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Peripheral Neuropathy Induced by Chemotherapy: A Review

S. Neelamani Durga[1], L. Renuka[2], N. Phani Satyavathi[3], P. Bhanuji Rao[4]
Doctor of Pharmacy (PHARM D), Sri Venkateswara College Of Pharmacy, Etcherla, Srikakulam, Andhra Pradesh, India

Abstract: ***This review explores the management of peripheral neuropathy in cancer patients, focusing on the clinical manifestations and underlying mechanisms of specific neurotoxic chemotherapy drugs. Chemotherapy-induced peripheral neuropathy (CIPN) is a frequent and often dose-limiting side effect of cancer treatments.***
Around 30–40% of patients receiving neurotoxic chemotherapy will experience CIPN, with varying degrees of severity among individuals.
This condition is usually sensory-based, causing pain, and can result in long-term complications for survivors. As cancer survival rates rise, the prevalence and impact of CIPN's lasting effects are expected to grow.
Keywords: Peripheral Neuropathy, Chemotherapy, Platinum Agents, Vinca Alkaloids, Taxanes, Epothilones.

I. INTRODUCTION

Peripheral neuropathies in cancer patients are most commonly caused by neurotoxic chemotherapy drugs, a condition that damages the peripheral nerves known as chemotherapy-induced peripheral neuropathy (CIPN). It's a common side effect of chemotherapy and can affect the nerves' sensory, motor, and autonomic functions. In rare cases, they may be a result of paraneoplastic, immune-mediated, or neoplastic neuropathies.

CIPN is frequently painful and can limit the dose of chemotherapy, and with the improvements in cancer survival rates, its prevalence is expected to rise. Approximately 30-40% of patients undergoing neurotoxic chemotherapy experience CIPN, significantly contributing to the annual healthcare costs.

Well-known chemotherapy agents like platinum compounds, vinca alkaloids, and taxanes are established causes of CIPN, but newer drugs can also cause this side effect, despite their more targeted mechanisms of action. This review will explore how peripheral neuropathy is managed in cancer patients and provide an updated understanding of the neurotoxic mechanisms and clinical manifestations of specific chemotherapy drugs.

II. EVALUATING PERIPHERAL NEUROPATHY IN CANCER PATIENTS

When evaluating a cancer patient with neuropathy, it's important to determine whether they have chemotherapy-induced peripheral neuropathy (CIPN). This involves assessing the drugs they've received, their cumulative doses, and the characteristics and timeline of their symptoms.

First, identify if the patient has been treated with neurotoxic chemotherapy agents, such as taxanes, platinum drugs, vinca alkaloids, thalidomide, or bortezomib, which are strongly linked to CIPN. Other drugs like cyclophosphamide or methotrexate are less likely to cause neuropathy, with only a few cases reported. Next, consider the method of drug administration.

For example, methotrexate is rarely neurotoxic unless given intrathecally, and bortezomib's neurotoxicity is reduced with subcutaneous use.

Third, evaluate whether the drug doses align with the likelihood of CIPN. Symptoms typically begin within the first two months of treatment, progress while chemotherapy is ongoing, and stabilize once treatment ends. Although CIPN is generally dose-dependent, some drugs have specific patterns, such as paclitaxel and oxaliplatin causing acute neurotoxicity, or cisplatin causing symptoms to worsen after discontinuation (a phenomenon known as "coasting"). It is uncommon for CIPN to develop weeks or months after the final dose of neurotoxic chemotherapy.

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Table 1 – Chemotherapeutic agents causing peripheral neuropathy

Platinum agents
Cisplatin
Carboplatin
Oxaliplatin
Vinca alkaloids
Vincristine
Vinblastine
Taxanes
Paclitaxel
Docetaxel
Epothilones
Ixabepalone
Newer agents
Bortezomib
Thalidomide
Lenolidamide

Before diagnosing chemotherapy-induced peripheral neuropathy (CIPN) in cancer patients, it's important to rule out other causes of neuropathy. Metabolic and endocrine-related neuropathies are uncommon in cancer patients, although individuals with diabetes mellitus may have an increased risk of developing CIPN. Paraneoplastic neuropathies typically occur at the onset of cancer, but can occasionally emerge during treatment, such as in the dysimmune neuropathies seen in lymphomatous disorders. Direct cancer cell infiltration can cause neuropathy in conditions like neurolymphomatosis, leukemia, and rarely carcinoma, sometimes mimicking chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). Paraproteinemias are linked to various neuropathies, and treatments for diseases like Waldenström's disease and multiple myeloma may involve drugs that cause CIPN, such as proteasome inhibitors and thalidomide, complicating the determination of the neuropathy's origin. Graft-versus-host disease following bone marrow transplantation can lead to neuropathies such as Guillain-Barré Syndrome (GBS). Additionally, chronic autoimmune neuropathies and amyloid deposition in peripheral nerves, which can occur in paraproteinemic neuropathies, multiple myeloma, and Waldenström's disease, should also be considered.

III. CLINICAL PRESENTATIONS AND NEUROTOXICITY IN CIPN

Chemotherapy-induced peripheral neuropathy (CIPN) is primarily caused by neurotoxic effects on neurons, with sensory symptoms being more prevalent than motor or autonomic symptoms. The condition may result from anatomical changes, like distal axonal degeneration, or from physiological changes in the nerves. Neuropathic pain in CIPN is often worsened by peripheral nerve hyperexcitability, which is linked to altered bioenergetics and ion channel activity, as well as central sensitization. The role of non-neuronal cells like Schwann cells in CIPN is not fully understood.

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CIPN typically develops in a dose-dependent manner after multiple cycles of chemotherapy, with the severity often correlating with the dose. However, newer biological agents may cause more unpredictable reactions. If CIPN occurs, it may require dose adjustments or discontinuation of the chemotherapy agent, although stopping the treatment may hinder cancer treatment. Oncologists must carefully balance the risk of CIPN-related quality of life issues with the potential benefits of cancer treatment. It is important to note that conventional electrophysiological tests may not accurately reflect the patient's symptoms, making them unreliable for monitoring therapy. Moreover, common clinical trial tools, like the Common Terminology Criteria for Adverse Events, lack sensitivity in assessing neuropathy. To improve evaluation, new methodologies like the EORTC QLQ-CIPN20, a 20-item questionnaire, and the CIPN-R-ODS, a scale built using Rasch analysis, have been developed. These tools aim to provide better assessments of symptoms and CIPN-related disability, and will likely be used in future clinical trials. Incorporating quantitative neurological exams and neurophysiology, like the Total Neuropathy Score clinical version (TNSc), will further improve CIPN evaluation. Although the TNSc has undergone Rasch analysis in CIPN patients, further validation studies are needed.

The main symptoms of CIPN stem from damage to dorsal root ganglion neurons or their axons, resulting in acral pain, sensory loss, and occasionally sensory ataxia. While motor, autonomic, and cranial nerve symptoms can occur, they are less common. Most cases of CIPN involve axonal damage, typically presenting as dying-back neuropathy. However, there are notable exceptions. Platinum compounds (carboplatin, cisplatin, and oxaliplatin) are known to cause sensory neuropathy, which is likely due to the permeability of the blood-nerve barrier at the dorsal root ganglion. When diagnosing CIPN and establishing a link to chemotherapy, clinicians must consider the variability in signs and symptoms. Neurophysiology can be helpful in distinguishing between different types of neuropathies, such as sensory neuropathy, length-dependent sensory motor neuropathy, or small fiber neuropathies.

The clinical characteristics of the different medication classes are separately reviewed below.

IV. PLATINUM COMPOUNDS

Platinum compounds, like cisplatin, have been in use for over four decades, but they often cause side effects such as hearing loss, tinnitus, sensory neuropathy, kidney damage, and bone marrow suppression. While the latter two can be managed through hydration or bone marrow stimulators, the neuropathy and ototoxicity require dose reduction or discontinuation of the drug to prevent further damage. About 20% of patients treated with cisplatin experience significant hearing loss, and 40% develop tinnitus, with these effects often being permanent. Although the neuropathy and hearing loss are related to dosage, there is no clear connection between them in terms of mechanism or risk. Platinum-based drugs, including cisplatin, oxaliplatin, and carboplatin, often cause long-term sensory nerve damage in 30–40% of patients, with cisplatin being the most commonly linked to peripheral neurotoxicity. Carboplatin is less neurotoxic than cisplatin. The treatment schedule and formulation of the drug can impact toxicity. In addition to the chronic neuropathy seen with all platinum-based compounds, oxaliplatin can also cause acute neuropathic pain, especially with cold-induced symptoms in the hands, face, and mouth. These symptoms are most severe after the second or third cycle of treatment and can last for a few days. One notable feature of platinum-based chemotherapy-induced peripheral neuropathy (CIPN) is the "coasting" phenomenon, where symptoms worsen for several months after treatment ends, which can be distressing for both patients and doctors. Much research into the cause of platinum-based CIPN has focused on damage to sensory neurons in the dorsal root ganglia. Platinum drugs (cisplatin, oxaliplatin, carboplatin, and their analogs) form DNA adducts in these neurons, leading to apoptosis due to abnormal cell cycle entry, which aligns with the clinical presentation of neuropathy. Additionally, damage to mitochondria, caused by disruptions in mitochondrial DNA transcription, is believed to contribute to the "coasting" effect. While DNA repair processes in the nucleus can fix platinum-DNA adducts, mitochondria lack these repair mechanisms, resulting in long-term mitochondrial dysfunction after treatment stops.

A. Anti-microtubule Agents

1) Taxanes

Taxanes (such as paclitaxel, docetaxel, and cabazitaxel) are commonly used in cancer treatment and typically lead to a dose-dependent, painful, length-dependent sensory neuropathy due to axonopathy, which may improve after stopping treatment. Notably, cabazitaxel tends to have less cumulative toxicity but may cause dysgeusia. Additionally, more than half of patients treated with paclitaxel experience an acute, transient pain syndrome, characterized primarily by aching musculoskeletal pain. This syndrome is not clearly linked to nerve damage, though it shares several features with chemotherapy-induced peripheral neuropathy (CIPN) and might correlate with paclitaxel-induced CIPN. Taxanes work by stabilizing the dynamic assembly of microtubules, and while they primarily kill cancer cells by disrupting cell division via microtubule interference, their mechanism for causing CIPN is less understood. Microtubules are crucial for axonal transport, and taxanes can disrupt this process, potentially leading to neuropathy.

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Other research shows that taxanes can damage mitochondria, possibly contributing to metabolic axonal failure in CIPN. A recent study in zebra fish suggests that paclitaxel-induced neuropathy might depend on interactions between skin nerve endings and epidermal keratinocytes through the matrix metalloproteinase MMP-13.

2) Vinca Alkaloids

Vinca alkaloids are primarily used for treating hematologic cancers and tend to cause a length-dependent sensory neuropathy, often accompanied by some motor involvement. These drugs may lead to long-term residual neuropathy, especially in paediatric and young adult patients. They can also cause vascular issues like Raynaud's syndrome. Although rare, these drugs can cause cranial nerve and autonomic dysfunction. Unlike taxanes, which stabilize microtubules, vinca alkaloids destabilize them. Despite this difference, both drug types seem to affect axonal transport and mitochondrial function similarly in neurons. Recent studies suggest that SARM1, a protein that promotes axonal degeneration, plays a significant role in neuropathy caused by vincristine, a vinca alkaloid. Genetic deletion of SARM1 in mice prevents vincristine-induced neuropathy, suggesting its importance in this process.

3) New Anti-microtubule Agents

In recent years, new chemotherapy drugs that affect microtubule dynamics have emerged. Eribulin and ixabepilone, used in breast cancer treatment, cause axonal sensory motor peripheral neuropathy. These drugs bind to the same site and have similar effects on microtubules as taxanes. A novel approach involves conjugating chemotherapy agents with tumor-targeting antibodies, as seen in brentuximab vedotin, which links an antibody targeting CD30 (found in lymphoma) to a microtubule toxin (monomethyl auristatin E). Despite its targeting to lymphoma cells, brentuximab vedotin can cause peripheral neuropathy in 30-50% of patients. Similarly, ado-trastuzumab emtansine combines an antibody against HER2 (found in breast cancer) with emtansine, which inhibits microtubule polymer assembly, and is frequently associated with peripheral neuropathy.

B. Proteasome Inhibitors

Bortezomib works by inhibiting proteasomes, the key machinery responsible for protein degradation in cells, thereby exerting its anti-cancer effects. However, it can cause a painful, small fiber-predominant axonal sensory neuropathy, particularly affecting distal axons, though this is a reversible condition. Recent findings show that subcutaneous administration reduces the severity and occurrence of this neuropathy. A small group of patients may develop a severe, immune-mediated polyradiculoneuropathy. Newer proteasome inhibitors like carfilzomib and ixazomib seem to have a lower risk of causing chemotherapy-induced peripheral neuropathy (CIPN). Despite the potential broad cellular effects of proteasome inhibition, bortezomib's neurotoxicity is thought to stem from its interference with microtubules and mitochondria, disrupting axonal transport and function in sensory neurons. Other possible mechanisms include the accumulation of ubiquitinated proteins in the nucleus and changes in protein transcription within sensory ganglion neurons.

C. Thalidomide

Long-term use of thalidomide, which is used to treat multiple myeloma, can result in a sensory-predominant neuropathy. The neurotoxic effects were already recognized when the drug was first used as a sedative in the 1960s, with up to 75% of patients experiencing persistent deficits during long-term follow-up. Newer formulations such as lenalidomide and pomalidomide have less neurotoxicity. Thalidomide and its analogs are believed to cause neuropathy through their anti-angiogenic properties, and impaired angiogenesis may also play a role in CIPN caused by other drugs.

D. Immune Checkpoint Inhibitors

Though not directly neurotoxic, immune checkpoint inhibitors—used in cancer treatments like melanoma—can cause immune-mediated neuropathies. Drugs such as ipilimumab and tremelimumab target CTLA-4, stimulating cytotoxic T cells to attack cancer cells. Pembrolizumab and nivolumab, which target PD-1 receptors, have also been linked to neurological side effects, including both peripheral and central nervous system disorders, some of which can be life-threatening. These side effects often resemble inflammatory demyelinating neuropathies or vasculitic neuropathies. Although these issues are rare, they can occur in up to 3% of patients. Treatment usually involves stopping the immune checkpoint inhibitor and administering immunotherapy, such as steroids or IVIG, which has been found effective for many patients.

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V. TREATMENT AND PREVENTION OF CIPN

Currently, there are no known preventive treatments for chemotherapy-induced peripheral neuropathy (CIPN). It's difficult to understand why chemotherapy drugs that target rapidly dividing cancer cells also affect non-dividing neurons. Developing agents to prevent CIPN is challenging because any potential treatment may interfere with the chemotherapy's effectiveness. Preventative approaches would need to separate the neurotoxic effects from the cancer-killing effects or identify features unique to neurons, such as receptor sensitivity to growth factors. Despite extensive research, this approach has not yet led to an effective preventive treatment. Another strategy is to identify patient-specific risk factors to personalize chemotherapy regimens. Many factors likely contribute to CIPN susceptibility, including dosage, method of drug delivery, concurrent medications, age, pre-existing neuropathy, and cancer type. For example, diabetes has been shown to increase CIPN risk in lung cancer patients treated with platinum and taxane drugs. Genetic factors are also relevant, as patients with Charcot-Marie-Tooth (CMT) disease, particularly CMT1A, are more vulnerable to CIPN, especially when treated with vinca alkaloids. Studies have linked specific genetic variants to increased susceptibility to neurotoxic effects from drugs like paclitaxel, vincristine, platinum compounds, bortezomib, and thalidomide. These findings have been based on various research methods, including candidate gene analysis, genome-wide association studies, and the study of gene expression. However, the identified genetic factors only show a small increase in susceptibility, and reproducibility has been a challenge, possibly due to population differences. No genetic test is available yet to predict an individual's risk for CIPN, but advances in genetic and epigenetic sequencing and bioinformatics hold promise for future success in this area.

Currently, the main treatment for CIPN is to stop or reduce the offending chemotherapy drug and address symptoms like neuropathic pain. The National Cancer Institute has sponsored 15 clinical trials focused on CIPN prevention (e.g., alpha-lipoic acid, intravenous calcium/magnesium, vitamin E, acetyl-L-carnitine, and glutathione) and symptomatic treatments (e.g., nortriptyline, gabapentin, lamotrigine, amifostine, topical amitriptyline/ketamine, duloxetine). Of these treatments, only duloxetine has shown effectiveness for treating neuropathic pain in established CIPN. Other medications, like gabapentin and topical treatments, are often used off-label.

A. Long-term effects and Future directions in research

One of the main long-term effects of chemotherapy on the nervous system is chemotherapy-induced peripheral neuropathy (CIPN), which often results in persistent chronic pain. Additionally, there is growing discussion about a new late effect in cancer patients and survivors: long-term cognitive impairments following chemotherapy. Studies in animal models and humans have shown evidence of this phenomenon, commonly referred to as "chemo brain." The underlying cause is believed to be the impact of chemotherapy on neurogenesis in the adult brain, and cranial radiation may enhance this effect. While this area of research is attracting increasing attention, there is still limited data available. As the number of cancer survivors grows, this will likely become a key focus of future studies.

VI. CONCLUSIONS

With the development of newer and more targeted chemotherapy drugs, there was optimism that CIPN would become less of a major issue. However, many of the older drugs that cause CIPN remain essential in cancer treatment. In addition, several new therapies also result in CIPN as a dose-limiting side effect, either due to direct toxicity or immune-mediated processes. As cancer treatments improve and survival rates increase, the long-term effects of CIPN continue to cause substantial hardship for cancer survivors.

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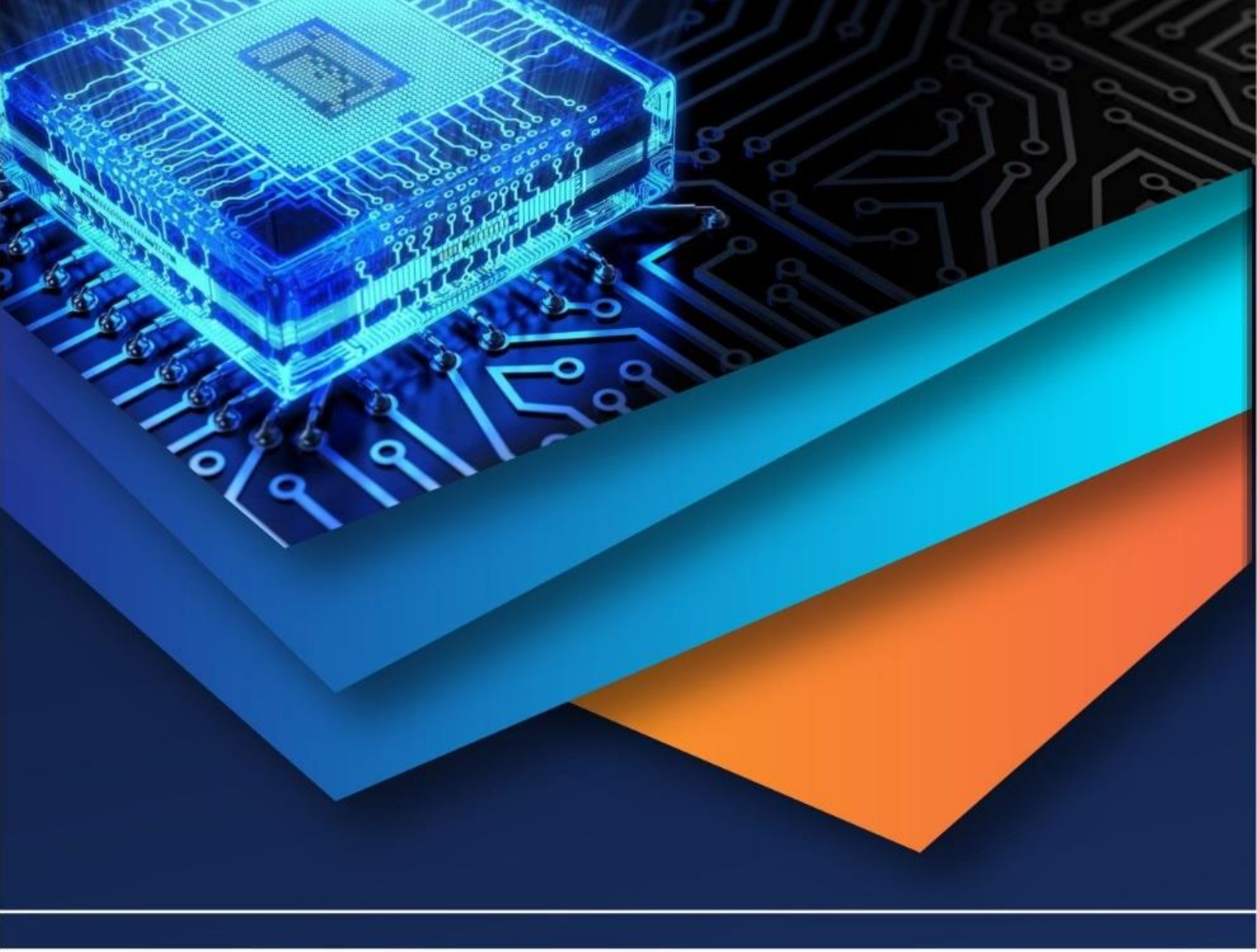
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