Incidence of atrial fibrillation in patients with history of paroxysmal supraventricular tachycardia

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Patients (pts) with history of paroxysmal supraventricular tachycardia (PSVT) have a higher risk of atrial fibrillation (AF) than other pts. The purpose of the study was to evaluate the incidence of AF in pts seen for PSVT and to look for the factors of AF.

Population: 1099 pts aged from 5 to 85 years were consecutively studied for spontaneous PSVT that was confirmed by electrophysiological study (EPS). Pts with anterograde conduction through an accessory pathway were excluded.

Methods: The history of spontaneous AF was noted. Clinical factors (age, gender, heart disease) and electrophysiological factors were noted. Pts with anterograde conduction through an accessory pathway were excluded.

Results: 62 pts developed documented paroxysmal or permanent AF or flutter (6 %). Several clinical factors were associated with AF: pts were older than 1037 pts without AF (59±13 years vs 50±19, p < 0.0009); they were more frequently men (35/62; 56 %) than other pts 383/1037; 37 %) (p < 0.002). Associated heart disease (HD) (ischemic, valvular, hypertensive HD) was more frequent in pts with AF (17/62; 27 %) than in pts without AF (66/1037; 6 %) (p < 0.0000). There were no differences at EPS concerning the mechanism of reentry: paroxysmal atrioventricular (AV) node re-entrant tachycardia (AVNRT) was noted in 48/62 pts with AF (77 %), 712/1037 pts without AF (69 %) (NS); reentry in a concealed accessory pathway (AVRT) was noted in 8 pts with AF (14 %) and 191 pts without AF (18 %) (NS); atypical AVRT was noted in 6 pts with AF (10 %) and 134 pts without AF (13 %) (NS). The induction or the spontaneous occurrence of AF during electrophysiological study was more frequent in pts with AF (17/62; 27 %) than in pts without AF (113/1037; 11 %) (p < 0.0000).

Conclusions: The incidence of AF was 6 % in 1099 consecutive patients who had PSVT. The risk was correlated with the classical factors of AF, the older age, the male gender and the presence of HD. The mechanism of the reentry does not change the incidence of AF but the induction of AF is more frequent than in other patients. Thus, patients with PSVT and with these risk factors should be carefully followed.

Respective roles of transactivating function-1 and -2 of estrogen receptor alpha in the vasculoprotective actions of estradiol

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Full length 66kDa estrogen receptor alpha (ER) stimulates target gene transcription through two activation functions (AF), AF-1 in the N-terminal domain and AF-2 in the ligand binding domain. Another physiologically expressed 46kDa ER isoform lacks the N-terminal A/B domains and is consequently devoid of AF-1. To evaluate the involvement of ER AF-1 and AF-2 in the vasculoprotective actions of estradiol (E2), we generated a targeted deletion of the ER A/B domain in the mouse named ERAF-10 mice, and a targeted deletion of amino acids 543-549 and thus deficient in AF-2 (named ERAF-20 mice).

Both basal endothelial NO production was increased by E2 administration in a similar extent than in control mice. E2 similarly decreased fatty streak deposits at the aortic root from both ovariectomized 18 week-old ERAF-14/+ -LDL-r-/- (Low Density Lipoprotein receptor) and ERAF-10 LDL-r-/- mice fed with a hypercholesterolemic diet. We conclude that ER AF-1 is not required for the vasculoprotective actions of E2, whereas it is necessary for the effects of E2 on its reproductive targets. Thus, Selective Estrogen Receptor Modulators stimulating ER independently of the A/B domain and thereby with minimal activation of ER AF-1 could retain beneficial vascular actions, while minimizing the sexual effects.

The results concerning ERAF-20 mice are in process and will be available at the end of 2010, and the precise role of AF2 in these actions will be presented.
ular (LV) remodelling after acute MI, but the mechanism of this improvement has never been assessed. We evaluated the relationship between ECFC levels and microvascular obstruction (MVO), and the impact of this relation on infarct size and LV remodelling at 6 months as assessed by magnetic resonance imaging (MRI).

Methods: 109 pts aged 75 years old, admitted with a first MI within 12 hours of onset of symptoms were enrolled. Peripheral blood samples were drawn to assess number of ECFC colonies (culture cells). Measurements of infarct size by MRI were performed at day 5 and 6 months.

Results: ECFC colonies were detected in 51/109 pts (47.2%) at admission (ECFC pos pts). At 5 days, MVO was more frequently observed (63% vs 33%; p=0.003) and of greater magnitude (7±6% vs 5±5%; p=0.0004) in ECFC pos patients versus ECFC neg pts respectively. At 6 months, there was a significantly greater reduction in infarct size in ECFC pos pts (~33.7±33.2% vs ~15.1±24.6%, ECFC pos vs ECFC neg respectively; p=0.003). This reduction in infarct size was associated with a significant improvement in LV ejection fraction and a significant reduction in LV end diastolic and systolic volumes in ECFC pos pts. A significant positive correlation was observed among ECFC pos pts between MVO at day 5 and infarct size at 6 months (r=0.58, p<0.0001), while the number of ECFC colonies was significantly correlated with the relative change in infarct size at 6 months MRI (r²=0.33, p<0.0001).

Conclusion: The presence of ECFC colonies is associated with a reduced degree of microvascular obstruction early after myocardial infarction, leading to reduced infarct size and positive LV remodelling at 6 months and can be considered as a marker of microvascular integrity in acute MI pts.

Validation of assessment of circulating oxidative stress markers by the Free Oxygen Radicals Testing (FORT) assay among patients with an acute myocardial infarction.

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Background: Free oxygen radicals play an important role in the pathogenesis of many diseases including cardiovascular diseases, diabetes, cancer and aging. Several methods were developed for the direct or indirect measurement of oxygen free radical and its by-products. Using a new Free Oxygen Radicals Testing (FORT) the current study is designed first to validate the device and to investigate the potential relationships between the ROS and clinical or biological factors in human serum from a population of men with an acute myocardial infarction (AMI).

Methods: We first determined the effect of storage, variability and reproducibility of the FORT test in serum. Then we used the test in 66 patients from our bio bank of AMI patients.

Results: FORT values vary between 324 and 1198 FORT units, with a median value of 581 (494-754) FORT units. Among the risk factors, 17% of patients are diabetic, and 20% are obese. In univariate analysis, the FORT values seem to be influenced by age (r=-0.161, p=0.195), presence of diabetes (p=0.102), a history of MI (p=0.181), LVEF <40% (p=0.005) and treatment with -blockers before admission (p=0.053), with ST-Elevation MI (p=0.058), levels of CRP (r=0.438, p<0.001), the rate of neutrophil (r=0.203, p=0.107) and peak CK (r=0.274, p=0.028). The analysis of multiple linear regression showed that CRP (p=0.023), LVEF <40% (p=0.001) and presence of diabetes (p=0.039) were independent predictors of serum FORT levels. This statistical model can explain 45% of the variance in the FORT levels.

Conclusions: The variability of the FORT on serum is minimal and thus reproducibility can be attained. FORT assay is stable when stored at 20 °C for a couple of months or at 4 °C for a few days. FORT correlation with CRP, LVEF and status of diabetes provides an interesting insight and a good link between oxidative stress and inflammation in patients with an AMI.

The polymorphism Trp719Arg in the kinesin-like protein 6 is associated with the presence of late outgrowth endothelial progenitor cells in acute myocardial infarction

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Background: Much attention is focused on genetic polymorphisms associated with coronary artery disease. A candidate gene controversy associated with risk of coronary events is the kinesin-like protein 6 (KIF6), which is correlated with Trp719Arg polymorphism. In acute myocardial infarction (AMI), endothelial progenitor cells, particularly endothelial colony forming cells (ECFC) are mobilized from bone marrow and correlated with infarct size reduction. We investigated whether there was a relationship between presence of ECFC in AMI at admission and genetic status with regard to the KIF6 Trp719Arg polymorphism (rs20455).

Methods: Forty five patients aged 75 years old referred for a first STEMI or non STEMI AMI. Peripheral blood samples were drawn on admission. Isolated peripheral blood mononuclear cells were obtained by Hypaque-Ficoll density gradient centrifugation and cultured for 4 weeks. Cultured cells were phenotyped to assess the endothelial origin of ECFC. Genomic DNA was extracted in all patients and genotyping for allelic variations of KIF6 was performed.

Results: Subjects were divided into two groups comparing the (Arg/Arg) homozygote variants with patients having a Trp allele. The genotype frequencies were 55%, 31% and 13% for Arg/Arg, Arg/Trp and Trp/Trp respectively. Between groups, higher levels of Trp, CK and CK-MB were observed in the Arg/Arg group (respectively p=0.026, p=0.001 and p=0.031). ECFC were observed in 33% of patients with AMI. The percentage of patients without ECFC was significantly higher in the Arg/Arg group (p=0.033). No other significant differences were observed between groups.

Conclusion: In this report the Arg/Arg group showed a high number of ECFC-negative patients. A possible explanation might be the low mobilization of ECFC from bone marrow in this genotype since KIF6 is involved in cytokine stabilization. The altered amino acid Trp719Arg could decrease the ECFC release from the bone marrow in response to chemokines released at the onset of AMI.

Renin-angiotensin-aldosterone system polymorphisms: a role or a hole in occurrence and long-term prognosis of acute myocardial infarction at Tunisian older people

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Background: Myocardial infarction (MI) is one of the major causes of death all over the world. Because MI frequently occurs suddenly without any preceding clinical symptoms, the prediction of MI is clinically of great importance. Angiotensin II is produced primarily by angiotensin I-converting enzyme (ACE) within atherosclerotic lesions and ACE level correlates with the severity of vessel wall damage. We analyzed the evolution with age of the frequencies of the ID polymorphism of the ACE, A1166C of the angiotensin II AT1 receptor (AT1R), and M235T of the angiotensinogen (AGT) gene in a healthy population and we subsequent comparison to age- and sex-matched groups of MI patients.

Aim: To investigate the influence of increasing age on the incidence and remaining lifetime risk of myocardial infarction in a cohort of older men.