Editorial
Is Coenzyme Q10 an Effective Treatment for Heart Failure?

Coenzyme Q10 (CoQ10) is widely used by nutritionally oriented practitioners to treat heart failure. However, conventional review articles on heart failure either do not mention CoQ10 or conclude that additional research is needed before its use can be recommended. Are the natural-medicine doctors wasting their patients' money, or are the conventional doctors overlooking an effective treatment? The short answer, according to my clinical experience and reading of the medical literature, is the latter.

As a cofactor in the electron-transport chain, CoQ10 is required for the synthesis of adenosine triphosphate (ATP), the body's major form of stored energy. Since most cellular functions are dependent on an adequate supply of ATP, CoQ10 is essential for the health of virtually all human tissues and organs. Although CoQ10 can be synthesized in vivo, situations may arise in which the body's synthetic capacity is insufficient to meet CoQ10 requirements. Susceptibility to CoQ10 deficiency appears to be greatest in metabolically active tissues such as the heart.

Low levels of CoQ10 have been observed in both serum and myocardial tissue of patients with various types of cardiovascular disease. In patients with heart failure due to cardiomyopathy, oral administration of 100 mg/day of CoQ10 for two to eight months increased myocardial CoQ10 concentrations by 20-85%. Thus, if CoQ10 deficiency is a contributing factor to heart failure, CoQ10 supplementation could have clinical benefits.

Numerous clinical trials have demonstrated a beneficial effect of CoQ10 in patients with heart failure. A few of these are summarized below:

One hundred twenty-six patients with heart failure due to dilated cardiomyopathy (98% of whom were in New York Heart Association [NYHA] functional class III or IV) received 100 mg/day of CoQ10 for up to 66 months. The mean left ventricular ejection fraction increased from 41% at baseline to 59% after six months of treatment (p < 0.001) and remained stable thereafter with continued treatment. After two years, 84% of the patients were still alive, and after 5.5 years, 52% were alive. These survival rates are considerably better than published survival statistics for patients given conventional therapy (i.e., a two-year survival rate of 50% for symptomatic cardiomyopathy and a one-year survival rate of 50% for decompensated cardiomyopathy).

In a multicenter trial, 1,113 patients with heart failure (NYHA class III or IV) were randomly assigned to receive placebo or CoQ10 (2 mg/kg of body weight per day) for one year. The number of patients requiring hospitalization during the study for worsening heart failure was 38% less in the CoQ10 group than in the placebo group (p < 0.001). Episodes of pulmonary edema were about 60% less frequent in the CoQ10 group than in the placebo group (p < 0.001).

In a double-blind trial, 641 patients with heart failure awaiting heart transplantation were randomly assigned to receive, in double-blind fashion, 60 mg/day of Ultrasome-Q10 (a special preparation of CoQ10 designed to increase intestinal absorption) or placebo for three months. The patients receiving CoQ10 showed a significant improvement in the six-minute
walk test (from 270 to 382 meters; p < 0.0001), as compared with a deterioration in the placebo group (from 254 to 177 meters). Significant improvements were also seen in the CoQ10 group in dyspnea (p = 0.04), NYHA classification (from a mean of 3.1 to 2.4; p = 0.01), nocturia (p = 0.01), and fatigue (p < 0.001). These parameters did not improve in the placebo group.5

Although the majority of studies of CoQ10 for heart failure have shown beneficial effects, two well-controlled, double-blind trials reported negative results. In one of these studies, 55 patients with heart failure were randomly assigned to receive 200 mg/day of CoQ10 or placebo for six months. Although the mean serum CoQ10 concentration increased significantly in the active treatment group, there was no change in either group in left ventricular ejection fraction, peak oxygen consumption, or exercise duration.6 Similar negative results were seen in another double-blind trial.7

One possible explanation for the conflicting results is that there were differences in nutritional status among the different study populations. One of the negative studies was performed at an inner-city hospital and a Veterans Affairs Hospital. In this population, there may have been a relatively high prevalence of alcoholism (alcoholic heart disease) and dietary inadequacies. Those factors, combined with the use of nutrient-depleting loop diuretics, could have led to clinically significant deficiencies of magnesium, thiamine, or other nutrients that are needed for normal cardiac function. Just as a chain is only as strong as its weakest link, CoQ10 therapy may be effective only when a person is not deficient in other cardioprotective nutrients.

In order to resolve the conflicting studies of CoQ10 and heart failure, a trial should be performed in which CoQ10 is compared with a placebo in patients in whom all other nutritional deficiencies have been corrected. However, even without definitive proof of efficacy, it is reasonable to include CoQ10 as part of a comprehensive treatment program for heart failure, because it is safe and, with one exception, does not appear to interact with cardiac medications. CoQ10 may interfere with the effect of warfarin, according to some case reports and animal studies, although one clinical trial suggested that such an interaction is uncommon, if it occurs at all.8 To be on the safe side, CoQ10 should be used with caution and with appropriate laboratory monitoring in patients who are receiving warfarin.

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