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Homocysteine, hypothyroidism, and effect of thyroid hormone replacement

B Catargi [1], F Parrot-Roulaud, C Cochet, D Ducassou, P Roger, A Tabarin

Affiliations expand

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Abstract

Elevation of total plasma concentration of homocysteine (t-Hcy) is an important and independent risk factor for cardiovascular disease. Hypothyroidism is possibly also associated with an increased risk for coronary artery disease, which may be related to atherogenic changes in lipid profile. Because hypothyroidism decreases hepatic levels of enzymes involved in the remethylation pathway of homocysteine, we prospectively evaluated fasting and postload t-Hcy in patients before and after recovery of euthyroidism. Fasting and postload t-Hcy levels were higher in 40 patients with peripheral hypothyroidism (14 with autoimmune thyroiditis and 26 treated for thyroid cancer) in comparison with those of 26 controls (13.0 +/- 7.5 vs. 8.5 +/- 2.6 micromol/L, p < .01, respectively, and 49.9 +/- 37.3 vs. 29.6 +/- 8.4 micromol/L p < .001, respectively). On univariate analysis, fasting Hcy was positively related to thyrotropin (TSH) and inversely related to folates. Multivariate analysis confirmed TSH as the strongest predictor of t-Hcy independent of age, folate, vitamin B12, and creatinine. Thyroid hormone replacement significantly decreased fasting but not postload t-Hcy. We conclude that t-Hcy is elevated in hypothyroidism. The association of hyperhomocysteinemia and lipid abnormalities occurring in hypothyroidism may represent a dynamic atherogenic state. Thyroid hormone failed to completely normalize t-Hcy. Potential benefit of treatment with folic acid in combination with thyroid hormone replacement has to be tested given that hypothyroid patients were found to have lower levels of folate.

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