

# Overview of Diabetic Neuropathy: A Comprehensive Examination

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## Abstract

India has earned the unfortunate distinction of being the global epicenter for diabetes, with an alarming projection of 109 million individuals expected to be afflicted by the condition by 2035. The global influence of diabetic neuropathy is significant, with an estimated 132 million individuals affected as of 2010, constituting roughly 1.9% of the global populace. Notably, within the diabetic population, there exists a significantly heightened prevalence. The prevalence of sensory peripheral neuropathy stands at 60.4%, with an incidence rate of 8.76%. Trend is particularly concerning as individuals age, with the likelihood of neuropathy increasing over time. The pervasive nature of this disease extends beyond national borders, presenting a burgeoning global challenge that profoundly influences daily life. The repercussions of diabetic neuropathy are multifaceted, encompassing sensory impairments, motor dysfunction, and an array of complications that compromise overall well-being. Tasks as seemingly mundane as walking, grasping objects, or even maintaining balance become formidable challenges for those affected. Moreover, the socioeconomic ramifications of diabetic neuropathy are substantial, as individuals contend with reduced productivity, increased healthcare expenditures, and diminished quality of life. Furthermore, the burden extends to caregivers and healthcare systems, amplifying the strain on resources and exacerbating healthcare disparities. Essentially, the widespread and increasing occurrence of diabetic neuropathy emphasizes the critical necessity for comprehensive approaches focused on preventing, detecting early, and managing it effectively. Addressing this multifaceted challenge demands a concerted global effort, encompassing research, policy initiatives, public awareness campaigns, and accessible healthcare services. Only through collective action we can hope to mitigate the profound impact of this global health crisis and alleviate the suffering endured by millions worldwide.

**Keywords:** Diabetes capital, sensory peripheral neuropathy, global problem, repercussions, healthcare services

## INTRODUCTION

Diabetic neuropathy encompasses a diverse range of clinical conditions influenced by various contributing factors. Metabolic elements play a significant role in the development of chronic neuropathies, which often manifest gradually over time. Among the numerous forms of peripheral neuropathy, diabetic neuropathy ranks as first. It typically follows a pattern of nerve damage that ascends throughout the body, primarily affecting the sensory and motor nerves in the feet and, to a lesser extent, the hands.

During the initial stages, many individuals with peripheral neuropathy exhibit no noticeable symptoms, though subtle abnormalities may be detected upon examination, such as diminished vibration sense, light touch sensitivity, and thermal perception. As the condition progresses, patients

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commonly experience symmetrical sensations of pain, burning, tingling, and numbness in both feet, often resembling a "stocking-glove" distribution.

A key therapeutic objective in managing polyneuropathy involves preventing the need for toe and foot amputations. Despite ongoing research efforts, there is currently no definitive evidence from randomized clinical trials supporting the efficacy of disease-modifying treatments for diabetic sensory polyneuropathy.

Certain medications, including tricyclic antidepressants (such as imipramine, amitriptyline, and nortriptyline), offer moderate relief; however, dose escalation may lead to adverse effects like drowsiness, anticholinergic effects, and cardiac arrhythmia potentiation. Anticonvulsants like gabapentin and carbamazepine are also beneficial but may cause side effects such as dizziness, headaches, and diarrhea. Notably, two drugs, duloxetine (an antidepressant) and pregabalin, have been approved by the FDA for reducing neuropathic pain and have shown effectiveness in clinical trials.

## AIMS AND OBJECTIVES

To study the literature regarding the diabetic sensory polyneuropathy through modern medicine.

## CONCEPTUAL STUDY

### Historic Aspects

Historical accounts of diabetes rarely mention its neurological complications, with only occasional references to autonomic and painful neuropathies found in Eastern records. It wasn't until the 18th century that Western medical practitioners began delving into the study of diabetes and its associated complications. Subsequently, the research efforts of notable figures like de Calvi and Pavy in the 19th century definitively established the connection between diabetes mellitus and diabetic neuropathies. The groundbreaking discovery of insulin in 1921 sparked heightened interest and a more methodical approach to investigating diabetic complications [1].

### Previous Research Work

As per available information, it is found that nearly two research works on diabetic neuropathy have been reported in Banaras Hindu University. One is —Effect of *dashmūla* on diabetic neuropathy in the year 1986 and second one is —Role of *jīvanīya* and *balya* drugs in diabetic neuropathy in the year 1996. One clinical research —Effect of *atibalāmūla* and *bhūmyāmalakī* on diabetic neuropathy has been done at J.S. Ayurved Mahavidyalaya, Nadiad.

### Definition

Diabetic neuropathy pertains to the dysfunction of autonomic, motor, and/or sensory nerves due to metabolic or vascular irregularities in individuals with longstanding diabetes mellitus. Diabetic sensory polyneuropathy entails the emergence of symptoms and/or signs indicative of peripheral nerve dysfunction in diabetic individuals [2]. Neuropathies involve a gradual decline in nerve fiber function. Peripheral neuropathies stand as the most prevalent complication of diabetes mellitus (DM), impacting up to half of patients with type 1 and type 2 DM. In type 1 diabetes, distal polyneuropathy typically manifests following many years of sustained hyperglycemia. Conversely, individuals with type 2 diabetes may develop distal polyneuropathy within a few years of known poor glycemic control; in some cases, neuropathy may even be present at the time of diagnosis [3].

In terms of incidence and prevalence, India holds the title of the diabetes capital of the world, with an estimated 109 million diabetic individuals projected by 2035. Globally, diabetic neuropathy affects around 132 million people as of 2010, accounting for 1.9% of the population. Among observed diabetic patients, there is a higher prevalence (60.4%) and incidence (8.76%) of sensory peripheral neuropathy, which may escalate with advancing age [4].

## **Anatomy**

A review of the anatomy of the peripheral nervous system can facilitate understanding of the classification of diabetic peripheral neuropathy. Peripheral neurons can be categorized broadly as motor, sensory or autonomic [5].

Motor neurons have their origin in the central nervous system (CNS) and extend toward the anterior horn of the spinal cord. Exiting the spinal cord via ventral roots, they merge with other fibers in either the brachial or lumbar plexuses before reaching their target organs through peripheral nerves. Similarly, sensory neurons originate from the dorsal root ganglia, situated outside the spinal cord, and follow a comparable pathway to motor neurons. Autonomic neurons encompass both sympathetic and parasympathetic varieties [6]. In the peripheral nervous system, preganglionic fibers exit the central nervous system and connect with postganglionic neurons either within the sympathetic chain or in sympathetic ganglia. In diabetes mellitus (DM), smaller fibers are typically impacted initially. However, prolonged exposure to high levels of blood sugar leads to subsequent damage to larger fibers [7].

Factors contributing to diabetic neuropathy include inadequate management of blood sugar levels, older age, high blood pressure, prolonged duration of diabetes, abnormal lipid levels, lifestyle habits like smoking or alcohol consumption, the presence of the HLA-DR3/4 phenotype, potentially reduced insulin levels, and autoimmune mechanisms triggering nerve inflammation. The onset of symptoms of peripheral neuropathy is influenced by various factors including prolonged exposure to high blood sugar levels, elevated lipids, hypertension, smoking, increased height, and exposure to other potentially neurotoxic substances like ethanol [8]. Genetic predispositions may also contribute to this development. Both primary diabetes (types 1 and 2) and secondary diabetes from various causes have been associated with peripheral neuropathies, suggesting a common underlying mechanism related to chronic hyperglycemia. Strong evidence supporting the role of hyperglycemia comes from studies like the Diabetes Control and Complications Trial (DCCT). The association between impaired glucose tolerance and peripheral neuropathy suggests a potential impact of hyperglycemia on nerves that may vary depending on the dosage, yet this remains a topic of debate, especially in the context of type 2 diabetes and prediabetes [9].

Research by Jende et al. suggests that in type 1 diabetes, nerve lesions characteristic of diabetic neuropathy are primarily linked to poor blood sugar control and nerve function decline, whereas in type 2 diabetes, these lesions are associated with changes in lipid metabolism [10, 11]. Additionally, a study by Pai et al. found an association between variability in fasting plasma glucose levels and the risk of painful diabetic peripheral neuropathy in adults with type 2 diabetes. This study showed that higher variability in fasting plasma glucose, as measured by the coefficient of variation (CV), was associated with increased odds of developing painful diabetic peripheral neuropathy, even after adjusting for HbA1c levels [12].

Furthermore, research indicates a connection between obstructive sleep apnea and small-fiber neuropathy in type 2 diabetes, with Poly(adenosine diphosphate-ribose) polymerase activation being a possible mechanism behind obstructive sleep apnea association with diabetic peripheral neuropathy and endothelial dysfunction [13].

## **Pathophysiology**

The precise causes behind the onset of diabetic neuropathy remain incompletely understood, with various hypotheses proposed. It is widely acknowledged to involve a multifaceted process, incorporating mechanisms such as the polyol pathway, formation of advanced glycation end products, and oxidative stress [14].

## **Polyol Pathway Hyperactivity**

Metabolic irregularities are the main instigator of diabetic neuropathy. Elevated blood sugar levels, resulting from decreased insulin secretion or insulin resistance, lead to heightened activity in the polyol

pathway. The first step in this pathway involves aldose reductase, which catalyzes the transformation of glucose into sorbitol while converting nicotinamide adenine dinucleotide phosphate (NADPH) into NADP<sup>+</sup>. Sorbitol is then converted to fructose by sorbitol dehydrogenase, a process that involves the reduction of nicotinamide adenine dinucleotide (NAD<sup>+</sup>) to NADH. It is described that during hyperglycemic states, the affinity of aldose reductase for glucose is higher, generating intracellular osmotic stress.

The buildup of sorbitol occurs due to its inability to penetrate cell membranes. Interestingly, despite being observed in diabetic patients, this sorbitol accumulation doesn't appear to be the primary cause of nerve damage, as reported sorbitol concentrations in nerves are insignificant. However, the prevailing hypothesis suggests that the hyperactivity of the polyol pathway primarily contributes to pathogenesis by accelerating the consumption of cofactors like NADPH and NAD<sup>+</sup> [15].

As a consequence, there is a decrease in the regeneration of glutathione and an elevation in the generation of advanced glycation end products (AGEs), coupled with the initiation of diacylglycerol and multiple isoforms of protein kinase C (PKC). The depletion of glutathione is thought to be the main trigger of oxidative stress and may be linked to the accumulation of harmful substances.

### **Advanced Glycation End Products**

When there is an excess of glucose, it can react non-enzymatically with proteins, nucleotides, and lipids, forming advanced glycation end products (AGEs). These AGEs may potentially disrupt the integrity of neurons and their repair mechanisms by interfering with nerve cell metabolism and axonal transport [16].

### **Oxidative Stress**

The heightened generation of free radicals in diabetes can pose harm through various mechanisms that are not entirely comprehended. These mechanisms include direct harm to blood vessels, resulting in nerve ischemia, and the promotion of advanced glycation end product (AGE) reactions. Despite the lack of full comprehension of these mechanisms, the potential use of the antioxidant alpha-lipoic acid shows promise in alleviating neuropathic symptoms. This concept is visually represented in Flowchart 1, provided at the end of the article.

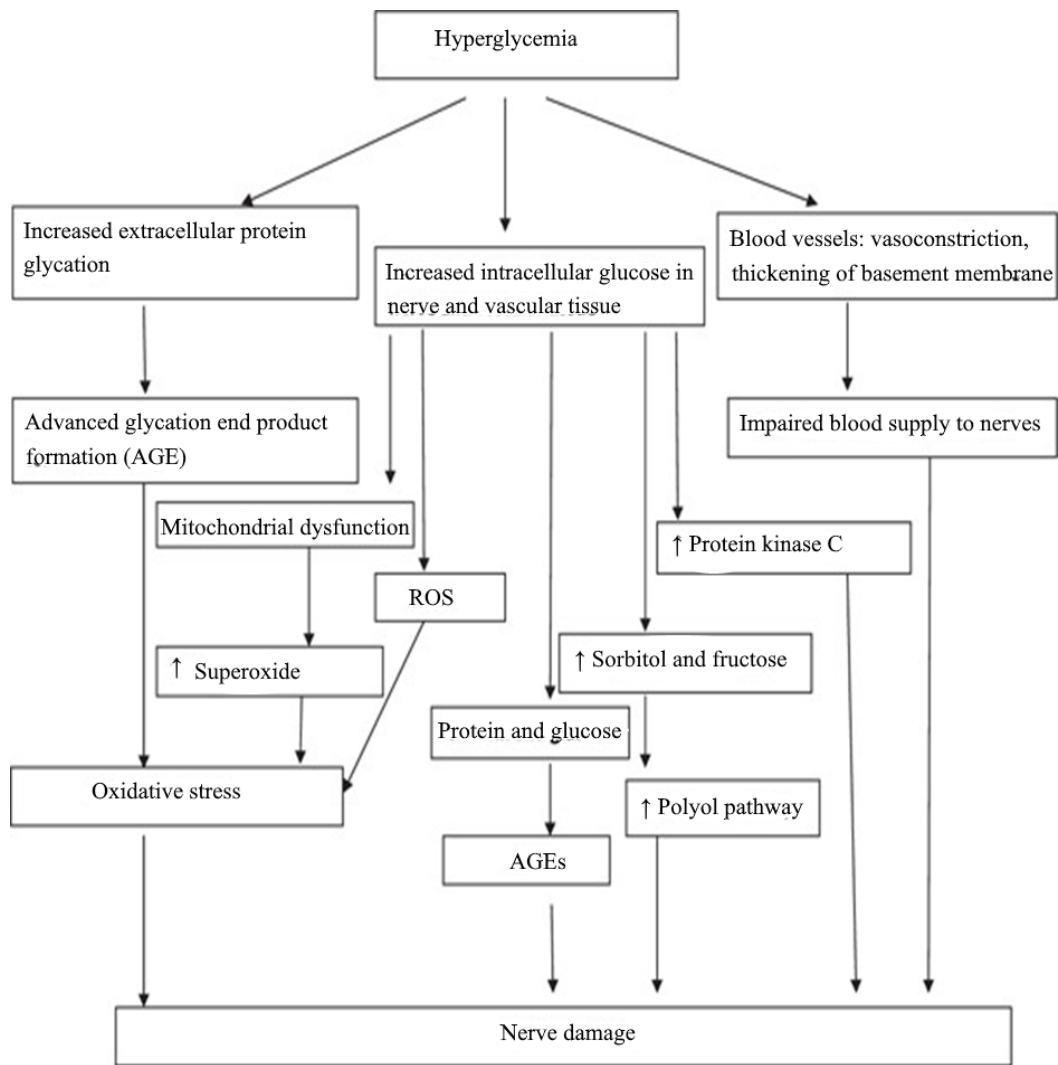
There are four types of diabetic neuropathy:

#### ***Peripheral Neuropathy***

Peripheral neuropathy, also referred to as distal polyneuropathy, stands as the most common form of neuropathy associated with diabetes. It impacts the nerves extending to the extremities, including the feet, legs, hands, and arms, with the feet and legs typically experiencing symptoms before the hands and arms [17]. The nerves reaching the feet are notably lengthy, originating from the lumbar region of the spine and traversing a considerable distance down the legs. Due to their extended length, these nerves are more susceptible to damage, which can lead to various foot issues associated with diabetes, including deformities, infections, ulcers, and, in severe cases, amputations. Symptoms of peripheral neuropathy can encompass numbness, tingling, burning sensations, sharp pains, cramps, increased sensitivity to touch, and impaired balance and coordination. These symptoms frequently intensify during nighttime and manifest symmetrically in both feet [18].

#### ***Proximal Neuropathy***

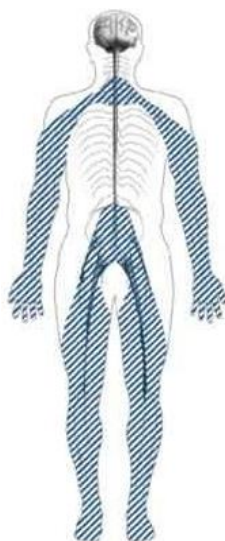
Proximal neuropathy is the second most common type of diabetic neuropathy (second only to peripheral diabetic neuropathy). It typically impacts older individuals with diabetes; unlike peripheral neuropathy, proximal neuropathy is also referred to as diabetic amyotrophy. The term "myo" in "amyotrophy" signifies muscle, indicating that this type of neuropathy can result in muscle weakness. It specifically affects the muscles in the upper part of your leg(s), buttocks, and hips as shown in Figure 1.



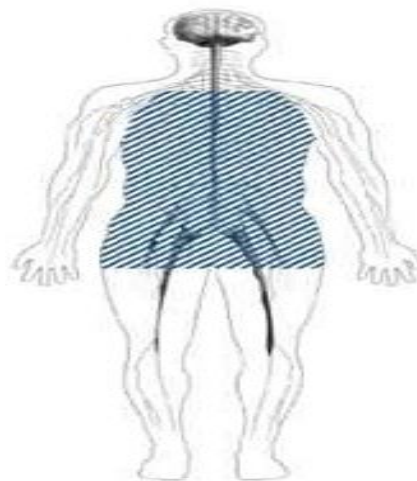
ROS: Reactive oxygen species

AGE: Advanced glycation end products

**Flowchart 1.** Pathology of diabetic neuropathy.



**Figure 1.** Peripheral neuropathy.



**Figure 2.** Autonomic neuropathy.

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Occasionally, proximal neuropathy may include nerve pain, particularly pain that radiates from the lower back down to the leg. This condition is medically termed radiculopathy, though commonly known as sciatica. When shooting nerve pain is present, this type of neuropathy may also be referred to as polyradiculopathy or diabetic amyotrophy. Typically, it resolves either with time or appropriate treatment [19].

### ***Autonomic Neuropathy***

The autonomic nervous system oversees the maintenance of the body's internal equilibrium, known as homeostasis. Autonomic neuropathy impacts the nerves that oversee heart function, blood pressure regulation, and blood glucose control, as illustrated in Figure 2. Furthermore, it can affect various internal organs, leading to complications with digestion, respiratory function, urination, sexual response, and vision. Additionally, this condition may impair the system responsible for restoring blood glucose levels to normal after a hypoglycemic episode, potentially causing a loss of warning symptoms associated with hypoglycemia.

### ***Focal Neuropathy***

It is also known as mononeuropathy. Focal neuropathy arises abruptly and affects specific nerves, usually in the head, torso, or legs. Symptoms of focal neuropathy may include difficulty focusing the eyes, double vision, pain behind one eye, facial paralysis (Bell's palsy), severe lower back or pelvic pain, pain in the front of the thigh, chest or abdominal pain, as well as pain along the shin or inside of the foot. Focal neuropathy can be painful and unpredictable, and it predominantly affects older adults with diabetes. However, it often improves spontaneously over weeks or months and does not necessarily require treatment does not cause long term damage [20].

## **SYMPTOMATOLOGY**

Diabetic neuropathy can present with a diverse range of sensory, motor, and autonomic symptoms, prompting the need for a systematic list of symptoms to effectively screen all diabetic patients for potential neuropathic complications.

### **Sensory Symptoms**

the onset of sensory neuropathy is typically gradual and often presents with a distribution pattern resembling stockings and gloves in the outermost parts of the limbs. Sensory symptoms can vary in nature, ranging from absence to presence, and may affect broad areas or specific spots. Negative sensory symptoms often include sensations of numbness or lack of feeling, likened by patients to wearing gloves or socks. Common manifestations include loss of balance, particularly noticeable when eyes are closed, and painless injuries due to diminished sensation. Positive symptoms may involve sensations such as burning, prickling pain, tingling, electric shock-like feelings, aching, tightness, or heightened sensitivity to touch [21].

### **Motor Symptoms**

Motor difficulties associated with diabetic neuropathy can manifest in various forms, ranging from weakness in distal, proximal, or focal areas. In the upper extremities, distal motor symptoms might involve challenges in fine hand coordination, impacting tasks like opening jars or turning keys. Early signs of foot weakness may include foot slapping, toe scuffing, or frequent tripping. Proximal limb weakness symptoms could entail difficulties in ascending or descending stairs, rising from a seated or lying position, experiencing falls due to weakened knees, and struggling to lift the arms above the shoulders.

### **Autonomic Symptoms**

Autonomic neuropathy can affect the cardiovascular, gastrointestinal, genitourinary systems, and sweat glands. Individuals with generalized autonomic neuropathies may experience symptoms such as ataxia, gait instability, or near-syncope/syncope. Furthermore, specific symptoms of autonomic neuropathies may vary based on the location of nerve damage, including gastrointestinal, cardiovascular, bladder, or sudomotor-related issues [22].

## **Symptoms of Diabetic Sensory**

### ***Polyneuropathy***

Symptoms of diabetic neuropathy often begin with sensations of numbness, tingling, sharpness, or burning in the feet, which may then spread to more proximal areas. Neuropathic pain can develop in some individuals, sometimes improving with better glycemic control. Typically, pain in the lower extremities occurs at rest and intensifies at night. As diabetic neuropathy advances, pain may diminish and eventually disappear, but sensory deficits in the lower extremities persist.

Peripheral neuropathy symptoms may include:

1. Pain
2. Sensations of burning, stabbing, or electric shocks
3. Numbness (loss of sensation)
4. Tingling
5. Muscle weakness
6. Impaired coordination
7. Muscle cramps or twitching
8. Reduced sensitivity to pain or temperature
9. Heightened sensitivity to even light touches

### ***Management***

The treatment of diabetic neuropathy remains largely unsatisfactory. It primarily involves strict glucose control to prevent further worsening of the neuropathy, as established neuropathies typically do not reverse. Chronic painful neuropathy poses challenges in treatment but may show some response to medications such as tricyclic antidepressants like amitriptyline, desipramine, nortriptyline, or selective serotonin-norepinephrine reuptake inhibitors like duloxetine, as well as anticonvulsants such as gabapentin, pregabalin, or carbamazepine.

### ***Initial Therapy and Counseling***

After confirming the diagnosis, providing patients with a comprehensive understanding of their condition, dispelling any fears or misunderstandings, and reassuring them that the pain may improve over time can be highly comforting. Basic physical interventions, such as utilizing a bed cradle to alleviate discomfort from hypersensitive skin caused by bedsheets, can offer relief. Additionally, guidance on appropriate footwear selection may be offered. For patients experiencing relatively mild pain, simple pain relievers or anti-inflammatory medications might adequately alleviate discomfort.

### ***Metabolic Control***

The most effective approach to achieving stable normal blood sugar levels is through pancreas or islet cell transplantation. However, this option is not widely feasible, primarily accessible to individuals with end-stage diabetic nephropathy who undergo combined pancreas and renal transplants, or in specific cases involving young individuals with type 1 diabetes.

Although randomized controlled trials of intensive insulin therapy for managing diabetic neuropathy are lacking, findings from several observational studies suggest that maintaining stable blood sugar levels is crucial. A recent study utilizing continuous glucose monitoring affirmed that erratic blood glucose control was associated with painful symptoms. There is no evidence indicating that individuals whose diabetes is not well-

### ***Pharmacological Management***

The evidence supporting the use of nonsteroidal anti-inflammatory drugs (NSAIDs) in diabetic polyneuropathy is limited. Some experts recommend considering their use for patients experiencing mild symptoms. However, caution is necessary when administering these agents to diabetic neuropathy patients due to the likelihood of renal impairment, which often serves as a contraindication for NSAID usage drugs [23].

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### ***Tricyclic Agents***

Several randomized, controlled studies support the use of tricyclic drugs in managing neuropathic pain. Despite being considered the first-line treatment for symptomatic neuropathy; their usage is limited due to the frequency and severity of side effects. As a result, newer anticonvulsants are being more frequently employed due to their improved side effect profiles.

The exact mechanism of action of tricyclic agents is not fully understood but may involve inhibiting the reuptake of norepinephrine and serotonin, as well as affecting sodium channels and N-methyl-D-aspartate receptors. The side effects, commonly expected and linked to anticholinergic actions, consist of dry mouth, blurred vision, cardiac irregularities, sedation, difficulty urinating, constipation, and low blood pressure upon standing. While nocturnal administration can mitigate sedative side effects, approximately one-third of patients may still find these agents intolerable. Among tricyclic drugs, amitriptyline and imipramine are commonly prescribed, although desipramine is preferred for its fewer anticholinergic side effects and reduced sedation.

### ***Selective Serotonin Reuptake Inhibitors***

Clinical trials investigating the efficacy of selective serotonin reuptake inhibitors (SSRIs) for treating diabetic neuropathy have yielded mostly underwhelming results. These medications function by inhibiting the presynaptic reuptake of serotonin without affecting norepinephrine. However, limited evidence suggests that paroxetine and citalopram, at doses of up to 40 mg/day, may offer some benefit based on findings from small controlled studies.

### ***Anticonvulsants***

These medications have been utilized for managing neuropathic pain for a significant period, yet there's only limited evidence supporting the efficacy of phenytoin and carbamazepine. Gabapentin has become widely prescribed for neuropathic symptoms. It shares a structural resemblance to the neurotransmitter  $\gamma$ -aminobutyric acid and was initially introduced as an anticonvulsant for complex partial seizures. While its side effect profile can still pose challenges, it appears to be less troublesome compared to tricyclics. Reported effects comprise sedation, dizziness, headaches, swelling in the feet, and increased body weight. Pregabalin, another compound active in the central nervous system and an analog of  $\gamma$ -aminobutyric acid, shows promise as a useful addition to anticonvulsants, beneficial in managing neuropathic pain, as suggested by preliminary evidence.

### ***Local Anesthetic Arrhythmic Agents***

Lidocaine functions by blocking sodium channels, thereby reducing sensitization of peripheral nociceptors and ultimately alleviating hyperexcitability in the central nervous system. While initial research indicated potential benefits of intravenous lidocaine for relieving neuropathic pain, concerns regarding potential side effects and the requirement for intravenous administration posed challenges. Due to these issues, lidocaine is not commonly utilized, primarily because of its side effects and the necessity for regular electrocardiogram monitoring during its use.

### ***N, methyl-D-Aspartate Antagonists***

This represents a relatively recent category of medications, encompassing dextromethorphan and memantine. Initial investigations into both drugs indicate some effectiveness in alleviating painful diabetic neuropathy, although additional studies are needed to confirm their efficacy.

### ***Opioid Analgesics***

Traditionally, opioids haven't been a common choice for managing diabetic neuropathic pain. However, recent trials involving two agents suggest their effectiveness. Tramadol, an opioid-like synthetic narcotic analgesic with central action, has been proven effective in a randomized, controlled trial. A follow-up study indicates its safe use for up to six months, providing sustained pain relief. Predictable side effects of both drugs include somnolence, nausea, and constipation, with addiction posing a concern. It's possible that opioids like tramadol and oxycodone could be considered as supplementary therapies for patients who do not respond to initial non-opioid medications.

## **TOPICAL AND PHYSICAL TREATMENTS CAPSAICIN**

This compound, derived from red pepper, works by depleting tissue of substance P, thus reducing chemically induced pain. While several controlled studies, analyzed collectively in meta-analyses, suggest some effectiveness in alleviating diabetic neuropathic pain, it might be more suitable for individuals experiencing localized discomfort rather than those with widespread generalized neuropathic pain.

### **Topical Nitrate**

Recent evidence indicates that reduced nitric oxide synthesis contributes to the development of diabetic neuropathy. Applying isosorbide dinitrate spray locally to the feet has shown effectiveness in alleviating overall pain and burning discomfort.

### **Other Physical Therapies**

Numerous physical therapies have been suggested for treating diabetic neuropathy. Controlled studies have shown the effectiveness of percutaneous nerve stimulation, static magnetic field therapy, and low-intensity interventions laser therapy, and monochromatic infrared light. However, these therapies have primarily been evaluated in small-scale single-center studies and need validation through larger trials. Electrical spinal cord stimulation has been employed to manage various chronic painful conditions, such as phantom limb pain, vascular disease, and severe neuropathy.

## **RESULT AND DISCUSSION**

Diabetic neuropathy refers to nerve damage affecting autonomic, motor, and/or sensory nerves due to metabolic or vascular abnormalities in individuals with longstanding diabetes mellitus (DM). It is the most prevalent complication of both type 1 and type 2 diabetes mellitus, affecting approximately 50% of patients. On a global scale, diabetic neuropathy impacts roughly 132 million people, which accounts for approximately 1.9% of the population as of 2010. In diabetic neuropathy, smaller nerve fibers are typically affected first, followed by larger fibers with prolonged exposure to high blood sugar levels.

The exact pathophysiology of diabetic neuropathy is not fully comprehended but is generally thought to encompass various factors, such as the polyol pathway, advanced glycation end products, and monochromatic infrared light products, and oxidative stress. Sensory neuropathy often manifests gradually and exhibits a characteristic distribution in the distal extremities, leading to symptoms such as burning pain, tingling, electric shock-like sensations, aching, tightness, or heightened sensitivity to touch.

Current treatments for diabetic neuropathy are inadequate. Strict glucose control is essential for preventing the progression of neuropathy, although established neuropathies typically do not reverse. Chronic painful neuropathy poses a challenge to treatment but may respond to medications such as tricyclic antidepressants (e.g., amitriptyline, desipramine, nortriptyline), selective serotonin-norepinephrine reuptake inhibitors (e.g., duloxetine), or anticonvulsants (e.g., gabapentin, pregabalin, carbamazepine).

## **CONCLUSION**

This observation stems from research indicating that individuals who fail to adhere to dietary guidelines are more prone to experiencing early onset symptoms. The progression of the disease is usually gradual, with symptoms exacerbating at night. A significant proportion of individuals reported experiencing symptoms within a maximum period of one year, emphasizing the prominence of diabetic sensory polyneuropathy as an early and prevalent complication of diabetes mellitus. Burning and tingling sensations are commonly cited symptoms, surpassing reports of pain and numbness. Furthermore, the diminished sensation in response to stimuli such as vibration, cold, and heat tends to be symmetrically distributed across both sides of the body.

This insight underscores the importance of dietary management in mitigating the risk of diabetic neuropathy development and progression. The gradual onset and worsening of symptoms during

nighttime highlight the chronic nature of the condition, necessitating early intervention and comprehensive management strategies. The prevalence of symptoms within a relatively short timeframe underscores the urgency of proactive screening and management approaches to address the impact of diabetic neuropathy. Additionally, the prevalence of sensory symptoms, particularly burning and tingling sensations, underscores the need for targeted therapeutic interventions to alleviate discomfort and enhance quality of life for affected individuals.

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