Evaluation and Management of Peripheral Neuropathy in Diabetic Patients With Cancer

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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.

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, Fabreguettes, Azulay, & Vague, 1996; Veves...