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# Morphological and Biomechanical Alterations of Achilles Tendon and Plantar Fascia in Type 2 Diabetes with Peripheral Neuropathy: A Shear Wave Elastography Study

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***ABSTRACT***
***Background:*** *Diabetes mellitus with peripheral neuropathy affects foot structures, potentially contributing to complications including ulceration and biomechanical dysfunction.*
***Objective:*** *To evaluate morphological and biomechanical properties of Achilles tendon and plantar fascia in diabetic patients with peripheral neuropathy using ultrasonography and shear wave elastography.*
***Methods:*** *This cross-sectional study compared 32 diabetic patients with peripheral neuropathy to 31 non-diabetic controls. Bilateral measurements of Achilles tendon and plantar fascia thickness were obtained using B-mode ultrasonography. Tissue stiffness was assessed using shear wave elastography. Statistical analysis included t-tests and Pearson correlations.*
***Results:*** *Cases were older (60±13 vs 50±14 years, p<0.001) with elevated fasting blood sugar (156±65 vs 82±10 mg/dl, p<0.001). Achilles tendon thickness increased bilaterally in cases (right: 6.0±1.2 vs 3.6±1.2mm; left: 6.08±1.19 vs 3.66±1.27mm; both p<0.001). Plantar fascia showed greater thickening (right: 4.9±0.8 vs 2.1±0.7mm; left: 5.1±1.0 vs 2.2±0.7mm; both p<0.001). Tissue stiffness was elevated in cases for Achilles tendon (3.40±0.70 vs 2.24±0.46 m/s, p<0.001) and plantar fascia (2.96±0.69 vs 1.77±0.42 m/s, p<0.001). Strong correlations existed between fasting blood sugar and all measurements (r=0.55-0.72, p<0.001).*
***Conclusion:*** *Diabetic peripheral neuropathy causes significant thickening and increased stiffness of foot structures, with plantar fascia more affected than Achilles tendon. These alterations correlate strongly with glycemic status, suggesting hyperglycemia as a key pathogenic factor.*
***Keywords:*** *Diabetes mellitus; peripheral neuropathy; shear wave elastography; Achilles tendon; plantar fascia; ultrasonography*

## I. INTRODUCTION

Diabetes mellitus represents a global health crisis characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both (1). India harbors approximately 77 million diabetic patients, ranking second globally, with projections indicating an increase to 109 million by 2035 (2). Among diabetes complications, peripheral neuropathy affects approximately 50% of patients, primarily involving the lower extremities and contributing significantly to foot morbidity (3).

The pathophysiology of diabetic peripheral neuropathy (DPN) involves multiple mechanisms initiated by chronic hyperglycemia. Advanced glycation end products (AGEs) form through non-enzymatic glycation of proteins, fundamentally altering tissue structure and function (4). These AGEs accumulate within collagen fibers, creating abnormal cross-links that increase tissue stiffness and reduce elasticity, particularly affecting load-bearing foot structures essential for normal biomechanics (5).

The Achilles tendon, the body's strongest and largest tendon, measures approximately 15 centimeters in length and undergoes significant mechanical loading during locomotion (6). Its unique spiral architecture, with fibers rotating 90 degrees before calcaneal insertion, creates differential stress distribution areas potentially vulnerable to diabetes-related changes (7). The plantar fascia, originating from the medial calcaneal tubercle and extending to the metatarsophalangeal joints, maintains the medial longitudinal arch and functions as a dynamic shock absorber (8). Composed predominantly of longitudinally arranged type I collagen fibers interspersed with elastic fibers, it provides essential viscoelastic properties for foot function (9).

Recent investigations reveal that diabetes induces significant structural alterations in both structures. Histopathological studies demonstrate collagen fiber disorganization, increased interfibrillar ground substance, and neovascularization in diabetic tendons (10). These morphological changes accompany biomechanical alterations including reduced compliance and altered stress-strain relationships, compromising normal shock absorption and potentially contributing to increased plantar pressures and ulceration risk (11).

While conventional ultrasonography provides morphological assessment, it cannot evaluate mechanical properties. Shear wave elastography (SWE) enables quantitative tissue stiffness assessment through acoustic radiation force-generated shear waves, with propagation velocity directly proportional to tissue stiffness ($E = 3\rho c^2$) (12). This technique offers operator-independent, reproducible measurements ideal for monitoring disease progression (13).

Previous elastography studies in diabetic populations report variable findings, potentially reflecting differences in patient selection and measurement techniques (14). Understanding relationships between glycemic control and musculoskeletal properties is crucial for developing targeted interventions. This study comprehensively evaluates Achilles tendon and plantar fascia in diabetic patients with established peripheral neuropathy using both conventional ultrasonography and SWE, addressing critical knowledge gaps regarding morphological and biomechanical consequences of diabetic neuropathy on weight-bearing structures.

## II. METHODS

**Study Design and Population**

This cross-sectional observational study was conducted at a tertiary care hospital from May 2023 to January 2024 after institutional ethics committee approval. Written informed consent was obtained from all participants.

**Participants**

Sample size calculation, based on previous studies reporting 2.0mm mean difference in Achilles tendon thickness with 1.5mm standard deviation, indicated 28 participants per group were required ($\alpha$=0.05, power=80%). We recruited 32 diabetic patients with peripheral neuropathy (cases) and 31 non-diabetic controls.

Inclusion criteria for cases included: confirmed type 2 diabetes mellitus; peripheral neuropathy diagnosed by abnormal monofilament test (10g), reduced vibration perception threshold (>25V), or abnormal nerve conduction studies; age 18-80 years. Control inclusion criteria: absence of diabetes (fasting glucose <100mg/dl, HbA1c <5.7%); no neuropathy history; age-matched within 5 years.

Exclusion criteria: Achilles tendon or plantar fascia pathology history; diabetic foot ulcers; peripheral vascular disease (ankle-brachial index <0.9); connective tissue disorders; recent lower limb trauma; BMI >40 kg/m²; pregnancy.

**Clinical Assessment**

Demographic data and medical history were collected through structured interviews. Peripheral neuropathy was assessed using 10g Semmes-Weinstein monofilament at ten sites per foot and vibration perception threshold using biothesiometer. Fasting blood glucose was measured after 8-hour overnight fast using hexokinase method.

**Ultrasound Examination**

All examinations were performed using Siemens ACUSON S2000 with 9L4 linear transducer (4-9 MHz) by a single experienced sonographer. Participants were positioned prone with feet hanging freely at 90-degree ankle flexion.

Achilles tendon thickness was measured 2-6cm proximal to calcaneal insertion in transverse plane, recording anteroposterior diameter perpendicular to tendon fibers. Plantar fascia thickness was measured at calcaneal origin in sagittal plane aligned with second metatarsal. Three measurements were averaged for each structure.

**Shear Wave Elastography**

Virtual Touch Quantification was performed immediately following B-mode assessment. A 10mm×5mm region of interest was positioned within each structure, ensuring perpendicular beam orientation to minimize anisotropy. Ten consecutive shear wave velocity measurements were obtained, recording median values in meters per second.

**Statistical Analysis**

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean±standard deviation, categorical variables as frequencies and percentages. Between-group comparisons used independent t-tests for parametric data and chi-square tests for categorical variables. Pearson correlations assessed relationships between variables. Multiple linear regression identified independent predictors. $P<0.05$ was considered significant.

## III. RESULTS

**Participant Characteristics**

The study included 32 diabetic patients with peripheral neuropathy and 31 controls. Cases were significantly older (60±13 vs 50±14 years, p<0.001) with male predominance in both groups (cases: 65.6%, controls: 80.6%, p=0.171). Median diabetes duration was 8 years (IQR 5-12). Mean HbA1c was 8.4±1.8% in available cases (n=28).

**Table 1: Demographic and Glycemic Parameters**

| Parameter | Cases (n=32) | Controls (n=31) | p-value |
|---|---|---|---|
| Age (years) | 60±13 | 50±14 | <0.001 |
| Male (%) | 65.6 | 80.6 | 0.171 |
| BMI (kg/m²) | 26.8±3.2 | 24.3±2.8 | 0.002 |
| FBS (mg/dl) | 156±65 | 82±10 | <0.001 |
| VPT right (V) | 32.4±8.7 | 12.3±3.2 | <0.001 |
| VPT left (V) | 31.8±9.1 | 12.1±3.5 | <0.001 |

FBS: Fasting blood sugar; VPT: Vibration perception threshold

**Morphological Findings**

Achilles tendon thickness was significantly increased bilaterally in cases. Right side measured 6.0±1.2mm in cases versus 3.6±1.2mm in controls (67% increase, p<0.001). Left side showed similar changes (6.08±1.19mm vs 3.66±1.27mm, p<0.001).

Plantar fascia demonstrated more pronounced thickening. Right side measured 4.9±0.8mm in cases versus 2.1±0.7mm in controls (133% increase, p<0.001). Left side thickness was 5.1±1.0mm versus 2.2±0.7mm (p<0.001).

**Table 2: Thickness Measurements**

| Structure | Cases | Controls | Difference (95% CI) | p-value |
|---|---|---|---|---|
| AT-Right (mm) | 6.0±1.2 | 3.6±1.2 | 2.4 (1.8-3.0) | <0.001 |
| AT-Left (mm) | 6.08±1.19 | 3.66±1.27 | 2.42 (1.8-3.0) | <0.001 |
| PF-Right (mm) | 4.9±0.8 | 2.1±0.7 | 2.8 (2.4-3.2) | <0.001 |
| PF-Left (mm) | 5.1±1.0 | 2.2±0.7 | 2.9 (2.5-3.3) | <0.001 |

AT: Achilles tendon; PF: Plantar fascia; CI: Confidence interval

**Biomechanical Findings**

Shear wave elastography revealed significantly increased stiffness in diabetic patients. Achilles tendon stiffness was 3.40±0.70 m/s in cases versus 2.24±0.46 m/s in controls (52% increase, p<0.001). Plantar fascia stiffness measured 2.96±0.69 m/s versus 1.77±0.42 m/s (67% increase, p<0.001).

**Table 3: Stiffness Measurements**

| Structure | Cases | Controls | Difference (95% CI) | p-value |
|---|---|---|---|---|
| AT Stiffness (m/s) | 3.40±0.70 | 2.24±0.46 | 1.16 (0.87-1.45) | <0.001 |
| PF Stiffness (m/s) | 2.96±0.69 | 1.77±0.42 | 1.19 (0.91-1.47) | <0.001 |

**Correlation Analysis**

Strong positive correlations existed between fasting blood sugar and all parameters. Strongest correlation was with right plantar fascia thickness (r=0.72, p<0.001), followed by left plantar fascia (r=0.69, p<0.001). Achilles tendon thickness showed moderate correlations (right: r=0.63, left: r=0.59, both p<0.001). Stiffness measurements correlated with glycemia (AT: r=0.55, PF: r=0.60, both p<0.001).

**Table 4: Correlations with Fasting Blood Sugar**

| Parameter | Correlation (r) | p-value |
|---|---|---|
| AT thickness right | 0.63 | <0.001 |
| AT thickness left | 0.59 | <0.001 |
| PF thickness right | 0.72 | <0.001 |
| PF thickness left | 0.69 | <0.001 |
| AT stiffness | 0.55 | <0.001 |

| Parameter | Correlation (r) | p-value |
|---|---|---|
| PF stiffness | 0.60 | <0.001 |

Inter-structural correlations revealed strong associations between Achilles tendon and plantar fascia measurements (r=0.78-0.82, p<0.01). Thickness-stiffness correlations were stronger for plantar fascia (r=0.71-0.76) than Achilles tendon (r=0.51-0.57).

**Bilateral Symmetry and Sex Differences**

No significant bilateral differences existed for Achilles tendon thickness in either group (cases: p=0.345, controls: p=0.367). Plantar fascia showed small but significant left-sided thickening in cases only (5.1 vs 4.9mm, p=0.031). Sex-stratified analysis revealed no significant differences between males and females within groups (all p>0.05).

**Table 5: Multiple Regression Analysis**

| Dependent Variable | Independent Predictor | β Coefficient | p-value |
|---|---|---|---|
| AT thickness | FBS | 0.58 | <0.001 |
| | Age | 0.22 | 0.042 |
| PF thickness | FBS | 0.65 | <0.001 |
| | Age | 0.14 | 0.186 |

Multiple regression identified fasting blood sugar as the strongest independent predictor of both Achilles tendon (β=0.58, p<0.001) and plantar fascia thickness (β=0.65, p<0.001) after adjusting for age, sex, and BMI.

## IV. DISCUSSION

This study demonstrates significant morphological and biomechanical alterations in Achilles tendon and plantar fascia of diabetic patients with peripheral neuropathy. The observed changes—67% increased Achilles tendon thickness, 133% increased plantar fascia thickness, and corresponding stiffness increases—represent substantial deviations from normal tissue properties with important clinical implications.

Our findings exceed previously reported changes. Evranos et al. documented 46% increased Achilles tendon thickness in diabetic patients without specific neuropathy assessment (15). Our greater magnitude likely reflects inclusion of patients with established neuropathy, representing advanced disease. The mean 6.0mm thickness substantially exceeds the 4-6mm normal range, indicating clinically significant alterations (16).

The 133% plantar fascia thickening represents one of the largest reported increases. Previous studies documented 30-80% increases, but our neuropathy-specific cohort may experience more severe changes (17). Mean thickness of 4.9-5.1mm exceeds the 4.0mm plantar fasciitis diagnostic threshold, yet patients were asymptomatic, highlighting distinct diabetic fasciopathy pathophysiology (18).

Differential changes between structures warrant consideration. The plantar fascia's greater susceptibility may relate to its complex loading patterns—tension, compression, and shear forces—unlike the primarily tensile Achilles tendon loading (19). Additionally, its proximity to plantar fat pad and role in arch maintenance may increase metabolic stress susceptibility.

Our elastography findings align with recent investigations. Fu et al. reported similar Achilles tendon stiffness increases (3.28±0.61 vs 2.31±0.48 m/s) in diabetic patients (20). Increased stiffness results from AGE accumulation causing abnormal collagen cross-linking, reduced proteoglycan content, and altered extracellular matrix composition (21). Interestingly, percentage stiffness increase (52% Achilles, 67% plantar fascia) was less than thickness increase, suggesting morphological changes may precede mechanical alterations.

Strong correlations between glycemia and tissue parameters (r=0.55-0.72) indicate dose-dependent relationships. Akturk et al. similarly reported significant HbA1c-Achilles tendon thickness correlations (r=0.64, p<0.001) (22). These findings suggest intensive glycemic control might prevent or ameliorate musculoskeletal complications, though specific studies are needed.

Clinical implications are substantial. Increased thickness and stiffness reduce ankle dorsiflexion, a recognized ulceration risk factor. Mueller et al. demonstrated 5.3-fold increased ulcer risk in diabetic patients with limited joint mobility (23). Altered plantar fascia properties compromise shock absorption, potentially increasing plantar pressures. Bus et al. reported 2.4-fold increased ulceration risk with elevated plantar pressures (24).

Bilateral symmetry suggests systemic rather than localized pathology, supporting bilateral assessment recommendations. Minor plantar fascia asymmetry may reflect subtle compensatory mechanisms. Sex-stratified analysis revealed no differences, indicating universal diabetes effects across genders.

Study strengths include neuropathy-specific focus, comprehensive morphological and biomechanical assessment, and standardized SWE protocols providing quantitative measurements. Limitations include cross-sectional design precluding temporal assessment, age differences between groups despite statistical adjustment, and lack of functional outcome measures.

Future research should investigate longitudinal progression, relationships to clinical outcomes, and intervention potential. Establishing whether intensive glycemic control or targeted therapies can reverse alterations has significant implications. Molecular pathway investigation may identify novel therapeutic targets. Standardized ultrasound and elastography protocols would facilitate multicenter studies and clinical implementation.

## V. CONCLUSION

Diabetic peripheral neuropathy causes significant morphological and biomechanical alterations in Achilles tendon and plantar fascia. Substantial thickness increases (67% Achilles tendon, 133% plantar fascia) and stiffness elevations (52% Achilles tendon, 67% plantar fascia) likely contribute to abnormal foot biomechanics and increased ulceration risk characteristic of diabetic foot disease.

Strong glycemic parameter correlations emphasize optimal diabetes management importance in preventing musculoskeletal complications. Greater plantar fascia changes suggest differential tissue susceptibility related to anatomical and biomechanical factors. Ultrasound and shear wave elastography provide valuable non-invasive assessment tools that could facilitate early detection, risk stratification, and treatment monitoring.

These findings enhance understanding of diabetic foot complication pathophysiology and support comprehensive management approaches addressing both metabolic and musculoskeletal aspects. Recognition and quantification of these alterations may improve outcomes for millions affected by diabetic peripheral neuropathy worldwide.

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