The effect of ubiquinone in diabetic polyneuropathy: a randomized double-blind placebo-controlled study.


Abstract

INTRODUCTION: Diabetic polyneuropathy aetiology is based on oxidative stress generation due to production of reactive oxygen species. Ubiquinone is reduced to ubiquinol and redistributed into lipoproteins, possibly to protect them from oxidation.

AIMS: To evaluate the impact of oral ubiquinone in diabetic polyneuropathy, and the role of lipid peroxidation (LPO) and nerve growth factor (NGF-β).

METHODS: We conducted a double-blind, placebo-controlled clinical trial, patients were randomized to ubiquinone (400 mg) or placebo daily for 12 weeks. Main outcomes were clinical scores, nerve conduction studies, LPO, NGF-β and safety.

RESULTS: Twenty four patients on experimental group and twenty five on control group met the inclusion criteria (mean age 56 years, 22% male and 78% female, mean evolution of type 2 diabetes mellitus 10.7 years). Significant improvement on experimental vs control group was found in neuropathy symptoms score (from 2.5 ± 0.7 to 1 ± 0.8, p<0.001), neuropathy impairment score (5.5 ± 4 to 3.1 ± 2.6, p<0.001), sural sensory nerve amplitude (13.0 ± 6.1 to 15.8 ± 5.1 μV, p=0.049), peroneal motor nerve conduction velocity (39.7 ± 5.0 to 47.8 ± 4.9 m/s, p=0.047), and ulnar motor nerve conduction velocity (48.8 ± 6.8 to 54.5 ± 6.1 m/s, p=0.046). There was a significant reduction of LPO in subjects treated with ubiquinone vs placebo (16.7 ± 8.6 and 23.2 ± 15.8 nmol/mL, respectively) with p<0.05, and NGF-β did not change (control 66.5 ± 26.7 vs. experimental 66.8 ± 28.4 pg/mL, p=0.856). No drug-related adverse reactions were reported.

CONCLUSIONS: Twelve weeks treatment with ubiquinone improves clinical outcomes and nerve conduction parameters of diabetic polyneuropathy; furthermore, it reduces oxidative stress without significant adverse events.

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