Effects of Coenzyme Q10 in Early Parkinson Disease: Evidence of Slowing of the Functional Decline

Archives of Neurology

October 2002; Vol. 59; pp.1541-1550

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FROM ABSTRACT:

Parkinson disease (PD) is a degenerative neurological disorder for which no treatment has been shown to slow the progression. The objective of this study was to determine if coenzyme Q10 supplementation could slow the functional decline in PD.

This is a randomized, placebo-controlled, double-blind trial that used 80 subjects with early PD, randomly assignment to placebo or co-enzyme Q10 at dosages of 300, 600, or 1200 mg/d.

All subjects underwent evaluation with the Unified Parkinson Disease Rating Scale (UPDRS) at the screening, baseline, and 1-, 4-, 8-, 12-, and 16-month visits. They were followed up for 16 months. The primary response variable was the change in the total score on the UPDRS from baseline to the last visit.

Results: [+ indicates an increased UPDRS score, indicating a worsening of their PD]: The adjusted mean total UPDRS changes were [rounded]:

+12.0	for the placebo group
+ 8.8	for the 300-mg/d group
+10.8	for the 600-mg/d group
+ 6.7	for the 1200-mg/d group

The difference between the 1200-mg/d and placebo groups was significant.

Conclusions: Coenzyme Q10 was safe and well tolerated at dosages of up to 1200 mg/d.

Coenzyme Q10 reduces the progression of PD disability and the benefit was greatest in subjects receiving the highest dosage [1200 mg/d].

Coenzyme Q10 slows the progressive deterioration of function in PD.

KEY POINTS FROM THIS STUDY:

1) "Parkinson's Disease (PD) is a degenerative neurological disorder that is characterized by resting tremor, slowness of movement, and muscular rigidity."

2) The cardinal pathological feature of PD is the loss of dopaminergic neurons in the substantia nigra pars compacta of the basal ganglia.

3) The causes of PD are both genetic and environmental; problems in the mitochondrial electron transport chain are documented and accepted.

4) There is evidence that a systemic insult to the mitochondria could preferentially injure nigral dopaminergic neurons leading to PD.

5) Coenzyme Q10 is the electron acceptor for mitochondrial proteins and also a potent antioxidant.

6) Evidence shows that PD patients have significantly lower levels of coenzyme Q10 in the mitochondria.

7) Oral supplementation with coenzyme Q10 has been shown to reduce the loss of dopamine and dopaminergic axons in the basal ganglia, and to significantly increase the concentration of coenzyme Q10 in mitochondria.

8) The coenzyme Q10 was taken 4 times each day, with breakfast, lunch, dinner and at bedtime. The wafers with active study drug contained 300 mg of coenzyme Q10 and 300 IU of vitamin E as a lipophilic carrier [Coenzyme Q10 is lipid absorbed]. Matching placebo wafers also contained 300 IU of vitamin E each.
At each visit, the subject was assessed for occupation performance, gait, balance, finances, domestic responsibility, and activities of daily living.

9) "Coenzyme Q10 was well tolerated; no dosage reductions were needed in any of the treatment groups."

10) The Unified Parkinson's Disease Rating Scale (UPDRS) scores have 3 parts: mental function; activities of daily living; motor function.
Reduced disability progression was noted with coenzyme Q10 supplementation of every dose and in all three measured parts.

11) "All groups receiving coenzyme Q10 had highly significant increases in the mean plasma level of coenzyme Q10 from baseline to the last visit."

12) "Our dosage-ranging study found that coenzyme Q10 was safe and well tolerated at the dosages of 300 to 1200 mg/d and that the 1200-mg/d dosage was associated with significant slowing of the worsening of PD."

13) "Our data are consistent with the hypothesis that mitochondrial dysfunction plays a role in the pathogenesis of PD and that treatments targeted at mitochondria might ameliorate the functional decline in PD."

14) "There have been numerous reports of the benefits of co-enzyme Q10 in patients with heart disease."

15) The dosage of coenzyme Q10 may be crucial in achieving favorable clinical outcomes:

- In this study the greatest benefit was found at a dosage of 1200 mg/d.
- In a study of Huntington disease the greatest benefit was found at a dosage of 600 mg/d [the highest dose used].
- In a congestive heart failure study, no benefit was seen at a dosage of 200 mg/d. (therefore the authors hypothesize that higher doses should be assessed).

16) "The mechanism(s) through which coenzyme Q10 exerted its beneficial effect cannot be determined from our clinical trial, but our data are consistent with an effect on mitochondrial function."

17) This data suggests that in treatment of neurological disorders such as PD and Huntington disease, dosages of coenzyme Q10 much higher than those previously used may be required.

18) "The benefit was greatest in the group receiving the highest dosage, 1200 mg/d. It is conceivable that a greater effect could be seen at even higher dosages of coenzyme Q10."

19) In this study, "coenzyme Q10 treatment at high dosages was safe and well tolerated and reduced the worsening of PD, as reflected in the total UPDRS score."

COMMENT FROM DAN MURPHY:

In looking at the data, supplementation with 1200 mg/d of coenzyme Q10 reduced the progression of Parkinson's disease by nearly half.