P2.04: CALCIUM INTAKE IS INDEPENDENTLY ASSOCIATED WITH INCREASED AUGMENTATION INDEX: RESULTS FROM A CROSS-SECTIONAL FOLLOW-UP STUDY OF TWO RHEUMATOID ARTHRITIS COHORTS


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aortic blood pressures were higher in female carriers of the 4 G/4 G genotype than female subjects carrying the 4 G/5 G and 5 G/5 G genotypes, (P = 0.014, P = 0.004 and P = 0.003 for central systolic-, diastolic- and mean arterial pressure, respectively). Adjustment for variables related to hypertension (age, BMI, DM, smoking, LDL-cholesterol, fasting glucose) had no effect on the associations. No association was found between PAI-1 genotype and brachial blood pressure in either men or women. Our findings show that the PAI-1 (4 G/5 G) polymorphism is associated with central arterial blood pressure in women. The genotype effect was independent of other risk factors related to hypertension, suggesting that impaired fibrinolytic potential may play an important role in the development of central arterial hypertension.

P2.06 ENDOTHELIAL DYSFUNCTION IS ASSOCIATED WITH ARTERIAL STIFFNESS IN HYPERTENSIVE PATIENTS WITH TYPE 2 DIABETES MELLITUS


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Both endothelial dysfunction and arterial stiffness are considered as independent predictors of cardiovascular mortality, but their interrelationship has been poorly explored. Therefore we evaluated the relationship between endothelial function and pulse wave velocity (PWV) in essential hypertensive patients with (DM+) or without (DM-) diabetes mellitus, on chronic pharmacological treatment.

51 DM+ patients and 51 DM- patients matched for age, gender and BP were included. Arterial and brachial PWV were measured by high resolution ultrasound and computerized edge detection system.

In DM+ PWV was higher (9.9 ± 1.8 vs 8.4 ± 1.4 m/s, p = 0.001) and PWV was related to BMI (r² = 0.45, p = 0.0006), systolic BP (r = 0.54, p = 0.0006) and BMI (r = 0.47, p = 0.0007), triglycerides (r = 0.54, p = 0.0006), systolic BP (r² = 0.17, p = 0.002), BMI (r² = 0.22, p = 0.009) resulted significant independent predictors of PWV. In the contra in DM-, PWV was related to age (r² = 0.35, p = 0.01), systolic BP (r² = 0.45, p = 0.001), but not to BMI. In multivariate analysis, only SBP (r = 0.02) was an independent predictor of PWV (r² = 0.23).

In hypertensive type 2 diabetic subjects on chronic pharmacological treatment, a decline in endothelial function seems to be independently associated with increased aortic stiffness, possibly suggesting a cause-effect mechanism. The association is not present in hypertensive euglycemic patients, who have lower aortic stiffness and endothelial dysfunction.

P2.07 MASKED HYPERTENSION IS “UNMASKED” BY LOW INTENSITY EXERCISE BLOOD PRESSURE

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Background: Masked hypertension (MH) independently predicts mortality but cannot be diagnosed from clinic blood pressure (BP). We sought to determine if MH could be identified from BP or pressure waveform analysis (PWA) at rest or during low intensity exercise.

Methodology: Brachial and estimated central BP (by PWA: Sphygmocor) were recorded at rest and during 10 minutes of cycling exercise (60-70% of age-predicted maximal heart rate) in 77 untreated subjects with a hypertensive response to exercise (HRE) (aged 54 ± 8years) and 61 patients with hypertension (HT) receiving therapy (aged 62 ± 8years). All subjects underwent 24 hour ambulatory BP monitoring (24hrABPM) and MH was defined as clinic systolic BP (SBP) <140mmHg and 24ABPM SBP >130mmHg.

Results: There were 44 (57%) HRE and 32 (52%) HT patients with MH. For the HRE group at rest, there were no significant differences between MH and normotensive subjects in any haemodynamic variable except brachial systolic BP, which was higher in MH subjects (127 ± 9 vs. 120 ± 10mmHg; p < 0.05). After correction for resting SBP, MH subjects had significantly higher brachial (187 ± 22 vs. 168 ± 15mmHg; p = 0.05) and central SBP (154 ± 17 vs. 141 ± 12mmHg; p < 0.05) during exercise, with greater changes in both from baseline (p < 0.05). No differences were observed in the HT group. Exercise brachial SBP predicted the presence of MH independent from all resting hemodynamic variables (β = 0.35; p < 0.001), and if >190mmHg, identified MH with 97% specificity (p < 0.001).

Conclusions: MH can be identified in untreated individuals from low intensity exercise brachial SBP. Exercise BP testing may be indicated in patients with borderline raised clinic brachial SBP.