Results: The exercise ECG showed an abnormal result in 69 patients (50%) (ST-segment depression ≥0.1mV and/or reproduction of the patient's usual symptoms). The ACH-test revealed abnormal coronary vasomotion (reproduction of ischemic symptoms, ischemic ECG shifts v- diffuse distal spasm) in 87 patients (64%). Such a result was significantly more often found in patients with a pathologic exercise ECG than in those with a normal exercise ECG (50.6%, 72%, vs 37.6%, 54%, p=0.034). There were no other statistically significant differences between patients with and those without a pathologic exercise ECG.

Conclusion: A pathologic exercise ECG does not represent a false negative finding in patients with angina and non-obstructed coronary arteries but is indicative of underlying microvascular disease.

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Clinical and anamnestic characteristics of patients with CAD depending on gene polymorphism T786C promoter of endothelial NO synthase.

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Objective: To investigate T-786C polymorphism of eNOS gene in Ukrainian patients with CAD and relationship between this polymorphism and clinical presentation.

Materials and methods: We examined 120 patients with CAD, stable angina pectoris II-III functional class who had haemodynamically significant coronary artery (CA) stenosis on selective coronary angiography (SCG). Control group included 35 persons without morphologic and atherosclerotic CA pathology. Examination included assessment of ankle-brachial index (ABI), diameter of lower extremities vessels and carotid arteries with evaluation of intima-media complex thickness (IMCT).

Allele polymorphism of eNOS gene was assessed by polymerase chain reaction with electrophoretic scheme of result detection.

Results and discussion: Genotyping T-786C polymorphism of eNOS gene in control group showed the following distribution of T/T, C/T and C/C genotypes: 45.7; 48.6 and 5.7%; in patients with CAD - 37.3; 40.8 and 21.7%, respectively. The ACH-test revealed abnormal coronary vasomotion (reproduction of ischemic symptoms, ischemic ECG shifts) in 87 patients (64%). Such a result was significantly more often found in patients with a pathologic exercise ECG than in those with a normal exercise ECG (50.6%, 72%, vs 37.6%, 54%, p=0.034). There were no other statistically significant differences between patients with and those without a pathologic exercise ECG.

Conclusion: A pathologic exercise ECG does not represent a false negative finding in patients with angina and non-obstructed coronary arteries but is indicative of underlying microvascular disease.

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Usefulness of the baseline lymphocyte count to predict platelet reactivity during clopidogrel therapy in patients with stable coronary artery disease.


Purpose: This study aimed to assess long-term prognosis of stable coronary artery disease (sCAD) in patients aged ≥75 years and to identify clinical predictors of cardiovascular and overall mortality.

Methods: From February 2000 to January 2007, 391 outpatients aged ≥75 years (median 78 years, interquartile range [IQR] 76-81 years, 66% male) with sCAD were recruited in this prospective cohort study. Associations of baseline variables with long-term cardiovascular and all cause death were investigated.

Results: After up to 11 years of follow-up (median 4 years, IQR 2-6 years), 89 patients died (23%, 5.4%/year), 35 from cardiovascular causes (9%, 2.1%/year). Multiple linear analysis identified family history of coronary disease (HR 4.28, 95% CI 1.22-15.02, p=0.02), baseline atrial fibrillation (HR 3.18, 95% CI 1.37-7.39, p=0.007), age (HR 1.61 per 5 year-increase, 95% CI 1.04-2.50, p=0.03), resting heart rate (HR 1.26 per 5 bpm-increase, 95% CI 1.09-1.47, p=0.003), and previous revascularization (HR 0.17, 95% CI 0.04-0.77, p=0.02) as independent predictors of cardiovascular death; and previous acute coronary syndrome (HR 4.93, 95% CI 1.49-16.30, p=0.009), baseline atrial fibrillation (HR 1.96, 95% CI 1.12-3.43, p=0.005), tobacco use (HR 1.69, 95% CI 1.00-2.84, p=0.049 for ex-smoking and HR 6.78, 95% CI 0.89-51.47, p=0.06 for active smoking), age (HR 1.58 per 5 year-increase, 95% CI 1.18-2.11, p=0.002), resting heart rate (HR 1.10 per 5 bpm-increase, 95% CI 1.00-1.22, p=0.05) and diastolic blood pressure (HR 0.97, 95% CI 0.94-0.99, p<0.01), as independent predictors of overall mortality.

Conclusions: In this study, 4-years overall mortality was 23% among elderly patients with sCAD. Simple clinical variables can identify patients at higher risk of mortality.

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Usefulness of the baseline lymphocyte count to predict platelet reactivity during clopidogrel therapy in patients with stable angina pectoris.

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Inflammation is one of the major factors affecting Platelet (PLT) function and PLT reactivity to Clopidogrel (CLO) in Stable Angina Pectoris (SAP) patients undergoing coronary artery stent implantation.

Methods: We investigated the predictive value of the preprocedural count of different types of white blood cells on PLT reactivity to Clopidogrel (CLO) in Stable Angina Pectoris (SAP) patients undergoing coronary artery stent implantation.

Results: The exercise ECG showed an abnormal result in 69 patients (50%) (ST-segment depression ≥0.1mV and/or reproduction of the patient's usual symptoms). The ACH-test revealed abnormal coronary vasomotion (reproduction of ischemic symptoms, ischemic ECG shifts v- diffuse distal spasm) in 87 patients (64%). Such a result was significantly more often found in patients with a pathologic exercise ECG than in those with a normal exercise ECG (50.6%, 72%, vs 37.6%, 54%, p=0.034). There were no other statistically significant differences between patients with and those without a pathologic exercise ECG.

Conclusion: A pathologic exercise ECG does not represent a false negative finding in patients with angina and non-obstructed coronary arteries but is indicative of underlying microvascular disease.

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Expression of ischemic preconditioning in patients with chronic coronary artery disease with and without diabetes mellitus.

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Background: Ischemic heart disease may, with the occurrence of acute events, deteriorate to myocardial dysfunction. Compensation mechanisms such as Ischemic Preconditioning (IP), a powerful intrinsic phenomenon of myocardial protection, can minimize the deleterious effects of events. Additionally, coronary artery disease (CAD) associated with diabetes mellitus is associated with worse outcomes. Although experimental studies have shown that diabetes interferes negatively with the development of IP, it’s still unknown whether diabetes can influence the expression of IP in patients with chronic CAD.

Objective: To evaluate and compare IP in chronic CAD patients with and without diabetes.

Methods: Diabetic and non-diabetic patients with chronic, stable CAD and preserved left ventricular systolic function were submitted to two sequential exercise tests (SETs) with 30-minute interval between them. Ischemic parameters were compared between diabetic and non-diabetic patients. Ischemic preconditioning was considered present when time to 1mm ST-segment deviation was greater in the second of two SETs. Tests were analyzed by two independent and experienced cardiologists.

Results: From 160 CAD patients who underwent SETs, 112 patients developed IP compared with 48 patients who did not (P=0.0001). From all patients, 75 were diabetic (group 1) and 85 were non-diabetic patients (group 2). The two groups were similar in terms of main prognostic parameters, besides previous infarction and cholesterol profile. In group 1, 54 (72%) patients developed IP and in group 2, 56 (65%) patients had IP. In diabetic patients, compared with the control group, the improvement in time to 1mm ST-segment deviation was quite similar (76 vs 66 seconds, respectively for groups 1 and 2; P=0.19). Improvement in time to the onset of angina during SETs were also similar between the groups (45.2 vs 76.7 seconds, respectively for groups 1 and 2; P=0.56).

Conclusion: In this study a significant number of diabetic and non-diabetic patients with coronary artery disease developed ischemic preconditioning. Additionally, diabetes mellitus appears not to affect the magnitude of this protective mechanism.