L-Carnitine

Forty patients with severe traumatic brain injury were randomized into 2 groups. The I-carnitine (LCA-) group received standard treatment with placebo while the (LCA+) group received I-Carnitine 2g/day for one week. Neuron specific enolase (NSE) was measured on days 1, 3 and 7 after the initiation of the study. Neurocognitive and neurobehavioral disorders were recorded on the first and third months.

Neurocognitive function and NSE significantly improved within one week in both groups. Patient mortality was similar in LCA+ and LCA- groups (P value: 0.76). Brain edema was present in 7 patients in LCA+ group and 13 patients in LCA-group (P value: 0.044). While there was no difference in NSE levels between the two groups. Neurological function was preserved in the LCA+ group with an exception of attention deficit, which was frequent in the LCA+ group.

Mahmoodpoor et al. concluded that despite improvements in neurobehavioral function and the degree of cerebral edema, 7-days of treatment with I-Carnitine failed to reduce serum NSE levels or improve mortality rate at 90days in patients with TBI¹⁾.

There is evidence in the literature for mitochondrial dysfunction in Parkinson's disease as well as fatty acid beta-oxidation, involving l-carnitine.

Gill et al. investigated I-carnitine in the context of microglial activation, suggesting a potential new strategy of supplementation for PD patients. Preliminary results from this studies suggest that the treatment of activated microglia with the endogenous antioxidant I-carnitine can reverse the effects of detrimental neuroinflammation in vitro²⁾.

1)

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Gill EL, Raman S, Yost RA, Garrett TJ, Vedam-Mai V. I-Carnitine Inhibits Lipopolysaccharide-Induced Nitric Oxide Production of SIM-A9 Microglia Cells. ACS Chem Neurosci. 2018 Jan 31. doi: 10.1021/acschemneuro.7b00468. [Epub ahead of print] PubMed PMID: 29370524.

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