

Topic 2 – Atherosclerosis, Haemostasis, Inflammation, AGE – B

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0036

Predictive value of clinical and electrocardiographic features in survivors of out-of-hospital cardiac arrest

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Background: Acute coronary lesions are known to be the most common trigger of out of hospital cardiac arrest (OHCA). But the diagnosis of acute coronary artery disease in survivors of out-of-hospital cardiac arrest is difficult. The aim of the present study was to assess the predictive value clinical and electrocardiographic features in diagnosing the presence of acute coronary lesions among out-of-hospital cardiac arrest patients.

Methods: Clinical and electrocardiographic data collected before coronary angiography were analyzed to determine whether they could be used to predict the presence of recent coronary – artery occlusion on angiography.

Results: 54 patients underwent coronarography angiography after OHCA; 42% of patients had ST-segment elevation and 57% of patients had other ECG patterns on post-restoration of spontaneous circulation (ROSC) ECG. Acute coronary lesions was found in 66% of patients; Significant coronary artery disease was observed in 83% of patients with ST-segment elevation and in 55% of patients with other ECG patterns on post-ROSC ECG ($p = 0.03$).

Significant coronary artery disease was observed in 100% of patients with chest pain before the arrest and in 55% of patients without chest pain ($p=0.02$). Chest pain has a good positive predictive value but a low negative predictive value in diagnosing the presence of acute or presumed recent coronary artery lesions (100% and 45%, respectively).

Conclusion: ST-segment elevation and chest pain before arrest after OHCA should not be considered as strict selection criteria for performing emergent coronary angiography in patients resuscitated from OHCA; even in the absence of ST-segment elevation on post- return of spontaneous circulation ECG, acute culprit coronary lesions may be present and considered the trigger of cardiac arrest.

0410

Validation of the GRACE risk score for predicting death within 6 months of follow-up in a contemporary cohort of patients with acute coronary syndrome: Algerian cohort

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Introduction and Objectives: The Global Registry of Acute Coronary Events (GRACE) risk score provides an estimate of the probability of death within 6 months of hospital discharge in patients with acute coronary

syndrome (ACS). Our aim was to assess the validity of this risk score in a contemporary cohort of patients admitted to an Algerian hospital.

Methods: The study involved 383 consecutive patients with ACS evaluated between January 2010 and January 2014. Their vital status was determined 6 months after hospital discharge and the validity of the GRACE risk score was evaluated by assessing its calibration and its discriminatory capacity.

Results: In total, 142 (37%) patients were admitted for ST-elevation myocardial infarction (STEMI) and 241 (67%) for non-ST-elevation myocardial infarction (NSTEMI). Percutaneous revascularization was performed in 249 (65%). The median GRACE risk score was 121 [interquartile range, 96-144]. Mortality 6 months after discharge was 4.9%. The calibration of the GRACE risk score was acceptable and its discriminatory capacity was excellent.

Conclusions: The GRACE risk score for predicting death within 6 months of hospital discharge was validated and can be used in patients with ACS. It would be wise to include the GRACE risk score in the medical records of these patients.

0204

Cardiac mitochondria vulnerability to reactive oxygen species: long term effects of statins in a model of hereditary hypercholesterolemia (Watanabe rabbits)

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Early primary prevention of atherosclerosis in high-risk patients is still a major challenge to decrease the burden of cardiovascular diseases. Treatment by statins is part of the strategies for prevention either by lowering LDL-CT or by their pleiotropic effects. Indeed, statins improve endothelial function, enhance the stability of atherosclerotic plaques, decrease inflammation and reactive oxygen species (ROS) production (at least at myocardium level).

Our aim was to determine the long-term effect of atorvastatin on mitochondrial function of myocardium focusing on ROS susceptibility. The Watanabe rabbit was used in this study as a hereditary hypercholesterolemia animal model.

Thirty-three Watanabe rabbits were randomly assigned to two groups: a control group without treatment and a group treated with atorvastatin (*per os* 2.5 mg. kg⁻¹.day⁻¹) from the age of 3 months. Blood was sampled monthly from the median artery of the ear for lipid analysis. The myocardium was sampled at the age of 3, 6, 9 and 12 months in both groups. Cardiac fibers were then permeabilized (using saponin). Maximal mitochondrial oxygen consumption was measured *in vitro* after incubating or not fibers with ROS (Fenton reaction) in order to assess the susceptibility of the mitochondrial function to ROS.

Decrease in blood lipids showed the efficiency of the atorvastatin treatment. After exposure to ROS, decrease in maximal mitochondrial oxygen consumption was significantly less important in treated than in control group from the age of 9 months (respectively 24.8±1.5% vs 40.9±2.8%, $p=0.0006$). No difference was observed in younger rabbits.

Our results show that long term atorvastatin treatment decreases mitochondrial vulnerability to ROS (6 months) in Watanabe heritable hyperlipidemic rabbits. This effect may be related to an increase in antioxidant capacities (measurements in progress) and/or decrease in ROS production (by mitochondria or NADPH oxidase) and/or lower potential ROS target.