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Oral L-carnitine supplementation increases trimethylamine-N-oxide but reduces markers of vascular injury in hemodialysis patients.

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Abstract

OBJECTIVES: Food or supplement-derived L-carnitine is changed to trimethylamine (TMA) by interstinal microbiota, which is further metabolized to trimethylamine-N-oxide (TMAO), being involved in the promotion of atherosclerosis in animal models. Meanwhile, carnitine deficiency has played a role in accelerated atherosclerosis in hemodialysis (HD) patients. However, effects of oral L-carnitine supplementation on circulating levels of TMAO and markers of vascular injury and oxidative stress in patients on HD remain unclear. In this study, we addressed the issue.

METHODS: Thirty-one HD patients with carnitine deficiency were treated with oral L-carnitine (900 mg/d) for 6 months. At baseline and after treatment, clinical variables including circulating levels of carnitine fractions, TMA, TMAO, advanced glycation end products (AGE), soluble forms of intracellular adhesion molecule-1 (sICAM-1), vascular cell adhesion molecule-1 (sVCAM-1), and malondialdehyde (MDA) were measured.

RESULTS: Oral L-carnitine supplementation significantly increased total, free, acyl carnitine, and plasma TMA and TMAO levels, whereas it decreased markers of vascular injury and oxidative stress such as sICAM-1, sVCAM-1, and MDA levels. TMA and TMAO levels at baseline were correlated with each other, and free carnitine was independently associated with TMAO levels. Furthermore, change in AGE values from baseline ([INCREMENT]AGE) was positively correlated with [INCREMENT]sICAM-1 ($P = 0.043$) and was a sole independent

determinant of [INCREMENT]sICAM-1 (R = 0.133, P = 0.043).

CONCLUSIONS: This study demonstrated that although oral L-carnitine supplementation was associated with increased TMAO levels, it might be beneficial on vascular injury in patients on HD. Vasculoprotective properties of L-carnitine supplementation in HD patients might be ascribed partly to its inhibitory actions on AGE.

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