

score. A weak positive correlation was detected between TO and Gensini score ($r:0.165$, $p:0.036$) and a weak negative correlation was detected between TS and Gensini score ($r:-0.193$, $p:0.013$). When HRV parameters of CAD and control groups were compared; SDNN, SDNN index and LFnu values were found significantly decreased in CAD group (SDNN: 116.3 ± 36.3 ms vs 131.8 ± 39.7 ms, $p:0.010$; SDNN index: 44.8 ± 15 ms vs 51 ± 16.7 ms, $p:0.013$; LFnu: 65.9 ± 20.2 vs 71.6 ± 14.6 , $p:0.041$ in patients with CAD and control group respectively). Also negative correlation between SDNN, SDNN index and Gensini score were found ($r:-0.356$, $p<0.001$ and $r:-0.270$, $p:0.001$ respectively). Furthermore TO is negative correlated with LFnu, HFnu, and LF/HF ratio ($r:-0.277$; $p<0.001$, $r:-0.243$; $p:0.002$, $r:-0.251$, $p:0.001$ respectively). TS is positive correlated with RMSSD and SDNN index ($r:0.203$; $p:0.009$, $r:0.170$, $p:0.030$ respectively).

Conclusions: Our data show that HRT and HRV variables are impaired in patients with CAD patients versus those with control group and weakly correlated with severity of CAD.

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Association of the Factor V Leiden Mutation (1691G>A, Arg534Gln) with Ischemic Stroke in Nonvalvular Atrial Fibrillation in Turkish Population

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Background: Factor V Leiden is an inherited disorder of blood clotting. It is a variant of human factor V that causes a hypercoagulability disorder. In this disorder, the Leiden variant of factor V cannot be inactivated by activated protein C. The gene that codes the protein is referred to as factor V. Mutation of this gene a single nucleotide polymorphism (SNP) is located in exon 10. As a missense substitution it changes a protein's amino acid from arginine to glutamine. Factor V Leiden gene is present in 3% of the general population. People who carry the factor V Leiden gene have a fivefold greater risk of thrombosis than the rest of the population. Atrial fibrillation (AF) is the commonest sustained cardiac arrhythmia, which confers a high risk of mortality and morbidity from stroke and thromboembolism. We wanted to investigate Factor V Leiden Mutation (1691G>A, Arg534Gln) in patients with AF who have had a stroke than in healthy controls.

Methods: The factor V Leiden mutation was analysed in 70 patients with nonvalvular AF who have had a stroke and 70 healthy individuals with no documented episode of AF matched for age, race and sex. The factor V Leiden mutation was identified by polymerase chain reaction method. Distribution of the factor V Leiden mutation alleles (allele G, allele A) and 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 distribution of AF patients who have had a stroke and control groups shown in the table. The frequency of GG normal genotype of the factor V Leiden was significantly lower in patients with AF patients who have had a stroke group compared with control group (57 (81.4%) vs 66 (94.3%), $p=0.02$). The frequency of GA genotype heterozygous mutant genotype was significantly higher in AF patients who have had a stroke group than control (12 (17.1%) vs 4 (5.7%), $p=0.034$). 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) was significantly higher in AF patients who have had a stroke group than control (14 (20%) vs 5 (7.1%), $p=0.026$).

Conclusions: In this study, our data suggest that the factor V Leiden mutation (1691G>A, Arg534Gln) may be associated with AF patients who have had a stroke from other clinical risk factors, but this should be associated in a much larger series of patients. Screening for this mutation may help in identifying patients at risk and in deciding the antithrombotic strategy.

Factor V Leiden Mutation (1691G>A, Arg534Gln) genotype frequencies

	AF patients with stroke (n:70)		Control (n:70)		P
	n:	%	n:	%	
GG genotype	57	81.4	66	94.3	0.020
GA genotype	12	17.1	4	5.7	0.034
AA genotype	1	1.4	0	0	0.316
GA + AA genotypes (Dominant genetic model)	14	20	5	7.1	0.026

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Effect of smoking on Tp-e interval, Tp-e/QT and Tp-e/QTc ratios as indices of ventricular arrhythmogenesis

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Background: Smoking may lead to ventricular arrhythmias and sudden cardiac death via altering ventricular recovery time dispersion indices such as QT interval and QT dispersion (QTd). Tp-e/QT ratio and Tp-e/QTc ratio are also known as predictors of ventricular arrhythmogenesis. The aim of our study was to assess effect of smoking on ventricular arrhythmogenesis using Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratios. **Methods:** 45 chronic smokers and 30 age- and sex-matched non-smoker controls were included in our study. Tp-e interval and Tp-e/QT ratio were measured by 12-lead electrocardiogram, and the Tp-e interval corrected for heart rate.

Results: QTd and corrected QT (QTc) were significantly higher in smokers group compared to controls (29.7 ± 5.6 vs 26.1 ± 4.4 and 36.1 ± 8.4 vs 31.1 ± 4.8 ms, $P=0.008$ and $P<0.007$, respectively). cTp-e interval, Tp-e/QT and Tp-e/QTc ratios were also significantly higher smokers (88.1 ± 8.5 vs 71.1 ± 6.8 and 0.24 ± 0.03 vs 0.17 ± 0.02 ms, $P<0.001$ for all). In smoker group, the amount of smoking was correlated both cTp-e interval and Tp-e/QTc ratio ($r=0.72$, $P<0.001$ and $r=0.55$, $P<0.001$, respectively).

Conclusion: We found in our study that cTp-e interval, Tp-e/QT and Tp-e/QTc ratios were increased in smokers and significantly correlated to the amount of smoking.

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Decreased transepical dispersion of repolarisation in hypertrabeculation/noncompaction cardiomyopathy patients

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Purpose: We aimed to evaluate the changes in the Tpeak±Tend interval (an index of transmural dispersion of repolarisation) with other traditional electrocardiographic indices of electrical dispersion in patients with hypertrabeculation/noncompaction (HT/NC) cardiomyopathy.

Methods: We enrolled 55 (38 men and 17 women; mean age 33 ± 15 years) HT/NC cardiomyopathy patients (mean LVEF $44 \pm 16\%$) and 18 controls (mean age 31 ± 11). Exclusion criteria were as follows:

valvular heart disease, other congenital heart diseases, documented coronary artery disease, pulmonary hypertension, chronic renal failure, diabetic neuropathy and patients using digoxin or antiarrhythmic drugs at the time of admission. Twelve-lead ECGs were recorded at a tape speed of 50 mm s-1. QT interval duration and corrected QT interval duration (according to the Bazett's formula) were measured manually after twofold magnification. The following parameters were analysed: QTp interval duration (from the onset of the QRS complex to the peak of the T wave); QTc interval duration (from the onset of the QRS complex to the end of the T wave); Tp±Te interval duration (from the peak to the end of the T wave); QT dispersion was measured from all 12 leads (I-V 6). Differences between groups were compared by using Mann Whitney-U test.

Results: Corrected values of the QTp, QTc and Tp±Te intervals in the controls and HT/NC cardiomyopathy patients are listed in Table. We found that minimum QT interval was significantly lower in the patient group than the controls ($p=0.016$). T wave negativity was more frequent in the HT/NC patients ($p=0.01$).

Conclusion: In HT/NC cardiomyopathy patients the transmural dispersion of repolarization is decreased and it may be associated with increased arrhythmias.