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Higher mortality seen in patients with frequent premature atrial contractions

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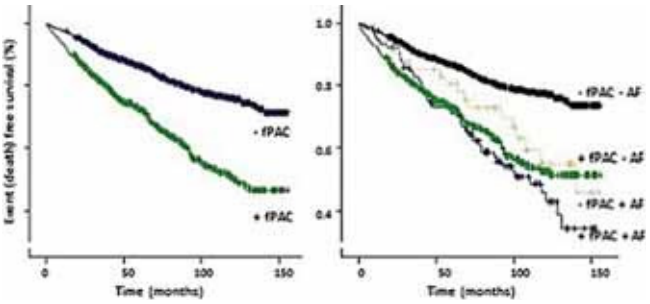
Purpose: Frequent premature atrial complexes (fPACs) have been reported as a risk factor for Atrial Fibrillation (AF). Some studies have linked fPACs to high mortality. More work is needed to establish this relationship.
Methods: We analyzed Holter recordings obtained between 2000 and 2010 of 1357 patients free of AF at baseline. Holter groups with fPAC (> 100/day) and infrequent PACs (< 100/day) were compared. Electronic medical records and EKGs were reviewed to ascertain baseline characteristics as well as occurrence of AF and death during median follow-up of 7.5 yrs. Logistic regression and Kaplan-Meier analyses were performed.
Results: Mean age was 64 yrs with 93% men. Mean BMI, A1c, left atrial size and average HR were 31.24 kg/m^2, 6.42%, 42.56 mm and 73 bpm, respectively. 37.31% of patients with fPACs died during follow-up as compared to 18.87% in non-fPAC group. After adjusting for demographics, medication use, co-morbidities, lab and echo findings, multivariate regression analyses confirmed fPACs to be independently associated with higher incidence of death (OR 1.44, 95% CI 1.07-1.92, P=0.015). Furthermore, mortality rates in patients with +fPAC/+AF, -fPAC/+AF, +fPAC/-AF and -fPAC/-AF were 48.11%, 36.73%, 34.3% and 17.8% respectively (p< 0.0001).



Kaplan-Meier survival curves

Conclusion: Patients with fPACs (> 100/day) have higher all-cause mortality, especially when they develop subsequent AF.

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Anti-arrhythmic drugs did not reduce progression from paroxysmal to sustained atrial fibrillation: from the Fushimi AF Registry

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Purpose: Atrial fibrillation (AF) increases the risks of thromboembolism and death, and the prevalence of AF is increasing significantly (reportedly, 0.6% of total population in Japan).
Progression from paroxysmal to sustained types (persistent or permanent) of AF is sometimes seen, but the effect of rhythm-control therapy on the progression remains elusive. We investigated the effect of anti-arrhythmic drugs (AAD) on the progression of AF.
Methods: The Fushimi AF Registry, a community-based prospective survey, was designed to enroll all of the AF patients living in Fushimi-ku, Kyoto, which is a typical urban district of Japan with a population of 283,000. At present, we have enrolled 3,821 Japanese AF patients (1.2% of total population) from March 2011 to December 2013. We investigated 1,398 paroxysmal AF patients at baseline who completed one-year follow-up.
Results: 457 paroxysmal AF patients (32.7%) were prescribed AAD at baseline; 107 (7.7%) for Vaughan-Williams class Ia, 17 (3.7%) for Ib, 318 (22.8%) for Ic, 13 (0.9%) for amiodarone, 34 (2.4%) for bepridil. 428 patients (30.6%) were prescribed single AAD and 29 patients (2.1%) were prescribed multiple AADs. Patients receiving AAD were more often male (61.7% vs. 55.6%; p=0.03) and younger (71.1 vs. 73.4 years of age; p< 0.01). Patients with AAD were less likely to have heart failure (11.2% vs. 21.0%; p< 0.01), diabetes (20.1% vs. 25.6%; p=0.02) and chronic kidney disease (28.5% vs. 34.6%; p=0.02). CHADS2 score was lower in patients receiving AAD (1.63 vs. 1.97; p< 0.01). HATCH score, a postulated scheme for the prediction of AF progression, was also lower in patients receiving AAD (1.56 vs. 1.97; p< 0.01). During the one-year follow-up period, progression of AF occurred in 79 patients (5.7%); 27 patients receiving AAD (5.9%) and 52 patients not receiving it (5.5%), with an odds ratio for patients receiving AAD of 1.07 (95% confidence interval [CI], 0.66 to 1.72; p=0.77) using univariate analysis, and 1.07 (95% CI, 0.65 to 1.73; p=0.79) using multivariate analysis. Furthermore, investigating each class of AAD, progression of AF occurred in 6 patients for Ia (5.6%), 2 patients for Ib (11.8%), 16 patients for Ic (5.0%), 2 patients for amiodarone (15.4%) and 3 patients for bepridil (8.8%). Progression of AF occurred in 25 patients with single AAD (5.8%) and 2 patients with multiple AADs (6.9%).
Conclusion: 5.7% of paroxysmal AF patients progressed to sustained AF in a year. Any class of AAD did not reduce progression from paroxysmal to sustained AF, during one-year follow-up of the Fushimi AF Registry in Japanese AF patients.

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Temporal changes in spatial characteristics of scar tissue in an experimental model of acute myocardial infarction: an MRI study

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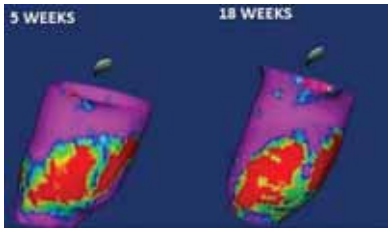
Background: Spatial characteristics of scar tissue including total extent of heterogeneous tissue (HT) and intrascar corridors of HT are related to cardiac mortality. However, little is known concerning evolution of scar architecture during the first months after acute myocardial infarction (AMI).
Methods: A transmural anterior AMI was created in 12 pigs. Myocardial scar was characterized 5 weeks and 18 weeks later with gadolinium ceMRI. End-diastolic and end-systolic left ventricular volumes (EDV and ESV), left ventricular mass and necrotic mass were quantified with QMASS 7.2® MEDIS. The ventricular wall was divided in 2 layers and the average of the subendocardium and subepicardium signal intensity (SI) was projected over 3D endocardial and epicardial shells. The SI was color coded, thus defining three different areas: 1) healthy tissue defined by SI < SI peak in normal myocardium, 2) core scar defined by SI greater than minimal SI in core scar and 3) heterogeneous tissue (HT) in between those extremes. A SI channel was defined as a corridor of HT differentiated by a lower SI from the surrounding scar.
Results: Mean EDV and ESV at 5 weeks after AMI were 114± 13 cc and 77± 13 cc, with a mean ejection fraction of 29± 9%. Mean increase of EDV and ESV at 18 weeks was 49± 15 cc and 31± 17 cc. Between 5 weeks and 18 weeks, mean reduction of endocardial and epicardial scar area was 5± 15 cm^2 and 3± 15 cm^2. At 5 weeks, 10 endocardial and 16 epicardial SI channels were observed. At 18 weeks, 20 endocardial and 18 epicardial SI channels were identified.



Conclusions: Spatial characteristics and architecture of scar change over time in the first months after AMI. A better knowledge of patterns of evolution of scar could be relevant for risk stratification in ischemic heart disease.

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Long term prognosis of intraventricular conduction delay in patients with structurally normal heart

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Purpose: Long-term prognosis of nonspecific intraventricular conduction delay (IVCD) remains unclear. This study was performed to elucidate long-term prognosis of IVCD in structurally normal heart.
Methods: Between 2000 and 2005, we evaluated the patients who underwent 12-lead electrocardiography. IVCD was defined QRS > 110ms without the criteria of complete or incomplete bundle-branch block. The patients were excluded if they had structural heart diseases, such as myocardial infarction, valvular heart disease, congenital heart disease and cardiomyopathy. In a matched-cohort design, 353 patients (IVCD group) (male 83.9% with a mean age of 52.2± 13.3 years) were matched for age and sex with 1060 patients (no IVCD group) who had neither IVCD nor structural heart diseases.
Results: During the follow-up of 6.9± 3.2 years, the cumulative incidence rates of complete atrioventricular block, heart failure, atrial fibrillation, sick sinus syndrome and all-cause mortality in the IVCD group were higher than no IVCD group (all P< 0.001, except for all-cause mortality (P=0.049)). And using univariate Cox's regression analysis, only the risk of atrial fibrillation was increased in the IVCD group (hazard ratio (HR) 1.97 [95% confidence interval (CI) 1.26–3.01], p=0.002). However complete atrioventricular block (HR 0.13 [95% CI 0.12–1.53], p=0.10), heart failure (HR 1.69 [95% CI 0.75–3.80], p=0.20), sick sinus syndrome (HR 0.39 [95% CI 0.13–1.46], p=0.16) and all-cause mortality (HR 0.79 [95% CI 0.49-1.73], p=0.79) were not increased in the IVCD group.