Alpha-Lipoic Acid for Diabetic Neuropathy

Four hundred forty-three patients with chronic painful neuropathy were treated with 600 mg per day of alpha-lipoic acid (ALA) for a mean period of five years. ALA was then stopped, and 293 patients were switched to gabapentin (600–2400 mg per day), while 150 patients remained untreated because they had no current symptoms. In the untreated group, 110 patients (73%) developed neuropathic symptoms as soon as two weeks after the end of treatment with ALA.

Comment: ALA has been reported to decrease oxidative stress and to prevent glycosylation of proteins (an early step in the formation of advanced glycation end products). Treatment with ALA has also been found to improve microcirculation in patients with diabetic neuropathy. Each of these effects of ALA would be expected to be of value for the prevention or treatment of diabetic neuropathy. Numerous clinical trials have found that ALA decreases symptoms in patients with diabetic neuropathy, but these trials have generally been short term (three weeks to four months). The results of the present study suggest that the beneficial effects of ALA persist for up to five years with continued treatment.

Food Additives Harmful for Kidney Patients

Two hundred seventy-nine patients with end-stage renal disease and hyperphosphatemia were randomly assigned to an intervention group or a control group. The intervention group received education on avoiding foods with phosphorus additives when purchasing groceries or visiting fast-food restaurants. The control group received usual care. After three months, the decline in serum phosphorus levels was 0.6 mg/dl greater in the intervention group than in the control group (p = 0.03).

Comment: Patients with end-stage renal disease have an impaired capacity to excrete phosphorus and are at risk of developing hyperphosphatemia, which can increase morbidity and mortality. Renal patients are therefore advised to restrict intake of high-phosphorus foods (such as meats, dairy products, whole grains, and nuts), and are also treated with phosphate binders. In recent years, phosphorus-containing food additives (primarily in processed and fast foods, particularly meats, cheeses, baked goods, and beverages) have become an important source of dietary phosphorus, contributing up to one-third of total phosphorus intake in the general population. The results of this study suggest that avoiding these foods could improve long-term outcomes in patients with chronic renal failure.

Potato Chips May Cause Heart Disease

Fourteen healthy volunteers (mean age, 35 years) ingested 160 g per day of potato chips for four weeks. The mean concentration of oxidized LDL increased significantly (p < 0.01): by 21% in nonsmokers and by 29% in smokers. The mean C-reactive protein concentration also increased significantly (p < 0.01): by 56% in nonsmokers and by 44% in smokers.

Comment: Oxidized LDL causes arterial injury, which is one of the earliest steps in the development of atherosclerosis. In addition, an elevated concentration of C-reactive protein (a marker of inflammation) is an independent risk factor for cardiovascular disease. Eating a relatively large amount of potato chips for four weeks increased both the oxidation of LDL and C-reactive protein levels, suggesting that overdoing it on potato chips can promote the development of cardiovascular disease. The adverse effects of potato chips are presumably due to free radicals and advanced glycation end products, which are formed during high-temperature cooking of potatoes in oil. Previous studies have shown that eating foods high in advanced glycation end products increases C-reactive protein levels. Emphasizing boiling and poaching of animal foods, as opposed to frying, grilling, and baking, can reduce the advanced glycation end products content of foods substantially.

Vitamin D Deficiency and Statin-Induced Myalgia

Serum 25-hydroxyvitamin D levels were measured in 621 patients receiving a statin drug, of whom 128 were experiencing myalgia. The mean 25-hydroxyvitamin D level was significantly lower in the symptomatic patients than in the asymptomatic patients (28.6 ng/ml vs. 34.2 ng/ml; p < 0.0001), whereas no difference was seen between groups with respect to factors that might influence vitamin D status, such as age, body mass index, and frequency of type 2 diabetes. A significantly higher proportion of symptomatic than asymptomatic patients had a low serum 25-hydroxyvitamin D level (defined as < 32 ng/ml or < 80 nmol/L) (64% vs. 43%; p < 0.0001). Of the 82 patients with myalgia and a low 25-hydroxyvitamin D level, 38 received vitamin D2 (50 000 IU per week for 12 weeks), while continuing statin therapy. The mean serum 25-hydroxyvitamin D level increased from 20.4 ng/ml to 48.2 ng/ml, and 92% of the patients experienced a resolution of the myalgia.

Comment: Myalgia is one of the manifestations of vitamin D deficiency and is also the most common side effect of statin drugs. The results of the present study suggest that suboptimal vitamin D status increases susceptibility to developing statin-induced myalgia, and that improving vitamin D status can reverse the myalgia. Paradoxically, treatment with statin drugs has been reported to increase 25-hydroxyvitamin D levels. Apparently, in some patients, the drug does not improve vitamin D status enough to prevent drug-induced myalgia.

Ahmed W et al. Low serum 25 (OH) vitamin D levels (<32 ng/mL) are associated with reversible myositis-myalgia in statin treated patients. Transl Res. 2009;153:11-16.