Table 1. AFSS outcome scores by EHRA classification and the correlation between the AFSS outcome scores and EHRA classification

	EHRA 1	EHRA 2	EHRA 3	EHRA 4	p value	Correlation coefficient (p value)
Total AF burden (range:3-30)	7.1±5.2	14.4±5.3	17.0±4.6	22.6±3.4	< 0.001	0.7 (<0.001)
Symptom severity (range 0-35)	3.6±4.7	8.8±5.8	17.7±6.6	23.8±7.4	< 0.001	0.7 (<0.001)
Health care utilization:						
Cardioversion (0-7)*	0.2±0.6	0.3±0.84	0.4±1.2	0.7±0.8	0.5	0.1 (0.09)
Emergency room visit (0-7)*	0.2±0.4	0.7±1.1	1.7±1.8	2.8±2.0	<0.001	0.4 (<0.001)
Hospitalization (0-7)*	0.2±0.4	0.6±0.8	1.0±1.1	1.8±1.6	0.001	0.4 (<0.001)
Specialist visit (0-7)*	1.5±1.5	2.5±2.3	2.5±2.0	2.8±1.8	0.2	0.2 (0.07)

Data are presented as mean±standard deviation. *The numbers indicate the range of scores. A correlation coefficient of > 0.6 indicate strong correlation. Increasing scores indicate increasing symptoms and severity. AFSS: Atrial Fibrillation Severity Scale EHRA:European Heart Rhythm Association class

Table 2. AFSS scores by SAF classification and the correlation between AFSS outcome scores and SAF classification

	SAF 0	SAF 1	SAF 2	SAF 3	SAF 4	p value	Correlation coefficient (p value)
Total AF burden (3-30)¶	5.7±5.1	9.2±5.0	15.2±4.6	17.6±3.8	21.4±4.7	< 0.001	0.75 (<0.001)
Symptom severity (0-35)¶	3.3±4.0	4.2±5.2	9.9±4.2	15.2±5.0	23.6±.6.8	<0.001	0.79 (<0.001)
Health care utilization:							
Cardioversion*	0.4±1.0	0.2±0.5	0.2±0.5	0.5±1.4	0.5±0.7	0.80	0.08 (0.40)
Emergency room visit*	0.3±0.5	0.5±1.1	0.6±1.0	1.3±1.6	2.5±1.9	<0.001	0.4 (<0.001)
Hospitalization*	0.3±0.5	0.5±0.8	0.5±0.6	0.7±1.1	1.4±1.6	0.08	0.2 (0.05)
Specialist visit*	1.7±1.5	1.8±1.8	2.8±2.6	2.2±2.1	2.7±1.7	0.47	0.18 (0.16)

Data are presented as mean±standart deviation. A correlation coeffcient of >0.6 indicates strong correlation. ¶The numbers indicate range of scores. *The score of the items in the health care utilization subscale ranges from 0-7. Increasing scores indicate increasing symptoms. AFSS: Atrial Fibrillation Severity Scale SAF: Severity in Atrial Fibrillation class

Table 3. Internal consistency of the three domains of the AFSS

	Internal consistency (Conbach α)
Total AF burden	0.85
Symptom severity	0.90
Health care utilization	0.67

AFSS: AF severity scale AF: atrial fibrillation

ORALS

OP-148

Non-Valvular Atrial Fibrillation in the Elderly; Preliminary Results from the National AFTER (Atrial Fibrillation in Turkey: Epidemiologic Registry) Study

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Objective: This study aimed at the assessment of the clinical approach to AF in the older population and the consistency with the guidelines based on the records of the multicenter, prospective AFTER (Atrial Fibrillation in Turkey: Epidemiologic Registry) study.
Methods: 2242 consecutive patients admitted to the cardiology outpatient clinics of 17 different tertiary health care centers with at least one AF attack determined on electrocardiographic examination, were included in the study. Among the patients included in the study, 631 individuals aged 75 years and older were analyzed.
Results: The mean age of the patients was determined as 80.3±4.2 years. The most frequent type of AF in geriatric population was the persistent- permanent type with a percentage of 88%. 60% of the patients with AF were female. Hypertension was the most common co-morbidity in patients with AF (76%). While in 16% of patients a history of stroke, transient ischemic attack or systemic thromboembolism was present, a history of bleeding was present in 14% of the patients. 37% of the patients were on warfarin treatment and 60% of the patients were on aspirin treatment. In 38 % of the patients who were on oral anticoagulant treatment, INR level was in the effective range.
Conclusion: The rate of anticoagulant use in the elderly with AF was 37% and considering the reason of this situation was the medication not being prescribed by the physician, one should pay more attention particularly in the field of treatment.

OP-149

Can Superiority of Rhythm Control be Expected in Young Patients with Non-Valvular Atrial Fibrillation (AF)?

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Introduction: Since transition disease has become a reality with the aging of the population, non-valvular AF appears as a new challenge in the scope of cardiology.

There are two strategies in the treatment of atrial fibrillation. One of them is cardioversion and maintenance of sinus rhythm with antiarrhythmic drugs; the other one is respect of AF and treatment with rate control drugs.

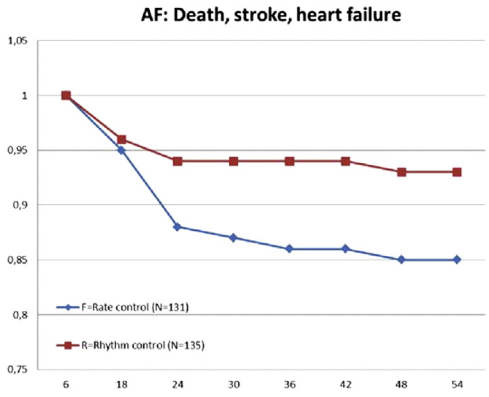
The two strategies were widely compared in the Affirm Study Without superiority of any of them. However, with a focus on the population studied in Affirm, we notice that it concerned old population at high risk of Stroke.

The aim of our study is then to compare the two strategies from another angle by targeting a population of younger and more active patients with AF.
Methods: We randomly assigned, in a prospective open-label study, 266 eligible patients with non- valvular AF, average age 52,41±9,6 years, 174 men (65,4 %,sex ratio 1,89), to undergo a rhythm control (R=131 patients) or a rate Control (F=131 pts) strategy.

The average follow -up (FU) was 27,18 months with 4 patients lost of view.

15 pts (8 and 7) were concerned by cross over from one strategy to the other.
Results: 240 pts (90%) pts were symptomatic. AF was paroxystic in 97 pts (36%), persistent in 61pts (23%) and permanent in 108pts (41%). 203 pts had comorbidities particularly hypertension in 142 (53%) and diabetes in 34 (13%). Isolated AF was present in 63pts (24%).219 pts (82%) were at low thromboembolic risk with a CHADS2 score ≤1. The hemodynamic status was favorable with an average LVEF of 64%.

There were fewer events in the R arm than in the F one, appearing late during FU but the difference wasn't statistically significant. There was no significant difference in survival without heart failure or ischemic stroke, respectively 83.8% [CI: 68.5- 100] and 56.6% [36.4-88], p=0.43.
Discussion-Conclusion: These results can be explained by the young age of the population with a low risk in which the events are rare and late in a chronic disease that requires treatment and long term follow up. For more than 2 years FU, superiority of rhythm control over rate control wasn't demonstrated despite the patients' profile that seemed to be favorable. However, given the shape of the survival curves, we can anticipate a superiority of rhythm control in the long term.



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