Hallux Ulceration in Diabetic Patients

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Original Research

Hallux Ulceration in Diabetic Patients

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ABSTRACT

We undertook a prospective cohort study to assess risk factors associated with hallux ulceration, and to determine the incidence of healing or amputation, in consecutive patients with diabetes mellitus who were treated over the observation period extending from September 2004 to March 2005, at the Jabir Abu Eliz Diabetic Centre, Khartoum City, Sudan. There were 122 diabetic patients in the cohort (92 males and 30 females) with an overall mean age of 58 ± 9 years. Fifty-three percent of patients had complete healing within 8 weeks and 43% healed within 20 weeks. The overall mean time to healing was 16 ± 8 weeks. In 32 (26.2%) patients, osteomyelitic bone was removed, leaving a healed and boneless hallux. The hallux was amputated in 17 (13.9%) patients; in 2 (1.6%) patients it was followed by forefoot amputation and in 7 (5.7%) patients by below-the-knee amputation. In 90 (73.8%) patients the initial lesion was a blister. In conclusion, hallux ulceration is common in patients with diabetes mellitus and is usually preceded by a blister. Neuropathy, foot deformity, and wearing new shoes are common causative factors; and ischemia, osteomyelitis, any form of wound infection, and the size of the ulcer are main outcome determinants. Complete healing occurred in 103 (85%) of diabetic patients with a hallux ulcer. Vascular intervention is important relative to limb salvage when ischemia is the main cause of the ulcer.

The development of a diabetic foot wound is a result of both local and general factors. An insensitive, deformed, and/or ischemic foot is likely to develop an ulcer (1). In addition to abnormal biomechanical factors (2), abnormal gylcosylation of keratin (3) and collagen (4) adds to the risk of ulceration. Uncontrolled diabetes of long duration, retinopathy, nephropathy (5), and smoking (6) also add significantly to the risk of cutaneous compromise (7). The neuropathic ulcer is commonly located beneath the head of the first metatarsal; however, it may develop at any site on the foot where weight bearing or contact pressure is applied. Approximately one third of all diabetic foot ulcers occur on the hallux (8). Wounds occur in this area because of a variety of factors, most significantly limitation of motion at the metatarsophalangeal joint (MTPJ) (9), which leads to increased plantar pressure on the hallux (10). When this latter effect is combined with repetitive stress, such as is the case with regular walking activity, it can result in a neuropathic foot ulceration (11). Jabir Abu Eliz Diabetic Centre is the only specialized center of its kind in Khartoum City, and many diabetic patients are seen daily for the care of foot ulceration in our clinic. The aim of this study was to assess hallux ulceration in regard to possible causes, management, and outcomes.

Patients and Methods

This was a prospective study performed between September 2004 and March 2005. All diabetic patients consecutively presenting with a hallux ulceration were included in the cohort. Patients were asked about their main presenting symptoms and any underlying suspected causative injury. The history included the type of diabetes, family history of diabetes, smoking habits, sexual function in males, and history of hypertension. General physical examination was carried out, and this included ophthalmological examination of the fundus. Local findings related to examination of the foot and the affected hallux were recorded. The main variables that were assessed included sensory neuropathy, ischemia, foot deformities, and evidence of osteomyelitis. Neuropathy was assessed using the 10-g Semmes-Weinstein monofilament and vibration sense using a 128-Hz tuning fork. The first was applied twice to 3 sites: first metatarsal head, hallux toe pulp, and fifth metatarsal head. Absent sensation was documented when 2 sites were unfelt by the patient. The tuning fork was applied percutaneously to the bony part of the dorsal aspect of the distal phalanx of the first toe. Limb ischemia was assessed by digital palpation of both dorsalis pedis and posterior tibial arteries. Doppler ultrasound was used in those patients when pulses were absent to manual palpation. Foot deformities were also recorded. The ulcer was measured using the longest diameter by a right-angle crossing diameter. The degree of wound sepsis was assessed clinically via local examination for the presence of erythema, swelling, warmth, or tenderness, and the presence of any purulent discharge, as well as its color and odor; specimens of the involved tissues were sent for bacteriological culture. Osteomyelitis was diagnosed using plain film radiographs, by means of identification of the presence of bone destruction and resorption, or when bone fragments

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Table 1
Prevalence of patient occupations (N = 122 patients)

Occupation	Count (%)
Retired	33 (27.0)
Housewife/homemaker	28 (23.0)
Small business	26 (21.3)
Government employee	17 (14.0)
Farmer	11 (9.0)
Laborer	7 (5.8)

were easily probed and removed during the clinical debridement of an infected ulcer communicating with the bone, thereby obviating the need for radiological assessment. Bone scan and magnetic resonance image (MRI) scans were not available in our center during the period of observation. Bone fragments were sent for bacteriological culture and sensitivity to confirm a diagnosis of osteomyelitis.

The management of the ulcer and its course and outcome were also documented using preformed case report forms. Blisters were incised and deroofed via excision of the epithelial covering, followed by gauze dressing using normal saline. Infected blisters were drained and swab specimens taken for culture and sensitivity. Empirical oral antibiotic was given to patients with an ulcer and localized cellulitis within 3-cm margins of the wound, using either oral cephalexin in a dosage of 500 mg every 6 hours for 7 days or oral clavulanate (125 mg)-potentiated amoxicillin (875 mg) every 12 hours for a week. Injectable third-generation cephalosporin (ceftriaxone) was given in the presence of systemic sepsis or cellulitis in a dosage of 1 g intravenously every 12 hours for 3 days until the results of bacteriological culture were available, when oral administration was commenced according to the result of the culture. Healing was defined as complete epithelialization in a patient who was able to return to wearing shoe gear and to ambulate in a weight-bearing fashion. Infected wounds were treated by sharp debridement using a scalpel or curette on a daily basis (12) until healthy tissues were established, or whenever granulations required freshening. Wound probing and toe squeezing were both useful to demonstrate sinus tracts that required debridement. The final outcome was assessed in regard to the main possible determinant factors, namely ischemia, osteomyelitis, any type of wound infection, and wound size as determined by the largest diameter (cm). The data were analyzed using SPSS, Version 12.0 (SPSS, Inc., Chicago, IL); the chi-square test was used to determine statistical significance. Statistical significance was defined at the 5% ($P \leq .05$) level for all of the computations.

Table 2
Prevalence of clinical characteristics of diabetic patients with hallux ulceration (N = 122 patients)

Vascular assessment [N = 122]			
Palpable posterior tibial artery	106 (86.9%)		
Palpable dorsalis pedis artery	102 (83.6%)		
Sensory neuropathy [n = 105 (86.1%)]			
	Normal	Diminished	Absent
10 g monofilament nylon	15 (14.3%)	65 (61.9%)	25 (23.8%)
128 Hz vibration sense	15 (14.3%)	65 (61.9%)	25 (23.8%)
Structural and skin deformity [N = 122]			
Hallux valgus	34 (27.8%)		
Callus formation	38 (31.2%)		
Hammer toe	13 (10.7%)		
Hallux rigidus	7 (5.7%)		
Initial lesion a blister, and cause [n = 90 (73.8%)]			
Spontaneous	42 (46.7%)		
Followed new shoe	41 (45.6%)		
Walking bear foot	7 (7.8%)		
Location on hallux [N = 122]			
Plantar	77 (63.1%)		
Dorsum	21 (17.2%)		
Medial	17 (13.9%)		
Lateral	7 (5.8%)		
Bilateral	3 (2.5%)		
Ulcer diameter (cm) [N = 122]			
< 1	8 (6.6%)		
≥1 to 1.5	10 (8.2%)		
>1.5 to 2	45 (36.9%)		
>2 to 3	40 (32.8%)		
>3 cm	19 (15.6%)		
Involved side [N = 122]			
Right hallux	67 (54.9%)		
Left hallux	55 (45.1%)		
Type of shoes [N = 122]			
Open shoes	91 (75.0%)		
Modified shoes	15 (12.0%)		
Regular closed shoes	16 (13.0%)		

Table 3
Prevalence of types of injury causing hallux ulceration in diabetic patients (N = 122 patients)

Cause	Count (%)
Spontaneous	43 (35.1)
New shoe	41 (33.6)
Direct trauma	12 (10.0)
Puncture	10 (8.2)
Foreign body	8 (6.5)
Heat exposure	7 (5.8)
Tight wound dressing	1 (0.8)

Results

Our cohort consisted of 122 diabetic patients with hallux ulceration. Of these, 92 (75.4%) were male, and 30 (24.6%) were female. The overall mean age was 57.9 ± 9.2 years. Table 1 shows the prevalence of the different occupations associated with the cohort. Of the 33 retired workers, 27 (81.8%) had either cataract or retinopathy. Table 2 shows the prevalence of several clinical characteristics related to hallux ulceration in the patients in our cohort, and Table 3 shows the prevalence of the different etiological factors related to hallux ulceration. The most prevalent risk factor was peripheral sensory neuropathy, which was present in 85.7% of the cohort. Most (> 83%) of the patients displayed at least one palpable pedal pulse, 68.9% of the hallux ulcers developed either spontaneously or in association with new shoe gear, and 33.6% of the patients displayed either hallux valgus or hallux limitus. Table 4 shows the prevalence of various risk factors associated with the hallux ulceration, and factors leading to delayed wound healing in diabetic patients, along with the statistical probability that the risk factor contributed the presence of the hallux ulcer. Table 5 depicts the duration of time required to heal the hallux ulcer, either by means of secondary intention or via primary or tertiary closure following amputation.

One hundred and three (84.4%) patients presented initially with an ulcer, rather than a blister or wound with an intact overlying skin barrier, of which 52 (42.6%) patients had an ulcer with cellulitis, and 15 (12.3%) displayed signs and symptoms consistent with septicemia. In 11 (9%) patients, the ulcer followed incision and drainage of a hallux abscess in our center, and in 8 (6.6%) patients it followed debridement of wet gangrene of the hallux. Osteomyelitis was evident in 32 (26.1%) patients, of whom 9 (7.4%) patients presented with gross hallux edema and radiological evidence of osteomyelitis. Ninety (73.8%) patients gave a history of a blister as an initial lesion, which was treated at home by drainage and/or excision of the margins using needles and scissors, cleansing and dressing that consisted of boiled saline (boiled with table salt), and gauze (Figure 1). After several days or weeks, if the wound had not healed, then the patient would present to our clinic, often with frank sepsis. Late, dry blisters were treated conservatively if no active sepsis was present, and continued until the wound sloughed. The blister followed wearing new shoe in 41 (33.6%) patients. In 7 (5.8%) patients the blister followed walking on a hot surface after the slippers (open shoes) had been removed or dropped off, and we suspect that the patient was unaware of the risk because of peripheral neuropathy. In the remaining 43 (35.1%) patients, no identifiable cause was found. Fifty-two (42.6%) patients had an infected hallux ulcer upon arrival to our clinic, and all of these

Table 4
Risk factor associated with the presence of hallux ulceration and factors leading to delayed wound healing in diabetic patients (N = 122 patients)

General risks for ulcer development	Count (%)	*P* value*
Diabetes for >10 y	101 (82.8)	.002
Positive family history of diabetes	90 (73.8)	.002
Impotence	67 (54.9)	.004
Smokers	47 (38.5)	.02
Hypertension	41 (33.6)	.03
Eye problems	38 (31.2)	.04
Local risks for ulcer development		
Sensory neuropathy	86 (70.5)	.003
Foot deformity	44 (36.1)	.025
Wearing new shoes	41 (33.6)	.03
Callus	40 (32.8)	.03
Factors associated with delayed healing		
Ulcer >3 cm diameter	19 (15.6)	.002
Presence of osteomyelitis	32 (26.2)	.001
Infection	52 (42.6)	.04
Ischemia	16 (13.1)	.05

* Probability calculated using chi-square test.

were patients presenting more than 2 weeks after the onset of a hallux wound. Fifty (41%) patients presented to our clinic within 2 weeks of the onset of a hallux wound, whereas 36 (29.5%) presented between 2 and 4 weeks, and another 36 (29.5%) presented more than 4 weeks after the onset of ulceration.

Sixty-five (53.3%) patients had immediate care at our center whereby the ulcer underwent regular dressing changes using normal saline until healing was complete. Forty-four patients (36.1%) carried on treatment at home, after receiving initial therapy in the clinic, using any available antiseptic solution of which 10 (8.2%) patients used a form of concentrated spirit (ethanol) that worsened the ulcer. Fifteen (12.3%) patients returned with signs and symptoms consistent with wound sepsis and septicemia. Infected wounds were managed by initial sharp surgical debridement and dressing using 10% povidone iodine. Intravenous systemic antibiotic was also used initially for a minimum of 3 days, or until the results of bacteriological cultures from blood and/or deep wound tissues were available. Probing the wound for osteomyelitis and extraction of bone fragments for culture and sensitivity was undertaken routinely, if deep wounds were present. Of 77 bacteriological cultures, 56 (72.7%) grew *Staphylococcus aureus*. Probing was relied on as a means of determining the presence (high likelihood) of osteomyelitis, and obviated the need for urgent radiological examination of the involved, as radiological services were not available at all times. Palpation, probing, and simply squeezing between 2 fingers hallux tissues suspected of being infected, often produced a discharge strongly suggestive of infection, and indicated the need for thorough surgical debridement. The mean overall duration of time to healing was 16 ± 8 weeks, at which time the patients were fully ambulatory with the use of biomechanically modified shoe gear (Table 5).

Only 3 (2.5%) patients had bilateral hallux ulceration, of which 1 ulcer was essentially healed at the time of presentation, and only the persistent ulcer was considered in this study. In 32 (26.2%) patients, bones with osteomyelitis were removed (hallux fillet), and the healthy residual soft tissues enabled the boneless hallux to heal satisfactorily (Figure 2). Amputation of the distal phalanx was

Table 5
Time to healing of hallux ulceration (N = 122 patients)

Weeks	Count (%)
< 2	10 (8.2)
≥ 2 to 4	26 (21.3)
≥ 4 to 8	29 (23.8)
≥ 8 to 16	29 (23.8)
≥ 16 to 24	24 (19.6)
≥ 24	4 (3.3)

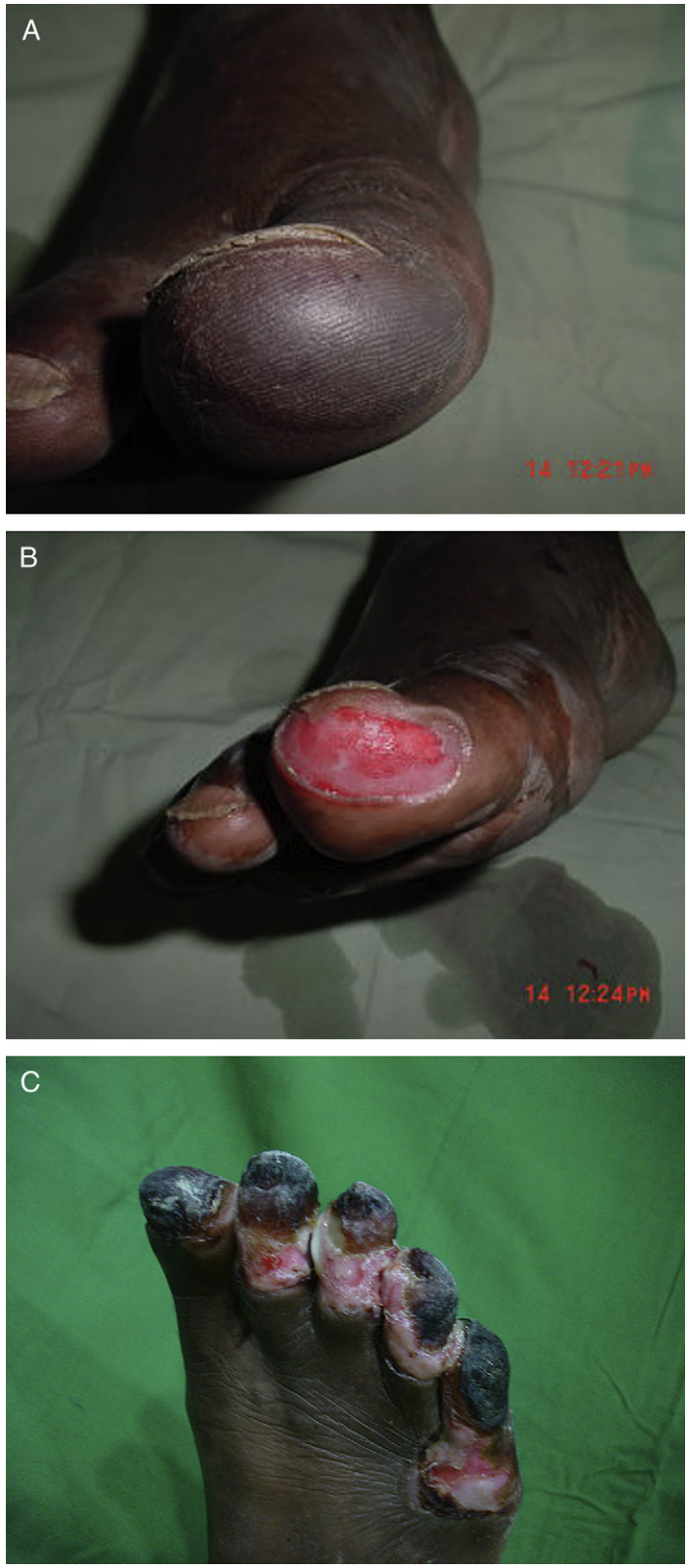

Fig. 1. Hemorrhagic blister of the hallux (*A*) before and (*B*) after drainage and excision of the skin. (*C*) Late dry skin gangrene in another patient, managed conservatively.

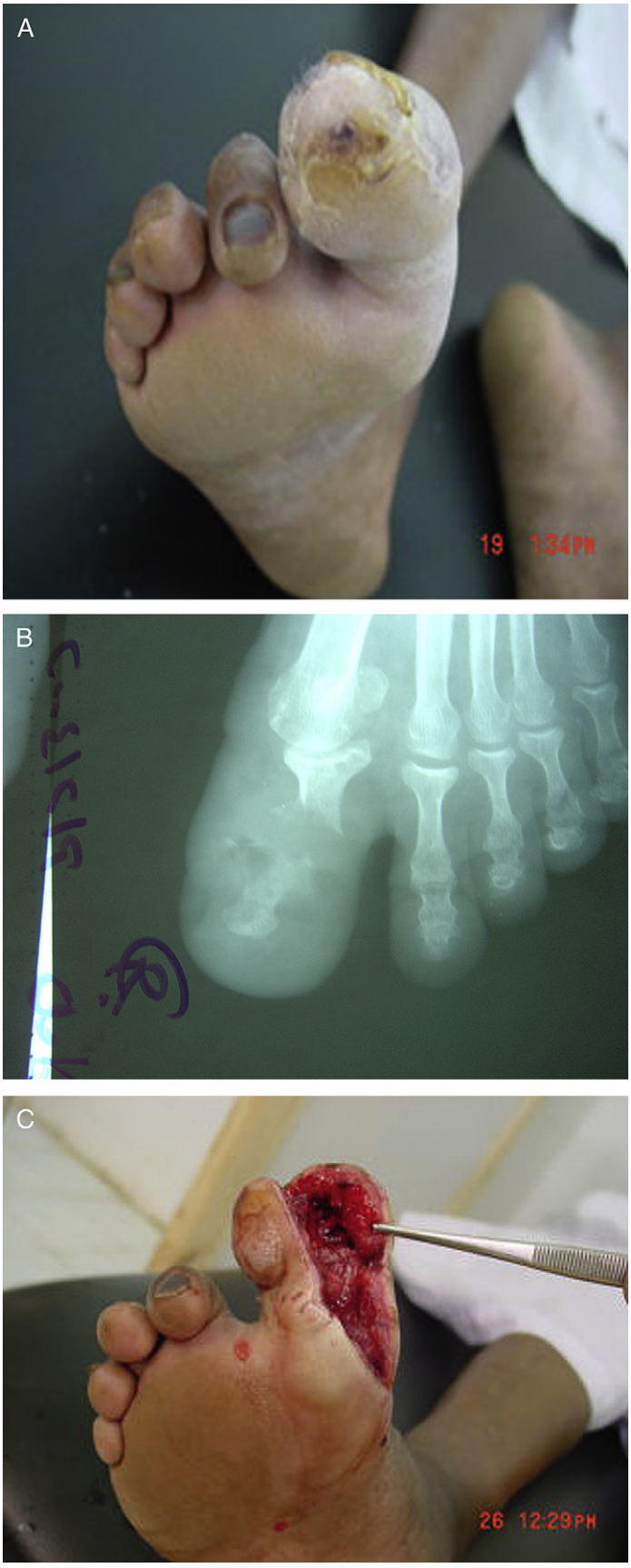

Fig. 2. Osteomyelitis of the hallux, (*A*) clinical and (*B*) radiographic appearance. (*C*) Surgical excision of the infected and necrotic soft tissues and bone.

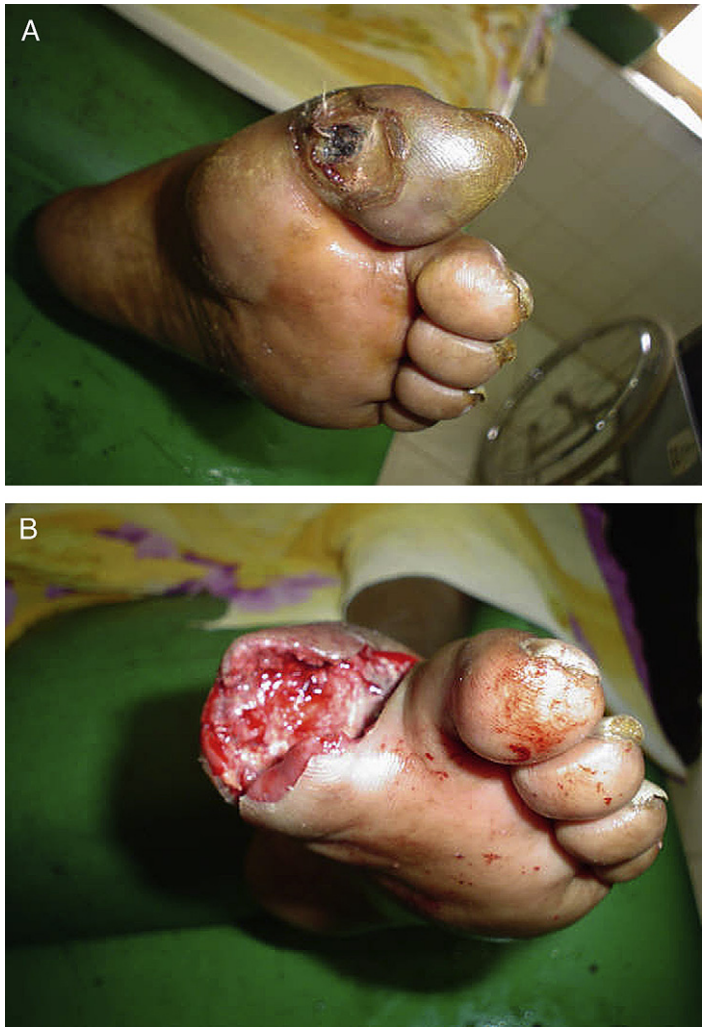

Fig. 3. (*A*) Hallux pulp ulceration with underlying osteomyelitis. Because of the extensive involvement of underlying bone, treatment consisted of hallux amputation (*B*), which went on to full healing after 6 weeks.

undertaken in 6 (4.9%) patients, and complete hallux amputation was carried in 17 (13.9%) patients, of which 2 (1.6%) ischemic patients ended up undergoing forefoot amputation and 7 (5.7%) ischemic patients eventually underwent below-the-knee amputation. Those patients with osteomyelitis of the hallux, with good limb circulation but necrotic skin and muscles, also ended up with hallux amputation (Figure 3). The combination of a larger ulcer ($>$ 1cm diameter) and osteomyelitis increased the likelihood of amputation or hallux fillet (χ^2 test, $P = .001$). Unfortunately, at our institution during the period of the investigation, none of the patients had the opportunity to undergo reconstructive vascular intervention; hence, amputation at a proximal level that was deemed suitable for healing was the intervention of choice for patients with ischemia and nonhealing wounds and/or osteomyelitis of the hallux.

Discussion

The diabetic foot is a common and serious problem, and foot ulcers precede 85% of nontraumatic lower extremity amputations (LEAs) (6). Approximately 3% to 4% of individuals with diabetes mellitus currently have foot ulcers or deep infections, and 15% of diabetic individuals develop foot ulcers during their lifetimes (6). Foot

disorders are a leading cause of hospitalization for diabetic individuals and account for billions of dollars in annual expenditure worldwide. Although these disorders cannot be prevented in all cases, dramatic reductions can be achieved with the use of a multidisciplinary approach to prevention and care (13). It remains sad to say, however, that by approximately 2 years following transtibial amputation, 36% of patients have died (14, 15), mostly because of concomitant cardiovascular disease that affects the brain, heart, and kidneys. In this study, males were affected more often than were females (75%, male-to-female ratio approximately 3:1); however, after the age of 65 years, the male-to-female ratio equalized. These findings, we feel, were because of the increased weight-bearing activity required of males in our population before age 65 years. The association of hypertension and ophthalmological involvement in nearly one third of patients was also a significant finding. The atherosclerosis associated with hypertension, in addition to impairment of peripheral sensation and vision dysfunction because of macular degeneration, combine to make the foot vulnerable to cutaneous compromise. Hallux ulceration was found to be common in elderly diabetic patients with impaired vision and peripheral neuropathy that inhibited stability during ambulation and gait. For these reasons, we recommend that the family members of those with these conditions be educated and trained to daily inspect and assist their elderly, neuropathic, diabetic family members in an effort to minimize hallux ulceration.

It was also interesting to note that hallux ulceration was preceded with a blister in more than 70% of the patients, and this was usually the result of wearing a new shoe, blunt trauma, or a puncture. Shoe-related minor trauma is the most frequent event leading to ulceration and amputation of the hallux in diabetic patients (16). It has been reported that half of all amputations in diabetic patients are preceded by injury from footwear (17), and specialized manufactured diabetic shoes are an important form of protection from foot ulceration (18). A systematic approach to off loading and proper shoe fitting, coupled with podiatric services, is available in only one center in Khartoum. Unfortunately, in Khartoum, we believe that patients lack an awareness of the importance of this service.

Several structural abnormalities of the hallux were encountered in this study, including hallux rigidus, lesser digital hammertoe, and hallux valgus. Bresater et al (19) reported callus formation and hallux valgus as the most frequent abnormalities associated with hallux ulceration. Nube et al (20) reported that hallux ulceration was associated with a more pronated foot type. Foot deformities may also be related to the type of footwear being used, and hallux ulceration has not been diagnosed in certain populations where diabetic individuals wear broad sandals with ample space for the toes (21).

The most common site of ulceration in our study was the planter surface of the hallux, with callus formation found in about 33% of the patients. This finding was similar to that noted in other observational studies (22, 23), which showed the greatest frequency of ulceration occurred under the metatarsal heads. In a multivariate study including 446 patients, hyperkeratosis was reported as 1 of 4 significant factors for development of foot ulceration, along with retinopathy, poor psychosocial status, and duration of diabetes (24).

Still further, in our study, 53.3% of the ulcers completely healed within 8 weeks in patients who were compliant with wound management in our diabetic center. Surgical debridement (12, 25) was the key method in our management and, in particular, sharp debridement (26) using a scalpel to remove necrotic tissues was done on a daily basis until the wound was rendered clean.

Sheehan et al (27) found that the best healing time was achieved within 12 weeks in 58% of patients, and Frykberg et al (22) reported healing time of the foot ulcer in 10 weeks in 55%. Piaggesi and colleagues (28) reported healing at 25 weeks in 79% of patients with neuropathic ulcers after conventional treatment, compared with 96% after excision of the ulcer and adjacent bone. However, in spite of good management, healing rates in large multicenter trials were 24% at 12 weeks and 31% at 20 weeks (29). Johnson et al (30) advised mandatory amputation for patients with hallux ulcer and bony involvement in addition to deep infection. However, removal of affected bone only or local amputation with antimicrobial therapy is a recommended optimum treatment in cases of osteomyelitis (31), and even though medical therapy alone has been advocated (32), we feel that surgical debridement is the mainstay of therapy for these wounds.

Overall, we evaluated 122 patients with hallux wounds related to diabetes mellitus. Of these, 32 patients with adequate limb perfusion were managed with removal of osteomyelitic bone ending with a boneless big toe rather than amputation, and this was met with high patient acceptance. Whether this procedure on the hallux will be associated with subsequent deformity and cutaneous compromise involving the second and third toes, as has been described (33), remains to be seen, and we continue to follow our cohort forward over time.

In conclusion, the main etiological factors leading to big toe ulceration were the degree of neuropathy and foot deformities, whereas the final outcome was determined by the degree of limb ischemia, size of the ulcer, and severity of sepsis as evidenced by osteomyelitis. Blisters that are likely to be preventable preceded most hallux ulcers, which appeared to be the result of tight shoes or heat exposure. Established hallux sepsis in the absence of critical ischemia warrants drainage and debridement, with removal of infected bone fragments in conjunction with intravenous broad-spectrum antibiotics. This must be accompanied with control of blood glucose and proper wound care until complete healing, and this usually takes up to 8 weeks of therapy. Thereafter, we recommend long-term ongoing surveillance of the patient, in an effort to minimize pedal morbidity.

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