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A Review on Retrospective Study of Risk Factors and Management of diabetic foot Ulcer

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Abstract

Diabetic foot ulcer (DFU) is a debilitating and severe manifestation of uncontrolled and prolonged diabetes that presents as ulceration, usually located on the plantar aspect of the foot. Approximately 15% of individuals with diabetes will eventually develop DFU, and 14%-24% of them will require amputation of the ulcerated foot due to bone infection or other ulcer-related complications. The pathologic mechanisms underlying DFU are comprise a triad: Neuropathy, vascular insufficiency, and secondary infection due to trauma of the foot. Standard local and invasive care along with novel approaches like stem cell therapy pave the way to reduce morbidity, decrease amputations, and prevent mortality from DFU. In this manuscript, we review the current literature with focus on the pathophysiology, preventive options, and definitive management management of DFU.

Keywords: Diabetes, Ulcer, Foot, Antibiotics, Revascularization, Cell therapy

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1. Introduction

A non-healing or poorly healing, break in the skin below the ankle in an individual with diabetes, these ulcers are critical in the natural history of the diabetic foot. The high incidence of Diabetic foot ulcer (DFU)and the associated mortality and morbidity are the most common reasons for hospitalization of diabetes patients.Early in the course of DM, patients experience serious foot sensitivity symptoms such as pain and tingling, while later stages of the disease.Course is characterized by negative symptoms such as numbness and weakness of the toes. With the progression of the disease, patients usually show mixed pain sensitivity and dullness, which lead to imbalance and unsteadiness and increase the likelihood of falls in addition, because of the increasing morbidity, DFU is a leading cause of non-traumatic amputation and is associated with an increased risk of death.



Fig.1. foot ulcer

The university of Texas classification system:

The classification system proposed by the university of Texas, takes some common clinical signals and symptoms

of DFU into to consideration by using a foreign to four matrices. They divided patients into four categories.

GRADE	INFECTED OR ISCHEMIC LESIONS
0	Pre or post ulcerative site
1	Super facial wound not involving tendon
2	Wound penetrating to tendon
3	Ulcer penetrating to bone of joint

Signs &Symptoms: Any changes to the skin or toenails, including cuts, blisters, Calluses or sores

- Discharge of fluids or pus
- Foul smell
- Pain
- Redness
- Skin discoloration
- Swelling
- Drainage or blood in
- Shoes or socks
- Blisters
- Sores

Causes: The common underlying Causes are

- Poor glycaemic control
- Calluses
- Foot deformities
- Improper foot care
- Ill –fitting foot wear
- Underlying peripheral neuropathy
- Poor circulation
- Dry skin

2. Epidemiology

Nearly 537 million people are currently suffering from diabetes mellitus worldwide. This is projected to rise to 783 million by the year 2045. Diabetes mellitus is the primary cause of non-traumatic lower limb amputations across the globe. This growing incidence of DM can lead to the increased prevalence of diabetic foot complications, which have become a serious medical, social, and economic concern of global importance. Diabetic foot ulcers are foot lesions that damage the skin, soft tissues, and bones in the legs and feet, generating an aggravated infection in diabetic patients and potentially leading to lower limb amputations. Recent reports showed that around 60 to 80% of these ulcers can heal, 10 to 15% can stay active, and 5 to 24% eventually result in limb amputation.

More than 85% of all amputations in diabetic foot patients are preceded by ulceration leading to severe gangrene or infection on average, about 6.4% of the worldwide population suffers from diabetic foot, with 2 to 5% yearly occurrence rates of ulcer or necrosis It is predicted that 19 to 34% of diabetic individuals will encounter foot disease at some stage in their lives .A systemic meta-analytical review has shown that diabetic foot ulcers (DFU) are more common in men (4.5%) than in women (3.5%) and more prevalent in type 2 diabetic patients (6.4%) compared to type 1 individuals (5.5%) Despite the rapidly growing prevalence of diabetic foot, only a few studies have been performed regarding its epidemiology.



Risk Factors: Anyone with diabetes is a risk of developing a diabetic foot ulcer. Here is a list of other risk factors that increase chances of developing foot ulcer.

- Peripheral neuropathy (sensory, motor, automatic)
- Foot deformity hammer toe
- Improperly fitted shoes
- Peripheral arterial disease
- Callus
- History of prior ulcer/amputation
- High planter foot pressures
- Limited joint mobility
- Uncontrolled hyperglycaemia
- Chronic renal insufficiency
- Poo knowledge of diabetes
- Abnormal foot structure
- Smoking
- Diabetes that is not well controlled
- Being over weight
- Poor circulation
- Walking bare foot
- Excessive alcohol use
- Aging
- High cholesterol levels

3. Clinical Manifestations

- Release of pus from the wound.
- Foul smell and pain.
- Redness around the Ulcer
- Skin changes
- Sores, ulcers, Blisters On the foot Over lower Leg
- Pain, difficulty in walking.
- Discoloration In Feet: black, Blue or red.
- Fever, skin redness Swelling.

Complications

Skin infections:

Diabetic ulcers on the feet are prone to skin infections, resulting in swelling around the wound site, foul smelling drainage, fever and chills. If the infections spread from the wound to the bone, the risk of amputation increases.

Abscess Formation:

An infected diabetic foot ulcer may lead to the formation of an abscess. Which is a painful accumulation of pus under the surface of the skin.

Sepsis:

If the infection is deep enough, it may enter the blood stream leading to sepsis. This is a potentially lifethreatening conditions when the body's immune system over reacts to an infection and starts to damage tissues and organs.

Foot deformities: Long term uncontrolled diabetes along with diabetic foot infection can weaken muscles and bones in the feet resulting in deformities. They may even cause Charcot foot, a serious complication of the joints, bones and soft tissue of the ankle or foot.

Gangrene:

Gangrene is the death of body tissue because of a bacterial infection or the loss of blood flow it is characterized by discoloration of the skin, unusual pus or discharge from the area and loss of sensation in the effected part of the body.

Foot amputation:

Many people with diabetes have peripheral arterial disease (PAD), which reduce blood flow to the feet and neuropathy, a condition that numbs pain usually in the hands and feet.Several infections, abscess and gangrene in the foot make it impossible for the foot ulcers to heal. In such cases the only option to prevent the infection from spreading to the blood stream is amputation of the effected foot.

Pathophysiology

DFU comprises a full-thickness wound involving the dermis, located in the weight-bearing or exposed area below the ankle. The Wagner system aids in categorizing the severity of the ulcer, ranking it on a scale of 1 to 5. The pathologic mechanisms of DFU are described in terms of a triad. This triad includes neuropathy, vascular insufficiency, and secondary infection due to trauma of the foot.

First, the lack of protective sensation in the feet predisposes patients with diabetes to developing trauma and ulcers. This sensory impairment occurs due to hyperglycemia-induced upregulation of aldose reductase and sorbitol dehydrogenase, which in turn increase the production of fructose and sorbitol. These glucose products accumulate and induce osmotic stress, thereby reducing nerve cell myoinositol synthesis and nerve conduction. Also, from a pathological stance, advanced glycation end-products (AGEs) must be considered. AGEs are non-enzymatic protein, amino acid, and DNA adducts which form from dicarbonyls and glucose. Diabetes is associated with the development of diabetic complications.

In addition to sensory neuropathy, diabetes can induce neuronal autonomic dysfunction that results in impaired sweat production, leaving the foot susceptible to dryness, skin cracking, and fissuring. Furthermore, motor neuron dysfunction can give rise to muscle wasting and structural abnormalities of the foot. These causes focally elevated pressures at various zones of the plantar foot and increases the risk of ulceration.



4. Diagnosis

Patient workup for Diabetic ulcers include:

Blood test: A complete blood count (CBC) should be performed. Leukocytosis may signal an associated diabetic foot infection. Wound healing is impaired by anemia. In the face of underlying arterial insufficiency, anemia may precipitate rest pain.

Radography:

Plain radiographs should be obtained in the setting of diabetic foot ulcers, as well as pre-ulcerative lesions. Weight-bearing radiographs of the foot are preferred, and three views are typically ordered. Radiography allows clinicians to assess for deformities that may be the driving force for ulcerations. Osteomyelitis can often be detected on plain radiographs; however, the images may appear normal in the first 2 weeks of the disease process. Soft tissue gas may be present in the setting of gas gangrene and necrotizing soft tissue infections, which require prompt surgical intervention.

Magnetic resonance imaging scan:

In the setting of osteomyelitis, MRI is said to be both specific and sensitive. MRI is also useful for evaluating deep space infections, infectious tenosynovitis, myositis, and septic arthritis. However, in the setting of Charcot arthropathy, it is difficult to distinguish that condition from osteomyelitis.

Computed tomography scan:

CT scanning can be used to identify osseous changes such as cortical erosions, pathologic fracture, and periosteal reactions. Because of resolution limitations, however, this modality may not be as useful in evaluating soft tissue infection, although injection of intravenous contrast may provide better visualization of deep space abscess formation.

Bone scans:

Bone scans can be beneficial when the use of MRI and CT scanning is contraindicated for evaluating osteomyelitis. However, although bone scans have high sensitivity for osteomyelitis, they have low specificity, resulting in an increased number of false-positive scans. Labeling with gallium and indium radionuclides may lead to more specificity for suspected osteomyelitis.

Ankle -Brachial Index

The systolic pressure in the dorsalispedis or posterior artery divided by the upper extremity systolic pressure is called

the ankle-brachial index (ABI) and is an indication of severity of arterial compromise

Pulse -Volume Recording

Pulse-volume recording (PVR), or plethysmography, uses pneumatic cuffs encircling the thighs, calves, ankles, feet, and, occasionally, toes to sense segmental volume changes with each pulse beat. The resulting tracings provide useful information about the hemodynamic effects of the arterial disease at each level.

Ultrasonography:

Duplex ultrasonography can provide images of arterial segments that help localize the extent of disease, and simultaneous Doppler measurement of flow velocity can help estimate the degree of stenosis. Duplex scanning is quite useful in visualizing aneurysms, particularly of the aorta or popliteal segments. Use of this technique probably is best left to the discretion of the vascular specialist.

Conventional angiography:

If vascular or endovascular surgical treatment is contemplated, angiography is needed to delineate the extent and significance of atherosclerotic disease. Major risks associated with conventional contrast-injection angiography are related to the puncture and to the use of contrast agents.

Bone biopsy:

If osteomyelitis is suspected, a bone biopsy is the gold standard for confirming the diagnosis. Bone biopsies are performed either through open technique or percutaneously, with or without the assistance of fluoroscopy. Osseous specimens are sent for histologic and microbiologic evaluation.

Preventive care:

Due to diabetes being a risk factor for the development of underlying peripheral vascular disease, the majority of DFUs are asymptomatic until advanced enough to recognize more severe signs and symptoms. During the diagnosis of DFU, neuropathy may mask ischemia and vice versa. Therefore, the primary preventative strategy is regular diabetic foot screening to allow early identification of DFU, followed by initiation of treatment if appropriate. Ultimately, early detection and management work to avoid further complications such as gangrene and amputation. **Noninvasive care:**

The most prevalent management modality for DFU is local care, in which many potential avenues of treatment can be utilized. These include wound dressings,human skin equivalents (HSEs), pressure offloading, total-contact casting (TCC), systemic hyperbaric oxygen, larvae therapy and topical growth factor.

Wound dressing:Wound dressings are the most basic and common treatment measure, and although they serve a vital purpose in the management of DFU, other methods have proven vastly more effective in comparison to or in adjunct with wound dressings.

Human skin eqivalen:

HSE is more effective compared to the standard treatment of saline-moistened gauze in reducing the rates of amputation and infection and in improving the rate of ulcer healing. One randomized controlled trial (RCT) assessed the effectiveness of Graftskin, a living skin equivalent indicated for use in noninfected, nonischemic DFU. In this study, Graftskin was applied weekly for a maximum of 4 wk or until complete healing occurred. **Offloding,TCC :**

Pressure offloading serves as one of the primary treatments of DFU, primarily in ulcers accompanied by neuropathy, with many variants being utilized. For ischemic DFUs, however, revascularization is more commonly used. Common methods of offloading include bed rest, wheelchair use, implementation of a crutch-assisted gait, total contact casting, use of felted foam, use of therapeutic shoes, and use of removable cast walkers. The most effective offloading treatment is TCC, in which full casts are applied by an experienced physiotherapist and are changed weekly for 2-3 wk or until healing has occurred. One RCT found that TCC was extremely effective in increasing ulcer healing and reducing infection when compared to traditional dressing changes and other offloading methods.

Larvae therapy:

Maggot therapy is another well-researched technique with respect to the treatment of chronic wounds in which maggots are placed on the wound area. This treatment method has been shown to significantly facilitate debridement. In one study, maggot therapy also enabled faster development of granulation tissue and more significantly decreased wound surface area compared to other topical treatments such as hydrogel dressings. Maggot therapy also had no effect on disinfection or complete healing rate for the wound.

Invasive treatment strategies:

Debridemen:

Debridement is a major component in the treatment of DFU, particularly due to its ability to alter the environment of the chronic wound through the removal of necrotic and nonviable tissue and foreign debris, which impede the healing process. Debridement may not always lead to complete healing of the DFU, but it serves as an important preliminary step in the treatment. Following debridement, the wound is further analyzed and if necessary, other treatment paths are pursued [55]. Debridement is commonly used in conjunction with other treatment modalities.

Skingrafting:

Skin grafting may serve as a solution when DFUs become more severe, offering a chance to replace the infected skin and promote the healing process. There are a variety of skin grafting techniques that may be used, including bioengineered or artificial skin, autografts (taken from the patient), allografts (taken from another person), or xenografts (taken from animals). A review article that analyzed 17 RCTs concluded that skin grafting and tissue replacement when used in conjunction with standard treatment led to an increase in the healing rate of DFU and slightly lowered the chance of amputation. However, evidence of long-term effectiveness is uncertain. Amputation: Amputation represents the final management option when treating DFU and is reserved for the most chronic levels of infection or deformity that render the foot nonfunctional. Amputation can be classified as either minor or major, with minor being the removal of a smaller area (e.g., removal of a toe or a part of the foot).

Prevention: Podiatrists recommend wearing clean, dry socks that don't have tight elastic bands, which restrict blood flow to the foot. Doctors advise people with diabetes to avoid walking bare foot and wearing saddles, which expose your feet to splinters concrete or sand, which may scrape or irritate the foot.

Methodology

Inclusion:We are case sheets collected in government gerenal hospital. 45-70 age peoples are included in this study. Female and male both patients are included in the study

Exclusion:Childrens are excluded in the study. Non diabetic foot ulcer patients excluded in this study



4. Conclusion

DFU Results in substantial morbidity and mortality in patients with diabetes. It also often leads to longer hospitalizations and associated increases in health care spending. Thus, prompt diagnosis and catered management is essential to management of this prevalent consequence of diabetes. Standard local and invasive care along with novel approaches like stem cell therapy pave the way to reduce morbidity, decrease the need for amputation, and prevent mortality due to DFU. Further research into newer modalities that aid in prompt and effective management will further help alleviate the healthcare burden of DFU.

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