and mean SWV value was calculated by averaging these values.

Statistical analysis

A Microsoft Excel spreadsheet was used to enter the data. In categorical variables, frequency and percentage were used as descriptors. Mean ± standard deviation was used for continuous variables. The Mann–Whitney *U*-test and Student's *t*-test were used to compare the means of the two groups. Spearman Correlation was used to study the correlation of thickness and stiffness of AT and PF with other variables.

Results

The study comprised 55 adult Type 2 DM patients, of which 29 (52.7%) were male and 26 (47.3%) were female. The youngest DM patient was 30 years old, whereas the oldest patient was 85 years old. The study also included 55 healthy nondiabetic volunteers, of which 25 (45.5%) were female and 30 (54.5%) were male. In our study, the youngest healthy

volunteer was 30 years old, and the oldest was 80. In the patient group, the mean duration of DM was 12.45 years. The HbA1c values ranged from 6.6% to 15.2%, with a mean of 9%.

On comparing the mean AT thickness of the DM patients and healthy volunteers, the mean AT thickness was found to be higher in the patients (5.66 ± 0.54 mm) than in the healthy volunteers (4.61 ± 0.39 mm) [Table 1], and the difference was statistically significant ($P < 0.001$). On comparing the mean PF thickness of the DM patients and healthy volunteers, the mean PF thickness was also found to be higher in patients (2.53 ± 0.51 mm) than the healthy volunteers (1.97 ± 0.19 mm) [Table 1], and the difference was statistically significant ($P < 0.001$). We also correlated AT thickness and PF thickness with the duration of diabetes, age of the patients, and HbA1c levels, but we could not find any significant association.

SWEI of the AT was performed in all 55 DM patients and 55 healthy volunteers. In the patient group, the mean SWV value was 5.53 ± 0.56 m/s, and in healthy volunteers, the mean SWV value was 7.25 ± 0.61 m/s. While the mean SWV of PF was 4.53 ± 0.89 m/s in DM patients and 6.28 ± 0.88 m/s in healthy volunteers. Hence for both PF and AT, the DM patients had considerably lower mean SWV values than healthy volunteers ($P < 0.001$) [Table 2]. The lower SWV values imply AT and PF softening in the diabetic group compared to the healthy volunteers [Figures 3 and 4]. On correlating AT and PF stiffness with the duration of diabetes, age of the patient, and HbA1c levels, no significant correlation was found.

Discussion

In this study, we observed softening and thickening of the AT and PF in DM patients as compared to the healthy volunteers with statistically significant difference. However, on correlating thickness and stiffness of AT and PF in DM patients with age, duration of diabetes, and HbA1c levels, no statistically significant correlation was found.

Water retention caused by the accumulating of hydrophilic proteoglycans and/or inflammation may also play a role in tendon and fascia getting thickened and softer in DM.[7,15] The AT in DM patients was studied using electron microscopy, and it revealed that the width of each collagen fibril had decreased, coupled with an increase in packing density and morphologic changes as compared to controls. The nonenzymatic glycation of proteins, which enables intermolecular cross-linking, may cause these morphologic changes in the tendons.[6] These modifications could impact the morphology and elasticity of

Table 1: Comparison of Achilles tendon and plantar fascia thickness between study groups

Subjects (mm)	Mean±SD		*P*
	Healthy volunteers	DM patients	
AT	4.61±0.39	5.66±0.54	<0.001
PF	1.97±0.19	2.53±0.51	<0.001

DM: Diabetes mellitus, AT: Achilles tendon, PF: Plantar fascia, SD: Standard deviation

Table 2: Comparison of Achilles tendon and plantar fascia stiffness shear wave velocity between study groups

Subjects (m/s)	Mean±SD		*P*
	Healthy volunteers	DM patients	
AT	7.25±0.61	5.53±0.56	<0.001
PF	6.28±0.88	4.53±0.89	<0.001

DM: Diabetes mellitus, AT: Achilles tendon, PF: Plantar fascia, SD: Standard deviation

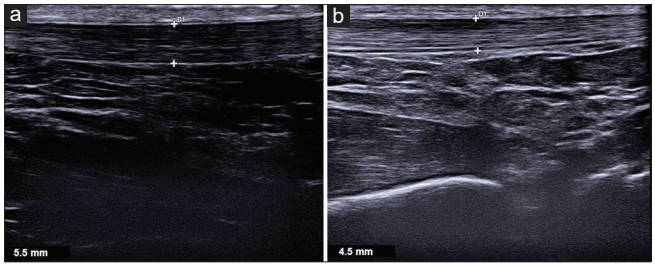

Figure 1: Sonographic images showing AT thickness in a DM patient (a) and a healthy volunteer (b). AT: Achilles tendon, DM: Diabetes mellitus

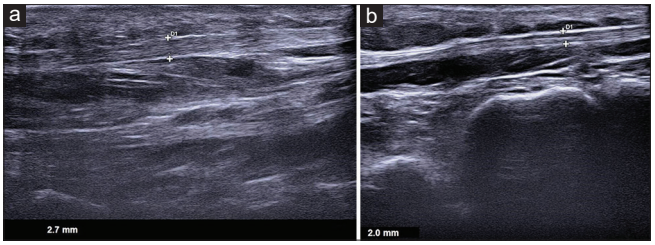

Figure 2: Sonographic images showing PF thickness in a DM patient (a) and a healthy volunteer (b). PF: Plantar fascia, DM: Diabetes mellitus

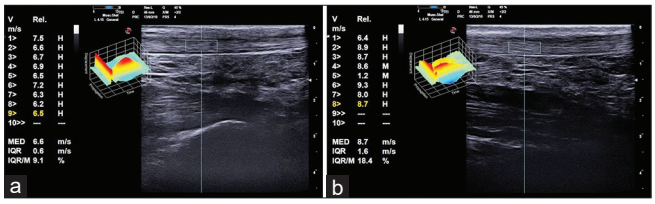

Figure 3: SWEI of AT in a DM patient (a) and a healthy volunteer (b) Showing shear wave velocities. SWEI: Shear wave elasticity imaging, AT: Achilles tendon, DM: Diabetes mellitus

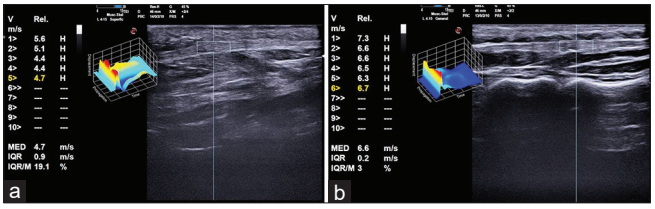

Figure 4: SWEI of PF in a DM patient (a) and a healthy volunteer (b) showing shear Wave velocities. SWEI: Shear wave elasticity imaging, PF: Plantar fascia, DM: Diabetes mellitus

different tissues and ligaments. Given that the PF is crucial in maintaining the foot's longitudinal arch during propulsion, it is possible to hypothesize that when the PF is abnormally thickened, foot assumes a rigid position and advances toward the ground as a "functional flat foot." This landing technique might subject AT to less stress.[4] PF needs enough flexibility to manage the tensile strain brought on by the synergistic action of AT and metatarsophalangeal joints to function properly. It has been shown that, under physiological settings, the PF elongates more quickly during the weight acceptance and mid-stance phases than it does during the toe-off and push-off phases. This behavior was related to the PF's elastic and collagen fibers histology composition. They can help with the early extension of fascia since they have a lower modulus of elasticity than collagen.[16] The foot's anatomical abnormalities brought on by diabetes, particularly in the AT and PF, impact how the foot loads during propulsion. Changes in the manner DM patients walk cause aberrant cumulative stress, which may cause the tendons to thicken as a protective mechanism.[4,17]

Compared to the healthy volunteers, DM patients' AT and PF were substantially thicker in the current study, with mean thicknesses of 5.66 ± 0.54 mm and 2.53 ± 0.51 mm, respectively. Numerous research examining the impact of diabetes on AT and PF have produced similar findings.[4,18-20] As the disease's duration lengthened, it would be expected that the soft-tissue changes caused by diabetes may become more apparent, but neither the AT thickness nor the PF thickness showed a significant correlation with the length of diabetes in our study.

The current study assessed the AT and PF using point shear wave elastography. We found significantly lower SWV values of AT and PF in DM patients compared with healthy volunteers, measuring 5.53 ± 0.56 m/s and 4.53 ± 0.89 m/s, respectively ($P < 0.001$). Only a few published studies have discussed the elastographic characteristics of AT and PF in DM patients, even though AT and PF thickening has been extensively studied. Evranos *et al*.,[19] who were the first to test AT by strain elastography in DM patients, found that the stiffness of AT was considerably reduced in patients with foot ulcers compared to controls.

Numerous studies have demonstrated that Achilles tendinopathy causes the tendon to soften.[13,21,22] A study found that DM patients with chronic tendinopathy have reduced neoangiogenesis, which may be one of the pathogenic causes of Achilles tendinopathy.[3] Another potential cause for tendon thickening is pathologic tendon thickening as compensation for structurally disorganized areas.[23] It has been proposed in a study by Wang *et al*. in 2000[24] that with thicker constructions, stress and hence the elastic modulus are reduced when the strain is constant. This can be one of the plausible explanations for lower SWV values observed in the AT of DM patients, as the thickened tendon results in reduced stress and an increase of cross-sectional area.

Previous studies have evaluated the changes in elasticity in patients with plantar fasciitis; however, there is a paucity of literature regarding the sonoelastographic characteristics of PF in diabetic patients.[25-27] Harish *et al*.[28,29] evaluated AT and PF with the help of sonoelastography in diabetic and healthy population in the Indian setting and found that patients with diabetes had thicker and softer AT and PF than the healthy population which matched our results.

Few of the limitations of our study were the exclusion of participants with foot ulcers and lack of follow-up to see effect of duration more objectively. In addition, because we did not distinguish between DM patients with and without peripheral neuropathy, we were unable to determine how peripheral neuropathy affected AT and PF.

Conclusion

The present study findings support the idea that DM alters AT and PF. The thickening and softening of AT and PF could change how the foot functions and perhaps contribute to the emergence of foot ulcers. For diabetic patients, B-mode ultrasound and shear wave ultrasound elastography may be helpful for identifying patients at risk of developing impaired foot biomechanics. More research involving a large number of patients is required to confirm this association.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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