Abstract

J Pediatr. 2010 Sep 29. [Epub ahead of print]

Rosuvastatin Lowers Coenzyme Q10 Levels, but not Mitochondrial Adenosine Triphosphate Synthesis, in Children with Familial Hypercholesterolemia.

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OBJECTIVE: To investigate whether statin therapy affects coenzyme Q10 (CoQ10) status in children with heterozygous familial hypercholesterolemia (FH).

STUDY DESIGN: Samples were obtained at baseline (treatment naïve) and after dose titration with rosuvastatin, aiming for a low-density lipoprotein cholesterol level of 110 mg/dL. Twenty-nine patients were treated with 5, 10, or 20 mg of rosuvastatin for a mean period of 29 weeks.

RESULTS: We found a significant (32%) decrease in peripheral blood mononuclear cell (PBMC) CoQ10 level (P = .02), but no change in PBMC adenosine triphosphate synthesis (P = .60). Uncorrected plasma CoQ10 values were decreased significantly, by 45% (P < .01). In contrast, ratios of plasma CoQ10/total cholesterol and CoQ10/low-density lipoprotein cholesterol remained equal during treatment.

CONCLUSIONS: In children with FH, rosuvastatin causes a significant decrease in cellular PBMC CoQ10 status but does not affect mitochondrial adenosine triphosphate synthesis in children with FH. Further studies should address whether (rare) side effects of statin therapy could be explained by a deterioration in CoQ10 status.

PMID: 20884007