Comparative Doppler Ultrasound Findings of Foot Arteries in Patients with Type 2 Diabetes Mellitus and Normoglycaemic Patients

# Introduction

**Abstract**

**Aim of the Study:** The aim of this study was to investigate lower extremity peripheral artery disease (LEPAD) in the foot arteries of patients with type 2 diabetes mellitus, with and without clinical symptoms of arterial insufficiency, using triplex Doppler ultrasound. **Materials and Methods:** Forty-seven consecutive adult subjects with type 2 diabetes mellitus (T2DM) and 47 age-matched and sex-matched non-diabetic controls were recruited (94 limbs each). Ankle-brachial index (ABI), fasting blood glucose assay, glycated haemoglobin assay and triplex sonography of the dorsalis pedis artery (DPA) and the distal posterior tibial artery (PTA) in both feet were performed. **Results:** The mean age of the subjects and controls were 60.21 ± 7.68 years and 56.81 ± 9.05 years (*P* > 0.05). The mean duration of diabetes mellitus was 10.4 ± 5.8 years. Crampy calf pain was the most common presenting symptom. Twenty-one (22.3%) of the 94 limbs of T2DM subjects had an abnormal ABI. Abnormal triplex Doppler waveform was seen in more than half of the PTA (57/94; 60.6%) and DPA (55/94; 58.5%). Forty-one (43.6%) of the 94 diabetic limbs had plaques in the PTA, while plaques were present in the DPA of 52 (55.3%) diabetic limbs. **Conclusion:** LEPAD is common in T2DM with a higher prevalence on triplex Doppler sonography compared to ABI values.

**Keywords:** *Ankle-brachial index, diabetes mellitus, diabetic foot, doppler ultrasonography, peripheral artery disease*

Diabetes mellitus (DM) is a metabolic disorder defined by chronic hyperglycaemia secondary to impaired insulin production, faulty insulin action, or a combination of the two.[1] Type 1 DM is caused by an autoimmune reaction in which the body’s immune system attacks the insulin-producing beta cells of the pancreas leading to a deficiency of insulin production. Type 2 DM results from the inability of the body’s cells to respond fully to insulin (insulin resistance).[1,2]

The current estimated prevalence of DM worldwide is 9.3% and is forecasted to reach 10.2% and 10.9% by 2030 and 2045, respectively.[3] In Nigeria, the prevalence of T2DM is 3.9% according to the current Diabetes Atlas of the International Diabetes Federation.[3]

Long term persistent hyperglycemia in T2DM leads to microvasculopathy (retinopathy, nephropathy, neuropathy) or macrovasculopathy (stroke, myocardial infarction, peripheral vascular diseases).[4,5]

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Atherosclerosis is one of the dreaded complications of T2DM.[6] Peripheral arterial disease (PAD)/Peripheral Vascular Disease (PVD) is an important manifestation of systemic atherosclerosis characterized by occlusive changes in the lower limb arteries.[7] PAD/ PVD is an umbrella term that encompasses various atherosclerotic and aneurysmal diseases affecting the extra-coronary circulation.[8] PAD affecting the lower limb arteries is termed lower extremity peripheral arterial disease (LEPAD).[9]

LEPAD is a frequent complication in patients with T2DM.[7] It affects over 200 million individuals globally. People with T2DM are 2–4 times more likely than the general population to have this condition.[9] Study-to- study variation in prevalence of LEPAD can be explained by differences in LEPAD definition, age and ethnicity, and disease duration.[9] The reported prevalence is higher when defined with abnormal ankle-brachial index (ABI) than when defined clinically.[9]

Patients with LEPAD, even without a history of myocardial infarction or ischemic stroke, have approximately the same relative risk of

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death from cardiovascular events as do patients with a history of coronary or cerebrovascular disease.[10,11] The lower the ABI, the greater the risk of cardiovascular events.[6,10] LEPAD is a major predictor of lower limb amputation in patients with diabetic foot wound (diabetic foot ulcer and diabetic foot gangrene).[12]

A prompt diagnosis of LEPAD in people with T2DM helps to initiate early treatment which can prevent attendant complications.[13] Thus, regular screening is considered an essential part of aggressive management as it can help to identify cases at risk of arterial disease/insufficiency so as to initiate intervention promptly.[7,14]

Conventional arteriography had been the gold standard for

hospital approved the study protocol before commencement (ADM/DCST/HREC/APP/2684).

### Subject selection

The study enrolled patients with T2DM who were ≥ 40 years old. They were recruited consecutively from the endocrinology clinic. Both newly diagnosed patients with T2DM and those already on anti-diabetic therapy were recruited. In all, 47 people with T2DM and 47 non-diabetic volunteer controls (Fasting blood glucose of 4.0 - 5.6 mmol/L, no history of leg pain or foot trauma, no systemic hypertension, dyslipidemia, smoking, sickle cell disease, or AIDS) constituted the participants.

**The sample size was calculated**[18] **using the following**

assessing the severity, location, and extent of LEPAD.[10] However, arteriography is invasive, uses ionising radiation, and iodinated contrast media. Consequently, several non-invasive

**formula**: *N* 

4 *Z*

*crit*

2 *p* 1 *p* *D*2

tests have been used to detect LEPAD in clinical practice. These tests include Computed Tomography Angiography (CTA), whole body Magnetic Resonance Angiography (WBMRA), Doppler ultrasonography and ankle-brachial index (ABI).[9] While CTA uses ionizing radiation and may not readily available; WBMRA is expensive, not readily available and may be unsuitable for claustrophobic patients.[10,11]

The ABI is cheap and inexpensive test but has low sensitivity in elderly individuals and people with T2DM.[15] In addition, medial arterial calcifications, incompressible arteries, operator skills and presence of diabetic peripheral neuropathy affect ABI measurement.[9] There is considerable variation in the sensitivity (range = 29–95%; median = 63%) and specificity (range = 58–97%; median = 93%) of ABI <0.9 as evidence of LEPAD.[9]

By contrast, Doppler ultrasound is a good imaging tool for diagnosing LEPAD, with a sensitivity of 93% and a specificity of 97%.[9] It is non-invasive, non-ionising, cost-effective, readily available, repeatable, and allows anatomical and hemodynamic (triphasic waveform indicates a normal artery without LEPAD) vascular studies independent of medial arterial calcifications.[9] Also, ultrasound-derived semi-quantitative scores might help improve the sonographic assessment of LEPAD.[16,17]

Diabetic foot disease is serious public health concern in our locality and worldwide. Most studies of LEPAD in T2DM in our population were based on clinical symptomatology and ABI. The aim of this study was to investigate the haemodynamic and sonomorphological changes in the foot arteries of patients with T2DM, with and without clinical symptoms of arterial insufficiency, using Doppler ultrasound.

# Materials and Methods

This article is based on a Fellowship dissertation submitted to the National Postgraduate Medical College of Nigeria. It was a prospective case-control study that was done from March 2019 to August 2019 at the radiology department of our tertiary institution. The Human Research and Ethics Committee of the

Where *N* = Sample size, *Zcrit*= Standard normal deviate = 1.96

corresponding to 95% Confidence interval (CI), *p* = Proportion of target population estimated to have LEPAD[13] which is 0.525, *D*= Desired level of precision taken as 0.3 (Based on assumed accuracy of arterial duplex ultrasound of about 70% in the pedal arteries,[19] 95% CI of ±15% was considered appropriate for this study), Substituting these into the formula yields *n* = 42.6 (Increased to 47 to allow for 10% attrition).

The initial diagnosis of diabetes mellitus was based any of the following criteria: fasting plasma glucose of ≥ 126 mg/dl (≥ 7.0 mmol/L) on two separate tests; symptoms of diabetes plus a random blood sugar of ≥ 200 mg/dl (≥11.1 mmol/l); two-hour plasma glucose ≥ 200 mg/dl (≥11.1mmol/l) during

an oral glucose tolerance test; or glycated hemoglobin (HbA1c) of ≥ 48 mmol/L (or ≥ 6.5 DCCT %).

Patients with type 2 diabetes mellitus were excluded if they had the following: previous revascularization surgery, history of foot trauma, co-existing Buergers disease (based on clinical history of on-and-off pain in the feet and hands in a patient who consumes tobacco), co-existing with systemic hypertension, and co-existing with sickle cell disease.[7,20]

### Clinical evaluation

Each subject had their weight (Kg) and height (m) checked to calculate their Body Mass Index (BMI) in Kg/m2 (i.e. BMI = Weight/height2). Clinical history was obtained from T2DM subjects to know the duration of illness (or age at diagnosis) and presence of symptoms of lower limb arterial insufficiency (intermittent claudication, foot pain at by resting, foot ulcer/gangrene).

Physical examination included a complete inspection of both feet noting the shape, any deformity, site and extent of foot lesions and evidence of gangrene. Arterial pulses in the posterior tibial and dorsalis pedis arteries were checked.

Blood screening for fasting blood glucose was done for all subjects (using Accucheck glucometer with test strips), while serum glycated haemoglobin concentration was assayed in

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the patients with T2DM only using portable kits (using PTS Diagnostics multi-test A1C System).

### Protocol for ankle-brachial index (ABI) measurement

The bilateral ankle-brachial index was obtained using handheld continuous Doppler device with 8.1 MHz transducer (Parks Medical Electronics, Aloha, Oregon, USA) and mercury sphygmomanometer with appropriate cuff size (about 12.5 cm wide; a cuff of 15 cm wide was used for the obese subjects). The established standard ABI measurement guidelines were adhered to.[15] The normal range for ABI was taken as 0.9– 1.3.[15]

### Ultrasound technique

Ultrasound examinations were done on a Toshiba Xario TUS-X200 (Toshiba Medical System Corporation, Japan) with

7.5 – 11 MHz transducer and Doppler function. Sonographic examinations of the dorsalis pedis artery was conducted in supine position with the limb bent at the knee at an angle of about 90°, while the posterior tibial artery (behind the medial malleolus) was examined with the patient in lateral position and the limb of interest dependent and slightly flexed in the knee.[14] These vessels were first identified on a transverse plane, after which the probe was rotated perpendicularly to demonstrate the arteries in the longitudinal plane. Doppler insonation was performed in the longitudinal plane using a sample gate of

* 1. mm placed centrally within the vascular lumen while the optimal Doppler angle of < 60o was maintained carefully.[14]

The following ultrasound assessments of the vessels were documented:

* B-mode
	+ 1. the external diameter of the vessels: measured from the echogenic outer margin of the near-wall to the outer margin of the far wall.[14]
		2. presence of plaque(s) described as an area along the vascular intima causing luminal narrowing which can be homogenous/heterogeneous, hypoechoic/hyperechoic/ dense calcific + acoustic shadowing.[21]
		3. Associated percentage diameter stenosis using the formula below.[21]

% *Diameter stenosis*  *A* *B* 100 / 1

*A*

Where:

A = Relative normal diameter distal to the stenosis

B = Luminal diameter at the level of the plaque/stenosis

* Spectral Doppler imaging to evaluate these parameters[14]:
1. the flow spectrum i.e., triphasic or biphasic or monophasic patterns
2. blood flow parameters, i.e., Peak Systolic Velocity (PSV), End-Diastolic velocity (EDV), Pulsatility Index (PI), and Resistance Index (RI).

If a segment could not be adequately evaluated, for instance because of severe calcifications, Doppler ultrasound was considered non-diagnostic.[14]

### Data analysis

IBM SPSS Statistics for Windows, version 20 (IBM Corp., Armonk, N.Y., USA) was used to analyze the data collected. Continuous variables like the age and vascular diameters are presented as mean (standard deviation) for each of the study groups. Categorical variables like gender, presence of plaque and spectral pattern are presented in percentages. Based on their individual ankle–brachial index, each limb of patients with T2DM were grouped into normal ABI and abnormal ABI and vascular sonographic changes were compared across the groups using Chi-square or Fischer’s exact test. A similar approach was used to compare association of sonographic vascular changes and serum HbA1c concentration (normal versus abnormal) and duration of DM (<10 years versus

≥10 years). Pearson’s test was used for correlational analysis

and students T-test for comparing continuous variables among the study groups. Significance level was set at *P* ≤ 0.05.

# Results

The study population comprised 47 subjects (Males = 16, Females = 31) with type 2 diabetes mellitus (T2DM) and 47 controls (Males = 21, Females = 26); each study group with 94 limbs. There was no statistically significant difference between the mean age of the subjects (60.21 ± 7.68 years) and the mean age of controls (56.81 ± 9.05 years) (*P* =0.052). The majority (37; 78.7%) of subjects with T2DM were 50 – 69 years old. The other characteristics of the study population are as shown in [Table 1].

The mean duration of diabetes mellitus was 10.4 ± 5.8 years. The duration of diabetes mellitus was <10 years in 19 subjects (40.4%), 10–19 years in 19 subjects (40.4%), 20–29 years in eight subjects (17.0%), and ≥30 years in one subject (2.1%).

Crampy calf pain relieved by resting (15; 31.9%), followed by absent arterial pulsations, was the most common presenting symptoms/signs of lower limb arterial insufficiency. Foot ulcer/ gangrene was seen in eight (17%) subjects with T2DM.

Seventy-three (77.7%) of the 94 limbs of the T2DM subjects had a normal ABI, while 21 (22.3%) had an abnormal ABI. The ABI was normal in 86 (91.5%) and abnormal in 8 (8.5%) of the 94 control limbs. The difference in the proportion of normal ABI and abnormal ABI between the subjects’ limbs and controls’ limbs was statistically significant (*P* = 0.009). However, there was no statistically significant difference between the mean ABI of the subjects’ right limbs vs. controls’ right limbs (*P* = 0.06), subjects’ left limbs vs. controls’ left limbs (*P* = 0.56), and subjects’ combined bilateral limbs vs. controls’ combined bilateral limbs (*P* = 0.06).

The B-mode and triplex Doppler ultrasound findings of the posterior tibial arteries and dorsalis pedis arteries are detailed in [Tables 2, 3], respectively.

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### Table 1: Characteristics of subjects with T2DM and Controls

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Variables** | **T2DM****n = 47** | **Controls****n = 47** | **Statistics** | **df** | **P value** |
| Gender, *n (%)* |  |  |  |  |  |
| Male | 16 (34.0) | 21 (44.7) | 1.114 | 1 | 0.291 |
| Female | 31 (66.0) | 26 (55.3) |  |  |  |
| **Age in years** |  |  |  |  |  |
| *Mean ± SD* | 60.21 ± 7.68 | 56.81 ± 9.05 | 1.966 | 92 | 0.052 |
| ***n (%)*** |  |  |  |  |  |
| 40-49 | 4 (8.5) | 10 (21.3) |  |  |  |
| 50-59 | 19 (40.4) | 16 (34.0) | 3.257 | 3 | 0.354 |
| 60-69 | 18 (38.3) | 17 (36.2) |  |  |  |
| >/=70 | 6 (12.8) | 4 (8.5) |  |  |  |
| **BMI** |  |  |  |  |  |
| *Mean ± SD (kg/m2*) | 25.98 ± 5.80 | 27.7 ± 5.26 | -1.505 | 92 | 0.136 |
| ***n (%)*** |  |  |  |  |  |
| Underweight | 1 (2.1) | 2 (4.3) |  |  |  |
| Normal | 24 (51.1) | 13 (27.7) | 5.505 | 3 | 0.138 |
| Overweight | 11 (23.4) | 17 (36.2) |  |  |  |
| Obese | 11 (23.4) | 15 (31.9) |  |  |  |
| **WHR** |  |  |  |  |  |
| *Mean ± SD (kg/m2*) | 0.94 ± 0.07 | 0.97 ± 0.08 | -2.053 | 92 | **0.043** |
| **FBG in *mmol/L*** |  |  |  |  |  |
| *Mean ± SD* | 7.65 ± 3.76 | 4.88 ± 2.55 | -1.804 | 92 | **0.002** |
| ***n (%)*** |  |  |  |
| Good control (FBG < 7.0) | 30 (63.8) | NA | - - - |
| Impaired control | 17 (36.2) | NA | - - - |
| **HBA1c in *%*** |  |  |  |  |  |

*Mean ± SD* 7.51 ± 2.25 NA - - -

|  |  |
| --- | --- |
| ***n (%)*** |  |
| Good control (HBA1c < 6.5) | 21 (44.7) | NA | - - - |
| Impaired control | 26 (55.3) | NA | - - - |

χ2 - chi square test statistic; T2DM- type 2 diabetes mellitus; BMI – body mass index; WHR – waist to hip circumference ratio; FBG – fasting blood glucose in mmol/L; HBA1c – glycated haemoglobin concentration in %; NA- not applicable

Thirteen (13.8%) of the diabetic limbs had hemodynamically significant (≥ 50%) luminal narrowing/diameter stenosis of the posterior tibial artery (PTA) compared to only 2 (2.1%) limbs with hemodynamically significant PTA stenosis among the control limbs (*p* = 0.001). Seven (7.4%) of the diabetic limbs had complete posterior tibial artery occlusion [Table 2] Abnormal posterior tibial artery waveform (not triphasic) was observed in 57 (60.6%) diabetic limbs, which was statistically significantly higher (*P* = 0.002) than the 40 (42.5%) abnormal PTA waveform among the control limbs [Table 2]. The posterior tibial artery mean Pulsatility index of the subjects was significantly lower (*P* = 0.03) than the posterior tibial artery mean Pulsatility index of controls [Table 2]. Similarly, the posterior tibial artery mean Resistive index of the subjects was significantly lower (*P* = 0.008) than the posterior tibial artery mean Pulsatility index of controls [Table 2].

Twenty-four (14.9%) of the diabetic limbs had hemodynamically significant (≥ 50%) luminal narrowing/diameter stenosis of the dorsalis pedis artery (DPA) compared to only one (1.1%) limb with hemodynamically significant DPA stenosis among the control limbs (*P* = 0.004). One (1.1%) of the diabetic limbs had complete dorsalis pedis artery occlusion [Table 3].

Abnormal (not triphasic) dorsalis pedis artery waveform was observed in 55 (58.5%) diabetic limbs [Figure 1], which was statistically significantly higher (*p* = 0.003) than the 39 (41.5%) abnormal DPA waveform among the control limbs. The dorsalis pedis artery mean Pulsatility index of the subjects (5.98 ± 4.60) was extremely significantly lower (*p* < 0.001) than the dorsalis pedis artery mean Pulsatility index of controls (8.17 ± 3.76) [Table 3].

A three-way comparison of T2DM subjects with normal ankle brachial index (ABI), T2DM subjects with abnormal ABI, and the controls was performed.

Posterior tibial artery (PTA) intimal plaques were more prevalent in diabetic limbs with abnormal ABI (57.1%; 12/21) than diabetic limbs with normal ABI (39.7%; 29/73) and the limbs of controls (31.3%; 20/94) (*P* = 0.001) [Table 4]. Similarly, loss of normal PTA pulsatile waveform was more frequent in diabetic limbs with abnormal ABI than diabetic limbs with normal ABI and the limbs of controls (76.2% vs. 56.2% vs. 42.6%, respectively) (*P* =0.005) [Table 4]. Also, the presence ≥ 50% PTA luminal diameter stenosis was statistically significantly more frequent in diabetic limbs with

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### Table 2: Ultrasound findings in the posterior tibial artery of T2DM subjects and Controls

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Ultrasound findings** |  | **T2DM****N = 94** | **Controls****n = 94** | **Statistics** | **df** | **P value** |
| B- mode | Diameter, |  |  |  |  |  |
|  | *Mean ± SD (mm)* | 1.99 ± 0.73 | 2.12 ± 0.51 | -1.490\* | 186 | 0.138 |
|  | Intimal plaques, *n (%)* |  |  |  |  |  |
|  | No plaque | 53 (56.4) | 74 (78.7) | 15.569\*\* | 3 | 0.001 |
|  | Hypoechoic | 7 (7.4) | 8 (8.5) |  |  |  |
|  | Hyperechoic | 29 (30.9) | 8 (8.5) |  |  |  |
|  | Calcified | 5 (5.3) | 4 (4.3) |  |  |  |
|  | Diameter stenosis *n (%)* |  |  |  |  |  |
|  | 0-15 % | 72 (76.6) | 91 (96.8) |  |  |  |
|  | 16-49% | 9 (9.6) | 1 (1.1) | 17.615\*\* | 3 | 0.001 |
|  | 50-69% | 6 (6.4) | 2 (2.1) |  |  |  |
|  | >/=70% | 7 (7.4)# | 0 (0.0) |  |  |  |
| Doppler mode | Spectral pattern *n (%)* |  |  |  |  |  |
|  | Triphasic (Normal) | 37 (39.4) | 54 (57.4) |  |  |  |
|  | Biphasic | 41 (43.6) | 38 (40.4) | 14.623\*\* | 3 | 0.002 |
|  | Monophasic | 10 (10.6) | 2 (2.1) |  |  |  |
|  | No flow | 6(6.4) | 0 (0.0) |  |  |  |
|  | Velocities/Velocity ratios*Mean ± SD* |  |  |  |  |  |
|  | PSV (cm/s) | 43.7 ± 20.7 | 45.6 ± 13.4 | -0.774\* | 94 | 0.440 |
|  | EDV (cm/s) | 9.37 ± 6.14 | 9.38 ± 3.75 | -0.017\* | 94 | 0.987 |
|  | RI | 0.88 ± 0.25 | 1.02 ± 0.44 | -2.701\* | 94 | 0.008 |
|  | PI | 6.20 ± 5.52 | 7.79 ± 4.36 | -2.195\* | 186 | 0.029 |

\*Independent sample t-test; \*\*χ2 - chi-square test statistic; #6 of these 7 subjects had complete (100%) posterior tibial artery occlusion. T2DM- type 2 diabetes mellitus; df-degreee of freedom; PSV- peak systolic velocity; EDV- end-diastolic velocity; RI-resistive index; PI- pulsatility index.

### Table 3: Ultrasound findings in the dorsalis pedis artery of T2DM subjects and Controls

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Ultrasound findings** |  | **T2DM****N = 94** | **Controls****n = 94** | **Statistics** | **df** | **P value** |
| B- mode | Diameter, |  |  |  |  |  |
|  | *Mean ± SD (mm)* | 2.60 ± 0.65 | 2.63 ± 0.51 | -0.327\* | 186 | 0.744 |
|  | Intimal plaques, *n (%)* |  |  |  |  |  |
|  | No plaque | 42 (44.7) | 71 (75.5) | 24.157\*\* | 3 | <0.001 |
|  | Hypoechoic | 5 (5.3) | 7 (7.4) |  |  |  |
|  | Hyperechoic | 38 (40.4) | 15 (16.0) |  |  |  |
|  | Calcified | 9 (9.6) | 1 (1.1) |  |  |  |
|  | Diameter stenosis *n (%)* |  |  |  |  |  |
|  | 0-15 % | 75 (79.8) | 90 (95.7) | 13.171\*\* | 3 | 0.004 |
|  | 16-49% | 5 (5.3) | 3 (3.2) |  |  |  |
|  | 50-69% | 12 (12.8) | 1 (1.1) |  |  |  |
|  | >/=70% | 2 (2.1)# | 0 (0.0) |  |  |  |
| Doppler mode | Spectral pattern *n (%)* |  |  |  |  |  |
|  | Triphasic (Normal) | 39 (41.5) | 55 (58.5) | 14.123\*\* | 3 | 0.003 |
|  | Biphasic | 41 (43.6) | 38 (40.4) |  |  |  |
|  | Monophasic | 13 (13.8) | 1 (1.1) |  |  |  |
|  | No flow | 1 (1.1) | 0 (0.0) |  |  |  |
|  | Velocities/Velocity ratios*Mean ± SD* |  |  |  |  |  |
|  | PSV (cm/s) | 43.9 ± 17.7 | 42.7 ± 14.9 | -0.503\* | 186 | 0.616 |
|  | EDV (cm/s) | 9.38 ± 7.49 | 8.74 ± 3.99 | 0.734\* | 186 | 0.464 |
|  | RI | 0.95 ± 0.12 | 0.98 ± 0.03 | -1.626\* | 186 | 0.106 |
|  | PI | 5.98 ± 4.60 | 8.17 ± 3.76 | -3.569\* | 186 | <0.001 |

\*Independent sample t test; \*\*χ2 - Chi square test statistic; #1 of these 2 subjects had complete (100%) dorsalis pedis artery occlusion. T2DM- type 2 diabetes mellitus; df-degree of freedom; PSV- peak systolic velocity; EDV- end diastolic velocity; RI-resistive index; PI- pulsatility index.

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**Figure 1: Triplex Doppler sonogram showing a severely stenosed dorsalis pedis artery (A) with abnormal monophasic waveform devoid of diastolic flow (B)**

### Table 4: Comparison of sonographic findings in foot arteries of T2DM patients with normal ABI, abnormal ABI and

**non-diabetic control subjects**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **USS Findings** | **Normal ABI** |  | **Abnormal ABI** |  | **Controls (C)** |  |  | ***p* values\*** |  |
|  | **(nABI)** |  | **(abABI)** |  |  |  | **nABI vs.** | **nABI vs.** | **abABI vs.** |
|  | **N = 73** |  | **N = 21** |  | **N = 94** |  | **abABI** | **C** | **C** |
| **Dorsalis pedis artery,*****n (%)*** |  |  |  |  |  |  |  |  |  |
| Intimal plaque | 34 (46.6) |  | 18 (85.7) |  | 23 (24.5) |  | **0.001** | **0.003** | **<0.001** |
| ≥50% diameter stenosis | 11 (15.1) |  | 3 (14.3) |  | 1 (1.1) |  | 0.929 | **0.001** | **0.003** |
| Loss of arterial resistance | 39 (53.4) |  | 16 (76.2) |  | 39 (41.5) |  | 0.062 | 0.125 | **0.004** |
| **Posterior tibial artery*****n (%)*** |  |  |  |  |  |  |  |  |  |
| Intimal plaque | 29 (39.7) | 12 (57.1) | 20 (31.3) | 0.156 | **0.009** | **0.001** |
| ≥50% diameter stenosis | 9 (12.3) | 4 (19.0) | 2 (2.1) | 0.432 | **0.008** | **0.002** |
| Loss of arterial resistance | 41 (56.2) | 16 (76.2) | 40 (42.6) | 0.098 | 0.081 | **0.005** |

\*p values are based on χ2 - Chi-square test statistic

abnormal ABI (19.0%; 4/21) and diabetic limbs with normal ABI (12.3%; 9/73) than the limbs of controls (2.1%; 2/94) [Table 4].

In the dorsalis pedis artery (DPA), intimal plaques were more prevalent in diabetic limbs with abnormal ABI (85.7%; 18/21) than diabetic limbs with normal ABI (46.6%; 34/73) and the limbs of controls (24.5%; 23/94) (*p* < 0.001) [Table 4]. Similarly, loss of normal DPA pulsatile waveform was more frequent in diabetic limbs with abnormal ABI than diabetic limbs with normal ABI and the limbs of controls (76.2% vs. 53.4% vs. 41.5%, respectively) (*P* =0.004) [Table 4]. The presence ≥ 50% DPA luminal diameter stenosis was statistically significantly more frequent in diabetic limbs with normal ABI (11/73; 15.1%) and diabetic limbs with abnormal ABI (3/21; 14.3%) than the limbs of controls (1/94; 1.1%) [Table 4].

The relationships between glycated haemoglobin concentration (HBA1C), ankle brachial index, and ultrasonographic changes in the foot arteries of subjects with T2DM are shown in [Table 5].

[Table 6] shows a multivariate logistic regression model depicting the relationship between ultrasound findings (outcome variables) and selected predictor variables (covariates).

# Discussion

LEPAD is a clinical disorder characterized by stenosis or occlusion of lower limb arteries, usually caused by atherosclerosis in those >40 years old. It is vital to diagnose PAD at the asymptomatic stage when treatment (lifestyle modification, medications, or revascularization procedures) would still be effective and cheaper.

Of the 94 bilateral lower limbs of subjects with T2DM in this study, 21 (22.3%) had an abnormal ABI, 57 (60.6%) had abnormal posterior tibial artery waveform, while 55 (58.5%) had abnormal dorsalis pedis artery waveform. In other words, evaluation of ABI in the T2DM yielded a LEPAD prevalence of 22.3%, while triplex Doppler sonography of the pedal arteries yielded a LEPAD prevalence of 58.5%-60.6%.

The prevalence of LEPAD in diabetic patients as reported by previous studies include Hur *et al.*[22] in South Korea (ABI = 5.6%, abnormal Doppler spectral waveform = 28.7%); Leoniuk *et al.*[14] in Poland (ABI = 0%, Doppler = 30.4%); Adebayo[23] in Ile-Ife, Nigeria (ABI = 19.7%, Doppler= 33.9%); Agboghoroma[24] in Jos, Nigeria (ABI = 38.5%); Shaheen *et al.*[7] in Pakistan (ABI = 46%, Doppler = 62%); Janssen[25] in Germany (Doppler = 54%); Akalu *et al.*[26] in Ethiopia

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### Table 5: Association between glycated haemoglobin concentration (HBA1C), ankle brachial index, and ultrasonographic changes in the foot arteries of T2DM subjects

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Variables,** |  | **HBA1C (*%*) *χ2*** | ***df*** | ***p* values** |
| ***n (%)*** | **< 6.5** | **≥ 6.5** |  |  |
|  | **N = 42** | **N = 52** |  |  |
| ***Ankle brachial index (ABI)*** |  |  |  |  |
| ABI category Normal | 36 (85.7) | 37 (71.2) 2.839 | 1 | 0.092 |
| Abnormal | 6 (14.3) | 15 (28.8) |  |  |
| ***Dorsalis pedis artery ultrasound*** |  |  |  |  |
| Intimal plaque Absent | 25 (59.5) | 17 (32.7) 6.767 | 1 | **0.009** |
| Present | 17 (40.5) | 35 (67.3) |  |  |
| ≥50% diameter stenosis Absent | 38 (90.5) | 42 (80.8) 1.727 | 1 | 0.189 |
| Present | 4 (9.5) | 10 (19.2) |  |  |
| Loss of arterial resistance Absent | 25 (59.5) | 14 (26.9) 10.172 | 1 | **0.001** |
| Present | 17 (40.5) | 38 (73.1) |  |  |
| ***Posterior tibial artery ultrasound*** |  |  |  |  |
| Intimal plaque Absent | 26 (61.9) | 27 (51.9) 0.941 | 1 | 0.332 |
| Present | 16 (38.1) | 25 (48.1) |  |  |
| ≥50% diameter stenosis Absent | 41 (97.6) | 40 (76.9) 8.351 | 1 | **0.004** |
| Present | 1 (2.4) | 12 (23.1) |  |  |
| Loss of arterial resistance Absent | 25 (59.5) | 12 (23.1) 12.931 | 1 | **<0.001** |
| Present | 17 (40.5) | 40 (76.9) |  |  |

*FBG – fasting blood glucose; χ2 – Chi square; df- degree of freedom.*

### Table 6: Multivariate logistic regression model showing the relationship between ultrasound findings (outcome

**variables) and selected predictor variables (covariates)**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Outcome variables** |  |  | **Covariates****Odds ratio****(95% confidence interval)** |  |  | **Naegel-****Kerke R2** |
|  | **Age ≥60yrs** | **Duration of****DM (≥10yrs)** | **BMI****≥30Kg/m2** | **FBG****(≥7mmol/l)** | **HBA1C (≥6.5%)** |  |
| Abnormal | 0.6 | 3.2\* | 1.2 | 2.5 | 1.6 | 0.186 |
| ABI | (0.2 – 1.7) | (1.1 – 9.2) | (0.3 – 4.0) | (0.7 – 8.5) | (0.4 – 5.6) |  |
| Dorsalis pedis artery |  |  |  |  |  |  |
| Intimal plaque | 1.7 | 2.6\* | 1.4 | 0.8 | 3.3 | 0.169 |
|  | (0.7 – 4.5) | (1.1 – 6.6) | (0.4 – 4.3) | (0.3 – 2.4) | (1.2 – 9.2) |  |
| ≥50% diameter stenosis | 1.9 | 0.7 | 2.7 | 0.8 | 2.4 | 0.079 |
|  | (0.5 – 7.0) | (0.2 – 2.1) | (0.6 – 11.4) | (0.2 – 3.2) | (0.6 – 9.9) |  |
| Loss of arterial resistance | 1 | 0.6 | 1.5 | 1.1 | 3.7\* | 0.163 |
|  | (0.4 – 2.7) | (0.2 – 1.5) | (0.5– 4.8) | (0.4 – 3.2) | (1.4 – 10.3) |  |
| Posterior tibial artery |  |  |  |  |  |  |
| Intimal plaque | 1.8 | 0.4 | 3.4\* | 0.4 | 2.1 | 0.153 |
|  | (0.7 – 4.6) | (0.2 – 1.1) | (1.1 – 11.0) | (0.1 – 1.1) | (0.8 – 5.9) |  |
| ≥50% diameter stenosis | 2.1 | 0.7 | 0.6 | 1.3 | 11.1\* | 0.223 |
|  | (0.5 – 8.9) | (0.2 – 2.5) | (0.1 – 3.6) | (0.3 – 5.0) | (1.3 – 97.1) |  |
| Loss of arterial resistance | 3.2\* | 0.4 | 1.5 | 1.7 | 3.8\* | 0.280 |
|  | (1.1 – 9.0) | (0.2– 1.1) | (0.4 – 5.0) | (0.5 – 5.4) | (1.3 – 11.2) |  |

*\*p value< 0.05; BMI- body mass index; ABI- ankle-brachial index*

(Doppler = 30.7%); Ali[27] in Sudan (Doppler = 14%); and Das *et al.*[28] in India (Doppler = 25.5%). The reported prevalence of LEPAD is affected by the method of detection (history of intermittent claudication vs. palpation of arterial pulses vs. ABI vs. arterial intima-media thickness vs. Doppler arterial waveform abnormality vs. Doppler velocimetry and indices vs. conventional/CT/MR angiography), arterial site evaluated

(femoropopliteal vs. infrapopliteal arteries), age of the study population, race, and the duration of diabetes mellitus.[14,23]

Of all the sonographic parameters for detecting LEPAD, arterial intima-media thickness and arterial waveform are the most sensitive for early detection of peripheral artery disease.[14,23,29] Compared to conventional angiography (gold standard), triplex

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Doppler sonography has a sensitivity of 77–92% and specificity of 92–98%.[30]

This study evaluated two pedal arteries - PTA and DPA. These two arteries were also the only vessels assessed in some of the previous studies.[14,20,27] Janssen[25] examined the anterior tibial artery (ATA) and the PTA only, while Das *et al.*[28] analysed the ATA, PTA, and the DPA. Some previous studies evaluated all the lower limb arteries from the common femoral artery (CFA) to the DPA.[7,31-33] These different arterial sites studied can also affect the sonographic prevalence of LEPAD.

Creager *et al.*[34] noted that the prevalence rates of atherosclerosis in the lower limb arteries are 80 – 90%, 40 – 50%, and 30% in the femoropopliteal arteries, DPA, and aortoiliac arteries, respectively. Although LEPAD is said to be more prevalent in the femoropopliteal arteries, angiopathic changes in the infrapopliteal arteries (especially the DPA) are often more severe in diabetic limbs and are more predictive of the risk of amputation.[7,20,28] Even among the studies that examined allthe lower limb arteries, two[7,31] noted that changes in the DPA were the most significant. In a third study,[32] DPA changes were second to the femoropopliteal arterial abnormalities in hemodynamic significance.

In this study, 41 (43.6%) of the 94 diabetic limbs had plaques in the PTA. Plaques were present in the DPA of 52 (55.3%) diabetic limbs. Arterial intimal thickening, plaques, and calcifications were also reported by some of the previous researchers including Seth *et al.* (47.7% in DPA, 50.8% in PTA),[20] Janssen (54%),[25] and Das *et al.* (73.3%).[28] The disparity in the plaque rates may be due to the differences in patient age, diabetes duration, and co-existing systemic hypertension and hyperlipidaemia in some of the other studies.

The pathogenesis of lower limb arterial intimal thickening in peripheral artery disease entails an initial compromise of arterial function (abnormal arterial stiffness, flow-mediated dilation and loss of elasticity), followed by structural damage like fatty degeneration and foam cell formation which lead to intimal-medial thickening, plaque formation, and clogging of the arterial lumen.[35]

The mean luminal diameters of the PTA and DPA in the diabetic limbs were 1.99 ± 0.73 mm and 2.60 ± 0.65 mm, respectively. These values were not significantly different from those of controls. The arterial diameter values are broadly similar to those reported by Seth *et al.* (PTA = 2.2 ± 0.9 mm, DPA = 1.8 ± 0.8 mm),[20] and Leoniuk *et al.* (PTA = 2.2 ± 0.42 mm, DPA = 1.98 ± 0.39 mm).[14]

The mean pulsatility index (PI) and mean resistivity index (RI) of the PTA in the diabetic limbs were 6.20 ± 5.52 and

0.88 ± 0.25, respectively; while the mean PI and mean RI of the DPA were 5.98 ± 4.60 and 0.95 ± 0.12, respectively. The mean RI of this study are similar to those reported by Seth *et al.* (PTA = 0.95 ± 0.90, DPA = 0.82 ± 0.31).[20] However, the mean PI are lower than those of Seth *et al.* (PTA = 10.83 ± 17.8, DPA = 8.9 ± 11.7).[20] The wide disparity in pulsatility indices could be due to an error in statistical reporting by Seth *et al.*

Their Pulsatility indices are skewed in distribution and should have been reported as median values rather than as mean values.[20] Janssen[25] documented that a PI <1.2 in the ankle arteries (ATA or PTA) is a reliable criterion for the diagnosis of critical limb ischemia in diabetic patients with polyneuropathy, with a sensitivity of 87% and a specificity of 62%.

In conclusion, LEPAD is common in T2DM with a higher prevalence on triplex Doppler sonography compared to ABI values. The limitations of this study include: the exact time of onset of diabetes was not accurately known; however, the time of diagnosis was used to estimate the duration of the disease; no conventional angiography data to serve as gold standard; and selection bias (prevalence among those presenting in a tertiary hospital often differs from the community prevalence).

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### Conflicts of interest

There are no conflicts of interest.

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