P61: PSYCHOLOGICAL DETERMINANTS OF TARGET ORGAN DAMAGE IN HYPERTENSIVE PATIENTS: FOCUS ON TYPE A PERSONALITY AND LEFT VENTRICULAR MASS INDEX

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and diabetes. However, scanty data are available on the role of psychological factors on arterial stiffness. The aim of the current cross-sectional study was to evaluate the association between depression, anxiety, perceived stress, Type A personality, and Type D personality and Pulse Wave Velocity (PWV) in a cohort of hypertensive patients, using baseline examination data of the TIPICO project.

Methods: A total of 259 outpatients (ages 18–80 years) followed by the Hypertension Unit of S. Gerardo Hospital (Monza, Italy) affected by essential hypertension were recruited. Psychometric tests, clinical BP, and laboratory data were evaluated. Patients were asked to complete a battery of psychological questionnaires under the guidance of a psychologist.

Results: At T0 mean age was 55.9 ± 10.1 years, SBP and DBP were 135.6 ± 17.7 and 82.5 ± 9.1 mmHg and PWV was 8.6 ± 2.1 m/s. The multivariate stepwise linear regression analysis showed that age (beta = 0.284, p < 0.001), pulse pressure (beta = 0.369, p < 0.001), dyslipidemia (beta = 0.130, p = 0.012), family history of CV disease (beta = -0.123, p = 0.017), and depression (beta = 0.126, p = 0.014) were significantly and independently associated with PWV.

Conclusion: Among psychological factors, higher levels of Type-A personality is related to higher LVMI, while Type-D personality, anxiety, depression and stress are not associated.

P62
BLOOD PRESSURE LOWERING HALTS CAROTID ARTERY STIFFENING IN HYPERTENSIVE PATIENTS: THE CATOD STUDY
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Background: We anticipate that in vascular outpatients followed over time, measured changes in arterial stiffness will be the multifactorial result of measured pressure-decrease, age-related degeneration, wall stress homeostasis, and medical treatment. Carotid ultrasound enables assessment of carotid pulse wave velocity (cPWV, via Bramwell-Hill), geometry (relative wall thickness, RWT = 2*IMT/diameter), and intrinsic material stiffness (Young’s-modulus, via Moens-Korteweg). We investigated changes in these carotid properties over time, and their interrelationship. To check whether the change in measured cPWV could be merely due to pressure-dependence, we calculated based on the stiffness index [p2] the theoretical pressure-dependent change in cPWV [3].

Methods: Hypertensive outpatients (n = 147) were assessed at baseline and 3.5 ± 1.1 year follow-up, and were stratified according to baseline-to-follow-up change in diastolic blood pressure (∆DBP) into three groups: decreasedDBP (∆DBP < -7 mmHg), constantDBP (-7 mmHg ≤ ∆DBP ≤ 7 mmHg) and increasedDBP (∆DBP > 7 mmHg), with the cut-off being twice the typical DBP measurement error [1].

Results: The theoretical pressure-dependent change in cPWV was 0.4 ± 1.3 m/s lower (p < 0.001, n = 147), corroborating the anticipated multifactorial conditions. Table 1 shows no changes in cPWV, RWT and Young’s-modulus for decreasedDBP. For constantDBP, both cPWV and Young’s-modulus were increased at follow-up. IncreasedDBP showed increases in cPWV and Young’s-modulus and a decreased RWT. The latter implies a 9.2 ± 10.7 kPa increase in circumferential wall stress (p < 0.001, in contrast to a 5.3 ± 6.9 kPa decrease (p < 0.001) in decreasedDBP (p < 0.05 for between groups).

Table 1. Changes in measure carotid properties with 3.5-year follow-up.

<table>
<thead>
<tr>
<th></th>
<th>∆cPWV [m/s]</th>
<th>∆RWT [-]</th>
<th>∆Young’s-modulus [MPa]</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 147</td>
<td></td>
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<tr>
<td>DecreasedDBP</td>
<td>-0.1 ± 1.4</td>
<td>0.72</td>
<td>0.00 ± 0.04</td>
</tr>
<tr>
<td>ConstantDBP</td>
<td>+0.6 ± 1.4</td>
<td>&lt; 0.001</td>
<td>0.00 ± 0.04</td>
</tr>
<tr>
<td>IncreasedDBP</td>
<td>+0.5 ± 1.5</td>
<td>0.029</td>
<td>-0.02** ± 0.04</td>
</tr>
</tbody>
</table>

∆s calculated as follow-up — baseline. *: p < 0.05 compared to decreasedDBP. **: p < 0.05 compared to constantDBP.

Conclusions: In this outpatient cohort, with clear DBP reduction, there is a discontinuation of carotid stiffening, but no reversal. In patients with increased DBP, progressive carotid stiffening appears driven by impaired wall stress homeostasis.

References