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Diabetic Foot Ulcer Overview: A Complication Care Study

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ABSTRACT

Diabetic foot complications are contributing to both mortality and morbidity among the diabetic population leading to physical, physiological and financial burden for the patients and community. The risk of ulceration and amputation among diabetic patient's increases by two to four folds with the progression of age and duration of diabetes regardless of the type of diabetes Foot ulceration is a preventable condition, where simple interventions can reduce amputations by up to 70% through programs that could reduce its risk factors. The outcomes of education on foot self-care practices among patients with diabetes depend on the type of education provided. The management of diabetic foot ulcers remains a major therapeutic challenge which implies an urgent need to review strategies and treatments in order to achieve the goals and reduce the burden of care. Prevention of diabetic foot ulceration is critical in order to reduce the associated high morbidity and mortality rates, and the danger of amputation. It is essential to identify the "foot at risk," through careful inspection and physical examination of the foot followed by neuropathy and vascular tests. Regular foot examination, patient education, simple hygienic practices, provision of appropriate footwear, and prompt treatment of minor injuries can decrease ulcer occurrence by 50% and eliminate the need for major amputation.These key educational elements for diabetes patients at low risk of complications are captured with CARE.

Keywords: Diabetic foot complications, foot self-care practices, amputation, risk of ulceration.

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INTRODUCTION

Diabetes mellitus (DM, is a group of metabolic disorders in which there are high blood sugar levels over a prolonged period [1].

Diabetes is due to either the pancreas not producing enough insulin or the cells of the body not responding properly to the insulin produced [2].

There are three main types of diabetes mellitus:

- Type 1 DM results from the pancreas's failure to produce enough insulin. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". The cause is unknown.
- Type 2 DM begins with insulin resistance, a condition in which cells fail to respond to insulin properly. As the disease progresses a lack of insulin may also develop. This form was previously referred to as "non insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes". The most common cause is excessive body weight and insufficient exercise.
- Gestational diabetes is the third main form, and occurs when pregnant women without a previous history of diabetes develop high blood sugar levels[3].

Comparison of type 1 and 2 diabetes [4]

Feature	Type 1 diabetes	Type 2 diabetes
Onset	Sudden	Gradual
Age at onset	Mostly in children	Mostly in adults
Ketoacidosis	Common	Rare
Autoantibodies	Usually present	Absent
Endogenous insulin	Low or absent	Normal, decreased or increased
Concordance in identical twins	50%	90%
Prevalence	~10%	~90%

Epidermiology

Diabetes mellitus occurs throughout the world, but is more common (especially type 2) in the more developed countries. The greatest increase in prevalence is, however, occurring in low- and middle-income countries [5].

Diabetes prevalence is increasing rapidly; previous 2013 estimates from the International Diabetes Federation put the number at 381 million people having diabetes. The number is projected to almost double by 2030[6,7].

India had more diabetics than any other country in the world, according to the International Diabetes Foundation. The average age on onset is 42.5 years. Nearly 1 million Indians die due to diabetes every year[8].

Diabetes currently affects more than 62 million Indians, which is more than 7.1% of the adult population [9].

SIGNS AND SYPTOMS

The classic symptoms of untreated diabetes are weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger) [10].

Symptoms may develop rapidly (weeks or months) in type 1 DM, while they usually develop much more slowly and may be subtle or absent in type 2 DM.Several other signs and symptoms can mark the onset of diabetes although they are not specific to the disease. In addition to the known ones above, they include blurry vision, headache, fatigue, slow healing of cuts, and itchy skin. Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes [11].

DIAGNOSIS

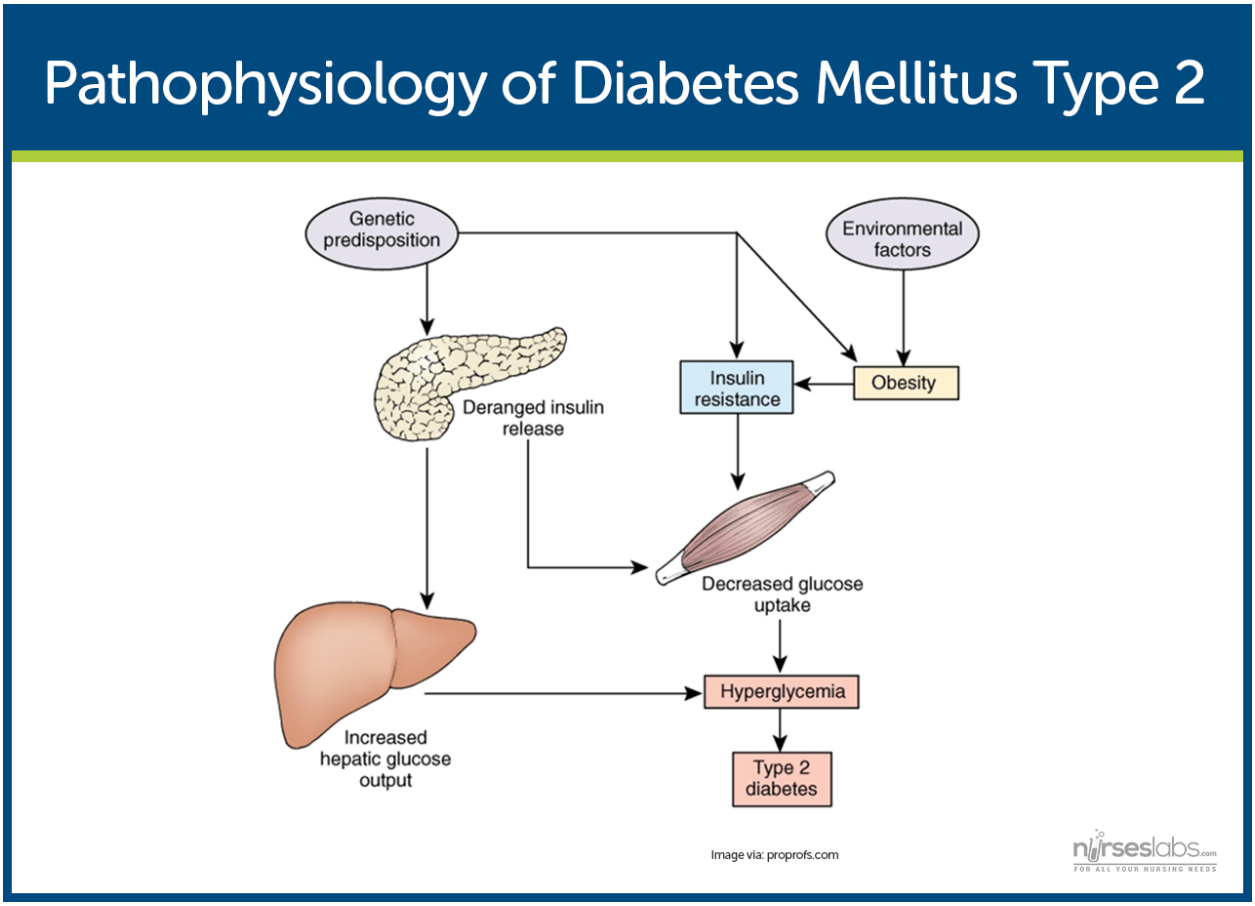
Diabetes mellitus is characterized by recurrent or persistent high blood sugar, and is diagnosed by demonstrating any one of the following[12]:

- Fasting plasma glucose level ≥ 7.0 mmol/l (126 mg/dl)
- Plasma glucose ≥ 11.1 mmol/l (200 mg/dl) two hours after a 75 g oral glucose load as in a glucose tolerance test
- Symptoms of high blood sugar and casual plasma glucose ≥ 11.1 mmol/l (200 mg/dl)
    - Glycated hemoglobin (HbA_{1C}) ≥ 48 mmol/mol (≥ 6.5 DCCT %) [13].

WHO diabetes diagnostic criteria [14,15]

Condition	**2 hour glucose**	**Fasting glucose**	**HbA_{1c}**	
Unit	mmol/l(mg/dl)	mmol/l(mg/dl)	mmol/mol	DCCT %
Normal	<7.8 (<140)	<6.1 (<110)	<42	<6.0
Impaired fasting glycaemia	<7.8 (<140)	≥6.1(≥110) & <7.0(<126)	42-46	6.0–6.4
Impaired glucose tolerance	≥7.8 (≥140)	<7.0 (<126)	42-46	6.0–6.4
Diabetes mellitus	≥11.1 (≥200)	≥7.0 (≥126)	≥48	≥6.5

PATHOPHYSIOLOGY

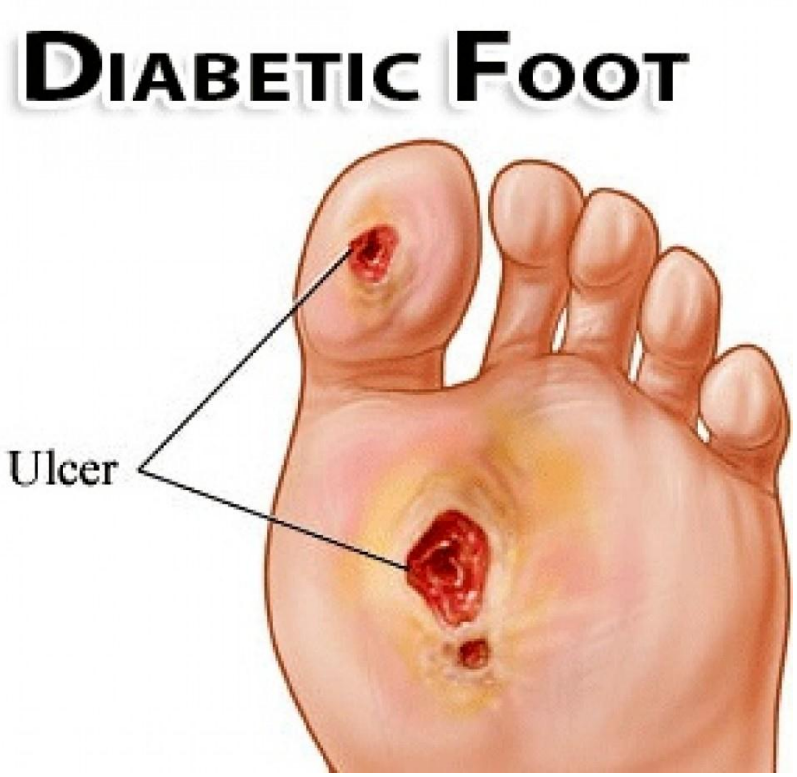


EVALUATION OF THERAPEUTIC OUTCOMES

The A1C is the current standard for following long-term glycemic control for the previous 3 months. It should be measured at least twice a year in patients meeting treatment goals on a stable therapeutic regimen. • Regardless of the insulin regimen chosen, gross adjustments in the total daily insulin dose can be made based on A1C measurements and symptoms such as polyuria, polydipsia, and weight gain or loss. Finer insulin adjustments can be determined on the basis of the results of frequent SMBG. • Patients receiving insulin should be questioned about the recognition of hypoglycemia at least annually. Documentation of frequency of hypoglycemia and the treatment required should be recorded. • Patients receiving bedtime insulin should be monitored for hypoglycemia by asking about nocturnal sweating, palpitations, and nightmares, as well as the results of SMBG. • Patients with type 2 DM should have a routine urinalysis at diagnosis as the initial screening test for albuminuria. If positive, a 24-hour urine for quantitative assessment will assist in developing a treatment plan. If the urinalysis is negative for protein, a test to evaluate the presence of microalbuminuria is recommended. • Fasting lipid profiles should be obtained at each follow-up visit if not at goal, annually if stable and at goal, or every 2 years if the profile suggests low risk. • Regular frequency of foot exams (each visit), urine albumin assessment (annually), and

dilated ophthalmologic exams (yearly or more frequently with abnormalities) should also be documented. • Assessment for influenza and pneumococcal vaccine administration and assessment and management of other cardiovascular risk factors (e.g., smoking and antiplatelet therapy) are components of sound preventive medicine strategies[17].

DIABETIC FOOT ULCER



Diabetic foot ulcer is a major complication of diabetes mellitus, and probably the major component of the diabetic foot. A key feature of wound healing is stepwise repair of lost extracellular matrix (ECM) that forms the largest component of the dermal skin layer [18].

Diabetic foot complications are contributing to both mortality and morbidity among the diabetic population leading to physical, physiological and financial burden for the patients and community [21]. The risk of ulceration and amputation among diabetic patients increases by two to four folds with the progression of age and duration of diabetes regardless of the type of diabetes [22] Foot ulceration is a preventable condition, where simple interventions can reduce amputations by up to 70%through programs that could reduce its risk factors [23].

People with diabetes often develop diabetic neuropathy due to several metabolic and neurovascular factors. Peripheral neuropathy causes loss of pain or feeling in the toes, feet, legs and arms due to distal nerve damage and low blood flow. Blisters and sores appear on numb areas of the feet and legs such as metatarso-phalangeal joints, heel region and as a result pressure or injury goes

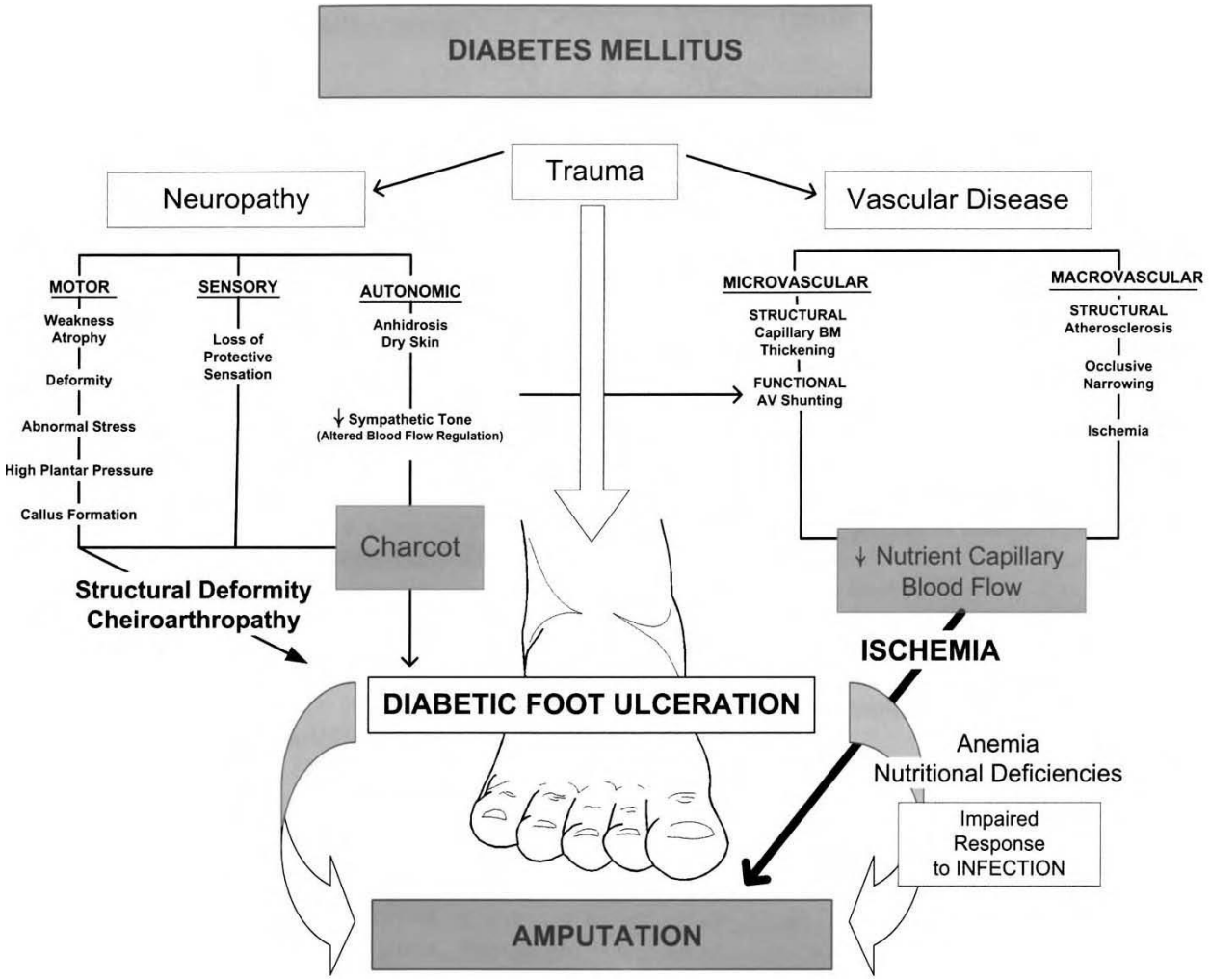
unnoticed and eventually become portal of entry for bacteria and infection. Risk factors implicated in the development of diabetic foot ulcers are infection, older age [24]diabetic neuropathy, peripheral vascular disease, cigarette smoking, poor glycemic control, previous foot ulcerations [25]

DIGONOSIS

Identification of diabetic foot in medical databases, such as commercial claims and prescription data, is complicated by the lack of a specific ICD-9 code for diabetic foot and variation in coding practices. The following codes indicate ulcer of the lower limb or foot:

- 707.1 Ulcer of lower limbs, except pressure ulcer
- 707.14 Ulcer of heel and midfoot
- 707.15 Ulcer of other part of foot
- 707.19 Ulcer of other part of lower limb

PATHOPHYSIOLOGY: [26]



One or more codes, in combination with a current or prior diagnosis of diabetes may be sufficient to conclude diabetic foot:

- 250.0 Diabetes Mellitus
- 250.8 Diabetes with other specified manifestations

TREATMENT

The gold standard for diabetic foot ulcer treatment includes debridement of the wound, management of any infection, revascularization procedures when indicated, and off-loading of the ulcer [27].

Debridement

Debridement should be carried out in all chronic wounds to remove surface debris and necrotic tissues. It improves healing by promoting the production of granulation tissue and can be achieved surgically, enzymatically, biologically, and through autolysis.

Surgical debridement, known also as the "sharp method," is performed by scalpels, and is rapid and effective in removing hyperkeratosis and dead tissue. Particular care should be taken to protect healthy tissue, which has a red or deep pink (granulation tissue) appearance [28].

Dressings

Ulcers heal more quickly and are often less complicated by infection when in a moist environment. The only exception is dry gangrene, where the necrotic area should be kept dry in order to avoid infection and conversion to wet gangrene. A wound's exudate is rich in cytokines, platelets, white blood cells, growth factors, matrix metalloproteinases (MMPs), and other enzymes. Most of these factors promote healing via fibroblast and keratinocyte proliferation and angiogenesis, while others, such as leukocytes and toxins produced by bacteria, inhibit the healing process. Moreover, it has been reported that local concentrations of growth factors [platelet-derived growth factor-beta (PDGF-beta), transforming growth factor-beta] are low in patients with chronic ulcers [29].

Negative-Pressure Wound Therapy

Negative-pressure wound therapy (NPWT) has emerged as a new treatment for diabetic foot ulcers. It involves the use of intermittent or continuous subatmospheric pressure through a special pump (vacuum-assisted closure) connected to a resilient open-celled foam surface dressing covered with an adhesive drape to maintain a closed environment. The pump is connected to a canister to collect wound discharge and exudate. Experimental data suggest that NPWT optimizes blood flow, decreases tissue edema, and removes exudate, proinflammatory cytokines, and bacteria from the wound area.It should be performed after debridement and continued until the formation of

healthy granulation tissue at the surface of the ulcer. Currently, NPWT is indicated for complex diabetic foot wounds [30].

Antibiotics

The length of antibiotic courses depend on the severity of the infection and whether bone infection is involved but can range from 1 week to 6 weeks or more. Current recommendations are that antibiotics are only used when there is evidence of infection and continued until there is evidence that the infection has cleared, instead of evidence of ulcer healing. Choice of antibiotic depends on common local bacterial strains known to infect ulcers. Microbiological swabs are believed to be of limited value in identifying causative strain.[7] Microbiological investigation is of value in cases of osteomyelitis [31].

Wound dressings

There are many types of dressings used to treat diabetic foot ulcers such as absorptive fillers, hydrogel dressings, and hydrocolloids. Biologically active bandages that combine hydrogel and hydrocolloid traits are available, however more research needs to be conducted as to the efficacy of this option over others [32].

Other treatments

Ozone therapy – there is only limited and poor-quality information available regarding the effectiveness of ozone therapy for treating foot ulcers in people with diabetes [33].

Growth factors - there is some low-quality evidence that growth factors may increase the likelihood that diabetic foot ulcers will heal completely [34].

DIABETIC FOOT CARE

Annual foot screening attendance

All patients with diabetes should expect to receive an annual foot screening examination by an appropriately trained healthcare professional and they should feel confident enough to ensure that this takes place. At each step of the foot screening examination, the patient should be told what the healthcare professional is checking or testing for; for example, that testing for sensation is performed to check for nerve damage, rather than for poor circulation [35,36,37].

Glycaemic control

Numerous clinical studies have demonstrated the positive relationship between reductions in HbA_{1c} and the reduced risk of microvascular complications of diabetes, including neuropathy and foot ulcers [38,39]. This relationship needs to be explained to patients in a language they can

understand, together with tackling misconceptions, such as amputation being an inevitable consequence of having diabetes and the link between neuropathy and ulceration.

Checking their own feet

Demonstrations and visualizations of foot complications and self-care practices may therefore help to engage and empower patients and reinforce that early detection of problems or changes by themselves is key to preventing serious complications [40].

Reporting changes in their feet

Perhaps just as importantly as checking their feet, patients also need to be aware that, if they find changes in the colour of the skin, skin breaks, skin swelling, or if they feel pain or numbness in their feet, they should alert their general practitioner or other healthcare professional promptly.

CONCLUSION

The outcomes of education on foot self-care practices among patients with diabetes depend on the type of education provided.

The management of diabetic foot ulcers remains a major therapeutic challenge which implies an urgent need to review strategies and treatments in order to achieve the goals and reduce the burden of care. Prevention of diabetic foot ulceration is critical in order to reduce the associated high morbidity and mortality rates, and the danger of amputation. It is essential to identify the "foot at risk," through careful inspection and physical examination of the foot followed by neuropathy and vascular tests. Regular foot examination, patient education, simple hygienic practices, provision of appropriate footwear, and prompt treatment of minor injuries can decrease ulcer occurrence by 50% and eliminate the need for major amputation.

These key educational elements for diabetes patients at low risk of complications are captured with

Care:

Control: control blood glucose levels

Annual: attend your annual foot screening examination

Report: report any changes in your feet immediately to your healthcare professional.

Engage: engage in a simple daily foot care routine

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