

Effect of vitamin E on serum aminotransferase and thioredoxin levels in patients with viral hepatitis C.

Mahmood S, Yamada G, Niiyama G, Kawanaka M, Togawa K, Sho M, Ito T, Sasagawa T, Okita M, Nakamura H, Yodoi J.

Department of Internal Medicine, Center for Liver Diseases, Kawasaki Hospital, Kawasaki Medical School, Okayama, Japan.

OBJECTIVES: Oxidative stress induces cellular responses such as cell death, gene activation and cell proliferation, in the liver. Vitamin E (Vit. E) has been found to protect the liver against oxidative stress in animal experiments. Thioredoxin (TRX) is a stress inducible, multifunctional protein, secreted during oxidative stress. This study evaluated effects of Vit. E on serum TRX and aminotransferase levels in hepatitis C virus (HCV) patients, partly non-responsive to initial interferon (IFN), with higher than average level of serum alanine aminotransferase (ALT) after receiving anti-inflammatory drug treatment. **METHODS:** Seventeen HCV patients (male = 3; female = 14) of age 62 +/- 7.65 years receiving anti-inflammatory drug therapy, at least 6 months prior to Vit. E administration, were given d-alpha-tocopherol 500 mg/day, orally, for a period of 3 months. ALT, aspartate aminotransferase (AST), TRX and Vit. E were measured at 0, 1, 2 and 3 months and 1 month after end of treatment. As controls, the same patients biochemical data, 3 months from the start of therapy were used. Patients were divided into three categories: total patients "T", low ALT group "L" (ALT < 70 IU/l) and high ALT group "H" (ALT > 70 IU/l), respectively. **RESULTS:** The ALT level was lowered, significantly in group H, in the 1st, 2nd, 3rd and 1-month post therapy, compared to the initial value. But group L showed little or no change in ALT. Post Vit. E therapy, in groups T and H, the TRX level was elevated but remained below initial levels, whereas in group L, TRX level remained significantly lower than the pretreatment value. Groups T and L, showed significant reduction ($p < 0.05$) in serum TRX levels in the 2nd and 3rd month. Group H showed a tendency towards TRX reduction, but not significantly. Serum Vit. E levels increased significantly ($p < 0.0001$) from the 1st to 3rd month in all three T, H and L groups. **CONCLUSION:** Oxidative stress induced liver damage is reduced by Vit. E in patients with viral hepatitis C, particularly those with initial ALT levels > 70 IU/l. Vit. E treatment causes reduction of oxidative stress markers as TRX and ALT in sera. Therefore, Vit. E can act as a supportive therapy to combat liver damage caused by oxidative stress, in such patients with continuously high levels of ALT even after anti-viral and anti-inflammatory drug therapy.