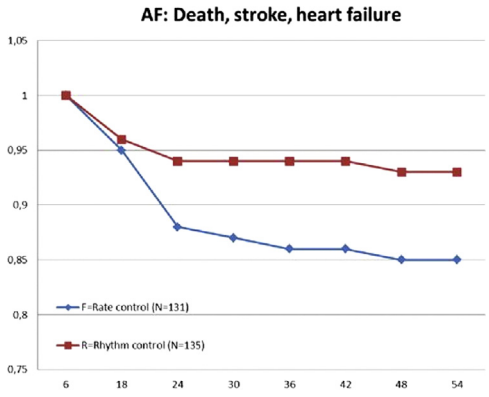

OP-150

Novel Fibroinflammatory Markers in Non-valvular Atrial Fibrillation: Galectin-3, Lcn2/NGAL, P3NP

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Introduction: Atrial fibrillation is the most common cardiac arrhythmia increasing the risk of stroke and death. Inflammation is an important factor involved in the initiation, maintenance and recurrence of AF and The abnormal inflammatory state may also cause a prothrombotic state resulting thromboembolism. In the present study we aim to investigate whether serum levels of galectin-3, Matrix Metalloproteinase 9 (MMP-9) and Procollagen III N-Terminal Propeptide (PIIINP),Human Lipocalin-2/ NGAL and NLR differ in patients with AF compared to patients with sinus rhythm in the guidance of serum levels of MMP-9 and Hs-CRP. We also evaluated associations of these markers with atrial structural remodeling which was interpreted by measuring the left atrial volume index.
Methods: The study population included 85 patients who were seen in our outpatient clinic between March 2012 and January 2012. 52 patients who diagnosed with non-valvular AF recruited into AF group. End-stage hepatic or renal disease, malignancy, any prior blood transfusions, carotid artery disease, prior transient ischemic attack, ischemic or hemorrhagical stroke and oral anticoagulant usage were exclusion criteria in our study. 35 age-matched patients with sinus rhythm recruited into control group. Serum levels of Galectin-3, Matrix Metalloproteinase 9 (MMP-9) and Procollagen III N-Terminal Propeptide (PIIINP) were measured by using a commercial enzyme-linked immunoassay kits and each assay was carried out in duplicate. Galectin-3, NGAL, MMP-9 and PIIINP levels were measured by using sandwich ELISA (Human Galectin-3 ELISA kit, eBioscience; Lipocalin-2/NGAL Elisa kit, BioVendor Research and Diagnostic Products; Human Matrix Metalloproteinase 9, Bio-Medical Assay; Human Procollagen III N-Terminal Propeptid, Bio-Medical Assay).
Results: There were significant differences between the groups in terms of inflammatory and remodeling markers except NGAL levels. We showed significantly higher levels of Galectin-3, MMP-9, PIIINP in in NVAF group compared to control group (1166 pg/ml (1126-1204) & 1204 pg/ml (1166-1362) p=0.001 Mann-Whitney U test, 146±88 pg/ml & 429±302 pg/ml p<0.0001 Student-t test, and 1426±Student-t test respectively). Hs1230 pg/ml & 6590±4594 pg/ml p<0.0001 -Crp and NLR level were also higher in NVAF (2.1±1.0 & 2.7±1.1 p=0.02 Student-t test and 4.2±1.9 mg/L & 6.0±4.7 mg/L p=0.04 Student-t test, respectively).

In correlation analyses, NLR showed quite significant correlation with LAVi whereas Hs-CRP did not (p=0.007 r=0.247, pearson test & p=0.808 r=0.025, pearson test,respectively). Moreover, Galectin-3, MMP-9, and PIIINP had strong positive correlation with LAVi (p=0.021 r=640, spearman test & p=0.004 r=0.319 pearson test, & p=0.004 r=0.325 pearson test,respectively).
Conclusion: As a result of this data we suggest that galectin-3 and PIIINP can be used as novel targets in AF patients in order to decrease degree of fibro-inflammation in the atria.

OP-151

The Genetics Polymorphism of Beta-Fibrinogen 455 G/A in Atrial Fibrillation Patients with Ischemic Stroke

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Background: Atrial fibrillation (AF) is the most commonly observed arrhythmia in clinical practice and associated with increased cardiovascular morbidity and mortality. Compared to healthy population, nonvalvular AF has a 2-7 fold increased risk of ischemic stroke. The mechanisms of thrombus formation in AF are still investigated. Fibrinogen plays an active role during the coagulation process. Increased plasma fibrinogen levels were shown to be associated with the coronary heart disease, peripheral artery disease and venous thrombosis. Beta-fibrinogen 455 G/A polymorphism is a gene mutation that may lead to alterations in the activity of fibrinogen. We wanted to investigate Beta-fibrinogen 455 G/A polymorphism in patients with AF who have had a stroke than in healthy controls.
Methods: The Beta-fibrinogen 455 G/A polymorphism was analysed in 70 patients with AF who have had a stroke 65 healthy individuals matched for age, race and sex. Because ethnic differences have been reported for Beta-fibrinogen 455 G/A polymorphism. The Beta-fibrinogen 455 G/A gene polymorphism was identified by polymerase chain reaction (PCR) method. Distribution of the Beta-fibrinogen 455 G/A gene polymorphism alles (allel G, allel A) genotypes (Normal (GG) genotype, heterozygous (GA) or homozygous (AA) mutant genotype) were identified in study population. Demographic characteristics and risk factors for AF and stroke were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. Genotype and allel distribution of nonvalvular AF patients with ischemic stroke and control groups shown in the table. The frequency of GG genotype of Beta-fibrinogen 455 G/A polymorphism was significantly lower in patients with AF who have had a stroke group compared with control group (p<0,05). The frequency of GA heterozygous genotype was similar between groups. The frequency of AA homozygous mutant genotype of Beta-fibrinogen 455 G/A polymorphism was significantly higher in patients with AF who have had a stroke group compared with control group (p<0,05). Between the two groups were compared according to the dominant genetic model (GA+AA vs. GG), The number of patients carrying at least one A mutant allele (GA+AA) were significantly higher in patients with AF who have had a stroke group than controls (p<0,05). With respect to allelic distribution (G vs A, additive model), the frequency of the A mutant allele was significantly higher in CAE patients (p<0,05).
Conclusions: In this study, we found that the frequency of β-fibrinogen 455 G/A gene polymorphism was higher in patients with AF who have had a stroke group compared to control subject. However, further large-sized studies are required for determining relationship between β-fibrinogen 455 G/A gene polymorphisms and patients with AF who have had a stroke group.

The Beta-Fibrinogen 455 G/A Polymorphism genotype and allel frequencies

	AF patients with Ischemic Stroke (n:70)		Control (n:65)		
	n:	%	n:	%	
GG genotype	23	32.9	36	55.4	0.008
GA genotype	27	38.6	25	38.5	0.990
AA genotype	20	28.6	4	6.2	0.001
GA+ AA genotypes (Dominant genetic model)	47	67.1	29	44.6	0.008
A allel	67	47.8	33	25.3	0.001