# REVIEW ARTICLE

## A REVIEW ARTICLE ON DIABETIC FOOT

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**INTRODUCTION:** Foot infections are the most common problems in patients with diabetes. These individuals are predisposed to foot infections because of a compromised vascular supply secondary to diabetes. Local trauma and/or pressure (often in association with lack of sensation because of neuropathy), in addition to microvascular disease, may result in various diabetic foot infections that run the spectrum from simple, superficial cellulitis to chronic osteomyelitis. Globally, diabetic foot infections are the most common skeletal and soft-tissue infections in patients with diabetes. The incidence of diabetic foot infections is similar to that of diabetes in various ethnic groups and most frequently affect elderly patients. There are no significant differences between the sexes. The mortality risk is highest in patients with chronic osteomyelitis and in those with acute necrotizing soft-tissue infections.

### PRECIPITATING CAUSES OF FOOT ULCERATION AND INFECTION:
- Friction in ill-fitting or new shoes.
- Untreated callus.
- Self-treated callus.
- Foot injuries (unnoticed trauma in shoes or when walking barefoot).
- Burns (for example, excessively hot bath, hot water bottle, hot radiators, hot sand on holiday).
- Corn plaster.
- Paronychia.
- Artefactual (self-inflicted foot lesions are rare; occasionally failure to heal is due to this cause).
- Heel friction in patients confined to bed.
- Foot deformities (callus, clawed toes, bunions, pes cavus, hallux rigidus, hammer toe, Charcot's foot, deformities from previous trauma or surgery, nail deformities, edema)

**NEUROPATHY:** More than 60% of diabetic foot ulcers are the result of underlying neuropathy.[1,2] The development of neuropathy in affected patients has been shown in animal and in vitro models to be a result of hyperglycemia-induced metabolic abnormalities.[3,4,5] One of the more commonly described mechanisms of action is the polyol pathway.[4] The hyperglycemic state leads to an increase in action of the enzymes aldose reductase and sorbitol dehydrogenase. This results in the conversion of intracellular glucose to sorbitol and fructose.

The accumulation of these sugar products results in a decrease in the synthesis of nerve cell myoinositol, required for normal neuron conduction. Chemical conversion of glucose results in

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a depletion of nicotinamide adenine dinucleotide phosphate (NADP) stores, which are necessary for the detoxification of reactive oxygen species and for the synthesis of the vasodilator nitric oxide. There is a resultant increase in oxidative stress on the nerve cell and an increase in vasoconstriction leading to ischemia, which will promote nerve cell injury and death. Hyperglycemia and oxidative stress also contribute to the abnormal glycation of nerve cell proteins and the inappropriate activation of protein kinase C, resulting in further nerve dysfunction and ischemia.

Neuropathy in diabetic patients is manifested in the motor, autonomic, and sensory components of the nervous system[1] Damage to the innervations of the intrinsic foot muscles leads to an imbalance between flexion and extension of the affected foot. This produces anatomic foot deformities that create abnormal bony prominences and pressure points, which gradually cause skin breakdown and ulceration. Autonomic neuropathy causes the overlying skin to become dry and increasingly susceptible to tears and subsequently to infections.The loss of sensation as a part of peripheral neuropathy exacerbates the development of ulcerations. As trauma occurs at the affected site, patients are often unable to detect the insult to their lower extremities. As a result, many wounds go unnoticed and progressively worsen as the affected area is continuously subjected to repetitive pressure and shear forces from ambulation and weight bearing.

**VASCULAR DISEASE:** Peripheral arterial disease (PAD) is a contributing factor to the development of foot ulcers in up to 50% of cases.[6,7] It commonly affects the tibial and peroneal arteries of the calf. 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. Further, the hyperglycemia causes an increase in thromboxane A2, a vasoconstrictor and platelet aggregation agonist, which leads to an increased risk for plasma hypercoagulability. Moreover, smoking, hypertension, and hyperlipidemia are other factors that are common in diabetic patients and contribute to the development of PAD.[5] cumulatively, this leads to occlusive arterial disease that results in ischemia in the lower extremity and an increased risk of ulceration in diabetic patients.

Impaired host defenses secondary to hyperglycemia cause defects in leukocyte function and morphologic changes to macrophages. Bagdade et al. demonstrated that leukocyte phagocytosis was significantly reduced in patients with poorly controlled diabetes, and improvement of microbiocidal rates was directly correlated with correction of hyperglycemia. Decreased chemotaxis of growth factors and cytokines, coupled with excess of metalloproteinases, impede normal wound healing by creating a prolonged inflammatory state. Fasting hyperglycemia and the presence of an open wound create a catabolic state. Negative nitrogen balance ensues secondary to insulin deprivation, caused by gluconeogenesis from protein breakdown. This metabolic dysfunction impairs the synthesis of proteins, fibroblasts and collagen, and further systemic deficiencies are propagated which lead to nutritional compromise. Research indicates impairment of the immune system with serum glucose levels ≥150 ml/dl. Patients with diabetes tolerate infection poorly and infection adversely affects diabetic control. This repetitive cycle leads to uncontrolled hyperglycemia, further affecting the host's response to infection.

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**ASSESSMENT OF DIABETIC FOOT ULCERS:**

**HISTORY:** A complete history should be obtained, including duration of disease, insulin dependence, previous complications or ulcerations and assessment of recent glycemic control.[7]Past medical history should focus upon related complications or comorbidities such as renal, liver, cardiovascular disease, neuropathy and retinopathy. A current medication list should be obtained, including past or current antibiotic use. Social history must not be overlooked, including use of tobacco or alcohol, quantity of weight-bearing and ambulation level, diet and exercise and home support network. Finally, patients should be questioned regarding smoking because smoking is linked to the development of neuropathic and vascular disease. History that takes into consideration previous ulceration or amputation. The history should also include any neuropathic symptoms or symptoms that are suggestive of peripheral vascular disease.

**CLINICAL EXAMINATION:** Visual inspection of the bare foot should be performed in a well-lit room. The examination should include an assessment of the shoes; inappropriate footwear can be a contributing factor to the development of foot ulceration. Visual inspection of the foot, should include checking between the toes for the presence of ulceration or signs of infection. The presence of callus or nail abnormalities should be noted. Additionally, a temperature difference between feet is suggestive of vascular disease.

The foot should also be examined for deformities. Hyperextension of the metatarsal-phalangeal joint with interphalangeal or distal phalangeal joint flexion leads to hammer toe and claw toe deformities, respectively. The Charcot arthropathy is another commonly mentioned deformity found in some affected diabetic patients. It is the result of a combination of motor, autonomic, and sensory neuropathies in which there is muscle and joint laxity that lead to changes in the arches of the foot. Further, the autonomic denervation leads to bone demineralization via the impairment of vascular smooth muscle, which leads to an increase in blood flow to the bone with a consequential osteolysis.

Detailed wound descriptions such as length, width and depth of the wound, colour and consistency of drainage, and character of wound base (granular, fibrous or necrotic) should be documented.

In examining for vascular abnormalities of the foot, the dorsalis pedis and posterior tibial pulses should be palpated and characterized as present or absent. Local signs of infection may include pain/tenderness, erythema, edema, purulent drainage and new-onset malodour. Systemic signs of infection include anorexia, nausea, vomiting, fever, chills, night sweats, change in mental status and a recent worsening of glycemic control. However, patients with DM may have an impaired neuroinflammatory response and not manifest typical signs of infection.

Claudication, loss of hair, and the presence of pale, thin, shiny, or cool skin are physical findings suggestive of potential ischemia. If vascular disease is a concern, measuring the ankle brachial index (ABI) can be used in the outpatient setting for determining the extent of vascular disease and need for referral to a vascular specialist. ABI below 0.91 are suggestive of obstruction. However, in patients with calcified, poorly compressible vessels or aortoiliac stenosis, the results of the ABI can be complicated.[8] If there is a strong suspicion of vascular disease, the patient should undergo vascular imaging as an alternate method of testing to determine the extent of disease and possible ischemia.

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Plain film radiography is important for the initial assessment of infection of soft tissue and osseous structures, deformity and foreign bodies. Soft tissue emphysema represents an emergent situation and must be treated immediately. Osteomyelitis appears as permeative radiolucencies, periosteal reaction and destructive changes on plain films following 30–50% loss of bone mineralization. Plain films are considered 67% specific and 60% sensitive for Osteomyelitis. Magnetic resonance imaging (MRI) is the most specific and sensitive non-invasive test to evaluate Osteomyelitis and is also useful for the evaluation of a probable abscess or sinus tract formation. Bone scans, such as the white blood cell labeled Indium-111, Technetium-99m HMPAO and Sulfur Colloid Marrow Scan, may prove beneficial between distinguishing acute and chronic infections, with the latter useful for identifying Osteomyelitis from Charcot neuroarthropathy by specifying bone marrow reactivation and neutrophil production.

Laboratory values are essential to establish a baseline and assess on the response to treatment.[9] Armstrong et al. found that fewer than 50% of DFI patients mounted an elevated white blood cell (WBC) in his study of 28 hospitalized DFI, with the mean WBC count being $11.9 \pm 5.4 \times 10^3$ cells/mm. A metabolic panel should also be ordered for the assessment of renal function, electrolytes, acidosis, and blood glucose level. Hemoglobin A1C levels provide a barometer of glycemic control averaged over the previous 2–3 months. Acute phase reactants, including erythrocyte sedimentation rate (ESR) and C-reactive protein level (CRP) are markers of inflammation that are elevated in response to inflammation, tissue injury and infections. Based on a study by Butalia et al,[10] an ESR >70 mm/hr significantly increases the probability of OM. Fleischer et al. concluded a CRP >3.2 mg/dl was a useful marker for differentiating OM from cellulitis. Hypoalbuminemia can result from decreased albumin production secondary to protein malnutrition, defective synthesis due to hepatocyte damage, deficient intake of essential amino acids, increased loss through inadequate GI and renal function and commonly through acute and chronic inflammatory states.

**CLASSIFICATION OF DIABETIC FOOT ULCERS:** If an ulcer is discovered, the description should include characteristics of the ulcer, including size, depth, appearance, and location. There are many classification systems used to depict ulcers that can aid in developing a standardized method of description.

One of the most popular systems of classification is the Wagner Ulcer Classification System, which is based on wound depth and the extent of tissue necrosis. Ddisadvantage of this system is that it only accounts for wound depth and appearance and does not consider the presence of ischemia or infection. The University of Texas system is another classification system that addresses ulcer depth and includes the presence of infection and ischemia. Wounds of increasing grade and stage are less likely to heal without vascular repair or amputation.

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| Table 1. Wagner Ulcer Classification System | |
|---|---|
| **Grade** | **Lesion** |
| 1 | Superficial diabetic ulcer |
| 2 | Ulcer extension involving ligament, tendon, joint capsule, or fascia with no abscess or osteomyelitis |
| 3 | Deep ulcer with abscess or osteomyelitis |
| 4 | Gangrene to portion of forefoot |
| 5 | Extensive gangrene of foot |

| Table 2. University of Texas Wound Classification System | |
|---|---|
| **Stages** | **Description** |
| Stage A | No infection or ischemia |
| Stage B | Infection present |
| Stage C | Ischemia present |
| Stage D | Infection and ischemia present |
| | |
| **Grading** | **Description** |
| Grade 0 | Epithelialized wound |
| Grade 1 | Superficial wound |
| Grade 2 | Wound penetrates to tendon or capsule |
| Grade 3 | Wound penetrates to bone or joint |

**TREATMENT MODALITIES:** The first principle isglycemic control, second to treat any infection; the third is to establish whether any associated ischaemia is amenable to revascularization; the fourth is to keep forces applied to the ulcerated part to a minimum; and the fifth is to improve the condition of the wound or ulcer by wound-bed preparation, topical applications, and removal of callus. Once the wound has healed, attention can be turned to the prevention of ulcer recurrence.

**Glycemic Control:** Measurement of glycosylated Hb is the standard method for assessing the long-term glycemic control.[11] Various studies show that there is almost a direct relationship of foot lesions with increasing Glycated Hb i.e. poorer blood sugar control. Patients who had an HbA1c level >10% manifested with various types of foot lesions. Each 2% increase in the HbA1c increases the risk of lower extremity ulcer by 1.6 times and the risk of lower extremity amputation by 1.5 times.

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**Eradication of infection:** The antibiotic regimen chosen should be based on the anticipated spectrum of infecting organisms. The combination of an aminopenicillin and a penicillinase inhibitor has the required activity, but other options include a quinolone plus either metronidazole or clindamycin. Intravenous options for soft-tissue infection include imipenem and gentamicin. Vancomycin, Teicoplanin, Rifampicin, or Linezolid should be used for methicillin-resistant S aureus (MRSA). Beta-lactams and quinolones are concentrated intracellularly at the site of infection, and clindamycin penetrates bones well. The parenteral route is preferred if the foot is severely ischaemic or in cases of systemic illness.

**Remediable macrovascular disease:** Clinical evidence and positive non-invasive tests for macrovascular disease, such as an ankle/brachial pressure index below 0·8, toe systolic pressure of less than 30 mm Hg, reduced transcutaneous oxygen tension, and abnormal duplex waveform on ultrasonography, indicate the need for assessment by a vascular surgical team. Options for revascularisation include angioplasty, thrombolysis, and bypass surgery. Distal bypass to the pedal vessels is increasingly common, though with regional variations. Despite a huge increase in revascularisation procedures in the past 20 years, the effect on the rate of major amputation has been disappointing. Transluminal angioplasty of the iliac arteries in conjunction with surgical bypass in the distal extremity may be implemented, and efficacy has been demonstrated in diabetic patients.

**Off-loading:** It is unrealistic to tell a patient to immobilise the foot for the time required for healing, and immobilisation carries the risk of thrombosis, muscle wasting, depression, and secondary ulceration elsewhere. Instead, custom-made orthotic devices and plaster or fibreglass casts are used to off-load the wound while allowing the patient to remain partly active. These devices can greatly lower plantar pressures, but patient compliance is poor. Off-loading devices might be impractical for patients who are frail or susceptible to falls, and a disadvantage of devices that cannot be removed is interference with bathing and showering.

**Ulcer Management:** Ulcers heal more quickly if their surface is clean and if sinuses are laid open. Vigorous and repeated sharp debridement of the wound is recommended. Complete excision of neuropathic ulcers did lead to healing in a mean of and 47days, as opposed to 129 days in non-randomised controls managed more conservatively. Necrotic material can also be removed with debriding agents (enzymes, hydrogels, and hydrocolloids). Larval therapy (maggots) to clean the wound bed, though not immediately appealing, does merit further study. Antiseptics containing iodine and silver have also been promoted. Attention has also focused on controlling edema.

There is considerable interest in the therapeutic potential of growth factors. Two trials have shown significant, but small, benefit from recombinant platelet-derived growth factor (becaplermin). G-CSF has been found to enhance the activity of neutrophils in diabetic patients.[12] G-CSF accelerated the resolution of infection in a pilot study, and results from a randomised trial suggested a reduction in amputation done for osteomyelitis that has yet to be substantiated. Some of the effects of allografts might result from their capacity to release growth factors, but

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promotion of angiogenesis might be another explanation. This approach is expensive, but can be used in selected patients.

The selection of wound dressings is also an important component of diabetic wound care management.The characteristics of specific dressing types can prove beneficial depending on the characteristics of the individual wound. 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. A complete discussion of the various classes of wound dressings is beyond the scope of this review; however, an ideal dressing should contribute to a moist wound environment, absorb excessive exudates, and not increase the risk for infections.

If infection is suspected in the wound, the selection of appropriate treatments should be based on the results of a wound culture. Tissue curettage from the base of the ulcer after debridement will reveal more accurate results than a superficial wound swab. In the case of deep tissue infections, specimens obtained aseptically during surgery provide optimal results.

Gram-positive cocci are typically the most common pathogens isolated. However, chronic or previously treated wounds often show polymicrobial growth, including gram-negative rods or anaerobes. The most frequent isolated organisms were Staphylococcus aureus, Staphylococcus epidermidis, and Streptococcus species. Among anaerobes, Peptostreptococcus magnus and Bacteroides fragilis were isolated from ulcers with ischemic necrosis or deep tissue involvement. Among hospitalized patients, the prevalence of methicillin-resistant Staphylococcus aureus (MRSA) in DFI is 15–30%.

Although a detailed discussion of the range of antibiotic therapy is beyond the scope of this review, common classes of agents used include cephalosporins, fluoroquinolones, and penicillin/B-lactamase inhibitors.

The possibility of underlying osteomyelitis should be considered with the presence of exposed bone or bone that can be palpated with a blunt probe. If osteomyelitis is diagnosed, the patient may undergo surgical excision of the affected bone or an extensive course of antibiotic therapy.

Consideration is also given to the presence of underlying ischemia because an adequate arterial blood supply is necessary to facilitate wound healing and to resolve underlying infections. Patients with evidence of decreased distal blood flow or ulceration that does not progress toward healing with appropriate therapy should be referred to a vascular specialist.

A number of adjunctive wound care treatments are under investigation and in practice for treating diabetic foot ulcers. The use of human skin equivalents has been shown to promote wound healing in diabetic ulcers via the action of cytokines and dermal matrix components that stimulate tissue growth and wound closure. However, the present data for most of these modalities are not considered sufficient for routine implementation in the treatment of diabetic wounds.

One of the more popular adjunctive therapies in use are hyperbaric oxygen therapy (HBOT). HBOT is the delivery of oxygen to patients at higher than normal atmospheric pressures. This results in an increase in the concentration of oxygen in the blood and an increase in the diffusion capacity to the tissues. The partial pressure of oxygen in the tissues is increased, which

stimulates neovascularization and fibroblast replication and increases phagocytosis and leukocyte-mediated killing of bacterial pathogens in the wound. Although small randomized studies have demonstrated an improvement in the rate of wound healing and a decrease in the number of amputations, other studies contest these data. The quality of the studies to date has been poor, and their findings have not been confirmed in a large, blinded, and adequately powered randomized trial.

**PREVENTION:**

**Primary prevention:** Improved blood-glucose control will reduce microvascular complications, and reduction in cardiovascular risk factors will render the foot less susceptible to ischaemia from macrovascular disease. Routine surveillance will detect patients whose feet are at risk, and they should receive targeted care. The case for primary prevention might seem self-evident, but is not yet evidence based.[13]

**Secondary prevention:** Early detection of potential risk factors for ulceration can decrease the frequency of wound development. Efforts should be made to reduce abnormal pressure loading, which might involve cushioning in frail and immobile people and individually fitted footwear in those who are mobile, but such interventions need to be properly targeted. Education should focus on foot care, maintaining good glycemic control, wearing appropriate footwear, avoiding trauma, and performing frequent self-examinations. Education improves knowledge and illness-related behaviour, and led in one trial to a three-fold reduction in re-ulceration and amputation within 13 months.

**CONCLUSION: Ulcer outcome:** In a cohort of 156 people, only 108(69%) healed after primary treatment; 34(22%) healed after surgery and 14(9%) died unhealed. In deep infections, the rate of healing without surgery dropped to 48%, with a median healing time of 24 weeks; with surgery this rate increased to 60% and 18 weeks. In trials of off-loading techniques, 21–50% of patients healed within 30 days, and 58–90% within 12 weeks. Rates and speed of healing are best in ulcers that are mainly a result of neuropathy. Piaggesi and colleagues reported 79% healing at 25 weeks in neuropathic ulcers after conventional treatment, compared with 96% after excision of the ulcer and adjacent bone However, despite good management, healing rates in large multicentric trial were 24% at 12 weeks and 31% at 20 weeks.

**Patients' Outcome:** Since peri-operative and postoperative mortality rates are high, crude data for amputation incidence are insufficient; survival, functional outcomes, and quality of life should be assessed by measures such as the SF-36 health survey, Barthel index, walking and walking stairs questionnaire, and Euroqol-5D.Using the psychological adjustment to illness scale and hospital anxiety and depression scale, Carrington and colleagues showed worse adjustment to illness and significantly more depression in patients with active ulcers than in diabetic controls. In addition to these generic measures, one disease-specific scale has recently been developed and validated and its use considered for future clinical studies. Outpatient dressings and nursing time

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contribute more to the cost of care for ulcer patients in Europe. These costs are met by various care agencies and could be difficult to collate.

Patients with diabetes are at an increased risk for developing foot ulcerations. The consequences of persistent and poorly controlled hyperglycemia lead to neuropathic and vascular abnormalities that cause foot deformities and ulceration. The feet of diabetic patients should be examined at least annually to determine predisposing conditions to ulceration. Treatment plans should be based on examination findings and the individual risk for ulceration.

If ulcers are present, the treatment strategy should include offloading, debridement, and appropriate dressings. Further, the presence of infections should be determined by clinical findings and appropriate wound cultures and treated based on the culture results. If evidence for ischemia is present, revascularization may be indicated to restore arterial blood flow and increase the chance for limb salvage. There are adjunctive therapies available that can also contribute to the overall healing process of the wounds in affected patients. By conducting a periodic foot survey in diabetic patients and incorporating the appropriate basic and specialized care as warranted, the risk of ulceration and its associated morbidities can be reduced.

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