P4.19: AORTIC STIFFNESS, REFLEXION WAVE AND ARTERIAL HYPERTENSION UNDER ANTI-ANGIOGENIC DRUGS

M.A. Alivon, J.G. Giroux, P.B. Boutouyrie, F.G. Goldwasser, S.L. Laurent


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P4.17  INFLUENCE OF ESTIMATED WALL SHEAR RATE INDICES ON CAROTID ARTERY INTIMA-MEDIA THICKNESS AND INTIMA-MEDIA COMPLEX ECHOCOGENCY

Peninsula Medical School, Peninsula NIHR Clinical Research Facility,
University of Exeter, Exeter, United Kingdom

Introduction: Grey scale median of the carotid artery intima-media complex (IM-GSM) is a recently introduced measurement to characterise the arterial wall. Wall shear stress is thought to influence intima-media thickness (IMT) and to play a major role in the development of atherosclerosis. However, the relationship between wall shear stress and IM-GSM is not well understood. This study examined the relationship between estimated wall shear rate (WSR) indices and IM-GSM as well as estimated WSR indices and IM-GSM.

Methods: Data from 156 middle-aged and older individuals (66.1 ± 6.7 years, 34.1% with hypertension, 18.8% with diabetes, 13.5% with hyperlipemia and 41.6% with obesity or overweight, in this study.

Data from 156 middle-aged and older individuals (66.1 ± 6.7 years, 34.1% with hypertension, 18.8% with diabetes, 13.5% with hyperlipemia and 41.6% with obesity or overweight, in this study.

Results: IMT and IM-GSM were independent determinants of WSR and mean WSR were independent determinants of IMT, and mean and diastolic WSR were independent determinants of IM-GSM after adjustments for potential confounders, OPT was independently associated with aortic PWV. There were significant correlations between WSR and IMT as well as estimated WSR and IM-GSM.

Conclusion: These results support the relationship between serum OPT and arterial stiffness in postmenopausal women, independent of the traditional cardiovascular risk factors and inflammation. At the same time, MGP was not found to be a predictor of arterial stiffness.

P4.19  AORTIC STIFFNESS, REFLEXION WAVE AND ARTERIAL HYPERTENSION UNDER ANTI-ANGIOGENIC DRUGS

M. A. Alivon 1, J. G. Giroux 2, P. B. Boutouyrie 1, F. G. Goldwasser 2, S. L. Laurent 1
1Inserm U970-Hôpital Européen Georges Pompidou, Paris, France
2Oncology Service- Hôpital Cochin, Paris, France

Objective: Sorafenib and Sunitinib are anti-angiogenic drugs (AAD) used in an increasing number of cancers. The most common side effect is arterial hypertension. We hypothesize that AAD lead to an early damage of large arteries which can be translated by an increase of aortic stiffness determined by the pulse wave velocity measurement (PWV).

Material and Method: In a longitudinal study, 32 patients have been treated with Sunitinib or Sorafenib. Subjects have been explored during a visit before the introduction of the treatment and then every two weeks for 2 months. Measured parameters are blood pressure, PWV, central pressure, augmentation index (Alx).

Results: 38% of the subjects have developed an early arterial hypertension requiring anti-hypertensive treatment. The initial values of brachial SBP was predictive from SBP changes under AAD while PWV and Alx was not. Furthermore, in patients who developed hypertension and required treatment with 5 to 10 mg amlodipine has been effective at decreasing SBP and Alx.

Conclusion: This study suggests that blood pressure at inclusion increases the risk of developing acute hypertension with AAD. It also shows that effective vasodilatation could be achieved despite small vessels disruption by AAD.

P4.20  ASSOCIATION OF A SINGLE NUCLEOTIDE POLYMORPHISM IN CYP2C8 WITH MYOCARDIAL INFARCTION IN BULGARIAN POPULATION

G. A. Atanasova 1, R. T. Tzveva 2, M. T. Tzvekova, assoc.prof3, R. K. Kaneva, assoc.prof4, V. M. Mitev 5
1Galva Naydenova, Pleven, Bulgaria
2Reni Tzvekova, Sofia, Bulgaria
3Maria Tzvekova, Pleven, Bulgaria
4Radka Kaneva, Sofia, Bulgaria
5Vanyo Mitev, Sofia, Bulgaria

Cytochrome P450 2C8 is a polymorphic enzyme responsible for the biosynthesis of vasoactive substances from arachidonic acid. Inter-individual differences in the action of these substances might be important in the pathogenesis of cardiovascular diseases such as acute myocardial infarction (AMI).

In the present study we analyzed the association of a genetic variant in CYP2C8 and the morbidity of AMI in Bulgarian population. The study included 99 AMI patients and 370 control subjects. To determine the genotypes of the samples real time PCR with predesigned TaqMan SNP Genotyping Assays (Applied Biosystem) was used.

<table>
<thead>
<tr>
<th>Hemodynamic variables by tertiles of HOMA Index*</th>
<th>T1 (&lt; 0.94) (n = 32)</th>
<th>T2 (0.94-1.90) (n = 28)</th>
<th>T3 (&gt; 1.90) (n = 30)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral SBP (mmHg)</td>
<td>111.7 ± 2.0</td>
<td>114.3 ± 1.8</td>
<td>123.2 ± 2.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Peripheral DBP (mmHg)</td>
<td>76.8 ± 1.4</td>
<td>79.8 ± 1.3</td>
<td>84.7 ± 1.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Peripheral PP (mmHg)</td>
<td>33.5 ± 1.0</td>
<td>35.0 ± 0.9</td>
<td>38.4 ± 1.0</td>
<td>0.004</td>
</tr>
<tr>
<td>Central SBP (mmHg)</td>
<td>103.7 ± 1.7</td>
<td>106.7 ± 1.5</td>
<td>115.3 ± 1.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Central DBP (mmHg)</td>
<td>77.8 ± 1.5</td>
<td>81.1 ± 1.3</td>
<td>86.1 ± 1.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Central PP (mmHg)</td>
<td>25.5 ± 1.5</td>
<td>28.1 ± 1.3</td>
<td>29.2 ± 1.0</td>
<td>0.071</td>
</tr>
<tr>
<td>MBP (mmHg)</td>
<td>92.5 ± 1.5</td>
<td>95.7 ± 1.3</td>
<td>102.3 ± 1.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>61.3 ± 1.9</td>
<td>65.0 ± 1.7</td>
<td>66.0 ± 1.9</td>
<td>0.213</td>
</tr>
<tr>
<td>PWV (m/s)</td>
<td>5.64 ± 0.17</td>
<td>5.71 ± 0.10</td>
<td>6.34 ± 0.9</td>
<td>0.012</td>
</tr>
<tr>
<td>Augmentation Index (%)</td>
<td>6.7 ± 3.2</td>
<td>6.1 ± 3.8</td>
<td>6.9 ± 2.2</td>
<td>0.082</td>
</tr>
</tbody>
</table>

*Values are expressed as means ± SEM