Coronary Care Unit Patient Profile and in-Hospital Mortality

	Group 1 (n=2041)	Group 2 (n=1181)
Age average (year)	59	63
Female (%)	24	28
Acute coronary syndrome (%)	79	72
ST elevated ACS (All of ACS) (%)	59	40
Non ST elevated ACS (All of ACS) (%)	41	60
Rhythm and conduction abnormalities (%)	9	13
Heart failure and pulmonary edema (%)	7	9
Cardiogenic shock and cardiopulmonary arrest (%)	1,9	1,3
Others (Valvular heart diseases, aort dissection, pericardial diseases, syncope, pulmonary diseases, acute pulmonary embolism, etc) (%)	3	4,7
Hospital mortality (%)	9	4,4
Hospital mortality of ACS (%)	6	4,3
Hospital mortality of ST elevated ACS (%)	6	5
Hospital mortality of non ST elevated ACS (%)	5	4,1
Hospital mortality of rhythm and conduction abnormalities (%)	11	9
Hospital mortality of heart failure and pulmonary edema (%)	11	8
Hospital mortality of cardiogenic shock and cardiopulmonary arrest (%)	65	47

PP-314

The Relationship between Endothelial Nitric Oxide Synthase Gene Polymorphism (G894T) and Isole Coronary Artery Ectasia

Atilla İçli[1], Ahmet Altınbaş[2], Yasin Türker[3], Habil Yücel[4], Salaheddin Akçay[5], Recep Sütçü[6], Fatih Aksoy[2], Akif Arslan[2], Şenol Tayyar[2], Bayram Ali Uysal[2]
[1]Department of Cardiology, Ahi Evran University Education and Research Hospital, Kırsehir, [2]Department of Cardiology, Suleyman Demirel University, Isparta, [3]Department of Cardiology, Duzce University, Düzce, [4]Department of Cardiology, Isparta State Hospital, Isparta, [5]Department of Cardiology, Celal Bayar University, Manisa, [6]Department of Biochemistry, Katip Celebi University, İzmir

Background: Coronary artery ectasia (CAE) is defined as local or generalized aneurysmal dilatation of the coronary arteries. Although the etiology of CAE has not been identified completely, the most frequent cause is coronary atherosclerosis. It is known that an expansive remodelling occurs in atherosclerotic coronary arteries due to plague rupture and increased plague burden particularly in early stages. Endothelial nitric oxide synthase (eNOS) has important role in modulating smooth muscle tonus and vessel diameter. Polymorphism of the eNOS (G894T) gene has been associated with altered function of this gene and its products. Experimental and clinical data suggesting that; in the absence of eNOS, endothelial functions and luminal remodeling is impaired, the vessel wall thickness is increased, atherosclerosis accelerated and got complicated.In this study, we investigated the eNOS gene polymorphism (G894T) in patients with CAE.
Methods: Sixty five patients with isolated CAE (mean age 53±7 years) and 65 controls with normal coronary angiograms (mean age 51±7 years) were included in the study. eNOS G894T gene polymorphisms were analysed by polymerase chain reaction and restriction fragment length polymorphism. For each polymorphic position, one of three possible patterns may be obtained: Normal (GG) genotype, heterozygous (GT), or homozygous (TT) mutant genotype. Demographic characteristics and major risk factors for atherosclerosis were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. Genotype distribution of CAE and control groups shown in the table. The frequency of the GT heterozygous genotype was significantly higher in CAE group than controls (38 (%58,5) vs 22 (%33.8), p=0.005). Between the two groups were compared according to the dominant genetic model (GT+TT vs. GG), The number of patients carrying at least one T mutant allele (GT+TT) was significantly higher in CAE than controls (43 (%66.2) vs 24 (%36.9), p=0.001). With respect to allelic distribution (G vs T, additive model), the frequency of the T mutant allele was significantly higher in CAE patients. (48 (%36.9) vs 26 (%20), p=0.004).

Endothelial nitric oxide synthase gene (G894T) polymorphisms genotype and allel frequencies

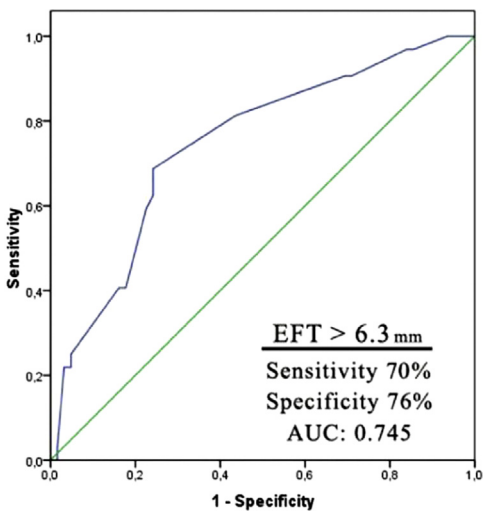
	CAE (n:65)		Controls (n:65)		
	n:	%	n:	%	P
GG genotype	22	33.8	41	63.1	0.001
GT genotype	38	58.5	22	33.8	0.005
TT genotype	5	7.7	2	3.1	0.244
GT + TT genotypes (Dominant genetic model)	43	66.2	24	36.9	0.001
T allel	48	36.9	26	20	0.004

PP-315

Association of Epicardial Fat Thickness with TIMI Risk Score in NSTEMI/ USAP Patients

Fırat Özcan, Osman Turak, Uğur Canpolat, Selçuk Kanat, Iskender Kadife, Sedat Avcı, Ahmet Işleyen, Muhammed Cebeci, Derya Tok, Fatma Nurcan Başar, Dursun Aras, Serkan Topaloğlu, Sinan Aydoğdu
Cardiology Clinic, Türkiye Yüksek İhtisas Training and Research Hospital, Ankara

Background: The association of epicardial adipose tissue (EAT) with coronary artery disease has been shown in previous studies. Furthermore the relationship between EAT and acute coronary syndrome was studied recently. Herein, we investigated the relationship between EAT thickness and the Thrombolysis in Myocardial Infarction (TIMI) risk score for non-ST elevation myocardial infarction (NSTEMI) and unstable angina pectoris (USAP).
Methods: A total of 144 patients with NSTEMI/USAP were included. Study population was divided into two sub-groups according to TIMI risk scores as group I: ≤4 (n=86) and group II: >4 (n=58). Stepwise multivariable logistic regression analysis was used to assess the independent association of clinical parameters with TIMI risk score.
Results: EAT thickness was higher in Group II compared to Group I (8.2±2.1 vs 6.2±2.2, p<0.001). Also patients in Group II showed higher rate of multivessel disease and Gensini score (p<0.001). In univariate linear regression analysis, EAT was positively correlated with TIMI risk score and Gensini score. Multivariate regression analysis showed that EAT thickness (OR: 1.56, 95% CI: 1.17-2.08, p=0.003), LVEF (OR: 0.93, 95% CI: 0.85-0.98, p=0.03) and Gensini score (OR: 1.36, 95% CI: 1.24-1.98, p=0.002) were independently associated with higher TIMI risk score.
Conclusion: In conclusion, EAT thickness is independently associated with TIMI risk score and may be an emerging risk factor for adverse events in NSTEMI/USAP.



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