Table 2. The comparison of left atrial diameters and functions between patients with and without interatrial block and P-terminal force

	Interatrial block (+) (n=42)	Interatrial block (-) (n=26)	P-value
Reservoir function(%)	35.5±6.5	46.2±8.9	<0.001
Conduit function (%)	15.8±3.5	21.2±5.1	<0.001
Pump function (%)	31.4±4.6	39.4±7.2	<0.001
Left atrial diameter (mm)	46.8±5.6	38.4±3.9	<0.001
	P-terminal force (+) (n=45)	P-terminal force (-) (n=23)	
Reservoir function(%)	37.2±5.9	44.8±6.7	<0.001
Conduit function (%)	15.9±3.7	20.7±5.2	<0.001
Pump function (%)	32.5±5.2	39.8±7.1	<0.001
Left atrial diameter (mm)	45.5±5.7	39.5±5.5	<0.001

PP-172

Evaluation of the Apolipoprotein B R3500Q Gene Mutation in Nonvalvular Atrial Fibrillation with Ischemic Stroke in Turkish Population

Atilla İçli[1], Nilgün Erten[2], Recep Sütçü[3], Salaheddin Akçay[4], Erdoğan Yaşar[1], Habil Yücel[5], Fatih Aksoy[5], Akif Arslan[5]
[1]Department of Cardiology, Ahi Evran University Education and Research Hospital, Kırsehir, [2]Department of Neurology, Giresun University, Giresun, [3]Department of Biochemistry, Katip Celebi University, İzmir, [4]Department of Cardiology, Celal Bayar University, Manisa, [5]Department of Cardiology, Suleyman Demirel University, Isparta

Background: The Apolipoprotein B (Apo B) gene mutations characterized by elevated low density lipoprotein cholesterol levels and premature coronary artery disease and/or myocardial infarction. The Apo B gene is located on chromosome 2. The Apo B gene mutation is a single base substitution (G to A) at nucleotide 10708 of the apo B gene. This mutation gives rise to the substitution of arginine for glutamine at residue 3500 of the apo B-100 molecule, within a region containing the putative receptor binding domain. Although various gene polymorphsims have been studied in patients with nonvalvuler AF who have had a stroke, Apo B gene polymorphisms have not been studied previously. We investigated relationship between R3500Q mutation of the Apo B gene and nonvalvular AF with Ischemic Stroke.
Methods: The Apo B R3500Q gene mutation was analysed in 70 patients with nonvalvuler AF who have had a stroke and 70 healthy individuals with no documented episode of AF matched for age, race and sex. The Apo B R3500Q gene mutation was identified by polymerase chain reaction (PCR) method. Distribution of the Apo B R3500Q gene alles (allel G, allel A) and genotypes (Normal (GG) genotype, heterozygous (GA) or homozygous (AA) mutant genotype) 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. There was no statistical difference in genotype distribution among the groups. The genotype distribution in nonvalvuler AF who have had a stroke group was as follows: normal genotype (GG) frequency was 67 (95.7%) and heterozygous mutant genotype (GA) frequency was 3 (4.3%). The genotype distribution in control group was as follows: normal genotype (GG) frequency was 68 (97.1%) and heterozygous genotype (GA) frequency was 2 (2.9%). Homozygous genotype (AA) was not detected in both groups. There was no statistically significant difference betwen groups in genotype distributions.
Conclusıons: Our results suggest that the Apo B R3500Q gene mutation appears not to be associated with nonvalvular AF with ischemic stroke in Turkish population.

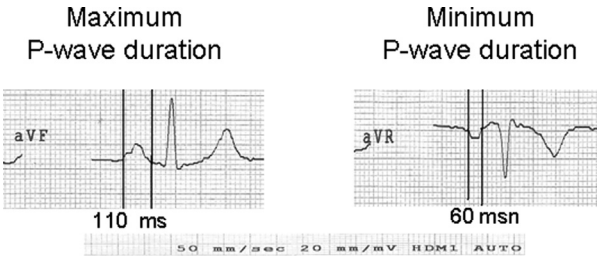
PP-173

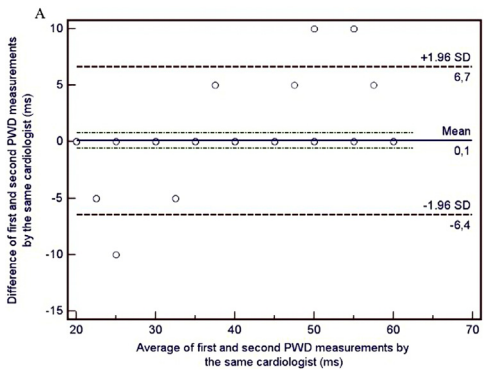
Increased P-wave Dispersion in Patients with New Diagnosed Lichen Planus

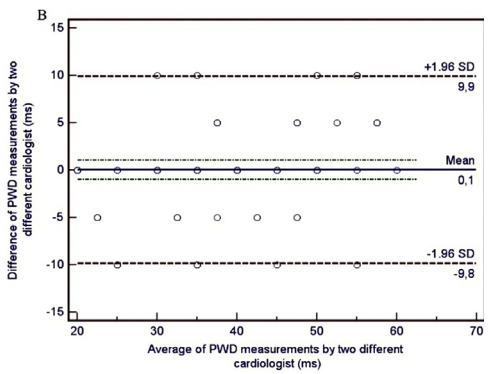
Musa Sahin[1], Serap Gunes Bilgili[2], Hakki Simsek[1], Serkan Akdag[1], Aytac Akyol[3], Hasan Ali Gumrukcuoglu[1], Mehmet Yaman[1], Yasemin Bayram[4], Ayse Serap Karadag[5]
[1]Yuzunci Yil University, Faculty of Medicine, Cardiology Department, Van, [2]Yuzunci Yil University, Faculty of Medicine, Dermatology Department, Van, [3]Van High Education and Reseach Hospital, Cardiology Department, Van, [4]Yuzunci Yil University, Faculty of Medicine, Microbiology Department, Van, [5]Medeniyet University, Faculty of Medicine, Dermatology Department, Istanbul

Purpose: Lichen planus is a chronic, inflammatory, and autoimmune mucocutaneus disease. There has been recent research emphasis on the strong association between inflammation both P-wave dispersion and dyslipidemia. The differences between the maximum and the minimum P-wave duration on electrocardiogram are defined as P-wave dispersion. Prolongation of P-wave dispersion has been demonstrated to be an independent risk factor for the development of atrial fibrillation. The aim of this the study was to investigate P-wave dispersion in patients with lichen planus.

Methods: Fifty-eight patients with lichen planus and 37 age and sex-matched, healthy controls were included in this study. We obtained electrocardiographic recordings from all participants and used them to calculate P-wave variables. We also assessed high-sensitive C-reactive proteins that an inflammatory marker and the lipid levels for each group. Results were reported as means ± standard deviations and percentages.
Results: P-wave dispersion were significantly higher in lichen planus patients than in the control group (39.9±12.9 ms, versus 32.4±11.8 ms, p=0.005, respectively). Also, high-sensitive C-reactive proteins (3.5±2.6, versus 1.7±1.1, p<0.001), LDL-cholesterol (129.6±31.7, versus 96.8±33.6, p<0.001) and, triglyceride (169.2±93.6, versus 110.5±52.7, p=0.001)levels were significantly higher in patients with lichen planus, compared to controls. There was a significant, positive correlation between high-sensitive C-reactive proteins and P-wave dispersion (r=0.549, p<0.001) in lichen planus patients.
Conclusıons: P-wave dispersion increased on surface electrocardiographic measurements in lichen planus patients. This result may be important in early detection of subclinical cardiac involvement. Increased P-wave dispersion should be considered, in terms of tendency to atrial fibrillation, in these patients.







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