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Diabetic foot in Nigeria - a review article

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Summary
Numerous institutional reports from Nigeria on diabetic foot (DF) have appeared showing variations from one institution to another in the last 40 years. In the author's opinion, this is the first overall review on DF care in Nigeria to put all the pictures together and then compare with global literature. The reports showed varied male-female preponderance, occurrence mainly among the low socio-economic group patients, mostly involved in one form of trading or another, with a peak incidence in the 6th decade, up from the 5th decade of the past. There is also a rising incidence of DF which has recently become an important indication for lower limb amputation in Nigeria. This review also discusses the peculiar situations of DF in Nigeria in relation to aetiopathogenesis, staging and classification, non-operative and operative treatment, and the way forward to reduce morbidity and mortality and improve the present disappointing outcome of care of DF patients in Nigeria. The roles of interventional radiology and revascularisation in DF management are highlighted.

Keywords: *Diabetes mellitus, diabetic foot, gangrene, amputation, Nigeria*

Résumé
Les nombreux rapports institutionnels du Nigeria sur la diabétique du pied (DF) ont paru montrer des variations d'une institution, l'un à l'autre dans les 40 années passées. Dans l'opinion de l'auteur, c'est la première révision totale sur DF au Nigeria pour réunir toutes les images et a comparées avec la littérature globale. Les rapports ont montré la prépondérance de la mâle-femme variée, événement principalement parmi les malades du groupe socio-économiques bas, principalement impliqué dans une forme de commerce ou un autre, avec une fréquence maximum dans la 6eme décade, en dès la 5eme décade. Il y a aussi une fréquence du soulèvement de DF qui est devenu une indication importante pour l'amputation du membre inférieure récemment au Nigeria. Cette révision discute aussi les situations particulières de DF au Nigeria par rapport à l'aetiopathogenesis, l'organisation et la classification, le traitement non chirurgical et chirurgical, le fait d'avancer la réduction de la morbidité et la mortalité, et améliorer le résultat décevant présent de soin de malades de DF au Nigeria. Les rôles d'intervention de la radiologie et la revascularisation dans la gestion de DF sont mis en valeur.

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Introduction

Diabetic foot (DF) is the most common cause of non-traumatic, lower extremity amputation in the developed[1], and recently in the developing [2-4] countries. It is the most devastating lower extremity complication of diabetes mellitus (DM) associated with more hospitalisations than all other complications of DM put together[5,6]. Characterised by prolonged hospital stay, DF terminally leads to single or serial amputation(s) and eventually to death within months to years of amputation [7, 8]. Indeed, it is the commonest indication for surgery in diabetics. The occurrence of DF in one foot places the other foot at high risk of developing DF [9]. Often beginning with minor trauma and progressing to cellulitis, superficial and deep sloughing of tissues, ulcer, abscess, osteomyelitis and eventually leading to foot gangrene [10]. The pathological changes in the foot of the diabetic have been blamed on a triad of poor immunity, vasculopathy and neuropathy acting singly or in synergy and leading to gangrene of the foot. Death usually occurs not distantly via end stage renal disease (ESRD), ketoacidosis, and/or encephalopathy.

Further research has implicated other contributory factors in producing DF. These include foot deformities (hallux rigidus and hammer toes), elevated plantar pressures in excess of 65 N/cm^2, history of amputation, lengthy duration of DM (>10 years), male sex, and poor diabetes control (evidenced by glycosylated haemoglobin (>9 %) [11]. Some socio-demographic characteristics are also associated with an increased risk of lower limb complications. These include age between 50 and 70 years as opposed to the younger than 50 years, being single as opposed to being married, treatment with insulin for insulin

dependent DM (Type 1 DM) and non-insulin dependent DM (Type 2 DM) compared to Type 2 DM not being treated with insulin, patients who needed help to reach the hospital before onset of the complications and those who did not attend hospital regularly [12]. In Nigeria as elsewhere, it is a great distress to lose a limb via amputation when certain measures could prevent this. The costs are enormous when computed for hospitalisation, treatment, man-hour losses in unearned income and unquantifiable psychological disturbance. The most humane means of control would include prevention of these complications by early diagnosis of DM and DF, early treatment of DM patients and an overall improvement in health care delivery in Nigeria.

Prevalence, age and sex distribution of DF

Statistics on DF worldwide [8, 13, 14] and specifically from Nigeria [8] is inadequate. Therefore, since the phenomenon of DF is tied to both DM and lower limb (LL) amputation, the information available on DM and LL amputation can be extracted for DF. Diabetes mellitus which was considered rare in Africa some 45 years ago by Vink and Angawa [15] has the earliest report in Nigeria by Kinnear [16] from Ibadan in 1963. Kinnear [16] stated that "diabetes is not at present a very common disease in Nigeria" because only 309 patients out of 80,000 admissions (0.39%) in 5 years were recorded. More recently, population studies unlike the hospital-based studies of Kinnear [16], carried out in 1988 in Lagos [17] showed a 1.5 % male and 1.9 % female prevalence whereas from the middle-belt region of Nigeria 1.43% prevalence[18] was obtained. Previously, diabetes was not an important indication for LL amputation from developing countries [3, 4], thought to be due to trauma and infections [4]. In recent times, the pre-eminence of DF in indications for LL amputation from developing countries suggests a new trend [3], now diabetes and trauma.

Kinnear [16] reported 3 cases of DF out of 309 DM patients (0.97%). Osuntokun *et al.* [19] in 1971 also suggested that DF was relatively an uncommon complication of DM because only 25 out of 832 DM patients (3%) had DF but Adetuyibi [20] in 1976 reported 3.8 % while Lawson *et al.* [21] in 1978 reported a prevalence of 5.4 % (57 DF out of 1,050 DM patients) in a 15 year-study. All these reports [16, 19-21] emanated from the University College Hospital Ibadan, in the south-western part of Nigeria where the first University teaching hospital in Nigeria is situated and perhaps patients seen there came from far and wide of Nigeria. A more recent data from Ilesha, south-western Nigeria by Ndububa *et al.* [22] in 1996 showed a 19.3 % prevalence (22 DF out of 114 DM patients). Certainly there is a rising incidence of both DM and DF.

Three other reports [4, 23, 24] from south-eastern Nigeria provide statistics on DF and LL amputations. In 1989, Onuba *et al.* [4] reporting from Calabar quoted a 13.9% prevalence of DF amputations. However, there were 18 upper limb and 18 LL amputations in their study, hence, in strict sense, the prevalence of DF lesion in LL amputations should be 27.8% (5 of 18 patients). In 1992, Osisioma *et al.* [23] reporting from Enugu recorded similar 27.8 % prevalence (30 DF out of 108 foot gangrene patients) while Anyanwu [24] from Onitsha in 1994 recorded a much higher prevalence of 69.0 % (29 DF amputations out of 42 LL amputations). Additionally, Bojuwoye [25] reporting from Ilorin, middle-belt region of Nigeria in 1995 found 17 DF lesions among 259 DM patients (6.6%) while Solagberu and Onawola [3] also from Ilorin in 2001 compiled amputation statistics and found DF indications in 38.1 % (16 DF amputations out of 42 LL amputations).

From the northern part of Nigeria, Ogirima et al [26] recorded 0.9 % prevalence (31 DF patients among 3301 DM patients seen at the Ahmadu Bello University Teaching Hospital, Kaduna) while Garba *et al.* [27] documented 10 DF out of 133 (7.5 %) LL amputations in a 10-year review from Zaria. It is remarkable that these prevalence rates (0.9 % and 7.5 %) of DF from DM and LL amputation statistics from northern Nigeria are the lowest in the country. It is quite possible that this is a truly low prevalence or that the unusually high rate of traditional bonesetters (TBS)-caused amputations when lowered will give the true incidence of DF amputation in northern Nigeria. This is because the northern part has the highest amputation rate in Nigeria from complications due to the TBS (35.3 %, 47 of 133 LL amputations, compared to Ilorin with 5.2 % [28] and Enugu with 25 %[29]). Further research in this area is readily obvious.

The sex distribution of DM and DF in most reports [16, 19, 21, 23, 24] confirmed male preponderance. Kinnear [16] in 1963 had attributed this to be the cultural habit of Nigerian males having greater access to health care rather than a strict gender difference. The evidence for this is reinforced when Bojuwoye [25] found greater female preponderance while Adetuyibi [20] and Ndububa et al. [22] found nearly equal sex distribution because that cultural habit is less pronounced now. On the contrary, actual gender difference with a male preponderance has been reported from outside Africa, at least in the United States of America [11]. This is attributed to smoking behaviour, activity level, hormonal differences, degree of compliance, level of denial, strength of social support mechanism, and quality of education as well as the higher

prevalence and severity of vascular disease, neuropathy and diabetes [11]. In general, women seem to have fewer complications and better prognosis than men because peripheral arterial vascular disease [30] and neuropathy [31] are lower in women with diabetes.

The age distribution of DM and DF worldwide affect all age groups. Some of the reports from Nigeria (Anyanwu [24], Ogirima *et al.* [26] and Umebese et al. [32]) however, did not include childhood DF. In a published comment [33], Ogirima [34] confirmed the absence of childhood DF in their series [26]. Some reports [8, 35] documented patients between 15 and 30 years with who presented with DF. The significance of this age variation of DF patients in Nigeria remains to be seen. What the literature suggests is better prognosis for the young DF patient unlike patients 50 years and above with this age limit being one of the risk factors for becoming a diabetic amputee [11, 14]. Further, the peak age incidence of DM among Nigerians has shifted upwards from the 5th decade [16, 19, 21] reported in the 1960's and 1970's to the 6th decade in the 1990's probably on account of improving longevity. However, the peak age for amputation for DF is still lower in Nigeria [3] than in the developed countries where peak age for amputation is above 60 years [36].

Other socio-demographic factors reported [16, 21, 22, 25] prevalence of low socio-economic status among DM patients, often with minimal or no education. However, multivariate analysis of these factors did not show any significant contribution in the level of formal education and DF[11]. The commonest occupation among the Nigerian patients is trading, many on petty scale [20, 22]. These realities conspire to determine the low purchasing power of DM patients, a situation responsible for inability to purchase drugs, comply with drug use, and attend clinic regularly predisposing the patients to develop DF [8, 20, 25]. It remains to be proved whether this is responsible for the lower peak age at which foot gangrene occurs and at which amputation is done for DF in Nigeria. Diabetes mellitus associated with malnutrition was described by Kinnear[16] in 1963. Notable differences in DF between the developed and the developing countries concern the dwindling practice of walking unshod, poor hygiene and poor quality footwear in the developing countries.

Table 1: Aetiopathogenesis of diabetic foot

Aetiology (from hyperglycaemia)		Pathogenesis
Poor Immunity[37]	1.	Glucose-C3 complex formed
	2.	Secretion of cytokines like interleukins, cachectin
	3.	Increased virulence of E coli, C albicans, P aeruginosa
Vasculopathy	1.	Athrosclerosis of small vessels (Goldenberg et al.)[35]
	2.	Pretibial distribution of vascular occlusion (LoGerfo and Coffman)[36]
	3.	Arteritis
Neuropathy	1.	Reduction in conduction velocity, nerve fibre
	2.	Axon loss
	3.	Segmental demyelination

Aetiopathogenesis

The classical triad of poor immunity, vasculopathy and neuropathy implicated in the aetiology of DF has been elucidated [8], Table 1. The hyperglycaemic environment facilitates formation of complexes between the thiol ester portion of C3 component of the complement system, important in bacteria phagocytosis [37]. The glucose and C3 (**Glu-C3**) complex formed is inactive in chemotaxis, bacteria adherence, opsonization and hence a defective phagocytosis results ensuring that bacteria exist unchallenged in the bloodstream [37]. In addition, the Glu-C3 complex stimulates the secretion of inflammatory mediators, for example cytokines such as interleukins, cachectin (tumour necrosis factor á) and others which cause tissue damage, a situation favouring infection. The virulence of Escherichia coli, Candida albicans and Pseudomonas aeruginosa, all of which are important in diabetic foot infection, is augmented by hyperglycaemic environment [37].

Goldenberg *et al.* [38] in 1959 had stated that the vascular problems of diabetics were in the small vessels (small arteries and arterioles) as evidenced by the presence of palpable pulses at all common points of palpation. Secondly, they alluded to the patchy nature of gangrene in diabetics unlike the massive gangrene seen in occlusive vascular disease in non-diabetics. These concepts had to change when LoGerfo and Coffman[39] in 1984 described atherosclerotic occlusion of the tibial and peroneal arteries, a sine qua non of diabetic atherosclerosis. Therefore, it was not surprising to see a diabetic patient with an ischaemic foot in the presence of a strong popliteal pulse. LoGerfo *et al.* [39] also found that the occlusive disease of tibial vessels occurs primarily in the leg, so that the arterial system in the foot is less frequently involved with athrosclerosis in the diabetic than IB the non-diabetic, thereby allowing for vein-graft reconstruction from the popliteal to the dorsalis pedis or posterior tibial artery to the ankle and making an amputation unnecessary. However, only univariate

analysis give significance to vasculopathy among factors responsible for DF [11].

The neuropathy leading to DF are recognized by endoneural oedema, reduction in nerve caliber and conduction velocity, segmental demyelination and axon loss; all of which are sequelae of hyperglycaemia [40]. Therefore, the patient walking barefooted or with ill-fitting shoe and with poor foot hygiene is particularly at risk of injury to the feet from peripheral neuropathy-a phenomenon common among poor rural and urban Nigerians. Kinnear [16] reported neuropathy rate of 33%, Osuntokun *et al.* [19] 48 %, while Bojuwoye [25] got 35.9 %—suggesting that at least 1 in 3 DM patients would have neuropathy in our environment. Globally [41], as is the practice here [8], patients' feet suffer neglect during outpatient visits and many minor lesions of the foot are missed at this crucial stage.

Table 2: Wagner's classification system of DF

Grade	Description
0	No open ulcerations present (pre or post-ulcerative skin)
1	Partial/Full thickness ulceration, but depth does not go beyond loss of skin
2	Deeper, tendon or joint capsule may be present
3	Open to bone, osteomyelitis may be present
4	Wet or gangrene plus or minus cellulites (partial foot gangrene)
5	Extensive gangrene indicating higher amputation (whole foot gangrene)

Table 3: Liverpool classification system for diabetic foot ulcers

Classification	Description
Primary	Neuropathic
	Ischaemic
	Combination of both (neuroischaemic)
Secondary	Uncomplicated
	Complicated (presence of cellulitis, abscess, or osteomyelitis)

Staging, scoring and classification (Tables 2-5)

In 1978, Wagner [42, 43] popularized a classification system for DF (Table 2). The Wagner system assesses ulcer depth and the presence of osteomyelitis or gangrene by using the following grades: grade 0 (pre or post-ulcerative lesion), grade 1 (partial/full thickness ulcer), grade 2 (probing to tendon or capsule), grade 3 (deep with osteitis), grade 4 (partial foot gangrene), and grade 5 (whole foot gangrene). By 1991, Laing and Klenerman [44] described diabetic foot ulcers as primary or secondary—the Liverpool Classification system, Table 3. In 1993, Jeffcoate *et al.* [45] from Nottingham, UK, proposed another classification aimed at being specific, flexible and simple for all health care workers to use whether specialist or not for all likely lesions they would encounter. Their classification failed to ignite the debate they hoped it would stimulate. Jeffcoate *et al.* [45] was based on infection, ischaemia and neuropathy, Table 1. It probably contributed to a more comprehensive classification described in 1996 from the University of Texas (UT) at San Antonio by Lavery *et al.* [46] which incorporated Jeffcoate *et al.* [45] and the elements of Wagner's classification [42, 43]. The UT system assesses ulcer depth, the presence of wound infection, and the presence of clinical signs of lower-extremity ischemia. This

Table 4: University of Texas classification of diabetic foot ulcer showing grade and stage

GRADE		0	I	II	III
STAGE	A	Pre- or post-operative lesion completely epithelialized	Superficial wound, not involving tendon, capsule or bone	Wound penetrating to tendon and capsule	Wound penetrating to bone and joint
	B	Infection (non ischaemic infected)	Infection (non ischaemic infected)	Infection (non ischaemic infected)	Infection (non ischaemic infected)
	C	Ischaemia (non infected ischaemic)	Ischaemia (non infected ischaemic)	Ischaemia (non infected ischaemic)	Ischaemia (non infected ischaemic)
	D	Both infection and ischaemia	Both infection and ischaemia	Both infection and ischaemia	Both infection and ischaemia

system uses a matrix of grade on the horizontal axis and stage on the vertical axis. The grades of the UT system are as follows: grade 0 (pre or post-ulcerative site that has healed), grade 1 (superficial wound not involving tendon, capsule, or bone), grade 2 (wound penetrating to tendon or capsule), and grade 3 (wound penetrating bone or joint). Within each wound grade there are four stages: clean wounds (stage A), nonischemic infected wounds (stage B), ischemic noninfected wounds (stage C), and ischemic infected wounds (stage D). By 1998, a scoring system was described from Benin City in Nigeria by Umebese *et al.* [32] called the Diabetic Foot Severity Score (DFSS), Table 5. The criteria include the colour of the foot, presence of foot pulses, sensation, and grade of ulcer, presence of calcification or osteomyelitis on plain radiographs and age of patients from 40 years and above. The deficiencies of the DFSS have been published [35] based on its incoherence with pathological anatomy of the foot, duplicity of scoring criteria and not reckoning with patients below the age of 40 years. The DFSS was not an improvement over the preceding classifications of Wagner's [42, 43], Laing *et al.* [44], Jeffcoate *et al.* [45] or Lavery *et al.* [46] and which were not referenced in Umebese *et al.* [32] paper. Lavery *et al.* classification [46] is definitely an improvement over Wagner's [42, 43] and is thus, recommended for use in Nigeria. A recent paper [47] comparing the two had confirmed the better predictive value of the UT system.

Table 5: Umebese et al.[32] : Diabetic foot severity system

1	Colour of foot lesion	Score
	Normal	3
	Darker discoloration	2
	Black	1
2	Peripheral pulses	Score
	Dorsalis pedis (DP) and Posterior Tibial (PT) palpable	4
	PT only	3
	DP only	2
	None	1
3	Sensation (light touch and pin prick)	Score
	Normal	3
	(Diminished) hypesthesia	2
	Insensibility to insensate	1
4	Ulcer grading	Score
	Ulcer/gangrene limited to 1 or 2 toes	5
	Full thickness ulceration of the dorsal skin only	4
	Ulcer involvement of more than 2 toes and ball of the foot	3
	Open putrid penetrating ulcer involving more than 50% of the sole of foot	2
	Whole foot gangrene with supramalleolar necrotising cellulitis	1
5	Foot plain radiographs	Score
	Normal	3
	Chronic osteomyelitis (OM) or Calcified peripheral vessels (CPV)	2
	OM + CPV	1
6	Age	Score
	40 years	3
	41-60 years	2
	61 years and above	1

Management of DF in Nigeria

Following clinical, radiological and laboratory evaluation of the patients, a diagnosis of DF is made; patients are managed jointly by the physicians and surgeons. All available reports from Nigeria used the sliding scale of urinary sugar level to determine the amount of insulin to administer for glycaemic control. This is inadequate because better blood sugar analysis is possible using the glucometer—although availability and poor finance can be blamed. Non-operative treatment of ulcers in the Nigerian series includes dressing with eusol and honey to de-slough the wounds. Superficial and deep sloughs are treated with debridement, and further dressings. Skin grafting is subsequently done once good granulation bed allows it. None of the series employed tissue flaps to cover wound defects.

Advanced cases merit various forms of amputation, the most common variety reported was the below knee major amputation representing 73.3% and 55% of DF amputations in the Lawson *et al.* [21] and Anyanwu [24] series. However, Lawson *et al.* [21] had more non-operative treatment (58.3%) than operative (41.7%), while Anyanwu [24] had 70.7% amputation rate and 29.3% non-operative treatment. For some reasons, Nigerians refuse amputation—refusal rate of 12.3%for DF [21] and 48% for tumours [48] have been reported. Reasons adduced included cultural abomination, social stigma as an amputee [25], and difficulty with getting prostheses [28]. This is evidenced by the reported low prosthesis use of 7.0% [28], and 7.5% [29] unlike the developed countries where it approaches 60% [49]. Vascular operative intervention is not reported in any of the Nigerian series probably due more to lack of angiographic evaluation than to shortage of vascular surgeons. However, global practice suggests that up to two thirds of limbs can be saved from revascularization procedure [11, 50]—a wake up call for updating the Nigerian practice. There is the need to embark

on the screening of the at risk group for DF among DM population [11]. Nigeria needs such a declaration like the St. Vincent Declaration [51] which targeted reducing the disease by 50% in 5 years in the UK. Such a giant leap in intention can be a product of multicentre co-operation and governmental funding.

Problems and Future Prospects

A myriad of problems confronts us in the care of DF patients in Nigeria. For example, the low level of literacy of majority of the patients [8, 24, 25, 33], poor earning power [22, 33], late reporting [8, 25], negative cultural and traditional beliefs and more recently the menace of faith healers [25, 52] who promise cure and more—a situation that detracts the DF patients from seeking orthodox care before complications set in. Positive challenges in our environment include making early and proper diagnosis of DF at the diabetic clinic, inadequate access to arteriography due to lack of equipment and the fact that vascular surgeons are in short supply. There is need to adequately fund research into DM and DF.

Areas of improvement would include establishing multidisciplinary foot clinics [6, 53], a measure that has improved the care of DF patients in the developed countries. Generally, an improvement in the Nigerian economy will impact positively on the individual DF patients and the institutions that care for them through equipment acquisition, manpower training and conduct of further research. Health education on early reporting, screening, legislation against unwholesome claims by traditional and faith healers, provision of prostheses and formation of DF amputee club to serve as counselling unit for others. Interventional radiology using angioplasty, stent insertion, loco-regional fibrinolysis and mechanical atherectomy—all of which can facilitate intravascular revascularization—should be acquired[54]. This is particularly suited for diabetics with multi-system impairment, who are unfit for surgical revascularization. The diagnosis of osteomyelitis in the presence of soft tissue infection and neuropathic bone changes is often difficult in DF. Scintigraphy using Technicium 99 methylene diphosphonate, Gallium 67 or Indium 111 autologous leukocytes has resolved this difficulty [55]. In the series by Solagberu, *et al.* [8], no osteomyelitis variety of DF was found, probably due to the limitation of plain radiographs which scintigraphy and magnetic resonance imaging (MRI) [56], presently unavailable in most of the centres in Nigeria, would have resolved. Needless to say, MRI acquisition would improve the overall evaluation and care given to DF patients.

Conclusion

The management of DF in Nigeria is still at its infancy. However, DF in the last 40 years in Nigeria has moved from being "rare" to being "common" in our practice setting. The rising LL amputation rate from DF has changed the old trend of indications for amputation to what had obtained in the developed countries, therefore, the need for adequate research in DF in Nigeria cannot be overemphasized. The progression of DF from minor foot trauma to foot gangrene, ending in amputation must change. Prosthetic technology and ready availability of prosthesis should reduce the high rate of refusal of amputation. A national symposium on DM and DF should issue a declaration like the St. Vincent's [51] to galvanize all issues raised here and energise Nigerian caregivers to effectively control DM, for as Lording said; "give diabetes an inch, it would take a foot" [57].

References

1. Armstrong DG, Lavery LA, Harkles LB. Who is at risk for diabetic foot ulceration? Clin Podiatr Med Surg 1998; 15: 11-19.
2. Naaeder SB. Amputation of the lower limb in Korle-Bu Teaching Hospital, Accra. West Aft J Med 1993; 12: 21-26.
3. Solagberu BA, Onawola KO. Lower limb amputations in a West African community: a new trend? Trop Doc 2001; 31: 250.
4. Onuba O, Udoidiok E. The scope of amputations in the developing countries. Postgraduate Doctor Africa 1989; 11: 118-121.
5. Barnett A. Prevention and treatment of the diabetic foot ulcer. Br J Nurs 1992; 2: 7-10.
6. Reiber GE. Diabetic foot ulcer. Financial implications and practice guidelines. Diabetes Care 1992; 15 Suppl 1: 29-31.
7. Thompson C, McWilliams T, Scott D, Simmons D. Importance of diabetic foot admissions at Middlemore Hospital. N Z Med J 1993; 106; 178-180.
8. Solagberu BA, Kuranga SA. Morbidity and mortality from diabetic foot. Postgraduate Doctor Africa 2002; 24: 10-12.
9. Apelqvist J. Wound healing in diabetes. Outcome and costs. Clin Podiatr Med Surg 1998; 15: 21-39.
10. Ahroni JH. The care of lower extremity lesions in patients with diabetes. Nurse Prac Forum 1991; 2: 188-192.
11. Lavery LA, Armstrong DG, Velsa SA, Quebedeaux TL, Fleischli JG. Practical criteria for screening patients at high risk for diabetic foot ulceration. Arch Intern Med 1998; 158: 157-162.

12. The Italian Study Group for the implementation of the St. Vincent Declaration. Risk factors for lower limb complications in diabetic patients. J Diabetes Complications 1998; 12: 10-17.
13. Diabetes Audit and Research in Tayside Scotland/ Medicines Monitoring Unit DARTS/ MEMO Collaboration. Diabetes and lower limb amputations in the community. A retrospective cohort study. Daibetes Care 1998; 21: 738-743.
14. Trautner C, Haastert B. Incidence of lower limb amputations and diabetes. Diabetes Care 1996; 19: 1006-1009.
15. Vink J de M, Angawa JOW. Prevalence of diabetes. East Afr Med J 1956; 33: 366-370.
16. Kinnear TWG. The pattern of diabetes mellitus in a Nigerian teaching hospital. East Afr Med J 1963; 40: 288-294.
17. Ohwovoriole AE, Kuti JA, Kabiawu SIO. Casual blood glucose levels and prevalence of undiscovered diabetes mellitus in Lagos metropolis Nigerians. Diabetes Res Clin Prac 1988; 4: 153-158.
18. Erasmus RT, Fakeye T, Olukoga E et al. Prevalence of diabetes mellitus in a Nigerian population. Trans R Soc Trop Med Hyg 1989; 417-418.
19. Osuntokun BO, Akinkugbe FM, Francis TI, Reddy S, Taylor GOL. Diabetes Mellitus in Nigerians: a study of 832 patients. West Afr Med J 1971; 20: 295-312.
20. Adetuyibi A. Diabetes in the Nigerian African. Review of long term complications. Trop Geo Med 1976; 28: 155-159.
21. Lawson EAL, Oyemade GAO, Adetuyibi A. The foot complications in the Nigerian diabetic patient. Niger Med J 1978; 8: 401-403.
22. Ndububa DA, Erhabor GE. Diabetes mellitus in Nigerian patients: a six-year review of hospital admissions. Nig med J 1996; 30: 134-138.
23. Osisioma ECO, Onuminya JE. Diabetic Foot Gangrene. Nigerian Journal of Surgical Sciences 1992; 2: 62-66.
24. Anyanwu SNC. The diabetic foot—a major surgical problem in our environment. Nig J Surg 1994; 1: 26-28.
25. Bojuwoye BJ. Clinical pattern, management and problems of diabetes mellitus in Ilorin, Nigeria. The Tropical Journal of Health Sciences 1995; 2: 1-5.
26. Ogirima MO, Asaku ME, Ukwenya AY, Udezue NO. Diabetes extremities in Kaduna. Nig J Surg Res 1999; 1: 21-24.
27. Garba ES, Deshi PJ, Ihejirika KE. The role of traditional bonesetters in limb amputations in Zaria. Nig J Surg Res 1999; 1: 21-24
28. Solagberu BA. Scope of amputations in a Nigerian teaching hospital. Afr J Med med Sci 2001; 30: 225-227.
29. Katchy AU, Chukwu COO, Nwankwo CO, Onabowale BO. Lower limb amputations in a specialist hospital: review of 120 patients. Orient J Med 1997; 9: 27-29.
30. Beach KW. Starndless DE. Arteriosclerosis obliterans and associated risk factors in insulin-dependent and non-insulin dependent diabetes. Diabetes 1980; 29: 882-888.
31. Franklin GM, Kahn LB, Baxter J, Marshall JA. Sensory neuropathy in non-insulin dependent diabetes mellitus. The San Luis Valley Diabetes Study. Am J Epidemiol 1990; 131: 633-643.
32. Umebese PFA, Ogbemudia AO. Mangement of diabetic foot; objective results in 40 patients using a new Diabetic Foot Severity Score. Nig J Surg 1998; 5: 10-12.
33. Solagberu BA. Diabetic foot: need for a multicentre study. Nig J Surg Res 2001; 3: 44
34. Ogirima MO. Diabetic foot: need for a multicentre study: reply. Nig J Surg Res 2001; 3: 44-45.
35. Solagberu BA. Comments on "Management of diabetes foot; objective results in 40 patients using a new Diabetic Foot Severity Score". Nig J Surg 2000; 7: 35-36.
36. The global Extremity Amputation Study Group. Epidemiology of lower extremity amputation in centres in Europe, North America and East Asia. Br J Surg 2000; 87: 323-337.
37. Margaret KH. Perspectives in diabetes. Handicaps to host defence, effects of hyperglycaemia on C3 and Candida albicans. Diabetes 1970; 39: 271-275.
38. Goldenberg S, Alex M, Joshi RA, Blumenthal HT. Nonartheromatous peripheral vascular disease of lower extremity in diabetes mellitus. Diabetes 1959; 8: 261-273.
39. LoGerfo FW, Coffman JD. Vascular and microvascular disease of the foot in diabetes. Implications for foot care. N Eng J Med 1984; 311: 1615-1619.
40. Thomas PK, Lascelles RG. The pathology of diabetic neuropathy. Q J Med 1966; 35: 489-509.
41. Birrer RB, Dellacorte MP, Grisafi PJ. Prevention and care of diabetic foot ulcers. Am Fam Physician 1996; 53: 601-611, 615-616.
42. Wagner FW. Orthoapedic rehabilitation of dysvascular lower limb. Orth Clin Nor Am 1978; 9: 325-350.
43. Wagner FW. Supplement: algorithms of foot care.

In: The diabetic foot. 3rd Ed. Levin ME, O'Neal LW, Eds. St. Louis, MO, CV Mosby 1983: 291-302.

44. Laing P, Klenerman L. The foot in diabetes. In: Klinerman L (ed): The foot and its disorders. London: Blackwell Scientific Publications; 1991: 139-152.
45. Jeffcoate WJ, MacFarlane RM, Fletcher EM. The description and classification of diabetic foot lesions. Diabetes Med 1993; 10: 676-679.
46. Lavery LA, Armstrong DG, Harkless LB. Classification of diabetic foot ulceration. J Foot Ankle Surg 1996; 35: 528-531.
47. Oyibo SO, Jude EB, Tarawneh I, Nguyen HC, Harkless LB, Boulton AJM. A Comparison of Two Diabetic Foot Ulcer Classification Systems. The Wagner and the University of Texas wound classification systems. Diabetes Care 2001; 24: 84-88.
48. Oyemade GAA, Junaid TA. Osteosarcoma of the bone: clinico-pathological features of sixty-nine cases in Ibadan, Nigeria. Int Surg 1982; 67: 553-555.
49. Nathan P, Jennifer K, David, Nicholas L, Crane TC. Natural history of the leg amputee. Am J Surg 1977; 133: 467-473.
50. Ebskov LB, Schroeder TV, Holstein PE. Epidemiology of leg amputation: the influence of vascular surgery. Br J Surg 1994; 81: 1600-1603.
51. Diabetes Care and Research in Europe: The St. Vincent Declaration. Geneva, World Health Organization, 1989 (ICP/CLR 034).
52. Ewins DL, Bakker K, Young MJ, Boulton AJ. Alternative medicine; potential dangers for the diabetic foot. Diabet Med 1993; 10: 980-982.
53. Knowles EA, Gem J, Boulton AJ. The diabetic foot and the role of multidisciplinary clinic. J Wound Care 1996; 5: 452-454.
54. Cotroneo AR, Citterio F, Cina A, Di Stasi C. The role of interventional radiology in the treatment of diabetic foot. Rays 1997; 22: 612-637.
55. Fox IM, Zeiger L. Tc 99m HMPAO leukocyte scintigraphy for the diagnosis of oateomyelitis in diabetic foot infection. J Foot Ankle Surg 1993; 32: 591-594.
56. Lipsky BA. Osteomyelitis of the foot in diabetic patients. Clin Infect Dis 1997; 25: 1318-1326.
57. Lording D W. The impact of diabetes on the lower limb. Give diabetes an inch and it will take a foot. Aust Fam Physician 1993; 22: 1583-1585; 1587-1590.