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Why You Should Consider The Use Of Supplements In The Management Of Diabetic Neuropathy



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Allen Jacobs DPM FACFAS
management-diabetic-neuropathy&redirect=http%3A%2Fwww.podiatrytoday.com

Supplements have proven useful for improved management of diabetes and diabetes-related complications such as neuropathy, wounds, cardiovascular complications, renal complications and ophthalmic complications.

In looking at practical and rational therapies for the management of neuropathy, Singleton and Smith noted that medication and lifestyle changes should aim at improving hyperglycemia and insulin resistance, and reducing obesity and dyslipidemia.¹ With reference to specific pharmacologic therapy, the authors noted that one should direct therapy at rational targets for known pathophysiology. Furthermore, these authors noted that treatment should aim at prevention of complications such as ulceration.

In the patient with diabetes, there are well-documented, specific deficiencies. However, **most podiatric physicians are not trained in the use of supplements for the treatment or prevention of diabetes-related pathology. Most podiatric physicians are not familiar with nutritional or supplement therapy,** and the literature relevant to the treatment of neuropathy. The majority of podiatric physicians tend to relegate supplement therapy to complementary alternative therapies rather than looking at the potential for disease prevention or resolution.

Examples of identified deficiencies include deficiencies of magnesium, calcium, zinc, riboflavin, folate and vitamin A for the healing of diabetic foot wounds. In the case of neuropathy, long axons with smaller cell bodies are most vulnerable to the effects of malnutrition, environmental insults and reduced vascular supply with impaired autoregulation. The result is decreased blood flow to the nerves, relative ischemia, hypoxia and oxidative stress.²

Chromium, for example, is a trace element required for glucose metabolism and lipid metabolism. The administration of chromium is effective for reducing glucose levels and improving glucose metabolism as well as decreasing insulin requirements. Chromium deficiency has been associated with increased serum glucose levels, cholesterol and triglyceride levels, and decreased HDL levels.³ Chromium deficiency has been associated with both peripheral neuropathy as well as central nervous system disorders.

Researchers have shown that the administration of chromium picolinate decreases hemoglobin A1c.³ Chromium increases insulin binding, insulin receptor site numbers and receptor fossa correlation.

Alpha lipoic acid is an antioxidant that increases insulin sensitivity and improves glucose metabolism diabetic neuropathy and vascular perfusion of tissues.

Polyphenols are antioxidants found in tea and dark chocolate. Researchers have found them to be associated with protective cardiovascular effects as well as increased glucose control and insulin activity.⁴ A common source of such polyphenols is green tea.

Cinnamon has an insulin-like activity. In a meta-analysis of 435 patients receiving cinnamon at a dose of 6 g per day, cinnamon had a beneficial effect on glycemic control with lowering of hemoglobin A1c as well as fasting blood glucose levels.⁵

We have long known zinc supplementation to be helpful in diabetic wound healing. Zinc improves the function of the immune system, providing antioxidant activity and glucose homeostasis. With reference to wound healing, zinc stabilizes cell membranes, is an enzyme cofactor and is required for cell mitosis and cell proliferation.⁶ Zinc is required for cell growth and proliferation, for protein, DNA and RNA synthesis. The administration of 50 or 20 mg of zinc per day improved wound healing within 24 hours.

The role of supplements in diabetic management can be summarized by the conclusions of Boulton and colleagues.⁷ They noted "emerging evidence of positive findings with some natural products... has been reported in glycemic parameters, markers of cardiovascular risk, and quality of life in individuals with type 2 diabetes. However, further investigation in well designed, adequately powered studies is needed."

A Closer Look At Diabetic Neuropathy

Diabetic neuropathy may manifest as sensory, motor or autonomic dysfunction. The most significant complications of diabetic neuropathy are associated with loss of sensation. Although many patients with diabetic neuropathy develop paresthesia and dysesthesia, the majority of patients affected by diabetic neuropathy suffer from loss of sensation, with associated ulceration, Charcot joint disease, infection and not infrequently, amputation. Additionally, motor neuropathy may manifest itself as aching pain, or a loss

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of intrinsic foot musculature and the development of bunion, hammertoe or metatarsal head prolapse. In addition, motor neuropathy may be associated with decreased knee and ankle reflexes, and weakness of the extrinsic foot musculature.

Diabetic autonomic neuropathy may be associated with decreased ability to sense the position of the toes and feet, vasomotor instability, neuropathic edema, or cold paresthesia.⁷ As the majority of patients with diabetic neuropathy suffer from decreased or loss sensation rather than paresthesia or dysesthesia, clinical examination by the podiatric physician is critical to determine the presence of neuropathy as the patient may not be aware of sensory loss. Examination with a 128 Hz tuning fork, pinprick, 10 g filament and ankle reflexes can determine the presence of diabetic neuropathy without the need of advanced diagnostic testing, with an 87 percent sensitivity.⁷ Therefore, the podiatric physician may generally diagnose diabetic neuropathy in its most common presentation without the need of sophisticated or advanced diagnostic modalities.

What You Should Know About The Treatment Of Diabetic Neuropathy

Remember that diabetic neuropathy is a metabolic disorder. Therefore, the treatment of diabetic neuropathy must include not only management of diabetes but reversal of those metabolic abnormalities that cause diabetic neuropathy. A variety of metabolic disorders have been associated with diabetic neuropathy. They include increased sorbitol accumulation, increased fructose, decreased myoinositol, glycosylated end products, reactive oxygen species, decreased nitric oxide, activated protein kinase C and decreased nerve growth factors, to name but some of the metabolic etiologies proposed for diabetic neuropathy.⁷

The treatment of diabetic neuropathy therefore must include interdiction of symptomatology such as paresthesia or dysesthesia, prevention of nerve degeneration and advancement of the neuropathic process, and the enhancement of nerve regeneration.⁹

Why You Should Consider Vitamin D Supplementation For Diabetic Neuropathy

We commonly consider vitamin D in discussions of bone metabolism and health. The effects of vitamin D deficiency include decreased calcium absorption, increased risk of osteoporotic fractures, delayed fracture healing, decreased neuromuscular coordination and muscle function. In addition, vitamin D plays a significant role in wound healing, participating in cell growth and differentiation, reversal of corticosteroid-induced epidermal atrophy, and the inhibition of hyperplastic epidermal tissue formation. This results in increased rates of wound healing and increased wound tensile strength.

Recently, researchers have defined the role of vitamin D in diabetic neuropathy. Soderstrom and coworkers noted that a significant number of patients with diabetic neuropathy suffered from vitamin D deficiency.⁹ This deficiency was most common in Hispanic Americans and non-Hispanic African-Americans. Therefore, it is possible that some patients with diabetic neuropathy may benefit from the simple administration of vitamin D.

Can Vitamin B12 (Methylcobalamin) Supplementation Have An Impact For Diabetic Neuropathy?

Increasingly, we have identified the role of vitamin B12 in diabetic neuropathy and the need to supplement B12 in the patient with diabetes. Metformin, the most commonly utilized drug for the treatment of diabetes, is associated with worsening of diabetic neuropathy due to the inhibition of folic acid and B12 absorption as a result of the effects of metformin.¹⁰

Authors have demonstrated that the malabsorption of B12 and decreased folate levels associated with metformin result in an increased homocysteine level and increased risk of neuropathy.^{11,12} The administration of B12 may be associated with a reversal of these effects. Homocysteine, when elevated, results in endothelial damage and decreased blood flow to peripheral nerve tissue by thrombus formation within the vascular supply to the nerve. In addition, homocysteine impairs the coupling of arginine and oxygen for the formation of nitric oxide, which is necessary to maintain vascular supply and normal nerve function.¹³ Cobalamin deficiency, which is exacerbated by the utilization of metformin, increases risk of neuropathy. B12 supplementation can be helpful in the reversal of this deficiency and neuropathy.

In addition to those patients utilizing metformin, researchers have demonstrated that vitamin B12 deficiency and folate deficiency are associated with increased levels of homocysteine, increased oxidative stress secondary to low levels of glutathione, and decreased total antioxidant activity.¹⁴ Bailey and colleagues noted that cobalamin deficiency is common and the majority of cases are subclinical.¹⁵ In addition, they have suggested that cobalamin serum testing is unreliable.

Solomon demonstrated that functional B12 deficiency is common in the elderly and common in 62 percent of patients with diabetic neuropathy.¹⁶ In these patients, normal serum cobalamin levels were associated with markers for B12 deficiency such as elevated levels of methylmalonic acid and the presence of neuropathy. Solomon showed that the administration of cobalamin reversed the effects of functional B12 deficiency in many patients.

Wyckoff and Ganji demonstrated that vitamin B12 deficiency may exist even in the presence of patients who presumably have a "large intake" of B12.¹⁷ They further demonstrated that classic markers of B12 deficiency such as macrocytic anemia are not reliable markers for B12 deficiency.

The administration of methylcobalamin at 1,500 mcg per day has been associated with normalization of hemoglobin A1c and improved motor conduction velocity, thereby suggesting that vitamin B12 therapy may be helpful in the reversal of diabetic motor neuropathy, or symptoms such as cramping.¹⁸ Others have demonstrated that the administration of methylcobalamin at 1,500 mcg daily was associated over three months with relieved muscle cramping, improvement in motor conduction velocity, reduction in neuropathic pain and improvement in two point discrimination, suggesting nerve regrowth.¹⁹

Yaqub and co-workers have demonstrated that methylcobalamin improves not only sensory but autonomic nerve dysfunction symptoms as well.²⁰ With reference to autonomic neuropathy, multiple studies have demonstrated the reversal of autonomic signs, autonomic symptoms and the normalization of autonomic nerve dysfunction with the administration of methylcobalamin.^{21,22} Authors have also described a potential neuroprotective effect together with reversal of symptomatic sensory and autonomic neuropathy.²³⁻²⁶

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Permalink (/comment/9045#comment-9045) Submitted by DrDPM (not verified) on Wed, 06/18/2014 - 21:40

Dear Dr Jacobs:

How about educating diabetics (especially those taking metformin) about eating chicken and salmon daily for lunch and/or dinner to replenish B12 and B3 levels in the body? These foods are high in B12 and B3. Is this treatment strategy also just as good as taking multivitamin B supplement?

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