Diabetic Foot Ulcers

Goals & Objectives

Course Description

Diabetic Foot Ulcers is a text-based online continuing education program for physical therapists and physical therapist assistants. The course presents contemporary information about diabetic foot ulcers including sections on epidemiology, etiology, symptomology, assessment, classification, management, and prevention.

Course Rationale

The purpose of this course is to present contemporary information about diabetic foot ulcers to physical therapists and physical therapist assistants. Physical therapists and physical therapist assistants will find this information pertinent and useful when developing and implementing rehabilitation programs that address the challenges and needs specific to individuals with diabetic foot ulcers.

Course Goals & Objectives

At the end of this course, the participants will be able to:

1. Identify the etiological mechanisms leading to diabetic foot ulcers
2. Recognize risk factors associated with diabetic foot ulcers.
3. List symptomology associated with diabetic foot ulcers
4. Classify diabetic foot ulcers using standardized methodologies
5. Identify the components of a comprehensive diabetic foot assessment
6. Recognize clinical signs and symptoms of wound infection
7. Identify and define the primary processes of foot ulcer management
8. Recognize and define alternative therapies
9. Identify and define the different types of foot amputations
10. Recognize strategies for preventing diabetic foot ulcers
11. Identify footwear considerations for individuals at risk for ulcers

Course Provider – Innovative Educational Services

Course Instructor - Michael Niss, DPT

Target Audience – Physical therapists and physical therapist assistants

Course Educational Level – Introductory; intermediate/advanced

Course Prerequisites – None

Method of Instruction/Availability – Online text-based course available continuously.

Criteria for Issuance of CE Credits - A score of 70% or greater on the course post-test

Continuing Education Credits – 3 hours

Determination of Credits – Mergener Formula: .9 x [-22.3+(0.00209 x 71,492) + (2.78 x 10) + (15.5 x 3)] = 180 minutes = 3.0 hours/ 3 CE units

Fees - $29.95

Conflict of Interest – No conflict of interest exists for the presenter or provider of this course.

Refund Policy - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

Diabetic Foot Ulcers

Course Outline

Introduction

Diabetes mellitus (DM) is a serious and complex disease affecting almost all the vital organs in the body. It is known to have many complications and one of the most distressing is Diabetic Foot Ulcer (DFU). DFU is prone to infections, chronicity and recurrence. A benign looking ulcer in a patient with diabetes often ends up in amputation. This can lead to severe morbidity and mortality. The successful DFU management strategies involve intensive prevention, early assessment and aggressive treatment by a multi-disciplinary team of experts. [1]

Epidemiology

It is estimated that 26 million Americans have diabetes[2]; and that approximately 15% of these individuals will develop a diabetic foot ulceration during their lifetime. The recurrence rate of foot ulcerations is approximately 28% at 12 months and nearly 100% at 40 months[3]. Unfortunately, chronic diabetic foot ulcers lead to more than 80% of non-traumatic amputations[2]. The good news is that, with proper care, it is estimated that at least 40% of diabetes-related amputations can be prevented.[3]

Etiology

Peripheral Neuropathy

Peripheral neuropathy is the most important causal pathway leading to foot ulceration and often leads to sensory deficit with the loss of protective pain sensation.[4] Peripheral neuropathy is damage to nerves of the peripheral nervous system, which may be caused either by diseases of or trauma to the nerve or the side-effects of systemic illness. The four cardinal patterns of peripheral neuropathy are polyneuropathy, mononeuropathy, mononeuritis multiplex and autonomic neuropathy. The most common form is (symmetrical) peripheral polyneuropathy, which mainly affects the feet and legs. The form of neuropathy may be further broken down by cause, or the size of predominant fiber involvement, i.e., large fiber or small fiber peripheral neuropathy. Frequently the cause of a neuropathy cannot be identified and it is designated as being idiopathic.

Symptoms of neuropathy depend on the type of nerves affected (sensory, motor, or autonomic) and where the nerves are located in the body. One or more types of nerves may be affected. Common symptoms associated with damage to the motor nerve are muscle weakness, cramps, and spasms. Loss of balance and coordination may also occur. Damage to the sensory nerve can produce tingling, numbness, and a burning pain. Pain associated with this nerve is described in various ways such as the following: burning, freezing, or electric- like, extreme sensitivity to touch. The autonomic nerve damage causes problems with involuntary functions leading to symptoms such as

abnormal blood pressure and heart rate, reduced ability to perspire, constipation, bladder dysfunction (e.g., incontinence), and sexual dysfunction.[3]

Sensory Neuropathy

Type-A sensory fibers are responsible for light touch, vibration, pressure, proprioception, and motor innervations to the intrinsic muscles of the foot. Type-C sensory fibers detect painful stimuli, noxious stimuli, and temperature. When these fibers are affected, protective sensation is lost which manifests as a distal, symmetric loss of sensation described as a “glove and stocking” distribution and proves to be the primary factor predisposing patients to ulcers and infection. Patients are unable to detect increased loads, repeated trauma, or pain from shearing forces. Injuries such as fractures, ulceration, and foot deformities therefore go unrecognized. Repeat stress to high-pressure areas or bone prominences, which would be interpreted as pain in the nonneuropathic patient, also goes unrecognized. Sensory dysfunction results in increased shearing forces and repeated trauma to the foot.[2]

Motor Neuropathy

Motor neuropathy is associated with demyelinization and motor endplate damage. It typically presents structural alterations of the dynamic anatomy of the foot and joints, causing weakness and wasting of small intrinsic muscles. This causes a loss of balance in the gait because of damage to the muscles. The atrophy of the interosseous and small intrinsic muscles of the foot acts to stabilize and hold the phalanges of the toes straight, as the long flexor and extensor tendons act through the insertions into the distal phalanges of the toes up into dorsiflexion, similar to a foot pressing the accelerator of the car. Alterations in the morphology of the structure of the foot, toes, forefoot and limited joint mobility impaired the ability of the foot to absorb and redistribute the forces relayed to impact the ground while walking. Effects on the foot include the reduction of motion and changes to the angle of the subtalar and first metatarsophalangeal joints (MTPJ). Vital musculoskeletal structures, such as equinus deformity through the shortening of the Achilles tendon and the collapse of the plantar fascia. In diabetic patients, the flexor tendons and extensor tips tend to be straight and rigid. If the intrinsic muscles are unable to do this, the toes shrink back to form what is called hammer toes and favor the thrust of one toe over another or a toe on the metatarsal head with the weight forced to the anterior surface with high force. On the other side, the contraction that the hammer toes cause on the plantar fat pad and the metatarsal heads reduces soft tissue plantar MTPJ, making them more susceptible to fracture of the skin, including the bone next to the formation of traumatic ulcers, owing to inappropriate weight loads. The mechanism is related to the high pressure exerted on the foot that occurs during the gait, in turn caused by motor neuropathy, which itself in turn causes structural changes in the anatomy and sliding of the fat pads of the foot. In addition to the shortening and thickening of the joints, the decreased capacity of distribution of plantar pressure in DM patients contributes to the development of high foot pressure and ensuing ulcerations. Excessive pressure and structural deformities in individuals with neuropathy is a prerequisite for the development of wounds.

Consequently, structural changes and offload pressure favors the formation of calluses

on various prominent parts of the foot, including the plantar region, the heel, the big toe, etc. The structure of the foot is a major determinant of plantar pressure. Although some structural factors are independent of the DM, others are predisposed to high pressure and appear to be a consequence of the disease.

Clawing toes: hyperextension of MTT phalange joints, usually accompanied by cavus foot and calluses on the dorsal surface of the fingers and the plantar surface of the metatarsal head or the tip of fingers.

Cavus Foot: under normal conditions the foot is shaped convexly due to the longitudinal medial arch that is extended from the head of the first MTT and the calcaneus; if this arch is abnormally high it produces an abnormal distribution of weight loads, favoring the formation of calluses in the forefoot and rearfoot.

Equinus Deformation: shortening of Achilles tendon (three muscles: lateral, internal gastrocnemius and soleus), falling of plantar fascia and facilitating abduct or adduct in the forefoot, beside the lost at the long flexor and extensor tendons that produce dorsiflexion.

1st toe rigid: it is due to hardening of the first MTT phalange joint with loss of dorsiflexion, resulting in excessive weight forces on the plantar surface and callus formation.

Joint stiffness: The limitation of joint movement is produced by the glycosylation of collagen and thickening of periarticular structures (tendons, ligaments, joint capsule, etc.) which favors deformities and plantar pressures, upsetting the biomechanics of the foot during walking by limiting plantar flexion and promoting equinus foot.

Deformity of the nail: Thickening or deformity of the nail atrophies the nail plate with convex deformity, causing pressure on the ridge tissues and, in turn, ingrown nail. The nail flange forms a callus in response to pressure and inflammation. As a result, the tissue of the trauma may become ulcerated and infected and penetrate the nail flange.[5]

Autonomic Neuropathy

Autonomic neuropathy is common in longstanding DM. In the lower extremities, autonomic neuropathy may result in arteriovenous shunting, resulting in the dilation of small arteries and producing distension of the foot veins, not alleviated through elevation of the foot. Neuropathic feet have a tendency to swell and to feel warm as a result of arteriovenous shunting. Autonomic neuropathy results in decreased autonomic nerve roots that innervate the sweat skin glands with appendage tissue, causing dryness of the skin and decreased elasticity, especially from the middle third of the leg down, where there is also discoloration of the skin. Dry, stiff skin produce cracks more easily forming splits, fissures or fractures of the skin and callusing around the foot injury, more frequently in the heel rim, plantar medial and first MTP -especially during the dryer months. These fractures or skin fissures can become infected, resulting in local cellulitis and then on to small longitudinal ulcerations.[5]

Charcot Arthropathy

Diabetic patients are especially prone to development of a neuroosteoarthropathy also known as Charcot foot (CN). Charcot neuroarthropathy is a chronic painless progressive degenerative arthropathy resulting from the disturbance in sensory innervations of the affected joint. The impairment of the autonomic nervous system due to DM causes an increase in local blood supply and the resting blood flow is much higher than in the normal patient. The sudden increase in blood flow causes calcium to dissolve, leading to osteoclastic activity of the bone and thus damaging the bone. Another theory is that the repetitive minor trauma to the insensate joints leads to fracture and disintegration. The production of pro-inflammatory cytokines leads to uncontrolled osteolysis in CN. The hallmark deformity associated with this condition is midfoot collapse, also known as “rocker-bottom” foot; which is prone to tissue breakdown and ulceration. There might be hallux valgus deformity and loose bodies in the joint cavity. The deformities associated with CN also predispose for recurrent ulcerations.

When the nerve gets injured, the patient is at a higher risk of getting a minor injury without noticing it until it becomes an ulcer. Clinically the patient may have signs of vascular insufficiency such as claudication, night pain or rest pain, absent peripheral pulses, thinning of skin, and loss of limb hair.[1]

Peripheral Vascular Disease

Coronary artery, cerebrovascular, and peripheral vascular disease (PVD) are the predominant manifestations of macrovascular disease in diabetes.[6] Compared to general population, diabetics are affected by atherosclerosis at a younger age, the atherosclerosis tends to progress at a much faster rate and results in higher rates of amputation. Its hallmark is the involvement of the tibioperoneal vessels with relative sparing of the pedal vessels. Characteristically, diabetic occlusive lesions spare the arteries above the knee but involve the infrapopliteal arteries with calcific single or multiple level disease. In more than 90% of patients, one or more of the large vessels at the ankle and in the foot are spared. In most cases, the peroneal artery in the calf remains patent and is the last of the three crural arteries to occlude prior to which it continues to provide pedal circulation via its terminal branches. Consequently, bypass to a single tibial or peroneal artery or a pedal bypass has the potential to provide good blood flow to the foot. Occlusive lesions affecting the foot and precluding revascularization are not common in diabetic patients.[2]

Peripheral arterial disease is a contributing factor to the development of foot ulcers in many cases. It commonly affects the tibial and peroneal arteries of the lower leg. Endothelial cell dysfunction and smooth cell abnormalities develop in peripheral arteries as a consequence of the persistent hyperglycemic state. There is a resultant decrease in endothelium-derived vasodilators leading to constriction.[3]

Decreased peripheral extremity circulation limits tissue resilience, leads to rapid death of tissue, and impedes wound healing. Wound healing and tissue regeneration depend on an adequate blood supply to the region. Ischemia due to vascular disease impedes healing by reducing the supply of oxygen, nutrients, and soluble mediators that are involved in the repair process. Purely ischemic diabetic foot ulcers are uncommon, representing only 10% to 15% of ulcers in patients with diabetes. More commonly, ulcers have a mixed ischemic and neuropathic origin. Once an ulcer is formed, the blood supply necessary to allow healing of the wound is greater than that needed to maintain intact skin. This leads to chronic ulcer development unless the blood supply is improved.[2]

Mechanically Induced

The foot is a complicated biologic structure containing 26 bones, numerous joints, and a network of ligaments, muscles, and blood vessels. An appreciation of the biomechanics required for walking is essential in understanding the etiology of foot ulcers. Gait is a complex set of events that requires triplanar foot motion and control of multiple axes for complete bipedal ambulation. Various external and internal forces affect foot function. The combination of body weight pushing down and the ground reactive force pushing up creates friction and compressive forces. Shear results from the bones of the foot sliding parallel to their plane of contact during pronation and supination.

Three mechanisms frequently play a part in the development of mechanically induced neuropathic ulcerations.

The first mechanism is usually the result of a quick traumatic event, like stepping on a sharp object such as a nail or a piece of broken glass, which results in piercing of the skin.

The second mechanism is application of chronic low grade pressure as would be seen with wearing an ill-fitting shoe. This creates focal areas of tissue ischemia over a bony prominence such as a bunion or hammer toe. If the pressure is maintained for a significant period of time, this leads to necrosis and ulceration.

The third mechanism involves a force of repetitive, moderate pressure. This accounts for the majority of diabetic plantar ulcers. Repetitive pressure >10 kg per cm acting on the foot during gait will contribute to the formation of this type of ulcer. In the absence of neuropathy, the repetitive pressure beneath prominent areas produces pain that prompts a sensate person to take measures to alleviate the discomfort. Such measures include limping or modification of gait to place weight on a different part of the foot, changing to more comfortable shoes, applying pads, or seeking medical treatment. The loss of protective sensation in the neuropathic patient lets foot wounds go undetected resulting in areas of inflammation and enzymatic autolysis culminating in tissue breakdown and ulceration. The location of this type of ulcer is predictable. Areas of increased pressure are commonly identified by areas of plantar callus formation, which conversely are the areas prone to ulceration in a neuropathic patient. Patients

have inadequate protective sensation during all phases of gait; therefore, high loads are undetected due to loss of pain threshold, which results in prolonged and increased forces. These problems manifest as abnormal pressure points, increased shearing, and greater friction to the foot. Because this goes unrecognized in the insensate foot, gait patterns remain unchanged, and the stresses eventually cause tissue breakdown and ulceration. Loss of protective sensation secondary to neuropathy can rapidly lead to ulceration at these high pressure zones if patient education and preventive measures are not taken.

Foot deformities or ill-fitting footwear enhance pressure points because they focus the forces on a smaller area. When the foot flattens too much or overpronates, the ankle and heel do not align during midstance and some bones are forced to support more weight. The foot strains under the body's weight, causing the muscles to pull harder on these areas, making it more difficult for tendons and ligaments to hold bones and joints in proper alignment. Over time, swelling and pain on the bottom of the foot or near the heel may occur. Bunions can form at the great toe joint, and hammertoe deformities can form at the lesser toes. Abnormal foot biomechanics resulting from limited joint mobility and foot deformities magnify shearing forces, resulting in increased plantar pressure on the foot during ambulation. This can represent critical causes for tissue breakdown. For instance, ischemic ulcers often develop on the dorsum of the foot, over the first and fifth metatarsal heads. A heel ulcer can develop from constant pressure applied while the heel is in a dependent position or during prolonged immobilization and bed rest. Although simple cutaneous breakdown is not infrequent because of shearing forces or direct trauma, healing is the rule unless the wound repair mechanisms are suboptimal due to impairment of perfusion, infection, or repeated, continuous traumatic insults. Lack of sensation allows the damage to cascade to ulceration. Lack of perfusion decreases tissue resilience and leads to rapid death of tissue and impedes wound healing for tissue repair. Broadly speaking, therefore, the progression to foot ulceration can be attributed to impaired arterial supply, neuropathy, musculoskeletal deformities, infection, or a combination of these factors.[2]

Risk Factors

Several clinical causal pathways have been identified. This allows the clinician to grade the primary risk factors associated with the onset of DFUs. The presence of peripheral arterial disease and a history of prior ulceration or amputation greatly increases the risk of complications beyond the introducing factors of peripheral neuropathy or deformity.[7] Early recognition and management of risk factors is important for reducing morbidity of foot ulceration. These risk factors include:

- age
- sex (being male)
- diabetes duration and type
- insulin use
- past history of diabetic foot ulcer (DFU) and amputation

- lower limb bypass procedures
- biomechanical factors such as glycemia level and poor glycemic control
- dyslipidemia
- sensory and autonomic neuropathy
- absence of reflex
- limited joint motion
- muscle weakness
- callus formation
- Charcot deformity
- hammer/claw toe deformity
- abnormal Achilles tendon reflex
- greater body mass (≥20kg)
- arterial insufficiency
- vascular disease
- skin dryness and fissure (caused by autonomic neuropathy)
- reduced skin oxygenation and foot perfusion
- diastolic hypertension
- impaired vision
- smoking
- alcohol consumptions
- lack of proper diabetes educations
- low income
- race (African Americans, Hispanic Americans and Native Americans face a higher risk)
- poor personal hygiene and self-care

Smoking, hypertension, and hyperlipidemia are considered as risk factors due to their effects on the increased occurrence of peripheral arterial occlusive disease in diabetics, which typically involves the tibial and peroneal arteries, but leaves the dorsalis pedis artery unaffected.[3]

The most important risk factors for the development of diabetic foot ulcers are: peripheral neuropathy (motor, sensory and autonomic), structural and anatomical deformities, environmental factors, peripheral vascular disease, a compromised immune system and poor metabolic control, in addition to social influences such as emotional, psychological and behavioral problems.[5]

Symptomology

Diabetic foot ulcers usually start with the following symptoms[3]:

- Atrophic integument
- Any break in the skin resulted from abnormal wear and tear, injury, or infection

- Sores, ulcers, or blisters on the foot or lower leg
- Persistent pain, which can be a symptom of sprain, strain, bruise, overuse, improperly fitting shoes, or underlying infection.
- Calluses and corns that may be a sign of chronic trauma to the foot
- A claudicating or difficulty walking that can be sign of joint problems, serious infection, or improperly fitting shoes
- Discoloration in feet: black, blue, or red
- Cold Feet
- Absent pulses
- Swollen foot or ankle
- Odor
- Fever or chills in association with a wound that can be a sign of a limb-threatening or life- threatening infection
- Redness, which can be a sign of infection, especially when surrounding a wound, or of abnormal rubbing of shoes or socks.
- Swelling of the feet or legs, which can be a sign of underlying inflammation or infection, improperly fitting shoes, or poor venous circulation.
- New or lasting numbness in the feet or legs, a sign of nerve damage from diabetes
- Signs of poor blood circulation, such as:
- Pain in the legs that increases with walking but improves with rest (claudication)
- Absence of pedal hair or pallor on elevation (coupled with other symptoms)
- Hard shiny skin on the legs
- Toenail fungus, athlete's foot, and ingrown toenails, which may lead to more serious bacterial infections
- Drainage of pus from a wound is usually a sign of infection. Persistent bloody drainage is also a sign of a potentially serious foot problem.

Clinical Presentation

Diabetic ulcers can occur anywhere on the foot but clinically the most frequent presentation is on the plantar surface. This predilection of diabetic ulcer on the plantar surface is related to the trauma that is developed in this area due to increased discharge pressure on the plantar surface during walking. Under normal conditions the foot has the ability to distribute the load equally over the entire surface (the forefoot, middle foot and rear foot) consequently preventing the development of ulcers. This capability is lessened in diabetics, first of all because of changes in the architecture of the foot related to the fundamental motor neuropathy that produces disorders in the mobility of joints, limited both by neuropathy as well as by the metabolic changes. Additionally, there is decreased mobility of the Achilles tendon, in turn creating an equinus deformity that directs plantar forces to the region of the forefoot.[5]

Overpowering by the extrinsic foot muscles can also lead to an equinus deformity at the ankle and a varus hindfoot. A cavovarus foot type can develop, leading to decreased range of motion of the pedal joints, an inability to adapt to terrain, and low tolerance to shock. In patients with “flatfoot” deformities, there is often excessive pronation and a hypermobile first ray that leads to an excessive amount of pressure beneath the second metatarsal. In neuropathic patients with this foot type, callus formation and subsequent ulcerations often develop beneath the second metatarsal head. In contrast, patients with a rigid cavus foot commonly ulcerate beneath the heel, the first metatarsal, and/or the fifth metatarsal. In patients with a “rocker bottom” Charcot deformity, the area beneath the cuboid is an area of increased risk.[2]

Ischemic ulcers

Ulcerations caused by ischemia are typically located on the tips of the toes and between the digits. The lesions often appear punched out and are painful but exhibit little bleeding. Ischemic ulcers are characterized by absence of bleeding, pain, and a precipitating trauma or underlying foot deformity. They also often develop on the dorsum of the foot and over the first and fifth metatarsal heads. Ischemic ulcers are uncommon on the plantar surface as the pressure is usually less sustained, and the perfusion is better. A heel ulcer can develop from constant pressure applied while the heel is in a dependent position or during prolonged immobilization and bed rest. It should not be a surprise that a patient with relatively mild symptoms of arterial insufficiency develops limb-threatening extremity ulcers. This is due to the fact that once an ulcer is present, the blood supply necessary to heal the wound is greater than that needed to maintain intact skin. A chronic ulcer will develop unless the blood supply is improved.[2]

Neuropathic ulcers

Neuropathic ulcerations typically occur at the heel or over the metatarsal heads on the plantar surface at pressure points but may also occur in less characteristic locations secondary to trauma. They usually are painless. The sensory neuropathy in the diabetic patient may allow the destructive process to go unchecked, with extension into the deep plantar space and minimal appreciation by the patient.[2]

Classification

Several schemes have been used to classify diabetic foot ulcers, but none of them has been accepted universally. The following are the most commonly used classifications.

Wagner-Meggitt Classification

Wagner-Meggitt, the most popular method, has been used for decades to classify DFUs in six grades based on the wound’s depths and extent of gangrene[3]:

Wagner Classification of Diabetic Foot Ulcer[1]

Grade 0	No ulcer in a high risk foot.
Grade 1	Superficial ulcer involving the full skin thickness but not underlying tissues.
Grade 2	Deep ulcer, penetrating down to ligaments and muscle, but no bone involvement or abscess formation.
Grade 3	Deep ulcer with cellulitis or abscess formation, often with osteomyelitis.
Grade 4	Localized gangrene.
Grade 5	Extensive gangrene involving the whole foot.

The University of Texas Wound Classification

The University of Texas classification is a more comprehensive scale and includes risk stratification and expresses tissue breakdown, infection and gangrene separately. Although Texas classification describes the grade of wound in more details, it does not include measures of neuropathy or ulcer area.[3] The University of Texas Wound Classification is a simple classification that considers grade (depth of the lesion) and stage (presence or absence of infection and ischemia). The 'grade' ranges from 0 (pre- or post-ulcerative completely epithelized lesion) to III (involvement of bone or joint). 'Stage' ranges from A (absence of both infection and ischemia), B (infection), C (ischemia) and D (infection and ischemia). The 'grade and stage' are combined to give the final classification.[1]

The University of Texas Wound Classification System[1]

Stage	Grade			
	0	I	II	III
A	pre- or post-ulcerative completely epithelized lesion	Superficial wound	Wound penetration up to tendon or capsule	Wound penetration Up to bone 0r joint
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

PEDIS

Another validated classification system for DFUs that includes the severity of infection is The PEDIS (perfusion, extent, depth, infection, and sensation) system.[1] PEDIS classification was proposed by the International Working Group on the Diabetic Foot and grades the wounds on the basis of five features: perfusions (arterial supply), extent (area), depth, infection and sensation. There are levels of 1 to 4 for each of these factors. The in-depth nature of this system is appropriate for the research community that desires this amount of detail.[3]

S(AD) SAD System

S(AD) SAD system builds upon the Wagner classification to include several additional categories: size (area, depth), sepsis, arteriopathy and denervation. The S(AD) SAD classification is a validated system with grades 0 to 3.[3]

RYB Color Classification

RYB Color Classification was developed for the nursing literature and has obtained considerable popularity. The system relies purely on a color scheme with no additional considerations. R/Red wounds are those that exhibit pale pink to beefy red granulation tissue and are deemed to be in the inflammatory or proliferative phase. Y/Yellow wounds are marked by pale ivory, yellowish green or brown color, slough of necrotic but moist tissue, and wound exudates. B/Black wounds are marked by black, brown or tan color, and desiccated eschar. The RYB classification is an easy and widely accepted system in the nursing literature and shows the continuum from acute to chronic wounds. Conversely, it is non-specific with no consideration of depth or size, and no consideration of the contributing factor of neuropathy.[3]

DEPA Scoring System

DEPA Scoring System, is the newest DFU classification system, in which D stands for depth of the ulcer, E for extent of bacterial colonization, P for phase of ulcer, and A for associated etiology. Ascending scores, from 1 to 3, are assigned for increasing levels of intensity in each category. For instance, an ulcer involving soft tissue receives a 2. Contamination of this ulcer receives a 1. The ulcer is in the inflammatory phase, generating a 2 score, and has an underlying bony deformity, generating another 2 score. Accordingly, this ulcer has a total score of 7. Ulcers with a total score of 6 or less are considered “low grade” ulcers. Recommended treatment measures include oral antibiotics (if infected), blood sugar control (type not specified) and debridement. Those with a total score of 7 to 9 are deemed “moderate grade” wounds that one would treat with parenteral antibiotics, insulin, debridement, healing promoting agents and pressure relieving methods. The “high grade” lesions, those with a total score between 10 and 12, require a conservative trial including parenteral antibiotics, insulin, debridement, healing promoting agents and vascular reconstruction. The authors of the DEPA classification system offered acute ischemia patients a below-knee amputation; however, other practitioners may offer revascularization or other interventions. The scores of 11 to 12 are prognostic for amputation and if these are heel ulcers, they were even more likely to lead to amputation. Scores of 10 or greater predict difficulty with healing, while scores of 6 or less indicate probable healing. Vascular impairment is one

of the most important causes of DFU, and in addition to hyperglycemia, is the main impediment in healing the ulcers. Smoking cessation, diet improvement and controlling total and LDL cholesterol, antiplatelet drug treatment, and maintaining an optimum blood pressure will help reducing the impact of vascular impairment on ulcer healing.[3]

Assessment

In diabetic patients with foot wounds, the clinician should perform a systematic examination to determine if the wound is infected and the degree of severity of said infection. The examination should start with vital signs, consciousness, complete review of the limb to the foot end, always removing the patient's shoes and socks, and should include a neurological, vascular, dermatological and musculoskeletal examination to determine the external characteristics of the skin, the shape and structure of the foot and alterations in the fingers, protruding metatarsal, heel, etc.[5]

History

As in all medical conditions the initial evaluation of a patient with a diabetic foot ulcer begins with a detailed history. A complete history will aid in assessing the severity and risk of foot ulceration. Important components of the history include[7]:

- duration of DM
- length of time the ulcer has been present
- etiology of the wound (if known)
- any self or professional treatment
- prior ulcer, infection, or amputation history; personal medical history
- allergies
- medications
- surgical history
- family history
- tobacco use
- alcohol abuse
- recreational drug use

The most common subjective complaints are those of neuropathic disease, which include history of numbness, paresthesias, and burning pain in the lower extremities. Patients often report previous episodes of foot ulcers and chronic skin infections.

Arterial insufficiency is suggested by a history of underlying cardiac or cerebrovascular disease, complaints of leg pain when walking, or impotence. Symptoms of arterial insufficiency occur because of inadequate perfusion to the lower extremity relative to its metabolism. Tissue hypoxia and the subsequent increase in concentration of lactic acid produce pain. Patients may complain of pain in the buttocks or calves brought on with activity and relieved with rest (intermittent claudication) or pain in the forefoot aggravated by elevation and relieved by dependency (rest pain).[2]

Vital Signs

The patient's temperature, respiratory rate, heart rate, and blood pressure in both upper extremities should be obtained. Fever may indicate the presence of an infected ulcer, and the presence of tachycardia and tachypnea may support the diagnosis of a septic foot.[2]

Inspection

A simple, rapid examination of the foot takes no more than one to two minutes.[7] Visual inspection coupled with an accurate history can determine the presence of a chronic vascular condition. In chronic arterial insufficiency, the arterioles are maximally dilated as a compensatory response to the chronic ischemia intensifying color changes. In acute arterial occlusion, the venules empty, leading to a chalky white appearance regardless of extremity position. Partial but inadequate perfusion either from an incomplete acute or chronic occlusion allows for pooling of blood in the venules, which may be red in the cold or blue at higher temperatures.

When the extremity is at the level of the heart, the pooled blood masks the color imparted by the arterial flow. Elevation of the extremity above the level of the central venous pressure allows the pooled venous blood to drain, enabling an accurate assessment of the degree of arterial flow. The normal extremity remains pink, whereas that with arterial insufficiency becomes pallid. Conversely, allowing the extremity to become dependent causes an intense rubor or cyanosis. The time of return of blood to the dependent extremity is a useful marker of the severity of the deficit (normally <20 seconds).[2] Trophic changes of the skin may include atrophic, shiny appearance with loss of hair, coolness to touch, cyanosis, and thickened nails[7]. Comparison of color and trophic changes between extremities gives a good indication of the severity of the process unless a bilateral deficit is present, in which case the experience of the examiner is required to make an accurate diagnosis.[2] From a clinical standpoint a significant sign of impending ulceration is the pre-ulcerative callus. This is seen as hyperkeratotic tissue with visible hemorrhage within the epidermal or dermal skin layers.[7]

Palpation

Skin temperature is a reliable indicator of the blood flow rate in the dermal vessels, though flow is governed primarily by constriction or dilation of the arterioles to maintain a constant core temperature. Nevertheless, the temperature of the skin as a marker of perfusion is useful and can be assessed by lightly palpating the skin with the back of the hand and comparing similar sites from one extremity to the other. An ischemic limb is cool, and demarcation of temperature gives a rough indication of the level of the occlusion. Again, assessment of temperature differences is confounded when both extremities are affected.[2]

A thorough physical examination should also include an evaluation of arterial outflow and the presence of peripheral arterial disease (PAD). This includes palpation of all pulses in the lower extremity, including the dorsalis pedis/tibialis anterior, posterior tibial, popliteal, femoral, and abdominal aortic pulses.[7]

Musculoskeletal Evaluation

The musculoskeletal examination is also fundamentally important to evaluate the patient with a diabetic foot ulcer. In the majority of patients an examination of the biomechanical contribution will reveal the cause of the ulcer. The common factor is a focal increased shear or vertical pressure. As such, a thorough examination for pedal deformity is of paramount importance. Overall appearance of the foot should be appreciated, followed by a detailed examination of specific deformities, including joint position, range of motion, and rigidity versus flexibility. Functional compensation at one joint for lack of motion of another is also commonly seen. For example, lack of motion of the great toe joint (hallux limitus) often leads to a compensatory increased motion at the hallux interphalangeal joint. This compensation increases plantar pressures at the joint with a subsequent DFU. Another highly important mechanical contributor to the creation of diabetic foot ulcers, especially those on the plantar forefoot area, is ankle joint equinus, or lack of dorsiflexion of the foot on the ankle during active walking.[7]

Ulcer Evaluation

Specific characteristics of the ulcer such as location, size, depth, and appearance should be recorded during the initial evaluation and with each subsequent follow-up visit to record progress and evaluate the treatment regimen. The margins of the ulcer should be undermined to evaluate the extent of tissue destruction. Ulcer extension to tendon, bone, or joint should be sought. A positive probe-to-bone finding has a high predictive value for osteomyelitis.[2]

A sterile stainless steel probe is used for assessing the ulcer to determine the depth and if there are sinus tracts present. Presence of granulation tissue or slough should be looked for in the floor of the ulcer to determine subsequent management. Diagnosing a soft tissue infection in patient with diabetes is sometimes difficult, as the signs of inflammation of the overlying ulcer may be absent. The infection is mainly diagnosed based on presence of clinical signs and symptoms such as redness, warmth, tenderness, purulent secretions and fever. Palpation of the bone at the base of the ulcer has been suggested as positive predictor of underlying osteomyelitis.[1]

In the diabetic patient with a neuropathic foot ulcer and concomitant PAD the wound appearance may be slightly different. In some situations, the wound will look similar to the well vascularized ulcer with the exception of a paler or light pink appearance to the wound base instead of a red, granular appearance. In more advanced neuroischemic wounds the appearance will be markedly different with a fibrous yellow appearance and an often irregular, sometimes punched out-appearing, shape.[7]

Imaging

Precise, comprehensive anatomic imaging is the cornerstone of successful revascularization of the ischemic lower extremity in patients with diabetes mellitus.

Contrast arteriography has been the mainstay for many years and remains the gold standard due to its superior image resolution and being the only modality used for both diagnosis and treatment. Coexistent renal insufficiency, however, makes conventional angiography impractical in significant percent of diabetic patients. CO_2 angiogram and noninvasive Doppler studies are other alternative imaging options in this patient population.

Duplex ultrasound is an integral component of diagnostic testing for the evaluation and management of arterial disease. This technology combines the acquisition of blood flow (pulsed Doppler spectral analysis) and anatomic information. Contemporary duplex ultrasound systems provide high-resolution imaging of tissue and vessel anatomy, including three-dimensional vessel reconstruction and evaluation of atherosclerotic plaque morphology. The duplex testing performed in the vascular laboratory is an extension of clinical assessment and is used to verify the presence and extent of disease, the involved arterial segment, and its severity.

Other noninvasive imaging methods useful in the assessment of patients with leg ulcers include plain radiography, MRI, MR angiography, and CT angiography.[2]

X rays are helpful to determine the depth of foot ulceration and to assess presence of bone infection or neuroarthropathy. In CN, radiographs may reveal bony erosions, fractures, subluxation/dislocation of multiple joints, osteosclerotic features or united fractures.

Magnetic Resonance Imaging has emerged as a popular investigation for many of the foot problems. In diabetic feet it is especially useful to detect infection and CN. It is used to evaluate the extent of foot infection by revealing the depth of ulceration, edema and localized fluid collections in the soft tissues, joints and tendon sheaths. Positron emission tomography demonstrates a high specificity for osteomyelitis.[1]

Vascular Assessment

The ankle brachial index (ABI) or toe-brachial index can be used to determine the extent of the vascular problem. The ABI is obtained by measuring the systolic blood pressures in the ankles (dorsalis pedis and posterior tibial arteries) and arms (brachial artery) using a handheld Doppler and then calculating a ratio. However, in patients with calcified, poorly compressible vessels or aortoiliac stenosis, the results of the ABI can be complicated.[3] Values below 0.9 suggests an obstruction, while ABI less than 0.4 is associated with tissue necrosis and a significant risk for amputation. Screening ABI every 5 years in patients with diabetes without any signs/symptoms of vascular insufficiency has been recommended.[1]

Pulse oximetry has also been reported to be as effective as ABI and the sensitivity of the test will be improved if used together with ABI.

Other investigations to detect vascular insufficiency include measuring absolute toe pressure, pulse volume recordings and angiography (CT, MRI or contrast). In-shoe and barefoot peak plantar pressure measurement has also been suggested to assess foot at-risk and prevent ulcers.[1]

Neurological Testing

The lower extremity neurologic examination is essential and should include testing for motor strength; deep-tendon reflexes; vibratory, proprioceptive, and protective sensation. Loss of protective sensation due to peripheral neuropathy is the most common cause of ulceration in the diabetic population.[2]

Sensory

The Semmes-Weinstein monofilament is a convenient, inexpensive, and painless evidence-based tool that should be utilized in the initial evaluation of all patients with diabetes mellitus as a screen for peripheral neuropathy.[2] This simple, rapid test is easily performed in the clinic. The 5.07 Semmes Weinstein monofilament is constructed to produce a standard 10 grams of force when bent and has been found to accurately predict the presence of ulceration. Ten sites are tested (plantar toes and metatarsal heads 1, 3, and 5; two points on the medial arch; one point on the heel; and one on the dorsum of the foot). If the patient is unable to feel 4 of 10 sites, he is diagnosed with peripheral neuropathy.[7] A positive Semmes-Weinstein monofilament result is a significant predictor of future ulceration and likely lower extremity amputation as well in patients with diabetes mellitus.[2]

Autonomic

The Heart Rate Variability (HRV) with deep breathing or orthostatic blood pressure is measured to detect autonomic neuropathy and any decrease or absence of HRV is considered the earliest sign of autonomic neuropathy in DM. Specialized tests for sudomotor dysfunction include thermoregulatory sweat testing, quantitative sudomotor axon reflex testing, silicone impressions, the Sympathetic Skin Response (SSR), and the quantitative direct and indirect axon reflex testing. These tests can be used in various combinations to localize the lesion of autonomic dysfunction (pre-ganglionic or post-ganglionic).[1]

Laboratory Investigations

The standard procedure involves measuring blood glucose level and urine for glucose and ketones. Other investigations like full blood count, blood urea, electrolytes, and creatinine levels should be monitored regularly. Glycosylated hemoglobin (HbA1C) is important to gauge the patient's overall glycemic control as HbA1c shows the mean blood sugar concentration best over previous weeks to months. Hepatic and renal function tests are necessary for monitoring the patient's metabolic status. Routine

wound cultures are not recommended since all wounds harbor microorganisms. However, in the presence of invasive infection, cultures from the deeper tissue will help to identify the causative microorganisms.[1]

Infection

Patients with diabetes appear to be more prone to various infections than their nondiabetic counterparts. Several factors increase the risk of development of diabetic foot infections including diabetic neuropathy, peripheral arterial disease, and immunologic impairment. Diabetes causes impairment in the functioning of polymorphonuclear leukocytes that can manifest as a decrease in migration, phagocytosis, and decreased intracellular activity. Some of the defects appear to improve with control of hyperglycemia underscoring the need for a tight and consistent control of hyperglycemia.

Undiagnosed clean neuropathic foot ulcers often convert to acute infections with abscess and/or cellulitis. Presence of peripheral arterial disease, neuropathy, or impaired leukocyte functions may reduce the local inflammatory response and classical signs or symptoms of local infection that makes the diagnosis of infection in a diabetic foot especially challenging.

Diabetic foot infections can be classified into those that are nonthreatening and those that are life or limb threatening. Non-limb-threatening diabetic foot infections are often mild infections associated with a superficial ulcer. They often have less than 2 cm of surrounding cellulitis and demonstrate no signs of systemic toxicity. These less severe infections can often be managed with local wound care, rest, elevation, and oral antibiotics on an outpatient basis.

A foot infection in a diabetic patient can also present with a more severe, life- or limb-threatening picture. In these patients, there is usually a deeper ulceration or an undrained abscess, gangrene, or necrotizing fasciitis. They tend to have greater than 2 cm of surrounding cellulitis, as well as edema of the affected limb. These more severe cases generally present with fever, leukocytosis, and hyperglycemia.

In contrast to nondiabetic individuals, complex foot infections in diabetic patients usually involve multiple organisms. These included a combination of gram-positive and -negative, as well as aerobic and anaerobic organisms. The most prevalent organisms typically are S. aureus, coagulase-negative Staphylococcus, group B Streptococcus, Proteus, Escherichia coli, Pseudomonas, and Bacteroides. Recently, methicillin-resistant S. aureus (MRSA) infection has become more common in diabetic foot ulcers and is associated with previous antibiotic treatment and prolonged time to healing. Anaerobic infections with Clostridium are also not uncommon.[2]

The foot has several compartments, which are inter-communicating and the infection can spread from one into another, and lack of pain allows the patient to continue

ambulation further facilitating the spread. The foot also has soft tissues, which cannot resist infection, like plantar aponeurosis, tendons, muscle sheaths, and fascia. A combination of neuropathy, ischemia, and hyperglycemia worsens the situation by reducing the defense mechanism.[6]

By definition, infection is characterized by the presence of purulent secretions or at least two of the classic signs of inflammation (erythema, hyperemia, edema, or swelling and pain) but these can be masked by lack of the sensitivity in the patient due to sensory neuropathy or impaired immune response.[5] Complaints of pain in an insensate patient should raise suspicion for an infection. Patients also complain of recalcitrant hyperglycemia and other constitutional symptoms such as fevers, malaise and chills, sometimes referred to as the 'diabetic flu', which should raise suspicion for a deep infection. Superficial infections typically show no signs of systemic toxicity and glycemic levels remain unaffected. Deep foot infections, in contrast, result in contiguous spread of erythema and edema with accompanying constitutional symptoms such as fever, chills, malaise, and occasionally blood glucose elevations.[7]

The types of diabetic foot infections can start from a simple paronychia onychomycosis, cellulitis, foot infection, deep tissue infection, septic arthritis, osteomyelitis, necrosis or gangrene. Infections can be painless, persist for days, weeks or months and progress rapidly even in a few hours.[5]

Osteomyelitis

Infection of bone underlying a foot ulcer is an especially difficult diagnostic and therapeutic problem. Osteomyelitis generally results from a contiguous spread of deep soft tissue infection through the cortex to the bone marrow.[6] Clinicians should assume that osteomyelitis is probably present if the bone is visible or palpable by probing. Diagnosing osteomyelitis in a patient with diabetic foot is often difficult. Major problems include differentiating soft tissue infection from bone infection and infections from non-infectious disorders (Charcot Foot). Plain radiography usually shows focal osteopenia, cortical erosions or periosteal reaction in the early stage and sequestration in the late stage.[6] Bone infection must usually be present for at least 2 weeks before it can be regarded as the cause of abnormalities seen on plain radiographs. Most nuclear medicine tests (e.g., technetium bone scans or labeled leucocyte scans) are more sensitive than plain radiography, but are relatively non-specific and less accurate than MRI. The gold standard test for osteomyelitis is a bone biopsy sample processed for culture and histology.[3] A simple clinical test is probing to the bone. A sterile metal probe is inserted into the ulcer if it penetrates to the bone it almost confirms the diagnosis of osteomyelitis. Chronic discharging sinus and sausage-like appearance of the toe are the clinical markers of osteomyelitis. Definitive diagnosis requires obtaining a bone biopsy for microbial culture and histopathology.[6]

Ulcer Management

The management of wounds requires meticulous care and early treatment by a

multidisciplinary foot care team. This management team may include: therapists, infectious disease specialists, microbiologists, podiatrists, nurses specializing in diabetes and physicians with knowledge of DM, all striving to provide quality care and good metabolic control. Optimal care and early detection of diabetic ulcers can greatly reduce the occurrence of infections.[5]

Debridement

Ulcers heal faster when the wound is clean as the devitalized necrotic tissues hinder cell migration and predispose it to infection and prohibit healing. Debridement of the wound may hasten healing by removing the dead necrotic tissue, particulate matter, or foreign materials, and reducing bacterial load. The conventional way is to use a scalpel and excise all unwanted tissues including callus and eschar (sharp debridement). Since the necrotic tissue often extends beyond the ulcer bed, liberal debridement of deeper tissue beyond the ulcer boundary is often necessary.

Other methods of wound debridement include physical debridement using wet-to-dry dressings (no longer recommended); hydrodissection or hydrocision with the use of high pressure saline beam; enzymatic debridement using enzymes like collagenase and papain as ointment preparations; autolytic debridement with the use of moisture retaining dressings; and biological debridement with use of larvae of common green bottle fly (*Lucilia sericata*). Occasionally sharp debridement is combined with other forms of debridement to achieve ulcer healing.[1]

Surgical (Sharp) Debridement

Surgical debridement is the fastest means, allowing the surgeons to accurately assess the severity and extent of the wound. It is the method of choice, especially in life- or limb-threatening infections with necrotic eschar or gangrene. It is also indicated for wounds with extensive or adhering eschar, in which the rapid clearance of necrotic tissue is required.

The drawbacks of sharp surgical debridement are that it is nonselective, and thus normal healthy tissue may be removed at the same time; bleeding; pain; and the need for anesthesia and the operating theater. However, in the presence of ischemic or neuroischemic ulcers, the management should aim toward restoring tissue perfusion prior to aggressive wound debridement or aggressive surgery to ensure wound healing and prevent the “dieback” phenomenon.[8] The limiting factors of sharp debridement include inadvertent bleeding, poor pain tolerance by the patient and lack of any objective markers to differentiate impaired and healthy tissue to ascertain the extent of debridement.[1]

Surgical debridement should be undertaken when absolutely necessary and should be performed with extreme caution to minimize damage to the healthy tissue. In some situations, when dry, noninfected gangrene, necrosis, or eschar tissue is present, they can be left in situ until autoamputation takes place, or the tissue spontaneously lifts off

the wound bed. Surgical intervention may be needed if wounds turn wet or become infected.[8]

Nonsurgical Debridement

Wet-to-dry dressing is one of the most commonly practiced nonsurgical methods of debridement. It is most effective for managing sloughy and minimally necrotic wounds. This technique is performed by leaving wet gauze in direct contact with wound surfaces and removing it when dried together with any adhering slough tissue. Unfortunately, this method causes excessive pain, as well as bleeding, and removes the new, healing epithelium when the dressing is changed.[8]

Autolytic debridement uses the inherent ability of the body to digest and remove necrotic tissue with endogenous enzymes or phagocytic cells. This approach is facilitated by a moisture-retention dressing, such as the application of a hydrogel dressing. This method is relatively easy to perform, requires limited technical skills, and involves minimal pain. It is indicated in wounds with a minimal necrotic load or that need more aggressive debridement requiring anesthesia in patients who are unable to tolerate pain. However, it is time-consuming and frequently causes maceration of the surrounding skin.[8]

Enzymatic debridement uses enzymatic agents such as collagenase and papain-urea to dissolve necrotic tissue. It is suitable for nonsurgical patients and can be effectively combined with moist wound healing. Papain is a broad-spectrum enzyme that is useful for bulk debridement, whereas collagenase is gentler on viable cells. Like autolysis-promoting agents, it can cause wound edge maceration, and additionally, the enzymatic agents are expensive.[8]

Moisture Balance

In general, delicate control of the wound and surrounding area moisture balance has been proven to accelerate wound healing in terms of reepithelialization, promote granulation tissue formation and prevent maceration of the surrounding skin. However, in the diabetic foot, moisture control needs to be linked closely to the treatment plan, which is made based on the patient's condition. For example, in an ischemic or neuroischemic foot where there is a dry gangrene and without infection, hydration of the wound/gangrene may not be appropriate, as the gangrenous part may be converted to wet gangrene and become infected. With adequate attention, the toe, foot, or ulcer can be allowed to be dry and become mummified, thus allowing autoamputation to take place.

When indicated, the wound can be kept in a balanced moisture healing environment as one would manage other chronic nonhealing wounds as part of the fundamental principles of wound bed preparation. To attain moisture balance, the clinician should create and maintain a warm, moist wound bed and avoid excessive periwound moisture that can cause surrounding skin maceration. Balanced moisture is required for the optimal effects of growth factors and cytokines within the wound to stimulate

proliferating cells, such as keratinocytes, endothelial cells, and fibroblasts. Excessive moisture in the wound contains matrix metalloproteinases and serine proteases that can break down or damage essential extracellular matrix materials.

The effect on surrounding skin, such as maceration, especially over the sole area, will reduce the host defensive barriers against microbial invasion provided by the thick skin in the region. On the other hand, in a dry condition, cellular activities will be inhibited, an eschar will form, and further tissue necrosis may occur at the wound bed.

Based upon the importance of moisture balance, a vast array of dressing materials and techniques has been developed. There is no one dressing that is perfect for a chronic wound during its course of healing, as the wound healing process is dynamic. Indications for dressing materials also need to change with respect to the wound conditions. For example, available moisture-retentive dressings include occlusive, semi-occlusive, absorptive, and hydrating dressings. In a highly exudative wound, an absorptive dressing such as foam will be appropriate, whereas in a dry wound eschar, an occlusive or semi-occlusive dressing such as a hydrocolloid, gel-based dressing such as a hydrogel, carboxymethylcellulose, or hydroactive hydrocolloid gel will be suitable to achieve the appropriate moisture balance.[8]

Dressings

Wound dressing, widely used to cure the infected wounds, is the most important component of a successful wound care. There are a number of available dressing types to consider. Although there is a shortage of published trials to support the use of one type of dressing compared to another, the characteristics of specific dressing types can prove beneficial depending on the characteristics of the individual wound. An ideal dressing should contribute to a moist wound environment, absorb excessive exudates, and not increase the risk for infections. Dressing changes and wound inspection should occur on a daily basis. Saline-soaked gauze dressings, for example, are inexpensive, well tolerated, and contribute to an atraumatic, moist wound environment. Foam and alginate dressings are highly absorbent and can aid in decreasing the risk for maceration in wounds with heavy exudates.

The process of autolysis is important in wound care. If an occlusive dressing is provided as a barrier to the outside environment, the body’s own phagocytic processes will provide debridement of wounds. These products range from occlusive films such as Tegaderm, which are permeable to air and water vapor, but impermeable to fluid and microorganisms to hydrocolloids such as DuoDerm, which are also occlusive but provide absorption of exudates in addition to maintaining a moist environment for autolysis. For heavily exudative wounds, there are a range of absorptive products, including various hydrophilic foam dressings, hydrogels, hydrofibers, and alginates, which can absorb up to 20 times their weight.

Silver dressings have been used for decades with little significant toxicity to cure infected wounds. Silver has a very broad spectrum of microbial coverage, including

yeast, fungi, mold, and even antibiotic-resistant organisms, when used at appropriate concentrations. Silver ion binds to negatively charged particles such as proteins, DNA, RNA and chloride ions. Silver sulfadiazine is known to release active silver ions gradually for a longer time. Nanocrystalline silver dressing contains two layers of high-density polyethylene net sandwiching a layer of rayon/polyester gauze. The outer layer is coated with a nanocrystalline (<20 nm), uncharged form of silver, and the inner layer helps maintain a moist environment for wound healing. This sandwich provides a sustained release of silver into the wound due to the low affinity of Ag0 to the negatively-charged particles in the wound. Other advantage of nanocrystalline silver dressing is less frequency of dressing changes compared the standard silver dressings, which must be changed up to 12 times a day. This brings less disruption to the wound healing bed.[3]

Negative Pressure Wound Therapy

Although several advanced debridement and dressing techniques have been developed to improve wound healing, achieving adequate wound closure is a major problem. Negative pressure wound therapy (NPWT) has emerged as an effective treatment for these complex wounds. This involves application of subatmospheric pressure to the wound through open-celled foam dressing in a closed environment. The pump is connected to a canister, which collects the wound exudates.[2] NPWT can drain away excessive wound exudates, reduce wound edema, contribute to improved tissue perfusion, and aid in reducing the wound size by promoting wound contraction and reducing the complexity of the wounds. The negative pressure exerted on the wound via the foam has microdeformation effects due to the stretching of small tissue blebs into the pores of the dressing. These stimulate changes within the cytoskeleton, resulting in cascades of biologic effects, including the stimulation of angiogenesis and formation of granulation tissue.[8] Once healthy granulation of deep complex wounds is achieved, restoration of intact skin barrier is of utmost importance to prevent infection, minimize wound contraction to maintain function, minimize cosmetic disfigurement, and to avoid volume depletion.[2] NPWT should not be used in active wound infections with excessive necrotic tissues. Instead, wound debridement should be undertaken until diminished or controlled infection before NPWT can be applied to the wound. In addition, a shorter cycle of NPWT application should be used in this wound.[8]

Epithelial Advancement

One of the key indicators of a healing wound is the progression of the wound edge in terms of epidermal cell (keratinocyte) migration and wound contraction. In a chronic diabetic foot ulcer, especially in a neuropathic ulcer, the presence of a thick callus or hyperkeratosis at the periphery of the wound will be an obstacle for keratinocyte migration and hence prevent epithelialization. At the same time, it is difficult to determine the true status of the ulcer edges, as the callus will obscure a full clinical assessment. If the patient continuously ambulates with the affected foot, pressure necrosis may develop under the callus, thus aggravating the ulcer. These adverse environments should be removed by proper debridement of all callus, slough, necrotic

tissue, nonviable cellular debris, and biofilm. Pressure redistribution in the diabetic foot is important, especially in the neuropathic patient. This redistribution can be achieved by specialized or customized, prescribed footwear or with the aid of walking crutches, frame, or wheelchair to offload pressure in the foot.[8]

Infection Management

Diabetic foot infections are difficult to manage due to the associated comorbidities affecting the patient such as neuropathy, peripheral vascular disease, immunopathy and nephropathy. Organisms such as methicillin resistant strains of Staphylococcus aureus, among others, pose a challenge to healthcare providers. Several factors such as prolonged hospital stays, exposure to surfaces and personnel who may have come into contact with resistant strains, and prolonged or prior antibiotic treatment can result in infections with these organisms.[7] It is important to control or restore microbial balance in the wound to a state that would not interfere with wound healing. If bacterial colonization is suspected to affect the progression of wound healing, or wound infection is clinically suspected, local therapy using a range of antimicrobial preparation or dressings should be initiated. Deep wound swabs and/or tissue cultures should also be taken for culture and sensitivities. Infections in diabetic foot ulcers are commonly polymicrobial and contain both aerobic and anaerobic bacteria. Slow-release silver dressings have gained in popularity due to their efficacy, low resistance and broad-spectrum antimicrobial actions, and effectiveness against Staphylococcus aureus, including methicillin-resistant Staphylococcus aureus (MRSA) and pseudomonas. Topical antiseptics such as aqueous chlorhexidine 0.5% and slow-release iodine have low tissue toxicity and broad-spectrum antimicrobial coverage. Normal saline and chlorhexidine are suitable for most wounds as cleansing or irrigating agents due to lower toxicity to the growing new tissues. Povidone may only be considered in grossly contaminated wounds. Acetic acid may be used for pseudomonas infections. Otherwise, toxic antiseptics such as povidone, acetic acid, or hydrogen peroxide should be avoided, as they are toxic to growing dermal and epidermal cells. Wound debridement or irrigation should be performed in the presence of necrotic or sloughy tissue to reduce bacteria loads and to disrupt the biofilms that protect the bacterial from antimicrobials. Systemic antibiotics are only indicated for active wound infections, ascending cellulitis, lymphangitis, osteomyelitis, or evidence of sepsis.[8]

Antibiotics are selected largely based on the probable causative organisms, taking into account any known local antibiotic resistance patterns. Patients with severe infections need parenteral treatment, at least initially; oral therapy is often adequate for those with mild or moderate infections.

Topical antimicrobials are often effective for mildly infected ulcers, however, some topical antiseptics can impair wound healing, but dressings containing silver or iodine seem to be safe, and possibly useful.

The aim of antimicrobial therapy is to cure the infection, not to heal the wound; extended treatment increases the risk of drug-related toxic effects and development of

antibiotic resistance. Antibiotic treatment without off-loading a plantar wound (i.e., the relieving of a mechanical load) is unlikely to result in ulcer healing.[3]

Surgical Revascularization

Patients with peripheral ischemia who have significant functional disability should undergo surgical revascularization if medical management fails. This may decrease the amputation risk in patients with ischemic DFUs. The procedures include open (bypass grafting or endarterectomy) or endovascular techniques (angioplasty with or without stent).[1]

It is imperative that flow-limiting arterial lesions be evaluated and reconstructed or bypassed. In general, the optimal strategy is to perform revascularization, if indicated, as soon as possible. Closure of the ulcer by primary healing or secondary reconstructive surgery will then be expedited. If revascularization of an ischemic ulcer is not possible for medical or technical reasons, amputation of the foot or limb will most likely result.

The long-held gold standard in vascular surgery for lower extremity revascularization procedures is the performance of an arterial bypass with autologous saphenous vein graft. Endovascular angioplasty stenting provides a less invasive alternative in indicated patients. Specifically, patients with short-segment disease in proximal locations, such as isolated iliac artery stenoses, are prime candidates for angioplasty and endovascular stent placement.

Contraindications to revascularization include nonambulatory patients and a foot with sepsis or excessive foot gangrene, precluding a functional foot despite adjunctive plastic surgical procedures such as skin grafts and free flaps.[2]

Wound Closure

After bacterial contamination has been controlled, small ulcers can usually be excised and closed immediately. Plastic surgical repair of these wounds can help avoid the production of inelastic scar tissue over weight-bearing surfaces.

Larger ulcers present a greater challenge. Simple closure of these wounds is often difficult because of preexisting bone deformity, tissue inelasticity, location of the defect, and superimposed osteomyelitis. Traditionally, split-thickness skin grafts (STSGs) are used to cover large areas of skin loss, granulating tissue beds, and tissue loss across joints in areas where contraction will cause deformity and where epithelialization alone will produce an unstable wound cover. STSGs currently represent the most rapid, effective method of reconstructing large skin defects and are preferred over full thickness grafts.[2]

DFUs with exposed tendon, ligament or bone require coverage with muscle flaps. Flaps can be either local (for smaller wounds) or free- flaps (for large area). Latissimus dorsi,

gracilis or rectus abdominis are the commonly used free flaps. The limitations of standard flaps include donor site morbidity, difficulty in shaping the flaps and interference with footwear.[1]

Off-Loading

There is no doubt that one of the most important parts of DFU treatments or prevention plans should be off-loading, meaning pressure relief on ulcer.[3] The aim is to reduce the plantar pressure by redistributing it to a larger area, to avoid shear and friction, and to accommodate the deformities.[1] High plantar pressure is usually caused by bony deformity or displacement of soft tissues, and may lead to ulceration and failure to heal. Ulcers can also be caused by contact between the dorsal surface of deformed toes and footwear that does not provide adequate toe room.[3] Total contact casting (TCC) is considered the best method of offloading as compared to a removable walking cast. Total casts should be properly made and changed at least weekly. However, when a total contact cast is unavailable or contraindicated, placing the patient in a wedge-type shoe or a walking boot, using flexible and rigid casting tape, complete bed rest or by using felt aperture padding have also been noted to reduce healing times.[7] A modified half-shoe can help off-load pressure from half of the foot. Felted-foam, soft polymeric insoles and orthoses with load-isolation regions are also used to smoothen the inner layers of shoes. For interdigital lesions, the close or overlapping toes must be separated. Ulcers on the plantar aspect of the heel take longer to heal than those on the forefoot in total contact casts and could benefit from special shoes without a rear-foot platform.[3]

DFU patients are encouraged to reduce their activity levels temporarily. Patients are typically less active in total contact casts than in healing shoes, presumably because of the bulk and weight of the irremovable device. Increased activity, with the consequent high cumulative load, can delay or prevent ulcer healing.[3]

If the conservative offloading attempts are ineffective, a surgical release (plantar fascial ligament resection) or surgical lengthening (tendoachilles lengthening) of the contracture may allow the forefoot to be more flexible when met with ground reactive forces thus healing the diabetic plantar foot ulcer. Surgical resection of the underlying bony prominence, termed internal off-weighting, is also an option. This surgical treatment may entail bony procedures such as an exostectomy, condylectomy, arthroplasty, metatarsal osteotomy or arthrodesis. These types of surgical procedures should only be performed by those with specific expertise in surgical reconstruction of the diabetic foot and ankle.[7]

Alternative and Adjunctive Therapies

Even when properly managed, the wounds may not heal in a timely fashion. Foot ulcers that do not heal in an expedient amount of time are expected to be more likely to become complicated by intervening infection, hospitalization, and amputation and, thus,

to be more costly because of the increased utilization of healthcare resources. Therapists generally rely on good clinical judgment and personal experience in deciding when to use more aggressive or more expensive technologies and interventions.

Many agents have been suggested to be used as adjuvants, to aid healing, in the treatment of diabetic ulcers. These therapies include topical agents for application to the wound bed (e.g., Recombinant PDGF, Regranex), systemic therapies (hyperbaric oxygen) to treat the patient, and skin substitutes (e.g., Apligraf, Dermagraft). These agents have shown promising results and have proven useful under specific circumstances.

Other investigational adjuvant therapies for diabetic foot include electrical stimulation of the ulcer bed, therapeutic ultrasound, application of electromagnetic fields, and therapeutic heat.[2]

Hyperbaric Oxygen Therapy

For over 50 years, Hyperbaric Oxygen Therapy (HBOT) has been a method applied to selected, serious cases of non-healing, and infection-complicated DFU resistant to other therapeutic methods. Application of the method was initially based on theoretical assumptions and then on experimental research.[4]

Hyperbaric Oxygen Treatment (HBOT) is most often used for diabetic wounds that have failed to resolve after a 30-day course of standard treatment. It is the delivery of pure oxygen to patients at higher than normal atmospheric pressures to compensate for decreased blood oxygen due to vascular impairment. The usual pressure for treating DFU is 1.4 to 3 atmospheres absolute pressure (ATA) - with an optimum of 2 ATA - for a compression time of 60 to 120 minutes - with an optimum of 90 minutes - during a course of multiple treatments.[3]

The precise mechanism of action of HBOT in DFU healing has not been uncovered yet. It has been theorized that increasing oxygen levels under pressure results in an increase in the concentration of oxygen in the blood and an increase in the diffusion capacity to the tissues, which, in turn, stimulates neovascularization and fibroblast replication and increases phagocytosis and leukocyte- mediated killing of bacterial pathogens in the wound. Many benefits of increasing the tissue oxygen have been claimed[3]:

- modulation of the production of nitric oxide
- promotion of cellular proliferation
- stimulation of capillary budding
- alteration of ischemic effect
- modification of the effect of growth factors and cytokines
- acceleration of collagen deposition
- reduction of edema
- modulation of the immune system response

- accelerated microbial oxidative killing
- enhancement of oxygen radical scavengers, thereby reducing ischemia reperfusion injury

HBOT-related complications are rare and involve claustrophobia, ear, sinus, or lung damage due to the pressure, temporary worsening of short sightedness, and oxygen poisoning.[4]

Maggot Therapy (MT)

Successful DFU therapy largely depends on regular wound debridement and creation of favorable humid conditions free from bacterial infection. Physical-mechanic means are the simplest ones to be used for wound debridement. During a surgical procedure, necrotic tissue, fibrin, and pathological granulation are removed with a scalpel, scissors, and a scraper (surgical debridement). However, the method is associated with a risk of intense bleeding; it is painful and imprecise. Debridement often extends beyond the necessary boundary, as it is difficult to separate and differentiate necrotic tissue, granulation, or poorly perfused tissue from a healthy one. That is particularly important in case of DFU, where debridement is a common procedure and the wound's area is small. Therefore, other, superior and more efficient methods of local therapy are sought.

Since the beginning of the 21st century, MT, also known as maggot debridement therapy (MDT), biosurgical debridement (BD) with maggots, or simply larval therapy, has been in the renaissance.

Maggots secrete digestive juices on the outside; necrotic tissue becomes digested and liquefied and absorbed in that form. Additionally, an additional mechanical debridement is caused by the specific mandibles or "mouth hooks" of the maggots and their rough body which both scratch the necrotic tissue. The therapeutic effect of MT is based in three mechanisms:

1. removal of necrotic tissue from the wound
2. antibacterial effect and destruction of bacterial biofilm
3. stimulation of healing processes.

The effect of wound healing stimulation is attributed to allantoin and urea in the maggot excretions and secretions. Ammonia, ammonium bicarbonate, and calcium carbonate change medium reaction from acidic into alkaline, inhibiting bacterial growth. Also, tissue irritation by moving maggots also speeds the process of wound healing up. Favorable effect on DFU healing is also associated with MT influence on disturbed mechanisms of the inflammatory process. Maggots remove necrotic tissue and do not digest bones, tendons, or viable tissues. They offer a precise, accurate, and delicate debridement. It seems that the level of wound debridement is not achievable with surgical methods. Maggots also clean off microorganisms.

MT is less effective in cases of highly discharging or dried wounds. Insignificant side

effects of the therapy include minor bleeding, pain, excessively induced exudates, increased body temperature, flu-like symptoms, allergic reaction, and skin maceration. There are practically no contraindications for MT in DFU therapy. Patient anxiety may constitute a limitation. *Lucilia sericata* maggot cultures are kept on sterile media, with sterile air flow, ensuring aseptic conditions. 1–3 mm long maggots are used for dressings. They are provided in two forms: open, for direct application on wound, or closed in a biobag. The network of the biobag is permeable and permits the migration of maggot ES to the wound. A wound qualified for MT requires no special preparation; edges of the wound are covered with an ointment protecting against digestive enzymes. Applied maggots, approximately 5–10 for one centimeter squared of the wound, may be covered by a nylon net, ensuring they remain in the wound area, and a humid dressing. A dry dressing is applied on top. As the outer dressing gradually becomes soaked, it should be changed. The dressing with maggots is maintained for 3 days, on average. After that period, the maggots should be removed by washing out the wound by saline. The procedure is repeated 1–4 times, if necessary. Closed dressings are more comfortable to use, because maggots cannot get out; however, they often cannot be applied on deep DFU with small, irregular area.[4]

Autologous Platelet-Rich Plasma

Making effective wound healing possible is the basic stage of the DFU curing process. Use of autologous platelet-rich plasma (PRP) in the form of local application of a gel obtained by centrifugation of full blood and addition of an activator, clotting agent, is designed for the creation of local conditions favorable to healing processes. PRP is defined as plasma fraction of autologous blood with a platelet count concentrated above the baseline. It is a repository of growth factors, cytokines, adhesion molecules and clotting agents, and leukocytes.

Platelets contain numerous natural growth factors released from their α granulations and stimulating healing processes. PRP is obtained by repeated centrifugation of autologous full blood. The resulting concentrate, combined with activating bovine thrombin, forms a gel that seals the wound. The gel is placed on wound bed and protected by a cover dressing. The dressing may stay in place for up to 7 days.

Platelets attach to the connective tissue, and growth factors are released via degranulation of α granulation in just 10 minutes after initiation of blood clotting processes. The majority of them is released during the first hour and is bound to membranous receptors in surrounding cells, activating intracellular signaling pathways. Following a rapid release of growth factors, platelets contained in PRP synthesize and secrete their additional quantities for subsequent 7 days. After that time, the healing function is taken over by macrophages.

PRPT is a rich source of locally active growth factors and cytokines that improve conditions of wound healing. Relatively simple and cheap production of PRP argues for continued interest in that adjunct method. It seems that specific cellular therapy

constitutes an additional and valuable option in therapy of DFU resistant to the conventional therapy.[4]

Phototherapy

Phototherapy, also known as photobiomodulation, low-level laser therapy (LLLT), involves the application of light (often laser light of a specific wavelength or a light emitting diode, LED) to stimulate cellular processes.

The effects of phototherapy are chemical and not thermal. Energy which is delivered to cells produces insignificant and minimal temperature changes, typically in the range of 0.1–0.5°C. Cellular responses are the result of changes in photoacceptor molecules, or chromophores. Photoacceptors take part in cellular metabolism and are not connected to a light response, such as chlorophyll which is a photoreceptor. Once the photon energy is absorbed, the photoacceptor assumes an electronically excited state, which in turn stimulates cellular metabolism by activating or deactivating enzymes which alter other macromolecules such as DNA and RNA. The energy which is absorbed by the photoacceptor can be transferred to other molecules causing chemical reactions in the surrounding tissue; this then gives rise to observable effects at a biological level. Photon energy is absorbed by the chromophores and there is an increase in adenosine triphosphate (ATP) and cell membrane permeability, which leads to activation of secondary messengers which in turn activate a cascade of intracellular signals. There is also an increase in mitochondrial membrane potential and proton gradient.[9]

Stem Cell Therapy

Stem cell therapy has emerged as a promising treatment modality aiming to address the underlying pathophysiology of DFU. Stem cells secrete chemokines and growth factors (especially EGF, VEGF and fibronectin), which promote angiogenesis and ECM remodeling to mobilize wound healing. Stem cells that have been studied for wound healing can be classified in two groups of allogenic and autologous, based on where their origins are. Placental or amnion-derived mesenchymal stem cells and embryonic stem cells are categorized as allogenic stem cells. On the other hand, bone marrow-derived endothelial progenitor cells, bone marrow-derived mesenchymal stem cells, hematopoietic stem cells, and mesenchymal stem cells derived from adipose tissue are the autologous stem cells. Placenta-derived MSCs are shown to be more effective in chronic wound healing. Also, isolated ESC- derived EPCs were shown to improve re-epithelialization when injected subcutaneously into or applied topically on to the wound.

Hematopoietic stems cells (HSCs), harvested from either bone marrow or peripheral blood, are shown to enhance wounds healing in both the inflammatory and proliferative phases of DFU.[3]

Amputation

Despite all efforts to treat ischemic and neuropathic ulcers, sometimes the lower-extremity is non-viable and amputation is inevitable. Non-traumatic amputations are high in poorer countries and in uneducated people in wealthy countries.

The indications for amputation in patients with diabetes are often multiple, mostly a non-healing ulcer, or frequent gangrene and infection occurring simultaneously. Whether primary minor amputation is beneficial in comparison with primary major amputation (below knee) is still controversial. Digital toe amputation eventually leads to limb loss, while a major amputation lowers the risk of re-amputation. Re-amputation is greatest in the first 6 to 12 months after the first amputation. Once hallux has gone under amputation, changes in mechanical force and pressure on the foot may increase the likelihood of developing further lesions, most probably within 6 months.[3]

Determining the level of amputation requires the trade-offs between vascularity and limb length. As a general principle, it is imperative to save as much limb length as possible. Clinical examination, ABI and transcutaneous oxygen measurements (before and after inhalation of oxygen) can be used to decide the level of amputation, but of these transcutaneous oxygen measurements are preferred.

The commonly performed amputations for ischemic DFUs include toe, Ray, transmetatarsal, tarsometatarsal (Lisfranc), midtarsal (Chopart), hindfoot and ankle (Pirogoff, Boyd, Syme's) and trans-tibial.[1]

Toe Amputations

Digital amputations are the most common amputations performed in the foot. Amputation of the great toe greatly reduces the thrust force during the gait, where the hallux with the flexor hallucis longus and flexor brevis play a fundamental role, with possible metatarsalgia of the 2nd and 3rd rays. The amputation of a central toe (2nd, 3rd or 4th) causes forefoot instability, not only because of the digitigrade contribution during the gait, but especially because it can cause deformities of the adjacent toes. For example, the space created after the amputation of the 2nd and/or 3rd toe, can create or worse a hallux valgus. The isolated amputation of the fourth toe, leaving in place the fifth, can also cause traumas and sub-dislocations of the latter.[10]

Ray Amputations

Amputation of the first ray causes, in addition to a deficiency in the boost phase of the step and the loss of plantar-flexion of the toe, a collapse of the medial column with a possible evolution towards a pronated and valgus foot. It may also lead to ulcerative lesions or stress fractures of the other rays.

It is important to keep the insertion of the anterior tibial tendon and peroneus longus tendon on the metatarsus, leaving a functionally valid foot, if necessary with the use of an orthotic prosthesis.

The amputation of a central ray is functionally more effective because it only slightly reduces the latero-medial diameter of the forefoot, without biomechanical deficits.

The amputation of the fifth ray can cause a varus, adducted and supinated foot, because of the prevalence of the posterior tibial tendon, with possible ulcerative lesions or stress fractures.

The amputation of the first two rays amplifies the problems caused by the amputation of the first isolated, increasing the probability of ulceration due to the overload on the lateral columns, creating difficulty in wearing a prosthesis. The risk of a subsequent more proximal amputation becomes higher.

Amputations of the last two rays: in this case there is the risk of developing an adduct, varus and supinated foot, but the distribution of the load is more balanced, because of the preservation of the 1st, 2nd and 3rd ray, increasing the possibility of using orthoses, reducing the risk of ulceration and a subsequent more proximal amputation. The amputation of two central rays creates a foot reduced in its latero-medial diameter, but functionally valid and biomechanically balanced.[10]

Transmetatarsal Amputation

Transmetatarsal amputation (TMA) is typically performed in patients with chronic osteomyelitis involving the forefoot, gangrene of the toes, or a non-healing ulceration with a previously resected first-ray. As mentioned earlier, a first-ray resection alters normal gait characteristics and patients are at higher risk for developing transfer lesions, putting them at risk for subsequent amputation. The resection of multiple central rays results in a nonfunctional foot and a TMA is indicated to prevent multiple amputations.

The amputation must be made at the bases of all the five metatarsals, leaving intact the areas of attachments of tibialis anterior and peroneus brevis on the first and fifth metatarsals. When appropriately balanced, the TMA can provide a functional foot. The peroneus brevis to peroneus longus transfer can maintain a plantigrade foot.

A rehabilitation program emphasizing protection of the residual foot for at least 3 months following surgery should be implemented to avoid complications. Patients are placed in a compressive type medication with a posterior splint and walking is allowed only with crutches without weight-bearing. However, patients may however require the use of an ankle and foot orthosis, with a forefoot filler. The brace may be worn in extra-depth shoes.[10]

Lisfranc Amputation

A Lisfranc disarticulation should be considered when there is inadequate soft tissue coverage for a TMA. Compared to a TMA, this more proximal amputation will result in a more pronounced muscle imbalance and deformity.

Postoperative management is similar to that described for the TMA and, initially, is

concentrated on protecting the residual foot and later may require the use of an ankle foot orthosis.[10]

Chopart's Amputation
The Chopart's amputation is a disarticulation through the midtarsal joint, leaving only the talus and calcaneus. Patients requiring a Chopart's amputation often present with infection extending proximally to the midfoot.

Compared to below-knee or Syme's amputations, it has some advantages: it is possible to use a shoe with a specific filler, avoiding a leg prosthesis, as for the other two amputations, it does not cause limb shortening. A bearing surface formed by the distal talus and the calcaneus is possible, and a skin cover at the calcaneus is present for a good stance.

The early postoperative care of the more proximal foot-sparing amputations focuses on protecting the residual foot. Patients are non-weight-bearing. Generally, after allowing for soft tissue healing, patients are transitioned to a removable cast walker. The need for bracing is the same as the need for a prosthesis with a below-knee amputation. Long-term management and choice of prosthetic will depend on the activity level of the patient.[10]

Syme's Amputation
A Syme's amputation involves a disarticulation of the ankle joint. This amputation preserves function of the knee with a long stump and independence by allowing patients to expend less energy walking than patients with higher-level amputations. The original technique included disarticulation of the foot at the ankle joint with resection of the malleolar projections.

Generally, patients are transitioned to a walking cast after 3 weeks and are fitted for a custom prosthetic when all wounds are healed and edema is controlled.[10]

Partial Calcanectomy
In cases of forefoot and midfoot pathology necessitating amputation, the calcaneus is maintained after removing the talus, and fused to the distal tibia. This gives stability and maintains some length to the remaining limb, preserves the distal flap and plantar fat pad. A partial calcanectomy is an alternative to below-knee amputation for calcaneal osteomyelitis with overlying tissue loss. Once healed, most patients maintain ambulation and improved quality of life is achieved by preserving a functional limb.

Patients are generally kept non-weight-bearing for a period of 6 weeks and then fitted for accommodative footwear.[10]

Prevention

Diabetic ulcers of lower extremity are a chronic problem with significant recurrence rates. Therefore, long-term maintenance must be addressed even for healed ulcers. This includes identification of high-risk patients, education of the patient, and institution of measures to prevent ulceration. High-risk patients should be identified during the routine foot examination performed on all patients with DM. Patient education should emphasize careful selection of footwear, daily inspection of the feet to detect early signs of poor-fitting footwear or minor trauma, daily foot hygiene to keep the skin clean and moist, avoidance of self-treatment of foot abnormalities and high-risk behavior (e.g., walking barefoot), and prompt consultation with a health care provider if an abnormality arises. Any diabetic patient admitted to acute care setting should have their feet examined on admission. If it is judged that their feet are at risk of new ulceration, preventive steps should be taken immediately, which includes provision of a pressure mattress and suitable protective footwear. Those with new ulcers should be referred promptly to an expert multidisciplinary team for expert assessment and management.[2]

Patient education and self-care practices like maintaining foot hygiene and nail care should be promoted. Skin should be kept moisturized with the application of topical moisturizers after washing the feet gently with soap and water. Harsher measures like hot soaks, heating pads and topical agents such as hydrogen peroxide, iodine and astringents are better avoided.

There is direct correlation between glycemic control and ulcer formation. Smoking and alcohol consumption should be minimized. Offloading and appropriate footwear to relieve focal high pressure areas is recommended for foot at-risk. Other comorbidities like hypertension and hyperlipidaemia which predispose to vascular occlusion should be treated.[1]

Early detection of potential risk factors for ulceration can decrease the frequency of wound development. It is recommended that all patients with diabetes undergo a foot examination at least annually, to determine the predisposing conditions to ulceration. Patients should be educated regarding the importance of maintaining good glycemic control, wearing appropriate footwear, avoiding trauma, and performing frequent self-examinations.[6]

Prevention Summary[6]

1. Primary prevention: Screening of high risk feet and proper advice on preventive footwear
2. Secondary prevention: Management of trivial foot lesions such as callus removal, treatment of nail pathologies, blisters, and so on.
3. Tertiary prevention: Prompt referral to a specialist for advanced foot lesions.

Footwear

While the presence of peripheral neuropathy is the leading risk factor for diabetes related foot ulceration, a pivotal event, such as trauma from footwear, is also needed for most ulcers to occur. The purchase and wearing of appropriate footwear is therefore an important process for all individuals with diabetes, especially for those who demonstrate loss of protective sensation from peripheral neuropathy. This patient group, who are unable to feel pressure and/or pain caused by inappropriate or ill-fitting shoes are more likely to develop blisters, callus and corns. These early complications are pathological and are warning signs that require prompt intervention if ulceration and potential amputation are to be avoided. Health professionals involved in the care of people with diabetes need to define the individuals' level of risk for developing foot complications and thus tailor footwear advice accordingly. Risk stratification is determined following a basic foot assessment, which includes evaluation for the presence of peripheral neuropathy, peripheral arterial disease and foot deformity. In addition to the foot assessment, other factors that should to be considered include the individuals' activity levels, occupation and level of mobility. For example, individuals who are employed on a work site may require steel capped footwear in order to comply with industry standards. Risk stratification should be re-assessed and upgraded on a yearly basis, given the potential for progression and development of new risk factors over time.[11]

Low Risk

People with no risk factors for ulceration Individuals with no identifiable risk factors (no peripheral neuropathy, peripheral arterial disease, foot deformity or history of amputation) on foot assessment are at low risk for developing foot ulceration. A commonsense approach to footwear selection and use is advisable. Individuals can usually be safely accommodated in a wide range of off the shelf footwear, provided they are correctly fitted and appropriate for the activity to be undertaken.[11]

At Risk

People with peripheral neuropathy and/or peripheral arterial disease Individuals with peripheral neuropathy and/or peripheral arterial disease on foot assessment are 'at risk' of developing foot ulceration. Correctly fitted footwear, especially with regards to length and width, is essential. This is especially important for individuals with peripheral neuropathy who may have a tendency to purchase poorly fitting shoes in order to stimulate some sensory feedback. The majority of individuals in this category should be able to be accommodated in off the shelf footwear. The criteria for selecting appropriate footwear should again be applied and individuals should be advised to have themselves fitted in the afternoon in order to accommodate any dependent edema. New shoes should be worn in, with wearing time gradually increased over a 1 to 2 week period. When shoes are removed, the individual should check their feet for signs of pressure, trauma and ulceration. Footwear should also be checked daily, inspecting for signs of wear and tear, and ensuring that there is no foreign object within the shoe or penetrating through the sole.[11]

High Risk

People with abnormal foot shape or history of amputation Individuals assessed as having an abnormal foot shape, including previous amputation, are at high risk of developing foot ulceration. The severity of the foot deformity will directly influence footwear prescription but in general the requirement will be for medical grade footwear and custom molded foot orthoses. Custom-made medical grade footwear and orthoses should be considered for individuals with severe deformities and additional modifications, such as rocker soles (for pressure relief) may be indicated. Medical grade footwear and custom molded foot orthoses should be reviewed for wear and be replaced as required. A referral to a health professional with the knowledge and skills for prescribing appropriate footwear is recommended. Individuals with a previous foot ulcer are at high risk of developing further ulcers in the future. Even though a past ulcer does not define the footwear needs of the individual, it should be seen as an alert that leads the individual to being prioritized in acquiring prompt access to appropriate footwear.[11]

Shoe Features

Shoe features that clients should be aware of when purchasing footwear[11]:

- Uppers - These should be made from leather or a combination of materials (such as those used in sports shoes) with smooth inner lining and no bulky seams at the toe area.
- Correct length - 1 cm from end of longest toe when client is standing.
- Correct depth - Accommodate the toes without causing pressure.
- Correct width - The sides of the shoe should not bulge over the last (sole) when worn.
- Low heels - Less or equal to 2 cm.
- Fastening - Adequate fastening such as laces or Velcro to keep the foot from sliding forward.
- Cushioned outer and inner soles - Approximately 0.5-1 cm thick under the forefoot.
- Enclosed heel - Open backed shoes can result in injury to the skin around the heel and usually require the individual to claw their toes in order to keep them on, also increasing risk of ulceration.
- Soles - Non slip

Supplemental Reading

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Diabetic Foot Ulcers

Post-Test

1. Peripheral neuropathy effects the _____ nervous system.
   A. Sensory
   B. Motor
   C. Autonomic
   D. All of the above

2. Midfoot collapse or “rocker-bottom” foot is most commonly associated with ____.
   A. Charcot arthropathy
   B. equinus deformation
   C. hallux rigidus
   D. pes cavus

3. Which of the following is NOT an identified risk factor for diabetic foot ulcer?
   A. Callus formation
   B. Systolic hypotension
   C. Smoking
   D. Impaired vision

4. Which type of ulcer is painful, has minimal bleeding, and is typically located on the tips or between toes?
   A. Ischemic
   B. Neuropathic
   C. Peptic
   D. Autonomic

5. Which wound classification system categorizes wounds based on grade (depth of lesion) and stage (presence of infection or ischemia)?
   A. Wagner-Meggitt Classification
   B. The University of Texas Wound Classification
   C. PEDIS
   D. DEPA Scoring System

6. Which of the following is NOT normally utilized as part of a typical diabetic foot ulcer evaluation?
   A. Thermometer and blood pressure cuff (sphygmomanometer)
   B. Sterile stainless steel probe
   C. A 5.07 Semmes-Weinstein monofilament
   D. Bornstein densiometer

7. Which of the following is toxic to growing dermal and epidermal cells and should be avoided?
   A. Hydroactive hypocolloid gel
   B. Silver sulfadiazine
   C. Antiseptics such as povidone, acetic acid, and hydrogen peroxide
   D. All of the above

8. Which of the following is correct concerning alternative and adjunctive therapies?
   A. Hyperbaric oxygen therapy injects 4-5 atmospheres of oxygen directly into the wound and may cause a pulmonary embolism.
   B. The primary risk of maggot therapy is that they frequently digest bone, tendon, and other viable tissue.
   C. The effects of phototherapy are chemical and not thermal.
   D. Autologous platelet rich plasma is infused intravenously to help fight against infection.

9. The _____ amputation is a disarticulation through the midtarsal joint, leaving only the talus and calcaneus.
   A. Lisfranc
   B. Chopart's
   C. Syme's
   D. Modified Taylor

10. The correct shoe length for an individual with diabetes is _____.
    A. 5 mm from the end of the great toe in nonweight bearing
    B. 5 cm from the end of the great toe in when standing
    C. 1 cm from the end of the longest toe when standing
    D. 1 inch from the end of the longest toe in nonweight bearing

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