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Diabetic Neuropathy: Education as a Therapeutic Strategy

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Abstract

Diabetes mellitus (DM) is a chronic and progressive non-communicable disease (NCD) characterized by increased blood glucose levels due to partial or absolute deficiency of pancreatic beta cells in producing insulin. It is estimated that by the year 2045, the number of affected people worldwide will exceed 700 million[1] and in the SACA region (South America, Central America, and the Hispanic Caribbean region of the IDF), it will reach 49 million people.

Keywords

Diabetic neuropathy; Chronic hyperglycemia; Nerve biopsy; Neuromodulators; Analgesia.

Introduction

Diabetes mellitus (DM) is a chronic and progressive non-communicable disease (NCD) characterized by increased blood glucose levels due to partial or absolute deficiency of pancreatic beta cells in producing insulin. It is estimated that by the year 2045, the number of affected people worldwide will exceed 700 million[1] and in the SACA region (South America, Central America, and the Hispanic Caribbean region of the IDF), it will reach 49 million people. DM constitutes a clinical condition of significant prevalence in our context; according to IDF-2019 (International Diabetes Federation) [1]. the age-adjusted comparative prevalence in adults aged 20 to 79 years was 7%, and the proportion of undiagnosed DM in the same age group was 51.8%; that is, 1 in 15 adults

aged 20 to 79 years has undiagnosed DM. Furthermore, diabetes caused 243,200 deaths during 2019 in adults aged 20 to 79 years in the Americas region [2]. In Venezuela, according to the IDF, the prevalence of DM in adults was 12.6% in the year 2021 [2].

Diabetic neuropathy is the most common complication of diabetes mellitus (DM), affecting up to 50% of patients with type 1 and type 2 DM. It involves the presence of symptoms or signs of peripheral nerve dysfunction in individuals with diabetes, once other possible causes have been excluded [3,4,5].

Chronic hyperglycemia is associated with secondary damage to various organs, leading to chronic complications such as nephropathy, retinopathy, neuropathy, and cardiovascular and cerebrovascular disease. However, considering the clinical and epidemiological complexity of diabetes from a holistic perspective, it is essential to take into account different biopsychosocial factors that may influence this chronic condition in a bidirectional manner [6].

The first data linking DM to an increased risk of stroke dates back to the 1970s, identifying it as one of the main risk factors. Epidemiological studies today reveal that individuals with DM have twice the risk of suffering a stroke, as well as a higher risk of recurrence after a first episode. Moreover, 30% of stroke patients have DM, whether previously diagnosed or newly detected (of which 90-95% have type 2 DM) [7].

The complexity of diabetes in its origins, clinical manifestations, acute and chronic complications, costs, and psycho-emotional and socioeconomic impact on the patient, their environment, and the public health system requires managing therapeutic aspects in a comprehensive and cyclical manner. Comprehensive management is essential because it considers the individual, their experience, and the processes as a whole, while cyclical management implies a continuous series of activities allowing the patient to acquire knowledge, skills, and abilities, and especially apply daily self-care. This fosters the generation of new knowledge, which leads to changes, innovations, and improvements in glucose values to optimize their quality of life.

Given this scenario, a plausible option to minimize its impact on the population is the management of diabetes through education and prevention, defined by the WHO [8]. as the set of measures aimed not only at preventing the onset of the disease, such as the reduction of risk factors, but also at halting its progression and mitigating its consequences once established. This goal can be achieved by providing education to at-risk patients and their family groups. From this perspective, the authors propose critically reviewing the influence of education as an auxiliary strategy in the treatment of neuropathy in patients with diabetes mellitus.

Methods

A retrospective cross-sectional narrative review was designed through the analysis of primary articles in scientific journals between the years 2015 and 2025. The search was conducted in bibliographic databases covering electronic journals in health sciences: Medline (Pubmed), Proquest, EBSCO, Virtual Health Library, and SciELO. The following descriptors were determined: diabetes mellitus, neuropathy, and therapeutic education in diabetes. The inclusion criteria for the reviewed articles were original studies of primary information or previous reviews, the content and scientific importance of the subject, methodological quality, efficacy and reliability of the information, and publications in Spanish or English.

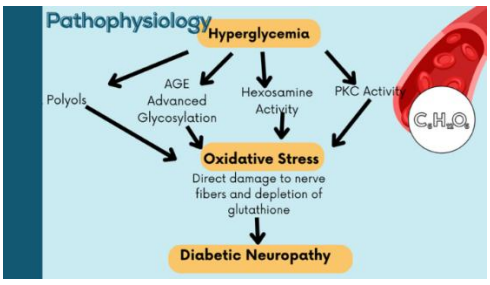
Results

The most important risk factor in the development of diabetic neuropathy (DN) is sustained hyperglycemia, accompanied by dyslipoproteinemia and obesity. Evidence in the literature shows that strict glycemic control reduces the incidence of DN in type 1 diabetes (T1D); however, data for type 2 diabetes (T2D) are not as precise [9]. In 2018, Pantalone et al [10], demonstrated that, after 10 years of T2D diagnosis, poor control or worsening glycemic control over time constitutes a contributing factor in the development of diabetic neuropathy. The coexistence of hypertension, dyslipidemia, smoking, alcohol consumption, and physical inactivity are elements to consider in favoring the onset of DN. Elevated levels of low-density lipoprotein (LDL), total cholesterol, and triglycerides have been associated with diabetic neuropathy.

Neuropathy is a disorder of the peripheral nervous system that affects the conduction of nerve impulses due to various causes. Its pathophysiology involves several mechanisms:

Persistent hyperglycemia, along with toxic substrates, affects multiple cells in the peripheral nervous system, including Schwann cells, axons, and neurons in the dorsal root ganglion, as well as vascular cells. The permanent increase in plasma glucose diverts into the polyol, hexosamine, and pyruvate pathways, and the enzymes aldose reductase and sorbitol dehydrogenase convert it into sorbitol and fructose. The accumulation of sorbitol and fructose leads to a reduction in nerve myo-inositol and the activity of membrane Na + /K + -ATPasa, resulting in impaired axonal transport and structural degradation of nerves, which causes abnormal action potential propagation, an increase in reactive oxygen species, and a loss in the production of adenosine triphosphate (ATP), as well as mitochondrial dysfunction [11]. The increased polyol flux leads to plasma hyperosmolarity due to the accumulation of sorbitol in the neuronal cytoplasm thanks to the action of aldose reductase [12]. The hexosamine pathway modifies transcription factors that promote endothelial fibrosis and atherosclerosis. The pyruvate pathway overloads the tricarboxylic acid cycle, which depletes ATP. The culmination of the pathophysiological process through these pathways will be accompanied by DNA damage, apoptosis, and activation of pro-inflammatory signals with consequent neurodegeneration13; in summary (Figure 1), we can point out that hyperglycemia promotes:

- **Axonal degeneration:** direct damage to the axon due to ischemia.
- **Demyelination:** loss of the myelin sheath, leading to defective nerve conduction.
- **Inflammation:** immune infiltration causing damage to nerve fibers.
- **Mitochondrial dysfunction:** affects ATP production, generating oxidative stress and neuronal damage.
- **Microvascular insufficiency:** pro-inflammatory environment.



In cases of focal or asymmetrical diabetic neuropathy syndromes, vascular injury or autoimmunity may play

more significant roles [14].

A cross-sectional case-control study conducted by Gastoł et al 15 indicated that in patients with Type 1 Diabetes (T1D), epigenetic factors are involved in the development of autonomic neuropathy. Patients with T1D who had autonomic neuropathy showed differences in genetic methylation compared to T1D patients without neuropathy [15].

A study by Jende et al [16], demonstrated that in patients with T1D, the predominant nerve injuries of symmetric distal diabetic neuropathy develop in relation to poor glycemic control and loss of nerve conduction, whereas in Type 2 Diabetes (T2D), these injuries arise in association with lipid metabolism changes [16].

Pai et al [17], indicated that in adults with T2D, there is an association between fasting plasma glucose variability and the risk of painful diabetic peripheral neuropathy. Using the coefficient of variation (CV) for fasting plasma glucose, the researchers found that, after considering HbA1c, the odds ratios for developing painful diabetic peripheral neuropathy were 4.08 and 5.49 for the third and fourth quartiles of CV, respectively, compared to the first quartile [17].

Dabelea et al [18], found that among adolescents and young adults diagnosed with Type 1 or Type 2 diabetes during childhood or adolescence, the age-adjusted prevalence of peripheral neuropathy was higher in those with T2D than in patients with T1D (17.7% vs. 8.5%, respectively). After adjusting for established risk factors measured over time, the odds ratio of peripheral neuropathy in T2D patients compared to those with T1D was 2,52 [18].

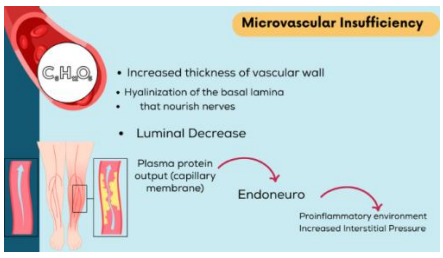
Clinical Classification

1. Based on distribution

- **Mononeuropathy:** affects a single nerve (e.g., Carpal Tunnel Syndrome).
- **Multiple Mononeuropathy:** compromises several nerves asymmetrically (e.g., Vasculitis).
- **Polyneuropathy:** affects multiple nerves symmetrically, most commonly seen in metabolic diseases like diabetes.

2. Based on the affected function

- **Sensory:** causes paresthesia, dysesthesia, and loss of sensation.
- **Motor:** weakness and muscle atrophy.
- **Autonomic:** affects involuntary functions such as sweating and blood pressure regulation



3. Based on etiology

- **Metabolic:** Diabetes mellitus.
- **Toxic:** Alcoholism, chemotherapy, heavy metals.
- **Genetic:** Charcot-Marie-Tooth.
- **Autoimmune:** Guillain-Barré syndrome.
- **Infectious:** Leprosy, HIV, herpes zoster.

Diagnosis

The diagnosis is based on a combination of:

- **Medical history and neurological examination:** evaluation of reflexes, muscle strength, and sensitivity.
- **Nerve conduction studies and electromyography (EMG):** detect alterations in nerve conduction.
- **Laboratory tests:** glucose, glycated hemoglobin, vitamin B12, kidney and liver function, and inflammatory markers.
- **Nerve biopsy:** in specific cases to evaluate inflammatory or metabolic processes.
- **Genetic studies:** for hereditary neuropathies.

Pharmacological Therapy

Treatment depends on the cause and symptoms:

- **Analgesia:** paracetamol and NSAIDs for mild pain.
- **Neuromodulators:** Gabapentin and pregabalin for neuropathic pain.
- **Tricyclic antidepressants (amitriptyline) and serotonin-norepinephrine reuptake inhibitors (duloxetine, venlafaxine):** effective in managing chronic pain.
- **Corticosteroids and immunotherapy:** for autoimmune neuropathies such as Guillain-Barré or vasculitis-related neuropathy.
- **Nutritional supplements:** vitamin B12 in cases of deficiency, alpha-lipoic acid for diabetic neuropathy.
- **Treatment of the underlying cause:** strict glucose control in diabetes, cessation of toxic substances, antiviral treatment for infections.
- Additionally, therapy should be complemented with physiotherapy and lifestyle changes to improve the patient's quality of life.

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