REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FASCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLITUS TYPE II – A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY

BY

REG. NO. BS0121004

Dissertation

Submitted to

KAHER, Belagavi, Karnataka

In partial fulfilment of the requirements for the degree of

M.D.

IN

RADIO-DIAGNOSIS

DEPARTMENT OF RADIO-DIAGNOSIS

JAWAHARLAL NEHRU MEDICAL COLLEGE

BELAGAVI-590010, KARNATAKA

DEC 2024/ JAN 2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

BELAGAVI, KARNATAKA

Endorsement by the HOD/Principal/ Head of the Institution

This is to certify that the dissertation entitled **"REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FASCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLITUS TYPE II- A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY"** is a bonafide research work done by **REG NO. BS0121004.**

Dr. SANTOSH D. PATIL
M.D. (Radio-Diagnosis)
Professor & HOD
Dept. of Radio-diagnasis
J.N. Medical College, BELAGAVI-10.
KMC Reg No. 58456

Dr SANTOSH D. PATIL
M.D. RADIO-DIAGNOSIS

Professor and Head,
Department of Radio Diagnosis,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date: 26/06/2024
Place: Belagavi

Dr N. S. MAHANTASHETTI
M.D. PAEDIATRICS

PRINCIPAL
J.N. Medical College.
BELAGAVI- 590 010

Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

KAHER
BELAGAVI
J.N. MEDICAL COLLEGE

Date : 26/06/2024
Place : Belagavi

UNDERTAKING

I, **Reg. No. BS0121004,** hereby declare that the information and the data mentioned in my dissertation entitled **"REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FASCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLITUS TYPE II- A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 26/06/2024
Place: Belagavi

REG. NO. BS0121004

PLAGIARISM ACCEPTANCE LETTER

KLE JAWAHARLAL NEHRU MEDICAL COLLEGE
(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)
(Recognized by National Medical Commission, New Delhi)
Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)
Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 25-06-2024

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: "REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FASCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLITUS TYPE II – A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY" has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **04%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BS0121004
Postgraduate Student,
2021-22 Batch,
Department of Radio-Diagnosis
J. N. Medical College, Belagavi.

INSTITUTIONAL ETHICAL CLEARENCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed - to-be-University)
Accredited 'A++' Grade By NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (Govt.)

JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http://www.jnmc.edu
E-Mail : drmc@jnmc.edu

Phone: (+91-0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 47 **Date: 27/09/2022**

To,

REG. NO.BS0121004
PG Student in Radiodiagnosis,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FASCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLITUS TYPE II – A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY."** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

ABSTRACT

Introduction: Diabetes mellitus, one of the commonest chronic non-communicable diseases has been associated with collagen vascular changes including tendinitis, fasciitis and tendinopathy in various fascia and tendon of both upper and lower limbs.

Objective: To compare the plantar fascia thickness and Achilles tendon thickness of diabetics to that of a control group and correlate the thickness of these with other parameters.

Methodology: A one-year hospital based study done in the department of Radiodiagnosis at the KLES DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI with 80 subjects. Subjects with complaints of heel pain were considered . 40 patients in each group, Group A considering as case group: known case of diabetics and group B: control group, having non-diabetics. USG on GE LOGIQ P9 R2 machine equipped with a 7.5–12 MHz high frequency linear array transducer.

Results: The plantar fascia thickness among diabetic and non-diabetic was 4.76 ± 0.974mm and 3.56 ± 0.514 on left foot. 4.76 ± 1.007 and 3.56 ± 0.516 among diabetic and non-diabetic respectively in right foot. The thickness of left foot Achilles 5.67 ± 0.881mm and 3.613 ± 0.515 mm respectively among diabetic and non-diabetic patients. Whereas the right foot AT thickness was 5.665 ± 0.888 and 3.615 ± 0.465 respectively, which had significant increase in diabetics ($p < 0.05$) From Spearman's rho Correlation, BMI, age, FBS and duration of diabetes had significant positive correlation with the increased thickness of AT and PF in bilateral foot.

Conclusion: Diabetes has significant positive correlation with the thickness of plantar fascia and the achilles tendon which further has the significant positive correlation with BMI, Age, FBS and the duration of diabetes. Hence, it would be better to add USG of plantar fascia and achilles tendon as one of the follow up investigation for early diagnosis and management.

Keywords: Diabetes Mellitus, Achilles tendon, planter fascia, Sonography

LIST OF ABBREVIATIONS USED

DM	DIABETES MELLTIUS
T2DM	TYPE 2 DIABETES MELLITUS
PF	PLANTAR FASCIITIS
ATT	ACHILLES TENDON THICKNESS
PFT	PLANTAR FASCIA THICKNESS
ATR	ACHILLES TENDON RUPTURE
AGES	ADVANCED GLYCATION END-PRODUCTS
DPN	DISTAL POLYNEUROPATHY
BMI	BODY MASS INDEX
FBS	FASTING BLOOD GLUOCSE
AMPK	5’ ADENOSINE MONOPHOSPHATE ACTIVATED PROTEIN KINASE
EGR1	EARLY GROWTH RESPONSE PROTEIN 1
TGF-β1	TRANSFORMING GROWTH FACTORE BETA 1
GAGs	GLYCOSAMINOGLYCANS
MRI	MAGNETIC RESONANCE IMAGING
USG	ULTRASONOGRAPHY
VISA	VICTORIAN INSTITUTE OF SPORTS ASSESSMENT
DFU	DIABETIC FOOT ULCER

TABLE OF CONTENTS

SR. NO.	CONTENTS	PAGE NO.
1	INTRODUCTION	1-2
2	OBJECTIVES	3
3	REVIEW OF LITERATURE	4-28
4	MATERIAL AND METHODS	29-32
5	RESULTS	33-47
6	DISCUSSION	48-52
7	STRENGTH AND LIMITATIONS	53
8	CONCLUSION	54
9	SUMMARY	55
10	BIBLIOGRAHY	56-63
11	ANNEXURE I- CONSENT	64-66
12	ANNEXURE II- PROFORMA	67
13	ANNEXURE III- CLINICAL IMAGES	68-70
14	ANNEXURE IV- KEY TO MASTERCHART	71
15	ANNEXURE V- MASTERCHART	72-73

LIST OF TABLES

TABLE. NO.	TABLE DESCRIPTION	PAGE NO.
1	Distribution of subjects according to demographic details over groups	33
2	Distribution of subjects according to different variables over groups	35
3	Distribution of subjects according to duration of diabetes (years)	35
4	Distribution of subjects according to different variables over groups	36
5	Spearman's rho Correlation between different variables in diabetic group over PFT and AT.	39
6	Spearman's rho Correlation between BMI over PFT and AT in non diabetic group.	44

LIST OF GRAPHS

GRAPH. NO.	GRAPHS DESCRIPTION	PAGE NO.
1	Distribution of subjects based on age over groups.	33
2	Distribution of subjects based on gender over groups.	34
3	Distribution of subjects based on BMI (Kg/m^2) over groups.	34
4	Distribution of subjects based on duration of diabetes (years).	36
5	Mean plot of Plantar Fascia thickness of left side over groups	37
6	Mean plot of Plantar Fascia thickness of right side over groups.	37
7	Mean plot of Achilles Tendon of left side over groups	38
8	Mean plot of Achilles Tendon of right side over groups	38
9	Scatter plot of Age (years) in diabetic group and Achilles tendon thickness on left side.	40
10	Scatter plot of Age (years) in diabetic group and Achilles tendon thickness on right side	40
11	Scatter plot of BMI (Kg/m2) in diabetic group and Achilles tendon thickness on left side	41
12	Scatter plot of BMI (Kg/m2) in diabetic group and Achilles tendon thickness on right side	41
13	Scatter plot of HBA1c (%) in diabetic group and Achilles tendon thickness on left side	42
14	Scatter plot of fasting blood glucose (mg/dl) in diabetic group and Achilles tendon thickness on left side	42

15	Scatter plot of fasting blood glucose (mg/dl) in diabetic group and Achilles tendon thickness on right side	43
16	Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on left side in diabetic group	43
17	Scatter plot of BMI (Kg/m2) in non diabetic group and Achilles tendon thickness on left side	44
18	Scatter plot of BMI (Kg/m2) in non diabetic group and Achilles tendon thickness on right side	45
19	Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on left side in diabetic group	45
20	Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on right side in diabetic group	46
21	Scatter plot of duration of diabetes (years) and Achilles tendon on left side in diabetic group	46
22	Scatter plot of duration of diabetes (years) and Achilles tendon on right side in diabetic group	47

LIST OF FIGURES

FIGURE. NO.	FIGURE DESCRIPTION	PAGE NO.
1	Anatomy of sole depicting Plantar fascia	5
2	Schematic representation of achilles tendon	9
3	Schematic representation of pathophysiology of type 2 diabetes: Insulin overview	12
4	Image illustrating the pathophysiology of plantar fasciitis in diabetic patients	15
5	Pathophysiology of tendinopathy among diabetics	16
6	Illustration of Haglund's deformity	18
7	X ray imaging of Plantar fasciitis	19
8	The ultrasound after interacting with the tissue, undergoes one of the above mechanisms based on the tissue density. Which is almost equivalent to the optical law.	20
9	Plantar fasciitis on imaging	22
10	Clinial grading system and USG grade for PF	23
11	Representative ultrasound images for measuring Achilles tendon thickening by Corrigan P et al	24
12	MRI showing thickened and edematous Achilles ten- don.	25
13	Grading of tendinopathy	25

INTRODUCTION

Diabetes Mellitus is one of the epidemic disease affecting 463 million people worldwide and has been predicted to increase incidence by 33.8% which might be 700 million patients by 2045. According to the recent statistics by World health organisation, 77 million Indian population aged 18 years and above are suffering from type 2 diabetes. Whereas almost 25 million are prediabetics.[1,2]

It is one of the frequent metabolic diseases characterized by chronic hyperglycemia which is due to defects in insulin secretion, insulin action/insulin resistance or even be both. In this, the metabolic abnormalities in carbohydrates, proteins and lipids would be resulting from the importance of insulin, which is basically an anabolic hormone leading to a myriad of chronic impairments of micro as well as macroangiopathic involvement and polyneuropathy. This would be further worsening to involve the underlying fasciae and even the bone.[3]

About fasciae, these have a major role in proprioception, transmitting the force of muscular force component, vascularization of the skin above them, maintaining its tropism and the healing local wounds. These are a dynamic multifaceted meshwork of connective tissue comprising diverse cells settled at the extracellular matrix and nervous fibers.[4] Each constituent in this takes its particular state in the fasciae adapting, various ways to the diverse stimuli. Also, the thickening of collagen, fragmentation of elastic fibers and impairment in glycosaminoglycans, specially the hyaluronan would be leading to stiffness, changes in gliding force as well. With these cascading repercussions at the cellular and molecular levels, the further complications of fascia would be progressed.[4-6]

Glycation end products will be accumulated in the musculoskeletal cells has been observed to the major pathological behind progression of the underlying disease and other

related complications. Among the diabetic patients, Frozen shoulder, rotator cuff tears, Dupuytren's contracture, trigger finger and Cheiroarthropathy are the commonest complications of upper extremities. Meanwhile, Achilles tendon tightness, referred as eqinus, heel spurs and increased plantar fascia thickness (PFT) are the most common musculoskeletal conditions in the lower extremity.[6,7]

Plantar fascia (PF) and Achilles tendon (AT), are the two major fasciae of the foot playing an important role in maintaining the lower limb biomechanics. Recent studies have interpreted with the evidences that structural changes and altered stiffness of AT in diabetic patients might increase the foot load and accelerate the occurrence of diabetic foot. In clinical practice, because the AT is thick and superficial, it is easy to examine its thickness and stiffness in patients with diabetes.[8]

DM also predisposes to plantar fasciopathy, which is characterized by thickened plantar fascia (PF), and loss of the normal organized PF architecture. The pathophysiology leading to involvement of the PF and AT in DM can be summarized as follows: sustained hyperglycemia leads to increased glycosylation of proteins, resulting in accumulation of "advanced glycosylation end products" in soft tissue and in thickening and avascularisation of the AT and PF. [9,10]

This increased ATT and PFT in DM have been considered an expression of soft-tissue damages. This factor, besides neuropathy, vasculopathy, and metabolic disorders, may contribute to the onset of ulcers in the diabetic foot. The muscles, cartilages, tendons, ligaments, all might experience structural changes even before the onset of diabetic neuropathy, and might then result in alteration of the overall function of the foot–ankle complex. [8-10]

OBJECTIVES OF THE STUDY

Primary objective:

- The objective of this study is to compare the plantar fascia thickness and Achilles tendon thickness of diabetics to that of a control group.

Secondary objective:

- Correlation analysis between plantar fascia thickness and Achilles tendon thickness with demographic data such as body mass index, duration of diabetes, fasting blood glucose level, HbA1c, sex and age of the patient.

REVIEW OF LITERATURE

PLANTAR FASCIA

The plantar fascia is a thick band of connective tissue that supports the foot's plantar arch. It originates at the calcaneal tuberosity of the hindfoot, ultimately inserting into the periosteum at the base of the toes' proximal phalanges. This thick fibrous aponeurosis that originates at the medial calcaneal tubercle and helps support the arch of the foot. It is thought that repetitive tensile overload from standing for long periods of time or running causes changes in the aponeurosis that can be either acute or chronic.

More recently, the term plantar fasciosis has been introduced to de-emphasize the idea that inflammation is the cause of pain. Histopathologic studies have shown that patients with diagnosed plantar fasciitis have more disorganization of fibrous tissue similar to degenerative tendinosis rather than inflammation.[11,12]

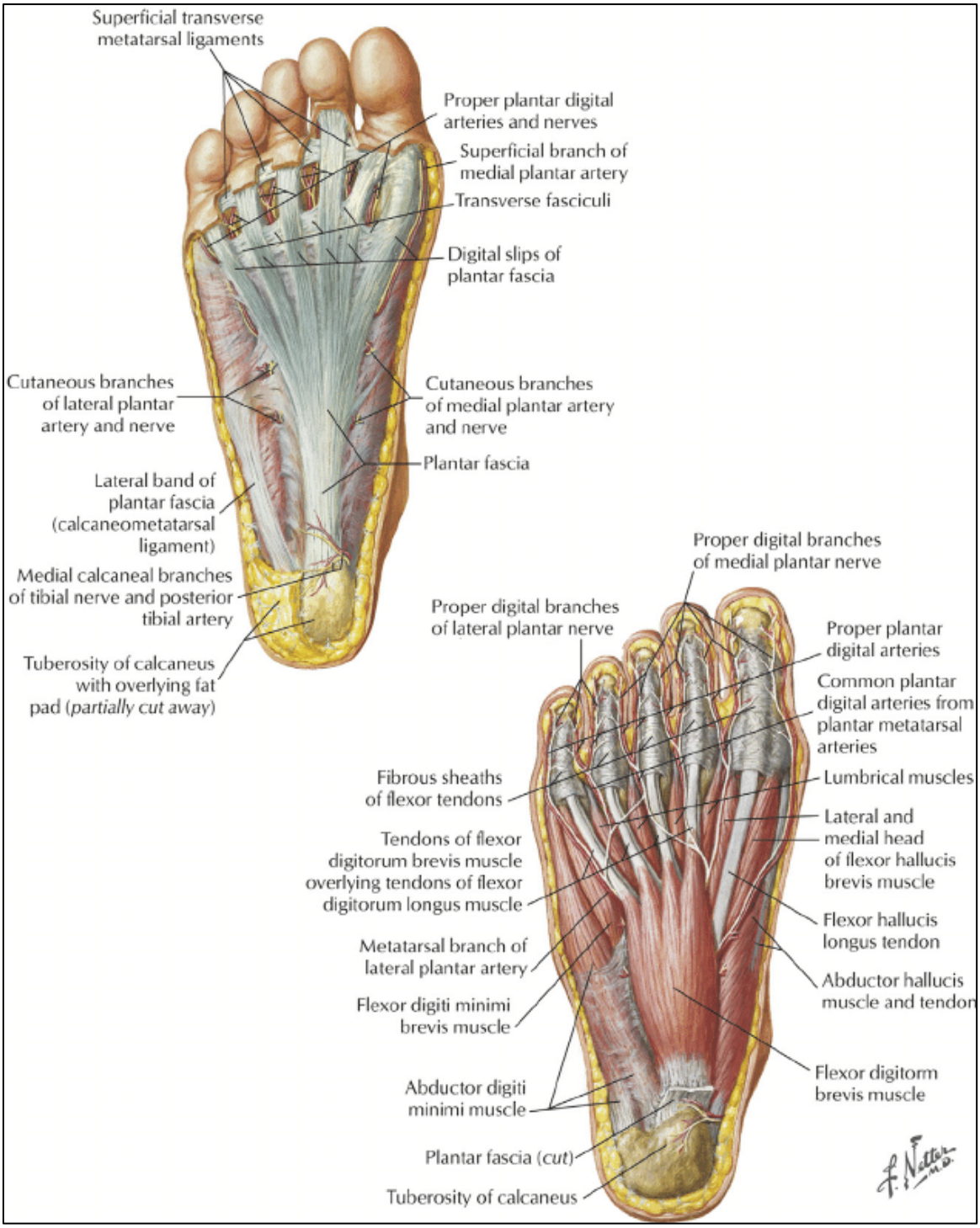


Figure 1: Anatomy of sole depicting Plantar fascia[13]

PLANTAR FASCIITIS

Plantar fasciitis, a prevalent and often vexing condition, arises from the degenerative irritation of the plantar fascia origin at the medial calcaneal tuberosity of the heel and its surrounding perifascial structures. The plantar fascia comprises 3 segments originating from the calcaneus and plays a pivotal role in maintaining the normal biomechanics of the foot, providing essential arch support, and serving as a shock absorber.[14]

An absence of inflammatory cells characterizes this condition despite its name. In the United States, millions of individuals suffer from heel pain each year, with plantar fasciitis being a primary culprit.[15] While multifactorial in its origins, overuse stress is often the leading cause, presenting with sharp localized pain at the heel and, occasionally, a heel spur. Non-surgical approaches are the primary mode of management, yet recurrent pain can be frustrating for both patients and healthcare providers.[16,17]

ETIOLOGY OF PLANTAR FASCIITIS

Plantar fasciitis is often an overuse injury primarily due to a repetitive strain causing micro-tears of the plantar fascia. Still, this condition can occur due to trauma or other multifactorial causes. Some predisposing factors are pes planus, pes cavus, limited ankle dorsiflexion, prolonged standing or jumping, and excessive pronation or supination. Pes planus can cause increased strain at the origin of the plantar fascia. Pes cavus can cause excessive strain on the heel because the foot does not effectively evert or absorb shock. Tightness in the gastrocnemius, soleus, and other muscles situated in the posterior leg is common for patients with this condition. Tight muscles can alter the normal biomechanics of ambulation.[18,19]

Approximately half of the patients with this condition will also have heel spurs, but the spurs are not the cause. Plantar fasciitis is often associated with runners and older adults, but other risk factors include obesity, heel pad atrophy, aging, occupations requiring prolonged standing, and weight-bearing. Plantar fasciitis is associated with various seronegative spondyloarthropathies, but there are no known systemic factors in approximately 85% of cases.[14,20]

RISK FACTORS FOR PLANTAR FASCIITIS[13,21-23]

Intrinsic risk factors	
Anatomic	Obesity
	Pes planus (flat feet)
	Pes cavus (high-arched feet)
	Shortened Achilles tendon
Biomechanic	Overpronation (inward roll)
	Limited ankle dorsiflexion
	Weak intrinsic muscles of the foot
	Weak plantar flexor muscles
Extrinsic risk factors	
Environmental	Poor biomechanics or alignment
	Deconditioning
	Hard surface
	Walking bare foot
	Prolonged weight bearing
	Inadequate stretching
	Poor footwear

DIFFERENTIAL DIAGNOSIS[13]

Possible differential diagnoses include the following:

- Calcaneus injury
- Infection
- Sickle cell bony pain
- Bone contusion
- Neuropathic pain
- Tendinitis
- Osteoporosis
- Malignancy

ACHILLES TENDON

This is the thickest and strongest tendon with the average length of 15cm, varying between 11 to 26 cm. The average width is about 4.5 to 8.6 cm. At the midsection, it reduced between 1.2 to 2.6 cm. This would be more rounded at an average of 4 cm above the calcaneus a width of 3.4 cm (2.0–4.8 cm) at its insertion site over the posterior surface of the calcaneus.[24,25]

Its origin lies close to the middle of the calf, and fuses with the gastrocnemius muscle proximally. The gastrocnemius is a fusiform muscle formed by two heads, medial and lateral, each separately crossing the knee joint. The medial and lateral heads fuse in a single muscle belly occupying the posterior superficial compartment of the lower leg.

Together with the gastrocnemius, it forms the three-headed triceps surae, which acts to plantarflex the ankle joint via its conjoint tendon, the Achilles tendon. The AT presents three main vascular areas: the peroneal artery supplies the midsection, while the posterior tibial artery supplies the proximal and distal sections. The relatively poor vascularisation of the mid-substance of the tendon might explain the frequent incidence of pathology at this site.[24-26]

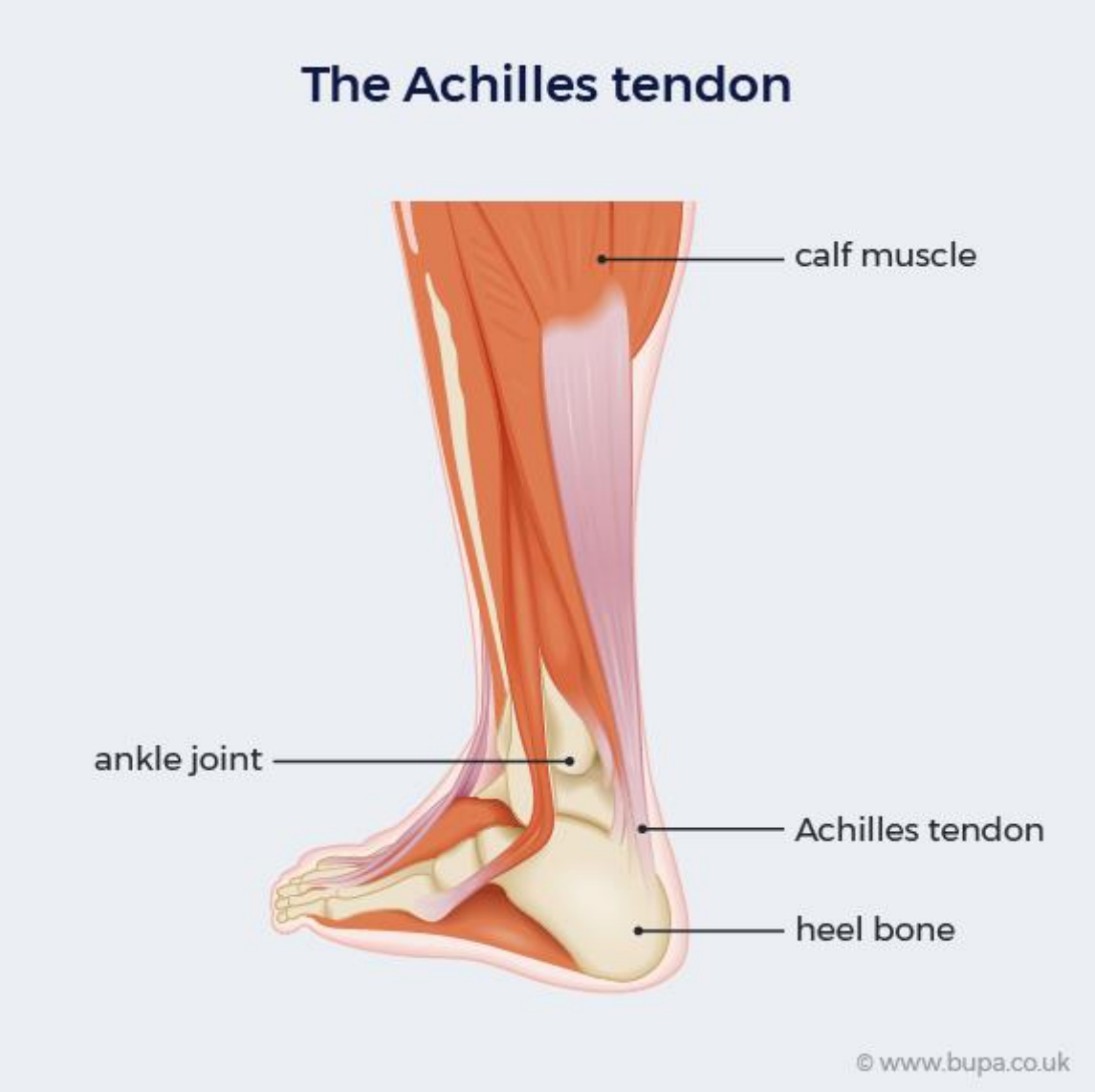


Figure 2: Schematic representation of achilles tendon[-27]

ACHILLES TENDINOPATHY: ETIOLOGY

The term "Achilles tendinopathy" refers to tendinitis or acute inflammation and tendinosis which is referred as chronic inflammation. Achilles tendinopathy will be characterized by pain, inflammation and Achilles tendon stiffness.

- **Intrinsic factors:** Anatomic variations, age, sex, metabolic dysfunction, foot cavity, dysmetria, muscle weakness, imbalance, gastrocnemius dysfunction, anatomical variation of the plantaris muscle, tendon vascularization, torsion of the Achilles tendons, slippage of the fascicle and lateral instability of the ankle.
- **Extrinsic factors:** These include mechanical overload, constant effort, inadequate equipment, obesity, medications such as corticosteroids, anabolic steroids, fluoroquinolones, improper footwear, insufficient warming or stretching, hard training surfaces, and direct trauma, among others. [28-30]

DIABETES MELLITUS

Diabetes mellitus is derived from the Greek word *diabetes* --- siphon - to pass through and the Latin word *mellitus* meaning sweet. The term "diabetes" was first used by the scientific scholar **Apollonius of Memphis** around 250 to 300 BC. Ancient Greek, Indian, and Egyptian civilizations discovered the sweet nature of urine was the primary condition and hence the propagation of the word Diabetes Mellitus came into being. [31]

Later they analysed that the Diabetes mellitus (DM) is a metabolic disease, involving inappropriately elevated blood glucose levels.

Type 2 Diabetes Mellitus

Type 2 diabetes mellitus (T2DM) accounts for around 90% of all cases of diabetes which is predominant among those aged more than 45 years. Most common pathology is insulin resistance.

PATHOPHYSIOLOGY OF TYPE 2 DM

Type 2 Diabetes Mellitus (T2DM) is one of the most common metabolic disorders worldwide and its development is primarily caused by a combination of two main factors: defective insulin secretion by pancreatic β-cells and the inability of insulin-sensitive tissues to respond to insulin

Major risk factors for the development of Type 2DM are;[31,32]

- Genetic predisposition
- Family history of diabetes
- Obesity
- Lower physical activity/ Sedentary lifestyle

PATHOLOGY DUE TO IMPAIRED INSULIN SECRETION[31-34]

As we all know, β-cells are responsible for insulin production, which is synthesized as pre-proinsulin. In the maturation process, pre-proinsulin undergoes a conformational modification carried out with the help of several proteins in the endoplasmic reticulum (ER) to yield proinsulin.

Proinsulin is translocated from the ER to the Golgi apparatus (GA), entering into immature secretory vesicles and being cleaved into C-peptide and insulin. Once matured, insulin is stored in granules until insulin release is triggered. Insulin release is primarily triggered by a response to high glucose concentrations.

When circulating glucose levels increase, β-cells take in glucose mainly through the glucose transporter 2 (GLUT2), a solute carrier protein that also works as a glucose sensor for β-cells. Once glucose enters, glucose catabolism is activated, increasing the intracellular ATP/ADP ratio, which induces the closing of ATP-dependant potassium channels in the plasma membrane.

This leads to membrane depolarization and opening of the voltage dependant Ca^{2+} channels, enabling Ca^{2+} to enter the cell. The rise in the intracellular Ca^{2+} concentration triggers the priming and fusion of the secretory insulin-containing granules to the plasma membrane, resulting in insulin exocytosis.[35]

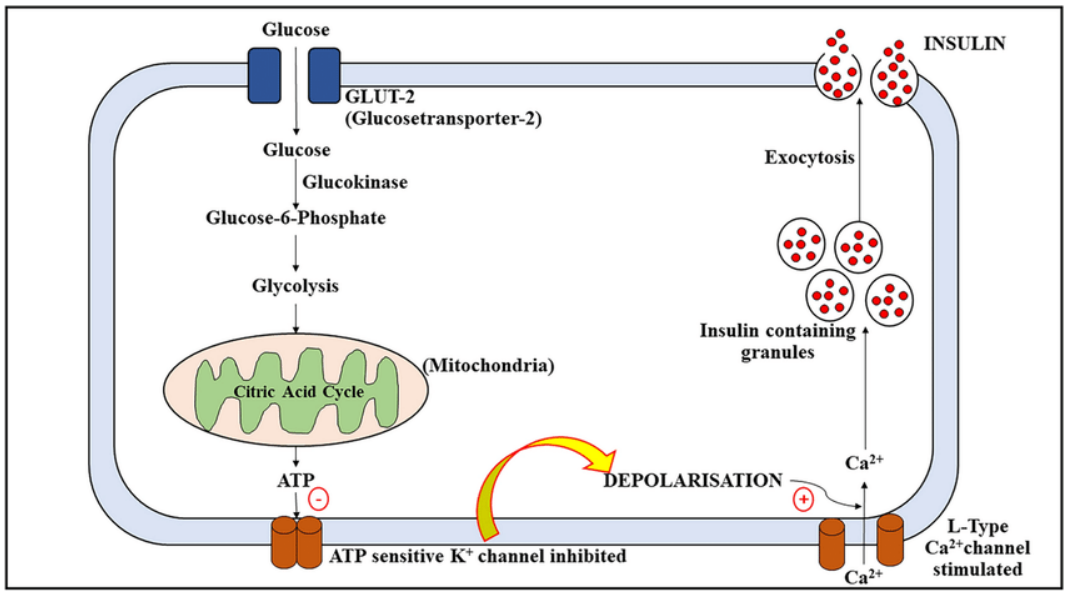


Figure 3: Schematic representation of pathophysiology of type 2 diabetes: Insulin overview

DISEASE STATEMENT

In 2021, the estimated age-adjusted prevalence of diabetes mellitus was 10.6% (95% CI 9.2–12.0%) in China and 9.6% (95% CI 8.5–10.6%) in India, accounting for 145 million and 74 million people, respectively, or 41% of the global number of adults with diabetes

mellitus. The large burden of diabetes mellitus in India and China has been shaped by various behavioural, geographical, socioeconomic and cultural factors in each country.[36]

ASSOCIATED MUSCULOSKELETAL DISEASE CONDITIONS OF CHRONIC DIABETES

Diabetes associated with a multitude of chronic impairments related to micro- and macroangiopathic involvement and polyneuropathy. These comprise cardiovascular complications, stroke, retinopathy, nephropathy, adhesive capsulitis, Dupuytren's contracture, crystal-induced arthritis, and plantar fasciitis (PF). Neuropathic complications or previous foot ulcers are associated with an increased prevalence of PF in diabetic patients.[37]

PF and AT are the most frequent causes of activity-enhanced foot pain in the adult population. The overall prevalence of this local, non-infectious inflammation is estimated to be 10%. [38]

PATHOGENESIS OF FASCIITIS AND TENDINOPATHY AMONG DIABETIC PATIENTS

Some research groups favour a degenerative origin. PF is characterized primarily by degeneration of the plantar fascia as a result of repetitive micro-tears that give rise to a local inflammatory response without systemic repercussions. Shortening of the gastrocnemius-soleus complex may play a crucial role in the development of PF. Other research groups have stressed the apparent involvement of metabolic factors in the pathogenesis of PF, along with mechanical overuse. This could particularly be the case in DM patients. In this view, PF in DM foot is linked to advanced glycation end-products (AGEs).[38,39]

Indeed, AGEs induce collagen crosslinking, ultimately leading to altered collagen structures and secondary mechanical dysfunction. In vitro, glycation disrupts the organization of collagen, resulting in an irregular fibril density and morphology. Electron microscopy has revealed a reduced density of tenocytes and fibroblasts along with an increased density of collagen. Of note, AGEs are increased in DM. Their presence has been directly associated with increased plantar fascial or Achilles tendon thickness in diabetic subjects; in contrast to non-diabetic subjects.

Hyperglycemia itself can modify redox homeostasis, particularly the polyol pathway, thereby leading to cellular edema. DM is also associated with decreased tendon neovascularization. The density of capillaries per unit of surface area is reduced, thereby leading to reduced blood flow.

This alteration could reduce vessel and nerve growth. The addition of a sensitive neuropathy and reduced nerve ingrowth decrease the distress signals and ultimately re-promote tendon overuse and damage. Distal polyneuropathy (DPN) is common in diabetic patients, with a prevalence of at least 50%. DPN is defined as a loss of sensitivity beginning distally in the lower extremities that may also be characterized by pain and significant morbidity. Half of the patients are asymptomatic and therefore remain at high risk for insensate lesions to their feet. Assessment of diabetic patients at least once a year for the presence and severity of DPN is recommended.

This can be performed using a graduated tuning fork (128 Hz), 10-g monofilament testing, or by the ankle jerk reflex. In the presence of DPN, other causes of neuropathy must be excluded, including vitamin deficiency, renal failure, or thyroid disorders. The precise contribution of DPN in the development of PF has yet to be elucidated as the

available data have remained limited. Studies assessing the association have indicated that PF is independent of the presence of moderate to severe DPN.

Obesity contributes by promoting the inflammation and structural alterations due to increased adipokines, lipocalin-1, serum amyloid A-3, and adiponectin, can disrupt the cellular functions of chondrocytes and tenocytes thereby potentially leading to tendon disruption. [38-40]

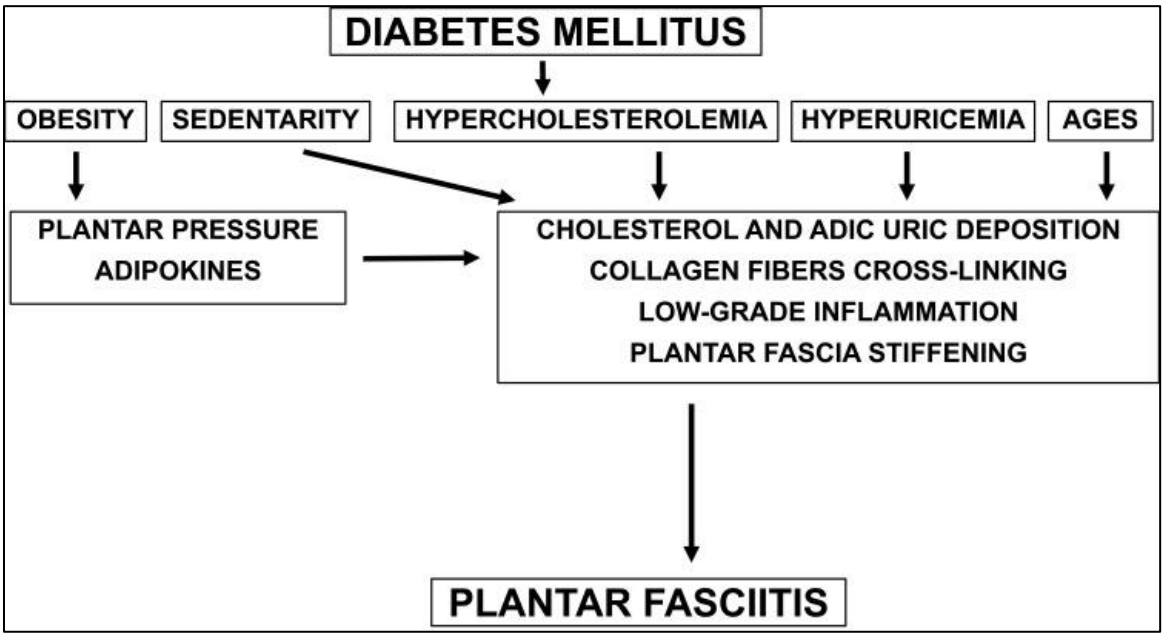


Figure 4: Image illustrating the pathophysiology of plantar fasciitis in diabetic patients[38]

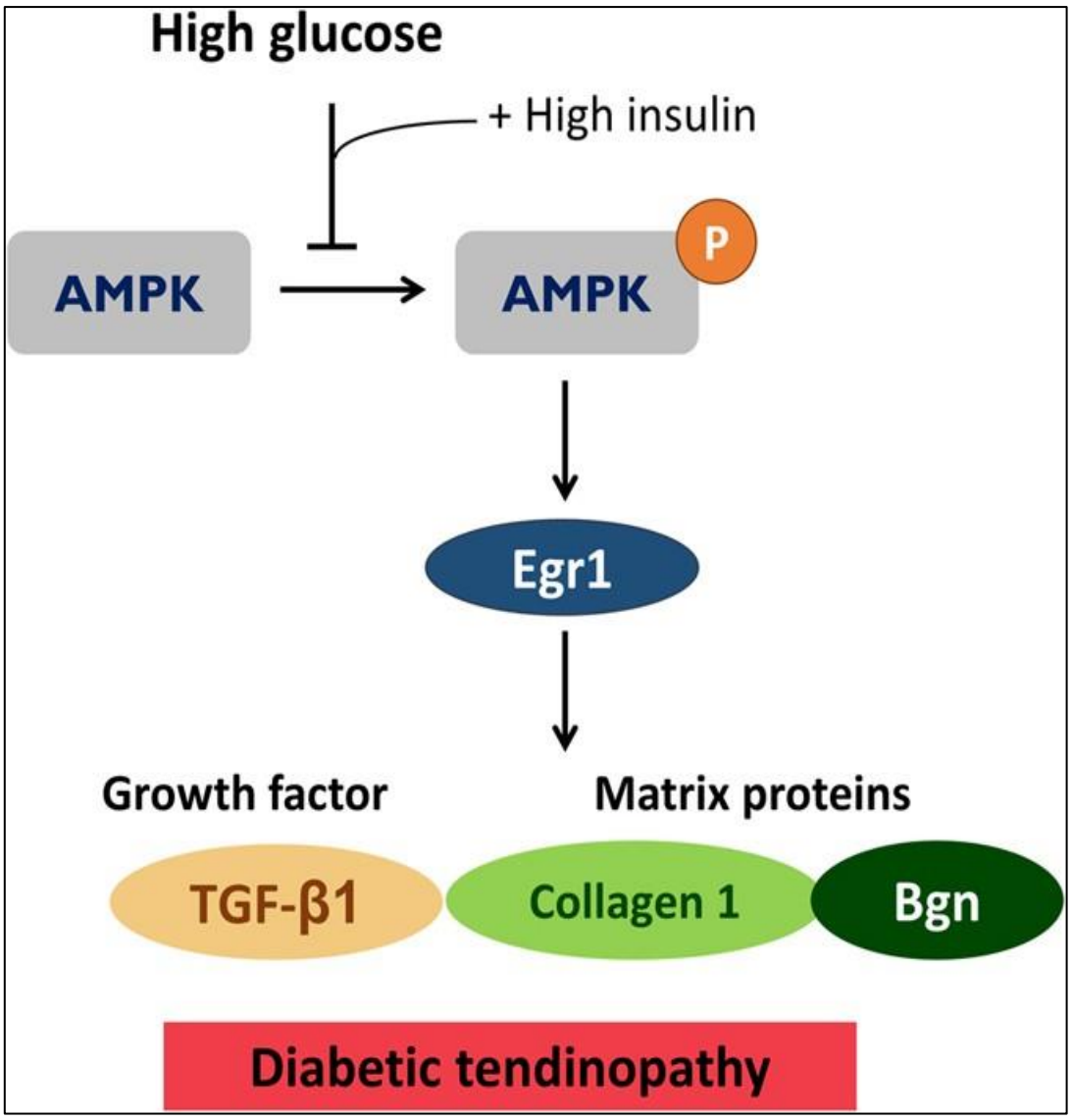


Figure 5: Pathophysiology of tendinopathy among diabetics[41]

The above image describes that the expression of Egr1 by inactivating AMPK signalling pathway will be resulting in the further suppression of downstream tendon-related genes, including TGF-β1, type 1 collagen, and other metalloproteins. The accumulation of these will be irregular leading to conformational changes of the tendons including thickening and pain at the site. Further the pressure over this will be leading to difficulty in movements.[41]

DIAGNOSING PF AND TENDINOPATHY AMONG THE PATIENTS WITH DIABETES

PF is essentially a clinical diagnosis, relying on pain localized in the inferior heel with a distinct area of tenderness that is exacerbated by walking, while it wanes with resting. The pain usually dissipates to a certain degree over the course of the day, although it can flare up after periods of prolonged standing. Upon physical examination, passive dorsiflexion of the ankle and toes (windlass test) usually induces pain. [42]

Furthermore, physicians should actively examine the gastrocnemius-soleus complex. A lack of ankle dorsiflexion beyond 10° reflects shortening of the gastrocnemius-soleus complex, which is associated with PF. The differential diagnosis is extensive, but it typically comprises neurological causes such as progressive polyneuropathic pain; or compression of the first branch of the lateral plantar nerve as "Baxter neuropathy" . Soft tissue involvement can indicate retro-calcaneal non-infectious bursitis, Achilles tendinopathy, or plantar fascia rupture. [42,43]

Heel pain originating from the bone typically includes a history of contusion, fracture, or Haglund's syndrome, which is an entity defined by the presence of insertional Achilles tendinopathy, retrocalcaneal bursitis and calcaneal prominence which is named as Haglund deformity. [44]

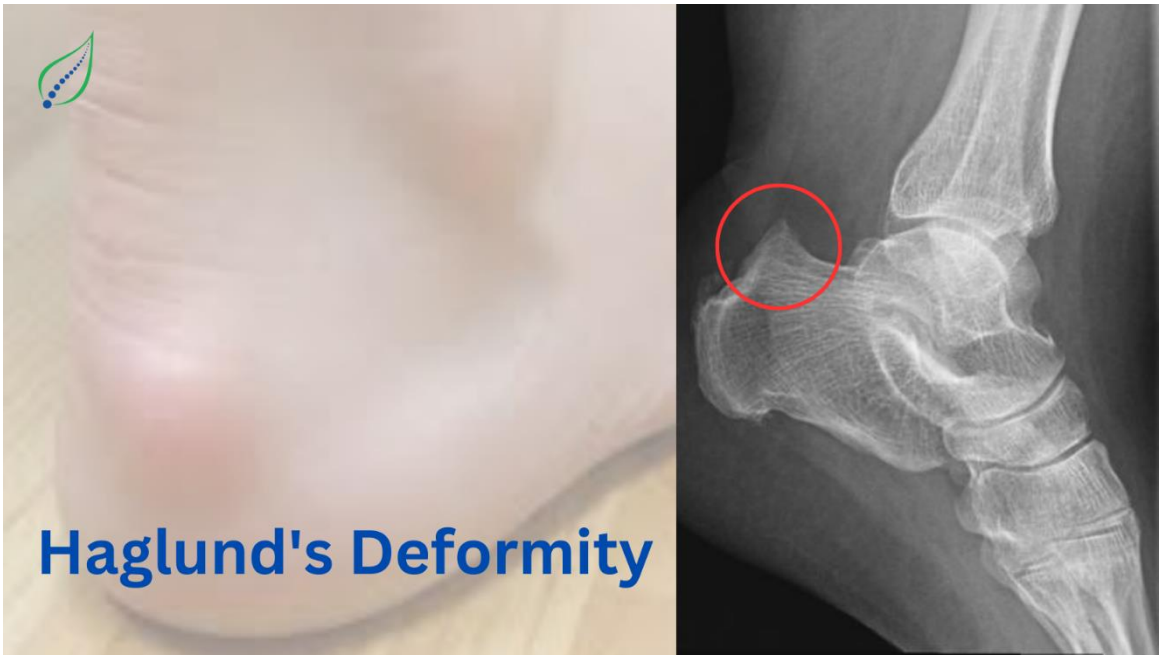


Figure 6: Illustration of Haglund's deformity[45]

As for all diabetic foot problems, an underlying osteomyelitis should be ruled out, especially if skin changes are observed, chronically ulcerated, or inflamed. By contrast, acute or chronic diabetic foot ischemia is unlikely to occur exclusively at the plantar fascia. Blood tests are not decisive, as inflammatory markers would only be increased by concomitant systemic inflammatory disease, but not by a local (mechanically triggered) inflammation. Imaging is not required for the initial assessment of PF, although it can be helpful to rule out alternative etiologies of heel pain, especially in diabetic foot.

Plain X-rays can also reveal calcaneal spurs. Although international experts have questioned the diagnostic value of calcaneal spurs for the primary diagnosis of PF, they might nonetheless be indicative of PF severity. In addition, conventional X-rays may be helpful to detect a (stress) fracture. [43]

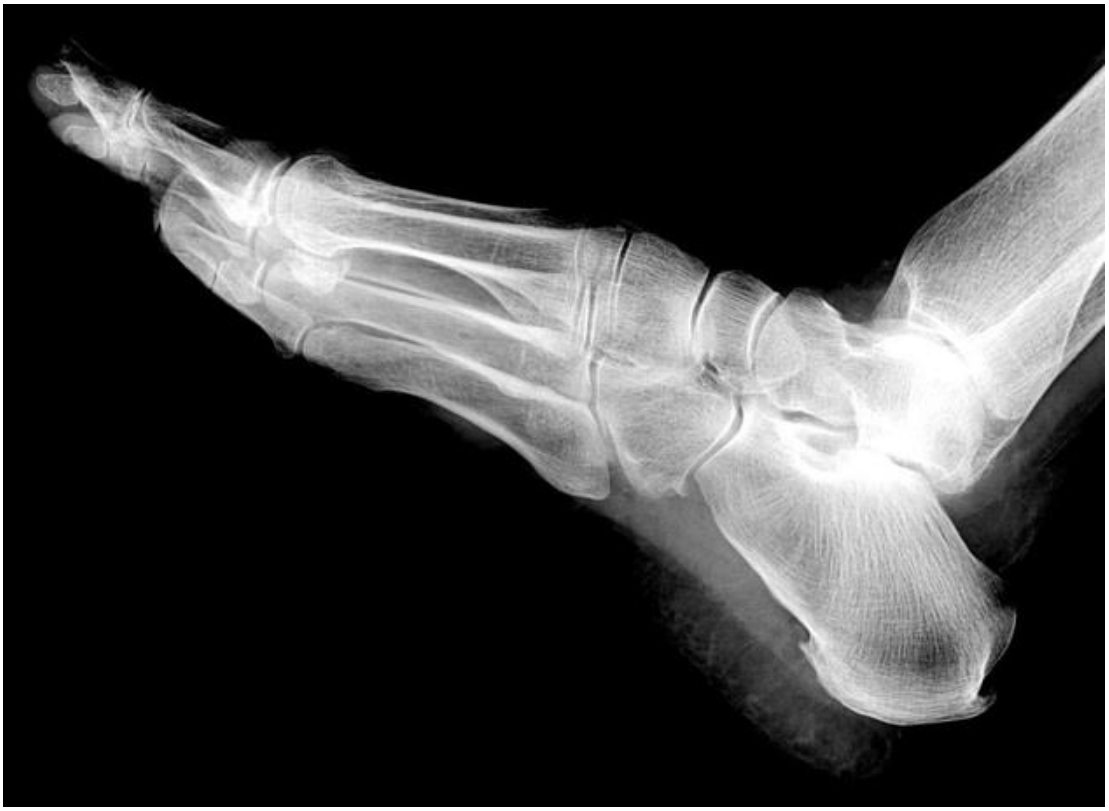


Figure 7: X ray imaging of Plantar fasciitis[45]

Sonographic findings of PF comprise fascial thickening, hypoechogenicity at the calcaneal insertions, and the loss of fibrillar structures. Ultrasound may also be valuable for distinguishing PF from diabetic foot infections or tumors. The MRI characteristics of PF include thickening of the plantar fascia and increased signal on delayed T2 sequences. Out of all of the available imaging modalities, MRI is considered to be the most sensitive technique for diagnosing PF.[45]

ULTRASONOGRAPHY AND EVOLUTION

Ultrasound implies acoustic energy with a frequency of 20,000 hertz which is above human hearing. The diagnostic sonographic scanners operate at a frequency ranging from2 to 18 megahertz. Higher frequencies have a correspondingly smaller wavelength, and can be used to make sonograms with smaller details.[46]

TISSUE INTERACTION WITH THE ULTRASOUND WAVE

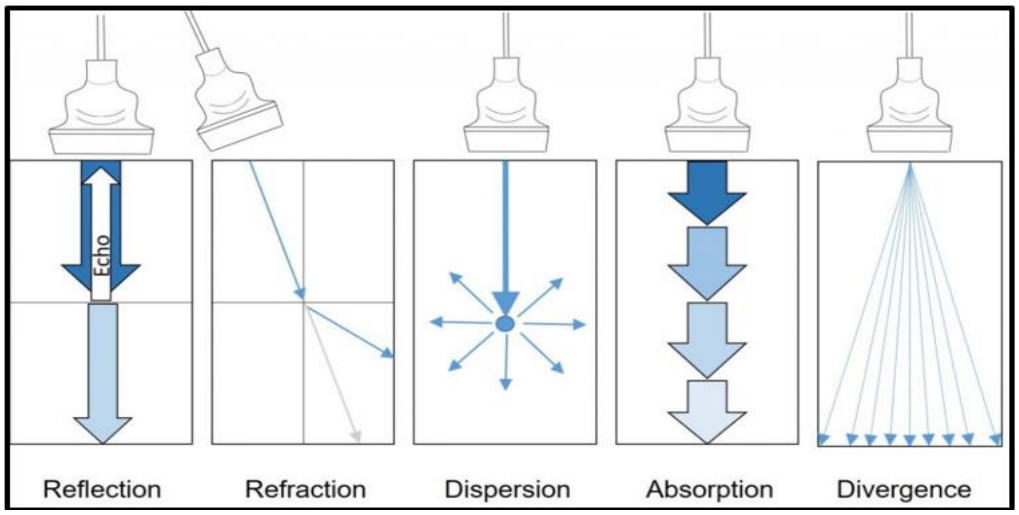


Figure 8: The ultrasound after interacting with the tissue, undergoes one of the above mechanisms based on the tissue density. Which is almost equivalent to the optical law.[48]

DIFFERENT MODES OF ULTRASOUND:

A mode: Amplification B: Brightness and M: Motion modes are the three major modes

- A-mode: A-mode is the simplest type. Single transducer scans a line through the body with the echoes plotted on screen as a function of depth. Therapeutic uses aimed at a specific tumor or calculus, to allow for pinpoint accurate focus of the destructive wave energy.
- B-mode: In B-mode ultrasound, a linear array of transducers simultaneously scans a plane through the body that can be viewed as a two-dimensional image on screen.
- M-mode: M stands for motion. In m-mode a rapid sequence of B-mode scans whose images follow each other in sequence on screen enables doctors to see and measure range of motion, as the organ boundaries that produce reflections move relative to the probe.[49]

PLANTAR FASCIA ON USG

The PF consists of three bundles: central, lateral and medial. The central component is proximally thick and distally thin and is the thickest of the three. It arises from the medial tubercle of the calcaneus and extends distally becoming broader and covering the plantar surface of the flexor digitorum brevis muscle. Distally, it divides into five digitations that insert into the metatarsophalangeal joints.

The lateral portion is also proximally thick and distally thin. It arises from the lateral margin of the medial calcaneal tubercle, covers the plantar surface of the abductor digiti minimi muscle and inserts into the fifth metatarsal joint capsule. The medial portion is thinner than the others. It arises from the midportion of the central bundle, covers the plantar surface of the abductor hallucis muscle and inserts into the first metatarsal joint capsule.

The mean maximal thickness of the PF has been reported as 4.0 mm in its central bundle, 2.3 mm in its lateral bundle and 0.6 mm in its medial bundle. Overall, PF thickness is greater in men than in women. Below is the USG imaging illustrating the plantar fascia.[50-52]

IMAGING FOR OF PLANTAR FASCITIIS

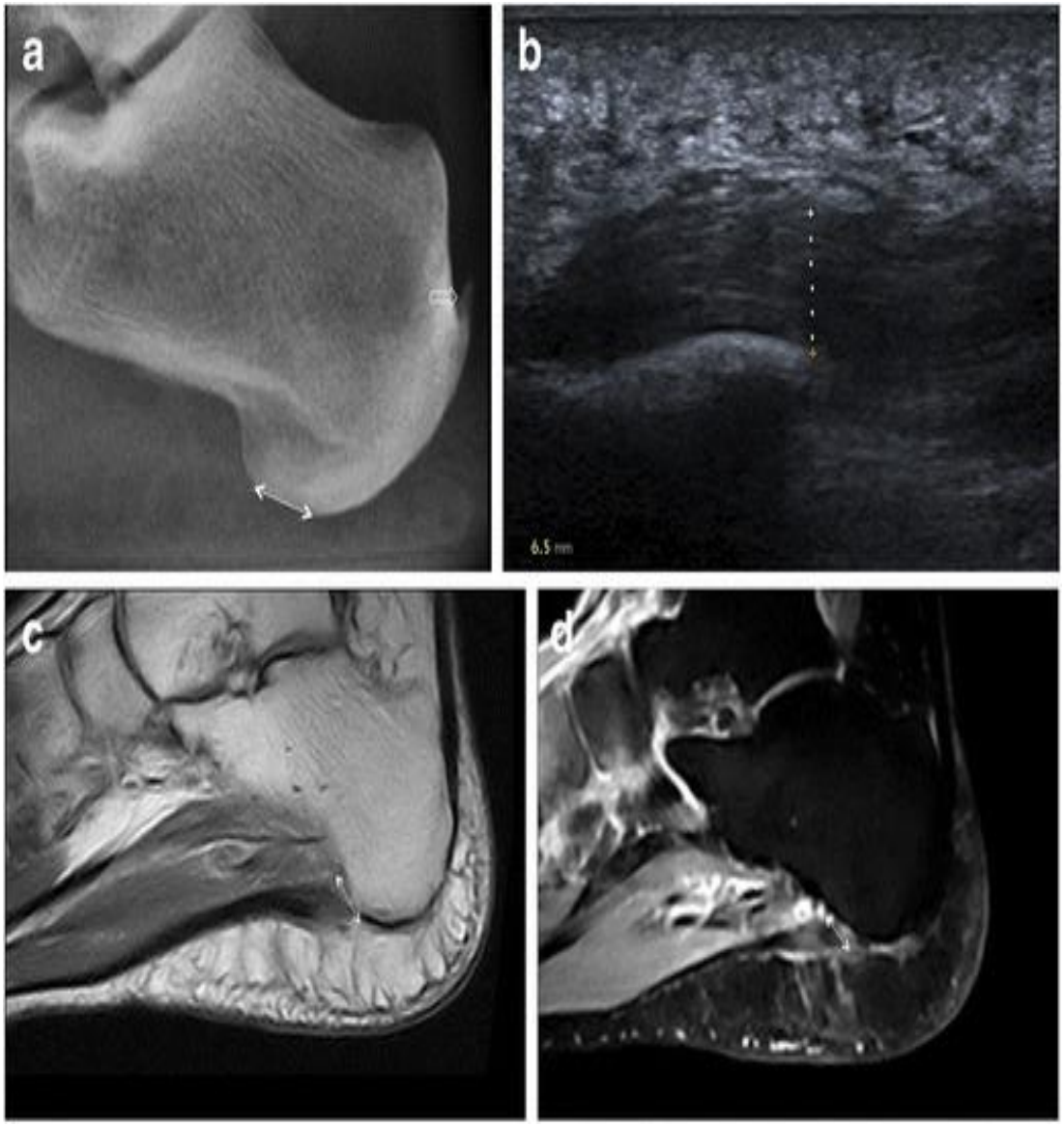


Figure 9: Plantar fasciitis on imaging.

Lateral plain radiograph highlights an increase in the distance between subcutaneous fat and intrinsic muscles of the foot at the calcaneal insertion of the PF as an indirect sign of plantar fasciitis (double-head arrow); calcific enthesopathy of the Achilles tendon is also seen (open arrow) (a). On ultrasound, plantar fasciitis presents with PF thickening (dashed line, 6.5 mm), a hypoechoic appearance and loss of fibrillar pattern (b). MRI confirms thickening of the PF at its calcaneal origin (double-head arrow) with intrasubstance areas

of intermediate and high signal intensity on T1-weighted (c) and fluid-sensitive (d) images, respectively

A Clinical Grading System To Direct Treatment When Using Diagnostic Ultrasound To Evaluate Plantar Fascia Thickness

Barrett Plantar Fasciopathy Ultrasound Grading System					
Plantar Fascia Thickness (mm)					
		I	II	III	IV
	A	<4 mm	4 mm – 5.5 mm	5.5 mm – 7.5 mm	
		None or Mild	None or Mild	None or Mild	
	B	<4 mm*	4 mm – 5.5 mm	5.5 mm – 7.5 mm	>7.5 mm
		Moderate	Moderate	Moderate	Moderate
	C		4 mm – 5.5 mm	5.5 mm– 7.5 mm	>7.5 mm
			Severe	Severe	Severe
Staging*					
Stage 1	Symptoms present <6 months				
Stage 2	Symptoms present <2 years				
Stage 3	Symptoms present >2 years				
Treatment Plan					
	Conservative care				
	Non-invasive intervention				
	Aggressive intervention				

Figure 10: Clinial grading system and USG grade for PF[53]

RADIOLOGY OF ACHIILES TENDON

Often shows thickening and rounding of the affected portion of the tendon. A cutoff value of 1 cm in anteroposterior diameter is usually used for diagnosis. There is also evidence of neovascularization, which, if present, is usually indicative of a poorer outcome and more severe clinical symptoms.

Additional signs are like increased Kager fat pad echogenicity which is common in chronic tendinopathy and thickening of a hypoechoic paratenon. Anterio-posterior diameter, structural variations and the vascularisation will be observed while conducting the USG for Achilles tendinopathy.[54]

Victorian Institute of Sports Assessment (VISA-A) is the commonly used grading system to analyse the Achilles tendinopathy which includes the components of evaluating pain, function and effect on activity of AT. score below 60 is usually found in AT patients while healthy individuals will be in the range above 95. Attaining a VISA-A score over 90 could be considered full recovery from AT.[55]

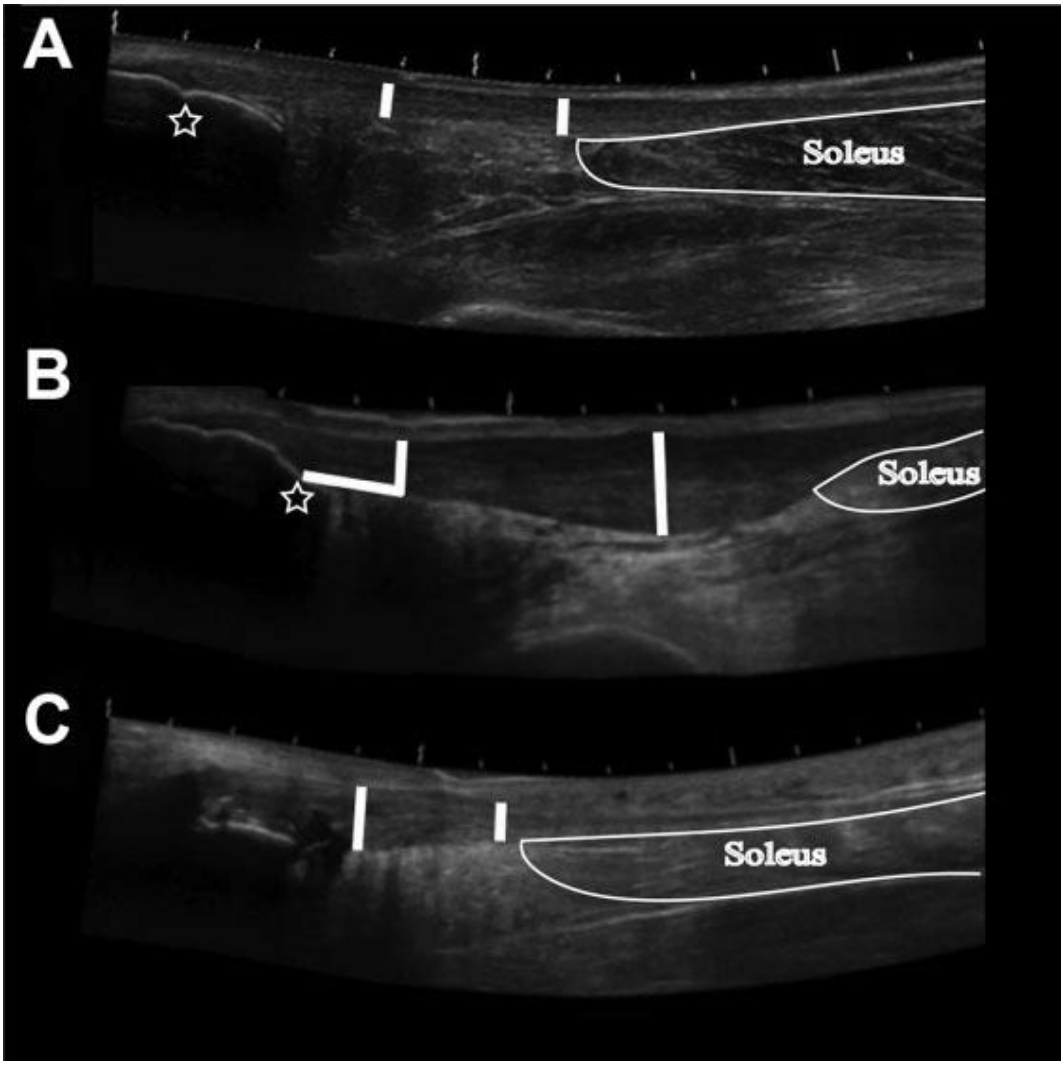


Figure 11: Representative ultrasound images for measuring Achilles tendon thickening by Corrigan P et al. [56]

In the above evidence, the thickening was calculated as: (Thickest location - thickness at the reference location).

(A) Shows the healthy Achilles tendon, close to zero thickness.

(B) Shows the Midportion Achilles tendinopathy: 2 cm proximal to the osteotendinous junction.

(C) Indicates at Insertional Achilles tendinopathy just immediate to distal to the soleus myotendinous junction.

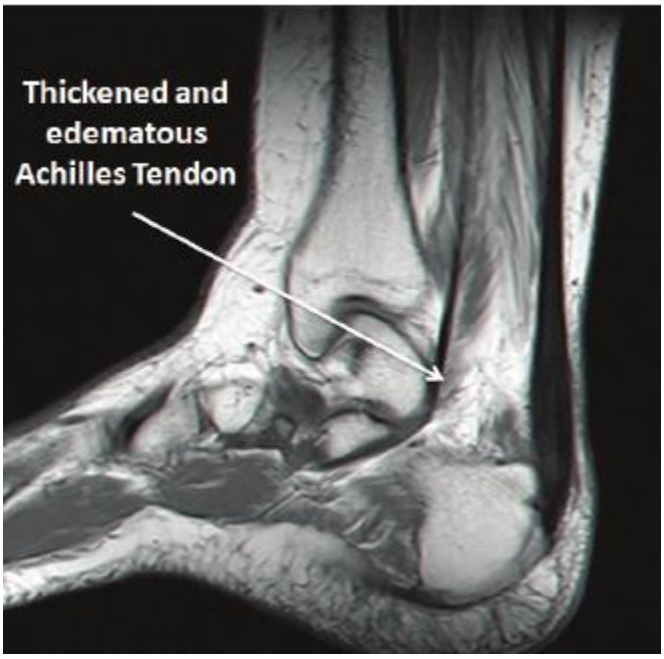


Figure 12: MRI showing thickened and edematous Achilles ten- don.[57]

Vascularisation grading system	Imaging pattern
Grade I	One vessel into the tendon
Grade II	Two vessels into the tendon
Grade III	Neovascularisation involving less than 50 % of tendon thickness
Grade IV	Neovascularisation involving from 50 % to 90 % of tendon thickness
Grade V	Neovascularisation involving more than 90 % of tendon thickness

Figure 13: Grading of tendinopathy[58]

REAL TIME SONOELASTOGRAPHY for PLANTAR FASCIA AND ACHIILES TENDON

Sonoelastography was performed by applying gentle compression with the hand-held transducer on the heel. The force applied to the plantar fascia was adjusted according to the quality factor set on the machine and it was displayed on the screen. A quality factor equal to or greater than 60 indicated the optimal compression force. During the examinations, the B-mode image and the elastogram were displayed concurrently side by side on the screen.

The elastogram appeared within a rectangular region of interest as a color-coded image superimposed over the B-mode image. The color represented the relative stiffness of the tissues within the region of interest and ranged from red (hard) to blue (soft) in a continuous spectrum. Thus, green indicated medium stiffness. During the examination, the right half of the screen, which showed the elastogram, was covered to avoid intentional influence by the examiner.

Elastogram images chosen for the analysis were those obtained with B-mode sonography that were of good quality (plantar fascia was horizontal and with clear upper and lower borders) and with the quality factor greater than 60. Three images of each plantar fascia were recorded and all of them were sent to picture archiving and communication system for imaging analysis. Twelve plantar fasciae of six non- selected volunteers were re-evaluated within 3 days of testing by the original physiatrist and another physiatrist (S.M.). These data were then used to analyze intrarater and interrater reliability for imaging analysis of sonoelastography.[59-61]

Saroha A et al (2023) had analysed 55 diabetic patients and 55 healthy volunteers for the changes in Achilles tendon and plantar fascia on USG elastography. They observed

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.[62]

Dixit R et al was another cross-sectional study (2020), had conducted sonoelastographic on 61 healthy volunteers and 81 patients with type 2 DM. They observed that The AT thickness was measured in the proximal, middle and distal portions. Alterations in echo pattern were noted. The patients were found to have thicker tendons than the healthy volunteers ($p < 0.01$). Alterations in the echo pattern of the AT were more common in patients compared with healthy volunteers ($p < 0.01$).[60]

The shear wave velocity in the distal one-third of the AT was measured using shear wave elasticity imaging. Mean shear wave velocity values obtained were lower in patients compared with healthy volunteers ($p < 0.001$). No significant difference was found in the sonoelastographic findings of the AT in patients with and without PN. We conclude that there is softening, thickening and alterations in echo pattern of the AT in the form of hypoechogenicity, loss of fibrillar pattern and calcification at insertion in patients with type 2 DM, and these alterations could occur independent of onset of PN.[60]

Similarly, Umelo DO et al (2021) found that the mean ATT was higher in diabetic subjects compared to the control subjects: 5.56 ± 0.65mm and 5.59 ± 0.61mm among case group in right and left foot. Whereas among the healthy population, it was 4.72 ± 0.44mm and 4.77 ± 0.40mm in right and left foot respectively with significant p value of <0.0001.

The optimal cut-off point of ATT for identifying the risk of PN in the feet of diabetics was determined to be > 5.75mm with an accuracy of 83.3%.[63]

Even the sound touch elastography (STE) by Huang X et al, who considered Young 's modulus (E) value for analysing the fate of tendon and fascia among diabetics and compared it with control groups, had reported that E values of the three segments of ATs in T2DM patients were lower than the healthy controls ($P < 0.05$). E value is a measurement index of STE and is considered a reliable biomechanical index for reflecting soft tissue stiffness.

In both groups, the E values of the distal segments were lower than those of the middle segments, and the latter were lower than those of the proximal segments with significant P value < 0.05. The E value of each segment of AT was inversely related to FPG, HbA1c, and diabetes duration ($P < 0.05$). The best cut-off points for the E values of the three segments of the AT for detecting diabetic tendinopathy were 347.44 kPa, 441.57 kPa and 484.35 kPa, respectively.[64]

Khor BYC et al (2021) had conducted systemic analysis 35 non-randomised observational studies were suitable for inclusion. Within these, 20 studies evaluated plantar tissue thickness, 19 studies evaluated plantar tissue stiffness, 9 studies evaluated Achilles tendon thickness and 5 studies evaluated Achilles tendon stiffness outcomes. No significant differences in plantar tissue thickness were found between people with and without diabetes in 55% of studies (11/20), while significantly increased plantar tissue stiffness was found in people with diabetes in 47% of studies (9/19). Significantly increased Achilles tendon thickness was found in people with diabetes in 44% of studies (4/9), while no significant differences in Achilles tendon stiffness were found between people with and without diabetes in 60% of studies (3/5).[65]

MATERIALS AND METHODOLOGY

Source of Data: A one-year Hospital based case control study was conducted in the department of Radio-diagnosis, patients with complaints of heel pain who met the inclusion criteria, from January 2023 to December 2023 at KLE's Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study Design: Hospital based case control study

Study Period: January 2023 to December 2023

Sample Size: The minimum sample size formula based on mean and standard deviation is

$$\mathbf{n} = \frac{(z_\alpha + z_\beta)^2(s_1^2 + s_2^2)}{(\bar{X}_1 - \bar{X}_2)^2}$$

where zα is linked with the level of significance and zβ is linked with the power of the test. For 5% level of the significance zα =1.96 and zβ =0.84 for 80% power of the test.

Ref: Sonographic Evaluation of the Achilles Tendon and Plantar Fascia of Type 2 Diabetics in Nigeria. Babalola Ishmael Afolabi1, Oluwag bemiga Oluwole Ayoola1, Bukunmi Michael Idowu1*, Babatope A. Kolawole2, Adeleye Dorcas Omisore1

The parameter considered in the calculation is left PF thickness

$\bar{X}_1$ is the mean of the first group (1.97) and $\bar{X}_2$ is the mean of the second group (1.70).

s1 is the standard deviation of the first group (0.4) and s2 is the standard deviation of the second group (0.2).

With these values the sample size obtained was 22.

To get confirmative results the sample size was increased to 40

There were two groups with 40 cases in each group.

The two groups had constituted;

Diabetics (cases) under evaluation: Group A

Non-diabetics (controls) under evaluation: Group B

Inclusion Criteria:

- Patients being referred to radio-diagnosis department for USG scan with complaints of heel pain
- Patients diagnosed with Type II Diabetes Mellitus
- Patients of age > 25 years
- Patients who give consent to take part in the study

Exclusion criteria:

- Patients diagnosed with type I Diabetes mellitus
- Patients of age < 25 years
- Patients with history of recurrent foot trauma, congenital ankle deformities, amputation involving lower limb or is a known case of rheumatoid arthritis
- Patients not willing to take part in the study

DETAILED PROCEDURE

Study was started after obtaining clearance from institutional ethics committee. Cases and controls were included based on the above mention inclusion and exclusion criterias. A written informed consent was obtained from all the study participants. A pre- structured Performa was used for collection of clinical data. A detailed history, associated risk factors such obesity, deranged lipid profile, sedentary life style and other significant parameters were noted. Height (m) and weight (kg) were noted to calculate the BMI by dividing their weight in kilograms by square of their height in metres. FBS (Fasting blood gluocse) and

HbA1c were noted for further correlation. The cut off used for HbA1c for diabetes > 48mmol/mol (6.5%) and FBS for diabetes > 125mg/dl.

The above-mentioned study population who met the inclusion criteria were subjected for ULTRASONOGRAPHY on GE LOGIQ P9 R2 machine equipped with a 7.5–12 MHz high frequency linear array transducer. Patients were examined in prone position with legs extended with their feet extending beyond the edge of the couch. All the studies were performed in two dimensions by scanning across the tendon & fascia by moving the probe medial to lateral and distal to proximal. Examination was done on real-time two-dimensional grey-scale and the images had been stored securely on a portable drive. The measurements taken by ultrasound was then be compared with the clinical details to look for correlation. Further groups constituting diabetics for evaluation of Achilles tendon, diabetics for evaluation of Plantar fascia, non-diabetics for evaluation of Achilles tendon and non-diabetics for evaluation of Plantar fascia had been analysed and the observed results are represented as tables and graphs below. No follow up was done or either required for this study.

EQUIPMENT: GE LOGIQ P9 R2 ULTRASONOGRAPHY MACHINE

STATISTICAL METHODS:

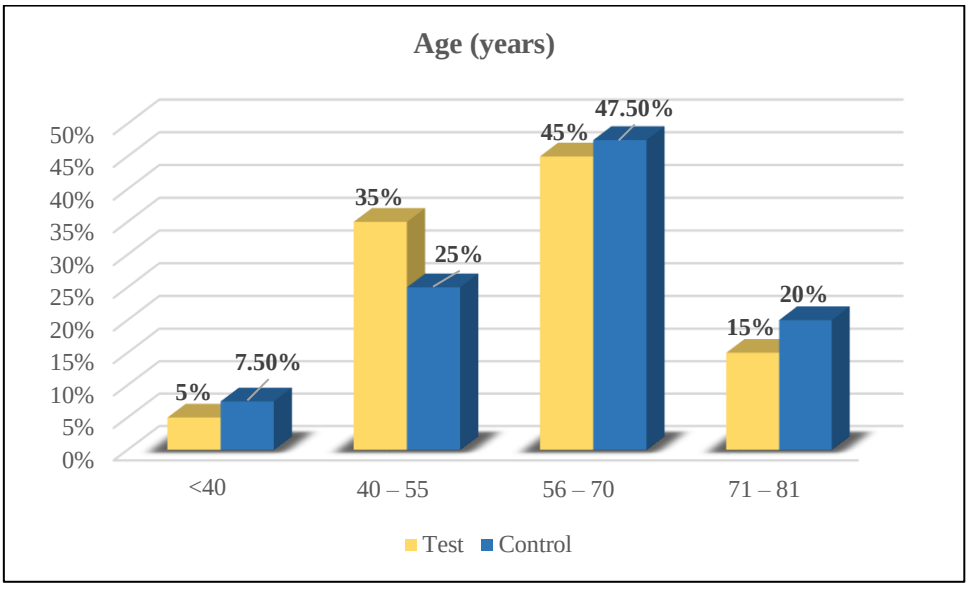
Data was analysed using SPSS software version 21 and Excel. Categorical variables were given in the form of frequency table. Continuous variables were given in Mean ± SD/ Median (Min, Max) form. Categorical variables are analysed by Chi square test. Ordinal data was analysed by Independent t test and Mann Whitney U test. Normality was analysed by Shapiro wilk test. Spearman's rho correlation coefficient was used to detect linear relationship between numerical variables. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS:

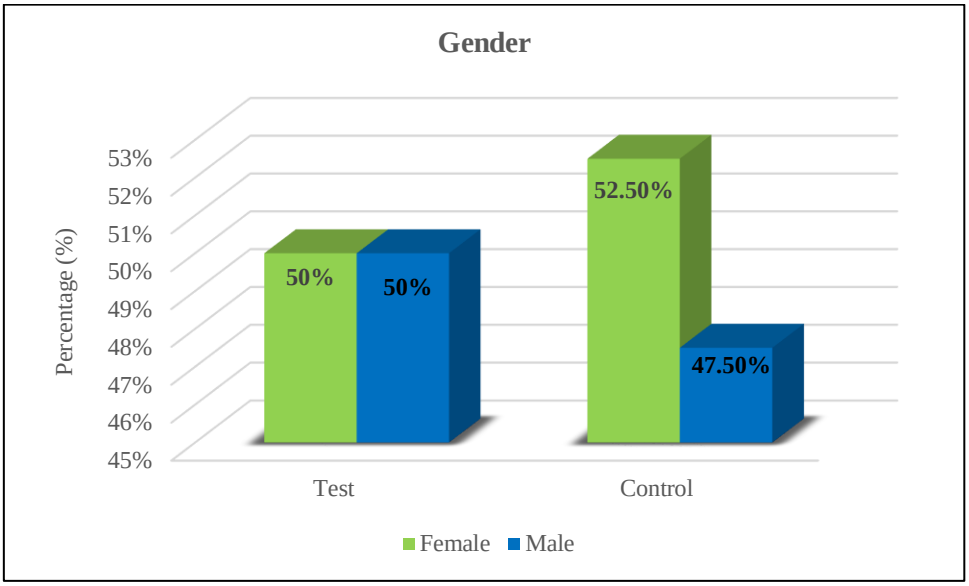
Data contains information of 40 subjects each in two group who underwent Real time Ultrasonography in evaluation of the Plantar Fascia thickness and Achilles Tendon thickness in Diabetes Mellitus Type II. The following tables give the summary of data.

Table 1: Distribution of subjects according to demographic details over groups

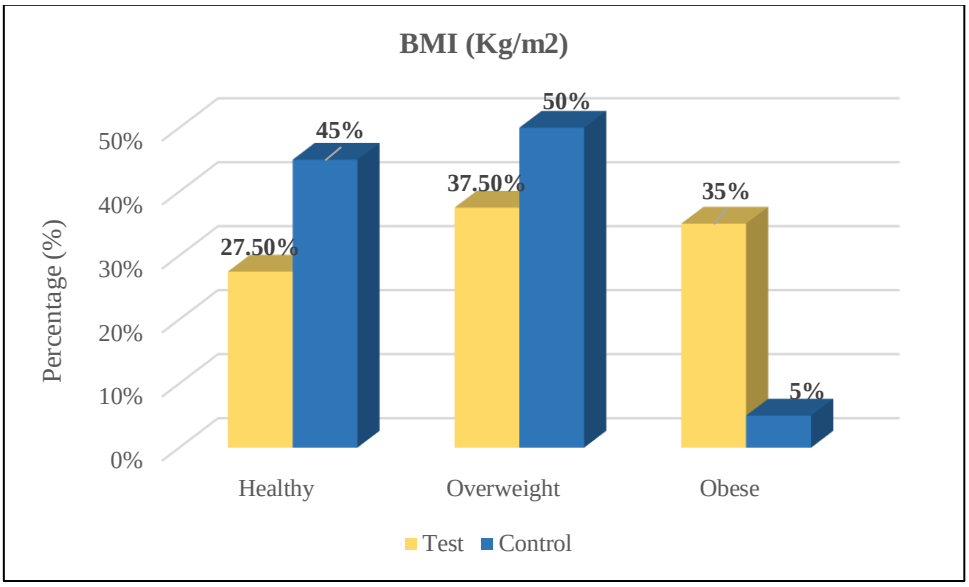
Variable	Sub Category	Groups	
		Test (A)	Control (B)
Age (years)	<40	2 (5%)	3 (7.5%)
	40 – 55	14 (35%)	10 (25%)
	56 – 70	18 (45%)	19 (47.5%)
	71 – 81	6 (15%)	8 (20%)
Gender	Female	20 (50%)	21 (52.5%)
	Male	20 (50%)	19 (47.5%)
BMI (Kg/m^2)	<24.9 Healthy	11 (27.5%)	18 (45%)
	25-29.9 overweight	15 (37.5%)	20 (50%)
	>30 Obese	14 (35%)	2 (5%)



Graph 1: Distribution of subjects based on age over groups.



Graph 2: Distribution of subjects based on gender over groups.



Graph 3: Distribution of subjects based on BMI (Kg/m^2) over groups.

Table 2: Distribution of subjects according to different variables over groups

Variable	Sub Category	Groups		p-value
		Test (A)	Control (B)	
Age (years)	Mean ± SD Median (Min, Max)	59.52 ± 11.1 60 (38, 81)	60.70 ± 11.46 62.5 (39, 80)	0.642^{t}
BMI (Kg/m^2)	Mean ± SD Median (Min, Max)	28.07 ± 3.149 29.1 (22, 32.6)	25.63 ± 2.78 25.4 (18, 30.1)	**<0.001*^{MW}**

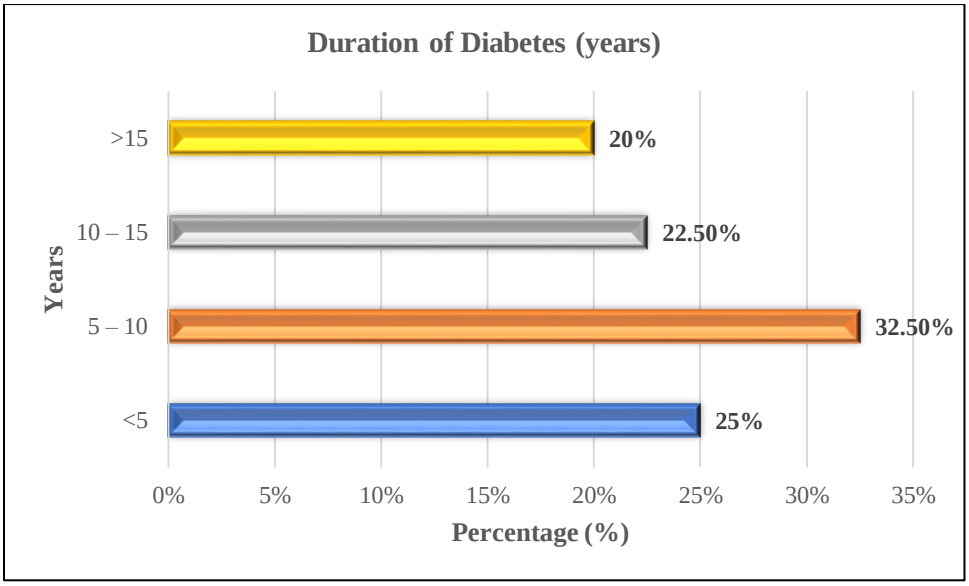
*Abbreviation: t- independent test, MW- Mann Whitney U test, *- indicates statistical significance*

From independent t test, it can be observed that, there is no significant difference in mean of age over groups. From Mann Whitney U test, it can be observed that, there is significant difference in mean of BMI over groups indicating significant positive association of BMI with diabetes.

Table 3: Distribution of subjects according to duration of diabetes (years)

Variable	Subcategory	Number of subjects (%)
Duration of diabetes (years)	<5	10 (25%)
	5 – 10	13 (32.5%)
	10 – 15	9 (22.5%)
	>15	8 (20%)

Out of 40 subjects, 13 (32.5%) of them had diabetes from 5-10 years, 10 (25%) of them had for less than 5 years.



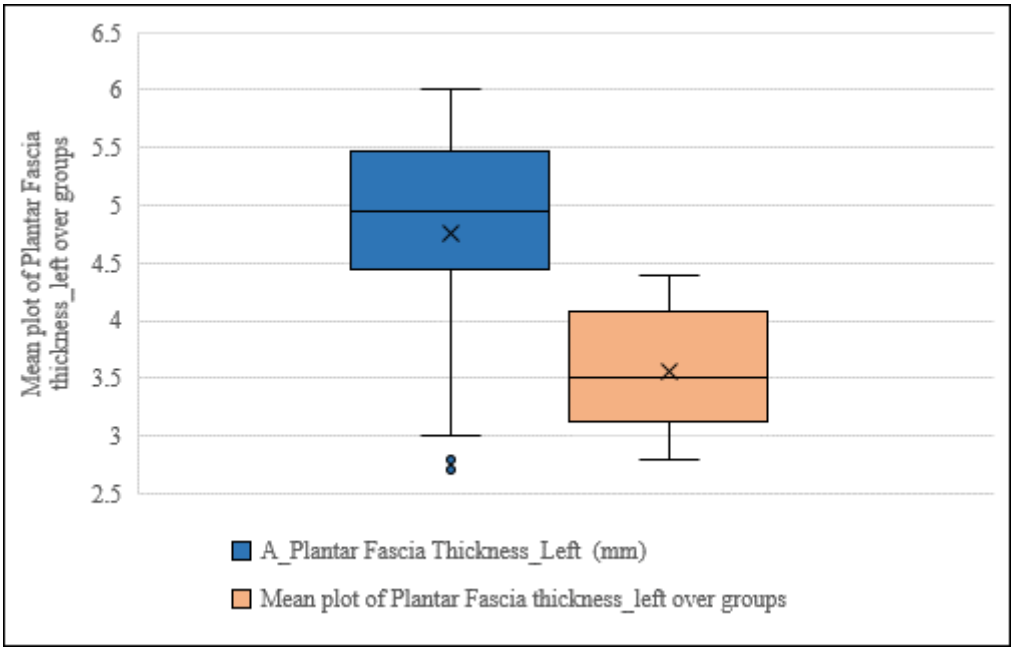
Graph 4: Distribution of subjects based on duration of diabetes (years).

Table 4: Distribution of subjects according to different variables over groups

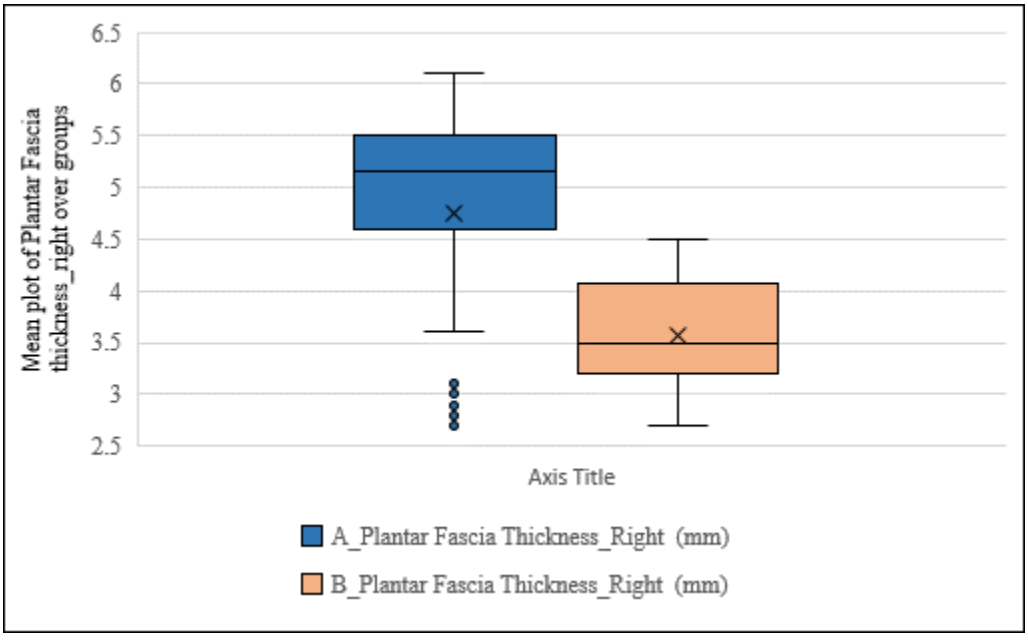
Variable	Subcategory	Groups		p-value
		Test (A)	Control (B)	
Plantar Fascia Thickness (Left) (mm)	Mean ± SD Median (Min, Max)	4.76 ± 0.974 4.95 (2.7, 6)	3.56 ± 0.514 3.5 (2.8, 4.4)	**<0.001*^{MW}**
Plantar Fascia Thickness (Right) (mm)	Mean ± SD Median (Min, Max)	4.76 ± 1.007 5.15 (2.7, 6.1)	3.56 ± 0.516 3.5 (2.7, 4.5)	**<0.001*^{MW}**
Achilles Tendon Thickness (Left) (mm)	Mean ± SD Median (Min, Max)	5.67 ± 0.881 6.1 (4, 6.7)	3.613 ± 0.515 3.55 (2.8, 4.6)	**<0.001*^{MW}**
Achilles Tendon Thickness (Right) (mm)	Mean ± SD Median (Min, Max)	5.665 ± 0.888 6 (4, 6.8)	3.615 ± 0.465 3.5 (3, 4.5)	**<0.001*^{MW}**

*Abbreviation: MW- Mann Whitney U test, *- indicates statistical significance*

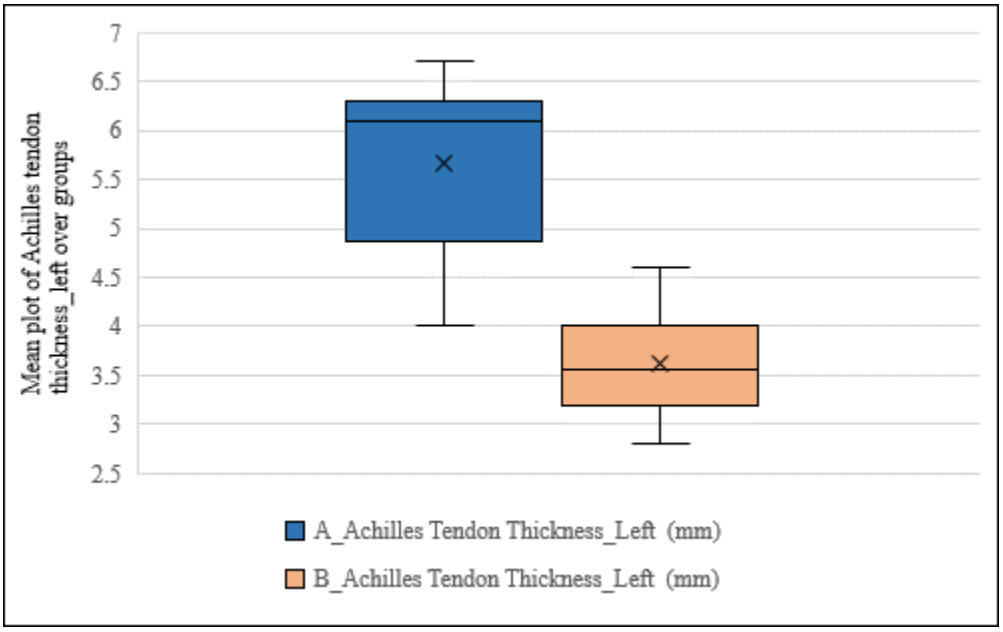
From Mann Whitney U test, it can be observed that, there is significant difference in mean of Plantar Fascia Thickness (right and left), Achilles Tendon Thickness (right and left) over groups.



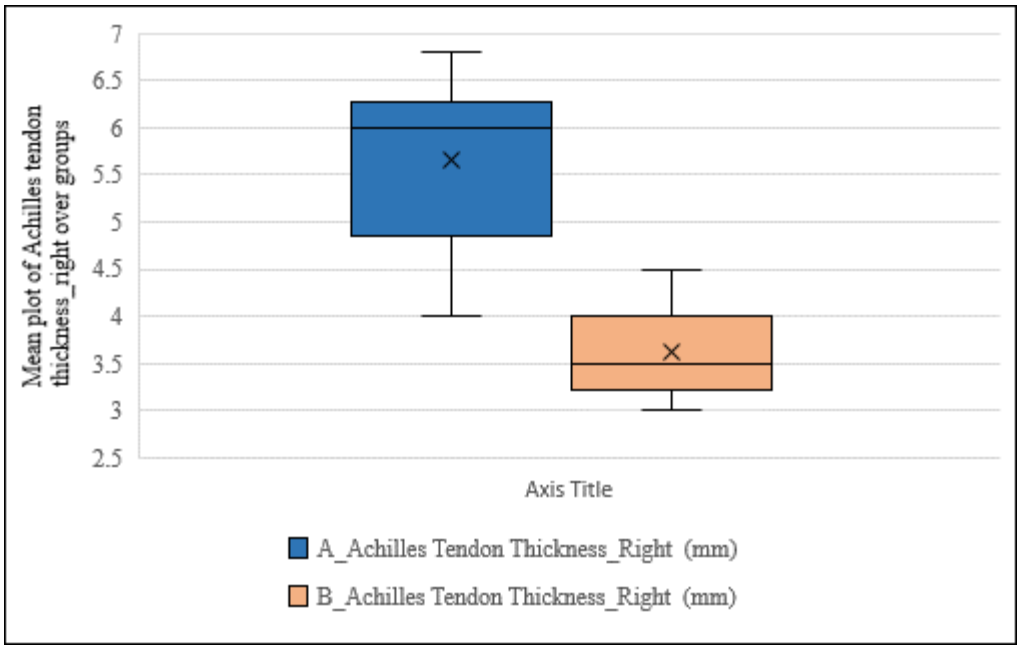
Graph 5: Mean plot of Plantar Fascia thickness of left side over groups.



Graph 6: Mean plot of Plantar Fascia thickness of right side over groups.



Graph 7: Mean plot of Achilles Tendon of left side over groups.



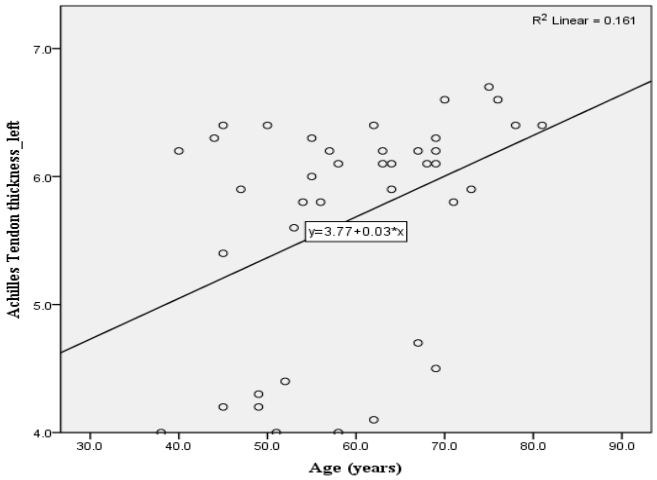
Graph 8: Mean plot of Achilles Tendon of right side over groups.

Table 5: Spearman's rho Correlation between different variables in diabetic group over PFT and AT.

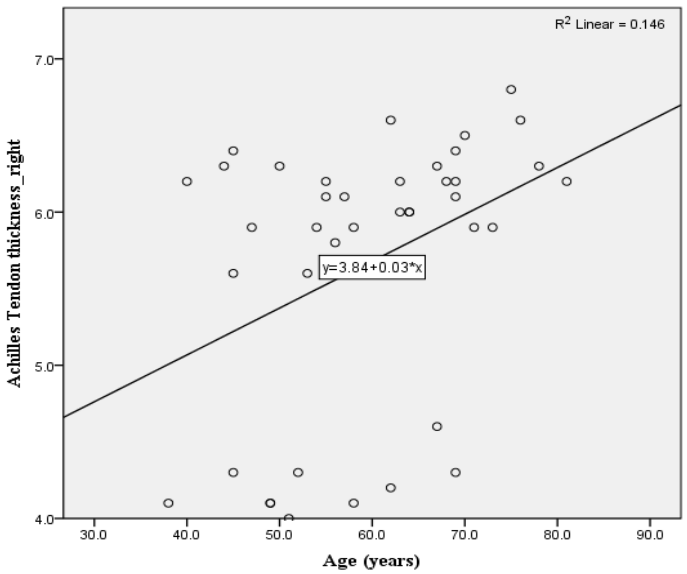
		Plantar Fascia Thickness (Left) (mm)	Plantar Fascia Thickness (Right) (mm)	Achilles Tendon Thickness (Left) (mm)	Achilles Tendon Thickness (Right) (mm)
Age	Correlation Coefficient	0.135	0.122	0.409	0.400
	p-value	0.405	0.453	**0.009**	**0.011**
	N	40	40	40	40
BMI (Kg/m2)	Correlation Coefficient	0.231	0.209	0.392	0.426
	p-value	0.151	0.195	**0.012**	**0.006**
	N	40	40	40	40
HBA1c (%)	Correlation Coefficient	-0.130	-0.124	0.322	0.266
	p-value	0.425	0.445	**0.043**	0.097
	N	40	40	40	40
Fasting Blood Glucose (mg/dl)	Correlation Coefficient	0.259	0.277	0.689	0.627
	p-value	0.107	0.083	**0.003**	**0.004**
	N	40	40	40	40
Duration Of Diabetes (Years)	Correlation Coefficient	0.335	0.312	0.603	0.585
	p-value	**0.034**	**0.050**	**<0.001**	**<0.001**
	N	40	40	40	40

From Spearman's rho Correlation, it can be observed that, statistically significant ($P < 0.05$) results were obtained among diabetic group in the correlation of Left and right AT with Age. R=0.409, 0.400 were obtained in the right AT and left AT respectively.

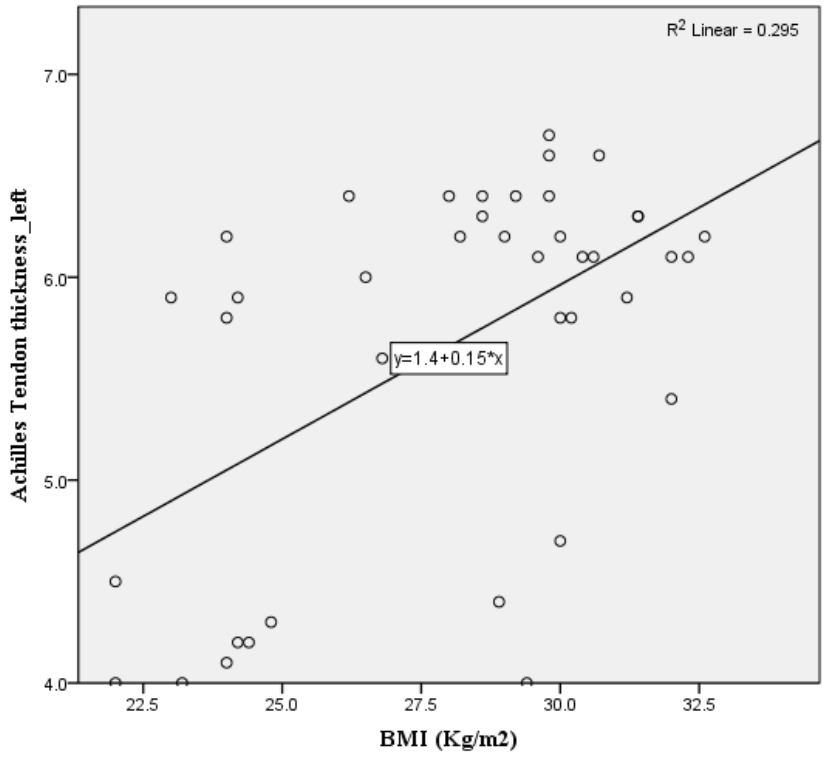
It can be observed that, statistically significant ($P < 0.05$) results were obtained among diabetic group in the correlation of Left and right AT with BMI. R=0.392, 0.426 were obtained in the right AT and left AT respectively. It can be observed that, statistically significant ($P < 0.05$) results were obtained among diabetic group in the correlation of Left and right ATT with Fasting blood glucose. R=0.689, 0.627 were obtained in the right AT and left AT respectively. The correlation was statistically significant among duration of diabetes (years) with left and right PFT and also with left and right AT.



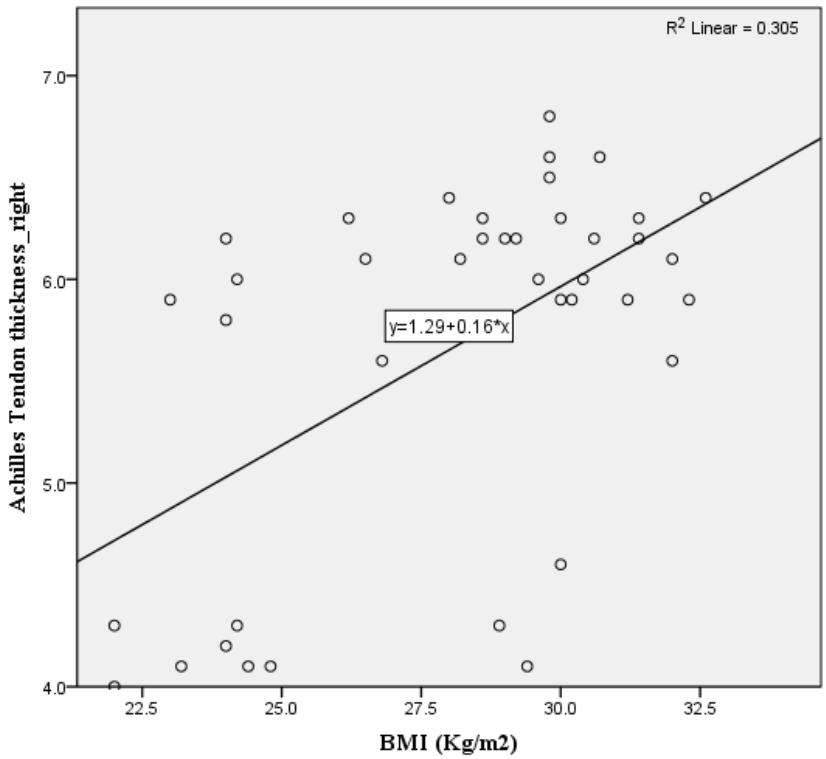
Graph 9: Scatter plot of Age (years) in diabetic group and Achilles tendon thickness on left side.



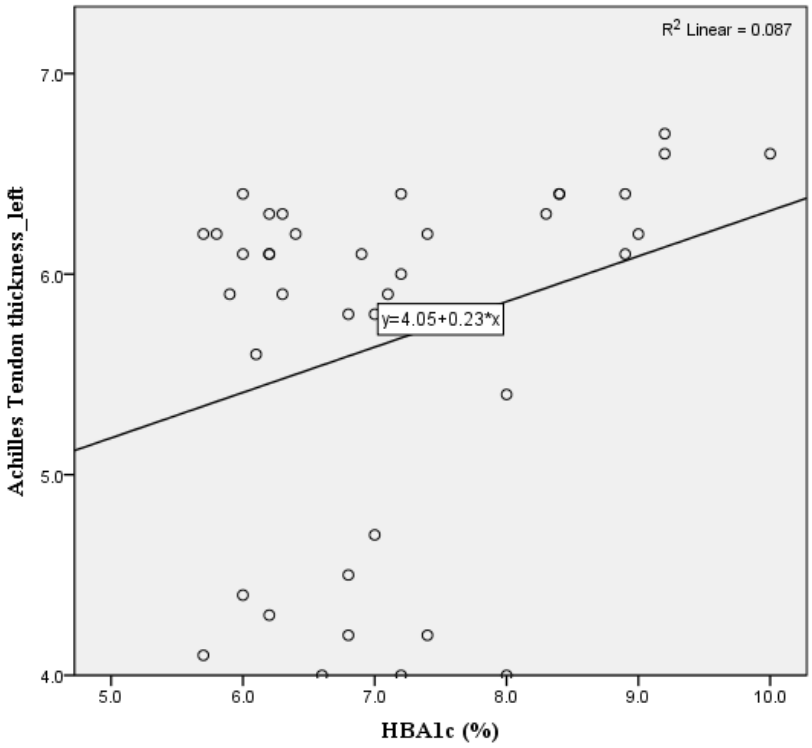
Graph 10: Scatter plot of Age (years) in diabetic group and Achilles tendon thickness on right side.



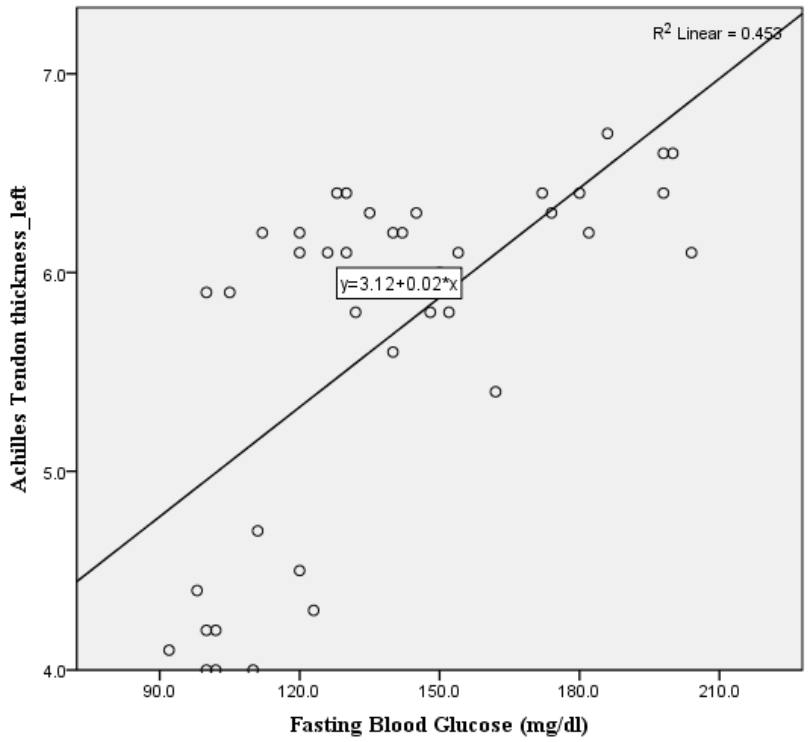
Graph 11: Scatter plot of BMI (Kg/m2) in diabetic group and Achilles tendon thickness on left side.



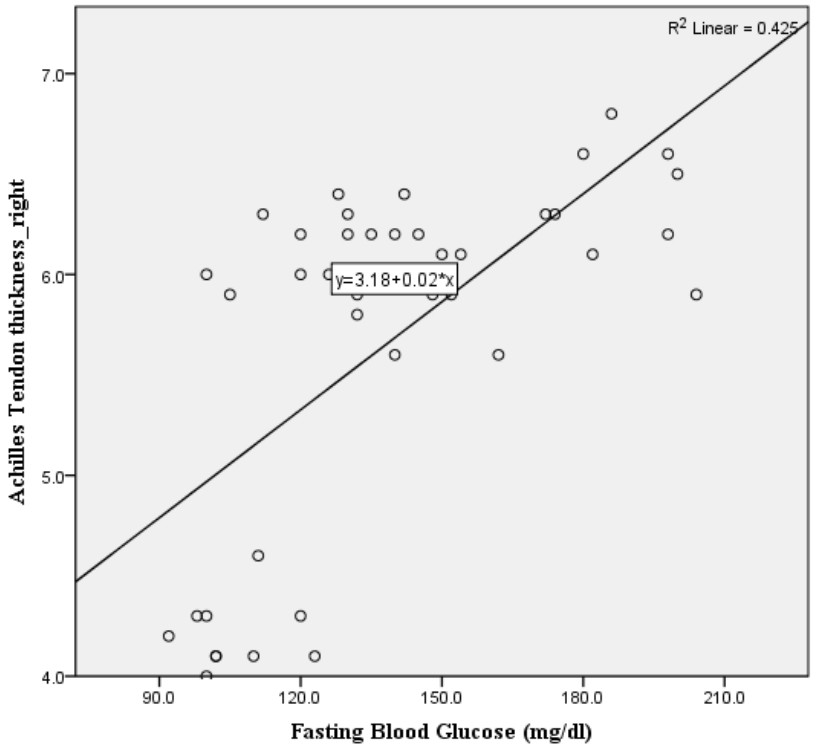
Graph 12: Scatter plot of BMI (Kg/m2) in diabetic group and Achilles tendon thickness on right side.



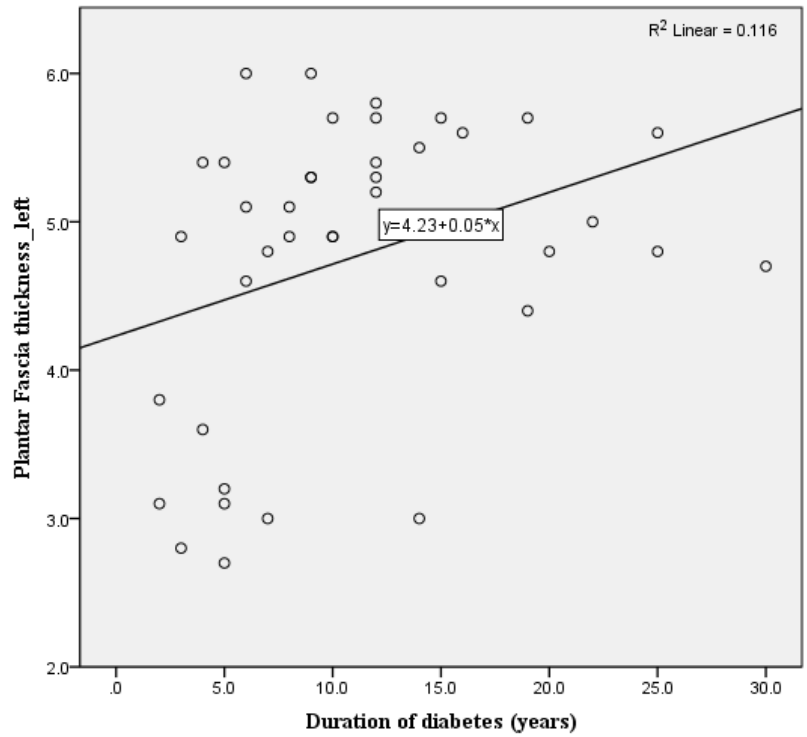
Graph 13: Scatter plot of HBA1c (%) in diabetic group and Achilles tendon thickness on left side



Graph 14: Scatter plot of fasting blood glucose (mg/dl) in diabetic group and Achilles tendon thickness on left side



Graph 15: Scatter plot of fasting blood glucose (mg/dl) in diabetic group and Achilles tendon thickness on right side

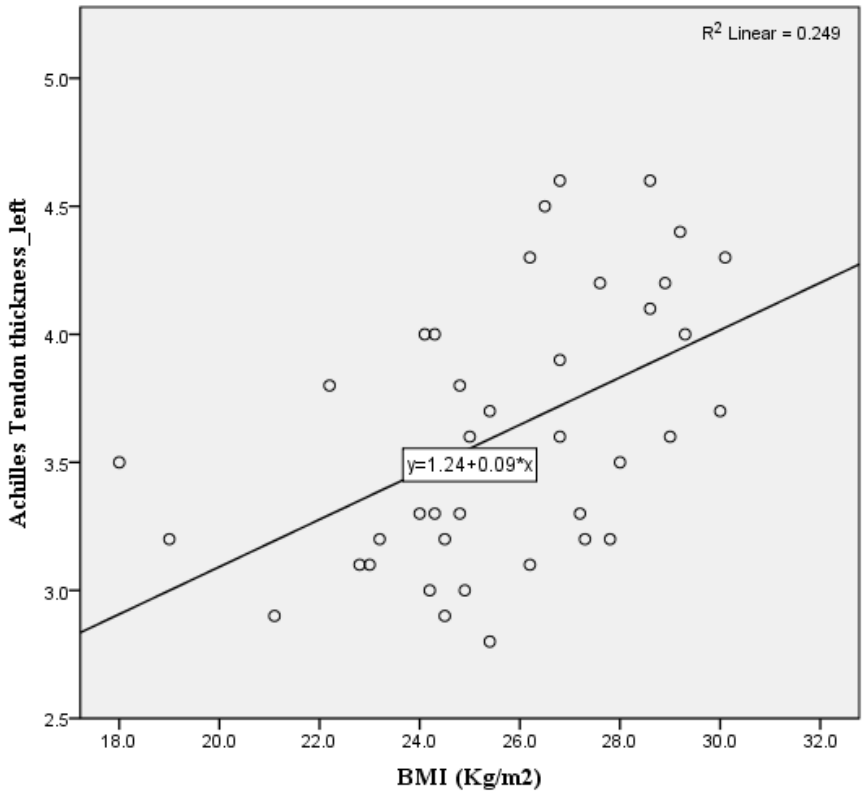


Graph 16: Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on left side in diabetic group

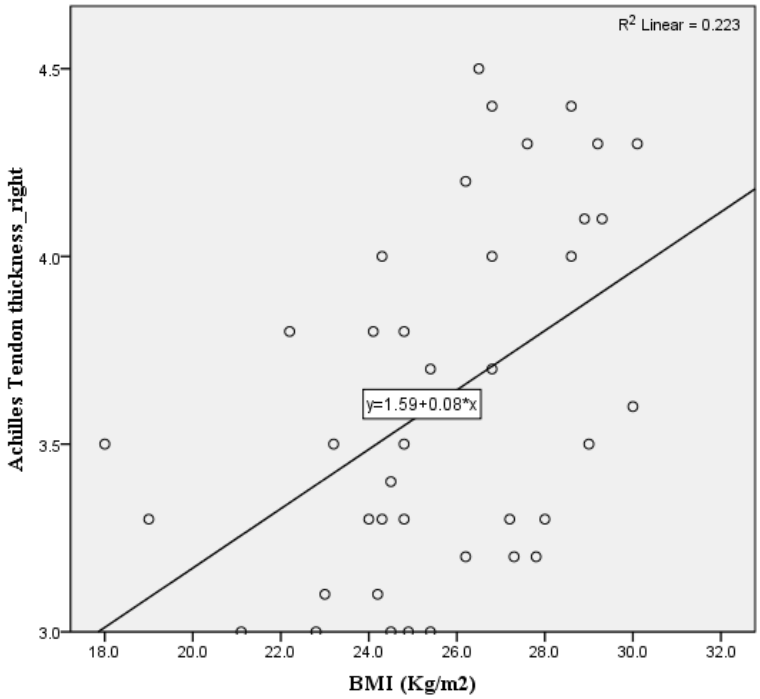
Table 6: Spearman's rho Correlation between BMI over PFT and AT in non diabetic group.

		Plantar Fascia Thickness (Left) (mm)	Plantar Fascia Thickness (Right) (mm)	Achilles Tendon Thickness (Left) (mm)	Achilles Tendon Thickness (Right) (mm)
BMI (Kg/m2)	Correlation Coefficient	0.186	0.190	0.525	0.477
	p-value	0.250	0.240	**0.001**	**0.002**
	N	40	40	40	40

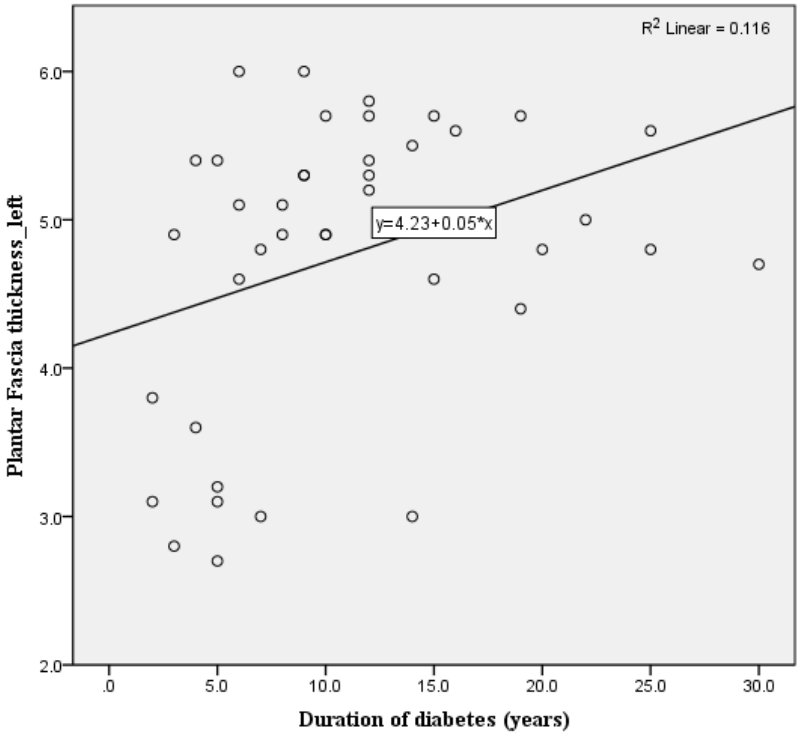
From Spearman's rho Correlation, only the AT showed significant values with BMI, based on this correlation with the highest correlation value noted in the left AT (R=0.525).



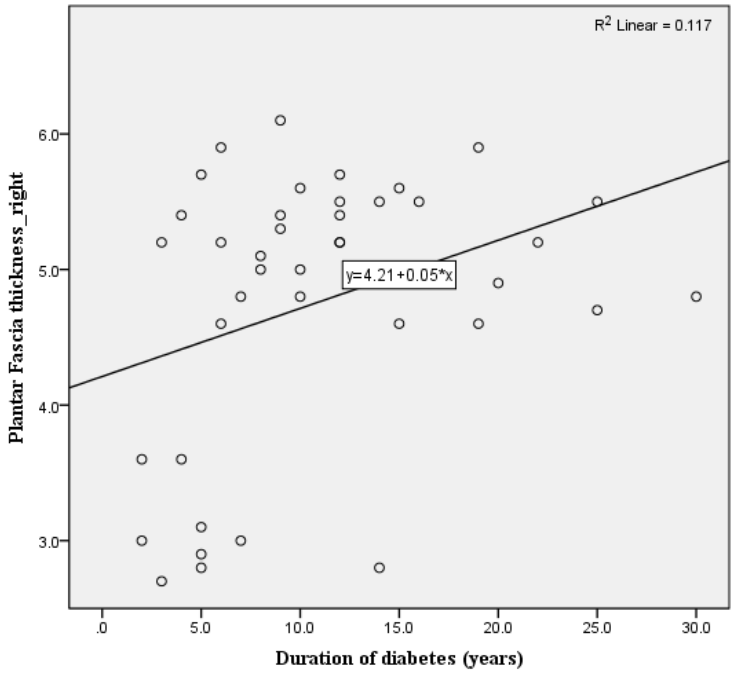
Graph 17: Scatter plot of BMI (Kg/m2) in non diabetic group and Achilles tendon thickness on left side



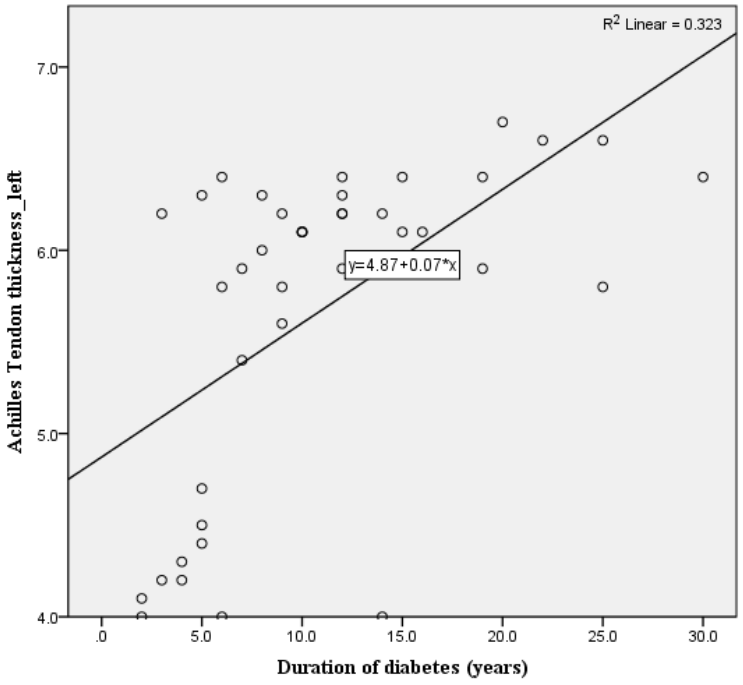
Graph 18: Scatter plot of BMI (Kg/m2) in non diabetic group and Achilles tendon thickness on right side



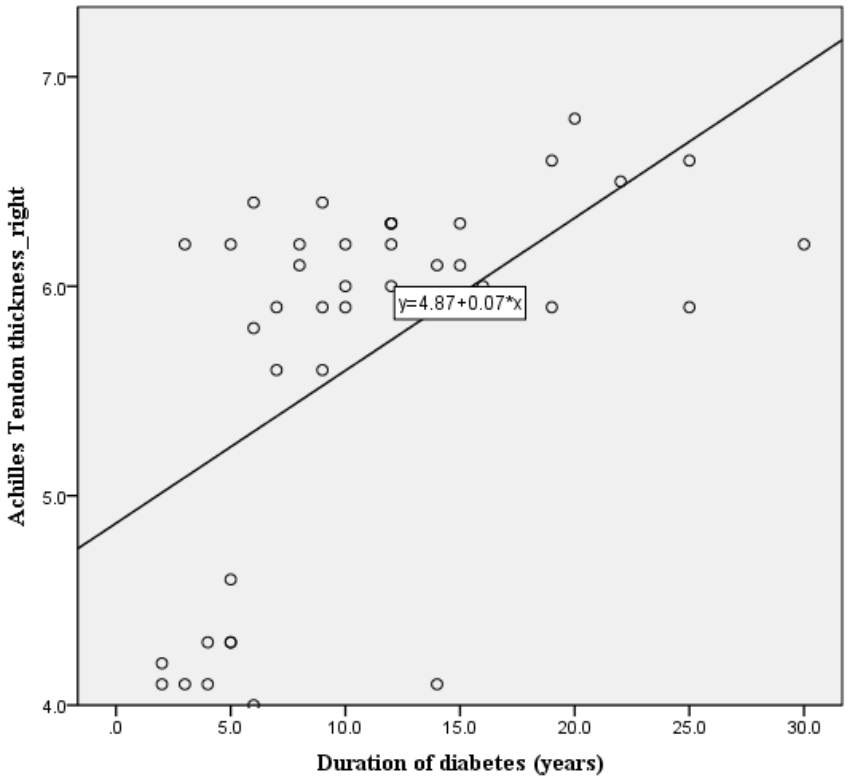
Graph 19: Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on left side in diabetic group



Graph 20: Scatter plot of duration of diabetes (years) and Plantar Fascia thickness on right side in diabetic group



Graph 21: Scatter plot of duration of diabetes (years) and Achilles tendon on left side in diabetic group



Graph 22: Scatter plot of duration of diabetes (years) and Achilles tendon on right side in diabetic group

DISCUSSION

Based on the available evidences, it had been postulated that, among the diabetic patients, irrespective of the type, hyperglycaemic non-enzymatic glycation of proteins would be leading to accumulation of glycosylated end-products in soft tissues including the Achilles tendon and plantar fascia (PF). This would further lead to locomotor concerns as well as inflammation with severe pain. The data mining on this concern had revealed that there were not many clinical trials who had observed for the changes among achilles tendon and plantar fascia among the same population.[7,8] Hence, the present study was conducted to evaluate the sonographic findings in the plantar fascia as well as the achilles tendon in patients with type 2 diabetes mellitus (DM) and compared these with non-diabetic population. We had included 40 each in diabetic group, designated as group A and non-diabetic group, designated as group B.

In our study, majority of the patients in either group were aged between 56 to 70 years and also, the distribution of gender was almost similar with no statistically significant difference.

ACHILLES AND PLANTAR FASCIA THICKNESS DIABETICS VERSUS NON-DIABETICS

The plantar fascia thickness among diabetic and non-diabetic was 4.76 ± 0.974mm and 3.56 ± 0.514 on left foot. Whereas in right foot, the thickness was 4.76 ± 1.007 and 3.56 ± 0.516 among diabetic and non-diabetic respectively. The thickness of Achilles tendon in left foot was 5.67 ± 0.881mm and 3.613 ± 0.515 mm respectively among diabetic and non-diabetic patients. Whereas the right foot Achilles tendon thickness was 5.665 ± 0.888 and 3.615 ± 0.465 respectively. Like our outcome, even in Umelo DO et al mean ATT, mean thickness in right and left foot of cases were, 5.56 ± 0.65mm and 5.59 ±

0.61mm. Whereas among the healthy population, 4.72 ± 0.44mm and 4.77 ± 0.40mm in right and left foot respectively (<0.0001). We can observe that our control group had comparatively thinner AT than their subjects. [63]

We observed that, there is significant difference in mean of Plantar Fascia Thickness (right and left), Achilles Tendon Thickness (right and left) over groups. Unlike our study, Harish SC et al found significantly thicker PF in diabetic patients compared to healthy volunteers. The mean thickness were 3.66 ± 0.89 mm and 2.67 ± 0.66 mm among diabetic and non-diabetics respectively ($p < 0.0001$). Addition to this, they had even observed that the thickening was more evident in those with neuropathy as compared to those without PN but did not attain statistical significance.[60] Afolabi BI et al also observed significant correlation between diabetics and increased thickness of the AT and PF of both foot.[10]

This was in consistent with the outcome of Abate M et al.[7] Whereas Huang X et al had considered Young ‘s modulus (E) value for analysing the fate of tendon and fascia among diabetics and compared it with control groups.[64] They observed that there will be increased thickness as well as stiffness among the diabetic group. Even in Khor BYC et al, though there was increased thickness of both AT and PF in both foot of the diabetics, thickness of PF did not have significant difference while AT thickness was significantly higher.[65]

As per the available explanation, the accumulation of GAGs and other pro-inflammatory markers due to insulin resistance among diabetics is the pathology behind this observation.

CORRELATION WITH EACH PARAMETER

DEMOGRAPHY: Age and gender

In our study, incidence of patients aged between 56 – 70 years were more in both groups, accounting for about 18 (45%) and 19 (47.5%) among group A and group B respectively. 50% each were males and females among Group A. In group B, 21 (52.5%) females and 19 (47.5%) males were there. The average age in group A and B respectively were 59.52 ± 11.1 years and 60.70 ± 11.46 years with the p value of 0.642, which did not have any correlation. This distribution of age and gender did not have any significant difference between the two groups. From Spearman's rho Correlation, we observed that there was statistically significant positive correlation ($P < 0.05$) obtained among diabetic group in the correlation of Left and right AT with respect to the Age. R=0.409, 0.400 were obtained in the right AT and left AT respectively but no such correlation was observed with respect to the plantar fascia thickness.

This could be probably due to age related changes as well. Also, as the diabetics are more prone for increased thickness of the tendon, this could have been significant change among elderly diabetics than non-diabetics. Favouring this statement, Stenroth L et al who had analysed the age-related changes in achilles, had reported that elderly individuals had comparatively increased thickness.[66] Jha DK et al reported that the chances of thickening of PF are in positive association among >45-year-old individuals.[67] Whereas Tillander et al had observed no much difference in the thickness of achilles even among symptomatic recreational runners compared to non-runners.[68] Unlike our study, Narindra et al had observed positive correlation between the age and plantar fascia thickness. Harish SC et al who analysed for the PF thickness between diabetic and non-diabetic individuals also did not find any correlation with the age.[69]

BMI

The distribution of BMI was 11 (27.5%), 15 (37.5%) and 14 (35%) in group A were with BMI between <24.9 Healthy, 25-29.9 overweight and >30 obese respectively. Whereas in group B, 18 (45%), 20 (50%) and 2 (5%) were <24.9 Healthy, 25-29.9 overweight and >30 obese respectively, with significant association of BMI with diabetes. Also, we observed that statistically significant ($P < 0.05$) results were obtained among diabetic group in the correlation of Left and right AT with BMI. R=0.392, 0.426 were obtained in the right AT and left AT respectively. Meanwhile even among non-diabetic patients, we observed that the thickness of Achilles and Plantar fascia had significant positive correlation with the BMI and not with any other parameters. Also, few evidences suggest that these changes imply that, along with thickness and stiffness, the factors such as tissue load, in the presence of structural tissue changes are collectively crucial for the development of DFU rather than either factor alone. Siddiqui R et al as well had observed BMI being one of the independent risk factors for increased thickness of tendon and fascia.[70]

Hence, obese individuals are more prone to tendon injury due to the increment in tendon thickness which causes bulging of the AT due to higher loads and diabetes among them would be the added risk. Also, the obese patients being higher risk for insulin resistance, they might further develop DM which would be viscous cycle with tendon and fascia related changes. Similarly, Ahn HS et al, a population based clinical research as well had reported higher BMI having greater association with achilles tendon rupture ATR and AT with positive hazard ratio [HR] of 3.49 and 1.96 respectively. This was even positive with increased waist circumference.[71] Also, they opined that inclusion of DM cases would yield the same outcome. Jha DK et al also reported the positive association between BMI and thickness of PF among their study population.[67]

DURATION OF DIABETES AND GLYCEMIC PARAMETERS

Of 40 subjects in the diabetic group, 13 (32.5%) of them had diabetes from 5-10 years, 10 (25%) of them had for less than 5 years. The correlation was statistically significant among duration of diabetes (years) with left and right PFT and with left and right AT. Also, we found statistically significant ($P < 0.05$) results were obtained among diabetic group in the correlation of Left and right ATT with Fasting blood glucose. R=0.689, 0.627 were obtained in the right AT and left AT respectively. Even in Afolabi BI et al there was a significant correlation with *r* value of 0.314 ($P < 0.05$) between the age and the duration of diabetes which were having significant correlation with increased thickness of AT as well as PF but they did not have such correlation with FBS. This could be explained by as, with increasing duration of diabetes, the soft-tissue changes associated with diabetes might be further increased.[10] Severe damages to both locomotor structures and functions of diabetic patients would be expected as the duration progress, which will further lead to formation of DFU.

STRENGTH OF OUR STUDY

Unlike other studies, we had compared AT and PF thickness among same population, which gives better inputs about diabetes related changes in both AT and PF.

LIMITATION

- Lesser sample size
- As it is a single centred study conducted at our hospital, it can not be generalized to the population
- We did not distinguish between DM patients with and without peripheral neuropathy, we were not able to determine how peripheral neuropathy affects AT and PF.
- We did not take into consideration peripheral vascular disease
- Study is not compared with gold standard for diagnosis and hence sensitivity in diagnosing can not be established

CONCLUSION

There was no significant association between the diabetes incidence and age or gender was observed. The plantar fascia thickness among diabetic and non-diabetic was 4.76 ± 0.974mm and 3.56 ± 0.514 on left foot. Right foot, the thickness of PF 4.76 ± 1.007 and 3.56 ± 0.516 among diabetic and non-diabetic respectively. The thickness of Achilles tendon in left foot was 5.67 ± 0.881mm and 3.613 ± 0.515 mm and right foot was 5.665 ± 0.888 and 3.615 ± 0.465 respectively among diabetic and non-diabetic patients, which had significant increase in diabetics ($p < 0.05$)

From Spearman's rho Correlation, BMI, age, FBS and duration of diabetes had significant positive correlation with the increased thickness of AT and PF in bilateral foot.

SUMMARY

With the available data, we had observed that diabetes, the chronic commonest non-communicable disease has been significantly associated with collagen vascular changes. This has led to the development of tendinitis, fasciitis and tendinopathy in various fascia and tendon of both upper and lower limbs. Of these, plantar fascia and achilles tendon have been observed to be affected the most. Thickness and stiffness of these have been statistically as well as clinically significant but there were dilemmatic outcomes in various clinical researches. Sono-elastography has been one of the most reliable mode of investigations. Hence, we had conducted this present study by including 40 patients in each group, Group A considering as case group: known case of diabetics and group B: control group, having non-diabetics. Our main objective was to compare the plantar fascia thickness and Achilles tendon thickness of diabetics to that of a control group as well as analyse the correlation with various parameters.

We conducted USG on GE LOGIQ P9 R2 machine equipped with a 7.5–12 MHz high frequency linear array transducer. We observed that there was no significant difference in the demographic details of the study population. The plantar fascia thickness among diabetic and non-diabetic was 4.76 ± 0.974mm and 3.56 ± 0.514 on left foot. 4.76 ± 1.007 and 3.56 ± 0.516 among diabetic and non-diabetic respectively in right foot. The thickness of left foot Achilles 5.67 ± 0.881mm and 3.613 ± 0.515 mm respectively among diabetic and non-diabetic patients. Whereas the right foot AT thickness was 5.665 ± 0.888 and 3.615 ± 0.465 respectively, which had significant increase in diabetics ($p < 0.05$) From Spearman's rho Correlation, BMI, age, FBS and duration of diabetes had significant positive correlation with the increased thickness of AT and PF in bilateral foot.

BIBLIOGRAPHY

1. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N et al. IDF Diabetes Atlas Committee. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. Diabetes Res Clin Pract. 2019 Nov;157:107843.
2. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018 Feb;14(2):88-98.
3. Banday MZ, Sameer AS, Nissar S. Pathophysiology of diabetes: An overview. Avicenna J Med. 2020 Oct 13;10(4):174-188.
4. Pirri C, Fede C, Pirri N, Petrelli L, Fan C, De Caro R, Stecco C. Diabetic Foot: The Role of Fasciae, a Narrative Review. Biology (Basel). 2021 Aug 7;10(8):759.
5. Picke AK, Campbell G, Napoli N, Hofbauer LC, Rauner M. Update on the impact of type 2 diabetes mellitus on bone metabolism and material properties. Endocr Connect. 2019 Mar 1;8(3):R55-R70.
6. Choi YJ, Chung YS. Type 2 diabetes mellitus and bone fragility: Special focus on bone imaging. Osteoporos Sarcopenia. 2016 Mar;2(1):20-24.
7. Abate M, Schiavone C, Salini V, Andia I. Occurrence of tendon pathologies in metabolic disorders. *Rheumatology (Oxford)*. 2013;**52**(4):599–608.
8. Giacomozzi C, D'Ambrogi E, Uccioli L, et al.. Does the thickening of Achilles tendon and plantar fascia contribute to the alteration of diabetic foot loading? *Clin Biomech* 2005; 20: 532-539.
9. 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.

10. Afolabi BI, Ayoola OO, Idowu BM, Kolawole BA, Omisore AD. Sonographic Evaluation of the Achilles Tendon and Plantar Fascia of Type 2 Diabetics in Nigeria. J Med Ultrasound. 2019 Apr-Jun;27(2):86-91.
11. Chen DW, Li B, Aubeeluck A, Yang YF, Huang YG, Zhou JQ, Yu GR. Anatomy and biomechanical properties of the plantar aponeurosis: a cadaveric study. PLoS One. 2014 Jan 2;9(1):e84347. doi: 10.1371/journal.pone.0084347. PMID: 24392127; PMCID: PMC3879302.
12. Latt, Daniel & Jaffe, David & Tang, Yunting & Taljanovic, Mihra. (2020). Evaluation and Treatment of Chronic Plantar Fasciitis. Foot & Ankle Orthopaedics. 5. 247301141989676. 10.1177/2473011419896763.
13. Image source: Internet https://www.netterimages.com/sole-of-foot-superficial-dissection-labeled-multiple-publications-general-anatomy-frank-h-netter-4485.html
14. Buchanan BK, Sina RE, Kushner D. Plantar Fasciitis. [Updated 2024 Jan 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK431073/
15. Bm SA, Tiwari V, Bakde AM, Dwidmuthe S, Roy M. Ultrasonographic Assessment of Indian Patients With Plantar Fasciitis and Its Clinical Correlation: A Prospective Observational Study. Cureus. 2023 Mar;15(3):e35764.
16. Mørk M, Soberg HL, Hoksrud AF, Heide M, Groven KS. The struggle to stay physically active-A qualitative study exploring experiences of individuals with persistent plantar fasciopathy. J Foot Ankle Res. 2023 Apr 15;16(1):20.
17. Bolgla LA, Malone TR. Plantar fasciitis and the windlass mechanism: a biomechanical link to clinical practice. J Athl Train. 2004 Jan;39(1):77-82.

18. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for Plantar fasciitis: a matched case-control study. J Bone Joint Surg Am. 2003 May;85(5):872-7.
19. Mørk M, Soberg HL, Hoksrud AF, Heide M, Groven KS. The struggle to stay physically active-A qualitative study exploring experiences of individuals with persistent plantar fasciopathy. J Foot Ankle Res. 2023 Apr 15;16(1):20.
20. Khired Z, Najmi MH, Akkur AA, Mashhour MA, Bakri KA. The Prevalence and Risk Factors of Plantar Fasciitis Amongst the Population of Jazan. Cureus. 2022 Sep 21;14(9):e29434.
21. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for Plantar fasciitis: a matched case-control study. J Bone Joint Surg Am. 2003 May;85(5):872-7.
22. Schwartz EN, Su J. Plantar fasciitis: a concise review. Perm J. 2014 Winter;18(1):e105-7.
23. Khired Z, Najmi MH, Akkur AA, Mashhour MA, Bakri KA. The Prevalence and Risk Factors of Plantar Fasciitis Amongst the Population of Jazan. Cureus. 2022 Sep 21;14(9):e29434.
24. Apaydin N, Bozkurt M, Loukas M, Vefali H, Tubbs RS, Esmer AF. Relationships of the sural nerve with the calcaneal tendon: an anatomical study with surgical and clinical implications. *Surg Radiol Anat.* 2009;31(10):775–780.
25. Del Buono A, Chan O, Maffulli N. Achilles tendon: functional anatomy and novel emerging models of imaging classification. Int Orthop. 2013 Apr;37(4):715-21.
26. Doral MN, Alam M, Bozkurt M, et al. Functional anatomy of the Achilles tendon. *Knee Surg Sports Traumatol Arthrosc.* 2010;18:638–643.
27. Image source: https://www.bupa.co.uk/health-information/muscles-bones-joints/achilles-tendinopathy

28. Tu P. Heel Pain: Diagnosis and Management. Am Fam Physician. 2018 Jan 15;97(2):86-93.
29. Maffulli N, Kenward MG, Testa V, Capasso G, Regine R, King JB. Clinical diagnosis of Achilles tendinopathy with tendinosis. Clin J Sport Med. 2003 Jan;13(1):11-5.
30. Maffulli N, Sharma P, Luscombe KL. Achilles tendinopathy: aetiology and management. J R Soc Med. 2004 Oct;97(10):472-6.
31. Sapra A, Bhandari P. Diabetes. 2023 Jun 21. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 31855345.
32. Malek R, Hannat S, Nechadi A, Mekideche FZ, Kaabeche M. Diabetes and Ramadan: A multicenter study in Algerian population. Diabetes Res Clin Pract. 2019 Apr;150:322-330.
33. Rorsman P., Ashcroft F.M. Pancreatic beta-Cell Electrical Activity and Insulin Secretion: Of Mice and Men. *Physiol. Rev.* 2018;98:117–214.
34. Mathur P, Leburu S, Kulothungan V. Prevalence, Awareness, Treatment and Control of Diabetes in India From the Countrywide National NCD Monitoring Survey. Front Public Health. 2022 Mar 14;10:748157.
35. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C. Pathophysiology of Type 2 Diabetes Mellitus. Int J Mol Sci. 2020 Aug 30;21(17):6275.
36. Ke C, Narayan KMV, Chan JCN, Jha P, Shah BR. Pathophysiology, phenotypes and management of type 2 diabetes mellitus in Indian and Chinese populations. Nat Rev Endocrinol. 2022 Jul;18(7):413-432.

37. Gefen A. Plantar soft tissue loading under the medial metatarsals in the standing diabetic foot. *Med Eng Phys*. 2003;**25**(6):491–499. doi: 10.1016/S1350-4533(03)00029-8
38. Gariani K, Waibel FWA, Viehöfer AF, Uçkay I. Plantar Fasciitis in Diabetic Foot Patients: Risk Factors, Pathophysiology, Diagnosis, and Management. Diabetes Metab Syndr Obes. 2020 Apr 22;13:1271-1279.
39. Connizzo BK, Bhatt PR, Liechty KW, Soslowsky LJ. Diabetes alters mechanical properties and collagen fiber re-alignment in multiple mouse tendons. *Ann Biomed Eng*. 2014;**42**(9):1880–1888.
40. Abate M, Schiavone C, Salini V, Andia I. Management of limited joint mobility in diabetic patients. *Diabetes Metab Syndr Obes*. 2013;**6**:197–207
41. Wu YF, Wang HK, Chang HW, Sun J. High glucose alters tendon homeostasis through downregulation of the AMPK/Egr1 pathway. Scientific Reports 7(1):44199
42. De Garceau D, Dean D, Requejo SM, Thordarson DB. The association between diagnosis of plantar fasciitis and Windlass test results. Foot Ankle Int. 2003 Mar;24(3):251-5.
43. Gariani K, Uckay I, Lipsky BA. Managing diabetic foot infections: a review of the new guidelines. *Acta Chir Belg*. 2014;**114**(1):7–16.
44. Yuen WLP, Tan PT, Kon KKC. Surgical Treatment of Haglund's Deformity: A Systematic Review and Meta-Analysis. Cureus. 2022 Jul 31;14(7):e27500.
45. https://www.spinalogy.com/haglunds-deformity
46. Carovac A, Smajlovic F, Junuzovic D. Application of ultrasound in medicine. Acta Inform Med. 2011 Sep;19(3):168-71
47. Quarato CMI, Lacedonia D, Salvemini M, Tuccari G, Mastrodonato G, Villani R, Fiore LA, Scioscia G, Mirijello A, Saponara A, Sperandeo M. A Review on

Biological Effects of Ultrasounds: Key Messages for Clinicians. Diagnostics (Basel). 2023 Feb 23;13(5):855.

48. Ultrasound imaging: http://famus.org.uk/modules/ultrasound-theory-module
49. Zander D, Hüske S, Hoffmann B, Cui XW, Dong Y, Lim A, Jenssen C, Löwe A, Koch JBH, Dietrich CF. Ultrasound Image Optimization ("Knobology"): B-Mode. Ultrasound Int Open. 2020 Jun;6(1):E14-E24.
50. Ehrmann C, Maier M, Mengiardi B, Pfirrmann CW, Sutter R. Calcaneal attachment of the plantar fascia: MR findings in asymptomatic volunteers. *Radiology*. 2014;272(3):807–814.
51. McNally EG, Shetty S. Plantar fascia: imaging diagnosis and guided treatment. *Semin Musculoskelet Radiol.* 2010;14(3):334–343.
52. Draghi F, Gitto S, Bortolotto C, Draghi AG, Ori Belometti G. Imaging of plantar fascia disorders: findings on plain radiography, ultrasound and magnetic resonance imaging. Insights Imaging. 2017 Feb;8(1):69-78.
53. https://www.hmpgloballearningnetwork.com/site/podiatry/point-counterpoint-epf-it-necessary-recalcitrant-plantar-fasciitis
54. Li HY, Hua YH. Achilles Tendinopathy: Current Concepts about the Basic Science and Clinical Treatments. Biomed Res Int. 2016;1(1):6492597.
55. Sierevelt I, van Sterkenburg M, Tol H, van Dalen B, van Dijk N, Haverkamp D. Dutch version of the Victorian Institute of Sports Assessment-Achilles questionnaire for Achilles tendinopathy: Reliability, validity and applicability to non-athletes. World J Orthop. 2018 Jan 18;9(1):1-6.
56. Corrigan P, Cortes DH, Pohlig RT, Silbernagel KG. Tendon Morphology and Mechanical Properties Are Associated With the Recovery of Symptoms and

Function in Patients With Achilles Tendinopathy. April 2020; 8(4):232596712091727

57. Oshri Y, Palmanovich E, Brin YS, Karpf R. Chronic insertional Achilles tendinopathy: Surgical outcomes. Muscle lig tendon. Apr 2012; 2(2): 91-95
58. Buonon AD, Chan O, Maffulli N. Achilles tendon: Functional anatomy and novel emerging models of imaging classification. December 2012; International Orthopaedics 37(4): 715 -722
59. Wu CH, Chen WS, Park GY, et al.. Musculoskeletal sonoelastography: a focused review of its diagnostic applications for evaluating tendons and fascia. *J Med Ultrasound* 2012; 20: 79-86.
60. Harish SC, Dixit R, Singh S, Garg S. Evaluation of the plantar fascia in patients with diabetes mellitus: the role of sonoelastography. Pol J Radiol. 2022 Sep 9;87:e500-e505.
61. Latt LD, Jaffe DE, Tang Y, Taljanovic MS. Evaluation and Treatment of Chronic Plantar Fasciitis. Foot Ankle Orthop. 2020 Feb 13;5(1):2473011419896763. doi: 10.1177/2473011419896763. PMID: 35097359; PMCID: PMC8564931.
62. 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 Feb 13;31(4):282-286.
63. Umelo DO, Ugboma EW, Nwankwo NC, Wekhe CN. Ultrasonographic evaluation of the achilles tendon in patients with type 2 diabetes: a dual center study in port harcourt. The Nigerian health J. 2021; 21(1): 228-249

64. Huang X, Chen X, Chen X, Chi P, Wang P, Zhan X, Zou C, Wang L, Dong Y. Sound touch elastography of Achilles tendons in patients with type 2 diabetes mellitus versus healthy adults. Diabetol Metab Syndr. 2023 Aug 21;15(1):174.
65. Khor BYC, Woodburn J, Newcombe L, Barn R. Plantar soft tissues and Achilles tendon thickness and stiffness in people with diabetes: a systematic review. J Foot Ankle Res. 2021 Apr 28;14(1):35.
66. Stenroth L et al https://doi.org/10.1152/japplphysiol.00782.2012
67. Jha DK, Wongkaewpotong J, Chuckpaiwong B. Effect of Age and BMI on Sonographic Findings of Plantar Fascia. J Foot Ankle Surg. 2023 Jan-Feb;62(1):125-128.
68. Tillander B, Gauffin H, Lyth J, Knutsson A, Timpka T. Symptomatic Achilles Tendons are Thicker than Asymptomatic Tendons on Ultrasound Examination in Recreational Long-Distance Runners. Sports (Basel). 2019 Dec 5;7(12):245.
69. Narindra LHRNO, Herinirina NF, Rakotonirina H, Andrianah GE, Ranoharison HD, Randriamboavonjy R, Ahmad A. Thickness of the Plantar Fascia in Asymptomatic Subjects. J Med Ultrasound. 2019 Feb 26;27(3):121-123.
70. Siddiqui R, Mriza EH, Javed R, Al-Qahtani M. Investigation of the Tissue Degenerative Impact of Increased BMI in Achilles Tendon via Strain Elastography and Finite Element Analysis. Curr Med Imaging. 2023;19(6):587-595.
71. Ahn HS, Kim HJ, Suh JS, Kazmi SZ, Kang TU, Choi JY. The Association of Body Mass Index and Waist Circumference with the Risk of Achilles Tendon Problems: A Nationwide Population-Based Longitudinal Cohort Study. Clin Orthop Surg. 2023 Jun;15(3):488-498.

ANNEXURE-I

INFORMED CONSENT FORM

'REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FSCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLTIUS TYPE II – A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY'

Name of Student/Principal Investigator: DR
POST GRADUATE STUDENT
(M.D RADIODIAGNOSIS)
JNMC, BELAGAVI

Name of Guide/Co Investigators: DR
PROFESSOR
DEPT. OF RADIODIAGNOSIS,
JNMC, BELAGAVI

Objective:

- **Primary objective**: The objective of this study is to compare the plantar fascia thickness and Achilles tendon thickness of diabetics to that of a control group.
- **Secondary objective:** Correlation analysis between plantar fascia thickness and Achilles tendon thickness with demographic data such as body mass index, duration of diabetes, fasting blood glucose level, sex and age of the patient.

Introduction: You are being invited to participate in this study for ultrasonographic evaluation of the plantar fascia and Achilles tendon.

Explanation of procedure:

If, you agree to be a part of the research study, you will be asked the relevant history and will be subjected to relevant investigations.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: Dr **********, 9535XXXXXX, ********@gmail.com. If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study '**REAL TIME ULTRASONOGRAPHY IN EVALUATION OF THE PLANTAR FSCIA THICKNESS AND ACHILLES TENDON THICKNESS IN DIABETES MELLTIUS TYPE II – A ONE YEAR HOSPITAL BASED CASE CONTROL STUDY'**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE -II

PROFORMA FOR DATA COLLECTION

NAME_____

AGE_____

SEX: **OP/IP NO _____**

MOBILE NO: _____

ADDRESS _____

BMI:_____

HbA1c:_____

FASTING BLOD GLUCOSE:_____

DURATION SINCE DIAGNOSIS OF DIABETES MELLITUS TYPE II:-

_____

TREATMENT MODALITY (INSULING/ ORAL HYPOGLYCEMIC DRUGS) :

_____

USG NUMBER: _____

COMORBIDITIES: _____

PRESENTING COMPLAINTS:-

USG FINDINGS:-

(a) LEFT PLANTAR FASCIA THICKNESS -

(b) RIGHT PLANTAR FASCIA THICKNESS-

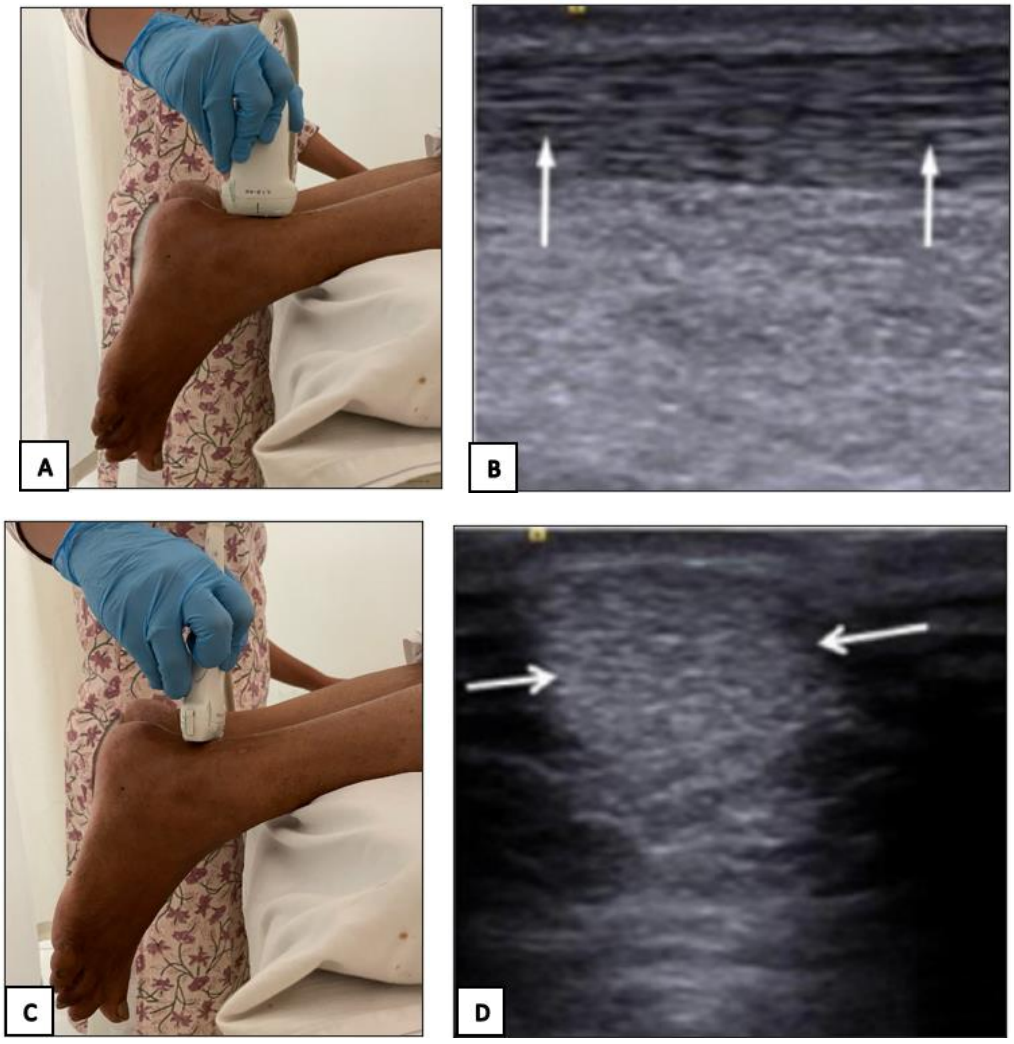
(c) LEFT ACHILLES TENDON THICKNESS-

(d) RIGHT ACHILLES TENDON THICKNESS-

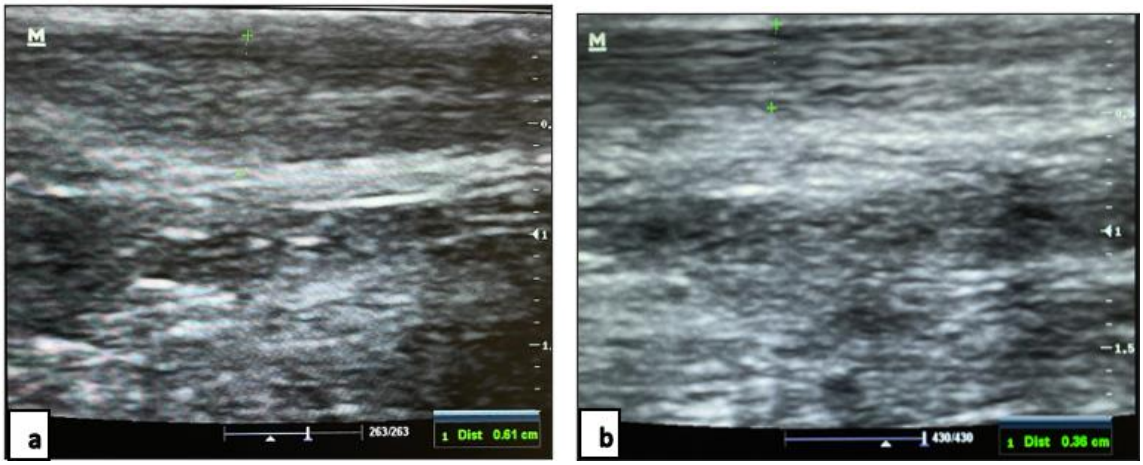
ANNEXURE- III

CLINICAL IMAGES

ACHILLES TENDON

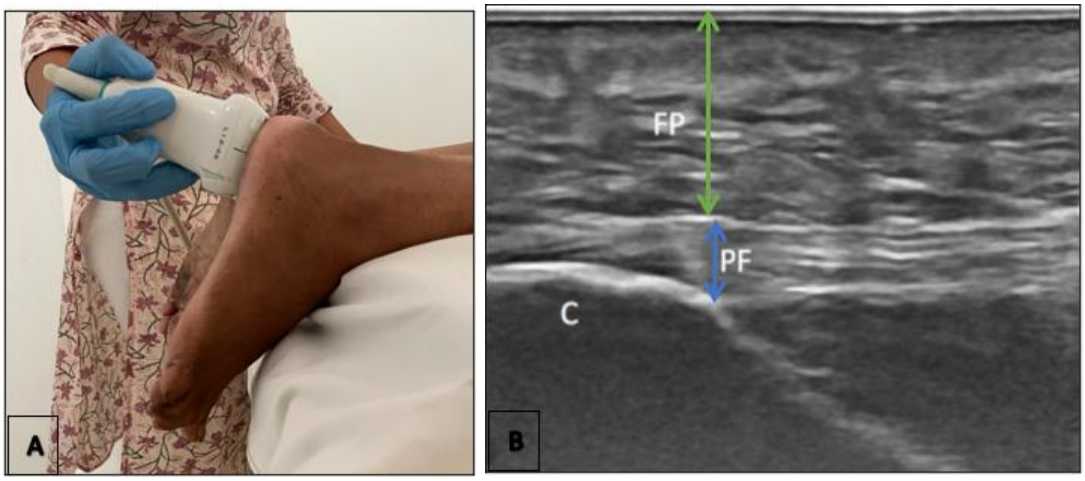


Ultrasonography of a normal Achilles tendon showing the transducer position and equivalent sonographic image in longitudinal/long axis view (A,B) and transverse/ short axis view (C,D)

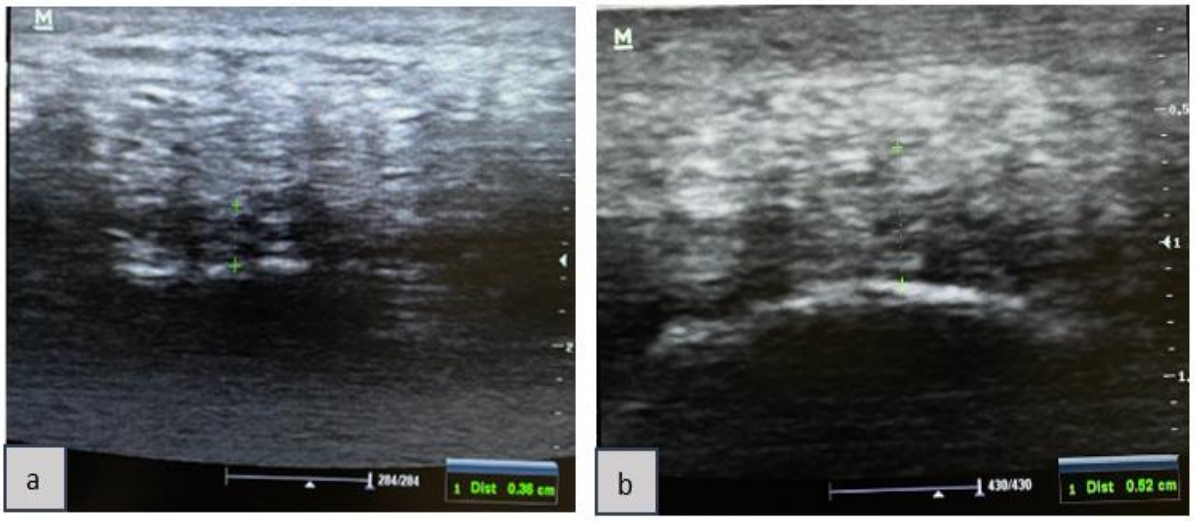


Sonographic images showing AT thickness in (a) DM patient and (b) Non diabetic patient

PLANTAR FASCIA



A. The transducer was placed over the plantar aspect of the hindfoot.
B. Long-axis sonogram of the plantar fascia. C, calcaneus; FP, fat pad (green line shows thickness); PF, plantar fascia (blue line shows thickness).



Sonographic images showing Plantar Fascia (PF) thickness (a) Non diabetic patient (b) and DM patient

ANNEXURE- IV

KEY TO MASTERCHART

FBS	FASTING BLOOD GLUCOSE
DRT OF DM	DURATION OF DIABETES MELLITUS
PFT	PLANTAR FASCIA THICKNESS
ATT	ACHILLES TENDON THICKNESS
YRS	YEARS
BMI	BODY MASS INDEX

ANNEXURE- V MASTERCHART

CASE	AGE (YRS)	SEX	DRT OF DM (YRS)	BMI (Kg/m2)	HBA1c (%)	FBS (mg/dl)	PFT (LT) (mm)	PFT (RT) (mm)	ATT (LT) (mm)	ATT (RT) (mm)
A1	63	M	12	29	6.4	140	5.2	5.2	6.2	6.2
A2	54	F	9	30	7	148	6	6.1	5.8	5.9
A3	38	M	2	23.2	6.6	102	3.8	3.6	4	4.1
A4	69	M	15	32	6.9	154	4.6	4.6	6.1	6.1
A5	45	M	4	24.2	7.4	100	3.6	3.6	4.2	4.3
A6	75	F	20	29.8	9.2	186	4.8	4.9	6.7	6.8
A7	76	F	25	30.7	10	198	5.6	5.5	6.6	6.6
A8	49	M	4	24.8	6.2	123	5.4	5.4	4.3	4.1
A9	68	F	10	30.6	6	130	4.9	5	6.1	6.2
A10	78	F	15	28.6	8.4	172	5.7	5.6	6.4	6.3
A11	44	F	12	31.4	8.3	174	5.3	5.4	6.3	6.3
A12	57	M	14	28.2	9	182	5.5	5.5	6.2	6.1
A13	51	M	6	22	8	100	5.1	5.2	4	4
A14	47	F	7	23	7.1	105	3	3	5.9	5.9
A15	69	F	5	22	6.8	120	3.2	3.1	4.5	4.3
A16	56	F	6	24	6.8	132	4.6	4.6	5.8	5.8
A17	81	F	30	29.2	8.9	198	4.7	4.8	6.4	6.2
A18	70	M	22	29.8	9.2	200	5	5.2	6.6	6.5
A19	69	M	9	32.6	7.4	142	5.3	5.3	6.2	6.4
A20	67	M	5	30	7	111	2.7	2.8	4.7	4.6
A21	73	F	19	31.2	6.3	132	5.7	5.9	5.9	5.9
A22	55	M	8	26.5	7.2	150	4.9	5	6	6.1
A23	62	M	2	24	5.7	92	3.1	3	4.1	4.2
A24	63	M	10	29.6	6.2	120	5.7	5.6	6.1	6
A25	71	F	25	30.2	7.2	152	4.8	4.7	5.8	5.9
A26	62	M	19	29.8	8.4	180	4.4	4.6	6.4	6.6
A27	58	F	10	32.3	8.9	204	4.9	4.8	6.1	5.9
A28	55	F	5	28.6	6.2	135	5.4	5.7	6.3	6.2
A29	64	M	12	24.2	5.9	100	5.8	5.7	5.9	6
A30	53	F	9	26.8	6.1	140	5.3	5.4	5.6	5.6
A31	49	M	3	24.4	6.8	102	2.8	2.7	4.2	4.1
A32	67	M	12	30	5.7	112	5.7	5.5	6.2	6.3
A33	45	F	6	28	6	128	6	5.9	6.4	6.4
A34	58	M	14	29.4	7.2	110	3	2.8	4	4.1
A35	69	F	8	31.4	6.3	145	5.1	5.1	6.3	6.2
A36	40	F	3	24	5.8	120	4.9	5.2	6.2	6.2
A37	45	F	7	32	8	162	4.8	4.8	5.4	5.6
A38	50	M	12	26.2	7.2	130	5.4	5.2	6.4	6.3
A39	52	M	5	28.9	6	98	3.1	2.9	4.4	4.3
A40	64	F	16	30.4	6.2	126	5.6	5.5	6.1	6

CASE	AGE (Yrs)	SEX	BMI (Kg/m2)	HBA1c (%)	FBS (mg/dl)	PFT (LT) (mm)	PFT (RT) (mm)	ATT (LT) (mm)	ATT (RT) (mm)
B1	62	M	29	5.2	100	4.3	4.1	3.6	3.5
B2	45	F	24.3	4.6	86	3.2	3.2	4	4
B3	40	M	19	4.9	89	2.8	2.7	3.2	3.3
B4	51	M	21.1	4.8	102	3	3	2.9	3
B5	65	M	26.2	5.3	120	2.9	3	3.1	3.2
B6	80	F	28.6	5	110	3.4	3.5	4.1	4
B7	64	F	22.2	4.8	85	4.2	4.3	3.8	3.8
B8	72	M	29.2	5.2	92	3.6	3.6	4.4	4.3
B9	40	F	18	5.2	94	4	3.9	3.5	3.5
B10	66	F	25	5.1	100	4.4	4.4	3.6	3.6
B11	76	M	26.2	4.8	104	4.2	4.3	4.3	4.2
B12	55	F	24.8	4.9	96	3.1	3.4	3.3	3.3
B13	68	M	25.4	4.6	99	3.2	3.3	3.7	3.7
B14	70	F	26.8	4.9	86	3	3	3.6	3.7
B15	42	F	24.2	5.1	84	4.3	4.4	3	3.1
B16	79	F	26.5	5.2	87	4.1	4.2	4.5	4.5
B17	58	M	23	4.5	90	3.2	3.2	3.1	3.1
B18	67	M	24.5	4.6	92	3.1	3.2	3.2	3.4
B19	56	F	23.2	4.3	103	3.5	3.5	3.2	3.5
B20	68	M	24.3	5.1	91	3.9	3.8	3.3	3.3
B21	75	M	24.9	4.2	97	2.9	2.9	3	3
B22	64	F	27.6	4.8	96	4.4	4.5	4.2	4.3
B23	78	F	28.6	4.9	104	4.2	4.2	4.6	4.4
B24	46	M	22.8	5.2	94	3.2	3.5	3.1	3
B25	72	F	24.8	5.1	89	3.7	3.8	3.5	3.5
B26	54	M	25.4	4.3	100	3.6	3.5	2.8	3
B27	63	F	30.1	4.9	84	4.1	4.2	4.3	4.3
B28	59	M	28.9	4.6	86	4.4	4.3	4.2	4.1
B29	73	F	26.8	4.6	84	3.6	3.4	4.6	4.4
B30	39	F	24	4.8	93	3.4	3.2	3.3	3.3
B31	61	F	27.8	5	109	3.2	3.3	3.2	3.2
B32	52	M	29.3	5.3	110	4	4	4	4.1
B33	64	F	24.8	4.9	97	3.8	3.6	3.8	3.8
B34	69	M	26.8	4.6	115	3	3.2	3.9	4
B35	47	M	24.1	4.8	86	3.2	3	4	3.8
B36	52	M	27.3	5.1	94	3.5	3.6	3.2	3.2
B37	62	F	30	4.7	89	2.9	2.8	3.7	3.6
B38	57	F	27.2	5.2	92	2.8	2.8	3.3	3.3
B39	49	M	24.5	4.9	96	3.7	3.6	2.9	3
B40	68	F	28	5	95	3.4	3.3	3.5	3.3