Results:
The frequency of the GT heterozygous genotype was significantly higher in CAE group than controls (38% vs 22%, 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 vs 24, 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 than controls (43% vs 24% p = 0.001).

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

<table>
<thead>
<tr>
<th>Genotype</th>
<th>CAE (n=65)</th>
<th>Controls (n=65)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>GG genotype</td>
<td>22</td>
<td>33.8</td>
<td>41</td>
</tr>
<tr>
<td>GT genotype</td>
<td>38</td>
<td>58.5</td>
<td>22</td>
</tr>
<tr>
<td>TT genotype</td>
<td>5</td>
<td>7.7</td>
<td>2</td>
</tr>
<tr>
<td>GT + TT genotypes (Dominant genetic model)</td>
<td>43</td>
<td>66.2</td>
<td>24</td>
</tr>
</tbody>
</table>

T allele | 48 | 36.9 | 26 | 20 | 0.004 |

PP-315
Association of Epicardial Fat Thickness with TIMI Risk Score in NSTEMI/USAP Patients
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Background: The association of epicardial adipose tissue (EAT) with coronary artery disease and Gensini score (<0.001). In univariate linear regression analysis, 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 logistic regression analysis, EAT thickness was independently associated with higher 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 logistic regression analysis, EAT thickness was 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.

PP-314
The Relationship between Endothelial Nitric Oxide Synthase Gene Polymorphism (G894T) and Isolated Coronary Artery Ectasia
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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 plaque rupture and increased plaque 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.

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 assessed 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% vs 22%, 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 vs 24, 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% vs 26% p = 0.004).