The effect of hereditary thrombophilia on the formation of carotid artery disease: a pilot study

Introduction: Carotid artery disease (CAD) is the narrowing of carotid arteries due to atherosclerosis. It can cause stroke. Some hereditary determinants can affect atherosclerosis formation. In present study, we investigated the hereditary thrombophilia on the formation of CAD.

Patients and Methods: We evaluated the effects of Factor V LEIDEN, Factor V H1299R, Prothrombin G20210A, Factor XIII V34L, B-Fibrinogen -455 G>A, PAI-1 4G/5G, HFA1, MTHFR C677T, MTHFR A1298C, ACE I/D, APO B R3500Q, and APO E polymorphisms on CAD formation by using a ViennaLab CVD Strip Assay. Group A includes 41 patients (70.2 ± 8.6 years, 30 men) with CAD and Group B includes 39 healthy controls (67.3 ± 9.2 years, 28 men). Twenty patients had transient ischemic attack or stroke, 21 had carotid artery stenosis, more than 50 % in Group A. Hyperlipidemia is more frequent in Group A compared Group B (71%, 49%; p<0.05). Hypertension, smoking habit and diabetes mellitus are similar in both groups.

Results and Conclusion: Heterozygote form of Factor V H1299R, Factor XIII V34L, B-Fibrinogen -455 G>A, MTHFR C677T and MTHFR A1298C were more frequent in Group A compared with Group B significantly [(2.6%, 7.3% p<0.05), (12.8%, 19.5% p<0.05), (12.8%, 19.5% p<0.05), (20.5%, 34.1% p<0.05), (25.6%, 46.3% p<0.05)]. On the formation of CAD, Factor V H1299R, Factor XIII V34L, B-Fibrinogen -455 G>A, MTHFR C677T and MTHFR A1298C heterozygous mutation seems to be determinant (p<0.05). We have some difficulty on the explication that why heterozygous form is significant even though homozygous form is not significant. This is a pilot study. We will go on working on the project to evaluate the hereditary thrombophilia.