Diabetic Foot Management at the Primary Care Level

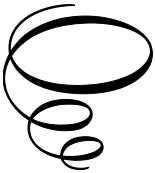
Diabetic Foot Management at the Primary Care Level:

An Evidence-Based Approach

Edited by

Hashim Mohamed

Cambridge Scholars Publishing



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This book is dedicated to a number of people, including my late father who passed away without realising his dream of seeing his young boy become a doctor, and to a unique lady, Badriya Al lenjawi, who is both my lovely wife and favorite coach, and our four children Ali, Houraa, Abdulla and Mohamed, and to those patients who suffered from diabetic foot ulcers, lost limbs, their families and those health care professionals who care for people with diabetic foot problems.

TABLE OF CONTENTS

Acknowledgements xii

Chapter 1 1
Epidemiology of the Diabetic Foot
Dr Hashim Mohamed
1.1 Risk of ulceration 2
1.2 Mechanisms of injury 3

Case Study – I 8

Case Study – II 12

Case Study – III 14

Chapter 2 15
Diabetic Foot Infection
Dr Hashim Mohamed 15
2.1 Risk of infection 15
2.2 Medical management of diabetic foot infections 16
2.3 Diagnosis and clinical presentation 17
2.4 Treatment 21

Case Study – IV 30

Case Study – V 32

Case Study – VI 34

Chapter 3 36
Risk of Ulceration
Dr Hashim Mohamed
3.1 Peak plantar pressure 36
3.2 Quantitative measurement 37
3.3 Conclusion 43

Case Study – VII 46

Case Study – VIII 48

Case Study – IX 50

Chapter 4 52
Assessment of the Diabetic Foot at Primary Care Level
Dr Hashim Mohamed
4.1 General inspection 52
4.2 Vascular assessment 57
4.3 Risk classification and referral/follow-up 59
4.4 Conclusions 60

Case Study – X 63

Case Study – XI 65

Chapter 5 68
Classification of Diabetic Foot Ulcers
Dr Hashim Mohamed
5.1 Wagner's classification 69
5.2 The University of Texas classification 70
5.3 Diabetic foot ulcer clinical evaluation, treatment and prevention .. 71

Case Study – XII 75

Case Study – XIII 76

Chapter 6 78
The Pathophysiology of Wound Healing
Dr Hashim Mohamed

Case Study – XIV 82

Case Study – XV 85

Case Study – XVI 87

Chapter 7 89
Diagnosis and Management of Diabetic Neuropathy in Primary Care
Georgios Ponirakis, Adnan Khan, Hamad M Almuhannadi,
and Rayaz A Malik
7.1 Why is it important to detect diabetic neuropathy
in primary care? 89
7.2 How to diagnose diabetic neuropathy 92
7.3 Autonomic neuropathy 96
7.4 Treatment of diabetic neuropathy 99
7.5 Treatment of painful diabetic neuropathy (Table 2) 99

Case Study – XVII 111

Chapter 8 114
Principles of Management of Acute and Chronic Diabetic Wounds
Dr Hashim Mohamed
8.1 Acute wounds 114
8.2 Chronic wounds 114
8.3 Diabetic ulcer 116
8.4 Local wound management principles 116
8.5 Adjuvant agents 120
8.6 Ulcer recurrence 121
8.7 The future 121

Case Study – XVIII 125

Chapter 9 128
Wound Care Products: The Past, Present and the Future
Dr Badriya Al Lenjawi
9.1 Introduction 128
9.2 Classification of dressings 130
9.3 Herbal wound therapy 135
9.4 Ultrasound 136
9.5 Lasers and wound healing 136
9.6 Natural honey and wound healing 137
9.7 Conclusion 141

Case Study – XIX 151

Chapter 10 153
The Charcot Foot in Diabetes
Dr Hashim Mohamed
10.1 Pathogenesis..... 153
10.2 Diagnosis 154
10.3 Medical treatment 156

Case Study – XX 161

Chapter 11 163
Surgical Management of Diabetic Foot Infections: A Stepwise Approach
Dr Huda Al Dhubaib
11.1 The stepwise approach 163
11.2 Diabetic foot infections management using a stepwise surgical approach 164
11.3 Conclusion 169

Case Study – XXI..... 171

Chapter 12 173
Diabetic Foot Radiographic Imaging
Jarrah Ali Al-Tubaikh
12.1 The radiographic densities 173
12.2 Diabetic angiopathy 174
12.3 Diabetic osteopathy..... 175
12.4 Diabetic infections 178
12.5 Osteomyelitis 180

Case Study – XXII..... 183

Chapter 13 184
Local Wound Care and Dressings for Diabetic Ulcers
Dr Abdurrahman Almurayshid and Afsaneh Alavi MD, MSc
Introduction..... 184
13.1 Cleansers..... 185
13.2 Debridement..... 185
13.3 Topical antibiotics and antiseptics 186
13.4 Dressings..... 187
13.5 Advanced therapies for DFU 190
13.6 DFU local wound care recommendations (4) 191
13.7 Conclusion 192

Case Study – XXIII 194

Appendix: Miscellaneous Cases 197
Composed of, or containing, a variety of cases undergoing studies and observation

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CHAPTER 1

EPIDEMIOLOGY OF THE DIABETIC FOOT

DR HASHIM MOHAMED

Diabetic foot ulcer represents one of the most common complications of diabetes. Around 15% of individuals with diabetes will develop a foot ulcer during their illness [1-4]. However, the prevalence of diabetic foot ulcers varies between different populations, ranging from 2% to 10% [3,5-7]. On average, individuals with diabetic foot ulcers have around a 59% longer stay at the hospital compared to diabetic patients without ulcers [8]. Furthermore, approximately 85% of lower-extremity amputations are preceded by a diabetic foot ulcer [11-14]. Despite the recent advances in the management of type II diabetes, it is still the most common reason for non-traumatic lower-extremity amputations across the globe [2,3].

On average, the lower-extremity amputation rate is 50% higher in males compared to females and 15 to 40 times higher among individuals with diabetes compared to those without the disease [8,10,12,33]. Additionally, ethnic minorities, including African Americans and Hispanic Americans, carry a 1.5- to 2-fold higher risk of diabetes-related amputation than their Caucasians counterparts [3,4,16-19]. This could be related to several reasons, including poor education, lack of medical insurance, lower socio-economic status, cultural factors, lifestyle, healthcare-seeking behaviour and an external locus of control.

Survival rates post lower limb amputation are generally low in individuals with diabetes [3,4,13]. The average five-year survival rates are about 40%, with cardiovascular complications being the leading cause of death [17]. Recent data has demonstrated a 50% incidence of contralateral lower limb amputation within 2–5 years [3,13].

In the US, the total annual health care costs for individuals with diabetes was estimated to be $132 billion in 2002. The average price of treating a foot ulcer depends on several factors, including the country the patient is residing in, the status of the health system, medical insurance cover, ability to pay for treatment and comorbid conditions. However, in the US, the

average cost of foot ulcer treatment ten years ago was around $4,595 per ulcer [19,20]. There are additional costs related to having a diabetic foot ulcer or a lower limb amputation, including work days lost due to illness, low self-esteem, depression and poor quality of life.

1.1 Risk of ulceration

Risk factors for foot ulceration include improper footwear, foot deformities, trauma, peripheral neuropathy, peripheral vascular disease, limited joint mobility, abnormal foot pressures, impaired vision, and a history of ulceration or amputation [9,21,22]. Recent data revealed that peripheral sensory neuropathy in the face of unperceived trauma tends to be the leading cause of diabetic foot ulceration [8,23,24]. Neuropathy is responsible for 45% to 60% of all diabetic foot ulcerations, whereas 45% of diabetic foot ulcers have a combined neuropathic and ischaemic component [8,25].

Other types of neuropathy may contribute to the development of foot ulceration, including motor neuropathy manifested in atrophy or wasting of intrinsic muscles, ultimately leading to foot deformities such as prominent plantar metatarsal heads, hammer toe and foot drop [9,10,26-28]. Furthermore, the poorly-distributed weight of the body as a result of intrinsic muscle deformities and a reduced ankle motion will confer a higher-than-normal focal plantar pressure, especially at the forefoot, ultimately leading to ulceration, recurrence and/or recalcitrance of existing diabetic foot ulcers [29-32].

The situation is further aggravated when autonomic neuropathy leads to malfunctioning of the sweat gland leading to reduced moisture of the skin, consequently leading to dry skin, cracking and fissure formation, thereby creating a portal of entry for bacteria [33,34]. In individuals with a prolonged course of diabetes, especially if it is uncontrolled, may ultimately result in sympathetic and parasympathetic dysfunction. These are manifested by arteriovenous shunting, where the foot becomes oedematous and warm. Furthermore, in response to injury, the microvascular thermoregulatory dysfunction impairs normal tissue perfusion in the foot and its ability to heal. These deregulations may subsequently lead to diabetic foot ulcer formation [34-38].

Many factors, including injury, improper footwear and foot deformities resulting from neuropathy including hard callous formation, abnormal gait, prior ulceration, surgical interventions and reduced peripheral perfusion may result in an increased risk of ulceration [8,39-45]. The course and prognosis of diabetic foot ulceration are prolonged and complicated by the

presence of peripheral arterial disease imparting an elevated risk of amputation[12,44,45]. Individuals with peripheral arterial disease and/or those who continue to smoke will have an impaired ability to resolve infection due to low levels of oxygen as well as the reduced ability to deliver antibiotics to the site of infection. Hence, early aggressive management of lower-extremity ischaemia is of utmost importance for salvaging lower limbs [14, 45-47].

1.2. Mechanisms of injury

Although many pathophysiologic pathways have been suggested that lead to foot ulceration [48-51], two common mechanisms can lead to foot ulceration in individuals with diabetes [52,53]. The first mechanism of skin injury is related to repetitive shear prolonged low-grade pressure over a bony prominence (i.e. a hammer toe or bunion deformity). Classically, this happens if a person wears tight or ill-fitting footwear, thereby causing skin breakdown or formation of the wound over the bony area involved. Loss of protective sensation, coupled with footwear trauma in the presence of foot deformity and callous formation, becomes the leading cause of foot ulceration in individuals with diabetes [8,12,41].

The other common etiology of ulcer formation occurs due to prolonged repetitive moderate stress [52]. This usually happens on the plantar aspect of the foot and is related to anteriorly displaced or atrophied fat pads, prominent metatarsal heads and structural deformity of the foot. Structural risk factors related to ulcer development in the foot include rigid deformities such as hallux rigidus, hallux valgus, hammer toes, Charcot arthropathy and a limited range of motion of the small joints of the foot [11,41,45,54-56]. Various clinical studies have linked the likelihood of ulcer development with the presence of high plantar pressures [10,44,54,57].

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CASE STUDY – I

A 61-year-old male patient with type II DM of 35 years duration, a chronic heavy smoker with coronary artery disease, hypertension, severe peripheral artery disease, chronic renal failure on haemodialysis, previous two episodes of myocardial infarction and left midfoot amputation presented with a non-healing right plantar foot ulceration with an exposed calcaneus. His investigations revealed Hb 6.7 gm/dl, HCT 6.6, MCV 19.4, creatinine 357 micromole/litre, urea 18.9 mmol/l. During his hospital stay he had a vascular assessment, which showed severe calcification of anterior and posterior tibial arteries.

He was treated previously in the hospital with intravenous antibiotics and insulin, and attended the podiatry clinic for dressing with advanced wound products, including a hydrogel and silver-based wound product before presenting at the primary care health centre.

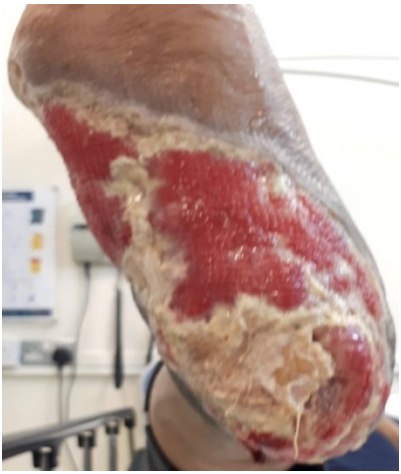

Figure 1-1: Exposed calcaneus and extensive necrotic tissue on presentation

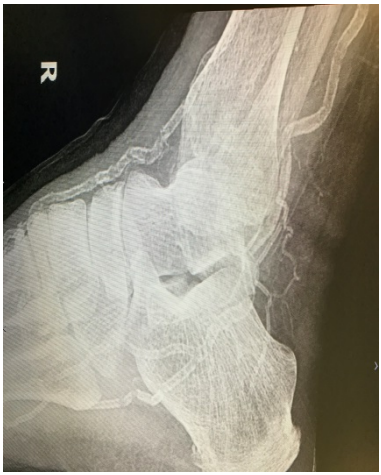

Figure 1-2: Showing complete calcification of anterior and posterior tibial arteries and its tributaries

The patient underwent extensive sharp debridement with a surgical blade (Figure 1-4); the wound was washed with normal saline via a 20 cc gauge syringe followed by the application of sterile raw honey, which was covered by a petroleum impregnated wound dressing. The wound was covered with cotton gauze and wrapped with a cotton bandage. The patient was instructed to strictly offload his foot using a pneumatic cast walker (Figure 1-3). An X-ray of the right foot revealed calcification of anterior and posterior tibial arteries (Figure 1-2). He was prescribed oral ciprofloxacin 500 mg bid for two weeks with daily dressing at the health centre. Eight weeks later, the plantar surface completely re-epithelized (Figure 1-5); however, the patient did not comply with the advice and removed the pneumatic cast walker and started to ambulate. This led to the haemorrhagic plantar surface with exposure of the calcaneal bone with subcutaneous bleeding and necrotic tissue formation (Figure 1-6).

The patient refused to go for angiography with the possibility of doing lower limb bypass and kept ambulating; this, in turn, led to the severe compromise of the blood flow to the toes resulting in the formation of dry gangrene of the second, third, fourth and fifth toes (Figure 1-7). The patient later stopped attending follow-up appointments.

Figure 1-3: Showing pneumatic cast walker

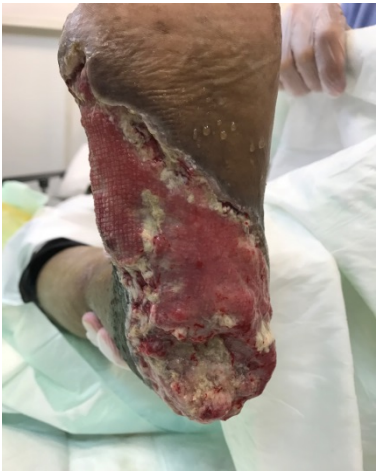

Figure 1-4: Plantar surface post debridement using surgical blade

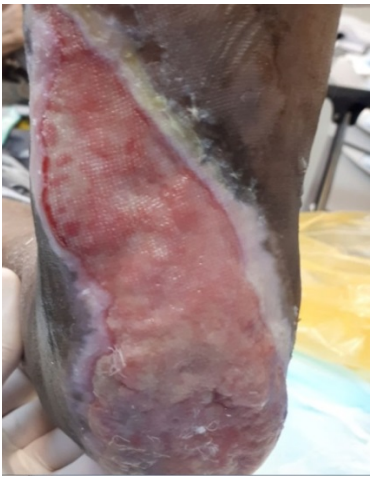

Figure 1-5: Complete epithelization of the plantar surface including the calcaneus eight weeks later

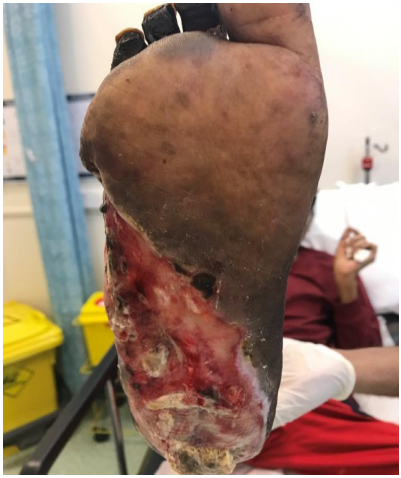

Figure 1-6: Trauma of plantar aspect leading to necrotic tissue and haemorrhagic spots formation and exposing of calcaneus with evident dry gangrene in the second, third and fourth toes

Figure 1-7: Dry gangrene of the second, third, fourth and fifth toes

Later upon inquiry, it was discovered that the patient was admitted by his family to the main hospital with an extensive lower limb infection and

was given intravenous meropenem antibiotic. He is waiting for below-knee amputation.

CASE STUDY – II

A 73-year-old female suffering from type II diabetes for the past 30 years, with hypertension, hypercholesterolemia, and chronic end-stage renal disease (stage IV), presented with a non-healing wound on the right big toe she had had for one month. She received treatment in the neighbouring health centre, but the wound failed to heal. History revealed that she had suffered this injury after placing her foot on a hot pot while cooking traditional food. The ulcer was dressed in the neighbouring health centre with Betadine alternating with antibacterial dusting powder for one month resulting in an ulcer of Wagner's stage III (Figure 1-8). On examination, her general condition was stable except for a BP value of 160/94 mmHg, HbA1c value of 9%, erythrocyte sedimentation rate (ESR) 50 mm/first hour, random blood glucose of 12 mmol/l. She had neuropathic feet (loss of protective sensation documented by absent pressure sensation as tested by 10 g monofilament) with a normal ABI.

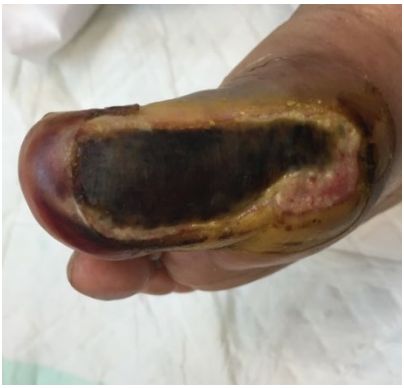

Figure 1-8: Thick black Escher formation with surrounding necrotic tissue, erythema and cellulitis

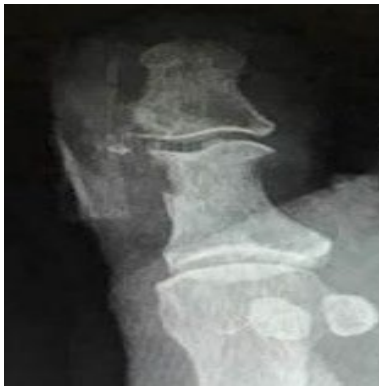

Figure 1-9: Showing punched out lytic lesion with cortical destruction of the shaft of the first proximal phalanx

After debriding the black eschar, the big toe revealed slough, necrotic tissue, and oozing pus with an exposed bone (Figure 1-8). The X-ray

revealed osteomyelitis of the first proximal phalanx shaft (Figure 1-9). She underwent daily dressing with natural honey and oral ciprofloxacin 500 mg twice daily for two weeks. The wound started to heal, but the area of necrotic bone (Figure 1-10) remained devoid of granulation tissue; hence curettage of osteomyelitis bone was performed using a sterile curette. Post curettage the wound was dressed with natural honey on a daily basis. Eight weeks later, the wound had completely healed and was covered by healthy skin (Figure 1-11). The patient was educated about her foot condition and how to examine it daily, including footwear, particularly therapeutic footwear.

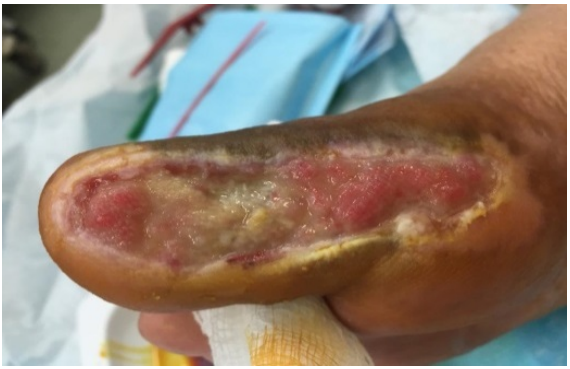

Figure 1-10: Showing healthy granulation tissue formation except in the proximal phalanx where necrotic tissue is apparent

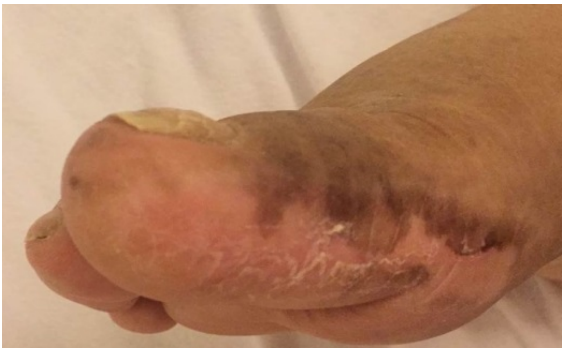

Figure 1-11: Complete healing of first big toe eight weeks later

CASE STUDY – III

A 35-year-old female suffering from type II DM for five years duration presented with right big hallux lesion showing hyper granulation at the base of the nail following previous trauma which was neglected by the patient (Figure 1-12).The patient underwent excision of the hyper granulation tissue after application of digital block using Xylocaine injection along with the removal of the devitalized nail (Figure 1-13).

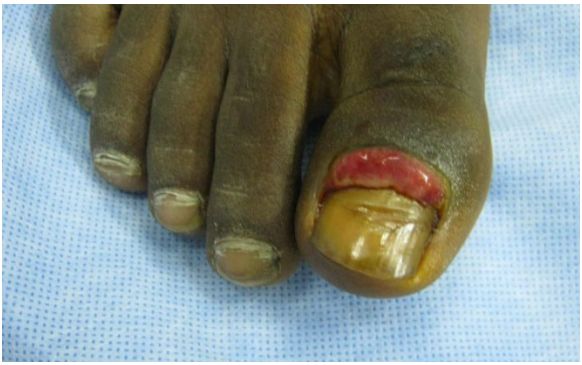

Figure 1-12: Right big hallux showing hyper granulation at the base of the nail

Figure 1-13: Post excision of the granulation tissue and nail

CHAPTER 2

DIABETIC FOOT INFECTION

DR HASHIM MOHAMED

2.1 Risk of infection

Individuals with diabetes have a higher risk of developing infections that are often more severe than in non-diabetics and carry a high risk of developing osteomyelitis[1]. Diabetic foot infections are usually polymicrobial in nature [2-8]. Prolonged impaired glycaemia results in dysfunction of the host leukocytes, thereby lowering their ability to fight bacterial pathogens, which is often complicated by the presence of peripheral vascular disease (ischaemia) because of reduced delivery of antibiotics to the site of infection.

As a result, the infection can spread rapidly and lead to significant and irreversible tissue damage [9]. Those individuals who do not have peripheral vascular diseases but suffer from peripheral sensory neuropathy will often experience a prolonged and problematic course of infection due to continued walking or delay in recognition [10,11]. Major predisposing factors leading to limb-threatening diabetic foot infections include prolonged hyperglycaemia, neuropathy, impaired immunologic responses and peripheral arterial diseases [12-14].

Foot infections are frequent among individuals with diabetes, especially following trauma; this, in turn, leads to increased risk of hospitalization and amputation. Diabetes confers a 30-fold higher lifetime risk of undergoing a lower-extremity amputation in individuals with diabetes compared with their counterparts [15,16]. Two-thirds of lower-extremity amputations are preceded by an infected diabetic foot ulcer [17,18], whereas infection-related lower-extremity amputation is surpassed only by the development of gangrene [19]. Statistically significant risk factors for developing a foot infection in individuals with diabetes include sustaining a foot wound, recurrent wounds that penetrated to the bone, peripheral vascular disease and wounds of long duration.

Additionally, neuropathy, previous history of amputation and peripheral vascular disease are each significantly and independently associated with infection, conferring 3.4-, 5.5- and 19.9-fold increased risk, respectively. Although studies in the developed world (USA) did not find any relationship between socio-economic factors, foot infections and ulceration, these findings cannot be mirrored in Third World countries where socio-economic factors are statistically linked to the development of foot ulceration [20].

2.2 Medical management of diabetic foot infections

2.2.1 Pathophysiology

Many predisposing factors related to the development of foot infections include immune system disturbances manifested by impaired phagocytosis, polymorphonuclear leukocyte migration, chemotaxis and intracellular killing [22].

Cellular immune responses, complement, and monocyte function is decreased as well [23,24].

2.2.2 Microbiological considerations

Before selecting suitable antimicrobial therapy, the clinician must be aware of the common etiologic agents responsible for diabetic foot infections along with any new antibiotic medications taken by the patient. Previous intake of antimicrobial agents can change the colonizing flora of skin ulcers [10,11]. It is worth noting that acute infections in previously untreated individuals often result from aerobic gram-positive cocci (often as monomicrobial infections). However, it is essential to be mindful that chronic ulcers often develop complex flora.

Determining the microbial aetiology of an infected wound will usually assist in subsequent management. The exact aetiologic agent(s) can be identified from specimens taken for culture and sensitivity. The sensitivity (true pathogens) can only be determined by obtaining deep tissue specimens aseptically by surgery. At a primary care level, an appropriate sample can be obtained by curettage or tissue scraping from the base of the ulcer with a scalpel after debriding the ulcer [10-13] unlike superficial swabs, which will only capture polymicrobial growing in the superficial flora.

The most common pathogen present in diabetic foot ulcers is *staph. aureus* [8]. Patients presenting at a primary care facility for daily dressing

may often be colonized with severe infections, including both aerobes and anaerobes [11,13]. Gram-negative Enterobacteriaceae usually colonize or are present in chronic or previously treated infections. In general, most patients are treated with wet dressings, including hydrogels or hydrotherapy. This approach confers a high chance of contracting *Pseudomonas* infections, especially in chronic wounds. Obligate anaerobic bacteria will usually colonize chronic wounds complicated by ischaemic necrosis or wounds penetrating deep tissues. Methicillin-resistant *S. aureus* is frequently isolated from hospitalized patients receiving antibiotic therapy [15]. However, they may present at primary care with a chronic wound in a patient attending a nursing home facility or even as an community-acquired infection in mobile patients.

2.3 Diagnosis and clinical presentation

Acute infection of a wound is usually diagnosed by the presence of systemic signs (e.g. chills, fever and leukocytosis), purulent secretions (pus), or two or more signs and/or symptoms of inflammation locally (warmth, pain, redness, induration and tenderness).

However, chronic wounds often have additional signs suggesting chronic infection, which include abnormal colouration, delayed healing, tissue friability, and on occasions, presence of foul odour. In patients with prolonged uncontrolled diabetes, peripheral neuropathy and/or ischaemia can either mask or mimic the signs and symptoms of inflammation and/or infection. Furthermore, chronic diabetic foot infections are often characterized by reduced signs of systemic toxicity, including low ESR level and reduced leukocytosis [19], even in those with limb-threatening conditions.

2.3.1 Clinical presentation

Evidence of peripheral vascular disease is present in almost two-thirds of patients suffering from a diabetic foot infection, with ~ 80% having lost their protective sensation [1]. Infections usually involve the forefoot, especially metatarsal heads, and the toes. Patients suffering from chronic diabetic foot ulcers do not often report pain, and more than half, including those with severe infections, do not have an elevated WBC count, C-reactive protein, and an elevated erythrocyte sedimentation rate or fever [19-21].

2.3.2 Assessing severity

The severity of the diabetic foot ulcer is assessed by the depth of the lesion, checking for ischaemia, and infection [22]. The diabetic foot ulcer must be thoroughly examined to look for necrotic material or foreign bodies and must be probed with a sterile metal probe or a cotton swab if a metal probe is not available. This is because deep-seated infections may often show a few superficial signs of infection.

Spread of infection to deep spaces must be suspected when there is inflammation distant from the skin wound, or when diabetic foot ulcers persist despite adequate antimicrobial therapy [23]. The primary care physician must be aware of what to keep and manage at the primary care level and what to refer urgently to the hospital when faced with a diabetic foot infection. Patients with a deep-seated infection must be admitted for possible surgical interventions at surgical podiatry clinics or hospital-based foot and ankle surgeons for incision and drainage, and metabolic control. The patient must be referred to the hospital if primary care has no offloading devices, if the patient is frail, suspected of having deep-seated infections and/or ischaemia, and is unlikely to comply with antibacterial therapy.

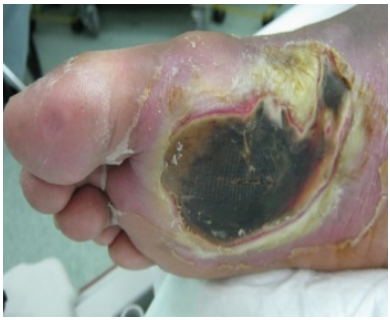

Figure 2-1: Deep infection of the forefoot and Escher formation on a neglected diabetic foot ulcer

2.3.3 Bone infection

Although diagnosing osteomyelitis is essential, it can also be challenging. Diagnostic, clinical and laboratory signs and symptoms are often unhelpful [36]. Osteomyelitis (contiguous spread of a deep soft tissue infection through the bone cortex to the marrow) may not be evident on plain X-ray during the first two weeks. Abnormalities commonly seen on X-ray are destructive bone