MINIMIZING THE RISK of Cholesterol-Lowering Therapy

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Medications prescribed to prevent heart problems may also create new risks for the patient. Hepatic hydroxymethyl glutaryl coenzyme A (HMG CoA) reductase inhibitors (statins), which have been available since 1987, have been used as prophylaxis against cardiac problems. In 1990, Folkers et al. disclosed a side effect of the statin lovastatin (Mevacor) that was potentially harmful to the heart and had not been previously disclosed in the package literature. Although the article by those authors was written about lovastatin in particular, statins in general work by blocking the production of cholesterol at the mevalonate pathway. Blocking this pathway also decreases the production of coenzyme Q10. The symptoms of coenzyme Q10 deficiency include cardiac disorders such as arrhythmias, angina, congestive heart failure, and fatigue; hypertension; and depression of the immune system.

In another study of cardiomyopathy, 137 patients who received coenzyme Q10 were compared with 182 patients who received conventional therapy without coenzyme Q10. In the group who received coenzyme Q10, the survival rate was approximately 75% at 46 months after the initiation of treatment. In those treated with conventional therapy without coenzyme Q10, the survival rate was about 25% at 36 months. The mean serum coenzyme Q10 level increased from 0.85 ± 0.26 µg/mL to a value ranging from 1.7 to 2.3 µg/mL in the group treated with coenzyme Q10. In the study cited, the improved cardiac function and prolonged survival rate in the group treated with coenzyme Q10 indicate that the correction of coenzyme Q10 deficiency is essential in patients with cardiomyopathy who undergo treatment with statins.

Additional Benefits of Coenzyme Q10 Therapy

Because coenzyme Q10 benefits patients with CHF, its use in combination with statins should be of value in patients who have a high cholesterol level. A US patent was issued to Merck & Co, Inc (Rahway, New Jersey), for the following therapy: “A method of counteracting HMG-CoA reductase inhibitor-associated skeletal muscle myopathy in a subject in need of such
treatment which comprises the adjunct administration of a therapeutically effective amount of HMG-CoA reductase inhibitor and an effective amount of Coenzyme Q₁₀ to counteract said myopathy.” Merck is also the assignee for US Patent 4,929,437, which applies to a pharmaceutical composition and method of counteracting elevated transaminase levels associated with HMG-CoA reductase inhibitor. The method described comprises the adjunct administration of an effective amount of a statin inhibitor and an effective amount of coenzyme Q₁₀. Merck was impressed enough with the potential effect produced by coenzyme Q₁₀ in conjunction with the use of statins to apply for and receive patents for that combination of therapies. It does not seem prudent to wait for Merck or any other drug company to apply for an Investigational New Drug application and to conduct trials specified by the Food and Drug Administration to prove that the combination of a statin and coenzyme Q₁₀ is safe and effective. Coenzyme Q₁₀ seems to produce important life-changing benefits such as increasing the pumping ability of the heart and improving the quality of life according to the NYHA classification scale. I would recommend this supplement for any patient or anyone taking a cholesterol-lowering medication; it produces little (if any) risk to patients. It is sold as a nonprescription drug, and if it is recommended by pharmacists, coenzyme Q₁₀ could improve the quality of life for those who use statins or have heart disease. Its use may even save the lives of those with coenzyme Q₁₀ deficiency.

Case Reports

Case Study 1

The patient, a 55-year-old white man with ischemic cardiomyopathy, was designated as having class III disease according to the NYHA classification of functional capacity. In May after his diagnosis, treatment was initiated with orally administered coenzyme Q₁₀ 100 mg, which was given once daily. When therapy was initiated, the patient’s baseline blood level of coenzyme Q₁₀ was 0.67 µg/mL, and his baseline ejection fraction was 60%. One month later, his blood level of coenzyme Q₁₀ had increased to 1.73 µg/mL, and his ejection fraction had increased to 74%. Data on the patient’s blood levels of coenzyme Q₁₀ and ejection fractions during a 3-year period indicate that daily therapy with orally administered coenzyme Q₁₀ maintained a therapeutic level of coenzyme Q₁₀ of 1.73 to 2.78 µg/mL and ejection fractions of 64% to 70%. During 3 years of therapy with coenzyme Q₁₀, the patient’s classification of ischemic cardiomyopathy had improved from class III to class II status, and his quality of life had significantly improved.

After 3 years of that therapy, 40 mg of Mevacor daily was added to the patient’s treatment regimen. Six months later, the patient’s NYHA classification status had

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steadily deteriorated from class II to almost class IV, which is life-threatening. During this decline, the patient exhibited clinical decompensation and chest pain and required surgical revision of one graft.

According to a review of the documentation of this patient’s clinical deterioration, his coenzyme Q10 blood level was 2.52 µg/mL when treatment with Mevacor was initiated. Approximately 6 months later, his blood coenzyme Q10 level had diminished to 1.15 µg/mL and further decreased to the very low level of 0.64 µg/mL after 5 subsequent months, at which time the ejection fraction had diminished to 54%. During the patient’s surgical revision and recovery, the oral administration coenzyme Q10 was not feasible for 3 weeks, but after that 3-week period, administration of coenzyme Q10 was resumed at 166 mg per day. One month after the reinitiation of therapy with coenzyme Q10, the patient’s blood level of coenzyme Q10 had increased to 1.39 µg/mL and stabilized at 1.55 µg/mL 2 months later (at which time the administration of Mevacor was reduced from 40 mg/day to 20 mg/day) and then to 1.66 µg/mL after 5 subsequent months. The reduction of the dosage of Mevacor from 40 mg to 20 mg daily and an increase in the dosage of coenzyme Q10 resulted in the cardiac stabilization of this patient with acceptable blood levels of coenzyme Q10 and ejection fractions. Clearly, the administration of MEVACOR over time significantly reduced the blood level of coenzyme Q10 and reduced the pumping of blood by the heart as monitored by the ejection fraction.

Case Study 2

A 46-year-old white man with dilated cardiomyopathy was classified as class III according to the New York Heart Association classification of functional capacity. His baseline blood level of coenzyme Q10 was 0.78 µg/mL and his control ejection fraction was 62% at the time of assessment. The patient’s record indicated that his blood level of coenzyme Q10 increased to the range of 1.79 to 2.31 µg/mL after treatment with 100 mg of coenzyme Q10, and his ejection fraction increased to the range of 68% to 71%. During that period of 2 years and 4 months, his cardiac function and his quality of life had improved from the NYHA class III to class I status, and his cardiac function stabilized at a clinically reasonable level.

Six months after that time, treatment with 20 mg daily of Mevacor was initiated, and during the subsequent 18 months, the patient’s coenzyme Q10 blood level had steadily declined (2.29 µg/mL, 1.82 µg/mL, 1.50 µg/mL, 1.12 µg/mL). At that time, therapy with Mevacor was terminated, and 5 months later, the patient’s coenzyme Q10 blood level had increased to 1.87 µg/mL. These data indicate that treatment with Mevacor reduced the blood level of coenzyme Q10 over time and that the effect reversed when therapy with Mevacor was terminated.

References


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