Diabetes & Metabolic Syndrome: Clinical Research & Reviews 10 (2016) 48–60

Contents lists available at ScienceDirect

Diabetes & Metabolic Syndrome: Clinical Research & Reviews

journal homepage: www.elsevier.com/locate/dsx

Review

The diabetic foot

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ARTICLE INFO

Keywords:
Diabetic foot
Pathophysiology
Management

ABSTRACT

Diabetic foot problems are responsible for nearly 50% of all diabetes-related hospital bed days. Approximately 10–15% of diabetic patients developed foot ulcers at some state in their life and 15% of all load in amputations are performed in patients with diabetes. There is a need to provide extensive education to both primary care physicians and the patients regarding the relationship between glucose control and complications encountered in the foot and ankle. The management of diabetic foot disease is focussed primarily on avoiding amputation of lower extremities and should be carried out through three main strategies; identification of the "at risk" foot, treatment of acutely diseased foot, and prevention of further problems. These are several obstacles in the management of DFI that include poor knowledge and awareness of diabetes and its complications, lack of appropriate podiatry services. These goals are possible only by the establishment of a dedicated team of podiatrist, endocrinologist, vascular surgeon and a pedorthist. The plastic surgeons, orthopaedic surgeons & diabetes teaching nurses/educator dedicated to foot care could be a part of the team. Identifying the patients with diabetes at risk for ulceration requires feet examination, including the vascular & neurological systems, skin conditions, and foot structure. Conservative management of foot problems has dramatically reduced the risk of amputation by simple procedures, such as appropriate foot wear, cleanliness, aggressive surgical debridement, regular wound dressing by simple wet-to-dry saline guage, and ulcer management.

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http://dx.doi.org/10.1016/j.dsx.2015.04.002
1871-4021/

1. Introduction

Diabetic foot problems account for more hospital admissions than any other long term complications of diabetes and are responsible for nearly 50% of all-diabetes-related hospital bed days. The diabetic foot is particularly at risk for complications because of its inability to tolerate stress. Diabetic foot ulcers (DFUs) are one of the most common and serious complications of diabetes and affects 15% of all diabetic patients and results in a high financial burden [1,2]. About 50% of all lower limb amputations are performed in people with diabetes. Diabetes associated lower extremity complications are emerging as noteworthy public health concern in both developing and developed countries.

The lifetime risk to a person with diabetes for developing a foot ulcer could be as high as 25% [3] and the primary factors in the development of these lesions are vascular insufficiency and peripheral neuropathy. Approximately 20% of diabetic patients with foot ulcer will primarily have inadequate arterial blood flow, ~50% will primarily have neuropathy, and approximately 80% will have both conditions [4,5]. Neuropathy, peripheral vascular disease, and reduced resistance to infections are recognized risk factors leading to the development of DFUs, which have all the characteristics of a chronic wound.

Conservative management of foot problems has dramatically reduced the risk of amputation by simple routine procedures such as good foot wear, chiropody, cleanliness, aggressive surgical radical debridement, off-loading, and ulcer management. Even that most dramatic of diabetic foot problems, Charcot's arthropathy, no longer means an inevitable progressions to amputation. Diabetic foot problems are not only an important complication, but they are also a preventable complication, bit they are also a preventable complication.

2. Pathophysiology and risk factor

Minor trauma both physical and mechanical, leading to cutaneous ulceration is the precipitating event for diabetic foot problems. The impact of diabetes complications mediated through micro-macrovascular disease is nowhere better exemplified than by feet. The presence of neuropathy, vascular insufficiency and an altered response to infection makes the patient with diabetes unique to foot problems. Peripheral neuropathy, high mechanical pressure resulting from structural deformities in the insensitive foot, inadequate arterial perfusion associated with peripheral artery disease, in addition to macro- and microvascular disease contribute to chronicity of diabetic foot ulcer. Although multiple factors contribute to the formation of foot ulcers in diabetes, oxidative/nitrosative stress, altered inflammation responses and impairment of the skin microcirculation have also emerged as critical intermediates.

Progressive atrophy of skin connective tissue is a critical intermediate in the formation of foot ulcer. Proliferation of skin fibroblast is reduced in diabetic patients [6,7]. Chronic, non-healing skin wounds of multiple etiologies demonstrate similar connective tissue abnormalities, including fibroblast numbers and proliferative capacity, reduced pro-collagen synthesis, and increased level of tissue degrading matrix metallo-proteinase (MMPs) [8]. Oxidative stress is also important in development of diabetic complications including neuropathy and foot ulceration. Hyperglycemic results in the increased production of vascular superdioxide (O^{2-}), thereby inactivating nitric oxide (NO) and contributing to vascular dysfunction. Additional, NO plays an important role in wound repair by promoting angiogenesis, migration and proliferation of fibroblasts, epithelial and endothelial cells and keratinocytes, Accumulation of AGEs has also been implicated in the pathogenesis of diabetic complications including impaired wound healing [9]. AGEs accumulate in diabetic wounds and after interaction interact with receptor of AGEs (RAGE), lead to expression of the pro-inflammatory molecules including endothelin-1, tumor necrosis factor alpha (TNF-α) and MMPs. TNF-α decreases the formation of tensile length of granulation tissue potentially by enhancing the generation of activated MMPs. Oxidative stress in diabetic vasculature may increase diacylglycerol and protein kinase C, thereby, contributing to vascular dysfunctions, skin small vessel disease and impaired skin perfusion.

All the risk factors (Table 1), especially uncontrolled glycemia, that are important to microvascular disease predispose to peripheral neuropathy and increase the risk of foot problems by altering the foot structure, physiology, and immune responses to trauma and infection. Other risk factors especially dyslipidemia, smoking and hypertension, that predispose to macrovascular disease also increase the risk of foot problems by disturbing the foot physiology, blood supply, and immune responses to trauma and infection.

The major underlying causes of diabetic foot disease are

- Peripheral neuropathy;
- Peripheral arterial disease;
- Infection secondary to trauma or ulceration; and
- Soft tissue and bony deformity

3. Peripheral neuropathy

A spectrum of peripheral neuropathy encounter in the lower extremity affects up to 60% with diabetes [10–12]. It is the most

Table 1
Patients at high risk for foot ulceration.

• Male sex	• Hypertension
• History of previous ulcer	• Poor glycemic control
• Smoking	• Sedantarism
• Ethnic origin as African American	• Skin changes
• Diabetes of more than 10 yr duration	Dyshydrosis
• Neuropathy: sensory, motor autonomic	Ingrown nails
	Mycotic toe nails
• Peripheral arterial disease	Evidence of poor hygiene
• Structural changes	Fissuring
Hammer toes	Chronic tinea pedis
Bunions	Chronic skin infections
Charcot's foot	• Abnormal gait
Pes cavus or planus	• Abnormal pattern of wear or shoes
Other pathological changes in shape	
Callus formation	
Bleeding into callus or under a nail	
Nephropathy, retinopathy	

common cause of leg pain in patients with diabetes. The average endocrine practice with a mixed age-range of patients would expect a neuropathy prevalence of 33%. Neuropathy is more prevalent with increasing age and duration of diabetes. Sensory neuropathy is usually the precipitant of painful symptoms. Of 100 patients with sensory neuropathy, up to 50 may be asymptomatic and only 1 would be expected to have significant symptoms that require treatment.

In diabetic patients, peripheral neuropathy encompasses both large and small fibers, with size describing related to the sensation of pain experiences in the diabetic patients and also disrupts temperature discrimination and automatic functions. Patient may present with burning, tingling, radiating pain beginning at the toes and progressing proximally on the foot and leg. Diminished sensibility to sensory stimuli including pain, temperature, touch, and vibration in the feet means that poorly fitting shoes or early signs of foot deformity or lesions may go unnoticed by the patient and uncorrected. Callus is prevalent in neuropathic ulcers and reduces the healthy potential of an ulcer, predisposing to infection.

Loss of neural supply to the intrinsic muscles of the foot leads to an imbalance between the flexor and exterior mechanisms, clawing to the toes, and increased prominence of metatarsals heads. A stoppage gait may develop. Loss of sweat gland function leads to dry skin predisposed to skin cracking and infection. The autonomic neuropathy results in a falsely warm foot secondary to altered blood flow. Loss of peripheral sympathetic vascular tone increases distal arterial flow and may lead to edema and osteopenia.

Although anti-convulsant medication (gabapentin, pregablin), vitamin supplementation, folic acid, thiamine have been used for the treatment of peripheral neuropathy, none have consistently shown an ability to restrict sensory loss. As peripheral neuropathy advances, the patient will become insensate with a noted loss of protective sensation distally in the extremities. This problem is irreversible, and can significantly elevate the risk for limb loss. Diabetic patients are unable to detect trauma to the foot and thus, does not respond by protecting or treating the area. Hence, even a smaller blister can progress to chronic ulcer because of delay in care.

4. Peripheral arterial disease

Diabetes induced peripheral arterial disease affects both small and large vessels in extremities. Reduced blood supply mimics and exacerbates the changes brought about by neuropathy. The large vessels that deliver arterial blood to the foot are the posterior tibial artery, anterior tibial artery and the peroneal artery. With advancing diabetic disease, one or all of the arteries may be compromised. The incidence of PAD in patients with diabetes is at-least four times that of non-diabetic individuals and increases with age and duration of diabetes. Hypertension and cigarette smoking, two classic risk factors for cardiovascular disease, also the predictors of PAD and the Epidemiological data suggest that an atherogenic mix of lipids and lipoproteins may, in particular, contribute to PAD and that hypertension, smoking, and perhaps hyperglycemia may interact to accelerate the process. Peripheral arterial disease has important distinctions in patients with diabetes. There is a prediction of PAD to primarily involve the tibia and peroneal arteries, but the foot arteries (dorsalis pedis, posterior tibial and plantar) are usually spared. Patients with diabetes frequently develop micro-arterial dysfunction–non-occlusive impairment involving arterioles and capillaries that begins early in life. There is increased micro-arterial pressure and flow leading to endothelial injury with sclerosis resulting in a limited capillary capacity with loss of auto-regulatory function including the abolition of a vasoconstrictor response. The leukocyte migration and oxygen diffusion is impaired. Increased arterio-venous shunting associated with autonomic neuropathy, an impaired hyperaemic response to heat and inflammation, the loss of a posterior vasoconstrictor response, increased capillary permeability leading to edema formation, and also the loss of other neurogenic regulatory responses that alter the diabetes patient's ability to response to injury. Besides, the intima and media of diabetic arteries frequently contain excessive calcium (Monckeberg's sclerosis), making them rigid and non-compressible. Results of non-invasive vascular laboratory test are thus, often incorrect or misleading. A hand held Doppler is the first means of detecting perfusion problems. If signals are any less than triphasic, assessment of segmental pressures (ankle brachial index (ABI) should be conducted, followed by vascular consultations.

An ABI of less than 0.50 in a chronic ulcer environment has highly likelihood of amputation [13]. An angiogram may reveal significant macrovascular disease requiring intervention, including angioplasty or by-pass, may also develop in diabetic patients with altered local blood flow and degranulation of vascular bone [14–16]. Hence, successful macrovascular intervention does not necessarily correlate with adequate perfusion of tissue.

Early signs of vascular compromise in the limbs should be identified before ulcer formation. The foot usually appears atrophic, evidence by lack of hair growth on the dorsum, cool temperature of the limb, and thin shining and atrophic skin. Patients usually complain pain in the limb as the ischemia progress, caused by the oxygen and nutrient deprivation to the tissues. This environment places the foot and ankle at the risk of ulceration. It is also important to determine the degree of perfusion loss to that area of foot or ankle, once an ulcer forms. Indirect blood flow to the area of ulceration may be sufficient to heal the ulcer but may take longer time to do so, placing at risk of infection or increasing depth. Rapid recognition of compromised blood flow and optimization of perfusion to the affected limb will support ulcer healing. In Charcot's arthropathy there is an arteriovenous shunt across the foot with distended dorsal veins (Wards sign).

5. Infections

The damage resulting from neuropathy, ischemia, or all three predisposes to foot infection. Infection may be bacterial (in association with ulcers) or fungal especially of toe nails. Infections are often undetected until limbs and sometimes life are threatened. The diabetic foot ulcers have an active and/or passive (biofilm) infections. Active infection includes the classic signs of ascending erythema, edema, purulence, increased drainage and malodour. Diabetic patients do not feel the progression of ulceration and signs and symptoms of infections (i.e. temperature, tachycardia, elevation of leukocyte count) are not manifested until late. The first sign of infection may be loss of blood glucose control or a flu like syndrome. These infections are usually polymicrobial and include aerobic gram positive cocci (*Staphylococcus aureus*), gram negative bacilli (*Escherichia coli*), *Klebseilla species* and *Proteus species*), and anaerobes (*Bacteroides* sp. and *Peptostreptococcus* sp.) [17,18]

Biofilms consist of bacterial colonies that form on the surface of chronic wounds and certainly play important role in ulcer healing. More than 60% of all infections are caused by biofilms. Biofilms are often the site of quorum sensing, influence the availability of key nutrients for biofilm formation, chemotaxis toward surface, mobility of bacteria, surface adhesion and presence of surfactants. The biofilm also provides a physical protection to bacteria because antimicrobial also are ineffective at penetrating the biofilms, decreasing the concentration acting on the bacterial cells within the biofilms and as a consequence there efficacy. Biofilms is present in 60% of chronic wounds but initially 6% of acute wounds [19,20]. These bacterial colonies are often multispecies and are

encased with glycocalyx matrix, making them resistant to oral, parental and topical antibiotics. Biofilms will reform within 10 h of debridement. A multimodal strategy is therefore, needed that includes sharp excision of the wound to disrupt the biofilm with immediate antimicrobial therapy to prevent its reformation.

6. Soft tissue and bone deformity

Most of the skin injuries on the feet of diabetic patients with neuropathy occur in the forefoot, with equal distribution on plantar and dorsal surface and those on plantar are frequent at site of high pressure. The two important forces experienced by the foot during ambulation are direct sagittal force and transverse force (side to side or front to back). The sagittal plane force is experienced on the plantar aspect of the foot during heel strike and forefoot push off. These shear forces are experienced between the foot and ground or foot and inside the shoe.

Repetitive pressure, shear from the walking and weight bearing or inappropriate foot wear, leads to increased plantar pressures, including callus formation and skin breakdown. This produces blister formation whereby the epidermis is dry, making is susceptible to tears and fissuring. Besides, there is atrophy of subcutaneous tissues, which negatively affects its ability to absorb shock during ambulation [21]. Furthermore, the tendons along with ligaments become stiff, lose their elasticity and its contracture, produces deformities such as hammer toe, which increases the likelihood of distal toe ulcer. Thus, the combination of compromised skin related structures and tendon contractures place the foot high risk for ulceration.

The foot deformity is frequently encountered in the diabetic foot Motor neuropathy, with imbalance between the flexor and extensor muscles in the foot, frequently result in the foot deformity with prominent metatarsals heads and clawing of the toes. The combinations of proprioceptive loss due to neuropathy and the prominence of metatarsals lead to increase in the pressure and load under diabetic foot [22].

Patients with later-stage peripheral neuropathy may not notice the change in foot structure and continue to ambulate, causing further subluxation/dislocation (mobility of joints). These deformities require conservative off loading or surgical reconstruction. Once the deformity has stabilized and no longer progresses, multidensity inserts in customized shoes or braces that offload and realign the foot and ankle are necessary.

A limb threatening example of joint deformity is Chacot neuropathy. Small fiber neuropathy may be prerequisite to the development of a Charcoid foot, i.e., autonomic pain fibers affected. Two important mechanistic processes have been involved in its pathogenesis: the neuropathy vascular theory and the neurotraumatic theory. The neurovascular theory invokes in increase in bone blood flow, perhaps including increased arterio-venus shunting due to reduction in tone of vasoconstrictor innervations, resulting in bone resorption ultimately leading to fracture and deformities [23]. Clinically, arterio venous shunting may be associated with prominent veins on the lower leg. In contrast, the neurotraumatic theory implicates repetitive trauma with fractures and progressive destruction of the architecture of the foot [24]. Precipitating causes for Charcot arthropathy that have been identified by a careful clinical history include trauma, infection, amputation and recent revascularization. The location of the radiological bony changes can give good clues as to the underlying diagnosis. For example, neuropathic osteoarthropathy is primarily an articular disease and is most common at the metatarsal heads and the development of a rocker bottom foot deformity. In Charcot arthropathy, in approximately 70% of subjects, the destructive processes primarily affects the mid foot and in 15% of the forefoot or rear foot is mainly affected. The forefoot is the commonest site for atrophic changes and may present as osteolysis of distal metatarsals. Hypertrophic disease affects mid foot distally and has been defined according to Eichen Holtz's classification system [25]. The "Ds" of Charcot arthropathy have been described:

- Dislocation;
- Debris;
- Disorganization; and
- Changes of bone density

Overt or subtle trauma may trigger a cascade of events that culminates in fractures, subluxations and dislocations of the bones in the foot or ankle. Charcot foot is characterized by edema, erythema and color. During the acute stage of Charcot neuroarthopathy there is gross instability of the affected joints. Immediate non-weight bearing is critical to prevent further collapse. If left untreated, these fractures, dislocations will proceed rapidly to ulceration and bone infection. After the Charcot neuro-arthropathy deformity has consolidated, bony prominences develop, causing area of high pressure resulting in ulcerations. Surgical reconstruction is not necessary if the deformity is shoe-able or braceable. All patients with diabetes and redness, swelling and increased foot temperature should be considered to be at risk of Charcot arthropathy and undergo an initial radiological assessment (Table 2). In Charcot arthropathy, there are no signs of systemic inflammation i.e., the white cell count and erythrocyte sedimentation rate (ESR) are not elevated. Reduced bone density of the lower limbs is also a feature of diabetic neuropathy and has been identified in patients with Charcot arthropathy. However, asymptomatic fractures are discovered in 22% of diabetic patients with neuropathy: often it is impossible to known whether patients presenting with traumatic foot fractures are at risk of developing Charcot arthropathy. All should be vigorously managed with off loading until the signs of acute inflammation have resolved.

Osteomyelitis is found associated with skin ulceration (90% results from spread from adjacent skin ulceration, cellulitis or abscess together with a sinus tract) and frequent at the metatarsal heads and distal phalanges. In practice, osteomyelitis most frequently occurs on the plantar aspects of the first and fifth metatarsal heads as well as distal phalanx of the great toe.

Radiographic findings in the acute Chacort foot (Fig. 1) may be very subtle and include mal-alignment and subluxation. The articular surface is often affected in subacute stages, becomes fragmented and may develop sub-chondral cysts and marginal erosions. With progression there is marked subluxation and osteoarticular destruction becomes severe. The X-ray findings of sclerosis, collapse and fragmentation of metatarsals heads have been reported. Fractures of the calcaneous are common as well as avulsion of the Achilles tendon. When X-ray evaluation has not revealed any abnormality, or when differentiation of Charcot changes from Osteomyelitis is required, additional imaging procedures are often performed. Magnetic resonance imaging (MRI) has emerged as the best method for the detection of Osteomyelitis with a specificity of up to 80%. This technique also allows determination of the extent of soft tissue infection, which can be very helpful in the planning of surgical approaches. In bone

Table 2
When should an X-ray be performed.

In a Charcot foot
• At presentation of a hot, red, swollen foot
• At 6 months to assess the efficacy of off-loading
• Prior to the transition from an air cast to bespoke shoes
• Subsequently if reactivation is suspected
In Osteomyelitis
• At presentation in a Wagner Stage 2 ulcer
• On reactivation of infection in previous osteomyelitis

Fig. 1. Charcot foot X-ray.

marrow infection, there is loss of the normal fatty marrow signal on T1 weighted images, a hyper-intense signal on T2-weighted or short tau inversion recovery (STIR) images and post gadolinium T1 weighted images are enhanced. Other procedures that may also be useful include a triple phase bone scan and white cell scan.

7. Prevention

Patient education is essential. Success in treatment and prevention of lower extremely diabetes related complications is only achieved with a motivated multidisciplinary approach where communication and collaborative efforts are at a high level with the goal of providing the right care to the right people at right time and in the right amount. Preventive strategies should include a multidisciplinary approach for both improved patient outcomes and short and long term cost effectiveness. A multidisciplinary approach is recommended for individuals with high risk feet and foot ulcers, especially those with a history of prior ulcer or amputations. One of the reasons is that diabetic patients have a complex co-morbid status associated with the presence of progressive peripheral neuropathy and sensitivity loss, poor vascular supply due to arterial disease involving small and large vessels and a compromised immune system. As a consequence, these patients require additional comprehensive testing and evaluation in order to establish the most appropriate plan of care. This will obviously, will be achieved by annual foot examinations, baseline testing, intervention and education which include:

- Evaluation of diabetic peripheral neuropathy and peripheral arterial disease.
- Regular evaluation of glycemic status and assessment of associated co-morbid conditions.
- Treatment and care of patients at high risk or with current wounds and ulcers, including podiatric care, wound and infective care, proper foot wear, off loading devices.
- Providing patient education.
- Following outcome measures longitudinally used to determine the effectiveness of the program and to detect areas that require further improvement.

Once the diagnosis of the diabetic peripheral neuropathy and/or peripheral arterial diseases is established, a multispecialty approach to foot care is appropriate and recommended at 3 to 6 months intervals. A comprehensive foot examination should comprise the following components: a general evaluation of the patient's medical history in the past; assessment of glycemic control; a comprehensive lower extremity foot examination. Peripheral neuropathy evaluation should comprise clinical examination (Pin prick sensation, light touch, vibration using a 128-Hz tuning fork at the apex of the hallus, ankle reflexes and pressure sensation using a 10-g monofilaments), quantitative sensory testing and electrophysiology if needed. Lower extremity vascular.

Status should be assessed by clinical examination of peripheral pulses, arterial Doppler studies and ankle and/or toe brachial pressure indices (bearing in mind that these can be falsely elevated by the presence of arterial calcification). The focus should be on identifying structural foot deformities including Charcot arthropathy using various imaging procedures and measurement of peak foot pressures if available. Low risk patients can be re screened annually. High risk patients should be scheduled for regular

podiatry evaluation at least once a quarter protocols for wound care consistent with standard wound care practices should be implemented including off loading with total contact cast, customized cast walkers, and healing sandals, wound debridement, glycemic control, infection control and lower extremity vascularization. The following instructions may help patient avoid foot ulceration and infection.

7.1. Foot hygiene

- Inspect the feet daily for blisters, cuts, scratches and reddened areas. One should always check between the toe.
- Wash feet daily with mild soap. Rinse and dry thoroughly, especially between the toes.
- Avoid extreme temperatures. Test water with hand or elbow before bathing.
- If your feet feel cold at night, wear socks. Do not apply a hot bottle, electric blanket or heating pad.
- Do not walk bare foot.
- Do not cut corns, use corn plasters, chemical agents for removal of corns and calluses.
- Wear clean intact socks appropriate for the shoes being worn.
- Trim nails with a slightly rounded edge.
- Avoid ''self bathroom surgery'' seek a qualified professional for treatment of all foot problems.
- For dry skin, use a very thin coat of a lubricating oil or cream except between the toes.

7.2. Shoes

- Wear well fitting shoes even if they are not stylish. Do not wear shoes without socks or stockings.
- Do not wear sandals or chappals with throngs between the toes.
- Change shoes during the day to relieve pressure areas.
- Try running or walking shoes for every day wear.
- Select dress shoes of soft leather and have them fitted carefully.
- Ask about therapeutic shoes if you have a foot deformity.
- Shake shoes out and inspect them before wear for areas that might cause blisters on rubbing.

7.3. Problems to report doctor

- Reports cuts, fissures, callus or breaks in the stem.
- Report changes in color or discoloration of the foot.
- Report ingrown nails.
- Report pain or loss of sensation.
- Report changes in architecture of the foot.

8. Assessment

All patients with diabetes should receive a through foot examination at least once in six months/annually; those with diabetic foot related complaints should be evaluated more frequently. Patients are often unaware of serious foot problems because of the masking effect of neuropathy and lack of education of foot care. Peripheral neuropathy, peripheral vascular disease, and bony deformity set the stage for ulceration. Loss of protective sensation is a major component of nearly all diabetic foot ulceration. A careful examination of the foot is indicated in all patients. Take a history inquiring about pain, discomfort, or numbness in the legs. Remember, diabetic neuropathy is a diagnosis of exclusion of other causes. A typical features that might suggest a non diabetic causes of neuropathy include rapid progression, foot drop, back and neck pain, marked asymmetry, weight loss, and family history. In long standing sensory motor neuropathy, small muscles wasting may be seen. Dry skin suggests co existing sympathetic dysfunction. Simple neuropathic assessment ideally would include.

- Pin prick sensation;
- Light touch;
- Vibration;
- Ankle reflexes; and
- Pressure sensation.

All these tests should be performed bilaterally, and the patients should be observed walking after the shoes have been impacted. Patients with severe sensory loss but no symptoms are often unsteady during normal gait because of the loss of proprioception. The diagnosis of diabetic neuropathy is normally a clinical one. Quantitative assessment of sensory modalities and electrophysiological studies may help to define the severity of the neuropathy but will not distinguish between neuropathy due to diabetes or other causes (malignant disease, toxins e.g. alcohol), infections e.g. HIV, uremia, metabolic disorders, inflammatory diseases and drugs (e.g. cytotoxic drugs, isoniazid).

8.1. Biothesiometer (the caliberated vibration threshold (vpt) meter)

It is a hand-held device with a rubber tractor that vibrates at 100 Hz. The device is connected to a basement displaying a linear scale of applied voltage, ranging from 0 to 100 V. The voltage is increased until the patient perceives a vibration. A mean of three readings measured in volts in generally used to determine the VPT for each foot. A VPT of more than 25 V had a sensitivity of 83%, a specificity of 63%, a positive likelihood ratio 0.22 (95% CI, 1.8–2.5), and a negative likelihood ratio of 0.27% (95% CI, 0.14–0.48) for predicting foot ulceration over four years. Semmes–Weinstein monofilament is an inexpensive, effective, reliable, valid, easy to use, and frequently used clinical indicator for detecting neuropathy. It is an effective or superior to more time-consuming tests of vibrations, temperature and peroneal nerve current perception in identifying patients at risk for ulceration. Semmes-Weinstein monofilament are caliberated nylon monofilaments, which generates a reproducible buckling stress, and identified, by manufactures assigned numbers that range from 1.65 to 6.65.

The higher the value of monofilament, the stiffer and more difficult to bend. Three SW monofilaments commonly used to screen patients at risk for peripheral neuropathy are 4.17, 5.07, 6.10. The forces required to bend these monofilaments are 1, 10, 75 g of forces respectively. The SW filaments are used to measure the patient's ability to sense appoint of pressure, as the repeated bouts of moderate amounts of unnoticed pressures are thought to be the primary mechanism for development of plantar ulcers in patients with diabetes and peripheral neuropathy. Although vibrating testing has demonstrated greater sensitivity, the SW monofilaments test is sensitive enough to identify patients with the highest risk of foot complications.

8.2. Ankle brachial index

It is the ratio of systolic blood pressure in the ankle to that in the brachial artery and helps in detection of peripheral vascular disease. An ABI of less than 0.90 indicates the presence of peripheral vascular disease, while higher than 1.1 may represent a falsely elevated pressure caused by medial artery calcifications. Risk of foot ulceration and transcutaneous oxygen tension is associated with an elevated risk of 0.80 (95% CI, 0.69–0.93; $P = 0.004$). A transcutaneous oxygen tension higher than 30 mm Hg correlates with a high likelihood of wound healing. Measuring

transcutaneous oxygen tension is not used frequently, as it requires expensive equipment and a trained technician.

Foot deformities have long been identified as risk factors for foot ulceration. The deformities like prominent metatarsal heads, clawed toes or hammer toes, rocker-bottom foot and prior amputation should be noted. The origin of the deformities may be congenital, idiopathic or traumatic. All of these contribute to an increase in plantar foot pressures and, with repetitive stress, the development of foot ulceration.

For evaluation and determination of this seventy of diabetic foot, various classification systems are in use now, that attempt to encompass different characteristics of an ulcer (namely this site, depth, the presence of neuropathy, infection and ischemia etc). The three main diabetic foot classification systems are commonly used in clinical diagnosis of diabetic foot. These are:

8.3. Meggit–Wagner classification

Most common and widely used Classification system is the Wagner Diabetic Foot classification System (Table 3). This system is basically anatomical with gradations of superficial ulcer, deep ulcer, abscess osteitis, gangrene of the fore foot, and gangrene of the entire foot. Only grade 3 addresses the problem of infection. In this system foot lesions are divided into different grades starting from grade 0 to grade 5. Grade 0 includes high risk foot but no active lesion and grade 5 includes gangrene of entire foot. But this system does not mention about ischemia or neuropathy and that is the drawback of this system (Fig. 2a–e).

8.4. Depth-ischemic classification

This classification is a modification of Wagner–Meggit system. The purpose of this classification system is to make the classification more accurate, rational, easier to distinguish between wound and vascularity of foot, to elucidate the difference among the grades 2 and 3, and to improve the correlation of treatment to the grade. Details of depth ischemic classification are presented in Table 4.

8.5. University of Texas classification system

Another popular system is the University of Texas San Antonio System which incorporates lesion depth and ischemia (Table 5). It is actually a modification of Wagner System. In this system each grade of Wagner System is further divided into stages according to the presence of infection or ischemia or combination of both. This system is somewhat superior in predicting the outcome in comparison to the Wagner System.

Ischemia and neuropathy can be difficult to differentiate; a thorough assessment is therefore, important (Table 6). The following should be inspected/examined:

- *Foot:* Wear; type; fit; pattern; foreign bodies;
- *Foot:* structure; distortion, e.g. Charcot's; pressure points; infection or ulcer between the toes; nails (for infection, length & whether in-growing); and
- *Skin:* whether dry; presence of fissures and calluses.

Table 3
Wagner–Meggitt classification system.

Grade	Lesion
0	No open lesion
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess, osteomyelitis, or joint sepsis
4	Local gangrene—fore foot or heel
5	Gangrene of entire foot

9. Wound evaluation

Wound evaluation (extent/size'' and ''depth/tissue loss items ''in the PEDIS systems [26] includes the evaluation of the size and depth of the wound, both of which should be determined after debridement. The duration and size of the ulcer relates directly to healing potential. Full thickness or deep wound of longer than 2 months duration is 79% less likely to heal. A chronic ulcer is defined as wound that does not decrease in size by 50% in one month. Measurements (length × width × depth) of the wound should be taken every one to 2 weeks to track changes in wound size. The wound should be fully explored for any pus pockets or communication to bone or hardware. The wound edges may have a rolled appearance indicating a chronic ulcer state. Senescent cells are found in the base and perimeter of the wound, preventing active wound healing and repair. Nonviable tissue (necrotic and fibrotic) may be evident in the wound bed, delaying wound healing. The wound with healing potential contains predominantly granulation tissue (bed of capillaries). The quality of the surrounding tissue should also be evaluated. Typically all diabetic foot ulcers have some levels of serous drainage unless there is an active infection, in which case purulence may be present.

9.1. Infection

The diagnosis of infection (''infections in the PEDIS System) is clinical, based on the presence of symptoms and signs of inflammation and must always be confirmed and classified according to the depth of involvements. In the PEDIS grading systems, these parameters are partially relevant to clinical managements and outcome: the involvements of the skin only (Grade 2), the involvement of deeper structures (grade 3), and the patients systemic inflammatory response (grade 4). Further, qualitative assessment should also be considered: cellulites (infections of the subdermis), necrotizing cellulites (infections-related tissues necrosis of the subdermis and dermis), necrotizing fasciitis (infection with involvements) of superficial fascia, presenting as sloughing of the skin and a violaceous color of the integument, without pus or abscess), wet gangrene (infection associated with blackish, necrotic tissues), and osteomyelitis. Different diagnostic procedures are indicated to categorize the patients into one of these groups. All patients should have a complete blood count with differential, ESR, CRP testing and, ideally, pro-calcitonin (PCT) testing. The most sensitive sign of infection is often recalcitrant hyperglycemia despite regular anti-hyperglycemic regimens. Recognising the insensitivity of classical signs and laboratory tests for the diagnosis of diabetic foot infections (DFI), Lipsky et al. [27] development a DFI wound scores that could also act a reliable and useful fool for predicting clinical outcome.

Another problems is determining the presence of osteomyelitis. The International Working Group on the Diabetic Foot has proposed consensus criteria [28] for diagnosing diabetic foot osteomyelitis (Table 7) that remains to be validated in a properly designed trial. Plain radiograph should be the initial imaging study, because in established cases, they often show characteristic pathological findings (cortical erosion, periosteal reaction, and mixed lucency and sclerosis). However, they are relatively insensitive, particularly in the first two weeks after infection. Furthermore, they do not permit the differential diagnosis of non-infections neuro-osteoarthropathy. Combining bone scans with other scintigraphic techniques, such as white blood cell scans

Fig. 2. (a) Image of Wagner grade 0. (b) Image of Wagner grade 1. (c) Image of Wagner grade 2. (d) Image of Wagner grade 3. (e) Image of Wagner grade 4. (f) Image of Wagner grade 5

(indium-111 leukocyte scans), improves specificity but these tests are expensive and time consuming and are, therefore, rarely used. MR imaging has a very high sensitivity for bone and soft-tissues infections and has gained wide acceptance in the management of DFI and can also be used for surgical planning. The criterion gold standard for diagnosis osteomyelitis is a characteristic histopathologically (acute or chronic inflammatory cells, or necrosis) associated with positive culture from a properly obtained bone specimen ideally obtained at the time of surgical debridement by fluoroscopic or computed tomography guided percutaneous biopsy.

In the absence of suspected osteomyelitis, bacteriological sampling, which must be done after mechanical debridement and cleansing of the wound with gauge socked in sterile physiological saline, is indicated if a DFU $\geq$ Grade 2 is clinically confirmed. The best sampling technique remains a matter of debate. While tissue biopsy and fluid aspirate are considered the gold standard for diagnosing wound infection, such invasive tests are infrequently performed. Superficial swabbing of the wound is discouraged, but swabbing the base of the ulcer is acceptable if it is the only possible option. Specimens must be placed in transport medium and be sent to the microbiology laboratory as quickly as possible.

Table 4
Depth ischemic classification system.

Grade lesion	
(0) No open lesions: may have deformity or cellulitis	
(A) Ischemic	(B) Infected
(1) Superficial ulcer	
(A) Ischemic	(B) Infected
(2) Deep ulcers to tendon, or joint capsule	
(A) Ischemic	(B) Infected
(3) Deep ulcers with abscess, osteomyelitis, or joint sepsis	
(A) Ischemic	(B) Infected
(4) Localized gangrene—forefoot or heel	
(A) Ischemic	(B) Infected
(5) Gangrene of entire foot	
(A) Ischemic	(B) Infected

Table 5
University of Texas classification system.

Stages	Grades			
	0	I	II	III
A	Pre-or post ulcerative lesions Completely epithelialized	Superficial wound not involving tendon capsule or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	With infection	With infection	With infection	With infection
C	With ischemia	With ischemia	With ischemia	With ischemia
D	With infection and ischemia	With infection and ischemia	With infection and ischemia	With infection and ischemia

9.2. Management

The severity of infected foot ulcer will determine the proper course of treatment. The treatment involves a multimodal approach that includes conservative and surgical interventions. Paramounts to ulcer healing in glucose control, which includes medical management, dietary/nutritional control and exercise. Regarding the direct care of the diabetic ulcer, there are four fundamental treatment principles. Optimization of perfusion, biofilm/infection control, debridement, and offloading. A major decision is whether the patient can be initially treated as an outpatient or need to be admitted to the hospital. Early superficial ulcers with minimal (<2 cm) cellulitis may be treated at home if there is no evidence of systemic toxicity and the patient is compliant, reliable, and has a vigilant support system. Hospitalization is indicated if there is no significant improvement within 24–48 h.

Inspection of wound to determine the extent of tissue destruction and sepsis is the first step. Carefully cleanse the area with an antiseptic solution, and with a sterile probe, forceps and scissors, unroof all encrusted areas. Inspect the wound to determine the extent of tissue destruction and possible bone and joint involvement. Little or no anesthesia is required because most of these have neuropathy. A multidisciplinary approach is effective protocol for treating superficial uncomplicated ulcers with limb threatening potential (Table 8).

9.3. Treatment of superficial uncomplicated ulcers and infections

The following guidelines apply in addition to the above guidelines shown in Table 8.

- Radical debridement of any necrotic, fibrotic, or hyperkeratotic tissue to a clean bleeding granular base.
- Apply plain gauze sponges wetted with saline solution to open the ulcer once or twice a day. New technology wound products may be used depending a wound environment.
- Treat fissures or cracks in the skin with an appropriate antibiotic ointment and a plain gauze outer dressing.
- Weight bearing is avoided until healing is ensured and resumed gradually. If weight bearing progresses too rapidly, acute Charcot's foot may result. Special devices used to help offload plantar ulcerations.

Table 6
Clinical features of neuropathic vs ischemic foot ulcer.

Neuropathic ulcer	Ischemic ulcer
Sensory defect	Not necessarily a sensory defect
Pulses present	Pulses absent
Subluxed metatarsals heads (cocked toes)	Foot structure retained
Ulceration of pressure points	Ulceration at points of ischemia, not pressure
Punched-out deep ulcer with surroundings callus	Superficial ulcer without callus

- Treat athletes foot with superficial bacterial super-infection with local antifungal cream or solution a plain gauze and an oral antibiotic.
- Foot should be modified to protect sensitive high risk areas. Once healed, these patients are regarded as high risk, and careful following, including modification of foot wear and orthotics, is recommended.

Numerous therapies to be effective for diabetic foot ulcer exist. There are numerous claims that dressings, ointments, solutions, cellular and/or tissue-based products, and other topical therapies heal diabetic foot wounds. However, there is a paucity of evidence to support that any of these any of these therapies promote wound healing but they should not be viewed as the sole agent for wound healing.

Debridement serves multiple functions that promote healing. It removes detritus, foreign material, and nonviable tissue, activates senescent cells by creating acute trauma to the wound bed and removes infection material associated with planktonic bacteria or biofilm. Debridement includes enzymatic debridement using collagenases, mechanical debridement using wet to dry dressing changes or whirlpool therapy, and biological debridement via maggot therapy. The preferred method is sharp debridement, which includes clinic based debridement and surgery based excisional debridement. Clinic based debridement involves the use of scalpels, scissors and curettes and is constrained by the inability to aggressively debride tissue and not a sterile environment. Surgery

Table 7
International Working Group on the Diabetic Foot Consensus criteria for diagnosing diabetic foot osteomyelitis.

Foot osteomyelitis	Grade O	Grade 1
Definite	Any 1 of the following • Positive bone culture and histology • Pus in bone at surgery • Detached bone in ulcer • Bony abscess on MRI	2 probable findings 4 possible findings 1 probable + 2possible findings
Probable	Any 1 of the following • Visible cancellous bone • MRI findings indicative of Osteomyelitis • Positive bone culture or histology	2 possible findings
Possible	Any 1 of the following • Cortex erosion on X-ray • MRI findings compatible with osteomyelitis • Positive probe-to-bone • Visible cortical bone • ESR > 70 mm/h • Chronic inflamed wound	
Unlikely	Any 1 of the following • Normal MRI • Normal bone scan • Acute ulcer without inflammation • Normal X-ray	

Table 8
Multidisciplinary approach is effective protocol for treating superficial uncomplicated ulcers with limb threating potential.

Active ulceration Peripheral neuropathy +/− PAD					
Non-limb threatening ulcer		Limb threatening Ulcer		Septic Ulcer	
Clinical evaluation	Patients evaluation	Clinical evaluation	Patients evaluation	Clinical evaluation	Patients evaluation
• Superficial • Minimal to no cellulitis • No bone or joint involvement • No significant ischemia • No toxicity	• Reliable • Will conform to treatment • Good support system • Evaluation of co-morbidities	• Check both feet and legs, compare • Ulcer probes deeply • Note location • May involve bone or joint • Cellulitis >2 cm • Lymphangitis, gas, crepitation • Ischemia +/− foot pulses, Doppler • Systemic toxicity, temperature, white blood cell, lymph nodes • Escher, callor, granulation tissues	• Unreliable • Poor support systems • Poor control of comorbidities	• Same as for limb threatening ulcer • Be sure to check for undermining deep tissue exposure, abscess, bone, and gas	• Same as for limb-threatening ulceration

Core	Adjuncts	Therapy	Therapy
• Offloading • Debridement • Dressing • Management of infections • Vascular evaluation if needed • Foot sparing surgery	• Control blood sugar • Treat comorbidities • Nutrition • Control edema • Physical and emotional therapy	• Admit to hospital – complete bed rest – protect wound and foot esp, heel • Employ core and adjunct • Control blood—stabilize comorbidities specially consult (cardiology, renal, infectious disease, etc) • Empirical broad spectrum antibiotics-adjust by culture and sensitivity • Labs and X-ray • Early surgical debridement-drainage and possible opens amputations • Meticulous wound care and dressing • Early vascular evaluation and Rx after control of infection • Foot sparing surgical vs amputation/ revisions • Close follow-up	• Immediate admission—complete bed rest-protect wound and foot, esp heel • Employ core and adjuncts • Intravenous broad spectrum antibiotic/fluid replacement and resuscitation • Labs and X-rays • Culture and sensitivity-blood culture • Control edema • Emergency aggressive surgical debridement—control all activity infection, including possible amputations • Stabilizing medically-blood glucose, cardiac (MI, CHF) renal, esp consult (cardiology, renal, ID. Etc) • Evaluate later for antihypertensive, beta blockers, calcium channel blocker, ACE inhibitors • Early vascular evaluation and Rx after control of infection • Close follow up

Adopted from Therapy for Diabetes Mellitus and Related Disorders, Fourth edition, by Harold E. Leboviz, page 517–518, ADA publications 2004.

based excisional aggressive removal of infection material and non viable tissue can be conducted. A combination of debriment strategies that includes all of the modalities described above should be used. Wounds should generally be debrided in the clinic every visit. Enzymatic and mechanical debridement can be performed in between clinic visits. Once the wound is sufficiently prepared in the clinic, the patient can be taken to the operating room for excisional debridement, after which the wound can be closed or covered with a split thickness skin draft. Large soft tissue defects requires local tissue flaps for closure and/or coverage of deep structures and bone.

Negative pressure wound therapy (NPWT) includes a family of devices consisting of specialized dressing, including adhesive drape and open cell foam, cut to fill the wound defects and capable of transmitting constant or intermittent pressure through the wound using a feedback control mechanism. Adding NPWT as part of a wound management strategy results in shortened hospital stays and a higher percentage of limb salvage, with consequent decreased overall medical costs.

As mentioned above, offloading is of clinical importance to successful ulcer healing, and is also perhaps the most difficult aspects of wound healing. Wheel chairs, crutches, and wheeled single-limb offloading platforms are some options that may used to completely offload a limb. Although the ideal situation is that the patient remains completely non-weight bearing. A common method of offloading advocated by Indian physicians is inserting a foam insole with a hole cut into the region of the plantar ulcer) into a one size larger shoe. However, such as insole is often ill fitting and can cause an "edge effect" or even new ulcers. An ideal off-loading device must be (1) patient compliant, (2) easy to apply (3)cost effective, (4) effective in wound healing, (5) comfortable for ambulation, (6) accommodated at all levels of health care systems, including rural setting. Although it may be difficult to achieve all these objectives through an offloading device, several techniques including total contact cast (TCC), walkers, air cast shows, crutch assisted walking, felted foam half-shows have been in use for the purpose. There is a lack of evidence-based research in India on the use of most of these off-loading devices comparing wound healing outcomes using TCC and conventional wound dressing except one study reported by Ganguly et al. [29] that showed that TCC was effective in healing 92% of foot ulcers as compared with 75% of wound dressings. Surgical interventions may be the most effective way to offload an ulcer. Tendon lengthening/re balancing, exostectomics, and bone/joint reconstruction can reduce the deforming forces that create a diabetic foot ulcer and contribute to its chronicity.

9.4. Treatment of limb threatening infections

The diabetic foot ulcer can have an active and/or passive (biofilm) infection. Active infection includes the classic signs of ascending erythema, edema, purulence, increased drainage, and

mal-odour. However, the diabetic patient is not able to mount a robust immune response. They often present without these signs, particularly when end stage renal disease is superimposed. Absence of leukocytosis may not reflect an active infection, although elevated blood glucose levels may. The degree of soft tissue infection and depth of infection will dictate the course of treatment. Surgical intervention, especially in a patient with systemic toxicity, should not be delayed even if the patient is not yet been stabilized medically and blood glucose is not controlled. Blood glucose control often requires the use of insulin and careful in patient monitoring.

Proper parental antibiotic therapy and wound care are essential to limb salvage. Whenever possible, deep infected tissue or bone should be cultured. Initial use of intravenous blood spectrum antibiotics is justified by the polymicrobial nature of these infections. Absorption of oral antibiotics may be inhibited by associated gastro-enteropathy, in hyperglycemic, seriously in patients. Soft tissue infections of the diabetic foot are often polymicrobial with gram positive species as well as gram negative bacteria, whereas bone infections are typically monomicrobial, this includes staphylococcal and streptococcal species as well as Pseudomonas and *E coli*.

9.5. Choice of an initial antibiotic or combination depends on

- Local bacterial resistance pattern.
- Proper antibiotic history.
- Gram stains of deep exudate.
- Appearance of wound and pus.
- Associated co-morbid conditions like renal, hepatic, cardiac impairment.

Changing antibiotics and duration of therapy depends on bacterial sensitivities and the response of the wound to surgical debridement. Biofilm consists of bacterial colonies that form on the surface of chronic wounds, and certainly plays a detrimental role in ulcer healing. Biofilm is present 60% of chronic wounds but in only 6% of acute wounds. These bacterial colonies are often multispecies and differ from planktonic bacteria. They have low metabolic activity and are encased with a glycocalyx matrix, making them resistant to oral, parenteral, and topical antibiotics. Biofilms will reform within 10 h of debridement.

To determine the extent of tissue destruction or bone or joint involvement, or plain X-rays are helpful initially but are not definitive. Scan and magnetic resonance may be useful in the differential diagnosis of osteomyelitis versus acute Charcot's disease. Surgical management of potentially serious foot ulcers in diabetes patient requires debridement that is extensive enough to ensure there is no undrained pus or necrotic tissue. Diabetes patient do not tolerate undrained infection, but they heal well if the infection is completely resolved and circulation is adequate. Dressings are begin with initial surgical management. It should consist of diluted isotonic antiseptic solution or saline applied to plain gauze and packed into the wound once or twice a day.

Assessment of the vascular status of the involved extremity is needed once sepsis is controlled. Non-invasive laboratory testing plays only a complementary role because of the peculiarities of diabetic peripheral arterial disease. In the case of critical ischemia, once the infection has been controlled, revascularization must be immediately considered. The criteria for revisualization should the patient's status, performance, the potential for cicatrisation, the site of the lesion, and the quality of the arterial distal runoff. The current revascularization options include conventional open surgery and endovascular intervention, which are not mutually exclusive and are often combined. Open surgical treatment includes endarterectomy for local lesions and peripheral bypass for long occlusions. Long term results are good, with 5 years secondary patency rates >70% and limbs salvage rates of 75 to 85% [30]. Endovascular options include angioplasty, with or without stenting, and atherectomy. However, restenosis rate is relatively high, especially in below—the-knee procedures for which it is high as 50% over a five year of period [31]. The result of the recent studies indicates that endovascular therapy might take a prominent place in the treatment of PAD, especially in patients with significant comorbidities, and thus applies even more to patients with DFI. Antiplatelet therapy should begin preoperatively and continues often an endovascular and surgical procedures.

10. Charcot's arthropathy

The term Charcot foot refers to bone and joint destruction that occurs in the neuropathic foot (Fig. 3). It is important to have a high index of suspicion for Charcot osteoarthropathy. Classically Charcot joint disease presents an unexplained swelling and erythema of the foot. Although there is often a history of trauma prior to the development of the Charcot's joint, the trauma can be so insignificant that patients are unable to recall a specific injury, It is often believed that profound sensory neuropathy makes this a painless process. In fact, patients may complain of mild to moderate discomfort. The pain, however is not in the proportion to the degree of bone and joint destruction seen on radiographs [32]. Charcot joint disease is one of the most mis-diagnosed entities involving the diabetic foot. Common misdiagnoses includes osteomyelitis, tendinitis, gout or acute aprain. There are two main classifications of Charcot new roarthropathy

Fig. 3. Charcot foot.

(Eichenholz and Brodsky), which describes disease progression and distributions respectively. The Echienholz classification describes the evolution of the condition overtime:

- Stage 1: destruction;
- Stage 2: Coalescene; and
- Stage 3: Consolidation.

Stage 0 has come into the use to classify the swollen, hot, usually rather painful foot in which plain foot radiograph is normally but MRI shows bone edema and stress fractures.

Charcot's osteoarthropathy can be divided into two phases:

- Acute Active Phase; and
- Chronic Stable Phase.

10.1. Acute active phase

The acute active phase is characterized by edema, localized warmth, erythema, and joint crepitus with range of motion examination. The foot is at least 2° hotter than the centre lateral foot. As the Charcot joint progresses to the next phase of coalescence, then temperature begins to equilibrate and joint crepitus diminished. Later on the active clinical phase, the signs are swelling, warmth and deformities, including the rocker-bottom deformity and medical convexity of the foot as well as hind deformition—X-ray reveals fragmentation, fractures, new bone formation, subluxation, and dislocation. The reconstruction or remodeling phase occurs over a period of months and years. This can eventually lead to a stable foot devoid of significant motion. Deformity in a Charcot foot can predispose to ulceration, which may become infected and leads to osteomyelitis. This may be difficult to distinguish from neuropathic bone and joint changes, as an X-ray, bone scan or MRI, appearances may similar. However, if the ulcer can be probed to bone, osteomyelitis is likely diagnosed.

Early diagnosis of Charcot foot is essential. Patients should have initially an X-ray examination which may be normal. It is possible to proceed to two investigations. A Technetium methylene diphonate bone scan will detect evidence of bone damage and also locate the site of damage. If the result of bone scan is possible, on MRI examination will describle in more details the nature of bone damage.

Treatment of the acute Charcot foot is directed at eliminating weight bearing forces that may lead to further destruction and deformity. This is best achieved by the use of crutches, a walker, or in the event of bilateral involvement, a wheelchair. A walking cast in the acute Charcot is not appropriate treatment. Non-compliance with non-weight bearing in the early stage of this disease will cause further fragmentation of bone, resulting eventually in greater deformity. Bisphonates may be helpful in the treatment of the Charcot foot [33] but are yet not fully established. An RCT of a single 90 mg pamidronate infusion has shown significant reduction of the markers of bone turnover and skin temperature in treated compared with control subjects, although the fall in skin temperature was similar to both groups. There was a similar findings in a recent study with alendronate [34]. Calcitonin has also been used in the acute stage and there was a more rapid transition to the stable chronic phase in the treated group compared with controls [35].

10.2. Chronic stable phase

The foot is no longer warm and red. There may still be edema but the difference in the skin temperature between the feet is less than 2 °C. X-ray shows fractures healing, sclerosis, and bone remodeling. The patients can now progress from a total contact to an orthotic walker, fitted with cradled molded insoles. However, to a rapid mobilization can be disastrous, resulting in further bone destruction. Extremely careful rehabilitation should be the rule. Finally, the patient may progress to foot wear with molded insole. No weight bearing is allowed as long as crepitus across joints and elevated skin temperature persist. Protected weight bearing can be instituted when the clinical examination and serial radiograph shows gradual resolution of the inflammatory process and healing of the involved joint. When appropriate, weight beating is instituted in a gradual manner and with protection. Weight-bearing is instituted with a protective brace and with 15 to 20 lb of pressure. This can be increased in 10 lb increments per week as long as there are no sign of reactivation. Should reactivation occur, patients should be returned to non-weight bearing until resolution of these symptoms. As weight-bearing progresses, the patient is eventually allowed to ambulate short distances without assistive devices.

The rocker bottom Charcot foot with planter bony prominence is a site of very high pressure, regular reduction of callus can prevent ulceration. If ulceration dioes occur, an exostectiomy may be needed. The most serious complications of a Charcot foot is instability of hind foot and ankle joint. This can lead to a flail ankle on which it is impossible to walk. Reconstructive surgery and orthodesis with a long term ankle foot orthosis, have resulted in better outcome and limb salvage.

11. Conclusions

Diabetes mellitus is global epidemic, and diabetic foot ulcer is one of the most serious and costly complications leading to severe economic and personal loss in the future. There is a need to provide extensive education to both primary care physicians and the patients regarding the relationship between glucose control and complications encountered in the foot and ankle. Peripheral neuropathy and peripheral vascular disease create an environment that will lead to ulceration and possible amputation. These are several obstacles in the management of DFI that include poor knowledge and awareness of diabetes and its complications, lack of appropriate podiatry services. This situation requires a concentrated effect involving a team of healthy care professional dedicated to the care of diabetes, especially related to foot complications. A proper knowledge of the diabetic foot in terms of its biomechanics etiology and treatment of the causative risk factors for DFI involving both clinical and surgical interventions and essential offloading devices to reduce wound pressure is requires among the health care providers to efficiently managing DFI in India. A multidisciplinary team approach is vital to the prevention and treatment of the diabetic foot. Besides this, imparting foot care education both to the physicians to update themselves and to the patients is also necessary to achieve successful outcome.

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