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Achilles tendon degeneration on ultrasound in type 2 diabetic patients

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Keywords
Achilles tendon, Achilles tendinopathy, tendon degeneration, tendinopathy, diabetes mellitus

Abstract

Aim of study: The main goal of this study was to compare the various degenerative changes in the Achilles tendon of type 2 diabetic patients to that of controls. The influence of diabetic peripheral neuropathy, duration of diabetes mellitus, age, and body mass index on the occurrence of degenerative changes was also evaluated. **Materials and methods:** The Achilles tendons of both limbs were evaluated with high-resolution ultrasound in 80 type 2 diabetics and 80 age/sex-matched controls. A 10 g Semmes Weinstein monofilament was used to examine for peripheral neuropathy. Anthropometric measurements and biochemical assessment of glycemic control (fasting plasma glucose and glycated hemoglobin) were also done. **Results:** The mean age of type 2 diabetic subjects and healthy controls was 60.9 ± 10.3 years (range 41–79 years) and 61.0 ± 10.3 years (range 40–79 years), respectively ($p = 0.963$). The median duration of diabetes mellitus was 42.0 months (range = 1–456 months). The prevalence of degenerative changes (calcifications, disorganized fibers and/or hypoechoic foci) was significantly higher in type 2 diabetic subjects than controls in both the right (55.0% *vs.* 18.8%, $p < 0.001$) and left (52.5% *vs.* 18.8%, $p < 0.001$) feet. **Conclusion:** The Achilles tendons of type 2 diabetic subjects have significantly more degenerative changes than their age/ sex-matched controls in our locality. Disorganized Achilles tendon fibers occur significantly more often among male than female type 2 diabetic subjects. Disorganization of Achilles tendon fibers and hypoechoic foci are significantly more prevalent in type 2 diabetic subjects with peripheral neuropathy than those without peripheral neuropathy. Body mass index did not affect the occurrence of degenerative changes in the Achilles tendon of participants.

Introduction

The Achilles tendon (AT) is the largest, toughest, and strongest tendon in the human body, formed when the aponeurosis of the soleus and gastrocnemius muscles combine[(1)]. It extends from the myotendinous junction distally to its insertion site on the upper half of the posterior surface of the calcaneus. It is approximately 15 cm long and 6 mm thick[(1)]. Tendinopathy is one of the musculoskeletal manifestations of diabetes mellitus. Diabetes mellitus (DM) causes pathological changes in the Achilles tendon, which could lead to structural or functional dysfunction or precipitate diabetic foot syndrome[(2)]. Diabetic tendon damage is a complex, multifactorial process characterized by excessive accumulation of advanced glycation end products (AGEs), inflammatory response dysfunction, neurovascularization, peripheral neuropathy, and vasculopathy[(2)].

In keeping with the application of musculoskeletal ultrasonography to diverse clinical situations[(3,4)], the AT has also been assessed in DM patients using ultrasound and magnetic resonance imaging, and the findings included increased AT thickness and structural abnormalities[(5–9)].

Aside from the thickening of the AT[10], other features, like abnormal morphology (twisted, curved, overlapping, and highly disorganized fibers), and calcific degeneration have been identified as degenerative changes affecting the AT. These alterations may be present in the initial stages of type 2 diabetes (T2DM)[11]. Since T2DM diagnosis is often made when some patients already have evidence of chronic complications, it implies that the effects of hyperglycemia would have been exerted for a while on tendon structures[11]. Altered AT fibrillary pattern, the presence of hypoechoic areas, and calcific foci within the tendon are common degenerative abnormalities[12].

While the stiffness of the Achilles tendon is not directly measured on B-mode ultrasonography, changes such as thickening and echotexture alterations (which have been shown in kinematics studies to alter the foot loading pattern and pose a considerable risk for plantar ulcer)[13,14] can be readily assessed on ultrasound.

This study aims to determine and analyze the prevalence, pattern, and modifying factors (age, body mass index, coexisting peripheral neuropathy, diabetes duration) of diabetic degenerative Achilles tendinopathy in patients with type 2 diabetes mellitus in our locality.

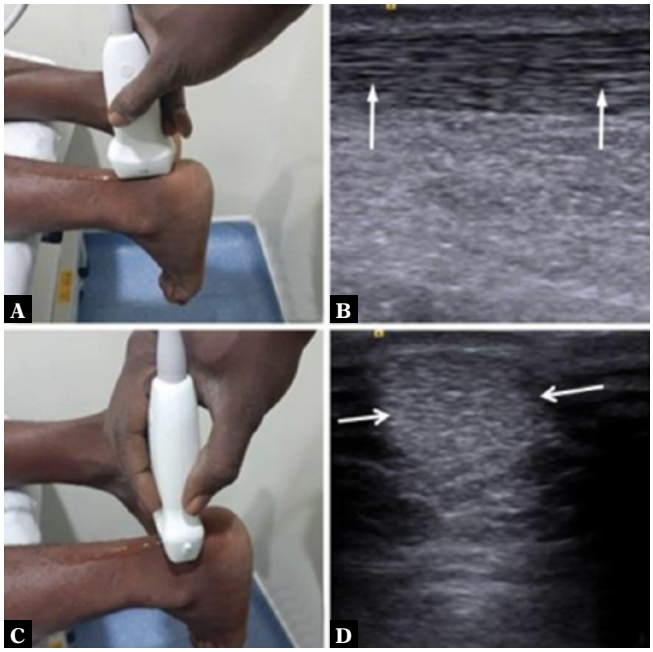

Fig. 1. *Ultrasonography of a normal Achilles tendon (arrows) showing the transducer position and equivalent sonographic image in longitudinal/long-axis view (***A, B***) and transverse/short-axis view (***C, D***)*

Materials and methods

Eighty (80) subjects with type 2 diabetes mellitus (T2DM) and 80 age/sex-matched controls were enrolled in this descriptive cross-sectional study. The ethics committee of the hospital approved the study protocol. All recruited participants gave written, informed consent.

All participants were ≥40 years old. The subjects were confirmed diabetic patients attending the endocrinology clinic of the hospital, while the controls were healthy volunteers with fasting blood glucose (FBG) of <6.1 mmol/L. Exclusion criteria were renal failure, dyslipidemia, history of peripheral vascular disease, smoking, Charcot neuroarthropathy, congenital ankle deformities, chronic heel pain, lower limb amputation, chronic steroid use, neurological disorders (besides those associated with DM), musculoskeletal diseases, rheumatoid disease, and bodybuilding[10].

The biodata, relevant clinical history, diabetes duration, and history of previous foot ulcers were documented. Weight, height, and body mass index (BMI) of the participants were recorded. A 10 g Semmes-Weinstein monofilament was used to assess for peripheral neuropathy (PN)[10]. Fasting blood glucose (FBG; for the entire study population) and glycated hemoglobin (HbA_{1c}; for diabetic subjects only) levels were determined using established standard methods[10]. Fasting blood glucose (FBG) was categorized as <5.6 mmol/L, 5.6–6.9 mmol/L, and ≥7.0 mmol/L to represent good FBG, impaired FBG, and poor FBG control, respectively. A MINDRAY® model DC-7 ultrasound scanner (Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China) with a 7.5–12.0 MHz high-frequency linear array transducer was used for Achilles tendon sonography. The first author performed all ultrasound scans to reduce interobserver variability.

After adequate exposure of the leg by rolling up clothing up to the knees, Achilles tendon (AT) sonography was performed with the participant lying prone on the examination couch, with their feet projecting beyond the edge of the couch and pointing downward, and the ankle in the neutral position at an angle of 90 degrees (Fig. 1). The Achilles tendons of both legs were scanned in longitudinal and transverse planes from the myotendinous junctions to their insertion site on the calcaneus bones. The presence or absence of disorganization of the tendon fibers, hypoechoic foci, and calcifications was documented.

The study data were analyzed using the IBM SPSS Statistics for Windows version 20 (IBM Corp., Armonk, N.Y., USA). The normality of data was determined using the Kolmogorov-Smirnov test. Descriptive and inferential analyses were applied as appropriate. Chi-square test was used to detect the association between the categorical variables, while the independent samples t-test was used to compare the mean values of continuous quantitative variables. A $p \leq 0.05$ was considered statistically significant.

Results

There were 80 type 2 diabetics (30 males and 50 females) and 80 non-diabetic controls (34 males and 46 females) matched for age and sex. The mean age of T2DM subjects and healthy controls was 60.9 ± 10.3 years (range

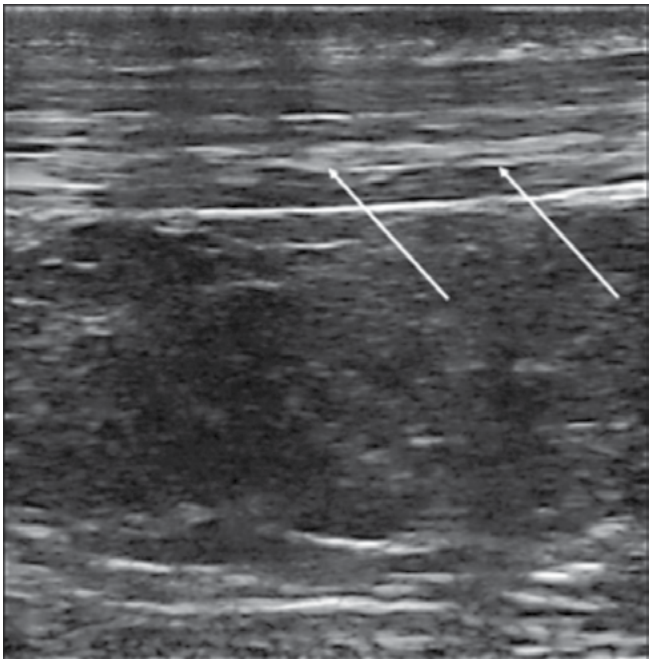

Fig. 2. *B-mode ultrasound image of the Achilles tendon (longitudinal view) showing disorganization of the Achilles tendon fibers (arrows)*

Tab. 1. *Comparison of the degenerative changes in the AT of T2DM and controls*

Variables	Study group, *n* (%)		χ2	df	*p*-value
	Diabetics (*n* = 80)	Controls (*n* = 80)			
Right AT calcifications					
Present	4 (5.0)	0 (0.0)		0.120*	
Absent	76 (95.0)	80 (100.0)			
Left AT calcifications					
Present	5 (6.3)	2 (2.5)		0.443*	
Absent	75 (93.8)	78 (97.5)			
Right AT disorganized fibres					
Present	43 (53.8)	14 (17.5)	22.919	1	**<0.001**
Absent	37 (46.3)	66 (82.5)			
Left AT disorganized fibres					
Present	41 (51.3)	12 (15.0)	23.728	1	**<0.001**
Absent	39 (48.8)	68 (85.0)			
Right AT hypoechoic foci					
Present	16 (20.0)	4 (5.0)	8.229	1	**0.004**
Absent	64 (80.0)	76 (95.0)			
Left AT hypoechoic foci					
Present	15 (18.8)	6 (7.5)	4.440	1	**0.035**
Absent	65 (81.2)	74 (92.5)			
Right AT calcification, disorganized fibres or hypoechoic foci					
Present	44 (55.0)	15 (18.8)	22.581	1	**<0.001**
Absent	36 (45.0)	65 (81.2)			
Left AT calcification, disorganized fibres or hypoechoic foci					
Present	42 (52.5)	15 (18.8)	19.867	1	**<0.001**
Absent	38 (47.5)	65 (81.2)			

** Fisher's exact test * df – degree of freedom; AT – Achilles tendon; T2DM – type 2 diabetes mellitus*

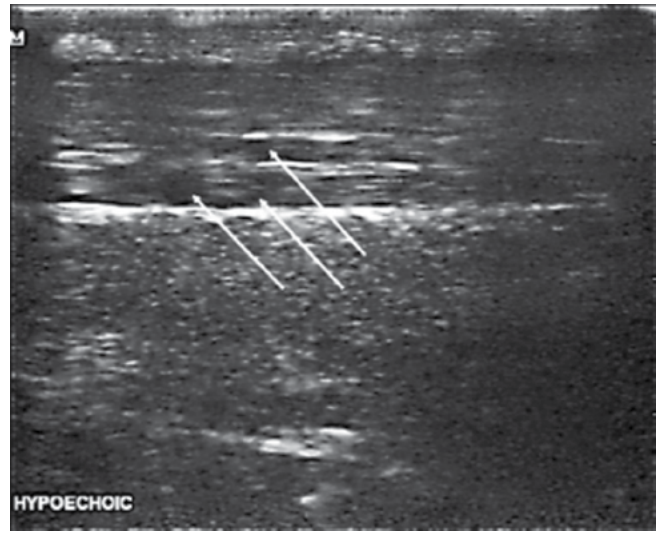

Fig. 3. *B-mode ultrasound image of the Achilles tendon (longitudinal view) showing hypoechoic foci (arrows) within the tendon fibers (arrows)*

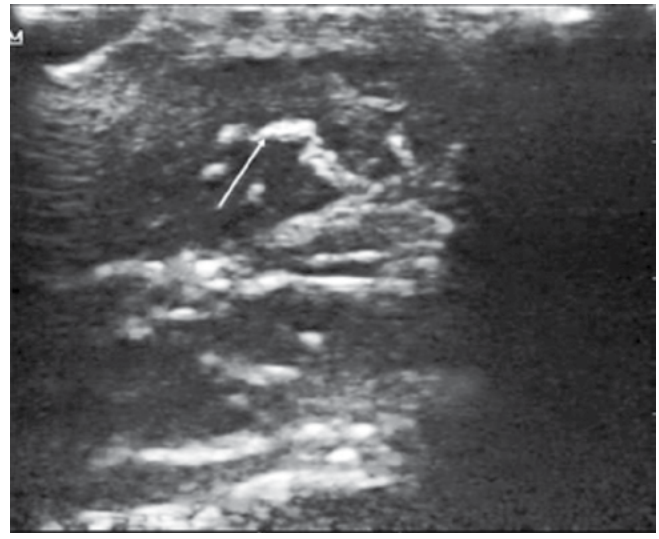

Fig. 4. *B-mode ultrasound image of the Achilles tendon (transverse view) showing the presence of calcific focus (arrow) within the Achilles tendon fibers*

41–79 years) and 61.0 ± 10.3 years (range 40–79 years), respectively (p = 0.963).

There was no significant difference between the mean BMI of the subjects (70.0 ± 12.1 kg/m²) and controls (67.0 ± 10.6 kg/m²); p = 0.141. The median duration of diabetes mellitus was 42.0 months (range = 1–456 months). Regarding peripheral neuropathy (PN), 56/80 (70%) of T2DM subjects had right foot PN, while 24/80 (30%) did not have right foot PN. Also, 50/80 (62.5%) had left foot PN, while 37.5% did not have PN in the left foot.

Most (48.8%) of the diabetic subjects had poor FBG control, 31.2% had impaired FBG level, while 20.0% had good FBG control. Glycated hemoglobin (HbA_{1c}) level was categorized as good (<7.0%) and poor (≥7.0%) glycemic

Tab. 2. *Association between the degenerative changes and characteristics of T2DM subjects*

Variables	Degeneration		t	*p*-value
	Present	Absent		
AT disorganized fibers				
Age (*mean ± SD*) **(years)**	63.8 ± 8.7	56.6 ± 11.0	3.132	0.003
BMI (*mean ± SD*) **(kg/m²)**	26.44 ± 4.36	25.14 ± 4.67	1.272	0.207
FBG (*mean ± SD*) **(mmol/L)**	8.7 ± 5.4	7.4 ± 2.4	1.409	0.163
HbA_{1c} (*mean ± SD*) **(%)**	8.7 ± 2.8	8.2 ± 2.4	0.853	0.396
DM duration *Median (IQR)* **months**	67.0 (16.0–116.8)	24.0 (6.0–61.8)	–2.294	0.022**
AT hypoechoic foci				
Age (*mean ± SD*) **(years)**	65.9 ± 7.2	59.7 ± 10.6	2.773	0.009
BMI (*mean ± SD*) **(kg/m²)**	25.86 ± 4.72	25.94 ± 4.49	–0.068	0.946
FBG (*mean ± SD*) **(mmol/L)**	9.7 ± 7.3	7.8 ± 3.4	1.004	0.330
HbA_{1c} (*mean ± SD*) **(%)**	9.5 ± 3.0	8.3 ± 2.5	1.757	0.083
DM duration *Median (IQR)* **months**	64.5 (28.5–103.0)	37.5 (8.8–96.8)	–0.848	0.396**
AT calcifications				
Age (*mean ± SD*) **(years)**	65.5 ± 7.2	60.6 ± 10.5	1.134	0.260
BMI (*mean ± SD*) **(kg/m²)**	27.87 ± 5.52	25.77 ± 4.42	1.100	0.275
FBG (*mean ± SD*) **(mmol/L)**	7.9 ± 3.0	8.2 ± 4.6	–0.144	0.886
HbA_{1c} (*mean ± SD*) **(%)**	9.3 ± 3.8	8.5 ± 2.5	0.508	0.632
DM duration *Median (IQR)* **months**	65.0 (21.5–206.3)	38.5 (7.5–96.3)	–1.042	0.298**
AT degenerative changes*				
Age (*mean ± SD*) **(years)**	63.8 ± 8.7	56.5 ± 11.2	3.094	0.003
BMI (*mean ± SD*) **(kg/m²)**	26.41 ± 4.32	25.15 ± 4.74	1.219	0.227
FBG (*mean ± SD*) **(mmol/L)**	8.8 ± 5.4	7.3 ± 2.2	1.740	0.086
HbA_{1c} (*mean ± SD*) **(%)**	8.8 ± 2.8	8.0 ± 2.2	1.395	0.167
DM duration *Median (IQR)* **months**	66.0 (17.0–116.5)	23.0 (6.0–62.0)	–2.218	0.027**

** Any of AT calcification, disorganized fibers or hypoechoic foci; ** Mann-Whitney U test; T2DM – type 2 diabetes mellitus; AT – Achilles tendon*

control. Most T2DM subjects (51; 63.8%) had HbA_{1c} level ≥7.0% while 29 (36.2%) had HbA_{1c} <7.0%.

Degenerative changes in AT in T2DM patients vs controls

More T2DM subjects had right AT disorganized fibers (Fig. 2), which was 43/80 (53.8%) compared with 14 (17.5%) controls (p <0.001) (Tab. 1). A significantly greater number of T2DM subjects had disorganized AT fibers in the left foot than controls (51.3% *vs*. 15.0%, p <0.001) (Tab. 1).

AT hypoechoic foci (Fig. 3) in both feet were more common in T2DM patients than controls. Twenty percent of T2DM subjects had hypoechoic foci in their right AT, while only 5% of controls had this feature (p = 0.004). Also, in the left AT, 18.8% of T2DM subjects had hypoechoic foci compared with 7.5% of control subjects (p = 0.035) (Tab. 1). The difference in the occurrence of calcific foci (Fig. 4) in the AT of both feet in T2DM subjects and those of control subjects was not statistically significant (both p >0.05) (Tab. 1).

The presence of either of calcific foci, disorganized fibers, and/or hypoechoic foci in the right AT was compared between T2DM subjects and controls; the prevalence was significantly higher among T2DM subjects than controls (55.0% vs. 18.8%, p <0.001). Similarly, in the left AT, 52.5% of T2DM subjects had one or more of degenerative features compared with 18.8% of controls (p <0.001) (Tab. 1).

Disorganized Achilles tendon fibers

The mean age of those with disorganized AT fibers was significantly higher than the mean age of those without disorganized fibers (p = 0.003) (Tab. 2). Disorganized AT fibers were most prevalent among subjects in their 70s, and least prevalent among those in their 40s (Tab. 3). Based on gender, 24/30 (80%) males had disorganized AT fibers while 24/50 (48.0%) females had features of Achilles tendon disorganization (p = 0.005) (Tab. 3). The prevalence among different BMI groups did not differ significantly (p >0.05), although a higher prevalence was seen among the obese, which was 11/17 (64.7%) compared with 19/37 (51.4%) in normal BMI group (Tab. 3).

The prevalence rates of disorganized AT fibers among T2DM subjects with time interval since diagnosis of <5 years, 5–10 years, and >10 years were 46.5%, 70.8%, 84.6%, respectively (p = 0.021) (Tab. 3). A higher proportion of T2DM subjects with PN had disorganized fibers (41/57, 71.9%) compared with the prevalence among T2DM subjects without PN (7/21, 30.4%) (p = 0.001) (Tab. 3).

The mean FBG level was higher among those with disorganized AT fibers at 8.7 ± 5.4 mmol/L compared with those without disorganized AT fibers at 7.4 ± 2.4 mmol/L (p =

Tab. 3. *Comparison of disorganized fibers of AT with characteristics of diabetics*

Variables	Diabetics' AT disorganized fibres			χ2	df	*p*-value
	Present	Absent	Total			
Age (years)						
40–49	3 (20.0)	12 (80.0)	16 (100)	12.315	3	**0.006**
50–59	11 (68.8)	5 (31.3)	16 (100)			
60–69	20 (69.0)	9 (31.0)	29 (100)			
70–79	14 (70.0)	6 (30.0)	20 (100)			
Gender						
Male	24 (80.0)	6 (20.0)	30 (100)	8.000	1	**0.005**
Female	24 (48.0)	26 (52.0)	50 (100)			
BMI (kg/m²)						
Normal	19 (51.4)	18 (48.6)	37 (100)	2.233	2	0.327
Overweight	18 (69.2)	8 (30.8)	26 (100)			
Obese	11 (64.7)	6 (35.3)	17 (100)			
DM Duration (months)						
<5 years	20 (46.5)	23 (53.5)	43 (100)	7.715	2	**0.021**
5–10 years	17 (70.8)	7 (29.2)	24 (100)			
>10 years	11 (84.6)	2 (15.4)	13 (100)			
Any peripheral neuropathy						
Present	41 (71.9)	16 (28.1)	57 (100)	11.757	1	**0.001**
Absent	7 (30.4)	16 (69.6)	23 (100)			
FBG						
<5.6 (mmol/L)	10 (62.5)	6 (37.5)	16 (100)	0.059	2	0.971
5.6–6.9 (mmol/L)	15 (60.0)	10 (40.0)	25 (100)			
≥7.0 (mmol/L)	23 (59.0)	16 (41.0)	39 (100)			
HbA_{1c}						
Poor (HbA_{1c} ≥7.0%)	32 (62.7)	19 (37.3)	51 (100)	0.442	1	0.506
Good (HbA_{1c} <7.0%)	16 (55.2)	13 (44.8)	29 (100)			

FBG – fasting blood glucose; HbA_{1c} – glycated hemoglobin; BMI – body mass index; AT – Achilles tendon

0.163) (Tab. 2). Similarly, the prevalence did not differ significantly among various FBG levels, i.e. good, impaired, and poor FBG ($p = 0.971$) (Tab. 4). Also, the prevalence of disorganized AT fibers did not differ significantly among those with HbA_{1c} ≥7.0% and HbA_{1c} <7.0% ($p = 0.506$) (Tab. 3).

Achilles tendon hypoechoic foci

The mean age of those with and without hypoechoic foci in either AT was 65.9 ± 7.2 years and 59.7 ± 10.9 years, respectively ($p = 0.009$) (Tab. 2). The prevalence of hypoechoic foci increased with increasing age from 0% among 40–49 years age group to 9/29 (31.0%) in those aged 60–69 years. Those aged 70–79 years had a prevalence of 5/20 (25.0%) ($p = 0.060$) (Tab. 4). There was no significant difference in the prevalence of AT hypoechoic foci between male and female T2DM subjects. Likewise, the rate of occurrence of AT hypoechoic foci among different BMI groups did not differ significantly (Tab. 4).

There was no statistically significant difference in the prevalence of tendoachilles hypoechoic foci between T2DM subjects with DM duration of <5 years (6/43; 14%), 5–10 years (7/24; 29.2%) and >10 years (3/13; 23.1%) ($p = 0.314$) (Tab. 4). In contrast, the rate of hypoechoic foci was significantly higher among T2DM subjects with PN (15/42, 26.3%) compared to those without PN (1/23, 4.3%) ($p = 0.031$) (Tab. 4).

The mean FBG was higher among those with AT hypoechoic foci (9.7 ± 7.3 mmol/L) than those without (7.8 ± 3.4 mmol/L) ($p = 0.330$) (Tab. 2). Despite this, the prevalence of hypoechoic foci did not differ significantly among those with good, impaired, and poor FBG levels ($p = 0.776$) (Tab. 4). A similar pattern was observed with glycated hemoglobin (Tab. 4).

Calcific foci in the Achilles tendon

There was no significant difference in the prevalence of AT calcifications among T2DM subjects based on age, gender, and body mass index ($p > 0.05$) (Tab. 5). The prevalence of AT calcifications was 4.7%, 8.3%, and 15.4% in subjects with DM duration of <5 years, 5–10 years, and >10 years, respectively. However, the observed differences in the prevalence were not statistically significant ($p = 0.295$) (Tab. 5). Among those with peripheral neuropathy, 5/57 (8.8%) subjects had AT calcifications compared to only 1/23 (4.3%) of those without peripheral neuropathy ($p = 0.667$). Also, the rate of AT calcifications did not differ significantly with the level of glycemic control, as measured by FBG and HbA_{1c} (both $p > 0.05$) (Tab. 5).

Tab. 4. *Comparison of hypoechoic foci of AT with characteristics of T2DM subjects*

Variables	Diabetics'			χ2	df	*p*-value
	AT hypoechoic foci					
	Present	Absent	Total			
Age (years)						
40–49	0 (0.0)	15 (100.0)	15 (100.0)			0.060*
50–59	2 (12.5)	14 (87.5)	16 (100.0)			
60–69	9 (31.0)	20 (69.0)	29 (100.0)			
70–79	5 (25.0)	15 (75.0)	20 (100.0)			
Gender						
Male	9 (30.0)	21 (70.0)	30 (100.0)	3.000	1	0.083
Female	7 (14.0)	43 (86.0)	50 (100.0)			
BMI (kg/m²)						
Normal	7 (18.9)	30 (81.1)	37 (100.0)	1.526	2	0.466
Overweight	7 (26.9)	19 (73.1)	26 (100.0)			
Obese	2 (11.8)	15 (88.2)	17 (100.0)			
Duration of DM (months)						
<5 years	6 (14.0)	37 (86.0)	43 (100.0)	2.320	1	0.314
5–10 years	7 (29.2)	17 (70.8)	24 (100.0)			
>10 years	3 (23.1)	10 (76.9)	13 (100.0)			
Any peripheral neuropathy						
Present	15 (26.3)	42 (73.7)	57 (100.0)			**0.031***
Absent	1 (4.3)	22 (95.7)	23 (100.0)			
FBS						
<5.6 (mmol/L)	4 (25.0)	12 (75.0)	16 (100.0)	0.506	2	0.776
5.6–6.9 (mmol/L)	4 (16.0)	21 (84.0)	25 (100.0)			
≥7.0 (mmol/L)	8 (20.5)	31 (79.5)	39 (100.0)			
Glycaemic control						
Poor (HbA_{1c} ≥7.0%)	11 (21.6)	40 (78.4)	51 (100.0)	0.216	1	0.642
Good (HbA_{1c} <7.0%)	5 (17.2)	24 (82.8)	29 (100.0)			

** Fisher's exact test; FBG – fasting blood glucose BMI – body mass index; T2DM – type 2 diabetes mellitus; AT – Achilles tendon*

Achilles tendon degenerative changes in controls

Among the controls, the prevalence of disorganized AT fibers and AT hypoechoic foci differed significantly across various age groups (both p <0.05) (Tab. 6). However, AT calcifications showed no significant difference across various age groups (p >0.05) among the controls. The was no significant difference in the prevalence of any of the three degenerative changes among the controls, males and females, or across BMI groups (p >0.05) (Tab. 6).

Discussion

The orientation of the collagen fibers in tendons is generally orderly and parallel in pattern. Diabetes mellitus causes increased production of advanced glycosylation end products, which leads to cross-linking within the collagen fibers, which can deteriorate the biomechanical function of tendons and ligaments[15]. In this study, the prevalence of degenerative features was significantly higher among T2DM subjects (55.0% in the right AT and 52.5% in the left AT) than controls (18.8% in both ATs) (p <0.001 for both). This higher prevalence in the subjects than in controls agrees with the findings by Batista *et al*.[7] and Abate *et al*.[12]

The degenerative changes observed among controls were majorly associated with advanced age as they were present mainly in those above 60 years of age. Similarly, among T2DM subjects, the mean age of those with degenerative changes was significantly higher than those without it. However, among those in their forties, degenerative changes were seen in only T2DM subjects, which suggests that the effects of diabetes mellitus on AT are independent of advanced age. Similarly, those with degenerative changes were older (both T2DM and controls) in the study by Abate *et al*.[12] In contrast, Batista *et al*.[7] found no association between age and degenerative changes among T2DM subjects. This disparity might result from the fact that the subjects in this study had a wider age range (40–80 years) compared with those of Batista *et al*. (52–78 years). D'Ambrogi *et al*.[16] showed that the effects of DM on AT were more difficult to evaluate beyond the threshold of 80 years of age[16].

There were no significant gender differences among the controls in the prevalence of any of the degenerative changes. However, in the T2DM subjects, a significantly higher rate was recorded among males than females for disorganized AT fibers. Abate *et al*.[12] did not observe any significant male/female differences, while sex differences were not evaluated by Batista *et al*.[7]

Tab. 5. *Achilles tendon calcifications compared by characteristics of T2DM subjects*

Variables	Diabetics' AT calcifications		Total	*p*-value*
	Present	Absent		
Age (years)				
40–49	0 (0.0)	15 (100.0)	15 (100.0)	0.512
50–59	1 (6.3)	15 (93.8)	16 (100.0)	
60–69	4 (13.8)	25 (86.2)	29 (100.0)	
70–79	1 (5.0)	19 (95.0)	20 (100.0)	
Gender				
Male	3 (10.0)	27 (90.0)	30 (100.0)	0.667
Female	3 (6.0)	47 (94.0)	50 (100.0)	
BMI				
Normal	3 (8.1)	34 (91.9)	37 (100.0)	0.564
Overweight	1 (3.8)	25 (96.2)	26 (100.0)	
Obese	2 (11.8)	15 (88.2)	17 (100.0)	
DM Duration (months)				
<5 years	2 (4.7)	41 (95.3)	41 (100.0)	0.295
5–10 years	2 (8.3)	22 (91.7)	24 (100.0)	
>10 years	2 (15.4)	11 (84.6)	13 (100.0)	
Any PN				
Present	5 (8.8)	52 (91.2)	57 (100.0)	0.667
Absent	1 (4.3)	22 (95.7)	23 (100.0)	
FBG				
<5.6 (mmol/L)	2 (12.5)	14 (87.5)	16 (100.0)	0.203
5.6–6.9 (mmol/L)	0 (0.0)	25 (100.0)	25 (100.0)	
≥7.0 (mmol/L)	4 (10.3)	35 (89.7)	39 (100.0)	
HbA_{1c}				
Poor (HbA_{1c} ≥7.0%)	3 (5.9)	48 (94.1)	51 (100.0)	0.662
Good (HbA_{1c} <7.0%)	3 (10.3)	26 (89.7)	29 (100.0)	

** Fisher's exact test; AT – Achilles tendon; FBG – fasting blood glucose; T2DM – type 2 diabetes mellitus; PN – peripheral neuropathy; BMI – body mass index; HbA_{1c} – glycated hemoglobin*

Overweight is a known risk factor for tendon degeneration[5]; however, in this study, both T2DM subjects and controls showed no significant influence of BMI on the occurrence of disorganized AT fibers, AT hypoechoic and calcific foci. This observation agrees with the findings of Batista *et al*.[7] Conversely, Abate *et al*.[5] found a significant association between BMI and degenerative changes. They observed echotexture disorders in 13.8% of tendons of healthy volunteers with normal BMI, while it was 34.3% in diabetic subjects who had BMI ≥25 kg/m² ($p = 0.02$).

The duration of DM was associated with disorganized AT fibers in this study. The prevalence of hypoechoic foci and calcifications increased with increasing duration of DM, but it was not statistically significant. Abate *et al*.[12] reported an association between the length of DM diagnosis and AT degenerative changes generally; however, the significance of the association of each of the assessed degenerative features was not stated. Batista *et al*.[7] could not establish a significant relationship between AT degenerative features and length of DM diagnosis.

The disorganization of AT fibers was significantly higher (71.9%) among T2DM subjects with peripheral neuropathy (PN) than those without PN (30.4%). Similarly, the presence of hypoechoic foci was also more statistically significant among T2DM subjects with PN (26.3%) than those without PN (4.3%). These observations align with the findings of Abate *et al*.[12], who also associated PN with AT degenerative changes. They stated that diabetic patients with AT degenerative changes had significantly increased diabetes duration and a significantly higher prevalence of PN than those without such AT changes. The difference between the DM duration of those with AT degenerative changes and those without AT degenerative changes was not significant in the study of Batista *et al*.[7]

Though increased degenerative changes were observed with an increased level of dysglycemia, there was no significant association between any of the AT degenerative changes and the level of glycemic control, as measured by fasting blood glucose and glycated hemoglobin. This agrees with the findings of Batista *et al*.[7], who observed a non-significant association between glycemic level and AT structural changes. According to Batista *et al*.[7], those with disorganized AT fibers had average FBG of 206.7 ± 13 mg/dL compared to 225.6 ± 43 mg/dL among those with organized AT fibers ($p > 0.01$). Also, no significant association was seen between tendon calcifications and FBG levels. Similarly, average HbA_{1c} among those with

Tab. 6. *Prevalence of various degenerative changes by characteristics of controls*

Variables	Present	Absent	Total	χ2	*p*-value
Disorganized AT fibers					
Age (years)					
40–49	0 (0.0)	16 (100.0)	16 (100.0)		**0.044***
50–59	2 (13.3)	13 (86.7)	15 (100.0)		
60–69	7 (25.0)	21 (75.0)	28 (100.0)		
70–79	7 (33.3)	14 (66.7)	21 (100.0)		
Gender					
Male	8 (23.5)	26 (76.5)	34 (100.0)	0.460	0.497
Female	8 (17.4)	38 (82.6)	36 (100.0)		
BMI (kg/m²)					
Normal	7 (17.5)	33 (82.5)	40 (100.0)	1.615	0.446
Overweight	8 (26.7)	22 (73.3)	30 (100.0)		
Obese	1 (10.0)	9 (90.0)	10 (100.0)		
AT hypoechoic foci					
Age (years)					
40–49	0 (0.0)	16 (100.0)	16 (100.0)		**0.035***
50–59	0 (0.0)	15 (100.0)	15 (100.0)		
60–69	2 (7.1)	26 (92.9)	28 (100.0)		
70–79	5 (23.8)	16 (76.2)	21 (100.0)		
Gender					
Male	3 (8.8)	31 (91.2)	34 (100.0)		1.000*
Female	4 (8.7)	42 (91.3)	46 (100.0)		
BMI (kg/m²)					
Normal	3 (7.5)	37 (92.5)	40 (100.0)		0.327*
Overweight	2 (6.7)	28 (93.3)	30 (100.0)		
Obese	2 (20.0)	8 (80.0)	10 (100.0)		
AT calcifications					
Age (years)					
40–49	0 (0.0)	16 (100.0)	16 (100.0)		1.000*
50–59	0 (0.0)	15 (100.0)	15 (100.0)		
60–69	1 (3.6)	27 (96.4)	28 (100.0)		
70–79	1 (4.8)	20 (95.2)	21 (100.0)		
Gender					
Male	0 (0.0)	34 (100.0)	34 (100.0)		0.505*
Female	2 (4.3)	44 (95.7)	46 (100.0)		
BMI (kg/m²)					
Normal	1 (2.5)	39 (97.5)	40 (100.0)		1.000*
Overweight	1 (3.3)	29 (96.7)	30 (100.0)		
Obese	0 (0.0)	10 (100.0)	10 (100.0)		

**Fisher's exact test; χ2 – chi-square; AT – Achilles tendon*

AT calcifications was 9.4 ± 1%, while those without calcifications had a value of 8.8 ± 0.3% ($p > 0.05$). No significant association was seen between HbA_{1c} levels and disorganized AT fibers. Abate *et al.*[12] did not report such a comparison.

Conclusion

This study demonstrated structural changes in Achilles tendons that are highly suggestive of biomechanical alterations. Type 2 diabetic patients have a higher prevalence of degenerative changes than non-diabetic controls. These changes are significantly influenced by PN and the duration of DM diagnosis. Further studies to evaluate progression and/or regression of these degenerative changes with clinical management are necessary.

Regarding study limitations, the duration of diabetes mellitus was estimated from the time of diagnosis in a hospital. This is a conservative estimation as subjects could have had the disease before reporting to the hospital.

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