RESEARCH LETTERS

Serum Level of Antioxidant Taurine in β-Thalassaemia Subjects: Possible Protection Against Thalassaemia Complications

The thalassaemias are hereditary anaemias that occur because of mutations in the globin genes that affect the rate of haemoglobin synthesis [1]. Oxidative stress, which has been a topic of considerable investigation, is implicated in a growing list of diseases to include the thalassaemia [2]. The antioxidant taurine has been considered among the very useful antioxidants that showed striking effects both therapeutically and protectively in different diseases [3,4]. Taurine was not considered in thalassaemias, and no data was found in the literature about the determination or use of taurine in thalassaemias. Therefore, from the present work we aimed at the evaluation of the serum level of taurine in β-thalassaemia subjects, and also evaluating the effect of oral taurine on lipid profile, liver functions, and coagulation time in β-thalassaemia carriers.

Serum taurine was measured by HPLC in 14 β-thalassaemia majors, 22 β-thalassaemia carriers, and 17 normal subjects. In addition, oral taurine (1000 mg/day) was supplemented to the carriers for 8 weeks.

Results indicated that both patients and carriers for β-thalassaemia have significantly low serum taurine levels compared to normal subjects ($p < 0.05$). The reduction of serum taurine was more pronounced in patients than in carriers compared to normal controls the values of which were: $43.23 \pm 2.45$, $49.86 \pm 3.76$, $56.55 \pm 4.60$ μmol/l respectively.

Although taurine supplementation (1000 mg/day) for 8 weeks did not result in any significant changes in the lipid profile of β-thalassaemia carriers, however, it resulted in a significant amelioration in the activity of liver enzymes (AST & ALT) which showed an upper-limit baseline values, the effect of taurine supplementation on ALT was more pronounced than the effect on AST. Moreover, the results regarding the coagulation time in
β-thalassaemia carriers revealed a hypercoagulable state with a baseline coagulation time (301.32 ± 44.66 sec) significantly lower than normal controls (337.40 ± 47.68 sec). Interestingly, oral taurine improved the coagulation time in the carriers, improvements were reported as early as the 4th week of supplementation (307.59 ± 39.64 sec) and continued also at the 8th week (325.5 ± 35.11 sec).

In the present work, clinical trial of taurine supplementation on β-thalassaemia majors was not possible, because those patients are under the management protocol provided by the haematology department at the Palestinian Ministry of Health. Therefore, we evaluated the patients biochemically and compared their parameters with carriers and normal controls. The correlation and linear regression analysis revealed a significant correlation between the reduction in serum taurine and the degree of iron overload in those patients ($r^2 = 0.60, p < 0.001$) and the complications of in β-thalassaemia disease as reported by the increased activities of AST & ALT ($r^2 = 0.63$ & $0.61$ respectively) and the hypercoagulable state ($r^2 = 0.65$) which showed a coagulation time of $271.29 ± 23.37$ sec. It was concluded that β-thalassaemia disorder is associated with a depleted levels of serum taurine, which justifies the necessity for clinical evaluation of oral taurine in β-thalassaemic patients which may ameliorate the complications specially for those with improper management and chelation therapy.

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References

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