Interaction of plasma homocysteine and thyroid hormone concentrations in the pathogenesis of the slow coronary flow phenomenon.

Department of Cardiology, Pamukkale University Faculty of Medicine, Denizli, Turkey.

BACKGROUND AND OBJECTIVE: The slow coronary flow (SCF) phenomenon is an angiographic observation and a well-recognized clinical entity characterized by delayed opacification of vessels in a normal coronary angiogram due to reasons yet unclear. Thyroid hormones exert significant effects on plasma homocysteine (Hcy) levels and microvascular resistance. Recently, several investigators have consistently reported that elevation of the plasma Hcy level can severely disturb vascular endothelial function and play a role in the pathogenesis of SCF. Accordingly, we investigated the levels of plasma Hcy and thyroid hormones and their relationship in patients with SCF.

METHOD: Forty-four patients with angiographically proven SCF (Group I) (mean age 55.5 +/- 10.4 years, 26 males) and 44 cases with normal coronary flow (NCF) pattern (Group II) (mean age 53.9 +/- 11 years, 22 males) with similar risk profiles were enrolled in the study. Coronary flow patterns of the cases were determined by the thrombolysis in myocardial infarction (TIMI) frame count method. The coronary TIMI frame counts were calculated separately for each coronary artery and their average was determined as the mean TIMI frame count for each subject. Serum levels of free tri-iodothyronine (fT3), free thyroxine (fT4), thyroid stimulating hormone (TSH) and Hcy were measured. Patients with thyroid disease or on medications with a potential to affect thyroid functions were excluded.

RESULTS: There were no statistically significant differences between the groups concerning the demographic characteristics and major cardiovascular risk factors. Mean TIMI frame counts of SCF and NCF groups were 45.9 +/- 12 and 23.3 +/- 3.7, respectively. fT4 (ng/dl) and TSH (microU/ml) levels of the two groups were similar (p > 0.05). The level of fT3, the active metabolite of the thyroid hormone family, was dramatically reduced in the SCF group when compared to the NCF group (2.3 +/- 0.2 vs. 3.0 +/- 0.3, p = 0.0001, respectively). Plasma Hcy levels of patients with SCF were found to be significantly higher than controls (12.2 +/- 4.9 vs. 8.5 +/- 2.9, p = 0.0001, respectively). Correlation analysis showed a significant negative correlation between the plasma fT3 and Hcy levels and the mean TIMI frame counts (r = -0.31, p = 0.003 vs. r = -0.66, p = 0.0001). Moreover, there was a significant positive correlation between the plasma Hcy levels and the mean TIMI frame counts (r = 0.58, p = 0.0001). Also, fT3 was the only significant determinant of the variance of Hcy in multiple regression analysis (r = -0.30, p = 0.005).

CONCLUSION: fT3 levels were decreased and plasma Hcy levels were increased significantly in patients with SCF as compared to controls. This finding suggests that thyroid hormones and/or a possible disturbance in their metabolism may be responsible for the elevated levels of plasma Hcy in patients with SCF and may play a role in the pathogenesis of the SCF phenomenon.

PMID: 17085937