SEVEN
publicações acadêmicas

Prevention techniques and effective management of diabetic foot: A literature review

https://doi.org/10.56238/innovhealthknow-042

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ABSTRACT
In the context of diabetic foot, peripheral neuropathy plays a crucial role. The loss of sensitivity and muscle strength in the feet due to neuropathy increases the risk of ulcers. Additionally, peripheral vascular disease and immunodeficiency are risk factors, making diabetic patients more susceptible to complications. Diabetic foot prevention involves a multidisciplinary approach, including regular foot exams, self-care education, lifestyle changes such as quitting smoking, keeping the feet clean and dry, wearing appropriate socks and comfortable shoes. Local care, including ulcer debridement and the proper choice of dressings, is essential for healing. Negative pressure therapy may be an option in severe cases. Furthermore, other conditions, such as Charcot arthropathy, venous stasis, deep vein thrombosis, and fractures, can be confused with diabetic foot infections and require differentiation through medical history, physical examination, and imaging studies. In summary, diabetes mellitus is a global health concern, with serious complications like diabetic foot. Prevention, education, and proper treatment play fundamental roles in managing this condition.

Keywords: Diabetic foot, Self-care, Prevention, Complications, Multidisciplinary.

1 INTRODUCTION

Type 1 diabetes or autoimmune diabetes. However, some patients with an absolute deficiency of insulin do not show evidence of autoimmunity, and they do not have any other known cause for the destruction of beta cells. The classification system of the American Diabetes Association (ADA) uses the term "idiopathic diabetes" or "Type 1B" to refer to these patients, who may encompass a range of non-autoimmune pathophysiological processes that lead to almost complete loss of beta cell function (COSTA; MOREIRA, 2021).

Type 2 diabetes is the most common phenotypic form of diabetes. Diabetes mellitus (DM) is a growing problem worldwide, regardless of the developmental status of the countries. In Brazil,

approximately 12.5 million people have DM, with an estimated progression to around 20.3 million comorbidity cases by 2045 (BRASIL, 2019).

With the significant increase in DM cases, complications inherent to the condition are also advancing, including foot-related disorders. Diabetic foot is a wound aggravated by an infection or other causes of neurological, vascular, or orthopedic origin. Typically, the problem occurs over time in comorbidity, leading to neuropathy along with poor perfusion, making patients more prone to injuries that they may not identify due to local insensitivity (MATTOS et al., 2019).

If diabetic foot is not treated properly, it naturally progresses to the development of ulcers, which can lead to amputations. Consequently, hospitalizations resulting from this complication are becoming more frequent, incurring high costs to public finances (MATTOS et al., 2019). Specialized attention is required to manage this condition and prevent the progression of diabetic foot, a complication that reduces the quality of life for patients.

2 PERIPHERAL NEUROPATHY

Pancreatic physiology is divided into two portions, one endocrine and the other exocrine, with the majority of pancreatic mass consisting mostly of acinar cells organized in acini. Acinar cells synthesize inactive digestive enzymes such as amylases, proteases, lipases, and nucleases. These enzymes are subsequently secreted into the pancreatic ducts and transported to the duodenum for activation. The duct cells produce mucus and bicarbonate-rich fluids, which are useful for neutralizing stomach acid (COSTA; MOREIRA, 2021).

The pancreas, in its endocrine function, is regulated by clusters of cells scattered within the pancreatic acinar tissue, known as the Islets of Langerhans. There are at least six types of pancreatic cells described. Among them, alpha cells make up about 15-20% of the islet cells and synthesize and secrete glucagon, glicentin, GRPP (glycentin-related pancreatic peptide), GLP-1, and GLP-2 (glucagon-like peptide 1 and 2). Beta cells are the most numerous, accounting for approximately 70-80% of the pancreatic islet cells. They are responsible for the synthesis and secretion of insulin and C-peptide, and to a lesser extent, they produce amylin, also known as IAPP (islet amyloid polypeptide), which acts as an insulin antagonist, among other peptides. Delta cells represent 5-10% of the cells (Montenegro JR et al., 2016).

Type 2 diabetes is responsible for over 90% of diabetes cases in the United States, Canada, and Europe. Type 1 diabetes accounts for another 5 to 10%, with the remaining cases attributed to specific etiological or pathophysiological factors. Known monogenic causes of diabetes (e.g., those causing maturity-onset diabetes of the young or neonatal diabetes) represent only a small fraction of cases. The genetic foundations of the common forms of Type 1 and Type 2 diabetes remain complex, with common gene variants contributing individually to only small degrees of risk or protection. A wide

range of phenotypic forms of diabetes with uncertain pathophysiology is emerging, which does not fit clearly into the Type 1 or Type 2 diabetes categories, collectively referred to as "atypical diabetes." Furthermore, the global epidemic of overweight and obesity has superimposed the pathophysiology of Type 2 diabetes on all other types (PERMUTT et al., 2019).

Type 1 diabetes is characterized by the destruction of pancreatic beta cells, leading to an absolute deficiency of insulin. This typically occurs due to autoimmune destruction of beta cells (Type 1A). Testing for islet cell antibodies (ICA) or other islet autoantibodies (antibodies to glutamic acid decarboxylase 65 [GAD65], insulin, tyrosine phosphatases, insulinoma-associated protein 2 [IA-2], IA-2 beta, and zinc transporter [ZnT8]) in the serum is important. A positive result is generally indicative of autoimmunity. Type 1 diabetes can occur in both children and adults and is characterized by hyperglycemia and varying degrees of insulin resistance and deficiency. Its prevalence increases significantly with the rise in the number of obese individuals. Insulin resistance and insulin deficiency can result from genetic or environmental influences, making it difficult to determine the exact cause in an individual patient. Furthermore, hyperglycemia itself can impair pancreatic beta cell function and worsen insulin resistance, a condition known as "glucotoxicity" (JUNIOR et al., 2020).

With the increasing prevalence of diabetes worldwide, distinguishing between Type 1 diabetes and atypical presentations of Type 2 diabetes has become increasingly challenging. Patients with Type 1 diabetes may develop an absolute need for insulin treatment over time. However, many Type 2 diabetes patients may experience beta cell dysfunction over time and require insulin for glycemic control (COSTA; MOREIRA, 2021).

Patients with Type 1 diabetes may coincidentally exhibit pathophysiological elements of Type 2 diabetes. In the past, poor metabolic control of Type 1 diabetes prevented most of these patients from experiencing weight gain. Intensive therapy now commonly used to control Type 1 diabetes has resulted in an increasingly similar prevalence of overweight and obesity in the Type 1 diabetes population and the non-diabetic population. Insulin resistance and other characteristics of Type 2 diabetes may be exhibited in such Type 1 diabetes patients, especially those with a family history of Type 2 diabetes (PENNA; HERMSDORFF; SARON, 2020).

2.1 PREVENTION OF DIABETIC FOOT ULCERS

It is estimated that 10 to 25% of the population with diabetes mellitus over the age of 70 develops foot lesions, and among them, 14 to 24% require amputation. This is a common cause of disability due to amputations, which significantly reduce the quality of life (MUZY et al., 2021).

The prevention of ulcers begins with regular foot examinations and public education, which can reduce the severe cases by half. Diabetics should frequently inspect their feet to check for the

presence of punctures, cuts, swelling, redness, excessive dryness, and discoloration (VICENTIN et al., 2020).

In addition to observation, lifestyle changes should be encouraged, such as quitting smoking, washing the feet with warm water, thorough drying of the feet including between the toes, avoiding the use of local alcohol and other substances that tend to dry the skin, cutting nails straight without leaving sharp edges, using seamless and elastic cotton socks, avoiding tight-fitting shoes, and being cautious about insects. The multidisciplinary team should guide patients with diabetes mellitus regarding the care and potential complications associated with non-compliance with appropriate treatment (RAMIREZ-PERDOMO; PERDOMO-ROMERO; RODRÍGUEZ-VÉLEZ, 2019).

Deep ulcers with chronic infections and/or prior antibiotic treatment are more likely to be caused by various pathogens. Wounds with extensive local inflammation, necrosis, foul-smelling drainage, or gangrene with signs of systemic toxicity should be presumed to have anaerobic organisms in addition to the above-mentioned pathogens. Potential pathogens include anaerobic streptococci, Bacteroides species, and Clostridium (WEINTROB et al., 2023).

Local care is an important mechanism to prevent worse outcomes. Debridement of necrotic tissue is essential for ulcer healing. The frequency of assessments and appropriate care can contribute more to wound healing than the type of debridement. Sharp debridement, the most widely used method, involves the use of a scalpel or scissors to remove necrotic tissue. Alternatively, the application of a hydrogel is suggested, but data supporting its effectiveness in promoting wound healing are limited. Enzymatic debridement (topical application of proteolytic enzymes like collagenase) may be more appropriate in certain scenarios (e.g., extensive vascular disease). Autolytic debridement can be a good option for patients with painful ulcers, using a semi-occlusive or occlusive dressing to cover the ulcer so that necrotic tissue is digested by enzymes naturally present in the wound tissue (ARAUJO; FILHO; BRANDÃO, 2022).

After debridement, ulcers should be kept clean and moist, but not excessively wet. Dressings should be selected based on ulcer characteristics, such as the extent of exudate, excessive dryness, or necrotic tissue. Some dressings simply provide protection, while others promote wound hydration or prevent excessive moisture. Adjunct therapies can help improve the healing of diabetic foot ulcers, including negative pressure wound therapy for extensive open wounds after debridement for infection and necrosis, or after partial foot amputation, provided there is no residual necrotic tissue or osteomyelitis. This technique involves the application of controlled subatmospheric pressure to the ulcer's surface, improving healing and increasing wound perfusion, reducing edema, reducing local bacterial load, and enhancing granulation tissue formation. This technique improves healing by reducing the duration of hospitalization, major complication rates, and costs (ARMSTRONG et al., 2022).

Several other conditions can lead to changes in the lower limb skin and may mimic an infection. Charcot arthropathy can be acute or chronic, and patients often present with sudden onset of unilateral warmth, redness, and swelling in the foot or ankle, often with a history of minor trauma. The affected foot may be notably warmer than the healthy contralateral foot. Alternatively, in some cases, patients may have a slow-progressing arthropathy with insidious onset of swelling over months or years. Occasionally, acute recurrent attacks may occur. The most frequently affected joints are the tarsal and tarsometatarsal joints, followed by the metatarsophalangeal joints and the ankle (MUÑOZ-DE-LA-CALLE; VIADÉ-JULIÀ, 2020).

In addition to Charcot arthropathy, venous stasis, deep vein thrombosis, and fractures can be differential diagnoses for diabetic foot. Generally, these conditions can be differentiated from infection based on clinical history, physical examination, and imaging findings. However, infection may coexist with other processes, and empirical antimicrobial therapy may be justified in some cases when the diagnosis is uncertain (WEINTROB et al., 2023).

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