CORRELATION BETWEEN CAROTID ATHEROSCLEROTIC DISEASE AND MULTI-VESSELS CORONARY DISEASE IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Introduction: Many studies about Atherosclerosis have documented that carotid plaques are predictive of coronary involvement.

Objective: To value the possible correlation between the extension of atherosclerotic disease in the carotid and coronary district.

Materials and Methods: Between January 2006 and May 2009, 191 patients with Acute Coronary Syndrome were underwent in-hospital coronary study and Eco-Color-Doppler of the epiaortic vessels. The pts positive to the Doppler were divided in two groups. Group A, 32 pts with only one carotid artery affected, and Group B, 116 pts with both carotid arteries affected.

Results: Among 191 pts underwent Eco-Color-Doppler of the epiaortic vessels, in 148 pts (77%) it was found a carotid artery involvement. In Group A 87.5% of pts have had a one- vessel coronary disease; 9.3% two-vessels disease and 3.1% three-vessels disease. In Group B 8.6% of pts have had an one-vessel disease; 34.4% two-vessels disease; 56.8% three-vessels disease.

Conclusions: The study have documented that greater is the number of carotid artery involved in the atherosclerotic disease and greater is the probability to find a coronary multi-vessels disease. Use of Carotid ultrasound improves the stratification of cardiovascular risk and predict presence and extent of coronary artery disease.

INSULIN RESISTANCE IS ASSOCIATED WITH METABOLIC SYNDROME BUT NOT WITH ANGIOGRAPHICALLY DETERMINED CORONARY ARTERY DISEASE

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Objective: Insulin resistance (IR) is the key feature of the metabolic syndrome (MetS) and in prospective studies predicts atherothrombotic events. Its association with directly visualised coronary atherosclerosis is unclear. We hypothesised that IR is associated with both angiographically determined coronary artery disease (CAD) and with the MetS.

Methods: We enrolled 986 consecutive patients undergoing coronary angiography for the evaluation of suspected or established stable CAD; significant CAD was diagnosed in the presence of significant coronarystenoses with lumens narrowing >50%. IR was determined by the HOMA index; the MetS was defined according to ATP III criteria.

Results: HOMA IR scores were significantly higher in MetS patients than in subjects without the MetS (6.4±2.1 vs. 2.2±2.0; p < 0.001). In contrast HOMA-IR did not differ significantly between patients with significant CAD and those who did not have significant CAD (3.9±1.4 vs. 3.2±1.4; p = 0.490). When both, the presence of MetS and of significant CAD were considered, HOMA-IR was significantly higher in patients with the MetS both among those who had significant CAD (7.2±2.8 vs. 2.3±1.4; p < 0.001) and among those who did not have significant CAD (5.3±5.7 vs. 2.1±1.4; p < 0.001) whereas it did not differ significantly between patients with significant CAD and subjects without significant CAD in patients with the MetS (7.2±2.8 vs. 5.3±5.7; p = 0.679) nor in those without MetS (2.1±1.4 vs. 2.3±1.2; p = 0.411). Similar results were obtained with the IDF definition of the metabolic syndrome.

Conclusion: IR is significantly associated with the MetS but not with angiographically determined coronary atherosclerosis.

ENDOTHELIAL PROGENITORS CELLS AND MICROPARTICLES IN HIV-INFECTED PATIENTS


Background: Nowadays the HIV infection is recognized as an inflammatory disease and can lead to endothelial dysfunction. Endothelial injury plays a critical role in coronary artery disease, but the assessment of this injury has been problematical. Recently, it has been shown that endothelial cells (EC) release microparticles (MP) on activation or apoptosis and that and evaluation of MP can provide useful information on EC status in patients with an increase cardiovascular risk.

Methods: We studied endothelial progenitors (EPC) cells and MP in HIV-infected naive patients and compared with HIV negative controls. Standard laboratory study included: lipid profile, glycaemia, C-reactive protein and apolipoprotein B. The EPC cells and MP were measure by flow citometry. The EPC were identified using the following markers: CD34+, KDR for definition and CD133+ for immature lineage cells. The MP were characterized with CD31+, CD42+ and CD51+.

Results: Thirty patients were included, 15 in each group. 73.3% were male with mean age 30.9 years. The lipid profile was significant only in HDL-c and LDL-c between the groups. In the HIV-infected group we observed 0.01% of CD34+/KDR+ and it was not isolated CD34+/CD133+ neither CD34+/KDR+. In this group it was also observed more release of MP CD51+ and CD131+/CD42+ comparing to the control group and CD31+ for the group of patients with AIDS were found.

Conclusion: Our results suggest a possible imbalance between EPC and MP. This new finding in HIV-naive patient may be associated with increased cardiovascular risk in the long term follow-up, and can be aggravated after antiretroviral therapy.

B2-MICROGLOBULIN, A NOVEL BIOMARKER OF PERIPHERAL ARTERIAL DISEASE, IS INDEPENDENTLY ASSOCIATED WITH AORTIC STIFFNESS IN THESE PATIENTS


Objective: Arterial stiffness is a prominent feature of vascular ageing and strongly predicts cardiovascular and total mortality. The [β2-microglobulin, (β2M)] a newly identified biomarker of peripheral arterial disease (PAD), is related to renal insufficiency, inflammatory and neoplastic diseases, but may also play a role in vascular dysfunction. However, the relationship between arterial stiffness and [β2M] has not been previously studied in patients with atherosclerosis. In the present study we examined possible association between [β2M] and arterial stiffness in patients with PAD and in healthy subjects.

Methods: Plasma [β2M] levels and parameters of arterial stiffness such as aortic pulse wave velocity (aPWV) and augmentation index (AIx) and were measured in 66 patients with PAD and in 66 apparently healthy subjects.

Results: Plasma levels of [β2M], aPWV and AIx were significantly increased in patients with PAD compared with controls (1858.1±472.8 vs 1554.5±297.0 m/s; P < 0.001; 9.9±2.2 m/s vs 7.6±1.6 m/s; P < 0.001; 28.9±14.1±11%; P < 0.001; respectively). There existed significant correlation between aPWV and [β2M] for the patient group (R = 0.47; P < 0.001), but not for the controls (R = 0.14; P = 0.26). In multivariate analysis, [β2M] remained independently associated with aPWV, fetuin-A, age and glomerular filtration rate in patients (R²=0.35, P < 0.001). We found no relationship between [β2M] and AIx in either group.

Conclusion: We demonstrated that among patients with PAD elevated plasma [β2M] levels were associated with higher aortic stiffness irrespective of distending pressure and cardiovascular disease risk factors. These data suggest that [β2M] may be involved in the pathogenesis of aortic stiffness in atherosclerosis.

DYSLIPIDEMIA AND CARdiovascular Risk In Northern Greece. ATHOS CARDIO GREECE STUDY

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Objective: To study the prevalence of dyslipidemia and its relation to cardiovascular risk in a Northern Greek population sample.

Methods: 3000 subjects (54% males and 46% females) with mean age 62±13 years from Northern Greece were examined during an epidemiological project the last three years. All subjects gave informed consent form and completed a questionnaire on personal and family medical history. Demographic/anthropometric characteristics and blood pressure were recorded. Biochemical parameters (total cholesterol, HDL, LDL, triglycerides) were measured by the Cholestech kit (dry chemistry method). The study sample was divided into two groups.

Group A: Patients receiving lipid lowering agents and/or having LDL >160 mg/dl.

Group B: Patients with LDL <160 mg/dl not receiving lipid lowering treatment. Systolic 15.0 was used for the epidemiological analysis. Data are presented as mean±standard deviation.

Results: Dyslipidemia was diagnosed in 54.6% of the subjects. 42% of the dyslipidemic group was not receiving lipid lowering agents. BMI and waist circumference were slightly higher over the dyslipidemics (28.2±4 kg/m² and 97.4±13 cm respectively) whereas hypetension, smoking and diabetes found to cluster in the same group. Cardiovascular risk score was 19% for the dyslipidemics and 29% for the dyslipidemics who smoked. Atheromatic index for the dyslipidemic group was not receiving lipid lowering agents. BMI and waist circumference were slightly higher over the dyslipidemics (28.2±4 kg/m² and 97.4±13 cm respectively) whereas hypetension, smoking and diabetes found to cluster in the same group. Cardiovascular risk score was 19% for the dyslipidemics and 29% for the dyslipidemics who smoked. Atheromatic index for the dyslipidemic group was not receiving lipid lowering therapy.

Conclusions: Dyslipidemia is a major cardiovascular risk factor. Despite that fact most subjects with dyslipidemia were not receiving lipid lowering therapy. The prevalence of risk factors was increased in the subjects with dyslipidemia and those were at higher risk of developing cardiovascular disease.