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[The influence of thyroid hormones on homocysteine and atherosclerotic vascular disease].

[Article in Polish]

Orzechowska-Pawiłojć A, Lewczuk A, Sworczak K.

Source

Department of Internal Medicine, Endocrinology and Haemostatic Disorders, Medical University of Gdansk, Poland.
ksworczak@poczta.onet.pl

Abstract

Several reports have appeared in the literature proving that hypothyroidism is associated with increased risk for cardiovascular disease, especially coronary heart disease. This increased risk for premature atherosclerosis is supported by autopsy and epidemiological studies in patients with thyroid hormone deficiency. Hypothyroid patients have increased diastolic blood pressure (as a result of increased systemic vascular resistance), altered lipid profile (elevated levels of total cholesterol, LDL-cholesterol and apolipoprotein B). More recently homocysteine, C-reactive protein, increased arterial stiffness, endothelial dysfunction and altered coagulation parameters have been recognized as a "new" risk factors for atherosclerosis in patients with thyroid hormone deficiency. The plasma total homocysteine concentration, an independent risk factor for atherosclerosis, is moderately elevated in overtly hypothyroid patients and it decreases with thyroid replacement therapy. Several experimental study have shown that hypothyroidism affects folate metabolism and the enzymes involved in the remethylation pathway of homocysteine (particularly 5,10-methylenotetrahydrofolate reductase - MTHFR). In hypothyroid condition the hepatic activity of flavoenzyme - MTHFR, is decreased. Thyroid hormone may affect the availability of FMN and FAD - necessary for stabilizing MTHFR. An impairment of enzyme involved in transsulfuration pathway is suggested. The increased serum creatinine level in hypothyroidism probably reflects a reduced glomerular filtration rate, which is linked to impaired renal homocysteine clearance and hyperhomocysteinemia.

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