Original Article

Sonographic Evaluation of the Heel Pad Thickness in Diabetics in Nigeria

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Abstract

Background: Diabetes mellitus is a chronic disease process affecting millions of people worldwide. Its prevalence is forecasted to reach a value of 7.7% by 2030. It carries severe morbidities and even mortality. Hyperglycemia and increased formation of advanced glycosylation end products causes the majority of soft tissue changes seen among diabetics. The effects are observed particularly in the heel pad and plantar fascia where thinning or thickening, fibre disorganization, calcification and hypoechoic foci are among the changes seen. **Methods:** This cross-sectional descriptive study was carried out at the Department of Radiology, OAUTHC, Ile Ife, Osun state, Southwest Nigeria. 40 years old and above subjects with Type 2 diabetes mellitus were recruited from the diabetic clinic of the institution and Ultrasound evaluation of the heel fat pad and plantar fascia were subsequently performed for those who met the criteria using ultrasound machine equipped with a 7.5- 12.0 MHz high frequency linear array transducer. **Results:** The mean heel pad thickness on the right feet was greater than that of the left in the study subjects. There was statistical significant difference in the heel fat pad thickness of diabetic subjects and the control group, in both feet. (P=0.000). The heel pad thickness is higher in diabetic subject than in non-diabetic control subjects. However, there was no statistical significant difference in the right and left heel fat pad thickness of the participants of this study (P value 0.6062). Only HPT was a statistically significant predictor of foot ulcers among other variables after binary regression was computed. Using Spearman's rank correlation to test the relationship between the BMI of diabetic subjects and mean heel pad thickness, it revealed a moderate positive correlation, with good statistically significance (Spearman's rho = 0.4397, P=0.0000). The relationship between the BMI of diabetic subjects and mean plantar fascia thickness showed a weak positive correlation, with good statistical significance (Spearman's rho = 0.2635, P=0.0008). **Conclusion:** The duration of diabetes mellitus did not determine the heel pad thickness and plantar fascia thickness. The findings in the study suggested that history of foot ulcer in the diabetic predispose them to have reduce HPT and further foot ulcers. Sonographic measurement of heel pad thickness can therefore be an additional imaging modality to evaluate and be used in the management of the diabetic patients' feet.

Keywords: Correlation, foot ulcer, heel pad thickness, ultrasound scan

Introduction

Diabetes mellitus (DM) is a chronic endocrine disease that comprises a group of common metabolic disorders which share the phenotype of hyperglycemia and affects millions of people worldwide. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors, and life-style choices. Depending on the etiology of the DM, factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiological changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system.[1] The prevalence of diabetes mellitus is gradually increasing especially with civilization. It is presently estimated at 5.1%

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worldwide and it is forecasted to reach a value of 7.7% by 2030.[2] Though the prevalence of both Type 1 and Type 2 DM is increasing worldwide, the prevalence of Type 2 DM is expected to rise more rapidly in the future because of increasing obesity and reduced physical activity levels.

In studying complications of chronic diabetes, retinopathy and nephropathy have naturally been the focus of many publications, due to the high prevalence of these complications.[3] Most of these studies addressed diabetic neuropathy as almost the only cause for the onset of the ulceration processes, thus delaying the development of clinical interest and basic research towards concurrent causes. As a result, the setup of screening and treatment for the prevention of neuropathic ulcers is still poorly effective compared with the human and economic efforts of the scientific community.[4,5] However, little attention has been paid to changes in the musculoskeletal system, which can contribute to the reduction of the general state of health of diabetic patients.[3,6] A different approach to the investigation of all the potential causes of the plantar ulceration process comes from the observation that the common sign of the distinct diabetic syndromes is hyperglycaemia, which promotes glycosylation of proteins and the consequent accumulation of advanced glycosylation end-products in most human tissues.[7]

Consequently, muscles, cartilages, tendons and ligaments might all experience structural changes even before the onset of diabetic neuropathy, and might then concur to alter the overall function of the foot–ankle complex during gait.[4] Attention should thus be paid to the modifications of main tendons, ligaments and other soft tissues that manage and control the foot.

The heel pad, located beneath the calcaneus bone, acts as an efficient shock absorber to reduce potential injury to the body during ambulation.[8] Diabetic foot ulcers; the end stage of a series of harmful cascades initiated by hyperglycemia, are caused by multiple pathologies[9] in which the altered foot mechanical properties resulting from the changes in fibrous structure may contribute in part to the development of foot ulceration.[10] It has been documented that diabetic ulcers frequently occur at pressure sensitive sites, commonly the heel, big toe, first, second and fifth metatarsal.[11] Structural changes have been observed in the sole of the foot of diabetic patients like decrease in the heel pad thickness, increase in density of collagen fibrils and decrease in the thickness of the sole over the first and second metatarsals.[12]

Diabetes mellitus is the most notable among medical conditions that predispose to plantar fasciopathy, a disorder characterized by thickened plantar fascia, disorganization of the normal reflective structure and loss of the normal organized plantar fascia architecture[13] Plantar fascia is among those tissues that may change their physiology and biomechanical function in the presence of chronic hyperglycaemia. Studies by Sharkey *et al*.[14] revealed that the plantar fascia, despite being a passive structure, actively influence the pressure acting on the metatarsal heads.

Plain radiography which is traditionally used for assessing the heel pad can only demonstrate bony abnormalities such as calcaneal spurs with poor soft tissue resolution. However, while taking a radiograph, a slight magnification of the heel pad thickness will result in less accurate measurements compared with real-time high resolution ultrasonography.[15] Computed Tomography scan has a better bone resolution compared to plain radiograph but both Imaging modalities utilize ionizing radiation.

On the other hand, Magnetic resonance imaging (MRI) can provide more information about soft-tissue changes in the heel fat pad and plantar fascia though it is expensive and lacks dynamic real-time assessment.[16] Also, optimal MRI is not widely available for routine use in our environment. High frequency Musculoskeletal Ultrasonography (MSK US) is being advocated as a valuable diagnostic tool for evaluating the sole of the foot. It has many advantages, including its non-invasive nature, low cost, portability, dynamic real-time assessment, and easy side-to-side evaluation of the soles of both feet.[17-19] This non-invasive technique is an ideal choice for direct and dynamic measurements comparison. In addition, the use of extended field imaging has helped in imaging larger anatomic structures and split-screen imaging is beneficial in comparing the changes of the heel-pad, because it permits accurate evaluation of the thickness and real time continuous imaging.[20] There are no available local studies evaluating the sonographic features of heel fat pad in diabetic patients. This study will aid in evaluating the influence of diabetes on heel pad thickness, plantar fascia thickness and other sonographic changes in these soft tissue structures. Also, relationships between these findings and relevant clinical and laboratory parameters were also evaluated.

Materials and Methods

This was a cross-sectional descriptive study carried out at the Department of Radiology of our institution.

Subject selection

The study was carried out on adult subjects with Type 2 diabetes mellitus who were aged 40 years and above. They were recruited from the diabetic clinic (Endocrinology Unit) of our institution. The participants included newly diagnosed diabetics and those on follow-up attending the clinic. The control group was individuals with fasting blood glucose <6.1 mmol/l and with no known history of diabetes mellitus.

Written informed consent was obtained from both patients and controls. Approval number: IRB/IEC/0004553 NATIONAL: NHREC/27/02/2009a.

Inclusion and exclusion criteria

Inclusion criteria for cases

The subject group included individuals attending the outpatient clinic of OAUTHC Ile Ife that were aged 40–80 years and have been diagnosed with Type 2 diabetes mellitus by the Endocrinologist based on the WHO 1997 criteria which include any of the following;

a. Fasting plasma glucose of 126 mg/dl (7.0 mmol/L) or higher on two separate tests
b. Symptoms of diabetes plus random blood glucose of 200 mg/dl (11.1 mmol/L) or higher
c. Two-hour plasma glucose >200 mg/dl (11.1 mmol/L) during an oral glucose tolerance test
d. Glycated hemoglobin (HbAic) of >48 mmol/L (>6.5%). The inclusion criteria were set about the least years of these patients diagnosed as having DM.

Exclusion criteria for the cases

1. Chronic smoking
2. Age over 80 years. Beyond this threshold, skeletal, neurological, or more general degenerative pathologies might render the analysis of the effects of diabetes more difficult
3. History of peripheral vascular, neurological (other than those of diabetic etiology), musculoskeletal, or rheumatoid disease
4. Any minor or major amputation
5. Charcot neuroarthropathy and hallux rigidus as a result of previous traumas. Not all patients had X-ray screening for Charcot neuroarthropathy. This will mean unnecessary exposure of patients to ionizing radiation. The feet and ankles were properly examined for swelling or any form of deformity
6. Chronic use of steroids
7. Congenital ankle deformities
8. Abnormal gaits. This is when there is a problem in the stance and swing phase of walking due to pain, weakness, or difference in the lengths of the limbs. The endocrinologist and or orthopedic surgeon determine if there is an abnormal gait.
9. Renal failure. It was determined by both the Creatinine level and glomerular filtration rate
10. Athletes and bodybuilders.

Inclusion criteria for the controls

1. Healthy volunteers comprising hospital staff, patient relatives, and individuals presenting in the Radiology department for other investigations (e.g., routine medical check-up) but are not diabetic
2. Fasting blood glucose in the range of 4.0–5.6 mmol/L
3. No known history of DM or foot ulcer
4. Age and sex were matched with those of DM patients.

Exclusion criteria for the controls

1. Subjects who did not meet the inclusion criteria for the control
2. Subjects with any exclusion criterion for cases
3. Persons who did not give their consent for whatever reason.

Equipment and materials

TOSHIBA® Real-time ultrasound machine: Model TUSF-30 (TOSHIBA MEDICAL SYSTEMS CORPORATION, OTAWARA SHI TOCHIGI 324–8550, JAPAN manufactured in the year 2012) equipped with a 7.5– 12.0 MHz high frequency linear array transducer and Accucheck glucometer with test strips.

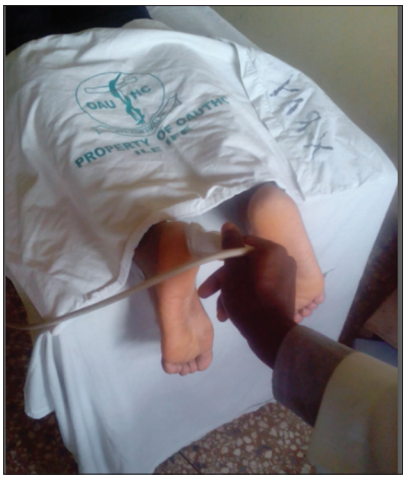

Figure 1: Showing patient and probe positioning in examining the heel pad in longitudinal axis view (Original)

Technique

The study group consisted of 80 diabetic patients and 80 age- and sex-matched controls.

Both groups were subjected to their respective inclusion and exclusion criteria.

Written informed consent was obtained from all participants.

Participants had their weight (kg) and height (m) measured. Their body mass index (BMI) was calculated. Venous blood was obtained in the morning following an overnight fast.

Fasting plasma glucose concentration was measured. In both the experimental and control group, ultrasound evaluation of the heel fat pad and plantar fascia were subsequently performed for those who met the above criteria.

To examine the heel pad, the patient laid in a prone position on an examination couch with the feet extending beyond the edge of the couch [Figure 1]. With the feet in a neutral position, a layer of acoustic gel was applied over the area of the heel of the foot. The linear array ultrasound transducer was placed on the coupling gel. It was applied as perpendicular to the heel pad as possible to prevent anisotropy. Depth and gain were adjusted to achieve acceptable imaging [Figure 2].

Scanning of the heel pad was done in longitudinal and transverse planes from the posterior margin of the sole of the foot to the junction between the hind and mid-foot. The (heel pad? Plantar fascia?) Thickness was then measured on a transverse plane at 3 cm from the posterior margin of the sole of the foot. The thickness of both right and left heel pads was measured three consecutive times using a gel stand-off technique to standardize the pressure on the heel pad. The mean value for each side was taken. Other morphological

changes like disorganization of the heel pad echotexture and the presence of calcaneal spur were documented. Each examination took an average of 5–10 min.

To examine the plantar fascia, participants laid prone on an examination table with feet overhanging the edge and toes pointing away from the body. A layer of gel was applied over the region of the plantar fascia. A linear array high-resolution transducer (7.5–12MHz) was then placed over it and the plantar fascia was examined from its calcaneal insertion to the region of the forefoot under the metatarsophalangeal joints. The thickness of the fascia was measured over the center of the arch at least 3 cm from its calcaneal insertion. This particular site was chosen because of its high reproducibility.

For each patient, three measurements of each of the right and left plantar fascia were performed, and the mean plantar fascia thickness was calculated for each foot.

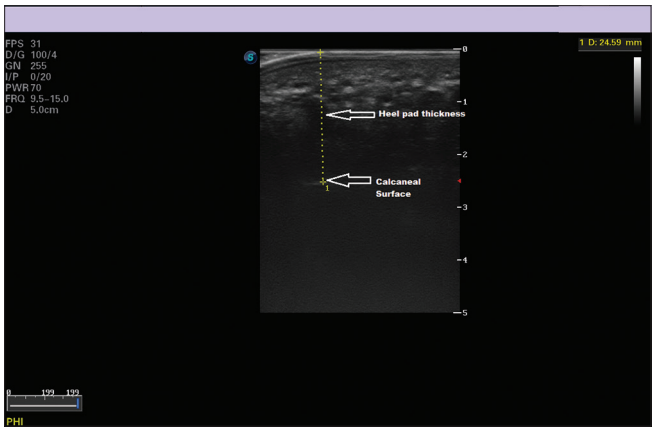

Figure 2: Sonographic image of the heel pad showing the heel pad thickness and the calcaneal surface (longitudinal view) (Original)

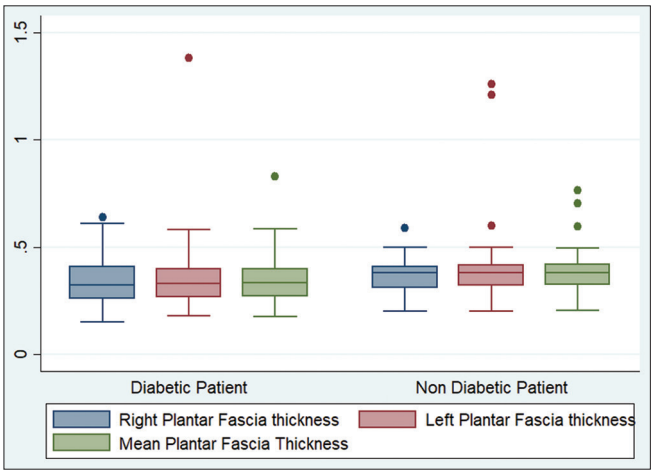

Figure 3: Box plot showing plantar fascia thickness in diabetic and non-diabetic patients (Original)

Table 1: Demographic characteristics of study subjects

Variables	Diabetic (*n*=80), *n* (%)	Nondiabetic (*n*=80), *n* (%)	Statistics	Df	*P*
Age (years)					
Mean±SD* (range)	59.5±10.4 (40-82)	60.2±7.4 (42-85)	0.473	158	0.6368
Male (mean±SD)*	61.6±8.4	60.1±8.2	0.7606	68	0.4495
Female (mean±SD)*	58.2±11.4	60.2±6.5	1.0034	88	0.3184
<50	15 (18.75)	7 (8.75)	0.473	158	0.6368
50-59	17 (21.25)	28 (35.0)			
60-69	33 (41.25)	37 (48.75)			
≥70	15 (18.75)	6 (7.5)			
Gender**					
Male	30 (37.5)	40 (50.0)	2.5397	1	0.111
Female	50 (62.5)	40 (50.9)			
BMI					
Mean±SD* (range)	27.06±6.08 (17.58-39.84)	27.77±4.39 (18.69-37.37)	0.8313	158	0.4071
Underweight	5 (6.25)	0	1.282	2	0.108
Normal	25 (31.25)	21 (26.25)			
Overweight	25 (31.25)	37 (46.25)			
Obese	25 (31.25)	22 (27.5)			

*Independent sample *t*-test was used to compare means, **χ^2. BMI: Body mass index, SD: Standard deviation

Table 2: Clinical parameters in type 2 diabetes mellitus patients

Variables	Diabetic (*n*=80)	Good control (FBG <7.0), *n* (%)	Poor control (FBG >7.0), *n* (%)
FBG (mmol/L)*, mean±SD (range)	7.6±3.5 (3.3-21.3)	47 (58.75)	33 (41.25)
Variables	**Diabetic (*n*=80)**	**HBAic <6.5**	**HBAic >6.5**
HBAic (%) mean ± SD (range)	7.02.71 (4.0-15.0)	44 (56.41)	34 (43.59)

*Independent sample *t*-test was used to compare means, Chi square test was used to compare proportion. SD: Standard deviation, BMI: Body mass index, FBG: Fasting blood glucose, HbAic: Glycated haemoglobin

Table 3: Relationship of diabetes mellitus duration with heel pad thickness

	n (%)	T2DM, mean±SD (range)	*t**	Df	*P*
Heel pad thickness (mm) (years duration)					
<6	43 (53.75)	16.4±2.8	0.1280	78	0.8984
>6	37 (46.25)	16.5±2.9			
Plantar fascia thickness (cm) (years duration)					
<6	43 (53.75)	3.6±1.1	1.8210	78	0.0724
>6	37 (46.25)	3.2±0.9			

*Independent *t*-test was used to compare means. SD: Standard deviation, T2DM: Type 2 diabetes mellitus patients

Table 4: Comparison of the body mass index with the heel pad and plantar fascia thickness

Variables	*n*	*t**	*P*
BMI_mean HPT	160	0.3134	0.0001
Diabetics	80	0.4397	<0.0001
Nondiabetics	80	0.3150	0.0044
BMI_Mean PFT	160	0.2635	0.0008
Diabetics	80	0.2629	0.0184
Nondiabetics	80	0.2231	0.0467

*Spearman's rank correlation was used to check relationship. BMI: Body mass index, HPT: Heel pad thickness, PFT: Plantar fascia thickness

Results

Characteristics of the study population

A total of 160 study participants were recruited comprising 80 adult subjects with diabetes mellitus aged 40 years and above, and an equal number of age and sex-matched control subjects. The epidemiologic statistics are listed in Table 1.

The diabetic group was further divided into well-controlled and poor-controlled subgroups, as shown in Table 2.

The mean duration of Diabetes Mellitus in the Diabetic subjects was 7.15 ± 5.62 years with a range of 0.3–34 years while the median was 6 years. The skewed distribution of the duration of DM led to the use of the median of 6 years in a grouping of DM patients to those with a duration of diabetes mellitus below 6 years and those above 6 years. Comparing the mean HPT and plantar fascia thickness of diabetic patients whose duration of diabetes mellitus is below 6 years with those >6 years showed no statistically significant difference between them ($P = 0.8984$) and ($P = 0.0724$), respectively, as shown in Table 3.

Comparison of the body mass index with the heel fat pad and plantar fascia thickness

Using Spearman's rank correlation to test the relationship between the BMI of diabetic patients and the mean of the HPT, it revealed a moderate positive correlation, with good statistical significance (Spearman's rho = 0.4397, $P < 0.0001$). The relationship between the BMI of diabetic patients and the mean of the plantar fascia thickness showed a weak positive correlation, with a statistically significant difference (Spearman's rho = 0.2635, $P = 0.0008$) [Table 4].

The relationship between the BMI of nondiabetic subjects and their mean heel pad fat thickness also showed a weak correlation with some statistical significance (Spearman's rho-0.3150, $P = 0.0044$). This was also the finding with the plantar fascia thickness (Spearman's rho-0.2231, $P = 0.0467$).

Heel pad thickness ultrasound measurements

Comparison between the heel pad thickness of diabetics and nondiabetics

Diabetic patients had significantly thicker heel pads compared to nondiabetic patients [Table 5].

Similar results were noted on the left where the range of the HPT of diabetics and nondiabetics were 10.4–24.0 mm and 12.0–19.7 mm, respectively, with a mean of 16.1 ± 2.8 mm and 14.1 ± 1.5 mm, respectively ($P < 0.001$) [Table 6].

Comparison of mean plantar fascia thickness in diabetics and nondiabetics

There was a statistically significant difference between the mean plantar fascia thickness in the diabetics compared to that of the nondiabetic patients ($P = 0.0142$). Statistically, a significant difference was noted between the right plantar fascia thickness of the diabetics and nondiabetic patients ($P = 0.0319$). There was however no statistically significant difference between the left plantar fascia thickness of diabetics and the controls ($P = 0.0516$) [Figure 3].

Relationship of diabetes mellitus duration with heel pad and plantar fascia thickness

The plantar fascia thickness, HPT of the right and left foot, and duration of Diabetes Mellitus in diabetics were not normally distributed hence Spearman's rank correlation was used to test the relationship between these continuous variables. A Spearman's rank correlation was used to test the relationship between duration of diabetes and mean HPT. This yielded a Spearman's rho of 0.0760 ($P = 0.5030$) meaning there was no linear relationship between these variables. Likewise with the mean plantar fascia thickness; Spearman's rho was 0.2130 ($P = 0.0579$). These showed that the duration of diabetes mellitus does not determine the heel pad fat and plantar fascia thickness.

Relationship of heel pad thickness and plantar fascia thickness with history of foot ulcers/foot ulcer status in diabetic patients

Diabetic patients with foot ulcer history had significantly thinner heel pads and thinner plantar fascia compared to diabetic patients without foot ulcer history [Tables 7 and 8].

Table 5: Comparison between the heel pad thickness of diabetics and nondiabetics

	Mean±SD (range)		*t**	Df	*P*
	Diabetic (*n*=80)	Nondiabetic (*n*=80)			
Heel pad thickness (mm)					
Right	16.8±2.9 (11.2-23.9)	14.2±1.6 (11.9-20.0)	7.0679	158	<0.001
Left	16.1±2.8 (10.4-24.0)	14.1±1.5 (12.0-19.7)	5.6740	158	<0.001
Plantar fascia thickness (mm)					
Right	3.4±1.0 (1.5-6.4)	3.7±0.7 (2.0-5.9)	2.1645	158	0.0319
Left	3.4±1.5 (1.8-12.8)	3.9±1.5 (2.0-12.6)	1.9611	158	0.0516

*Independent *t* test was used to compare means. SD: Standard deviation

Table 6: Comparison between the right and left heel pad thickness among the study participants

Heel pad thickness (mm)	Study participants (*n*=160), mean±SD (range)	*t**	Df	*P*
Right	15.5±2.7 (11.2-23.9)	4.5435	159	<0.001
Left	15.1±1.9 (10.4-24.0)			

*Independent *t*-test was used to compare means. SD: Standard deviation

Table 7: Relationship of heel pad thickness with history of foot ulcers/foot ulcers status of diabetic patients

Heel pad thickness (mm)	*n* (%)	T2DM, mean±SD	*t**	Df	P
No foot ulcers history	54 (67.5)	17.8±2.1	8.6274	78	<0.0001
Foot ulcers history	26 (32.5)	13.7±1.8			

*Independent *t*-test was used to compare means. SD: Standard deviation, T2DM: Type 2 diabetes mellitus patients

Table 8: Relationship of plantar fascia thickness with history of foot ulcers/foot ulcers status of diabetic patients

Plantar fascia thickness (mm)	*n* (%)	T2DM, mean±SD	*t**	Df	*P*
No foot ulcers history	54 (57.5)	3.7±1.1	3.7326	78	0.0004
Foot ulcers history	26 (32.5)	2.8±0.7			

*Independent *t*-test was used to compare means. SD: Standard deviation, T2DM: Type 2 diabetes mellitus patients

Determining the predictors of foot ulcers in T2DM subjects

A binary logistic regression was computed to determine the predictors of foot ulcers, which showed that HPT was a single significant predictor of foot ulcer ($P < 0.0001$) and could explain up to 60.44% of the variability in foot ulcers.

Discussion

In the present study, the prevalence of foot ulcers in T2DM patients was 2.5%. This figure is much lower than the 22.5% reported by Gooding *et al*.[12] in the United State of America. The observed disparity may be due to the difference in the study population and ethno-racial differences. However, a total of 26 (32.5%) out of the 80 diabetic patient had a history of foot ulcers. In our study, the heel pad was significantly thicker in diabetic patients compared to nondiabetic control subjects ($P < 0.001$). This finding is similar to what was observed by Gretchen *et al*.[15] (California, USA) in their high-resolution ultrasound scan evaluation of 38 diabetes patients and 10 healthy patients' HPT. They found that the mean HPT for diabetics was statistically larger than that for control ($P < 0.01$). On the other hand, in the study undertaken by Gooding *et al*.,[12] the HPT in control was found to be greater than that of the diabetics and was significant statistically. The HPT was then greater than that of diabetics with foot ulcer. The mean HPT and mean PFT of diabetic patients with foot ulcers or history of foot ulcers were greater than those without in this study. There was a statistically significant difference between the HPT of diabetic subjects with foot ulcers or a history of foot ulcers and those without a history of foot ulcers ($P < 0.001$).

The average HPT of healthy adults in the study done by Gooding *et al*.[15] was 16.6 mm while that of the diabetics was 17.8 mm. This was quite higher than what was recorded in the index study which measured 14.2 mm in nondiabetic control and 16.5 mm in diabetics. This disparity might have been due to racial differences between the two study groups. The mean HPT of males in the study done by Udoh *et al*.[21] among healthy Nigerians was 14.3 ± 1.24 mm while that of the females was 12.14 ± 1.26 mm which were similar to what was obtained in the index study where the average HPT in the nondiabetic males and females participants measured 14.6 ± 1.71 mm and 13.7 ± 1.06 mm respectively.

In this study, the plantar fascia thickness was slightly greater in the control group compared to that of the diabetic patients. This is quite different from what was found in the study of Abate *et al*.[22] who focused on the possible effects that BMI

might have on Achilles tendon and plantar fascia in recently diagnosed Type 2 diabetics. In all the examined fifty-one diabetic subjects, who were free from diabetic complications, the thickness of both the plantar fascia and Achilles tendon was increased compared to the controls ($P < 0.001$, $P = 0.01$, $P = 0.003$, respectively). The plantar fascia thickness and BMI values (r = 0.749, $P < 0.0001$) were significantly related. However, in the index study, there was weak positive correlation between the mean plantar fascia thickness and the BMI of diabetic patients (Spearman's rho-0.26) and the relationship was statistically significant ($P = 0.0184$).

In the study done by Akindeju *et al*.[23] in Benin city, Nigeria on four hundred and twenty apparently healthy volunteers comprising of participants with male to female ratio of 1:2.5. Study participants weighed between 37.0 kg and 123.0 kg (mean 68.5 ± 13.7 kg) while the BMI ranged from 14.2 kg/m^2 to 40.9 kg/m^2. The mean HPT for the study participants was 17.7 ± 2.5 mm. The mean HPT for the right foot was 17.7 ± 2.5 mm (range 11.9 mm–25.0 mm), while the mean for the left was 17.7 ± 2.4 mm (range 12.5 mm–25.0 mm). This was much higher than the mean HPT of nondiabetic patients in the index study which was 14.2 ± 1.5 mm (range 12.0–19.8 cm). The mean HPT for the right foot was 14.2 ± 1.6 mm (range 11.9–20.0 mm) and the mean for the left was 14.1 ± 1.5 mm (range 12.0–19.7 mm) The difference in mean HPT for the right and left foot was not statistically significant ($P = 0.320$) in the study done by Akindeju *et al*.[23] contrary to the finding in the index study ($P = 0.0319$) and ($P = 0.0142$) for the right and left foot respectively. The mean HPT for males was 18.2 ± 2.5 mm and for females 17.5 ± 2.4 mm; this was not statistically significant ($P = 0.950$ right foot and $P = 0.683$ left foot) in Akindeju *et al*.'s study. This is similar to what was found in the index study ($P = 0.9633$) but incongruent to the findings by Udoh *et al*.[21] where analysis of variance showed that there was a significant difference between the value of HPT obtained from the male subjects and those obtained from the females. This could be due to the difference in the subject sample size and probably because the study was carried out among healthy Nigerians only. The mean HPT on the right feet in both diabetic and control subjects in this study was greater than that of the left feet [Table 5].

Twenty-six patients with a history of foot ulcers were seen among the diabetic patients of this study (2 out of which were having foot ulcers). This could have been due to the fact that most of the diabetic patients have only been diagnosed a few years ago with the median duration of diabetes mellitus being 6 years. Longer duration of DM is associated with the development of calf shortening due to structural changes, glycated collagen of tendon fibers, and the presence of peripheral sensory neuropathy, contributing strong risk factors for forefoot ulceration. Harmful pressure distribution contributes to hyperkeratosis and ulcer formation in the plantar surface.[24,25] In general, the duration of diabetes mellitus did not determine the HPT and the plantar fascia thickness in the index study. A binary logistic regression was computed to determine the predictors of foot ulcers, only HPT was a statistically significant predictor of foot ulcers ($P < 0.0001$) among other variables [Table 9].

Table 9: Determining the predictors of foot ulcers in type 2 diabetes mellitus patients subjects

Variables	*n*	Statistic*	*P*
Mean HPT	80	3.80	<0.001
Mean PFT	80	1.40	0.161
Age	80	0.81	0.420
Gender	80	0.00	0.999
BMI	80	0.80	0.425
DDM	80	0.82	0.411

*Binary regression analysis was used to determine the predictor of foot ulcers. BMI: Body mass index, HPT: Heel pad thickness, PFT: Plantar fascia thickness, DDM: Duration of diabetes mellitus

After multiple regression analysis was run to predict HPT from age, gender, BMI, duration of diabetes, FBG, and HBAic, only BMI added statistically significantly to the prediction ($P < 0.0001$) in the index study.

Conclusion

The HPT of the T2DM was statistically different from that of the nondiabetics controls. The right HPT was also statistically different from that of the left in the diabetics. There was a statistically significant difference between the plantar fascia thickness of the diabetics and the nondiabetics. However, there was no statistically significant difference between the right and left plantar fascia thickness. There was no statistically significant difference in the HPT between males and females. There was however statistically significant difference between the HPT of T2DM with foot ulcers or history of foot ulcers and those without. HPT was shown to be a statistically significant predictor of foot ulcers among other variables.

The duration of diabetes mellitus did not determine the HPT and the plantar fascia thickness. There was a moderate positive correlation between BMI and HPT with some statistical significance. Similarly, the relationship between the BMI of diabetic patients and their average plantar fascia thickness showed a weak positive correlation with good statistical significance.

BMI was the only variable that added statistically significantly to the prediction of HPT following multiple regression analysis.

Recommendations

Sonographic measurement of the HPT and plantar fascia thickness can be an additional imaging modality in the evaluation of diabetic patients' feet.

Further local ultrasound study of the HPT with a larger population of diabetics should be carried out by researchers.

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Conflicts of interest

There are no conflicts of interest.

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