13.02: EXPOSURE TO URBAN AIR POLLUTANTS ALTERS ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS

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13.01 C-REACTIVE PROTEIN LEVELS ARE GRADUALLY ASSOCIATED WITH ADIPONECTIN AND ARTERIAL STIFFNESS IN NEWLY DIAGNOSED UNTREATED ESSENTIAL HYPERTENSIVE SUBJECTS: A UNIFYING APPROACH TO ATHEROSCLEROSIS

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Purpose: To examined the plausible correlations between hs-CRP levels, adiponectin and arterial stiffness in essential hypertensive patients.

Methods: In 148 newly diagnosed untreated non-diabetic essential hypertensive patients [96 men, mean age = 49 years, office BP = 150/97 mmHg], aortic stiffness was evaluated on the basis of carotid to femoral pulse wave velocity (PWV), by means of a computerized method (Complior SP). Venous blood samples were drawn for estimation of lipid profile and hs-CRP and adiponectin levels. All subjects according to hs-CRP values were divided into group A (hs-CRP < 1.29 mg/l), group B (hs-CRP = 1.3-2.39 mg/l) and group C (hs-CRP > 2.39 mg/l).

Results: Patients in group A (n = 51) compared to subjects in group B (n = 45) and C (n = 52) had lower office systolic BP and left ventricular mass index (P < 0.005 for all cases), while groups did not differ regarding lipid levels (P > NS). In the entire population, hs-CRP was positively associated with body mass index (r = 0.32, p < 0.001) and c-f PWV (0.412, p < 0.0001), while it was negatively correlated with adiponectin (r = -0.231, p < 0.005). Furthermore, patients in group C exhibited lower levels of adiponectin compared to group B and A (7.0±0.4 vs 8.9±5.1 vs 9.4±4.9 μg/ml, respectively; p < 0.05 for all cases) and more augmented PWV values (8.6±1.6 vs 8.2±0.9 vs 7.8±1.2 m/s, p<0.05, for all cases). Analysis of covariance revealed that adiponectin and PWV values remained different between groups after adjustment for confounding factors (p < 0.05).

Conclusions: Low-grade inflammation is associated in a graded fashion with proatherogenic processes linked with hypoadiponectinemia and arterial stiffening, even in the early stages of essential hypertension.

13.02 EXPOSURE TO URBAN AIR POLLUTANTS ALTERS ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS

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Exposure to urban air pollution, ultrafine particles or gas, is associated with acute cardiovascular mortality and morbidity. We investigated the effects of ambient air pollution on endothelial function in 40 healthy Caucasian men, previously described in the KLK study (JCI 2005), who spontaneously breathed ambient air pollution in Paris.

Endothelial function was measured by the % of brachial artery dilatation (dDr) and the absolute variation of shear stress (dSS) after hyperemia following 5 min hand ischemia and after 150 μg of TNT sublingual, using RF-bases echotracking device, at two visits 2 weeks apart. Air pollution level, (CO, NO, NO2, SO2, PM 2.5) were extracted from “Airparif” database, the day of echotracking. FDV was estimated by the slope of diameter-shear stress relationship. FDV was estimated by the slope of diameter-shear stress relationship.

Objective: To compare the acute flow-dependent vasodilatation (FDV) to a hand warming test (from 28°C to 44°C) and endothelium-independent vasodilatation (EIV) to sublingual glyceryl trinitrate (GTN 150 μg) of the brachial artery (BA) in 10 patients with an acute-phase Buerger disease, defined by an ADAR score ≥4 and a recent ulcer of the lower limbs (7 current), and 10 age- and sex-matched non-smokers healthy subjects.

Methods: BA diameter and shear stress were recorded by high definition echotracking. FDV was estimated by the slope of diameter-shear stress relationship.

Results: See the table.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median [IQR]</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buerger</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>FDV: Δ BA diameter (%)</td>
<td>6.8 (0.18;1.8)</td>
<td>3.7 (1.1;6.5)</td>
</tr>
<tr>
<td>Δ shear stress (%)</td>
<td>70 (32;100)</td>
<td>93 (57;125)</td>
</tr>
<tr>
<td>Δ BA diameter (mm)</td>
<td>30 (28;33)</td>
<td>17 (13;22)</td>
</tr>
<tr>
<td>Δ shear stress (%)</td>
<td>98 (40;137)</td>
<td>117 (76;251)</td>
</tr>
<tr>
<td>Δ slope diameter-shear stress (%)</td>
<td>0.89 (0.55;1.24)</td>
<td>0.28 (0.01;0.58)</td>
</tr>
</tbody>
</table>

Buerger’s patients had an enhanced flow-dependent response to the increase in shear stress due to hand warming by comparison with controls as shown by the higher slope of diameter-shear stress relationship.

Conclusions: Acute flow-mediated changes in brachial artery diameter during hand hyperemia and EIV to GTN were not impaired in patients compared to control, by contrast to what has been repeatedly suggested in the literature.

14.01 MOLECULAR DETERMINANTS OF ARTERIAL STIFFNESS

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Arterial stiffness has an independent predictive value for cardiovascular events. This review proposes an integrated view of the molecular determinants of arterial stiffness, based on a candidate gene approach, an analysis of the structure-function relationship in hypertension, and studies on gene expression profile in humans. In monogenic diseases of connective tissue (Marfan, Williams, and Ehlers-Danlos syndromes) and corresponding animal models, the precise characterization of arterial phenotype allows understanding the influence of abnormal, genetically-determined, wall components on arterial stiffness. These studies underline the role of extracellular matrix signaling in the vascular wall and the fact that elastin