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Review Article

Chemotherapy-Induced Peripheral Neuropathy as an Acquired *Vata Vyadhi*: Mechanisms, Supportive Care and Research Opportunities

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Abstract

Chemotherapy-induced peripheral neuropathy (CIPN) is a frequent and sometimes dose-limiting adverse effect of cancer treatment. It is most often associated with platinum compounds, taxanes, vinca alkaloids and proteasome inhibitors. Patients commonly experience distal numbness, tingling, burning pain, altered temperature sensation, weakness and impaired balance. The underlying injury differs among drugs but may involve axonal degeneration, disturbed microtubule-dependent transport, mitochondrial dysfunction, oxidative stress, neuroinflammation, ion-channel changes and damage to dorsal root ganglion neurons. Treatment options remain limited, and supportive care is therefore important. CIPN has no exact disease equivalent in classical Ayurveda. Nevertheless, its acquired onset after a toxic therapeutic exposure and its predominance of pain, altered sensation, weakness and impaired movement allow a careful interpretation within the broader framework of acquired *Vata Vyadhi*. Concepts such as *Dhatukshaya* and *Srotodushti* may further support hypothesis development. This narrative review brings modern neurobiology and Ayurvedic reasoning together without treating them as identical systems. *Abhyanga*, selected *Basti* procedures, rehabilitation and standardized *Vatahara* or *Rasayana* interventions deserve systematic investigation as supervised supportive measures. Future studies should use validated CIPN scales, objective neurological outcomes, oncology-specific safety screening and formal herb-drug interaction assessment. The aim is to improve symptoms, function and quality of life without compromising anticancer treatment.

Keywords: chemotherapy-induced peripheral neuropathy; Ayurveda; *Vata Vyadhi*; neurotoxicity; neuropathic pain; supportive care

INTRODUCTION

Better cancer survival has made long-term treatment toxicity increasingly important. Chemotherapy-induced peripheral neuropathy is particularly troublesome because it can affect both continuation

of treatment and everyday function. Neurotoxic agents may injure peripheral sensory, motor or autonomic pathways through several overlapping mechanisms, and symptoms can continue long after chemotherapy has ended.[1]

Clinical management remains difficult. The American Society of Clinical Oncology guideline does not recommend a pharmacological agent for routine prevention of CIPN and identifies duloxetine as the main evidence-supported option for established painful CIPN, with an expected benefit that is usually modest.[2] This gap has encouraged interest in exercise, rehabilitation, acupuncture and other supportive approaches, including carefully designed integrative care.

CIPN usually begins distally in a symmetrical stocking-and-glove pattern. Patients may describe numbness, tingling, burning, electric-shock-like pain, cold sensitivity, altered touch, weakness or poor balance.[3] The exact pattern varies with the drug, cumulative dose and individual susceptibility; some patients recover after treatment, while others have persistent symptoms and functional limitations.[4]

Ayurveda offers a functional language for pain, altered sensation, weakness and impaired movement. Classical descriptions of *Vata Vyadhi* are therefore relevant to clinical interpretation, but the comparison must be used correctly. Chemotherapy is a modern exposure, and CIPN was not described as a named classical disease. The useful question is not whether CIPN and *Vata Vyadhi* are identical, but whether selected principles of *Vata* pathology and management can guide safe, testable supportive-care strategies.[5]

AIM

To review CIPN from the perspectives of modern neurobiology and Ayurveda, with particular emphasis on its possible interpretation as an acquired *Vata Vyadhi*, contemporary supportive care and priorities for integrative research.

OBJECTIVES

The review describes the clinical features and mechanisms of CIPN; examines its relationship with *Vata Vyadhi*, *Dhatukshaya* and *Srotodushti*; reviews evidence-based supportive management; considers Ayurveda-based interventions suitable for investigation; and proposes practical safety and outcome standards for future research.

MATERIALS AND METHODS

A narrative review method was used. Contemporary publications were selected for the clinical presentation, mechanisms, assessment and management of CIPN, with particular attention to major reviews and professional guidelines. Classical Ayurvedic sources were considered for descriptions of *Vata*, *Vata Vyadhi*, pain, altered sensation, weakness, *Dhatukshaya* and general principles of *Vatahara Chikitsa*. Biomedical and Ayurvedic evidence were interpreted in their own contexts. The conceptual correlation presented here is intended to generate research questions, not to establish a direct classical diagnosis.

CLINICAL PROFILE OF CIPN

CIPN is toxic injury to the peripheral nervous system caused by anticancer treatment. Peripheral neurons are vulnerable because their long axons depend on continuous energy production, axonal transport and stable membrane excitability. The pattern and severity of injury depend on the drug, cumulative dose, schedule, combination treatment and individual factors. Oxidative damage is important in several drug classes and can reinforce mitochondrial dysfunction and neural injury.[6]

The syndrome is not biologically uniform. Taxanes disrupt microtubule dynamics, platinum compounds can injure dorsal root ganglion neurons, and vincristine affects axonal transport. These mechanisms overlap, which explains why patients may share a distal sensory phenotype even when the responsible drugs act differently.[7]

Table 1. Major anticancer drug groups associated with CIPN

Drug group	Examples	Typical clinical pattern
Platinum compounds	Cisplatin, oxaliplatin	Predominantly sensory neuropathy; oxaliplatin may produce acute cold sensitivity
Taxanes	Paclitaxel, docetaxel	Painful sensory neuropathy with numbness and distal sensory loss
Vinca alkaloids	Vincristine	Sensory-motor neuropathy; autonomic features may occur
Proteasome inhibitors	Bortezomib	Painful sensory neuropathy
Other neurotoxic agents	Thalidomide and related agents	Predominantly sensory neuropathy

CLINICAL PRESENTATION

Sensory symptoms are most common. They generally begin in the toes or fingers and spread proximally as neuropathy progresses. Numbness, tingling, pricking, burning, shooting pain, reduced touch sensation and altered temperature perception can occur alone or together. Oxaliplatin is especially associated with acute cold-evoked symptoms, while platinum-related injury also illustrates the vulnerability of sensory neuronal structures.[8]

Motor involvement may reduce grip, dexterity or walking ability, and autonomic symptoms may occur with certain drugs. Pain reflects altered excitability as well as structural injury; changes in sodium, potassium, calcium and transient receptor potential channels can contribute to spontaneous pain and thermal hypersensitivity.[9] The clinical impact is often greater than the sensory score alone suggests because impaired balance, sleep and confidence can restrict daily activity.

MECHANISMS OF CIPN

Axonal and microtubule injury

Long peripheral axons depend on efficient transport of mitochondria, proteins and vesicles. Taxanes stabilize microtubules and vinca alkaloids disrupt their assembly; both actions are useful against cancer but can interfere with normal neuronal transport. Distal axons then become vulnerable to energy failure and degeneration. Neuroimmune changes involving macrophages, Schwann cells and glial signalling can add sensitisation and help maintain pain after the initial exposure.[10]

Mitochondrial dysfunction and oxidative stress

Peripheral neurons have high energy demands. Damaged mitochondria produce less ATP and may generate excessive reactive oxygen species. Oxidative injury can then damage lipids, proteins and additional mitochondria, creating a self-reinforcing cycle. Experimental work on axon degeneration has placed mitochondrial health and axonal maintenance at the centre of several CIPN models.[11]

Dorsal root ganglion and neuronal excitability

The dorsal root ganglia are exposed to circulating agents and are particularly susceptible to platinum compounds. DNA injury, mitochondrial changes and altered sensory signalling contribute to the predominantly sensory nature of many cases. The relative importance of each mechanism differs among agents, so CIPN is best treated as a family of related toxic neuropathies rather than one uniform disease.[12]

AYURVEDIC INTERPRETATION

CIPN has no exact named equivalent in classical Ayurveda. A defensible interpretation is that it is a modern, treatment-acquired neurological toxicity whose clinical expression overlaps with functions governed by *Vata*. This preserves biomedical accuracy while allowing Ayurvedic reasoning to contribute to supportive-care research.

Why *Vata* is relevant

Vata is associated with movement, sensory perception, motor activity and neuromuscular coordination. Disturbance of these functions may present as *Ruja* (pain), *Toda* (pricking pain), *Supti* (numbness or altered sensation), *Stambha* (stiffness), *Kampa* (tremulous movement), weakness and impaired mobility. These descriptions resemble important features of CIPN at the level of clinical function. They do not imply that the underlying pathology is the same.

CIPN as an acquired *Vata Vyadhi*

CIPN begins after exposure to a defined therapeutic agent. It may therefore be conceptualized as an acquired or treatment-associated disorder in which peripheral neural injury produces a *Vata*-dominant symptom pattern. The sequence is clinically coherent: neurotoxic exposure leads to nerve injury, which leads to pain, altered sensation, weakness and impaired movement. The value of this model is that it suggests therapeutic questions; it is not proof of a classical diagnosis.

Dhatukshaya* and *Srotodushti

Cancer and chemotherapy can reduce nutritional reserve through fatigue, poor appetite, mucosal injury, weight loss or systemic stress. Within Ayurvedic reasoning, this depleted state may be understood as *Dhatukshaya*, which can favour the expression of *Vata*. *Srotodushti* offers another broad model for disturbed transport and tissue function. It may help organize Ayurvedic thinking about impaired neural communication, but it should not be directly equated with microtubule damage, mitochondrial movement or ion-channel dysfunction.

Table 2. Conceptual comparison of CIPN and Ayurvedic features

CIPN feature	**Possible Ayurvedic interpretation**
Neuropathic pain	*Ruja*, *Toda*
Tingling or pricking	*Toda*
Numbness or altered sensation	*Supti*
Motor weakness	Functional disturbance of *Vata*
Difficulty walking or poor coordination	Impairment of normal *Vata*-mediated movement
Tissue injury after chemotherapy	Acquired pathological insult
Systemic depletion	*Dhatukshaya*
Persistent functional impairment	Sustained disturbance of *Vata* functions

CURRENT SUPPORTIVE CARE

The first priority is careful neurological monitoring during treatment. When neuropathy becomes clinically significant, the oncology team may consider dose adjustment, delay, substitution or discontinuation of the causative drug. These decisions must remain with the treating oncologist because modification of anticancer treatment can affect disease control. European clinical guidance similarly emphasizes agent-specific recognition, follow-up and multidisciplinary management.[13]

Duloxetine and other medicines

Duloxetine has the best evidence for established painful CIPN. In a randomized clinical trial, it produced a modest improvement in pain and some functional outcomes.[14] It should not be described as reversing nerve injury, and it is not established as a preventive drug. Gabapentin and pregabalin are commonly used for other neuropathic pain conditions, but evidence specific to CIPN remains insufficient for a firm recommendation.

Exercise, rehabilitation and fall prevention

Drug-specific syndromes also matter. Paclitaxel can cause an acute pain syndrome that may precede or accompany later neuropathy and requires appropriate

clinical recognition.[15] Persistent sensory loss and reduced proprioception can increase fall risk, particularly when balance and lower-limb function are affected.[16] Practical care should therefore include footwear advice, safe lighting, removal of household obstacles, balance training and assistive devices when necessary.

Exercise is a promising supportive strategy. A multicentre randomized study suggested that structured exercise during chemotherapy may reduce symptoms or functional impact in some patients, although the evidence is not yet uniform across regimens and outcomes.[17] Rehabilitation should be individualized for weakness, loss of conditioning, gait instability and fear of falling. Assessment should also capture pain, sensory loss, balance and daily function rather than relying on one symptom alone.[18]

A clinically useful plan combines early recognition, agent-specific oncology decisions, symptom management, rehabilitation and prevention of secondary injury. This is consistent with modern understanding of chemotherapy-induced neurotoxicity and its variable recovery course.[19] Reviews of available treatment strategies also show why multimodal supportive care is necessary: no single medicine adequately addresses pain, numbness, balance and disability in every patient.[20]

POTENTIAL ROLE OF AYURVEDA

The limited treatment options create a meaningful opportunity for Ayurveda-based supportive research. Classical sources give a central place to *Snehana*, *Basti* and individualized management in disorders dominated by *Vata*.[21][22] Ayurvedic pharmacology also identifies *Vatahara*, *Balya* and *Rasayana* drugs that may provide candidates for systematic study.[23] The appropriate aim is not to replace oncology care, but to test whether supervised interventions can reduce symptoms, improve function or support quality of life.

Abhyanga as a research candidate

Abhyanga is a logical first intervention to investigate because it is non-invasive and consistent with *Snehana*-based *Vatahara* care. Standardized studies could measure pain, perceived numbness, sleep, functional ability, balance and quality of life. Massage and tactile stimulation may also provide comfort and relaxation. Safety screening remains essential: vigorous massage may be unsuitable in severe thrombocytopenia, skin infection, open wounds, fragile irradiated skin, venous thrombosis or unstable bone metastases.

Basti and individualized *Vata* management

Basti holds an important place in classical *Vata Vyadhi* management and deserves careful investigation rather than casual extrapolation. Active chemotherapy may be accompanied by neutropenia, thrombocytopenia, mucositis, gastrointestinal complications or immunosuppression. Rectal procedures may therefore be inappropriate for some patients. Any clinical trial must define eligibility, timing, infection precautions, hematological thresholds and oncology oversight before assessing benefit.

Rasayana and translational research

Rasayana provides a broader framework for resilience and recovery. Instead of making general claims of nerve regeneration, research can examine fatigue, functional recovery, inflammatory markers, oxidative stress and quality of life. Mitochondrial dysfunction is a major experimental target in CIPN, which makes carefully selected preclinical models particularly relevant.[24] Contemporary reviews of chemotherapy neurotoxicity also support studying several interacting mechanisms rather than pursuing a single universal target.[25]

AN INTEGRATIVE SUPPORTIVE-CARE MODEL

A rational model keeps oncology treatment at the centre. Standard care includes chemotherapy review, neuropathy assessment, duloxetine when appropriate for painful CIPN, rehabilitation and fall prevention. Ayurveda-based measures may be added only after clinical screening and should be standardized, supervised and documented. The outcomes should be patient-centred: less pain, better sensation or

function, improved sleep, safer mobility and better quality of life, achieved without interfering with anticancer treatment.

Other modalities remain investigational. Reviews have considered topical high-concentration capsaicin and several non-pharmacological strategies, but evidence and accessibility vary.[26] Prevention research has also produced inconsistent results, reinforcing the need to separate prevention from treatment and to avoid presenting an intervention as effective before adequate trials are completed.[27]

MECHANISTIC AND SAFETY PRIORITIES

Mechanistic research should proceed in stages. Standardized botanical candidates can first be screened in cellular and animal models for effects on mitochondrial function, oxidative injury, inflammatory signalling, neurite growth, axonal integrity, Schwann-cell function and sensory excitability. Positive laboratory findings should then lead to dose-finding and safety studies before larger clinical trials. This sequence is more convincing than moving directly from a classical indication to broad therapeutic claims.

Herb-drug interaction assessment is mandatory. Anticancer drugs often have narrow therapeutic windows, and herbal products may affect cytochrome P450 enzymes, transporters, renal elimination, platelet function or immune activity. Every study should document the chemotherapy regimen, concurrent medicines, botanical identity, extract composition, dose, adverse events and possible pharmacokinetic interactions. Patients should not begin herbal medicines during chemotherapy without discussion with the oncology team.

RESEARCH GAPS

Several gaps remain. No Ayurvedic diagnostic framework has been validated specifically for CIPN, and most correlations with *Vata Vyadhi* are conceptual. Standardized clinical trials are scarce, herb-drug interaction data are inadequate, and multi-component interventions often make it difficult to identify the active element. Different chemotherapy-related neuropathies are also frequently pooled despite important biological differences. These limitations create a substantial opportunity for doctoral and interdisciplinary work involving oncologists, neurologists, Ayurvedic physicians, pharmacologists and rehabilitation specialists.

DISCUSSION

CIPN lies at the difficult intersection of effective cancer treatment and treatment-related neurological disability. Modern evidence shows that axonal degeneration, microtubule dysfunction, mitochondrial injury, oxidative stress, neuroinflammation and altered ion-channel activity interact in different proportions according to the causative drug. This biological diversity explains why a single preventive or therapeutic solution has remained elusive.

Ayurvedic interpretation is most useful when it remains clinically grounded. *Ruja*, *Toda*, *Supti* and impaired movement reasonably place the phenotype within the functional domain of disturbed *Vata*, while *Dhatukshaya* offers a way to consider reduced resilience during cancer treatment. Renaming CIPN as *Vata Vyadhi*, however, would ignore the specific toxicity created by chemotherapy. Describing it as a modern acquired neuropathy with a *Vata*-dominant clinical expression is more accurate and more productive for research.

The greatest opportunity lies in supportive, not replacement, care. *Abhyanga*, selected procedures and standardized formulations can be studied for pain, sensory symptoms, balance, sleep and quality of life. A positive Ayurvedic contribution does not require exaggerated claims; it requires careful intervention design, meaningful outcomes and proof that benefit is achieved without increasing oncological risk.

CONCLUSION

Chemotherapy-induced peripheral neuropathy is a clinically important complication of several anticancer drugs. It can cause pain, tingling, numbness, altered temperature sensation, weakness, poor balance and long-term functional difficulty. The injury is multifactorial, with axonal damage,

disturbed transport, mitochondrial dysfunction, oxidative stress, neuroinflammation and altered sensory excitability all contributing.

CIPN has no exact classical Ayurvedic equivalent, yet its acquired onset and symptom pattern provide a reasonable basis for exploring it within the broader framework of acquired *Vata Vyadhi*. *Dhatukshaya* and *Srotodushti* can add useful conceptual dimensions when they are not treated as direct substitutes for molecular mechanisms.

Ayurveda-based supportive care deserves rigorous study. *Abhyanga*, carefully selected *Basti* procedures and standardized *Vatahara* or *Rasayana* interventions may eventually add useful options for patients with CIPN. Until adequate evidence is available, they should be supervised research candidates used alongside oncology care, not substitutes for chemotherapy review, symptom management, rehabilitation or safety monitoring. The strongest future model is collaborative and evidence-based, combining Ayurvedic clinical insight with oncology, neurology, pharmacology and rehabilitation science.

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