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 and without AF were compared.

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: typical 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 AVNRT 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 < 0.00000).

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 as in control mice. E2 similarly decreased fatty streak deposits at the aortic root from both ovariectomized 18 week-old Eraf-14/4, 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.

Metabolic and cardiovascular consequences of postnatal overfeeding in mice

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Cardiovascular and metabolic consequences of overweight are a growing concern in both western and developing countries. Several studies in mice have shown that postnatal overfeeding (OF) induces permanent moderate increase of body weight in the adult life; however, cardiovascular repercussions of postnatal OF are less known.

Immediately after birth, and during 3 weeks, litters of C57BL/6 mice were either maintained at 10 (normal-fed group, NF), or reduced to 3 in order to induce an OF situation. At weaning, mice of both groups received a standard diet. Measurements of body weight, and metabolic parameters (cholesterol, insulin and leptin) were performed in the plasma at 7 months. Cardiac function was assessed by echocardiography and the susceptibility to myocardial global ischemia and reperfusion was assessed ex vivo in isolated perfused heart.

OF led to an increase in body weight (+30%) as compared to NF group. Significant increases of plasma cholesterol (0.67 vs 0.78 g/l, p<0.05), insulin (0.98 vs 1.63 ng/ml, p<0.05) and leptin (0.39 vs 0.91 ng/ml, p<0.05) levels were observed in OF mice as compared to NF mice. In vivo, diastolic (97 vs 114 mmHg, p<0.05) and systolic (126 vs 140 mmHg) blood pressure were significantly higher in OF mice than NF mice. Moreover, LV shortening and ejection fraction were decreased in OF mice. Ex vivo, after 30 minutes of ischemia, hearts isolated from mice that underwent postnatal OF showed lower recovery of coronary flow (35% vs 55%, p<0.05), of developed ventricular pressure, of +dP/dt and -dP/dt. Moreover, infarct size evaluated after 2 hours of reperfusion was increased in OF group (31% vs 54%, p<0.05) as compared to NF.

These results show that OF induces metabolic and functional disturbances but also a higher susceptibility to cardiac functional damage after ischemia ex vivo. Complementary data are required to understand the cellular pathways implicated in these metabolic and cardiovascular modifications.