We assessed the I/D polymorphism of the ACE gene using conventional PCR in 422 symptomatic ischemic patients referred to our catheterization unit; a second independent PCR was performed in D/D patients using specific primers to avoid D allele underestimation. Patients were classified as having stable (n=176) or unstable angina (n=246). Both groups were in Hardy-Weinberg equilibrium. The genotype distribution and the D allele frequency (p) in the two groups are shown in the table.

Conclusion: The D allele of the ID polymorphism of the ACE gene seems not to be associated with a higher incidence of an unstable and the D allele frequency (p) in the two groups are shown in the table.

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Key Words: ACE polymorphism, unstable angina, angina presentation of CAD in a selected population of symptomatic patients seems not to be associated with a higher incidence of an unstable and the D allele frequency (p) in the two groups are shown in the table.

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Key Words: ACE polymorphism, unstable angina, angina

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ASSESSMENT OF FACTORS ASSOCIATED TO IN-STENT RESTENOSIS IN NON-INSULIN DEPENDENT DIABETIC PATIENTS

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Diabetic patients (pts) have a poorer outcome after percutaneous coronary interventions. Small vessel diameter and augmented neointimal proliferation have been associated with recurrence after coronary stenting (CS). The metabolic mechanisms at the basis of the artery disease and the healing process after percutaneous injury in diabetic pts are under investigation.

We studied prospectively the association between clinical, angiographic and biochemical parameters with restenosis (R) in 49 non-insulin dependent (NID) diabetic pts treated with CS that followed a strict metabolic control after the procedure and underwent protocol angiographic QCA at six months.

R was observed in 16 pts (32.7%). At univariate analysis R pts did not differ from non-R pts in terms of age, BMI, history of AMI, degree of CAD, length of the duration of NIDD, clinical presentation, and length of the stenotic coronary segment. R pts had smaller MLD after CS (2.86±0.46 vs 3.09±0.87 p=0.1) and received higher pressure for stent expansion (14.1±3.5 vs 11.4±5.9 p=0.06). The biochemical assessment revealed no differences as to the metabolic control of the diabetic state (basal glycemia, HbA1c, and basal insulin). R pts had a lower although ns glucose/insulin rate (23±2.1 vs 15.2±7 p=0.1), a significantly lower level of ApoA (1.3±0.2 vs 1.8±0.3 p=0.002), and higher levels of IGF-1 (221±43 vs 153±68 p=0.003). A significant correlation was found between ApoA and IGF-1 and the diameter of stenosis at follow-up QCA: r=0.469, p=0.016 and r=0.492, p=0.001 respectively. At multivariate analysis, ApoA and IGF-1 were confirmed as independent predictors of R.

Low levels of ApoA and high levels of IGF-1 may be involved in the mechanisms of in-stent R in NID diabetic pts. through the antiatherosclerotic effects of the former, and the proliferative effects of the later.

Key Words: PTCA, diabetes, coronary artery disease

P-466

ANG II INCREASES ATHEROSCLEROSIS IN APOE-DEFICIENT MICE: TEMPORAL DIFFERENCES INATHEROSCLEROSIS BETWEEN AORTIC ROOT AND DESCENDING AORTA

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Angiotensin II can affect almost every step in atherogenesis. Accordingly, a number of recent studies have shown that atherosclerosis in hyperlipidemic, apolipoprotein E-deficient (ApoE(0)) mice is augmented by angiotensin II infusion and reduced by angiotensin receptor blockers.

To determine the time course of the effects of Ang II we implanted osmotic minipumps in ApoE(0) mice to deliver saline or 1000 ng/kg/min Ang II from 4-16, 8-12, 16-20 and 16-32 weeks of age. Blood pressure was measured by tail-cuff every 2 weeks. Plasma renin concentration (PRC) was measured in orbital blood every 4 weeks. At the end of the study, the area of atherosclerotic lesions was measured at the aortic root and in the descending aorta.

In all mice treated with Ang II, systolic blood pressure increased by ~60 mm Hg and PRC was highly suppressed. In control mice, aortic root atherosclerosis increased steadily from ~40,000 μm² at 12 weeks to ~500,000 μm² at 32 weeks. Ang II infusion from 8-12 and from 4-16 weeks increased aortic root atherosclerosis 2.1- and 3.3-fold, respectively, whereas Ang II infusion from 16-20 and 16-32 weeks had no effect. In control mice lesion area of the descending aorta increased from 0.4% at 12 weeks to 2.2% at 32 weeks and Ang II infusion dramatically increased lesion area to 30-40% at all time points.

These observations demonstrate the potent atherogenic effects of Ang II and indicate that there are significant temporal and anatomical variations in the sensitivity of the vasculature to the effects of Ang II.

Key Words: Angiotensin, Atherosclerosis, Aorta

<table>
<thead>
<tr>
<th>patients</th>
<th>422</th>
<th>D/D (156)</th>
<th>I/D (205)</th>
<th>I/I (61)</th>
<th>(p)/D</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable angina</td>
<td>70 (39.7%)</td>
<td>81 (46%)</td>
<td>25 (14.3%)</td>
<td>0.628</td>
<td>0.58</td>
<td></td>
</tr>
<tr>
<td>Unstable angina</td>
<td>86 (35%)</td>
<td>124 (50.4%)</td>
<td>36 (16.6%)</td>
<td>0.602</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Key Words: ACE polymorphism, unstable angina, angina presentation of CAD in a selected population of symptomatic patients seems not to be associated with a higher incidence of an unstable and the D allele frequency (p) in the two groups are shown in the table.