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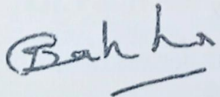
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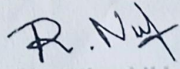
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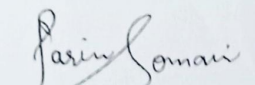
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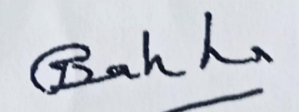
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Promising new approach to treating chronic diabetic foot

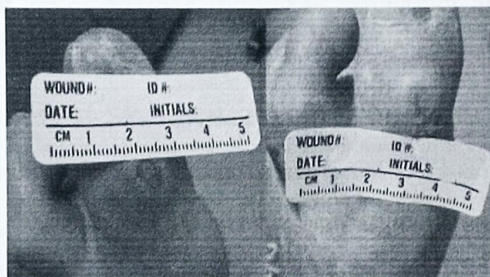
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Introduction

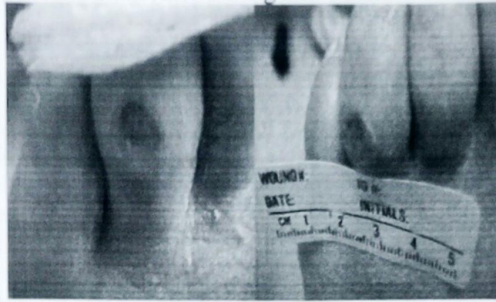
Diabetic foot ulcers, as shown in the images below, occur as a result of various factors, such as mechanical changes in conformation of the bony architecture of the foot, peripheral neuropathy, and atherosclerotic peripheral arterial disease, all of which occur with higher frequency and intensity in the diabetic population. [1,2]

Diabetic ulcer of the medial aspect of left first
Diabetic ulcer of the medial aspect of left first toe before and after appropriate wound care.



Diabetic ulcer of left fourth toe associated with
Diabetic ulcer of left fourth toe associated with mild cellulitis.
Non enzymatic glycation predisposes ligaments to stiffness.
Neuropathy causes loss of protective sensation and loss of

coordination of muscle groups in the foot and leg, both of which increase mechanical stresses during ambulation.



Diabetic foot lesions are responsible for more hospitalizations than any other complication of diabetes.^[3] Diabetes is the leading cause of non-traumatic lower extremity amputations in the United States, with approximately 5% of diabetics developing foot ulcers each year and 1% requiring amputation.

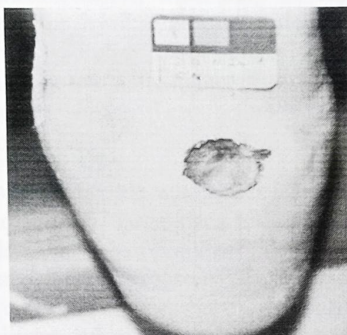
Physical examination of the extremity that has a diabetic ulcer can be divided into examination of the ulcer, examination of the feet, assessment of the possibility of vascular insufficiency,^[4] and assessment for the possibility of peripheral neuropathy.

Diabetic foot ulcers can be staged using the Wound, Ischemia, and foot Infection (WIFI) threatened limb classification system. This system allows communication between providers and provides risk stratification for major amputation.^[5] Blood work should be obtained, such as a complete blood count (CBC), a comprehensive metabolic panel, and hemoglobin A1c (HbA1c), as well as inflammatory markers when infection is suspected. Weight-bearing radiographs of the affected limb should be obtained.

The management of diabetic foot ulcers requires offloading the wound,^[6,7] daily saline or similar dressings to provide a moist

wound environment, ^[8] debridement when necessary, antibiotic administration with or without surgical intervention if osteomyelitis or soft tissue infection is present, ^[9,10] optimal control of blood glucose, and evaluation and correction of peripheral arterial insufficiency. ^[11]

All patients harboring diabetic foot ulcers should be evaluated by a qualified vascular surgeon and podiatric surgeon who will consider débridement, reconstructive surgery on bony architecture, vascular reconstruction, and options for soft tissue coverage.



It is prudent to address the underlying etiologies in diabetic foot ulcers for wound care modalities to be successful. Without addressing the osseous deformities and muscular imbalances, infections, and vascular insufficiency, there will be of minimal benefit in employing advanced wound care dressings.

Pathophysiology

Atherosclerosis and peripheral neuropathy occur with increased frequency in persons with diabetes mellitus (DM).

Trophic changes

Non-enzymatic glycosylation of skin and connective tissue, along with decreased collagen production in people with diabetes, result in alterations in the biomechanics in the diabetic foot. This is frequently seen in the Achilles tendon, where increased stiffness results in a contracture that limits ankle dorsiflexion, a condition known as equinus. Equinus has been associated with diabetic foot ulcers, as it increases plantar pressures in the forefoot and midfoot.

Diabetes-related atherosclerosis

Overall, people with diabetes mellitus (DM) have a higher incidence of atherosclerosis, thickening of capillary basement membranes, arteriolar hyalinosis, and endothelial proliferation. Calcification and thickening of the arterial media (Mönckeberg sclerosis) are also noted with higher frequency in the diabetic population, although whether these factors have any impact on the circulatory status is unclear.

Diabetic persons, like people who are not diabetic, may develop atherosclerotic disease of large-sized and medium-sized arteries, such as aortoiliac and femoropopliteal atherosclerosis. However, significant atherosclerotic disease of the infrapopliteal segments is particularly common in the diabetic population. Underlying digital artery disease, when compounded by an infected ulcer in close proximity, may result in complete loss of digital collaterals and precipitate gangrene.

The reason for the prevalence of this form of arterial disease in diabetic persons is thought to result from a number of metabolic abnormalities, including high low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL) levels, elevated plasma von Willebrand factor, inhibition of prostacyclin synthesis, elevated plasma fibrinogen levels, and increased platelet adhesiveness.

Diabetic peripheral neuropathy

The pathophysiology of diabetic peripheral neuropathy is multifactorial and is thought to result from vascular disease occluding the vasa nervorum; endothelial dysfunction; deficiency of myoinositol-altering myelin synthesis and diminishing sodium-potassium adenine triphosphatase (ATPase) activity; chronic hyperosmolarity, causing edema of nerve trunks; and effects of increased sorbitol and fructose.^[12]

Motor dysfunction of peripheral nerves in diabetic neuropathy leads to muscular imbalances in the diabetic foot. Muscle wasting of the intrinsic pedal muscles leads to overpowering of the spared extrinsic muscles, which results in significant forefoot deformities such as claw toes or hammer toes.^[13,14] Autonomic dysfunction of the peripheral nervous system may lead to sudomotor dysfunction. This will result in dry, cracked skin, which is more prone to injury and breakdown.^[15]

The result of loss of sensation in the foot is repetitive stress; unnoticed injuries and fractures; structural foot deformity, such as hammertoes, bunions, metatarsal deformities, or Charcot foot (see the image below); further stress; and eventual tissue breakdown. Unnoticed excessive heat or cold, pressure from a poorly fitting shoe, or damage from a blunt or sharp object inadvertently left in the shoe may cause blistering and ulceration. These factors, combined with poor arterial inflow, confer a high risk of limb loss on the patient with diabetes.

Charcot deformity with mal perforans ulcer of plan

Charcot deformity with mal perforans ulcer of plantar midfoot.

Etiology

The etiologies of diabetic ulceration include neuropathy,^[16] arterial disease,^[17] pressure,^[6] and foot deformity.^[18] Diabetic peripheral neuropathy, present in 60% of diabetic persons and 80% of diabetic persons with foot ulcers, confers the greatest risk

of foot ulceration; microvascular disease and suboptimal glycemic control contribute.

Discussion

A study by Naemi et al indicated that tissue mechanics may be associated with foot ulceration in patients with diabetic neuropathy, with an evaluation of 39 patients finding that the heel pad in nonulcerated feet tended to be stiffer than in ulcerated feet^[19]. These results were further elucidated in another study by Naemi et al, which reported that the risk of diabetic foot ulcer is higher in diabetic neuropathy patients who have greater plantar soft tissue thickness and lower stiffness in the area of the first metatarsal head.

Conclusion

The investigators found that adding the mechanical properties of plantar soft tissue (stiffness and thickness) to commonly evaluated clinical parameters improved specificity, sensitivity, prediction accuracy, and prognosis strength by 3%, 14%, 5%, and 1%, respectively.^[20]

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