Management of Peripheral Neuropathy Caused by Microtubule Inhibitors

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Any patient receiving an agent that targets microtubules (e.g., taxanes, vinca alkaloids, epothilones) is at some risk for encountering peripheral neuropathy. This article provides tools and discussion to aid nurses in managing peripheral neuropathy in their patients through early identification and education. Some patients are at higher risk than others based on their chemotherapeutic regimen, pretreatment history, and comorbidities. When interacting with at-risk patients, nurses should be alert for primarily sensory neuropathy that presents as loss of sensation, numbness, or tingling, beginning at the distal ends of the extremities and moving proximally with a stocking or glove distribution. Clinical assessments for neuropathy generally employ grading scales, questionnaires, quantitative sensory testing, and psychometric assessments; each has benefits and limitations. Patients who experience moderate or severe neuropathy may require a dose reduction or delay until symptoms resolve; these patients may need a lower dose for the next treatment cycle. No known agents have proven to be more effective at reducing the incidence and severity of microtubule-induced peripheral neuropathy than regular neurologic examinations, early intervention, and patient education. In this respect, nurses can make a substantial difference in the impact of neuropathy on treatment efficacy and patients’ quality of life.

At a Glance

- Patients who are receiving microtubule inhibitors are at risk for peripheral neuropathy, but nursing intervention can minimize the incidence of severe events.
- As nurses interact with patients, they should be alert to early signs of peripheral neuropathy; several tools can assist this process.
- To date, no agents have proven to be more effective at reducing the incidence and severity of microtubule-induced peripheral neuropathy than early intervention and patient education.

Microtubules form when multiple copies of two building block proteins, α- and β-tubulin, form long chains in an energy-dependent polymerization process (Checchi et al., 2003; Wilson, Panda, & Jordan, 1999). Vinca alkaloids, taxanes, and epothilones all bind to β-tubulin, but vinca alkaloids block its polymerization with α-tubulin; taxanes and epothilones keep existing microtubules from reorganizing, thereby disrupting their dynamic function (Checchi et al.; Wilson et al.) (see Figure 1).
The exact mechanism by which the microtubule inhibitors promote peripheral neuropathy is not yet clear. Microtubules are known to form the cytoskeletal framework needed to maintain the architecture of neuronal axons. They also transport various cellular components from the neuronal cell body along the axon to the distal synapses (Hollenbeck & Saxton, 2005). Preclinical studies suggest that microtubule inhibitors may cause neuropathy because they disrupt this anterograde axonal transport (Theiss & Meller, 2000). Neuropathy may thus progress as the neuron undergoes a “dying back” process (Argyriou et al., 2008), starting at the distal nerve endings and progressing to involve the neuronal body, axon, and the Schwann cells that form the myelin sheath around the axon. Therefore, neuropathic symptoms may lag weeks or months behind the onset of chemotherapy and may persist for weeks or months more after treatment has ended (Ocean & Vahdat, 2004).

Although microtubule inhibitor-associated peripheral neuropathy can be debilitating, the condition is reversible and manageable in most cases (Argyriou et al., 2008; Bhagra & Rao, 2007; Perez et al., 2007; Thomas et al., 2007). This article discusses how nurses can assess and manage peripheral neuropathy associated with microtubule inhibitors, including how to identify at-risk patients, recognize early-stage symptoms, and help patients cope with potential neuropathic events.

### Risk Factors for Peripheral Neuropathy

Several factors may predispose a patient for peripheral neuropathy while they are receiving microtubule inhibitors (Argyriou et al., 2008) (see Figure 2). Use of combination therapy is increasing, and patients may be at a greater risk for severe neuropathy if they receive two neurotoxic agents concurrently (Ocean & Vahdat, 2004). In monotherapy, patients who receive higher taxane doses per cycle exhibit a higher incidence of severe peripheral neuropathy (Harvey et al., 2006; Nabholtz et al., 1996; Ocean & Vahdat; Winer et al., 2004).

Patients undergoing more frequent dosing also may be more likely to develop neuropathy, but again, overall dose per cycle appears to be the key factor. Several phase III studies in breast or lung cancer have associated weekly dosing of paclitaxel with higher rates of severe neuropathy than dosing every three weeks (Belani et al., 2008; Schuette et al., 2006; Seidman et al., 2008; Sparano et al., 2008). However, phase II trials have shown that administration of ixabepilone once every three weeks may be associated with higher rates of severe (grade 3 or higher) neuropathy than using lower doses for five consecutive days each cycle (Low et al., 2005; Perez et al., 2007; Thomas et al., 2007).

A shorter infusion time and, therefore, a higher concentration of drug per unit of time also may increase risk for neuropathy. One study randomized 24 patients with various advanced cancers to receive either one-hour or three-hour infusions of 100 mg/m2 paclitaxel (Mielke et al., 2005). Subsequent pharmacokinetic assessments revealed that only the time during which paclitaxel concentrations were 0.05 mcml/l or higher was independently associated with developing neuropathy, regardless of whether the infusion was one or three hours. This observation may help explain why paclitaxel appears to produce a greater risk of peripheral neuropathy when infused over three hours than when infused over 24 hours (Markman, 2003; Smith et al., 1999).

Patients with comorbidities that are associated independently with peripheral neuropathy also are at elevated risk for more severe microtubule inhibitor-related neuropathy. For example, peripheral neuropathy is a well-recognized microvascular complication of poorly-controlled diabetes, and using microtubule inhibitors in these diabetic patients may exacerbate neuropathic symptoms (Wolf, Sadetzki, Catane, Karasik, & Kaufman, 2005). A retrospective review undertaken to pinpoint neurotoxicity events in diabetic patients with advanced ovarian cancer identified that 9 of 18 patients treated with paclitaxel experienced progression of neuropathic symptoms after treatment (Gogas et al., 1996). In addition, a pooled analysis of 945 ixabepilone-treated patients with breast cancer identified diabetes as a risk factor for severe neuropathy (Bristol-Myers Squibb Co., 2007a).

Many pretreated patients with cancer exhibit baseline neuropathy from their prior treatment regimens. As such, standard doses of taxanes and vinca alkaloids are more likely to produce disabling neuropathy in this subset of patients (Chaudhry, Chaudhry, Crawford, Simmons-O’Brien, & Griffin, 2003). Of interest, in one case study of six patients who developed grade 2 peripheral neuropathy during taxane-carboplatin combination therapy, switching from paclitaxel to docetaxel reportedly decreased or resolved neuropathic symptoms, allowing patients...
to continue therapy (Rose & Smrekar, 2003). Age and concurrent treatment with other neurotoxic drugs also may raise the risk of microtubule inhibitor-associated peripheral neuropathy, but data on such associations are limited (Lee & Swain, 2006).

**Symptoms and Assessment**

Neuropathy symptoms typically are characterized according to onset, distribution, severity, and functional impact (Argyriou et al., 2008; Bhagra & Rao, 2007; Lee & Swain, 2006). Upon physical examination, sensory neuropathy first appears as an elevated vibratory perception in the distal extremities, particularly the great toe, often in association with loss of pain and temperature sensation. Deep tendon and ankle reflexes may be reduced. Symptoms of neuropathy occurring during treatment with microtubule inhibitors generally are mild to moderate in intensity but may become severe and even painful in some cases (Lee & Swain).

Early identification of patients who are at risk for developing neuropathy is crucial to limiting and managing the condition. Identifying at-risk patients or those with mild symptoms is facilitated by regular peripheral nervous system assessments. For all patients facing a treatment regimen associated with neuropathy, a physical assessment including a full or partial neurologic examination should be conducted at baseline, before each chemotherapy cycle, and whenever any complaints arise that could indicate new or progressing neuropathic symptoms.

The examinations provide an opportunity to interact with the patient one on one for oncology nurses as well as the entire practice. In addition to assessing symptoms as dictated in the standardized examination, nurses can gather additional information by listening to patients or by simply observing (i.e., a patient may not actually voice a complaint but may display subtle difficulties with a manual task, such as holding a pen). Therefore, even nonclinical staff should be educated regarding common signs of neuropathy so that the practice can adopt a team approach to patient observation.

**Qualitative Methods to Characterize Neuropathy**

Physical assessments of sensory peripheral neuropathy should focus primarily on the extremities, which are the most commonly affected sites. Patients’ responses can be documented on pictorial representations of the extremities, such as the arms and hands and the legs and feet (see Figure 3). The assessments can use various techniques (Bissett et al., 1993; Wickham, 2007).

The following techniques ask patients to report a sensation, usually while averting the eyes. The examiner begins testing distally and works proximally until the patient can feel the sensation normally.

- **Light touch:** Brush a cotton swab over the palm or sole.
- **Pin prick:** Gently apply a pointed object to the tip of the finger or toe.
- **Vibration:** Tap a tuning fork and place it against the extremity, usually beginning at the great toe.
- **Temperature:** Place a test tube containing hot or cold water against the extremity.
- **Positional sense or proprioception:** Move a digit or extremity up and down.

Other techniques ask patients to discriminate between sensory stimuli without looking.

- **Two-point discrimination:** Ask the patient to distinguish between one pointed object placed against the extremity (e.g., the forearm) or two pointed objects placed about 0.5 cm apart.
- **Nociception:** Ask the patient to distinguish between a pointed object (painful stimuli) and a dull object (nonpainful stimuli).
- **Stereognostic sense:** Place a small familiar object (e.g., a key) in the palm of the patient’s hand and ask him or her to identify it by feeling.

The techniques should be used as part of the regular neurologic assessment, the results of which can be used to grade patients’ neuropathy using a standardized grading scale. In general, clinical practices grade neuropathy using toxicity criteria set by the World Health Organization, the Eastern Cooperative Oncology Group, or the National Cancer Institute (NCI) (Miller, Hoogstraten, Staquet, & Winkler, 1981; NCI, 2006; Oken et al., 1982). All of the grading scales use combinations of neuropathic measurements that are subjective (e.g., degree of paresthesia) or objective (e.g., deep tendon reflexes in the ankles and wrists) (Ocean & Vahdat, 2004; Wickham, 2006). The U.S. Food and Drug Administration (FDA) cites the NCI’s Common Terminology Criteria for Adverse Events (CTCAE) version 3 as their preferred source for toxicity grading scales (FDA, 2009) (see Table 1).

Grading neuropathy gives healthcare professionals a convenient, standardized cutoff for when to apply interventions recommended in prescribing instructions, such as dose reductions.
In addition, keeping a record of how a patient’s neuropathy changes over the course of two or more neurologic assessments taken at baseline and again at subsequent visits can provide information as to how or whether the neuropathy is changing over time. As a result, investigators have evaluated the use of various measurement tools and scales. For example, the Total Neuropathy Scale correlates with the CTCAE grading system but may be more sensitive at detecting changes in chemotherapy-induced peripheral neuropathy than CTCAE grade alone (Cavaletti et al., 2007; Smith, Beck, & Cohen, 2008). The Total Neuropathy Scale denotes separate grades for sensory symptoms, motor symptoms, pin sensitivity, vibration sensitivity, reflexes, autonomic symptoms, vibration sensation, sural amplitude, and peroneal amplitude. Each neuropathic assessment is scored from 0–4, with higher scores indicating greater extent or severity of neuropathy (Cavaletti et al., 2003).

Many tools using the techniques discussed in this article are available to determine the extent of chemotherapy-induced peripheral neuropathy. Several studies have focused on the use of the scales in clinical practice because qualitative scales are subject to variable user interpretation in the clinical setting. The Oncology Nursing Society (ONS) provides a tabulated, evidence-based summary of the validity of the tools (visit http://onsopcontent.ons.org/toolkits/evidence/Clinical/pdf/NeuropathyOverview.pdf).

The expanded Peripheral Neuropathy Scale, a self-reporting tool based on common patient complaints, grades 11 symptoms specific to neuropathy in the hands and feet. Peripheral Neuropathy Scale scores for hand-and-foot neuropathy accurately correlated with NCI common terminology criteria grades, supporting the validity of the tool. However, some items reportedly produced more consistent patient responses and more strongly correlated with peripheral neuropathy (e.g., numbness, tingling, stiffness) than others (e.g., difficulty buttoning, feeling the shape of an object held in the hand) (Almadrones, McGuire, Walczak, Florio, & Tian, 2004).

In addition, some questionnaires designed to assess quality of life include assessments of sensory, motor, and autonomic neurologic symptoms; most notable are two of the Functional Assessment of Cancer Therapy (FACT) questionnaires—the taxane-specific questionnaire (FACT-taxane) (Cella, Peterman, Hudgens, Webster, & Socinski, 2003) and the Gynecologic Oncology Group questionnaire with a neurotoxicity subscale (FACT/GOG-Ntx) (Calhoun et al., 2003). To access the questionnaires, visit www.facit.org. Both scales contain 11-item subsets designed to assess neurotoxicity, and the scores are added to produce a cumulative score; lower total scores indicate more severe symptoms of neurotoxicity. In patients with non-small cell lung cancer receiving carboplatin and paclitaxel, the FACT-taxane subset scores decreased from baseline after 6 or 12

![Figure 3. Evaluation Forms for Neurologic Examination of the Upper and Lower Extremities](image-url)
weeks of therapy, reliably indicating the onset of neurotoxicity (Cella et al.). Likewise, the neurotoxicity subscale of the FACT/GOG-Ntx questionnaire could differentiate over time between patients with known neurotoxicity and those who were chemotherapy-naïve (Calhoun et al.). In both studies, parallel neuropathic assessments by quantitative means validated the scales as measuring tools for neurotoxicity.

Quantitative Methods to Characterize Neuropathy

Quantitatively evaluating peripheral neuropathy is sometimes desirable or necessary. All tools and tests discussed in this section are available commercially, usually as kits with instructions for patient assessment. Sensory neuropathy also can be assessed using computer-based systems to measure a patient’s response to stimuli. Quantitative sensation testing (QST) systems use preprogrammed algorithms to evaluate a patients’ thresholds for touch (e.g., pressure), temperature (e.g., hot and cold stimuli), and vibration.

QST has been studied extensively in other neuropathic settings, such as diabetes (Dyck, Dyck, Larson, O’Brien, & Velosa, 2000), but less is known about the validity of QST in assessing chemotherapy-induced peripheral neuropathy. Limited studies have shown that QST may not be as sensitive as a standard clinical examination at diagnosing chemotherapy-induced peripheral neuropathy, particularly at early stages (Lee & Swain, 2006). One study indicated that great toe vibration threshold probably was the most sensitive QST measurement for detecting paclitaxel-induced neuropathy, but QST still was less sensitive than clinical examination (Forsyth et al., 1997). In oxaliplatin-treated patients, maintenance of a thorough patient history was as effective as QST at evaluating peripheral neuropathy (Rambaud et al., 2001). A trial aiming to better characterize paclitaxel-induced neuropathy in patients with breast cancer evaluated neuropathy with the Total Neuropathy Scale and toxicity grading as well as QST via vibration threshold, but each technique was used to measure different parameters. In the study, measured neuropathy did not significantly vary between techniques. However, the parameters measured by QST were more specific and may have required greater sensitivity (Augusto et al., 2008).

Psychometric assessments that measure responses to stimuli also can be used to quantify neuropathy, but few tests have been evaluated formally in clinical studies of chemotherapy-induced neuropathy. A study of multiple functional tests identified two hand-function tests that reliably identified early symptoms of motor neuropathy in patients receiving ixabepilone therapy: the Jebsen-Taylor Test of Hand Function and the Grooved Pegboard test (Lee et al., 2005). The Jebsen-Taylor Test uses seven separate assays and an array of small objects to test a wide range of common hand functions. In the Grooved Pegboard test, which assesses dexterity and hand-eye coordination, the patient is asked to insert grooved pegs into a grid of holes. The pegs are grooved on one side only, so each must be rotated with the hand to insert it into a hole.

Some tools are specifically designed to characterize sensory neuropathy. The Semmes-Weinstein microfilament test, which is used to measure sensory perception threshold on the skin, has undergone only limited evaluation in the context of chemotherapy; however, the tool has been validated as a screening test for diabetic neuropathy (Perkins, Olaleye, Zinman, & Bril, 2001). The test uses a series of monofilaments, each of which is greater in diameter than the previous one and is designed to apply a unique amount of force; the monofilaments are applied to the skin, and the patient is asked to report when he or she feels the sensation (Visovsky, Meyer, Roller, & Poppas, 2008). Alternatively, the examiner repeatedly uses a single monofilament and records the number of correct reports by the patient (Perkins et al.).

Regardless of how neuropathy is assessed, more than one healthcare provider likely will conduct assessments on a single patient throughout his or her course of treatment. Therefore, standardization and uniformity regarding how and when peripheral neuropathy is measured, supported by a good interoffice communication system, are crucial to detecting neuropathic onset and progression.

Management of Peripheral Neuropathy Caused by Microtubule Inhibitors

Patients experiencing clinically relevant neuropathy—anything more substantial than minimal numbness and tingling in the fingers and toes—should have their drug dose reduced or delayed (see Table 2). In the event of severe (grade 3 or higher) peripheral neuropathy, clinicians should reduce paclitaxel doses by 20% (Bristol-Myers Squibb Co., 2007b) or discontinue docetaxel therapy (Aventis Pharmaceuticals, 2005). Likewise, ixabepilone prescribing instructions suggest reducing ixabepilone doses by 20% in the event of grade 2 neuropathy that lasts for more than seven days or any grade 3 neuropathy event.

### Table 1. National Cancer Institute Grading Scale for Peripheral Neuropathy

<table>
<thead>
<tr>
<th>GRADE</th>
<th>SENSORY NEUROPATHY</th>
<th>MOTOR NEUROPATHY</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Asymptomatic; loss of deep tendon reflexes or paresthesia (including tingling) but not interfering with function</td>
<td>Asymptomatic; weakness on examination or testing only</td>
</tr>
<tr>
<td>2</td>
<td>Sensory alteration or paresthesia (including tingling) interfering with function but not interfering with ADL</td>
<td>Symptomatic weakness interfering with function but not interfering with ADL</td>
</tr>
<tr>
<td>3</td>
<td>Sensory alteration or paresthesia interfering with ADL</td>
<td>Weakness interfering with ADL; bracing or assistance to walk (e.g., cane, walker) indicated</td>
</tr>
<tr>
<td>4</td>
<td>Disabling</td>
<td>Life-threatening; disabling (e.g., paralysis)</td>
</tr>
<tr>
<td>5</td>
<td>Death</td>
<td>Death</td>
</tr>
</tbody>
</table>

ADL—activities of daily living

Note. The scales are not intended for grading sensory or motor neuropathy related to cranial nerves. For the cranial neuropathy scale, visit http://ctep.cancer.gov/protocolDevelopment/electronic_applications/docs/ctcaev3.pdf.

Note. Based on information from National Cancer Institute, 2006.
discontinuing treatment entirely if grade 3 neuropathy persists for more than seven days, and not administering the next dose until the neuropathy resolves to grade 1 or better (Bristol-Myers Squibb Co., 2007a). The package inserts for the vinca alkaloids vincristine and vinblastine do not provide specific recommendations for managing peripheral neuropathy, although the vincristine sulfate insert recommends a 50% dose reduction for patients with a direct bilirubin serum value above 3 mg/100 ml (Sicor Pharmaceuticals, 1999). Neurotoxicity is a dose-limiting clinical toxicity; therefore, clinical evaluations and individual patient histories should be used to detect the need for dosage modification (Sicor Pharmaceuticals).

To date, no interventions have generated evidence that is conclusive enough for ONS (2006) to recommend a treatment for chemotherapy-induced peripheral neuropathy. However, chemotherapy-induced peripheral neuropathy frequently is reversible once therapy has ended. In three pivotal trials that investigated ixabepilone as monotherapy or in combination with capecitabine in heavily pretreated patients with metastatic breast cancer, peripheral neuropathy consistently resolved to baseline within an average of five to six weeks (Perez et al., 2008).

Several chemoprotective agents are being evaluated in clinical studies for the prevention or treatment of chemotherapy-induced peripheral neuropathy: calcium and magnesium infusions, amifostine, glutamine, glutathione, vitamin E, gabapentin, and recombinant human leukemia inhibitory factor (Lee & Swain, 2006; Ocean & Vahdat, 2004). In addition, multiple pharmacologic agents have been used to treat nonmalignant neuropathy (Armstrong, Almadrones, and Gilbert, 2005) provide an extensive list); the interventions may be useful if chemotherapy-induced peripheral neuropathy is associated with neuropathic pain.

Glutamine has shown some promise in preventing paclitaxel-induced neuropathy (Ocean & Vahdat, 2004). In a study of patients with breast cancer receiving high-dose paclitaxel (825 mg/m²), 12 patients who received glutamine (10 g TID for four days beginning 24 hours after paclitaxel infusion) experienced significantly fewer incidences of severe dysesthesia and numbness in the extremities (p < 0.05) than 33 patients who did not receive glutamine (Vahdat et al., 2001).

Although the antiepileptic gabapentin relieves taxane-induced myalgia at doses of 300–400 mg (Nguyen & Lawrence, 2004), six weeks of gabapentin (2,700 mg target dose) failed to produce a statistically significant improvement in chemotherapy-induced peripheral neuropathy (Rao et al., 2007). However, another study demonstrated that a daily regimen of gabapentin (2,400 mg) and sustained-release morphine (60 mg) was significantly more effective than placebo at lowering pain scale scores in patients experiencing diabetic neuropathic pain (Gilmington et al., 2005).

Calcium or magnesium infusions may help to prevent oxaliplatin-associated neuropathy, but the chelating mechanism associated with calcium and magnesium may not be useful against neuropathy caused by microtubule inhibitors (Ocean & Vahdat, 2004). Studies of the compounds in a chemotherapy setting are in progress.

Fewer studies have evaluated interventions once neuropathy has developed, but two small studies reported promising results for acetyl-L-carnitine. At 1 g TID for eight weeks, the fat metabolism intermediate improved sensory and motor neuropathy grades and total neuropathy scores in patients who experienced severe neuropathy during paclitaxel or cisplatin therapy (Bianchi et al., 2005; De Grandis, 2007). Some evidence shows that patients also may gain relief from taxane-induced neuropathic pain by using amitriptyline (10–50 mg) (Seidman et al., 1998).

Although data in chemotherapy recipients are extremely limited, studies have explored nonpharmacologic interventions for peripheral neuropathy (e.g., acupuncture, physical activity) because evidence suggests that the treatments might aid in managing peripheral neuropathy associated with diabetes or HIV (Visovsky, Collins, Abbott, Aschenbrenner, & Hart, 2007). The benefits of other approaches, such as magnetic therapy, massage, and hydrotherapy, are not well supported by clinical neuropathy studies; however, the interventions may improve quality of life if they are pleasant experiences for patients, and they generally are harmless when they are well controlled (e.g., avoiding extremes in water temperature) and affordable to patients (Armstrong et al., 2005).

Physical activity as an intervention for neuropathy is associated with favorable (e.g., increased muscle strength, return to work) and unfavorable (e.g., worsening neurologic symptoms, pain requiring analgesics) secondary outcomes. A Cochrane analysis evaluating exercise as an intervention for peripheral neuropathy revealed a lack of well-designed studies in this area; most were not clearly randomized or controlled, and many had very small sample sizes. Among the few trials that met the inclusion criteria for the analysis, none were conducted in chemotherapy recipients and none could demonstrate that exercise therapy improved functional ability.
in patients with peripheral neuropathy (White, Pritchard, & Turner-Stokes, 2004).

To date, the most effective approach for preventing severe neuropathy is educational intervention, and nurses should provide patients with practical advice for detecting and managing peripheral neuropathy (Marrs & Newton, 2003; Visovsky et al., 2007) (see Figure 4). Patients should know the early signs and symptoms of neuropathy so that they can be reported while neuropathy is still mild in intensity. Patients also should be advised on how to avoid harmful situations if they lose sensation in their extremities and how to compensate for such losses to maintain their personal safety.

Patients also should be thoroughly educated regarding how neuropathic events might affect their treatment course. Some patients may be reluctant to report early symptoms of neuropathy to their healthcare team if they are afraid that their treatment will be discontinued. As demonstrated in this article, many neuropathic events can be addressed through a dose reduction or a temporary treatment delay while still preserving treatment efficacy. In addition, patients should be aware that early intervention is the most effective approach currently available to prevent severe events and that unreported early neuropathy that is allowed to progress is more likely to interrupt treatment and thus negatively impact efficacy.

Conclusions

Although no specific interventions are available to date for preventing or treating peripheral neuropathy, early identification of at-risk patients and mild peripheral neuropathic symptoms can effectively prevent most events from progressing to severe neuropathy. Moderate-to-severe (grade 2 or higher) neuropathy may warrant a dose reduction or delay; severe events may even require a treatment delay until symptoms improve to grade 1 or resolve completely. To prevent future severe events in these patients, healthcare providers should consider using a reduced dose in the next treatment cycle.

Nurses must accurately and continuously monitor for signs to identify patients who may encounter symptoms of chemotherapymduced peripheral neuropathy. Nurses also should include patients as a part of the healthcare team by encouraging them to report symptoms early and teaching them how to self-manage their neuropathic events. In these respects, nurse intervention can make the crucial difference in whether neuropathy significantly impacts patients’ quality of life during cancer treatment.

The author takes full responsibility for the content of the article but thanks Rebecca Goldstein, PhD, of StemScientific, supported by Bristol-Myers Squibb Co., for medical writing support. The author did not receive honoraria for this work. The content of this article has been reviewed by independent peer reviewers to ensure that it is balanced, objective, and free from commercial bias. No financial relationships relevant to the content of this article have been disclosed by the independent peer reviewers or editorial staff.

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References


Figure 4. Practical Advice for Patients About Managing Peripheral Neuropathy

Note. Based on information from Visovsky et al., 2007.
Clinical Journal of Oncology Nursing  •  Volume 13, Number 6  •  Peripheral Neuropathy Caused by Microtubule Inhibitors


