Original Research Article

PREVALENCE AND PREDICTORS OF LOWER EXTREMITY PERIPHERAL ARTERY DISEASE AMONG ADULTS WITH TYPE 2 DIABETES MELLITUS ATTENDING A TERTIARY HOSPITAL IN OWERRI, NIGERIA

ABSTRACT

Background

Lower extremity peripheral artery disease [LEAD] is common among patients with Diabetes mellitus (DM) and is under-diagnosed and under-treated. Early diagnosis and treatment will prevent associated cardiovascular events, minimize long term disability and improve quality of life. There is paucity of data on LEAD in Owerri and Southeastern Nigeria in general.

Study objectives

To determine the prevalence and predictors of LEAD among adults with type 2 diabetes mellitus (T2DM).

Study design: Cross-sectional analytical

Study site: Endocrinology Clinic, Federal Medical Centre, Owerri, Nigeria

Methodology: Two hundred and seventy (270) T2DM patients and 135 non-diabetic controls were recruited consecutively between January and June, 2016. Questionnaires were used to collect relevant information, followed by focused physical examination and anthropometry. A portable Ankle Brachial Index (ABI) kit was used for measurement of ABI and participants with values ≤ 0.9 were diagnosed as having LEAD. For participants with ABI ≥ 1.3, a toe pressure kit was used to measure their toe systolic pressure and those with toe brachial index (TBI) ≤ 0.7 were diagnosed as having LEAD. Fasting blood samples were also collected for assessment of glycated haemoglobin (HbA1c), fasting plasma glucose (FPG) and lipid profile. Data analysis was performed with SPSS version 22 and p-value < 0.05 was considered significant.

Results

The mean ages of the T2DM and control participants were 59.8 ± 10.7 and 59.6 ± 12.3 years respectively ($P = 0.89$) while their mean ABIs were 0.97 ± 0.18 and 0.99 ± 0.16 respectively ($P = 0.26$). The prevalence of LEAD was 31.1% and 27.4% among T2DM and control participants respectively ($P = 0.44$) while among the T2DM participants that had LEAD, 57 (67.8%), 26 (31.0%) and 1 (1.2%) had mild, moderate and severe LEAD respectively. The only predictor of LEAD among T2DM participants was absent/reduced dorsalis pedis artery pulsation (AOR = 3.57, CI = 1.13 – 11.29, $P = 0.03$).

Conclusions and recommendations

There is a high prevalence of LEAD among adults with T2DM but this is not significantly higher than the prevalence among non-diabetic individuals. Regular screening of T2DM patients for LEAD should be encouraged. There is also need for regular palpation of dorsalis pedis artery among adults with T2DM to identify those with absent or reduced pulsation which may be an indication of the presence of LEAD.

Keywords: Lower extremity peripheral artery disease, type 2 diabetes mellitus, prevalence, predictors

Abbreviations: LEAD: Lower extremity peripheral artery disease, ABI: Ankle brachial index, TBI: Toe brachial index, HbA1c: Glycated haemoglobin, T2DM: Type 2 diabetes mellitus

INTRODUCTION

Peripheral artery disease [PAD] comprises those entities which result in the obstruction of blood flow in the arteries, exclusive of the coronary and intracranial vessels (1) and can affect extra cranial carotid circulation, upper extremities arteries, mesenteric and renal circulation and arteries of the lower extremities. It has been shown that Peripheral artery disease is more common and progresses more rapidly with the likelihood of development of foot ulcers and gangrene in people with DM than those without DM. It is also associated with a higher risk of Ischaemic events (2)(3)(4) due to systemic atherosclerosis associated with DM (2). When PAD

affects the lower extremities, it is called lower extremity peripheral artery disease [LEAD]. While LEAD may be asymptomatic, the symptoms include intermittent claudication, rest pain, ulcers and gangrene. Asymptomatic LEAD is common among diabetic patients (5), with increased predisposition to development of diabetic foot ulcer [DFU] and lower extremity amputation. Despite increased morbidity and mortality associated with LEAD in diabetic patients, it has been shown to be frequently underdiagnosed and undertreated (6). Hence, early diagnosis and treatment of LEAD, reduce associated cardiovascular events, minimize long-term disability, and improve quality of life (2). The accurate assessment of the prevalence of LEAD in the diabetic population is confounded by various factors. Lower extremity peripheral artery disease is often asymptomatic and concomitant peripheral neuropathy may alter pain perception. Also two of the common clinical findings in LEAD which are absence of peripheral pulses and presence of claudication, are inadequate diagnostic indicators (7). However, using ABI as the index of diagnosis, the prevalence of LEAD among DM patients was observed to be between 10% and 30% (8)(9)(10)(11). Umuerri et al (5) in their study of adult T2DM patients in Benin Nigeria, using ABI < 0.90 as the basis for diagnosis of LEAD, observed a prevalence of 35.6%. On the other hand, Oyelade et al (12) in Ogbomoso, South West Nigeria observed a prevalence of 52.5%. The major risk factors for PAD are DM, cigarette smoking, advanced age, dyslipidaemia and hypertension (13). Others are hyperhomocystinaemia (14), obesity, positive family history, male sex and African-American or Hispanic origin (15)(16). Among these, hypertension, DM, smoking, and dyslipidaemia account for majority of cases of PAD and each of these was found to be significantly and independently associated with a higher risk of PAD after adjustment for the other three risk factors and confounders (17). While several studies have

been done globally on LEAD among people with DM, there is paucity of data on this in Southeastern Nigeria.

MATERIALS AND METHODS

This cross-sectional analytical study was carried out at the Endocrinology Clinic of Federal Medical Centre, Owerri which is one of the major tertiary health Institution in Imo state, Nigeria and serves as a referral Centre for Imo and surrounding States. Two hundred and seventy T2DM patients previously diagnosed using the American Diabetes Association (ADA) criteria (18) and 135 non-diabetic controls aged 30 years and above were recruited consecutively over the study period between January and June, 2016. Those with known haemoglobinopathies as well as pregnant women were excluded from the study. Ethical approval for the study was obtained from the ethics committee of the hospital while written informed consent was obtained from the participants. A pre-tested interviewer-administered questionnaire was used to obtain information on socio-demographics of the participants like age, sex, occupation and tribe as well as relevant medical history like duration of DM, current anti-diabetic therapy, Cigarette smoking, hypertension, duration of hypertension, past history of stroke and family history of Stroke. Blood pressure (BP) was measured with mercury sphygmomanometer (ACCUSON ENGLAND) using standard procedure, in sitting position after about 5 minutes rest and diagnosis of Hypertension was based on BP $\geq$ 140/90 mmHg (19) and/or taking antihypertensive drug. Phase I Korotkoff sound was used for systolic BP and Phase V for the diastolic BP. However, where phase V sound could not be obtained; phase 1V sound was used for diastolic pressure. Anthropometric indices were then measured for each participant using standard procedures (20)(21). Weight was measured to the nearest 0.5 kilogram using a standard weighing scale (Hanson, England) with the participants wearing light clothes and on bare foot. The scale was on a hard and flat surface,

and calibrated frequently using known standard 10 kg weight, while the pointer of the scale was adjusted to zero before each measurement. Height was measured to the nearest 0.1 cm using a Stadiometer (Avery England) in an erect position without foot wears, head scarf or caps. Body Mass Index (BMI) was then calculated by dividing the weight (W) in kg by the square of the participant's height (H^2) in meters i.e. BMI = W/H^2 in (kg/m^2) and all values were taken to the nearest one decimal place and was classified as underweight (<18.5 kg/m2), normal (18.5 - 24.9 kg/m2), overweight (25.0 - 29.9 kg/m2) and obese (≥30 kg/m2). Waist circumference (WC) was measured to the nearest 0.1 cm at the midpoint between the lower rib margin and the iliac crest while the hip circumference was measured to the nearest 0.1 cm at the point of maximum circumference of the buttocks using a non-elastic measuring tape. The Waist: hip ratio (WHR) was then calculated. This was followed by a detailed examination of the lower limbs for signs of impaired blood perfusion like sparse hair, dystrophic nail changes, cold extremities, ulcers, gangrene or amputation as well as palpation of the dorsalis pedis, posterior tibial and popliteal pulses to ascertain whether they were full, diminished or absent. A portable ABI kit (Hokanson USA) was used to assess the ABI with the participants in supine position, arms and legs at heart level and having rested for at least 20 minutes. The systolic blood pressure (SBP) on brachial, posterior tibial and dorsalis pedis arteries were measured bilaterally with appropriately sized cuffs applied above the elbows and maleoli for measurement of the arm and ankle pressures respectively. Ankle brachial index for each side was calculated by dividing the higher SBP on the dorsalis pedis artery and posterior tibial arteries on that side by the higher arm SBP (right or left). The lower of the ABIs on the right and left side of the body was then taken as the overall ABI (22)(23). Participants with ABI ≤ 0.9 were diagnosed as having LEAD (22). However, those with ABI ≥ 1.3 were assumed to have incompressible arteries, likely due to medial arterial

calcification (1)(22) and in such cases Toe Brachial Index (TBI) of ≤ 0.7 was used to diagnose co-existing LEAD (24)(25). Toe systolic pressure was measured using toe pressure kit (Hokanson USA). With participant in supine position, arms and legs at heart level and having rested for 20 minutes, the toe cuffs were placed snuggly on each of the big toes and the photo plethysmograph then connected on the pad of one of the big toes, making sure that it did not touch the cuff. The patient's pulse was then observed as a waveform on the chart recorder, after which the sphygmomanometer was connected to the toe pressure cuff and inflated slowly until the waveform disappeared and the toe pressure was noted. Inflation of the sphygmomanometer was then continued until 20 – 30 mmHg above the noted pressure and then slowly released at about 2 mmHg per declination until the waveform reappeared. The pressure at which the waveform reappeared was taken as the toe systolic pressure and the cuff completely deflated. The procedure was then repeated on the contra lateral big toe. The TBI for each side of the body was derived by dividing the toe systolic pressure on that side by the higher brachial systolic pressure (right or left) and the lower of the two sides then taken as the overall TBI (26)(27). About 10 mls of blood was collected from each of the participants, after overnight fast, using standard procedures and 4 mls was put into fluoride oxalate bottle for assessing FPG and HbA1c while 6 mls was put into plain bottle for serum lipid profile. The blood sample for lipid profile was allowed to stand for about 2 hours and then spun for 10 minutes using the centrifuge at 3000 rpm, after which the serum was separated. Glycated haemoglobin (HBA1C) was assessed with In2it HBA1C analyzer which uses Boronate affinity chromatography method while FPG was assessed using glucose oxidase method. Total cholesterol and triglycerides levels were determined by enzymatic (28) and colorimetric (29) methods respectively using Biosystems reagents while high density lipoprotein cholesterol (HDL-C) levels were determined by

precipitant method (30) using Randox HDL-Chol kit. Low density lipoprotein cholesterol (LDL-C) levels were then calculated using Friedwald formula (31).

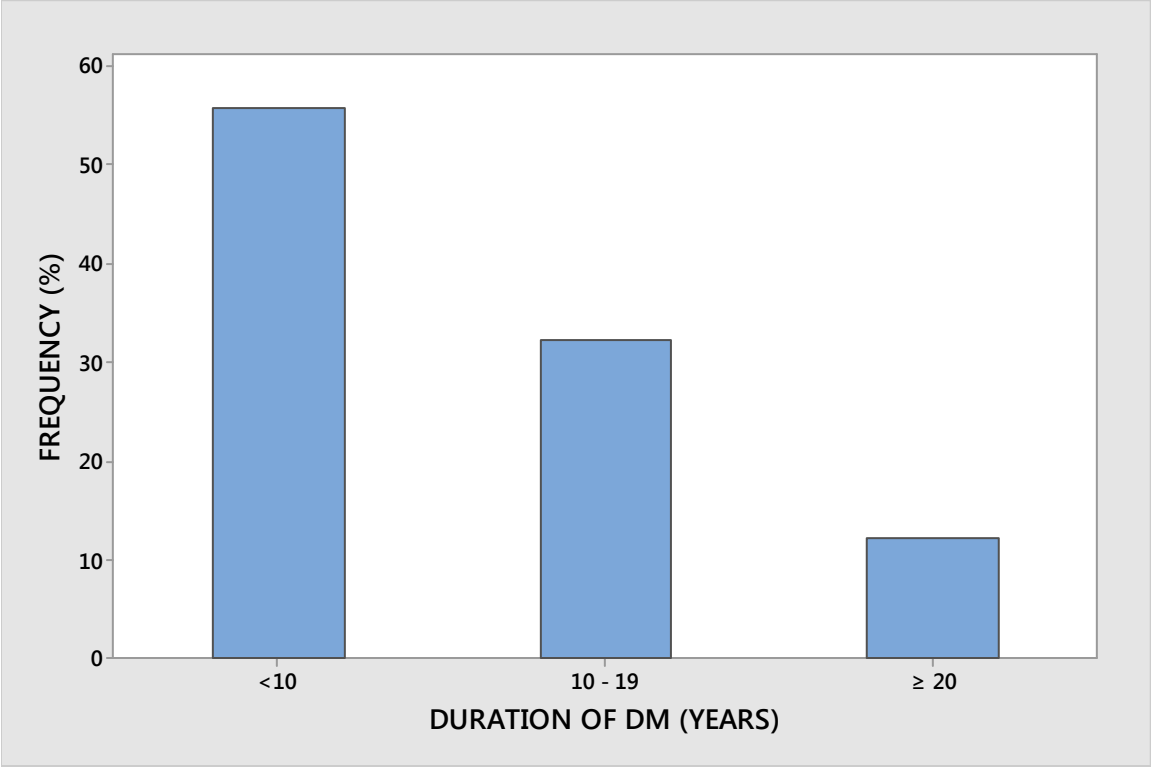
Statistical Analysis

The data obtained were entered and analyzed using statistical package for social sciences (SPSS) version – 22 (IBM, USA) and then presented as tables, graphs and charts. Continuous variables were described using mean ± standard deviation (SD) if normally distributed while non-normally distributed continuous variables were described using median (interquartile range). Categorical variables were compared using chi-square or Fisher's exact test, where appropriate, while differences in group means were ascertained using Student's t-test. Predictors of LEAD among Participants were determined using multivariate logistic regression analysis and *P*-value < 0.05 was taken as significant.

RESULTS

Two hundred and seventy (270) participants with type 2 diabetes mellitus who met the inclusion criteria as well as one hundred and thirty five (135) non-diabetic controls who did not have any of the exclusion criteria were recruited for the study. All the participants were Nigerians and majority (99.3% and 97.8% of T2DM and control participants respectively) were of Igbo tribe. Among the T2DM participants, 111 (41.1%) and 159 (58.9%) were males and females respectively while among the non-diabetic controls, 55 (40.7%) and 80 (59.3%) were males and females respectively. The mean ages of the T2DM participants and the non-diabetic controls were 59.79 ± 10.71 years and 59.61 ± 12.30 years respectively. The duration of DM among the T2DM participants ranged from 1 – 40 years, with a median duration of 8.0 (IQR 4.00 – 14.00) years. More than half (55.6%) of the T2DM participants have had DM for < 10 years. The

frequency of distribution of duration of DM among the T2DM participants is shown in the figure below:



DM: Diabetes mellitus

Figure 1: Bar chart showing duration of DM among participants with T2DM

The mean WC was 94.69 ± 11.38 cm and 90.64 ± 12.01cm among the T2DM and control participants respectively while their mean WHR were 0.94 ± 0.09 and 0.89 ± 0.08 respectively. The clinical characteristics of the T2DM and control participants are as shown in the table below:

Table 1: Clinical characteristics of study participants

VARIABLE	T2DM N = 270 n (%)	CONTROL N = 135 n (%)	Chi - square (X^2)	P – value
Gender				
Male	111 (41.1)	55 (40.7)		
Female	159 (58.9)	80 (59.3)	0.005	0.94
BMI				
Underweight	10 (3.7)	6 (4.4)		
Normal	78 (28.9)	46 (34.1)	2.26	0.52
Overweight	112 (41.5)	46 (34.1)		
Obese	70 (25.9)	37 (27.4)		
	Mean ± SD	**Mean ± SD**	**t – value**	**P - value**
Age (years)	59.79 ± 10.71	59.61 ± 12.30	0.14	0.89
Weight (kg)	71.97 ± 12.95	73.42 ± 16.8	0.99	0.32
Height (m)	1.63 ± 0.074	1.65 ± 0.096	2.45	0.01
SBP (mmHg)	133.53 ± 19.88	131.71 ± 22. 08	0.83	0.41
DBP (mmHg)	80.28 ± 12.86	81.36 ± 13.53	0.78	0.43
BMI (kg/m^2)	27.03 ± 4.76	26.89 ± 5.71	0.18	0.85
WC (cm)	94.69 ± 11.38	90.64 ± 12.01	3.29	0.001
WHR	0.94 ± 0.09	0.89 ± 0.08	5.46	0.0001
ABI	0.97 ± 0.18	0.99 ± 0.16	1.14	0.26

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WC: Waist circumference, WHR: Waist-hip ratio, ABI: Ankle brachial index

Intermittent claudication was present in 62 (23%) and 12 (8.9%) of the T2DM and control participants respectively. While 10 (3.7%) and 13 (4.8%) of T2DM participants had gangrene and limb amputation respectively; neither of these features was observed among the control participants. The table below shows the comparison of the clinical features of LEAD between T2DM participants and the controls.

Table 2: Clinical features of LEAD among study participants

VARIABLE	T2DM N = 270 n (%)	Control N = 135 n (%)	P-value (Fisher's exact test)
Intermittent claudication	62 (23.0)	12 (8.9)	0.000
Foot ulcer	47 (17.4)	1 (0.7)	0.000
Foot Gangrene	10 (3.7)	0 (0.0)	0.04
Amputation	13 (4.8)	0 (0.0)	0.01
Sparse hairs	57 (21.1)	11 (8.1)	0.001
Dystrophic nails	151 (55.9)	32 (23.7)	0.000
Cold extremities	54 (20.0)	10 (7.4)	0.001
Diminished DP pulse	119 (44.1)	32 (23.7)	0.000
Absent DP pulse	23 (8.5)	1 (0.7)	0.001
Diminished PT pulse	126 (46.7)	30 (22.2)	0.000
Absent PT pulse	20 (7.4)	2 (1.5)	0.01

DP: Dorsalis pedis, PT: Posterior tibial

The HbA1c among the T2DM participants ranged from 4.0% to 15.0% with a mean of 7.90 ± 2.24 %. Among them, 106 (39.4%) had HbA1c < 7.0% while 164 (60.6%) had HbA1c of ≥ 7.0%. The laboratory variables of the study participants are as shown in tables below:

Table 3: Laboratory variables of study participants

	T2DM (mean ± SD)	CONTROL (mean ± SD)	t - value	p-value
HbA1c (%)	7.90 ± 2.24	4.96 ± 0.66	19.91	0.000
FPG (mmol/L)	9.51 ± 4.69	4.98 ± 0.59	15.76	0.000
TC (mmol/L)	4.98 ± 1.17	4.62 ± 0.73	3.79	0.000
TG (mmol/L)	1.44 ± 0.55	1.30 ± 0.50	2.57	0.011
HDL-C (mmol/L)	1.27 ± 0.43	1.42 ± 0.43	3.31	0.001
LDL-C (mmol/L)	3.07 ± 1.12	2.65 ± 0.75	4.47	0.000

HbA1c: Glycated haemoglobin, FPG: Fasting plasma glucose, TC: Total cholesterol, TG: Triglycerides, HDL: High density lipoprotein cholesterol, LDL: Low density lipoprotein cholesterol, SD: Standard deviation

The prevalence of LEAD among T2DM participants in this study was found to be 31.1% while among the control participants it was 27.4%. Six (2.22%) of T2DM participants had ABI ≥ 1.30, out of whom 5 (83.3%) had TBI ≤ 0.70 and were thus diagnosed as having LEAD. Similarly, among the control participants, 3 (2.22%) had ABI ≥ 1.30, two (66.7%) of whom had TBI ≤ 0.70 and were diagnosed as having LEAD. The prevalence of LEAD was equally observed to be 20.0%, 29.6% and 41.1% among T2DM participants aged 30 – 49, 50 – 69 and ≥70 years respectively.

The associations between LEAD and some other variables among the T2DM participants in this study were determined using chi-square. This was then followed by multivariate logistic regression analysis using the variables with *P*-value ≤ 0.25. Subsequently, reduced or absent dorsalis pedis artery pulsation was observed to be the only independent predictor of LEAD among the T2DM participants (AOR = 3.572, 95% CI = 1.130 - 11.293 and *P* - value = 0.03). The associations between LEAD and some other variables and the multivariate logistic regression analysis for predictors of LEAD among the T2DM participants in this study are shown in the tables below:

Table 4: Associations between LEAD and some other variables among T2DM participants

VARIABLE	CHI –SQUARE	P – value
AGE	4.287	0.12
GENDER	1.424	0.23
DURATION OF DM	1.278	0.53
SMOKING	1.442	0.23
HTN	2.417	0.12
FAMILY HX OF LEAD	1.122	0.29
OBESITY	2.054	0.15
INT. CLAUDICATION	0.049	0.82
FOOT AMPUTATION	1.442	0.23
FOOT ULCER	4.889	0.03
FOOT GANGRENE	1.729	0.19
REDUCED/ABSENT DP	33.065	0.000
REDUCED/ABSENT PT	28.402	0.000
INCREASED HbA1c	0.320	0.57
DECREASED HDL-C	4.075	0.04
INCREASED LDL-C	1.603	0.21
INCREASED TG	3.129	0.08
INCREASED TC	2.356	0.13

DM: Diabetes mellitus, HTN: Hypertension, HX: History, Int: Intermittent, DP: Dorsalis pedis, PT: Posterior tibial, HbA1c: Glycated haemoglobin, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, TG: Triglycerides, TC: Total cholesterol, LEAD: Lower extremity peripheral artery disease

Table 5: Multivariate logistic regression for possible predictors of LEAD among T2DM participants

VARIABLE	B	ODD RATIO	CI	P – value
AGE	- 0.427	0.652	0.258 - 1.650	0.37
GENDER	0.170	1.185	0.631 - 2.227	0.60
SMOKING	- 0.816	0.442	0.114 - 1.711	0.24
HTN	- 0.188	0.829	0.435 - 1.579	0.57
OBESITY	0.282	1.325	0.653 - 2.689	0.44
FOOT AMPUTATION	0.717	2.049	0.483 - 8.687	0.33
FOOT ULCER	0.273	0.761	0.336 - 1.722	0.51
FOOT GANGRENE	- 0.463	0.629	0.122 - 3.253	0.58
REDUCED/ABSENT DP	1.273	3.572	1.130 - 11.293	0.03
REDUCED/ABSENT PT	- 0.145	0.865	0.266 - 2.818	0.81
DECREASED HDL-C	0.428	1.535	0.765 - 3.078	0.23
INCREASED LDL-C	- 0.013	0.987	0.425 - 2.292	0.98
INCREASED TG	- 0.266	0.766	0.400 - 1.469	0.42
INCREASED TC	- 0.464	0.629	0.265 - 1.490	0.29

LEAD: Lower extremity peripheral artery disease, CI: Confidence interval for odd ratio, HTN: Hypertension, DP: Dorsalis pedis, PT: Posterior tibial, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, TG: Triglycerides, TC: Total cholesterol

Among the 270 T2DM participants in this study, one hundred and fifty (55.6%) have had DM for < 10 years while 33 (12.2%) have had DM for ≥ 20 years. Nine (27.3%) and 44 (29.3%) of participants that have had DM for ≥ 20 years and < 10 years respectively were observed to have LEAD. The association between duration of DM and presence of LEAD among the T2DM participants in this study is as shown in table 6 below:

Table 6: Association between duration of Diabetes and frequency of Lower Extremity Peripheral Artery Disease among T2DM participants

Duration of DM (years)	LEAD		Chi square	p-value
	PRESENT n (%)	ABSENT n (%)		
< 10	44 (29.3)	106 (70.7)	1.278	0.53
10 – 19	31 (35.6)	56 (64.4)		
≥ 20	9 (27.3)	24 (72.7)		
TOTAL	84 (31.1)	186 (68.9)		

LEAD: Lower extremity peripheral artery disease

DISCUSSION

There were more females among the T2DM and control participants in this study. This pattern of gender distribution is similar to that observed by Okello et al (32) in their study involving 229 DM patients out of whom 63.7% were females as well as Umuerri and Obasohan in Benin, Southsouthern Nigeria in whose study females constituted 62.9% of the participants (5). This pattern of gender distribution may be attributable to the health care seeking pattern in the general population, as women have been shown to more readily seek medical attention than men (33). It has also been shown that women with DM prefer to attend government hospitals while men prefer to attend private hospitals and clinics (34) as they are more likely to get faster services in such private facilities. This could have also contributed to the pattern of gender distribution of participants in this study as it was conducted in a government hospital. Similarly, DM has been

shown to be more common in women than men (35) and this may equally offer some explanation for the gender distribution of participants in this study. The frequencies of most of the clinical features of LEAD were significantly higher among the T2DM participants than the controls in this study. Though Intermittent Claudication which is the major clinical presentation of LEAD was commoner among the T2DM participants, some of these participants that had LEAD did not have this symptom as it was only present in 23.0% as against the 31.1% that had LEAD, diagnosed based on ABI ≤ 0.90 and/or TBI ≤ 0.70. Similarly, in the study by Okello et al (32) only 21.0% of the participants had definite claudication while 24.0% were diagnosed of LEAD using ABI. This shows that a reasonable number of diabetic patients with LEAD are asymptomatic, likely due to co-existence of diabetic neuropathy which impairs their ability to perceive pain. Similarly, while foot ulcers, gangrene and amputation were observed among some of the diabetic participants in this study, none of these features was observed among the control participants. Presence of diabetic complications like neuropathy and vasculopathy predispose to development of foot ulcers which are readily infected as a result of associated compromised immunity, with subsequent development of foot gangrene and amputation. Thus diabetic patients should be properly educated on foot care to minimize the rate of development of these complications and advised to seek prompt medical attention whenever they notice any ulcer on their feet.

This study shows that the prevalence of LEAD is high among adults with T2DM but there is no statistically significant difference in the prevalence of LEAD between those with T2DM and the control group (P-value $=$ 0.44). The high prevalence of LEAD among adults with T2DM is likely due to the atherosclerosis associated with DM (36).The presence of other recognized risk factors for LEAD like dyslipidaemia (13) and central obesity (15) in the T2DM participants

could have equally contributed to the high prevalence of LEAD among them in this study. The absence of a statistically significant difference in the prevalence of LEAD between adults with T2DM and the control population in this study is likely due to the presence of other recognized risk factors for LEAD among the control group. For instance, there was no statistically significant difference in the prevalence of obesity [*P*-value = 0.52] and dyslipidaemia [p-value = 0.40] between the T2DM and control participants. The prevalence of LEAD among T2DM participants in this study is similar to that of 35.6% observed in Benin, Southsouthern Nigeria (5). On the other hand, the prevalence of LEAD among T2DM patients observed in this study is higher than the prevalence of 24% observed by Okello et al (32) in Uganda, East Africa. This difference may likely be due to the difference in the methods of measurement of ABI in the two studies. While in this study, the ABI for each side of the body was derived by dividing the higher of the SBP on the dorsalis pedis and posterior tibial artery on that side by the higher of the left and right brachial SBP, Okello et al (32) derived the ABI of their participants by dividing the higher of the dorsalis pedis and posterior tibial SBP on that side by the ipsilateral brachial artery SBP. Secondly, there were fewer diabetic participants in the study by Okello et al than in this study (229 vs. 270). The prevalence of LEAD among T2DM participants in this study was found to be higher among those ≥ 70 year. This is similar to what was observed by Oyelade et al (12) in Ogbomosho, Southwestern, Nigeria. Advancing age predisposes to atherosclerosis and is a recognized risk factor for LEAD. This may explain the higher prevalence of LEAD among the elderly. The high prevalence of LEAD among adults with T2DM translates to a high prevalence of the associated morbidity with the attendant negative effect on the productive capacity of the population. This buttresses the need for aggressive risk factor modification and institution of appropriate therapy for T2DM patients with LEAD as they are highly predisposed to ischaemic

events (37). There is also need for regular evaluation of people with DM for LEAD to enhance early diagnosis and prompt management of the condition.

Recognized risk factors for LEAD include DM, cigarette smoking, advanced age, dyslipidaemia, HTN, obesity, family history and male gender (13)(15). However, in this study, multivariate logistic regression analysis showed that only absent or reduced dorsalis pedis artery pulsation was the significant predictor of LEAD among T2DM participants. In a similar study, Aboyans et al (25) identified age, pack year of smoking and hypertension as the significant predictors of low ABI which is a marker of LEAD. Smoking was not among the identified significant predictors of LEAD among the T2DM participants in this study probably because there were more females among the participants and the percentage of smokers among them was low. Odenigbo et al (38) in Nnewi, Southeastern Nigeria also observed that smoking had no influence on the prevalence of PAD among adults with hypertension. Obesity was equally not identified as a significant predictor of LEAD in this study. This is likely because only about one-fifth (20.6%) of the T2DM participants in this study were obese. Patients with T2DM are usually encouraged to adopt a different lifestyle using physical exercises and an appropriate diet as part of their treatment with the aim of achieving weight loss and blood glucose control (39). Most patients with T2DM also take the oral antidiabetic drug Metformin, which has been shown to cause weight loss (40). There is thus need for regular evaluation of adult T2DM patients for absent or decreased dorsalis pedis artery pulsation as this will enhance early diagnosis of LEAD and appropriate treatment among them.

CONCLUSION

There is a high prevalence of LEAD among adults with T2DM but this is not significantly higher than the prevalence among non-diabetic individuals. Regular screening of T2DM patients for LEAD should be encouraged. There is also need for regular palpation of dorsalis pedis artery among adults with T2DM to identify those with absent or reduced pulsation which may be an indication of the presence of LEAD

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