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P2.39: INCREASED CARDIOVASCULAR RISK IN PATIENTS WITH A HYPERTENSIVE RESPONSE TO EXERCISE MAY BE EXPLAINED BY MASKED HYPERTENSION

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associated SNP (rs1081161) within the 9p21 region were genotyped. Aortic stiffness was decreased in male carriers of the rs10757274G and rs2891168G alleles. Accordingly, aortic compliance and distensibility were higher in men who carried the rs10757274G and rs2891168G alleles. Adjustment for age and mean arterial pressure had no effect on these associations. None of the SNPs were associated with either intima-media thickness or lumen diameter of the abdominal aorta. There were no associations between the T2D-associated rs10811661 SNP and any measure of aortic stiffness.

Impaired mechanical properties of the aortic wall may be a link between the association between chromosome 9p21 polymorphisms and vascular disease.

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INCREASED CARDIOVASCULAR RISK IN PATIENTS WITH A HYPERTENSIVE RESPONSE TO EXERCISE MAY BE EXPLAINED BY MASKED HYPERTENSION

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Background: A hypertensive response to exercise (HRE; defined as normal clinic blood pressure [BP] and exercise BP >210/105mmHg in men or >190/105mmHg in women) independently predicts incident hypertension and cardiovascular mortality. The mechanisms remain unclear but may be related to masked hypertension. This study aimed to assess the prevalence of masked hypertension and cardiovascular risk factors, including aortic reservoir function, in patients with a HRE.

Methods: Comprehensive clinical and echocardiographic evaluation (including central BP, aortic reservoir pressure, aortic pulse wave velocity by tonometry) and 24 hour ambulatory BP monitoring (ABPM) were performed in 81 untreated patients with HRE (aged 54±9 years; 60% male; free from coronary artery disease). Masked hypertension was defined as ABPM systolic BP (SBP) ≥130 mmHg and clinic BP <140/90 mmHg.

Results: Masked hypertension was present in 50 patients (62%). These patients had higher left ventricular (LV) mass index (92.1±17.8 g/m² versus 77.2±17.9g/m²; p=0.01) aortic reservoir pressure (104±9mmHg versus 97±10mmHg; p=0.001) and exercise SBP (226±15mmHg versus 210±15mmHg; p<0.001), despite no significant difference in aortic pulse wave velocity or central pulse pressure (p>0.05 for both). Aortic reservoir pressure was significantly correlated with peak exercise SBP (r=0.34; p=0.002). The strongest independent determinant of LV mass index was the pressure of masked hypertension (β=0.37; p=0.001).

Conclusions: Aortic reservoir pressure is significantly elevated, and masked hypertension highly prevalent in HRE patients with a normal resting office BP. This may help to explain increased risk in patients with a HRE and clinicians should suspect masked hypertension in this population.

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P2.40

CENTRAL AORTIC PRESSURE IS A BETTER DETERMINANT OF ANTI-HYPERTENSIVE RESPONSE THAN BRACHIAL PRESSURE IN YOUNG HYPERTENSIVE PATIENTS

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Aortic blood pressure (BP) has been shown as a stronger predictor of target organ damage than brachial BP. Whether pre-treatment aortic BP is a better determinant of early anti-hypertensive response than brachial BP is not known.

We analysed the pre and post treatment haemodynamic data on untreated hypertensive subjects (n=290) aged 51±0.6 years, who had received in random fashion ; ACE inhibitors, angiotensin receptor blockers, calcium antagonists & nebivolol (vasodilators) & non-vasodilating agents ; atenolol & thiazide diuretics. Data were analysed using JMP Version 7.1.

Baseline aortic systolic BP showed a better correlation with reduction in either brachial or aortic systolic BP compared with brachial systolic BP. In subjects < 50 years, baseline aortic systolic BP showed a stronger correlation with brachial systolic response (r=0.38, p<0.0001) than brachial systolic BP (r=0.28, p<0.001). Baseline brachial systolic BP showed a poor correlation with reduction in aortic systolic (r=0.19, p<0.05) compared with baseline aortic systolic BP (r=0.37, p<0.0001). In those >50 years, pre-treatment brachial (r=0.46, p<0.0001) and aortic (r=0.47, p<0.0001) systolic BP showed similar correlations. Baseline aortic systolic BP showed a better correlation with BP response to vasodilating anti-hypertensives

(r=0.42, p<0.0001) than non-vasodilator agents (r=0.36, p<0.0001). In multiple regression analysis, gender, vasodilator agents and baseline aortic systolic BP emerged as independent determinants of brachial BP response with no significant contribution from brachial systolic BP.

Aortic BP is a better determinant of BP response than brachial BP, especially in young hypertensive patients and particularly to vasodilating antihypertensives and may guide choice of initial anti-hypertensive agent in the young patient.

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P2.41

PULSE WAVE VELOCITY CORRELATES WITH LEFT VENTRICULAR SYSTOLIC FUNCTION IN NEVER-TREATED ESSENTIAL HYPERTENSIVES

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Introduction: Hypertension is associated with increased arterial stiffness. Arterial stiffness, which is a predictor of cardiovascular risk, has been shown to correlate with diastolic dysfunction of left ventricle in hypertensive patients.

Hypothesis: We assessed the hypothesis that arterial stiffness is associated with left ventricular systolic function in never-treated hypertensive patients.

Methods: We enrolled 195 consecutive essential hypertensives (mean age 50±12 years) with preserved left ventricular ejection fraction (LVEF>45%). Arterial stiffness was determined with carotid-femoral pulse wave velocity (PWV). LVEF was measured echocardiographically and calculated using the Teichholz method.

Results: Subjects were divided into tertiles according to PWV. There was a correlation of PWV tertiles with age (44±13 vs 49±11 vs 57±9 years at the 1st, 2nd and 3rd tertile, respectively). Mean blood pressure was similar across the tertiles (p=NS). We observed a stepwise decrease of LVEF with increasing PWV. (Figure) Multivariable regression analysis showed that the inverse correlation of LVEF with PWV was independent of age, sex and mean blood pressure (p=0.028, adjusted R² of model=0.241).

Conclusion: Higher PWV is an independent predictor of a lower LVEF in never-treated essential hypertensives. This finding provides further insights into the role of arterial stiffness in left ventricular function.

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P2.42

POSTURAL CHANGES HAVE A DIFFERENTIAL RESPONSE ON BRACHIAL, COMPARED WITH CENTRAL, SYSTOLIC BLOOD PRESSURE IN PATIENTS WITH HYPERTENSION

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Background: Clinic brachial blood pressure (BP) is typically recorded in the seated, supine and standing positions. However, it is unknown whether central BP may be differentially altered with postural changes, and this may have treatment implications. This study aimed to assess brachial and central BP during different postures in patients with hypertension compared with controls.

Methods: Study population comprised 41 patients with hypertension receiving medication (HTN; aged 60±7 years; 22 male), 26 untreated patients with masked hypertension (MaskHTN; 57±9 years; 19 male) and 36 normotensive controls (aged 54±9 years; 22 male). The average of two brachial and central BP's (by radial tonometry; SphygmoCor) were recorded in the seated, supine (after 3-5 minutes) and standing (after 2 minutes) positions.

Results: Supine brachial systolic BP (SBP) was significantly higher in patients with HTN (127±12 mmHg) and MaskHTN (130±10 mmHg) compared with controls (120±13 mmHg; p<0.05). As expected for the controls, seated brachial SBP was slightly, but not significantly (p>0.05), higher than both supine and standing positions. This non significant pattern was similar for central SBP in the controls and MaskHTN patients, but not patients with HTN, whose standing central SBP (109±12 mmHg) was significantly lower compared with the supine position (116±14 mmHg; p<0.05).

Conclusion: Posture has a differential effect on central, compared with brachial SBP in patients with treated hypertension. This highlights the importance of assessing central BP in these people, which may be particularly useful for managing patients with symptoms related to orthostatic hypotension.

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