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Muscle coenzyme Q10 level in statin-related myopathy.

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Abstract

BACKGROUND: Statin drugs (3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors) reduce the level of cholesterol by inhibiting the synthesis of mevalonate, an intermediary in the cholesterol biosynthetic pathway. Use of statin drugs has been associated with a variety of skeletal muscle-related complaints. Coenzyme Q10 (CoQ10), a component of the mitochondrial respiratory chain, is also synthesized from mevalonate, and decreased muscle CoQ10 concentration may have a role in the pathogenesis of statin drug-related myopathy.

OBJECTIVES: To measure the CoQ10 concentration and respiratory chain enzyme activities in muscle biopsy specimens from 18 patients with statin drug-related myopathy and to look for evidence of apoptosis using the TUNEL (terminal deoxynucleotidyl transferase-mediated deoxyuridine triphosphate nick-end labeling) assay.

DESIGN: An open-labeled study of CoQ10 concentration in muscle from patients with increased serum creatine kinase concentrations while receiving standard statin drug therapy.

SETTING: Neuromuscular centers at 2 academic tertiary care hospitals.

RESULTS: Muscle structure was essentially normal in 14 patients and showed evidence of mitochondrial dysfunction and nonspecific myopathic changes in 2 patients each. Muscle CoQ10 concentration was not statistically different between patients and control subjects, but it was more than 2 SDs below the normal mean in 3 patients and more than 1 SD below normal in 7 patients. There was no TUNEL positivity in any patients.

CONCLUSION: These data suggest that statin drug-related myopathy is associated with a mild decrease in muscle CoQ10 concentration, which does not cause histochemical or biochemical evidence of mitochondrial myopathy or morphologic evidence of apoptosis in most patients.

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Publication Types, MeSH Terms, Substances, Grant Support

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