Blood biochemical parameters in subclinical hyperthyroidism and Graves disease: possible increased cardiac risk in suppressed TSH patients

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Thyroid hormones play a central role in differentiation, development and energy metabolism. In hyperthyroidism, due to accelerated oxidative metabolism in the mitochondria, there is an augmented production of reactive oxygen species, leading to tissue and systemic oxidative stress. In this work, the presence of systemic oxidative stress was evaluated in human hyperthyroidism and subclinical hyperthyroidism. The subclinical hyperthyroid group (SHT) is characterized by reduced serum thyrotropin (TSH) levels, while free thyroxine (FT_4) and free triiodothyronine (FT_3) estimates are within the reference range. Subjects usually present no obvious symptoms of hyperthyroidism and the condition is commonly observed in patients given TSH-suppressive therapy with thyroid hormones as part of the treatment following thyroid cancer removal. The hyperthyroid group (HT) was selected among Graves disease patients, an organ-specific autoimmune disease caused by an immunological abnormality. Comparisons were carried out against a control group of euthyroid volunteers. Several blood biochemical parameters were measured, such as haemoglobin, blood cell count, AST, ALT, total cholesterol and fractions, triglycerides, blood glucose and reactive C protein (CRP), as well as levels of plasmatic thiobarbituric reactant substances (TBARS) and antioxidant vitamins (alpha-tocopherol, beta-carotene, lycopene, coenzyme Q10, ascorbic acid, and uric acid). Results show that in SHT group there was an increase in TBARS levels and in TBARS/α-tocopherol ratio; biochemical exams showed an increase in glycemia and CRP levels. In the HT group, results presented a decrease in α-tocopherol levels as well as an increase in glycemia and a discrete increase in PCR, taking it within the limits of cardiac risk. These alterations, associated to an insulinic resistance of hyperglycemic effects and a possible endothelial dysfunction, may be relevant in the planning of TSH suppressive therapy in thyroid cancer patients. Financial support: FAPESP, CAPES, CNPq.