Table 2. The laboratory findings of the study population

	Group 1	Group 2	Group 3	p Overall	p Group 1-3	p Group 2-3	p Group 1-2
INR, %	2.54 (2.01-2.99)	1.37 (0.94-1.95)	0.97 (0.85-1.21)	<0.001	<0.001	<0.001	<0.001
Hemoglobin, g/dL	12.0 (11.0-16.8)	12.0 (11.4-16.3)	13.0 (11.7-16.2)	0.193	-	-	-
WBC, x1000/μL	7.34 (3.92-11.3)	7.43 (3.91-11.2)	7.12 (4.31-11.0)	0.295	-	-	-
Platelet, x1000/μL	236 (119-442)	219 (135-382)	236 (151-400)	0.173	-	-	-
MPV, fL	7.56± 0.63	8.26± 0.63	7.63± 0.68	<0.001	0.710	<0.001	<0.001
PCT, %	0.174 (0.089-0.274)	0.203 (0.106-0.311)	0.183 (0.099-0.304)	<0.001	0.433	<0.001	<0.001
PDW, %	17.3 (14.2-19.4)	17.9 (13.7-20.5)	17.5 (15.4-19.7)	<0.001	0.165	0.004	<0.001

INR= International normalized ratio, MPV= Mean platelet volume, PCT= Plateletcrit, PDW= Platelet distribution width, WBC= White blood cell

Human platelet antigens -1 gene polymorphisms genotype and allel frequencies

	AF patients with stroke (n:70)		Control (n:65)		P
	n:	%	n:	%	
1a1a genotype	49	70	57	87.7	0.012
1a1b genotype	18	25.7	6	9.2	0.012
1b1b genotype	3	4.3	1	1.5	0.347
1a1b + 1b1b genotypes (Dominant genetic model)	21	30	7	10.8	0.006
1b allel	24	17.1	8	6.1	0.009

PP-157

Polymorphisms of the Human Platelet Alloantigens-1 in Nonvalvular Atrial Fibrillation Patients with Ischemic Stroke in Turkish Population

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Background: Atrial fibrillation (AF) is the commonest sustained cardiac arrhythmia, which confers a high risk of mortality and morbidity from stroke and thromboembolism. Altered platelet activation and platelet-dependent thromboembolism have been associated with the pathogenesis of cardiovascular or thromboembolic disorders, which include atherosclerosis, coronary disease and cerebrovascular disease. Platelet adhesion and activation are mediated by human platelet alloantigens (HPAs), a complex of platelet membrane glycoproteins (Gp) and other cellbound Factors. By altering platelet receptor sensitivity, polymorphisms in platelet Gp directly impact platelet susceptibility to activating stimuli, which is linked with an increased risk of atherothrombotic events, including acute myocardial infarction. We wanted to investigate HPA-1 polymorphism in patients with AF who have had a stroke than in healthy controls.
Methods: The HPA-1 polymorphism was analysed in 70 patients with nonvalvuler AF who have had a stroke and 65 healthy individuals with no documented episode of AF matched for age, race and sex. Because ethnic differences have been reported for HPA-1. The HPA-1 gene polymorphism was identified by polymerase chain reaction (PCR) method. Distribution of the HPA-1 gene polymorphism alles (allel 1a, allel 1b) genotypes (1a1a, 1a1b and 1b1b) were determined 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 AF patients who have had a stroke and control groups shown in the table. The frequency of 1a1a genotype of HPA-1 polymorphism was significantly lower in patients with AF patients who have had a stroke group compared with control group (49 (70%) vs 57 (87.7%), p=0.012). The frequency of 1a1b genotype heterozygous genotype was significantly higher in AF patients who have had a stroke group than control (18 (25.7%) vs 6 (9.2%), p=0.012). Between the two groups were compared according to the dominant genetic model (1a1b + 1b1b vs. 1a1a), The number of patients carrying at least one 1b mutant allele (1a1b + 1b1b) was significantly higher in AF patients who have had a stroke group than control (21 (30%) vs 7 (10.8%), p=0.006). With respect to allelic distribution (1a vs 1b, additive model), the frequency of the 1b allele was significantly higher in AF patients who have had a stroke (24 (17.1%) vs 8 (6.1%), p=0.009).
Conclusıons: In this study, our data suggest that the HPA-1 gene polymorphisms may be associated with AF patients who have had a stroke from other clinical risk factors, but this should be confirmed in a much larger series of patients. Screening for this mutation may help in identifying patients at risk and in deciding the antithrombotic strategy.

PP-158

Evaluation of Left Atrial Functions and the Electromechanical Delay Time by Echocardiography in Patients with prediabetes

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Objective: Prediabetes is a predictor of manifest diabetes mellitus (DM) and is known to be associated with increased cardiovascular mortality and morbidity. As the diabetic patients are at higher risk of developing atrial fibrillation (AF), a significant part of the patients with lone AF are also diabetic. Inter-atrial and intra-atrial electromechanical coupling time which can be measured by both prolonged P wave dispersion and tissue Doppler imaging are known as the non-invasive predictors of atrial fibrillation. Impairment of left mechanical functions could be associated with the increased risk of developing AF. In our study, we examined the atrial electromechanical coupling time which is measured by the tissue Doppler imaging (TDI), left atrial (LA) mechanical function by disc method, and P wave dispersion of the prediabetic patients.
Matherıal-Method: 50 prediabetic (22 M, 28 F; median age: 51±10 years) and 41 healthy subjects as control group included in this study. Atrial electromechanical coupling time was calculated from lateral mitral annulus (PA lateral), septal mitral annulus (PA septum) and right ventricular tricuspid annulus (PA tricuspid) by TDI. Left atrial volumes (maximum, minimum, and pre-systolic) were measured in the apical four-chamber view with the disk method and were indexed to body surface area. Left atrial mechanical functions (LAPEV, LAPEF, LAAEV, LAAEF, CV, LATEV) were evaluated. P wave dispersion was obtained by 12-lead electrocardiography and was calculated as subtracting the minimum P wave duration from maximum P wave duration period. The results of prediabetic and control groups were analysed.
Results: Inter-atrial (PA lateral-PA tricuspid) and left atrial electromechanical delays were found to be significantly longer in patients with prediabetes than the control group (21.5±10.5 vs 13.8±5.6 msec; p < 0.001, 12.5±8.1 vs 6.7±3.7, p < 0.001, respectively). Maximum and pre-systolic volumes were found to be similar in both groups (29.1±7.2 to 27.1±8.2, p=0.24, 18.6±4.4 to 17.8±6.6, p=0.14, respectively). In the prediabetic patients, LATEV, LAAEV, CV and LAAEF were found to be higher than the control group (18,8±6,3 vs 16,1±4,5, p=0,01; 8,7±3,1 to 5,7±2,4; p<0,001, 31,3±8,3 vs 27,5±9,7, p=0,047; 0,53±0,16 vs 0,31±0,13, p<0,001, respectively).
In prediabetic patients, P-wave dispersion was found to be longer than the control group (55,3±11,1 msec to 28.9±5.9 msec, p<0.001, respectively).
Conclusıon: Prolonged atrial electromechanical delay and prolonged PWD suggested that prediabetic population have an increased risk for development of AF than the normal population. Impaired left atrial mechanical functions could be a predictor of the heart failure and atrial fibrillation which may develop in future. In our opinion, in patients with presiabetes, some precautions are to be taken before the development of overt diabetes, may prevent such cardiovascular complications as AF and heart failure.

PP-159

Autonomic Dysfunction and Arrhythmic Disorders in Patients with Coronary Artery Disease

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Occurrence and progression of coronary artery disease (CAD) and heart failure (HF) are accompanied by worsening of autonomic regulation of circulation. In its turn, this contributes to development of supraventricular arrhythmias. In this connection, neural activity modulation may be an effective approach to treatment and prevention.
Methods: Estimation of sympathetic and parasympathetic activity in 40 patients with CAD and paroxysmal atrial fibrillation (AF) and in 40 healthy subjects was performed by Valsalva maneuver test, evaluation of heart rate variability (HRV) and blood

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