AYUSHDHARA
ISSN: 2393-9583 (P)/ 2393-9591 (O)
An International Journal of Research in AYUSH and Allied Systems

Review Article

AYURVEDIC PERSPECTIVE ON DIABETIC PERIPHERAL NEUROPATHY - A CONCEPTUAL REVIEW

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Article info
Article History:
Received: 20-02-2026
Accepted: 23-03-2026
Published: 06-05-2026
KEYWORDS:
Diabetic Peripheral Neuropathy, *Madhumeha*, *Prameha*, Ayurveda, *Dosha-Dushya* Involvement, Integrative Approach.

ABSTRACT

Diabetic Peripheral Neuropathy is one among the most prevalent and distressing complications of diabetes mellitus, significantly impairing the quality of life of affected individuals. **Aim and Objective:** The aim is to bridge traditional wisdom and explore diabetic peripheral neuropathy through an Ayurvedic perspective and correlate it with contemporary clinical understanding to provide an integrative approach for this complication. **Materials and Methods:** The present work is a conceptual review based on descriptions from Ayurvedic classics along with relevant modern medical literature. **Results:** Modern therapeutic options are mainly limited to symptomatic management and have minimal role in preventing disease progression. Ayurveda explains such conditions through the involvement of *Dosha, Dushya* and *Srotas*. In classical texts, *Prameha*, particularly *Madhumeha*, and its *Purvarupa* and *Upadrava* show similarities with the manifestations of diabetic peripheral neuropathy. **Conclusion:** Understanding the disease through both Ayurvedic and modern perspectives may help in developing a more comprehensive and integrative approach for its management.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Prolonged hyperglycemia leads to widespread metabolic disturbances and progressive tissue damage, forming the basis for long-term complications.[1] According to the World Health Organization (WHO), diabetes affects approximately 828 million adults worldwide and causes nearly 2 million deaths annually.[2] Chronic hyperglycemia results in progressive damage to blood vessels throughout the body. These complications are broadly classified into microvascular and macrovascular complications. Microvascular damage leads to retinopathy, nephropathy, and neuropathy, while macrovascular involvement contributes to cardiovascular morbidity.[3]

Among these complications, diabetic neuropathy is one of the most common and disabling. Neuropathy refers to functional or structural impairment of nerves due to sustained metabolic and vascular abnormalities associated with long-standing diabetes. When the nerves outside the brain and spinal cord are affected, it is termed peripheral neuropathy.

Diabetic Peripheral Neuropathy (DPN) is the most prevalent form of diabetic neuropathy, affecting approximately 30–50% of individuals with diabetes. It typically presents with pain, paresthesia, numbness, and muscle weakness in a characteristic stocking–glove distribution, significantly impairing quality of life.[4]

In Ayurveda, *Prameha* has been described by all three *Brihatrayees*, highlighting its importance and complexity. The chronicity of *Madhumeha* and its progression is known to cause *Upadravas*, many of which align closely with the modern understanding of diabetic neuropathic complications. Hence, revisiting

Access this article online
Quick Response Code
https://doi.org/10.47070/ayushdhara.v13i2.2524

this disease through the Ayurvedic framework offers an effective and holistic management.

Objectives

- To study Diabetic Peripheral Neuropathy (DPN) from both modern and Ayurvedic perspectives.
- To correlate the symptoms of DPN with Ayurvedic descriptions found in *Prameha purvarupa*, *Rupa*, and *Upadrava*.
- To explore Ayurvedic diagnostic and therapeutic strategies for the effective management of DPN.

MATERIALS AND METHODS

For this study, information has been collected from the classics of *Brihatrayees - Charaka Samhita, Susruta Samhita* & *Astanga Hridaya*, various books of modern medical science and various articles from online and offline medical journals using the key words diabetic peripheral neuropathy, *Prameha upadrava* etc.

RESULTS

Diabetic Peripheral Neuropathy

Diabetic Peripheral Neuropathy (DPN) is defined as the presence of symptoms and or signs of peripheral nerve dysfunction in diabetes after the exclusion of other causes (malignancy, chronic alcoholism, nutritional deficiency, infections, iatrogenic etc).[5] Though neuropathy is considered as a late complication, it can even be present at the onset of diabetes mellitus.

It can lead to a range of complications including foot ulcers, infections, charcot arthropathy, and eventual limb amputation.

Complications

Microvascular complications are among the most debilitating consequences of diabetes mellitus, arising from structural and functional alterations in capillaries and arterioles, particularly involving endothelial cells and their basement membranes.[6] While these changes occur systemically, their clinical significance is most evident in the retina, kidneys, and peripheral nerves, manifesting as diabetic retinopathy, nephropathy, and neuropathy. Diabetic neuropathy, a consequence of chronic hyperglycemia, includes a spectrum of nerve disorders and is broadly classified into polyneuropathy, mononeuropathy, and autonomic neuropathy, with clinical presentations ranging from transient forms (such as painful neuropathies and radiculopathies) to progressive sensorimotor polyneuropathy, often with autonomic involvement. These complications substantially impair quality of life and contribute to long-term morbidity in diabetic patients, underscoring the importance of early recognition and comprehensive management.[7]

Pathophysiology[8]

The pathogenesis of diabetic peripheral neuropathy is mediated through four interrelated mechanisms: (i) advanced glycation end-product formation, (ii) polyol pathway activation, (iii) protein kinase C signalling, and (iv) oxidative–mitochondrial stress.

First, chronic hyperglycemia promotes non-enzymatic glycation, wherein glucose irreversibly binds to proteins and lipids, forming advanced glycation end products (AGEs)-stable, abnormal molecules that accumulate over time and disrupt normal cellular structure and function. AGEs deposit within peripheral nerves and the endoneurial microvasculature, leading to structural protein modification, impaired axonal transport, inflammatory activation via AGE receptors, and reduced neural perfusion.

Second, excess intracellular glucose is predominantly metabolized through the polyol pathway, resulting in sorbitol accumulation within Schwann cells. This causes osmotic stress, depletion of myo-inositol, reduced antioxidant defense, and subsequent demyelination and axonal injury. Third, hyperglycemia induces protein kinase C (PKC) activation, which alters endothelial function, reduces nitric oxide bioavailability, increases pro-inflammatory mediators, and further compromises nerve blood flow. Finally, these metabolic disturbances collectively enhance oxidative stress and mitochondrial dysfunction, impairing ATP generation and axonal integrity, ultimately leading to progressive nerve conduction failure characteristic of diabetic peripheral neuropathy.

Clinical Features of Diabetic Peripheral Neuropathy (DPN)[9]

Diabetic peripheral neuropathy is a length-dependent, symmetrical neuropathy with a gradual onset, affecting sensory, motor, and autonomic nerves. Symptoms typically begin in the distal lower limbs and progress proximally in a characteristic stocking–glove distribution.

Sensory Features

Sensory involvement is the earliest and most frequent manifestation. Common features include:

- Numbness and reduced sensation in the toes and soles.
- Tingling, burning, or prickling sensations.
- Abnormal sensitivity to touch or temperature.
- Impaired vibration and position sense.

Motor Features

Motor involvement usually occurs in advanced stages and is characterized by:

- Distal muscle weakness, mainly in the lower limbs
- Reduced or absent ankle reflexes
- Gait instability and balance difficulty
- Foot deformities or foot drop in severe cases

Autonomic Features

Involve multiple systems

- Cardiovascular: Resting tachycardia, orthostatic hypotension
- Gastrointestinal: Gastroparesis, nausea, bloating, constipation or diarrhea
- Genitourinary: Urinary disturbances, erectile dysfunction
- Skin and pupils: Altered sweating, dry skin, impaired light response

Clinical Examination Findings

On examination, patients commonly show

- Loss of vibration and pinprick sensation
- Impaired proprioception
- Reduced or absent ankle reflexes
- Length-dependent sensory loss ascending from the feet.

Management of Diabetic Peripheral Neuropathy

- The cornerstone of diabetic neuropathy management is strict glycemic control, which may improve nerve conduction but may not completely reverse symptoms. Supportive care includes avoiding neurotoxic factors such as alcohol, correcting nutritional deficiencies (especially B vitamins), and relieving neuropathic pain.
- Acute painful neuropathy may resolve spontaneously within about a year, whereas chronic painful neuropathy often requires drug therapy such as tricyclic antidepressants, gabapentin, NSAIDs, or agents like carbamazepine and topical capsaicin.
- Amitriptyline is effective for burning neuropathic pain but may cause anticholinergic side effects; desipramine is often preferred due to better tolerability.

Ayurvedic View of DPN

Acharya Charaka advises that when a condition cannot be clearly diagnosed or classified, the physician should analyze the *Samutthana viseṣa* (causative factors), *Adhiṣṭhana* (site of manifestation), and *Vikara prakṛti* (nature and character of the disease) to determine its appropriate understanding and management.[11] From this perspective, DPN may be interpreted as an *Upadrava* of *Prameha*. DPN aligns with *Prameha upadrava* and *Poorvarupa*, indicating the chronicity of *Madhumeha*.

रोगारम्भकदोषस्य प्रकोपादुपजायते।
योऽन्यो विकारः स बुधैरुपद्रव इहोदितः ||११||
(Bhavaprakasha)

Upadrava has been defined as the disease which develops after the manifestation of the main disease.[12] Acharyas have described *Upadravas* for specific *Pramehas* like *Kaphaja*, *Pittaja* and *Vataja*, among them we can correlate *Pittaja* and *Vajata upadravas* like *Daha, Shola, Shosha* to the features of diabetic peripheral neuropathy. In the progressive stages of *Prameha*, there is excessive *Kshaya* of *Saumya dhatus* due to *Prabhuta avila mutrata*, which leads to *Dhatu shaithilya* in the body. This may correspond to the degeneration of the myelin sheath of peripheral nerves observed in DPN.

Samprapthi

The pathogenesis of Diabetic Peripheral Neuropathy (DPN) in Ayurveda can be understood as a progressive complication of *Madhumeha*, arising primarily due to a dual mechanism- *Avaranajanya* and *Dhatukshayajanya samprapti*.[13]

Excessive indulgence in *Kapha-pradhana nidanas* such as *Guru, Snigdha, Madhura ahara* and *Avyayama* causes *Agnimandya*, *Ama* formation, and impaired *Dhatu poshana*. The resulting increase in *Kleda* produces *Srotorodha*, especially of *Medovaha* and *Mutravaha Srotas*, causing *Prameha*. When left untreated, *Prameha* progresses to *Madhumeha*, as stated in *Aṣṭanga Hṛdaya* "कालेनोपेक्षिता सर्वा यद्यान्ति मधुमेहताम्".[14]

In the early stage, *Santarpana nidanas* cause *Kapha, Medas, Maṃsa,* and *Pitta vṛddhi*, leading to *Avaraṇa* of *Vata*. The obstructed *Vata* becomes *Prakupita* and draws *Ojas* and *Kleda* toward the *Basti pradesa*, producing *Madhumeha*. With disease chronicity, prolonged *Vata prakopa* and *Apatarpaṇa* lead to excessive *Kṣaya* of *Saumya dhatus*, resulting in *Dhatu saithilya* and depletion of *Mamsa, Meda, Majja,* and *Ojas*.[15] This *Dhatukṣaya*-dominant *Vata kopa* manifests as neurological deficits comparable to demyelination and axonal degeneration seen in DPN.

Rupa

The classical text Siddhanta Nidana describes several *Upadrava lakshanas* of *Madhumeha* that closely resemble symptoms of Diabetic Peripheral Neuropathy. These include *Karapadadaha* (burning sensation in palms and soles), *Teevra shula* in the nerves of the limbs "शाखागत नाडी-सन्धि तीव्र वेदना", *Supti (*numbness), *Harsha* (tingling sensation), *Shoola* (pain), *Mamsa-shosha* (muscle wasting), *Dourbalya* (weakness), *Stambha* (stiffness), tremors (*Kampa*), and fatigue (*Glani*). The progressive degeneration also leads to ocular involvement, referred to as "दृष्टि नाडी

वितान शोष", indicating optic nerve damage and potential visual disturbances- a known complication of chronic diabetes.[16] Hence, Diabetic Peripheral Neuropathy in *Madhumeha* is the result of both *Avarana* of *Vata* by excessive *Kapha* and *Medas* in the early stage and the subsequent *Dhatukshaya* and *Vata prakopa* in later stages.

In all types of *Prameha, Prabhuta mutrata* (excessive urination) and *Avila mutrata* (turbid urine) are consistently observed.[17] In cases resembling Diabetic Peripheral Neuropathy (DPN), *Daha*, *Pipelika sancaram iva sula*, and *Suptata* are common. *Pitta prakopa* causes *Daha, Vata prakopa* produces pain and paresthesia, while *Vata kapha* vitiation results in numbness. Features such as *Daurbalya, Maṃsa kṣaya*, and *Kampa* indicate chronic *Vata* aggravation with *Dhatu kṣaya*.

Clinical features are tabulated below

In the Ayurvedic understanding of diabetic peripheral neuropathy (*Prameha janya sannipatika vyadhi*), various sensory disturbances described in modern medicine can be correlated with classical *Lakshanas* mentioned in the context of *Prameha*, *Vata vyadhi*, and *Avṛta vata.* Numbness of limbs may be understood as *Supti*, typically arising due to *Medo-kaphavṛta vata*.[18] A burning sensation often experienced by patients corresponds to *Daha*, which is suggestive of *Pittanubandha* or *Raktavṛta vata lakshanas*.[19] Pricking sensations like needle pricks are denoted by *Suchibhiriva nistoda*, while sensations like ants crawling *Pipeelika sanchara* indicate *Mamsavṛta vata*.[20] Heaviness in limbs *Guruta* is often attributed to *Kaphavṛta vata*, whereas abnormal pain perceptions such as *Toda*, *Sparsavaiguṇya*, or *Shoola* arise due to *Sveda kṣhaya, Pitta-vata dushti*, or *Dhathu kshaya*.[21]

Motor symptoms such as *Mamsapacchaya soṣa* (muscle wasting) and *Daurbalya, Angasada* general weakness are often explained as *Prameha upadrava* due to *Dhatu kṣhaya*.[22] *Kampa* (involuntary movements) represent *Vataja kopa* and neuro-muscular degeneration seen in chronic *Prameha* stages.[23]

Table 1: Sensory Symptoms of Neuropathy[24]

S.No	Symptoms	*Lakshana*	References
1.	Numbness	*Supti*	*Medakaphavrita vata*
2.	Burning sensation	*Daha*	*Prameha purvarupa, Raktavrita vata, Prameha upadrava*
3.	Pricking sensation	*Suchibhiriva nistoda*	*Raktavrita vata*
4.	Tingling sensation	*Pipeelika sancharamiva*	*Mamsavrita vata*
5.	Heaviness	*Guruta*	*Kaphavruta vata*

Table 2: Motor Symptoms[25]

S.No.	Symptoms	*Lakshana*	Reference
1.	Weakness	*Dourbalya, Angasada*	*Prameha upadrava*
2.	Involuntary movements	*Kampa*	*Prameha upadrava*
3.	Wasting	*Mamsopachaya Sosha*	*Prameha upadrava*

Chikitsa

In the Ayurvedic management of Diabetic Peripheral Neuropathy, the primary focus lies on addressing both the underlying *Prameha* and the *Vata* vitiation due to *Avarana* and *Dhatu kshaya*. Hence, the treatment should include *Avarana hara chikitsa* along with *Pramehahara* approach. The core treatment goals include:

- *Pramehahara*
- *Kleda shoshana*
- *Srotorodha hara*
- *Dhatvagni deepana*
- *Vatanulomana*
- *Rasayana*

Formulations having *Vata-pitta hara*, *Ojovardhaka*, and *Rasayana* properties are especially beneficial in repairing nerve damage and improving vitality. Rejuvenating and nervine tonic herbs that are rich in anti-oxidants, like *Ashwagandha*, *Shatavari*, and *Guduchi*, play a vital role in restoring nerve function. Panchakarma procedures like *Abhyanga*, *Swedana*, and *Basti* help in pacifying vitiated *Vata*, promoting nerve conduction, and strengthening *Dhatus.*[27] The line of treatment should also aim at *Dhatu poshana*, *Bala vardhana*, and *Jeevaniya* action while ensuring *Kleda Shoshana*, *Srotorodha hara*, and *Dhatvagni deepana* to improve metabolic clearance and tissue nourishment.

DISCUSSION

Neuropathy in patients with diabetes is generally diagnosed through a combination of clinical symptoms, patient history, and physical findings. However, from an Ayurvedic point of view, the manifestation of such complications like DPN is not merely the result of high blood glucose but also due to prolonged continuation of *Nidanas* that disturb the *Dosha-dhatu-srotas* level, leading to irreversible changes in *Samprapti*. Therefore, accurate diagnosis and understanding of the disease pathology is essential before deciding the line of treatment.

The neurodegenerative changes seen in DPN such as demyelination, microvascular ischemia, and accumulation of AGE and sorbitol-closely resemble *Ama sanchaya* and *Srotorodha* that disturb *Vata gati*.[28] These features point towards *Avarana* of *Vata* by *Kapha*, *Meda*, and more precisely, *Rakta*, causing manifestations similar to *Raktavruta vata*.

In *Raktavruta vata*, the obstructed *Vata* within *Rakta vaha srotas* leads to symptoms like *Daha*, *Shoola*, *Shopha*, and *Sparsha asahatwa*, which shows a close resemblance to clinical presentations of DPN such as burning feet, tingling, and numbness. Moreover, the concept of *Upadhatu pradosha* involving *Sira, Snayu, and Kandara* explains peripheral nerve impairment, due to *Rakta dushti*. This indicates the need for a multidimensional therapeutic approach beyond mere glucose control. Therefore, line of management should ideally focus on *Raktaprasadana chikitsa* followed by *Vatahara* interventions. In *Rakta avruta vata*, Acharya Vagbhata has advised to adopt the *Vatarakta chikitsa*. The symptoms of diabetic peripheral neuropathy may be correlated with the stage of *Uthana vataraktam*.[29] The treatment protocol of *Uthana vatarakta* including *Alepana, Abhyanga, Parisheka, Upanaha* can be adopted here. However, it is essential to note that *in Prameha*, *Swedana* is to be done with caution. Hence, mild forms of fomentation such as *Mridu sweda* in the form of *Parisheka* and *Upanaha*, are more suitable. These features strongly indicate the need for a combined therapeutic approach that not only addresses *Prameha* but also targets *Rakta dushti*, clears *Avarana*, and restores *Vata* function. Hence, inclusion of *Raktaprasadana* and *Vatanulomana* measures alongside *Pramehahara chikitsa* becomes essential in the Ayurvedic management of DPN. Hence, addressing *Prameha* alone may not yield sufficient results unless the treatment also targets *Raktaprasadana* and relieves *Vata avarana*.

CONCLUSION

Diabetic Peripheral Neuropathy (DPN), a common yet debilitating complication of *Madhumeha*, demands more than symptomatic management. Rather than viewing it as a mere *Upadrava*, it should be addressed as a *Pradhana vyadhi*, requiring a comprehensive and individualized treatment approach. In Ayurveda the involvement of *Vata*, particularly *Rakthavruta vata*, aligns well with the clinical features of DPN such as *Daha, Shoola, Shopha,* and *Supthi* reflecting *Dushti* in *Rakta* and its *Upadhatus* like *Sira, Snayu,* and *Kandara*. Hence, integrating *Pramehahara, Vatahara,* and *Raktashodhana*, alongside both internal and external treatments, becomes essential. *Rasayana chikitsa* plays a pivotal role in *Dhatu kshaya* and halting disease progression. Understanding the chronic, cyclical nature of DPN and focusing on *Nidana parivarjana* and *Samprapti vighatana* form the cornerstone of effective long-term management.

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Cite this article as:
Meenu R Menon, Ratheesh P, Ajay Paul, Vinitha C, Ananthalekshmi R, Saranya K. Ayurvedic Perspective on Diabetic Peripheral Neuropathy - A Conceptual Review. AYUSHDHARA, 2026;13(2):384-389.
https://doi.org/10.47070/ayushdhara.v13i2.2524
Source of support: Nil, Conflict of interest: None Declared

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