



Paclitaxel - induced peripheral neuropathy in a breast cancer patient: A case report

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Abstract

Background: Paclitaxel is one among the drugs considered in the management of breast cancer. The issue, however, arises when the beneficial effect of this drug in any therapy is limited by the development of chemotherapy-induced peripheral neuropathy. PIPN is mainly sensory, dose-related, and has a significant impact on the patient's quality of life.

Case Presentation: A 52-year-old female with breast cancer developed neuropathic symptoms after two cycles of paclitaxel. Patient-reported symptoms included burning pain, numbness, and tingling of the extremities. Neurological examination and nerve conduction studies revealed axonal sensory neuropathy of the peripheral type. Differential laboratory diagnoses excluded the presence of diabetes, thyroid dysfunction, renal impairment, or vitamin

deficiencies. An assessment of causality using the Naranjo scale gave a score of 6, and application of the WHO-Uppsala Monitoring Centre (UMC) criteria classified the reaction as probable, i.e., neuropathy related to paclitaxel.

Management and Outcome: Pharmacological intervention comprising pregabalin, gabapentin, and amitriptyline, together with folic acid, vitamin B-complex, and calcium supplementations led to symptom amelioration within about two weeks. After stabilization, paclitaxel was readministered at 20 percent dose reduction. The patient tolerated the modified regimen well, no further progression of neuropathy, and thus, could continue with cancer treatment.

Conclusion: This case reveals that early recognition with a structured approach in the evaluation of PIPN is important. Pharmacological treatment targeted at the symptoms, supportive supplementation, and chemotherapy dose modification allowed the patient to undergo effective anticancer therapy without compromising neurological safety. Hence, individualized management plans, interdisciplinary cooperation, and frequent clinical reviews are necessary to enhance treatment outcomes and preserve the quality of life in patients receiving taxane-based regimens.

Keywords: Paclitaxel, Peripheral Neuropathy, Breast Cancer, Chemotherapy-Induced Neuropathy, Case Report

Introduction

Breast cancer is the most common malignancy in women around the globe, with systemic chemotherapy remaining one of the main therapeutic approaches. The paclitaxel, the taxane obtained from the Pacific yew tree (*Taxus brevifolia*), is perhaps one of the most effective agents in breast cancer treatment-adjuvant and metastatic. It has a cytotoxic effect on tumor cells by stabilizing microtubules and preventing their depolymerization so that mitotic spindle function is disrupted and mitosis stops. In addition to being effective, this drug causes many side effects, with peripheral neuropathy being the commonest and sometimes a dose-limiting toxicity.^[1,2,3]

Peripheral neuropathy caused by Paclitaxel acts mainly as a distal and symmetrical sensory neuropathy, causing sensations of numbness, tingling, and burning pain in the hands and feet. The severe cases can cause motor weakness and some functional debility. The incidence varies greatly, affecting 30–70% of patients, depending on the cumulative dose, treatment schedule, and individual susceptibility. The actual pathophysiology leading to PIPN includes microtubule accumulation in axons, cessation of axonal transport, mitochondrial dysfunction, and oxidative stress, culminating in axonal injuries. Symptoms appear mostly within weeks of initiating treatment and linger in many cases after the treatment has ceased, seriously compromising the patient's quality of life and possibly blocking continued cancer treatment in earnest.^[4,5,6]

PIPN diagnosis can pose a big challenge, with neuropathic symptoms sometimes being confused with those of diabetes mellitus, thyroid dysfunction, vitamin deficiencies, renal impairment, or other related neurotoxic agents. Therefore, a detailed clinical history, laboratory evaluation, and electrophysiological studies are mandatory for making a definitive diagnosis. Causality assessment tools like the Naranjo Adverse Drug Reaction Probability Scale and WHO-UMM criteria can strengthen the correlation between drug exposure and adverse neurological occurrences. In the context of breast cancer treatment, dose intensity, and treatment continuity are crucial: thus, a careful consideration of the neurotoxicity and its management must accompany the oncology efficacy.^[7,8,9]

Although PIPN is known to be an adverse effect, each case is interesting clinically because important lessons are to be learned in terms of timing, severity, and management of neuropathy as observed in actual practice. This report presents the case of a 52-year-old woman who was treated for breast cancer and who developed axonal sensory neuropathy amid treatment with paclitaxel. After careful clinical assessment, including ruling out differential diagnoses, assessment of causality, and tailored treatment interventions, the case highlights the need to address early recognition, multidisciplinary input, and individualized dose modification. Documenting this case may add to the existing literature advocating successful management of

PIPN, whereby methods allow for continuation of chemotherapy, decreasing neurological deficits, and ultimately preserving quality of life for patients.^[10,11,12]

Case Presentation

Patient Demographics

- **Age/Sex:** 52-year-old female
- **Ethnicity:** Indian
- **Occupation:** Homemaker
- **Residence:** Semi-urban area, living with husband and two children
- **Education level:** Secondary school completed

Chief Complaint

- **Tingling, numbness, and burning sensations** in both feet and fingers for 3 weeks.
- Difficulty in buttoning clothes, holding small objects, and prolonged walking.

History of Present Illness

Six months earlier, the patient had been suffering from stage IIIB invasive ductal carcinoma of the left breast. She underwent modified radical mastectomy, followed by adjuvant chemotherapy.

- When her four cycles of doxorubicin (60 mg/m²) and cyclophosphamide (600 mg/m²) had been completed, she was then given paclitaxel (175 mg/m² every 3 weeks).
- After her second cycle of paclitaxel, mild tingling started in her toes. This sensation worsened by the time she reached her fourth cycle and spread to her hands, interfering with her daily life.
- Associated signs such as motor weakness or autonomic dysfunction (constipation, urinary complaints, or orthostatic dizziness) were not seen with neuropathic symptoms.

Past Medical History

- **No history** of diabetes mellitus, hypertension, thyroid disorders, renal, or hepatic disease.

- **No history** of neurological disorders.

Past Medication History

- **Chemotherapy:**
 - Doxorubicin + Cyclophosphamide (4 cycles)
 - Paclitaxel (planned 4 cycles; neuropathy developed after 4th cycle)
- **Supportive medications:** Ondansetron, dexamethasone, proton pump inhibitor, and hematinic supplements.
- **No history** of neurotoxic drugs (isoniazid, antivirals, etc.)

Social History

- **Tobacco/Alcohol:** None
- **Diet:** Mixed diet, adequate nutrition
- **Family history:** Negative for cancer, neuropathy, or metabolic disorders
- **Drug allergies:** None known

Physical Examination

- **General:** Alert, oriented, ECOG performance status 1, BMI 24.5 kg/m²
- **Vital signs:** BP 124/76 mmHg, HR 82 bpm, Temp 36.8°C, RR 18/min

Systemic Examination

- **Cardiovascular:** Normal S1/S2, no murmurs
- **Respiratory:** Clear breath sounds, no rales/wheezes
- **Abdomen:** Soft, non-tender, no hepatosplenomegaly
- **Neurological:**
 - **Sensory:** Decreased vibration and pinprick sensation in stocking-glove distribution
 - **Motor:** Normal power (5/5) in upper and lower limbs
 - **Reflexes:** Ankle reflexes diminished; knee and biceps reflexes normal
 - **Cranial nerves:** Intact
 - **Gait:** Mild unsteadiness on tandem walking

Laboratory Investigations

Parameter	Result	Reference Range	Interpretation
Hemoglobin	12.1 g/dL	12–16 g/dL	Normal
Total Leukocyte Count	6,700/ μ L	4,000–11,000/ μ L	Normal
Platelet Count	245,000/ μ L	150,000–400,000/ μ L	Normal
Serum Creatinine	0.8 mg/dL	0.6–1.3 mg/dL	Normal
SGOT/AST	28 U/L	<40 U/L	Normal
SGPT/ALT	31 U/L	<40 U/L	Normal
Fasting Blood Glucose	94 mg/dL	70–110 mg/dL	Normal
HbA1c	5.3%	<5.7%	Normal
Thyroid Stimulating Hormone (TSH)	2.2 μ IU/mL	0.5–4.5 μ IU/mL	Normal
Serum Vitamin B12	412 pg/mL	200–900 pg/mL	Normal
Serum Folate	8.7 ng/mL	>4 ng/mL	Normal

Neuropathy Evaluation

Nerve Conduction Study (NCS)

Nerve Tested	Parameter	Patient Value	Normal Range	Interpretation
Sural Nerve (Sensory, lower limb)	SNAP Amplitude	4.5 μ V	>10 μ V	Reduced
	Conduction Velocity	38 m/s	>45 m/s	Slowed
Median Nerve (Sensory, upper limb)	SNAP Amplitude	8 μ V	>15 μ V	Reduced
	Conduction Velocity	42 m/s	>50 m/s	Slowed
Peroneal Nerve (Motor, lower limb)	CMAP Amplitude	6.5 mV	>5 mV	Normal
	Conduction Velocity	48 m/s	>45 m/s	Normal
Median Nerve (Motor, upper limb)	CMAP Amplitude	5.8 mV	>5 mV	Normal
	Conduction Velocity	51 m/s	>50 m/s	Normal

Summary: Findings indicate **axonal sensory neuropathy** with preserved motor function.

Total Neuropathy Score (TNS)

Component	Patient Finding	Score (0–4)
Sensory Symptoms	Tingling, numbness in hands/feet	2
Motor Symptoms	No weakness	0
Autonomic Symptoms	Absent	0
Pin Sensation	Decreased in stocking–glove	2
Vibration Sense	Reduced at toes/ankles	1
Strength	Normal	0
Reflexes	Ankle reflexes reduced	1

Total Score = 6/12 → Moderate Neuropathy

Causality Assessment:

Causality was assessed according to the Naranjo Adverse Drug Reaction Probability Scale and WHO-UMM criteria. The patient established a score of 6 in the Naranjo scale, suggesting a probable adverse drug reaction. According to the WHO-UMC, the reaction was labeled as probable/likely because of the following reasons:

- Appearance of the adverse event after initiation of paclitaxel.
- Worsened-onset of symptoms after repeated dosing with paclitaxel.
- Laboratory and comorbid evaluation not revealing another possibility.
- Partial resolution of the symptoms after reduction of dosage and supportive care

Final Diagnosis

On the basis of clinical presentation, neurological examination, and laboratory investigations, along with nerve conduction studies performed, a diagnosis was made of Paclitaxel-Induced Peripheral Neuropathy (PIPN), sensory predominant axonal type. These neuropathic symptoms occurred after the second cycle of paclitaxel and worsened with increasing doses, not occurring during earlier chemotherapeutic treatment with doxorubicin–cyclophosphamide. Laboratory values excluded diabetes, thyroid dysfunction, and vitamin deficiencies as alternative causes. Nerve conduction studies showed reduced sensory nerve action potentials characterized by axonal neuropathies. Paclitaxel was thus deemed the probable cause of this reaction by both the Naranjo Scale with a score of 6 and WHO-UMC criteria.

Day-wise Treatment Details

Day 1 – Presentation and Initial Evaluation

The patient presented with symptoms of progressively worsening tingling, numbness, and burning sensations occurring in both feet and hands, all of which affected daily activities. The pain was rated 7/10 on the Visual Analog Scale (VAS). Therefore, paclitaxel infusion was

withheld. Baseline laboratory values were all normal. Neurological examination showed diminished vibration and pinprick sensation in the lower limbs. Nerve conduction study demonstrated low sensory nerve action potentials, which were compatible with axonal sensory neuropathy.

Day 2 – Initiation of Supportive Therapy

Supportive therapy was commenced to manage neuropathy. Pregabalin 75 mg once daily and Amitriptyline 10 mg at bedtime were initiated for pain and sleep regulation. Folic acid 5 mg once daily, vitamin B-complex, and Calcium carbonate 500 mg twice daily were prescribed to support nerve health. The patient was counseled about lifestyle modifications including foot care, avoidance of trauma, use of supportive footwear, and monitoring for progression of sensory symptoms. Education focused on adherence to prescribed medications and early reporting of worsening symptoms. The initial response to supportive therapy was encouraging, with improved sleep and reduced anxiety.

Day 3 – Pain Control Optimization

With the persistence of neuropathic pain and slight relief, Gabapentin was prescribed at a 300-mg nighttime dose, to enhance analgesia with ongoing pregabalin and amitriptyline regimens. Topical capsaicin cream was prescribed for localized burning discomfort. Physiotherapy was commenced, including balance training, stretching, and gait stabilization exercises, for the prevention of function decline and enhancement of mobility. Mild symptomatic relief was felt by the patient at day end in the burning sensations, yet tingling and numbness continued. The treatment plan was very well tolerated with no systemic adverse effects observed. Sedation and drowsiness were kept under close monitoring.

Day 4 – Monitoring and Adjustments

By Day 4, the patient reported some improvement in the pain symptoms. Sedation did occur but did not interfere with his functional activities. Pregabalin 75 mg was continued once daily;

gabapentin, 300 mg at night with plans for titration. Renal and hepatic function tests were reviewed, remaining normal. The patient was advised to keep a daily pain diary to record symptom intensity, sleep quality, and overall functional status. The neurological examination showed no worsening of sensory deficits. The multidisciplinary team recommended continuing with the existing regimen on the strict condition of close observation for cumulative toxicity.

Day 5–7 – Symptom Stabilization

Over the course of a period of three days, a clinical case showed stabilization of symptoms while greatly improving. Pain intensity scores went down from 7/10 to 4/10, with neurological sensory examination revealing persistent but non-progressive sensory deficits. Physiotherapy exercises were continued, as the patient had reported better confidence in walking and less tiredness. Vitamin and folate supplementation were continued without adverse events. Emotional reassurance and adherence counseling were given as reinforcement. Patient's sleep had showed improvement, with the patient also expressing better tolerance to therapy. Laboratory results were unchanged. The clinical improvement suggested a stabilization of neuropathy, with an improvement in the quality of life and functional independence by the end of the first week.

Week 2 – Chemotherapy Reintroduction with Dose Modification

After a multidisciplinary reassessment, paclitaxel was cautiously reintroduced with a 20% dose reduction to strike a balance between efficacy and prevention of toxicity. Supportive therapies for neuropathy, including pregabalin, gabapentin, amitriptyline, folic acid, vitamin B-complex, and calcium, were maintained. Pain intensity remained stable at 4/10, with absence of new neurological symptoms. Hematological and biochemical parameters remained within the set norms, indicating systemic tolerance to the drug. The patient was advised to report the slightest worsening of symptoms at the earliest; weekly

follow-up visits for neurological examination were scheduled during chemotherapy.

Week 3–4 – Continued Follow-up

During the third and fourth weeks of treatment, the patient showed gradual improvement of symptoms. Pain intensity was scored at 3/10; supportive treatment was well-tolerated, with no major side effects. Pregabalin, 75 mg once daily, and gabapentin, 300 mg once at night, were continued. Physiotherapy gave support in regaining balance, mobility, and confidence about performing daily activities. Emotional counseling taught the patient how to cope with treatment-related anxiety, and as an outcome, the patient became more receptive to lifestyle changes. No new systemic abnormalities were detected on their follow-up investigations. The supportive regimen continued well in combination with paclitaxel at the reduced dose, thus exhibiting clinical stability and further improvement of neuropathic symptoms.

Week 6 – Re-evaluation and Long-term Planning

By the sixth-week follow-up, repeat nerve conduction studies revealed persistent axonal sensory neuropathy, without progression. Pain scores remained at a stable level of 3/10. No new deficit was noticed during neurological examination, and the patient was observed to make good improvements in carrying out daily activities. Supportive pharmacotherapy with pregabalin, gabapentin, and amitriptyline were kept ongoing, with tapering of gabapentin to be considered with symptom stability. Paclitaxel therapy (reduced dose) was continued without further complications. From this point, the patient remains under stringent follow-up with maintenance emphasis on regular neurological monitoring, adherence to supportive therapy, physiotherapy exercises, and lifestyle measures to improve quality of life and prevent neuropathic progression.

Outcome

After pharmacological therapy with pregabalin, gabapentin, and amitriptyline, along with supportive supplements, gradual abatement of neuropathic pain and tingling within two weeks was experienced. Neuropathy scores showed consistent improvement in terms of burning and numbness. Paclitaxel was resumed 20% dose-reduced at week 2, well tolerated with no further worsening of symptoms. Laboratory values remained stable, and no newly emerged systemic toxicities were found in follow-up. The patient continued chemotherapy at the reduced dose successfully with maintained symptom control, which means paclitaxel-induced peripheral neuropathy could be managed successfully without compromising cancer therapy.

Discussion

Paclitaxel is a cornerstone drug against breast cancer, and yet its clinical efficacy is routinely compromised by chemotherapy-induced peripheral neuropathy (CIPN). Typically, 30-70% of patients suffer paclitaxel-induced peripheral neuropathy (PIP), contingent on the cumulative dose and patient-related factors involved. It involves disruption of axonal transport by microtubule stabilization, mitochondrial dysfunction, and oxidative stress, which collectively impair the integrity of peripheral nerves. Sensory fibers are mostly affected, so the distal symmetric sensory neuropathy is manifested by burning pain, paresthesia, and numbness. Nerve conduction studies in our patient showed an axonal sensory neuropathy consistent with the prevailing reports wherein paclitaxel is mostly known to induce axonal and not demyelinating injury.^[13]

PIP is an extremely difficult diagnosis to distinguish from other kinds of neuropathies caused by different etiologies. Diabetes, thyroid dysfunction, alcoholism, and vitamin deficiencies are all known potential causes for peripheral neuropathy. In the present case, these considerations were carefully excluded by history, laboratory evaluation, and clinical examination to establish a stronger causality link with paclitaxel.

The Naranjo Adverse Drug Reaction Probability Scale showed a score of 6, whereas WHO-UMC causality criteria put this reaction under probable category. Adopting such structured assessment tools is very useful while making clinical decisions, as well as for pharmacovigilance. The literature also supports the use of causality assessment in oncology to guide dose modifications, supportive interventions, or other measures, thereby optimizing patient care without compromising on therapeutic efficacy.^[14]

PIP management is symptomatic and supportive, as no single therapy is curative. Drugs used for neuropathic pain include anticonvulsants such as pregabalin and gabapentin. Tricyclic antidepressants such as amitriptyline are frequently used in adjunctive management. The combination was found to provide almost the total relief of symptoms for our patient, in agreement with reports that conclude the usefulness of the two drugs in the treatment of CIPN. The patient was also supported with folic acid, vitamin B complex, and calcium supplementation for further support in nerve recovery. While the patient was symptomatically stabilized for about two weeks, paclitaxel was readministered at a 20% reduced dose. Dose modifications have been advocated for instead of discontinuation to help maintain oncological efficacy and minimize any further neurotoxicity.^[15]

This case highlights the clinical challenge posed by paclitaxel-induced peripheral neuropathy (PIP), a dose-limiting side effect typical of breast cancer patients receiving taxane-based chemotherapy. Our patient experienced distal burning pain, such as tingling and numbness, after the second cycle of paclitaxel, with nerve conduction studies confirming an axonal sensory neuropathy. Laboratory tests ruled out other causes, including diabetes, thyroid dysfunction, renal impairment, or vitamin deficiency. The Naranjo algorithm rated this reaction as probable, with a score of 6, and the WHO-UMC system rated it the same, thus establishing paclitaxel as the likely causative agent. A probable association was further supported by the absence of neuropathic signs during previous doxorubicin-cyclophosphamide treatment. This sequence

highlights the importance of conducting a systematic evaluation and a structured causality assessment in the diagnosis of chemotherapy-induced neuropathy.^[16]

In this case, the treatment was carefully set to relieve symptoms while maintaining cancer therapy. Pharmacologically, pregabalin, gabapentin, or amitriptyline could provide rapid improvement of neuropathic pain within two weeks, whereas supportive supplementation with folic acid, vitamin B-complex, and calcium helped in neuronal protection. Once stabilized, the patient could be rechallenged with paclitaxel, albeit at a 20% dose reduction, which was well tolerated and did not exacerbate neuropathy. Regular monitoring of neuropathy scores and laboratory parameters were ensured while continuing chemotherapy. This case highlights the importance of early diagnosis, prompt symptomatic management, and individualized dose reduction to prevent the progressive course of PIPN. It supports what has been mentioned in the literature-that with early intervention, patients can continue effective chemotherapy without compromising their long-term neurological disabilities.^[17]

Conclusion

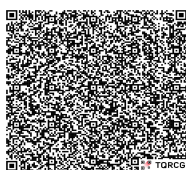
Paclitaxel remains a cornerstone in breast cancer management, yet the peripheral neuropathies are among the most distressing and dose-limiting toxicities. This case highlights how thorough evaluation, assessment of causality, and gradual approach-towards-treatment maximizes both patient safety and therapeutic outcome. This patient developed an axonal type of sensory neuropathy after paclitaxel therapy; the diagnosis was confirmed by nerve conduction studies following systematic exclusion of other etiologies. The strength of this association was made more probable, considering Naranjo scale and WHO-UMC probability assessment, and thus made more certain with respect to paclitaxel exposure. Early initiation of pharmacological treatment with pregabalin, gabapentin, and amitriptyline, along with some supportive supplements, brought relief from symptoms, and dose reduction of paclitaxel by 20% allowed safe

delivery of chemotherapy without worsening neuropathy. This case again reiterates the importance of early recognition and prompt institution of treatment with an individualized approach. Multidisciplinary integration and structure in monitoring would ensure symptom control without compromising on oncological efficacy, thus maintaining the overall quality of life.

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Access this Article in Online	
	Website: www.ijcrims.com
	Subject: Neuropharmacology
Quick Response Code	
DOI: 10.22192/ijcrms.2025.11.09.002	

How to cite this article:

Fatima Khader Unisa, Mohammed Sonia, Nalajala Akash, Sushma D R, Brinda Joshi, Krutika Dixit, Alapati Sathya Sai Guptha, Saniya Naaz, Arpitha C S,P. Joanna Grace. (2025). Paclitaxel - induced peripheral neuropathy in a breast cancer patient: A case report. *Int. J. Curr. Res. Med. Sci.* 11(9): 14-22. DOI: <http://dx.doi.org/10.22192/ijcrms.2025.11.09.002>