Recently, chemotherapy-induced peripheral neuropathy has received a great deal of attention. However, the interaction of diabetic neuropathy with potentially neurotoxic chemotherapy is far less understood. The incidence of type II diabetes has risen exponentially in the past two decades. In concert with the rise in type II diabetes, the number of individuals with diabetes who need chemotherapy for cancer also is expected to increase. Diabetic neuropathy and the neurotoxic effects of chemotherapy have a significant potential to cause functional disability. Diabetics may be most at risk for the effects of neurotoxic agents on peripheral nerve functioning, in addition to the other effects induced by chemotherapeutic agents. The purpose of this article is to review the evaluation, management, and clinical implications of peripheral neuropathy in patients with cancer and diabetes.

**At a Glance**
- Comorbid illnesses, such as diabetes, can result in increased incidence and severity of peripheral neuropathy associated with cancer treatments.
- Assessment of patients with diabetes undergoing treatment with known neurotoxic agents should include objective and subjective measures.
- Patients with cancer and diabetes require specialized patient education regarding safety measures and the importance of glycemic control.

Cancer remains a challenging problem for individuals with comorbid conditions, as underlying disease states can result in increased toxicities and morbidity associated with cancer treatments. Diabetes mellitus is a common comorbidity in the general population, with 8%–18% of patients with cancer affected (Psarakis, 2006). Diabetes is associated with the development of peripheral neuropathy in 37%–59% of affected patients (Dyck et al., 1995; Herman & Kennedy, 2005; Kastenbauer, Irsigler, Sauseng, Grimm, & Preger, 2004). Preexisting conditions may be present in the diabetic population because of long-term damage from poorly managed blood glucose, including renal, cardiac, and neuropathic complications (Psarakis). Diabetes-associated peripheral neuropathy may result in increased toxicities and morbidity when compounded by neurotoxic cancer treatments. Consequently, peripheral neuropathy (see Figure 1) that worsens in the presence of diabetes can lead to treatment delays, dose reductions, or treatment discontinuation. The purpose of this article is to review the evaluation, management, and clinical implications of peripheral neuropathy in patients with cancer and diabetes.

**Peripheral Neuropathy**

**Diabetic Peripheral Neuropathy**

Although its exact cause is unknown, the development of diabetic peripheral neuropathy has been associated with a decrease in sodium-potassium adenosine triphosphatase (Na⁺-K⁺-ATPase) activity and hyperglycemia that ultimately results in the accumulation of sorbitol and other metabolites in peripheral nerves, impairing nerve blood flow and leading to hypoxia, vascular degeneration, and sensory neuropathy (Low, Nickander, & Scionti, 1999). In addition, decreased Na⁺-K⁺-ATPase activity results in elevations in intra-axonal sodium and a blockage of nerve membrane depolarization (Nicolucci et al., 1996; Raccah, Fabregueettes, Azulay, & Vague, 1996; Veves, 1999).