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Research Paper

Walking On Thin Ice: Understanding and Preventing Diabetic Foot Ulcers

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ABSTRACT: *Diabetic foot ulcers (DFU) are a serious side effect of diabetes that frequently results in amputations, extended hospital stays, and infections. Peripheral artery disease (PAD), neuropathy, smoking, long-term diabetes, foot abnormalities, and a history of ulcers or amputations all raise the chance of developing DFUs. Usually, a combination of Gram-positive and Gram-negative bacteria, anaerobes, and fungi cause the infections in DFUs. Comprehensive care, such as blood glucose management, lifestyle modifications, routine foot assessments, appropriate footwear, and patient education, is the focus of prevention initiatives. For people who are at risk of DFUs, wearing shoes that fit properly, abstinence of smoking, and managing blood pressure and cholesterol are important preventive measures. the use of multiple treatments is needed for patients who already have foot ulcers. This includes debridement, pressure unloading, wound dressings, and maybe surgery. To remove necrotic tissue and to promote healing, debridement techniques include surgery, enzymatic, autolytic, and biological debridement (such as maggot therapy) are crucial. For the treatment of neuropathic ulcers, pressure modulation using methods such as total contact casts (TCC) is potential. Numerous cutting-edge wound dressings, such as hydrogels, hydrocolloids, and dressings infused with silver, help control wound moisture, enhance autolysis, and fight against infection. Revascularization, nonvascular foot surgery for abnormalities, or amputation in extreme circumstances are all possible surgical therapeutic options. strategies are required for long-term prevention and care for treating both the ulcer and the underlying causes of DFU. Ultimately, lowering the incidence of DFU and enhancing patient outcomes require a mix of efficient communication, patient education, and coordinated treatment among healthcare providers*

KEYWORDS: *Diabetic Foot Ulcers (DFU), Risk Factors, Debridement, Wound Dressing, Infection Management, Foot Care, Preventive Strategies*

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I. INTRODUCTION:

Adults with diabetes who have diabetic foot ulcers (DFU) are at high risk for avoidable disease effect. Infection, hospitalization, lower-extremity amputation, decline in functional status, and death are some of the consequences of foot ulcers. Approximately 19% to 34% of people will develop a foot ulcer in their lifetime, and this risk is increasing as people with diabetes live longer and have more complex medical conditions[1].Globally, there are more than 463 million cases of diabetes mellitus (DM) Numerous complications related to diabetes are possible for these patients. particularly these patients have significant morbidity and mortality due to foot issues. Patients with diabetes often experience foot-related conditions like ulcerations, gangrene, and infections. According to assumptions from the International Diabetes Federation, 9.1–26.1 million people with diabetes will get diabetic foot ulcers (DFUs) yearly. Moreover, the lifetime risk of foot ulcers for people with type 1 and type 2 diabetes is up to 34%[2].

A possibly deadly infection can originate from foot ulceration caused by diabetic peripheral sensory neuropathy, repetitive trauma from unprotected walking, rigid osseous deformities and soft-tissue contractures, and peripheral vascular disease[3].The complicating factor of underlying peripheral vascular disease renders majority of diabetic foot ulcers asymptomatic during the early stages of disease. In the latter more advanced stages, evidence of tissue loss becomes more evident, frequently occurring in the form of chronic non-healing

foot ulcers[4].In order to avoid consequences like DFU Primary preventive measures that are inexpensive, effective, and enable early detection and timely treatment of changes include self-care, daily foot self-examination, and clinical foot examination. self-care behaviours must be monitored using validated assessment tools. Additionally, it makes it possible to evaluate how well a patient is responding to treatment, pinpoint issues and requirements, and guide the development of a care plan, clinical management, and decision-making[5].

This review aims to provide an understanding of diabetic foot complications by concentrating on risk factors, underlying causes, and the most recent preventive techniques. It seeks to increase understanding of the seriousness of diabetes-related foot complications among medical professionals, patients, and caregivers, as well as the importance of early detection and treatment in averting serious consequences like ulcers, infections, and amputations.

1.1 EPIDEMIOLOGY:

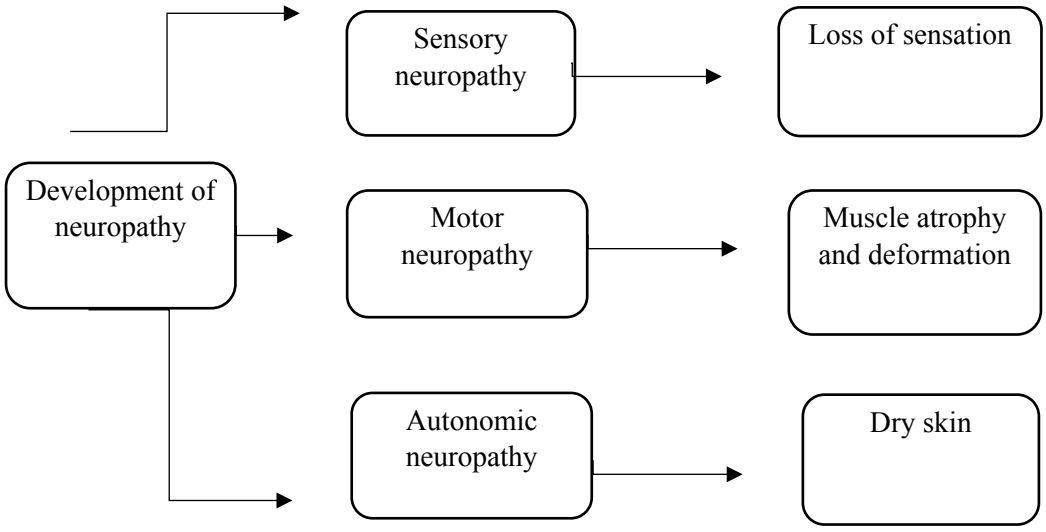
An annual incidence of 1.0 % to 4.1 % has been estimated for diabetic patients' lifetime risk of developing foot lesions (ulcers/gangrene). Although type 2 diabetic patients make up about 90 % of all diabetic patients, the incidence of these lesions appears to be similar in type 1 and type 2 diabetic patients. 15 % of these patients will eventually require an amputation due to ulcers. Patients with poor glycaemic control, male gender, history of diabetes for ten years, and pre-existing cardiovascular, renal, or retinal comorbidities are more likely to develop an initial foot ulcer. the rate of Foot ulcers occurs highest in Native Americans and lowest in Madrid, Spain. In North America, patients who belong to ethnic minority groups—particularly Hispanics and African Americans—as well as other patient groups without health insurance are more likely to have foot ulcers and amputations[6].

II. PATHOPHYSIOLOGY OF DFU:

For a diabetic patient due to the abnormal blood glucose levels ulceration or infections occurs. It is divided into three phases namely

1. The pre ulcer phase
2. Ulcer phase and
3. Recurrent ulcer phase[7]

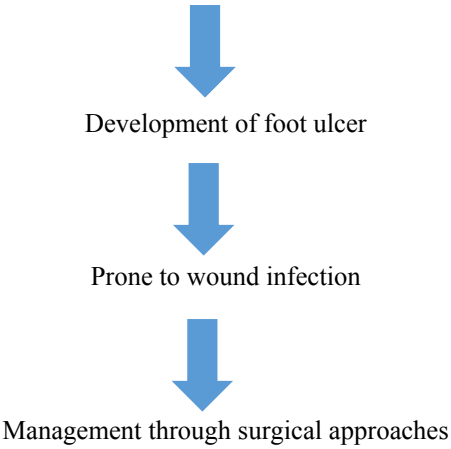
2.1 Pre ulcer phase:



People may experience this phase when there is improper education which may lead to dangerous period for ulcer development.

2.2 Ulcer phase:

Due to the triggering factors like loss of protection, peripheral vascular lesions and repeated injuries



2.3 Recurrent ulcer phase:

The healing phase known as the Recurrent Ulcer Phase is when clinical symptoms disappeared but there is still a high chance of recurrence. Peripheral vascular lesions, infection, neuropathy, and a high risk of relapse all have an impact on this recurrence risk. Strategies include increasing blood supply, using adjuvants appropriately, and implementing surgical or pharmaceutical treatments to successfully manage these hazards. These methods are intended to lessen the recurrence and improve the quality of life for those who suffer from recurrent ulcers[7]

2.4 PATHOGENESIS OF DFU:

Peripheral neuropathy:

"The presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after the exclusion of other causes" is the definition of diabetic peripheral neuropathy. This can have a catastrophic impact. Diabetes is a major cause of lower limb amputation, and about 50% of diabetics will get a foot ulcer in their lifetime[8]. Diabetes-related neurological conditions fall into one of three categories: sensory, motor, or autonomic neuropathy[9]. Diabetic peripheral neuropathy may occur in multiple forms. The common kind is distal symmetric polyneuropathy, which can be divided into three different groups: mixed small and large fiber, mostly small fiber, and primarily large fiber[10].Medical conditions such as insulin resistance, dyslipidemia and hyperglycemia cause metabolic pathways that are irregular, which in turn causes an imbalance in the redox status of the mitochondria, which in turn causes an excess of reactive oxygen species to develop in the cytoplasm and mitochondria. These disorders result in axonal destruction and loss of axon energy storage, which exacerbates peripheral nerve lesions and damages the foot's nerves. Due to Neuropathy in the lower extremities is frequently unnoticed in particular time, and the injury continues to experience continuous strain (including extended periods of walking or carrying heavy items.)Additionally, neuropathy results in muscle tissue imbalances and muscle wasting in the feet of individuals diagnosed with diabetes. During the time being, foot abnormalities like foot deformities may develop. fall, talon toes, and contracture of the calf muscle can develop, resulting in exacerbating diabetic foot ulcer[11].

Peripheral artery disease:

Peripheral arterial disease (PAD) is a significant factor in the occurrence of leg ulcers and amputations. A peripheral artery disease is the total or partial blockage of one or more of the upper and lower limbs' noncardiac, non intra cerebral arteries. This can result in tissue loss or decreased blood flow. It is typically caused by vascular wall atherosclerosis, but it can also develop from thrombosis, embolism, fibromuscular dysplasia, or vasculitis. In non-peripheral intra-cerebral and coronary arteries, atherosclerotic PAD may be a sign of systemic atherosclerosis. The lower limb arteries are the ones that are typically affected by diabetes mellitus (DM); these are typically the distal arteries, particularly the dorsalis pedis artery[12]. Studies have revealed that advanced glycation end products (AGEs) induce aberrant crosslinks in collagen, which results in vascular stiffness and reduced endothelial cell nitric oxide release. Nuclear factor kB (NFKB) is produced by endothelial cells when they form a AGE receptor complexes. Vascular cell adhesion protein 1 (VCAM-1) and proinflammatory cytokines are therefore expressed more frequently. After a while, endothelial cell function is compromised, which impairs blood vessel constriction and results in platelet aggregation, endothelial cell growth, and atherosclerosis [13].

Diabetic foot infection:

Diabetic foot infections (DFIs) are characterized by the attack and proliferation of microorganisms in diabetic wounds that do not heal. These infections are linked to changes in the host's inflammatory response and/or tissue destruction. Among the most dangerous and common complications in diabetics are infections. They are assumed to appear in roughly 60% of DFU cases and are a significant cause of morbidity for these patients. The development of infection is affected by a variety of wound microbiology factors, such as the microbial load, microbe diversity, presence of pathogenic microorganisms, and synergistic association among microbial species. Not only Gram-positive bacteria like S. aureus (methicillin-susceptible and methicillin-resistant strains), Streptococcus hemolytic, and C. striatum among the most commonly mentioned bacteria in DFUs, but also Gram-negative bacteria like P. aeruginosa, E. coli, A. baumannii, Proteus spp., Enterobacter spp., and Citrobacter spp., as well as some anaerobes deeper in the wound bed like Bacteroides spp., Prevotella spp., Clostridium spp., and Peptostreptococcus spp.[14] (table 1)

Table 1: Common Microorganisms in Diabetic Foot Ulcers

Microorganism type	microorganism	Gram positive /negative	Seen in
Gram-Positive	*Staphylococcus aureus*	Positive	Commonly found in skin infections
	Streptococcus pyogenes	Positive	Associated with necrotizing fasciitis
	Enterococcus faecalis	Positive	Often linked to polymicrobial infections
	Clostridium perfringens	Positive	Anaerobic; can cause gas gangrene
Gram-Negative	*Pseudomonas aeruginosa*	Negative	Known for resistance to multiple antibiotics
	Escherichia coli	Negative	Commonly associated with fecal contamination
	Klebsiella pneumoniae	Negative	Linked to hospital-acquired infections
	Proteus mirabilis	Negative	Can contribute to urinary tract infections
Anaerobic Bacteria	*Bacteroides fragilis*	Negative	Common in mixed infections
	Fusobacterium nucleatum	Negative	Associated with severe infections
	Peptostreptococcus spp.	Positive	Part of normal flora; can cause infections
Fungi	*Candida albicans*	Fungal	Common in moist environments
	Aspergillus spp.	Fungal	Rarely found; can cause opportunistic infections

III. RISK FACTORS:

Based on Wagner's classification, there is a relationship between the duration of diabetes. Therefore, people who have diabetes for more than 10 years have higher chances of developing DFU. Smokers are likely to develop DFU when compared with non-smoking populations. High neutrophil/lymphocyte ratios (NLR) are directly proportional to the development of DFU[15]. DFU is also associated with significant risks such as neuropathy, peripheral artery disease (PAD), foot deformities caused by motor neuropathy, and minor foot injuries, as well as infections and osteomyelitis [16]. DFU development also involves gram-positive bacteria such as Enterococcus species, Streptococcus agalactiae (group B streptococcus), Staphylococcus aureus, and Staphylococcus epidermidis. 15 % of patients get Methicillin-Resistant Staphylococcus. Aureus (MRSA) infections, which are especially dangerous because there are few effective treatment choices and the organism is extremely virulent. Anaerobes such as Porphyromonas, Clostridium, Prevotella, and Bacteroides fragilis are discovered in roughly one-third of cultures, whereas gram-negative bacteria like Pseudomonas aeruginosa and Enterobacteriaceae are identified in more than half of cases. The fact that 50% to 80% of infections are polymicrobial, which makes treatment more difficult, adds to this complexity[18]. Patients having a prior history of foot ulcers or amputations and cardiovascular diseases are also at a higher risk of developing complications again.

IV. CLINICAL MANIFESTATIONS:

Presence of the typical signs of inflammation, such as erythema, discomfort, tenderness, warmth, or induration, or purulent discharge from an ulcer. A bad odour, necrosis and the inability of the wound to heal or with discomfort at the location of a pressure point, callus, or other bony protrusion even with the best care are some of the indicators of infection.

Certain diabetic foot infections may show no or very little localized inflammation. example, those with neuropathy may experience decreased or absent pain and tenderness, but those with vascular disease may not experience erythema. [19]

V. PREVENTION STRATEGIES FOR DFU

DFU is a multifactorial and needs a multidisciplinary approach, prevention or prophylactic care is implicated in patients to reduce or avoid the risk of morbidity, LOS in hospital and also the amputation[20]. In order to guarantee proper care, patients with diabetes who seek treatment should be concurrently managed by a primary care physician and appropriately referred to endocrinologists, podiatrists, vascular surgeons, ophthalmologists, nephrologists, physical therapists, dietitians, and diabetic educators.[20] The prior step in prevention of DFU is creating a awareness in patients and to avoid the fear of amputation by means of proper communication. Due to lack of effective communication between patient and caregiver the effective treatment of DFU is diminished the overall quality of care.[21] since the DFU experience is made more challenging by inadequate communications.

The following are the components of diabetes complications treatment and prevention:

- Changes in lifestyle
- Regulation of blood pressure
- Control of lipids
- Quitting smoking
- Glycemic management

5.1 Patient education on Nail & Skin care [20]

➢ Patients with diabetes should examine their foot, skin and nail every day, Maceration, particularly between the toes, is typically the result of a fungal infection and needs to be closely monitored. It is advised to utilize a mirror to improve Examine the foot's plantar surface. If the patient is unable to see then ones individual help should to taken.

➢ At least once every day, the feet should be cleaned and dried. The interdigital areas between the toes need to be properly dried.

➢ Patients with diabetes, particularly those who have sensory neuropathy, should not apply heating pads to their bodies.

➢ The diabetic patients with peripheral neuropathy are recommended to wear special shoes during outdoor and even indoor.

➢ The Diabetic patients are insisted to change socks and maintain clean socks on daily basis.

➢ The nails are not recommended to cut in rounded fashion but to be cut in straight fashion.

5.2 FOOT EXAMINATION

All diabetics should have their feet examined annually to detect high-risk foot disorders such as peripheral vascular diseases, structural foot abnormalities, and loss of protective sensibility.[20]

5.3 VASCULAR EXAMINATION

Vascular Disease mostly include PAD, Peripheral artery disease is not most causing aspect for the DFU but it accounts only 30% in ulcer formation. All lower extremity pulses, including the femoral, popliteal, posterior tibia, and dorsalis pedis pulses, should be palpated as part of vascular examination. Pulse palpation is an acquired ability with a high degree of false positives and inter-observer variability and rates of false negatives. Ankle Brachial Pressure Index (ABPI) is a test that should be performed to the patients who crossed 50 years, if the test result is normal then the test should be performed for every 5 years.[21]

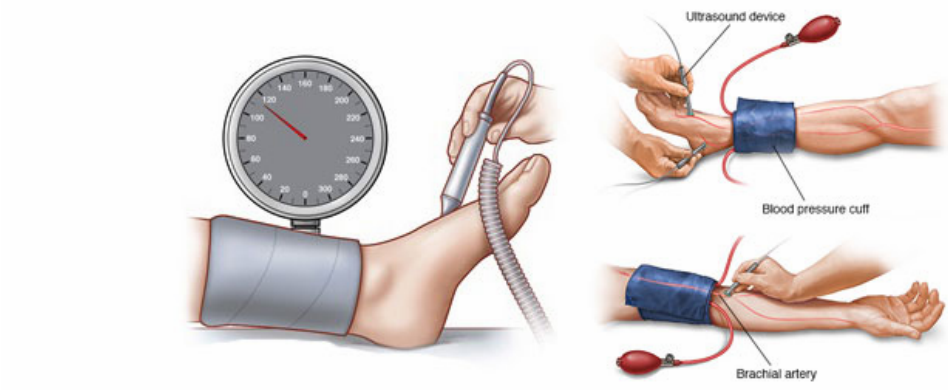


Figure (1) Ankle Brachial Pressure Index

5.4 PROTECTIVE SENSATION

To find the risk of DFU protective sensory test is to be performed, Simple, non-invasive tools like a calibrated vibration perception threshold, a Semmes-Weinstein 5.07/10 gram monofilament nylon wire, and a 128 Hz tuning fork can be used to assess the lack of protective sensation. Vibration Perception threshold (VPT) meter, or by a thorough physical assessment.[21]

5.5 FOOT DEFORMITIES AND BIOMECHANICS

The plantar aspect of the foot is subjected to excessive pressure due to joint limitations and foot abnormalities. The secondary cause of this restriction in joint mobility is non-enzymatic glycosylation of periarticular soft tissues and decreases the foot's capacity to allow for an increase in ambulatory ground reactive force.[21]

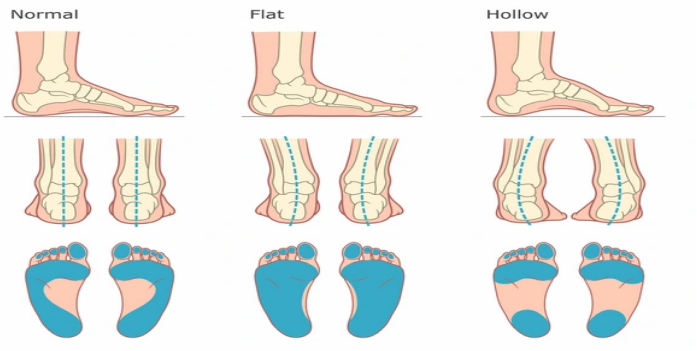


Figure (2)

5.6 ROLE OF FOOTWEAR AND ORTHOTICS

The footwear is not recommended to every patient nut to only some, based upon the evidence screened by he health care provider it is suggested.[23]

Table 2: List of terms and definition of footwear

Term	Definition
Abnormal foot shape	A foot form that prefabricated shoes are unable to fit. Hallux valgus, clawed or hammer toes, severe pes-planus or cavus foot types, excessively wide feet, flat feet, minor amputations, and Charcot foot are a few examples of these foot conditions.
Bespoke footwear	It is known as "Custom-made medical grade footwear".
Custom Footwear	It is known as custom-made medical grade footwear.
Custom made insole	An insole that is generally constructed in multiple layers and is tailored to the individual's foot using a 2D or 3D impression of the foot. Other features, like a metatarsal pad or metatarsal bar, might also be included in this. The purpose of the insole is to provide cushioning and plantar pressure redistribution by melding to the curve of the foot.
Depth- inlay	It is an extra depth footwear.
In-shoe orthosis/orthotic	This term refers to a device that is placed within the shoe to reduce pressure or change how the foot functions. either custom-made or prefabricated.
Metatarsal pad	A little pad is positioned close to the metatarsal head to transmit load more proximally and reduce focused pressure.
Metatarsal bar	To relieve focal stresses and transfer load more proximally, a bar that spans the entire forefoot or only a portion of it is positioned proximal to the metatarsal heads.
Shoe last	Most recently, it was utilized to produce shoes. The shoe's upper is pulled or melded over the bottom. The final shape determines the shape of the shoe, including the toe spring, heel pitch, and outsole. Various sizes of generically created lasts are utilized for prefabricated or off-the-shelf footwear.
Toe orthosis	This is especially used for the toe.

According to Australian guidelines they were 10 guidelines given to the people with diabetes on DFU.
For people at risk of ulceration[24]

- Encourage people to wear shoes that fit, shield, and accommodate their unique foot shapes.
- Encourage people to wear socks in order to lessen friction and shear.[24]
- Advice people to create awareness for the diabetic patients and also the caregivers about the importance of wearing the proper footwear.

For people at intermediate or high risk of ulceration[24].

- Tell diabetics who are at intermediate or high risk of developing foot ulcers to get shoes from a qualified expert to make sure they fit, protect, and take into account their unique foot shape.
- Encourage diabetics who are at intermediate or high risk of developing foot ulcers to always wear their shoes, both inside and outside.
- Encourage diabetics who are at intermediate or high risk of developing foot ulcers, as well as their family members and caregivers, to examine their feet. Every time you put on your shoes, make sure there are no foreign items inside or piercing the soles. feet, every time their shoes are taken off, to make sure there aren't any indications of unusual pressure, injury, or ulceration
- Medical-grade footwear, such as specially designed insoles or orthoses, should be prescribed for patients with foot deformities or pre-ulcerative lesions.
- Provide medical-grade shoes with specially designed insoles or orthoses that have been shown to reduce plantar pressure in the high-risk areas for patients who have healed plantar foot ulcers.

For people with Diabetic Ulceration[24]

- It is not particularly advised to treat plantar diabetic foot ulcers using footwear; instead, the right offloading devices should be prescribed.

Lifestyle modification plays a major in any of the disease control, before the Ulceration the diabetes is needed to controlled which is a root cause of the DFU. The first prevention strategy for the Diabetes is limiting the sugar intake, having regular exercise, Frequent exercise can help relieve stress, encourage weight loss, and lower blood sugar levels. The average adult should engage in moderate aerobic exercise, such as swimming, riding, or walking, for at least 150 minutes each week.

Weight control: It's critical to maintain a healthy weight, particularly if you have type 2 diabetes. This could be managed by reducing the calorie intake.

Stress management: People with diabetes can better control their disease by learning how to lower their stress levels. Cortisol levels are the key factors for the elevating the stress. These levels could be reduced by regular Meditation and Breathing exercise

Monitoring Alcohol Intake, Reducing the alcohol intake could reduce the complication of diabetes, a serious complication associated with diabetes is Diabetic Ketoacidosis (DKD) and also Lactic-Acidosis.

Quitting smoking is essential for people who are more susceptible to diabetic foot ulcers. Foot ulcers can form and worsen as a result of smoking's effects on circulation, oxygen delivery to the extremities, and wound healing. It can also make peripheral neuropathy worse and make infections more likely. Giving up smoking will greatly increase blood flow and general health, which will help the body repair wounds more quickly. This can lower the risk of consequences like foot ulcers for those with diabetes. Support groups, behavioural therapy, and nicotine replacement treatment are among methods for quitting smoking. All things considered, giving up smoking is an essential first step toward improved health and the avoidance of complications from diabetic foot.

VI. MANAGEMENT OF DFU:

There is a vital need of management of diabetic foot ulcers in order to prevent infection, improve healing and to decrease the risk of amputations. Management is done by debridements, pressure modulation by offloading techniques, wound dressing, Surgery etc

6.1 DEBRIDEMENT:

Debridement, means removal of foreign and infected materials from a wound management along with necrotic and senescent tissues, is considered may need to be the first and most essential therapeutic step and a reduction in the prospect that patients with DFU may need to amputate a limb[26]. Debridement appears to increase the synthesis of local growth factors while decreasing the number of bacteria. Moreover, this method lowers pressure, analyses the wound bed, and promotes wound drainage.[27]

the various types of Debridement can be performed are surgical, enzymatical, autolytical, mechanical, or biological. Of these techniques, it has been stated that surgical debridement is more productive in DFU healing.[28]

Table: 3 Debridement methods for Diabetic foot ulcers[29]

Method	description	Advantages	Disadvantages
Surgical or Sharp	Callus, nonviable tissues, and bone are removed with sterile tools until healthy tissue is visible.	economical; just needs simple surgical instruments.	needs skill to prevent the wound from getting bigger
Mechanical	methods such as pulsed lavage, high-pressure irrigation, wet-to-dry dressings, and hydrotherapy to wash wounds prior to additional debridement procedures.	removes necrotic tissue that has hardened effectively.	Not selective; can cause pain; may damage granulating tissue.

Enzymatic	usage of enzymes that break down proteins in necrotic tissue, such as streptokinase and streptodornase.	applying directly to necrotic regions.	Systemic absorption risk; not recommended for some patients; expensive.
Autolytic	Natural debridement that takes place in an environment of moist wounds with appropriate blood flow and drainage.	Cost-effective; suitable for extremely painful wounds.	time-consuming; prolonged care might be necessary.
Biological or MDT	application of Lucilia sericata, sterile maggots, to consume necrotic tissue while protecting healthy tissue.	distinguishes between necrotic and healthy tissue with effectiveness.	Potential resistance from medical professionals and patients; costly.

Surgical or sharp debridement is the removal of diseased and dead tissue, followed by a daily application of cotton gauze that has been moistened with saline. This kind of debridement is primarily intended to initiate an acute ulcer from a chronic one. Repeated surgical debridement is necessary if new necrotic tissue keeps forming.[29]

Maggot debridement therapy (MDT), sometimes referred to as larval therapy or maggot therapy, is an older form of debridement that falls under the biological debridement category. This method involves applying live, sterile Lucilia sericata larvae to the wound in order to debride, disinfect, and eventually heal it. This method is recommended for open wounds and ulcers with necrotic or gangrenous tissues, whether there is infection or not[30]

6.2 PRESSURE MODULATION:

The most crucial element in the treatment of diabetic patients' neuropathic ulcers is the application of offloading techniques, which is also known as pressure modulation. Evidence from recent studies shows that appropriate unloading improves DFU healing[31]. Few studies concentrate on the frequency and rate of wound healing with some of the commonly used clinical offloading techniques, despite the fact that many are currently in use. One of the most effective offloading techniques for the treatment of neuropathic DFU is total contact casts (TCC)[31]

6.3 WOUND DRESSING:

The finding of new dressings over the past few decades has been a significant advancement in DFU management. Dressings should ideally provide oxygen permeability, growth factor stimulation, moisture balance, protease sequestration, antimicrobial activity, and the ability to encourage autolytic debridement, which aids in the formation of granulation tissues and the re-epithelialization process. In the case of treatment with drugs, it should also have a longer duration of action, high efficacy, and enhanced sustained drug release. As a result, no single dressing can satisfy every need of a diabetic patient suffering from a foot ulcer. The causes of DFU, wound location, depth, quantity of scar or slough, exudates, state of wound margins, presence of infection, and pain all play a significant role in choice of dressing.[32]

There are three types of wound dressing: passive, active, and interactive. Because they provide enough protection and absorb a manageable volume of exudates, passive dressings are utilized for both defensive purposes and acute wounds. By promoting cellular activity and the release of growth factors, active and interactive dressings can alter the physiology of a wound[33]

The following are the primary classifications of dressings used for DFU: films, foams, alginates, hydrogels, hydrocolloids, and silver-impregnated (table 4)

Table: 4 Types of wound dressings

Type	Example	Notes	Advantages	Disadvantages
Hydrocolloids[34]	Granuflex, Duoderm	Gel-forming hydrocolloid matrix for moist healing	Long lasting, absorbent, and helps with autolysis**.**	risk of infected wounds, maceration, and Odor**.**
Hydrogels[35]	Aquaform, Aquaflo	Gels high in water content that absorb or rehydrate wounds.	absorbent, provides wetness, promotes autolysis.	Risk of infected wounds macerating.
Films[36]	Opsite	Thin, transparent layer; used in conjunction with other dressings	cheap and simple to work with.	Non-absorbent, may require wetting to remove; not recommended for infected wounds.
Alginates[35]	Sorbalgon Calcium Alginate	Highly absorbent and gel-forming substances	Vascular and bactericidal	Might require moistening
Foams[36]	Biatain, Tegaderm	Polyurethane foam that absorbs and holds onto moisture	Extremely protective and absorbent	Possibly bulky and dermatitis-causing
Silver impregnated[35]	Acticoat and Urgosorb Silver	Antimicrobial Wound Dressings	Antiseptic, Odor-Reduction	High Price

SURGERY:

Surgery will be necessary for almost all patients with severe infections and for some individuals with intermediate illnesses. In order to compress limb-threatening abscesses and remove nonviable tissue, a limb preservation group is required to be capable of performing both bedside and intraoperative incision and debridement. This component of limb preservation should be of interest and expertise to the surgeons providing this treatment. Revascularization should be performed as soon as the primary infection has been sufficiently treated if inadequate blood supply to the extremities limits the delivery of oxygen or antibiotics. Thus, immediate infection drainage—with or without partial foot amputation—takes preference over revascularization. [37] Diabetic lower limb revascularization has been utilized to treat limb salvage and incapacitating claudication. However, the following factors make the long-term results unsatisfactory: Firstly, diabetic lower limb ischemia is a multilayer arterial occlusive disease. Secondly, distal arteries are typically affected. Lastly, diabetic patients frequently have coronary and cerebral artery disease.[38]

The primary objective of nonvascular foot surgery is to treat abnormalities that raise plantar pressure using elective, preventive, curative, and emergency procedures.[39](table:5)

Table:5 types of nonvascular diabetic foot surgery

Type	Description	Examples
Elective	reconstructive surgeries on non-neuropathic individuals.	osteotomy, Achilles lengthening (TAL), etc.
Prophylactic	therapeutic methods for neuropathic individuals who do not have a wound to lower the risk of ulcers or re-ulceration.	Keller arthroplasty, TAL, Ex-ostectomy etc.
Curative	techniques that assist in the healing of open injuries.	Metatarsal head resection, Keller arthroplasty, toe amputation, etc.
Emergent	techniques to stop or slow the spread of an infection.	Incision & drainage, open amputation, fasciotomy, etc.

In one trial, participants were randomly assigned to one of two groups: one that received TCC alone, and the other that received treatment for Achilles Tendon-Lengthening (ATL) and TCC. According to their findings, all ulcers in the ATL group cured, and at seven months and two years, the probability of ulcer recurrence was 75% and 52% lower, respectively, than in the TCC group.[39]

VII. CONCLUSION:

Diabetic foot ulcers (DFU) must be prevented and managed with a thorough, observation and strategies that takes into account the condition's immediate risk factors as well as its underlying causes. Blood glucose management, healthy weight maintenance, regular exercise, stopping smoking, and managing comorbid disorders including dyslipidaemia and hypertension are the first steps toward effective prevention. Education is essential in ensuring that patients understand the significance of early medical attention for any indications of foot issues Vascular evaluations and foot examinations are crucial for early high-risk patient identification, and orthotics and customized footwear help relieve pressure and stop additional injuries. Amputations and infections can be avoided with early and efficient management of DFUs, which includes debridement, offloading techniques, advanced wound dressings, and, if required, surgical interventions. Healthcare providers play an equally significant role in promoting open communication and offering coordinated care, guaranteeing that patients receive customized care that is suitable to their special needs. Not only the incidence of DFUs be decreased by combining these management and prevention techniques, but overall patient outcomes, complications, and quality of life can be vastly improved.

REFERENCES:

[1]. McDermott, K., Fang, M., Boulton, A.J., Selvin, E. and Hicks, C.W., 2023. Etiology, epidemiology, and disparities in the burden of diabetic foot ulcers. Diabetes Care, 46(1), pp.209-221.
[2]. Stancu, B., Ilyés, T., Farcas, M., Coman, H.F., Chiș, B.A. and Andercou, O.A., 2022. Diabetic foot complications: a retrospective cohort study. International Journal of Environmental Research and Public Health, 20(1), p.187.
[3]. Andersen, C.A. and Roukis, T.S., 2007. The diabetic foot. Surgical Clinics of North America, 87(5), pp.1149-1177.
[4]. Lim, J.Z.M., Ng, N.S.L. and Thomas, C., 2017. Prevention and treatment of diabetic foot ulcers. Journal of the Royal Society of Medicine, 110(3), pp.104-109.
[5]. de Brito Belchior, A., Nascimento, F.G., de Sousa, M.C., da Silveira, A.B.M. and de Oliveira, S.K.P., 2023. Instruments for assessing foot self-care of people with diabetes: a scoping review. Revista Brasileira de Enfermagem, 76(3).
[6]. Kalish, J. and Hamdan, A., 2010. Management of diabetic foot problems. Journal of vascular surgery, 51(2), pp.476-486.

[7]. Jiang, P., Li, Q., Luo, Y., Luo, F., Che, Q., Lu, Z., Yang, S., Yang, Y., Chen, X. and Cai, Y., 2023. Current status and progress in research on dressing management for diabetic foot ulcer. Frontiers in Endocrinology, 14, p.1221705.
[8]. Hicks, C.W. and Selvin, E., 2019. Epidemiology of peripheral neuropathy and lower extremity disease in diabetes. Current diabetes reports, 19, pp.1-8.
[9]. Pop-Busui, R., Boulton, A.J., Feldman, E.L., Bril, V., Freeman, R., Malik, R.A., Sosenko, J.M. and Ziegler, D., 2017. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes care, 40(1), p.136.
[10]. Kumar, S., Ashe, H.A., Parnell, L.N., Fernando, D.J.S., Tsigos, C., Young, R.J., Ward, J.D. and Boulton, A.J.M., 1994. The prevalence of foot ulceration and its correlates in type 2 diabetic patients: a population based study. Diabetic medicine, 11(5), pp.480-484.
[11]. Noor, S., Zubair, M. and Ahmad, J., 2015. Diabetic foot ulcer—a review on pathophysiology, classification and microbial etiology. Diabetes & Metabolic Syndrome: Clinical Research & Reviews, 9(3), pp.192-199.
[12]. Soyoye, D.O., Abiodun, O.O., Ikem, R.T., Kolawole, B.A. and Akintomide, A.O., 2021. Diabetes and peripheral artery disease: A review. World journal of diabetes, 12(6), p.827.
[13]. Lapolla, A., Piarulli, F., Sartore, G., Ceriello, A., Ragazzi, E., Reitano, R., Baccarin, L., Laverda, B. and Fedele, D., 2007. Advanced glycation end products and antioxidant status in type 2 diabetic patients with and without peripheral artery disease. Diabetes care, 30(3), pp.670-676.
[14]. Lipsky, B.A., Berendt, A.R., Cornia, P.B., Pile, J.C., Peters, E.J., Armstrong, D.G., Deery, H.G., Embil, J.M., Joseph, W.S., Karchmer, A.W. and Pinzur, M.S., 2012. 2012 Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clinical infectious diseases, 54(12), pp.e132-e173.
[15]. Syauta, D., Mulaward, P., Prihantono, P., Hendarto, J., Mariana, N., Sulmiati, S., Kusumanegara, J., & Faruk, M. (2020). Risk factors affecting the degree of diabetic foot ulcers according to Wagner classification in diabetic foot patients. Annals of Medicine and Surgery, 60, 183-187. https://doi.org/10.1016/j.mcpsp.2021.100231
[16]. Mavrogenis, A. F., Megaloikonomos, P. D., Antoniadou, T., Igoumenou, V. G., Panagopoulos, G. N., Dimopoulos, L., Moulakakis, K. G., Sfyroeras, G. S., & Lazaris, A. (2018). Current concepts for the evaluation and management of diabetic foot ulcers. EFORT open reviews, 3(9), 513–525.
[17]. https://doi.org/10.1302/2058-5241.3.180010
[18]. Matheson, E.M., Bragg, S.W. and Blackwelder, R.S., 2021. Diabetes-related foot infections: diagnosis and treatment. American family physician, 104(4), pp.386-394.
[19]. Bader MS. Diabetic foot infection. Am Fam Physician. 2008;78(1):71-79.https://pubmed.ncbi.nlm.nih.gov/18649613/
[20]. Syauta, D., Mulaward, P., Prihantono, P., Hendarto, J., Mariana, N., Sulmiati, S., Kusumanegara, J., & Faruk, M. (2020). Risk factors affecting the degree of diabetic foot ulcers according to Wagner classification in diabetic foot patients. Annals of Medicine and Surgery, 60, 183-187. https://doi.org/10.1016/j.mcpsp.2021.100231
[21]. Mavrogenis, A. F., Megaloikonomos, P. D., Antoniadou, T., Igoumenou, V. G., Panagopoulos, G. N., Dimopoulos, L., Moulakakis, K. G., Sfyroeras, G. S., & Lazaris, A. (2018). Current concepts for the evaluation and management of diabetic foot ulcers. EFORT open reviews, 3(9), 513–525.
[22]. https://doi.org/10.1302/2058-5241.3.180010
[23]. Matheson, E. M., Bragg, S. W., & Blackwelder, R. S. (2021). Diabetes-Related Foot Infections: Diagnosis and Treatment. American family physician, 104(4), 386–394.
[24].
https://pubmed.ncbi.nlm.nih.gov/34652105/
[26]. Bader MS. Diabetic foot infection. Am Fam Physician. 2008;78(1):71-79.
[27]. https://pubmed.ncbi.nlm.nih.gov/18649613/
[28]. Tallis, A., Motley, T.A., Wunderlich, R.P., Dickerson Jr, J.E., Waycaster, C., Slade, H.B. and Collagenase Diabetic Foot Ulcer Study Group, 2013. Clinical and economic assessment of diabetic foot ulcer debridement with collagenase: results of a randomized controlled study. Clinical therapeutics, 35(11), pp.1805-1820.
[29]. Yazdanpanah, L., Nasiri, M. and Adarvishi, S., 2015. Literature review on the management of diabetic foot ulcer. World journal of diabetes, 6(1), p.37.
[30]. Chen, P., Vilorio, N.C., Dhatariya, K., Jeffcoate, W., Lobmann, R., McIntosh, C., Piaggesi, A., Steinberg, J., Vas, P., Viswanathan, V. and Wu, S., 2024. Guidelines on interventions to enhance healing of foot ulcers in people with diabetes (IWGDF 2023 update). Diabetes/Metabolism Research and Reviews, 40(3), p.e3644.
[31]. Edwards, J. and Stapley, S., 2010. Debridement of diabetic foot ulcers. Cochrane Database of systematic reviews, (1).
[32]. Paul, A.G., Ahmad, N.W., Lee, H.L., Ariff, A.M., Saranum, M., Naicker, A.S. and Osman, Z., 2009. Maggot debridement therapy with Lucilia cuprina: a comparison with conventional debridement in diabetic foot ulcers. International wound journal, 6(1), pp.39-46.
[33]. Armstrong, D.G., Nguyen, H.C., Lavery, L.A., Van Schie, C.H., Boulton, A.J. and Harkless, L.B., 2001. Off-loading the diabetic foot wound: a randomized clinical trial. Diabetes care, 24(6), pp.1019-1022.
[34]. Moura, L.I., Dias, A.M., Carvalho, E. and de Sousa, H.C., 2013. Recent advances on the development of wound dressings for diabetic foot ulcer treatment—A review. Acta biomaterialia, 9(7), pp.7093-7114.
[35]. Fu, T., Stupnitskaia, P. and Matoori, S., 2022. Next-generation diagnostic wound dressings for diabetic wounds. ACS Measurement Science Au, 2(5), pp.377-384.
[36]. Dumville, J.C., Deshpande, S., O'Meara, S. and Speak, K., 2013. Hydrocolloid dressings for healing diabetic foot ulcers. Cochrane Database of Systematic Reviews, (8).
[37]. Hilton, J.R., Williams, D.T., Beuker, B., Miller, D.R. and Harding, K.G., 2004. Wound dressings in diabetic foot disease. Clinical Infectious Diseases, 39(Supplement_2), pp.S100-S103.
[38]. Alven, S., Peter, S., Mbese, Z. and Aderibigbe, B.A., 2022. Polymer-based wound dressing materials loaded with bioactive agents: Potential materials for the treatment of diabetic wounds. Polymers, 14(4), p.724.
[39]. Wukich, D.K., Armstrong, D.G., Attinger, C.E., Boulton, A.J., Burns, P.R., Frykberg, R.G., Hellman, R., Kim, P.J., Lipsky, B.A., Pile, J.C. and Pinzur, M.S., 2013. Inpatient management of diabetic foot disorders: a clinical guide. Diabetes care, 36(9), pp.2862-2871.
[40]. Gu, Y.Q., 2010. Vascular surgery and diabetic foot revascularization. Chinese medical journal, 123(15), pp.2116-2119.
[41]. Frykberg, R.G., Wukich, D.K., Kavarthapu, V., Zgonis, T., Dalla Paola, L. and Board of the Association of Diabetic Foot Surgeons, 2020. Surgery for the diabetic foot: A key component of care. Diabetes/metabolism research and reviews, 36, p.e3251.