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

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aortic blood pressures were higher in female carriers of the 4G/4G genotype than female subjects carrying the 4G/5G and 5G/5G 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 (4G/5G) 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.

#### P2.04

### 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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**Background:** Population studies have indicated that both the presence of osteoporosis and serum levels of calcium are associated with an increased risk of cardiovascular disease.

**Objective:** To investigate the association between bone loss, calcium intake and levels of the reactive hyperemia index (RHI) and augmentation index (Alx), two measures of endothelial function and surrogate markers of cardiovascular disease, in a cohort of patients with RA.

**Methods:** Two hundred and thirty eight patients with early RA were comprehensively examined at baseline with registration of clinical and radiographic data. At the 15-year follow-up these examinations were repeated in 153 patients and additionally the RHI (ITAMAR) and Alx (Sphygmocor) were recorded.

**Results:** Calcium substitution, ever vs never was associated with lower levels of RHI and higher Alx  $\beta$ (SE) -0.12 (0.05)  $p = 0.03$  and 4.03 (1.19)  $p = 0.001$  respectively, in models that were adjusted for age, sex and CVD risk factors. Measures of bone mineral density or rate of bone loss were not significantly related to Alx or RHI. In models that were adjusted for current CVD risk factors, RA disease activity and use of disease modifying anti-rheumatic drugs, current use of calcium substitution was a significant independent predictor of Alx  $\beta$ (SE) 5.21 (2.38)  $p = 0.03$ , model  $r^2$  0.56, but not of RHI.

**Conclusion:** Calcium supplementation was associated with increased arterial stiffness in this cohort of patients with RA. Residual confounding cannot be ruled-out.

#### P2.05

### CENTRAL HEMODYNAMIC PARAMETERS AND ARTERIAL STIFFNESS IN PAGET'S DISEASE

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**Background:** Paget's disease of bone (PDB) is a common disorder characterised by increased, disorganised bone turnover in affected areas with overgrowth of immature woven bone. An increased cardiovascular risk has been reported in some studies of PDB, possibly related to arterial wall calcifications. The aim of this study was to evaluate central hemodynamic parameters and arterial stiffness in PDB patients.

**Methods:** 11 PDB patients and 11 control subjects matched by age, weight, height and cardiovascular risk factors, were enrolled. Anthropometric measures, metabolic profile and information about PDB features (extension, therapy, and genetic alteration of disease) were made available for all subjects. Central hemodynamic parameters and carotid-femoral Pulse Wave Velocity (PWVcf) were assessed non-invasively by Sphygmocor.

**Results:** The PDB and the control group were comparable for age (PDB vs Control:  $59 \pm 8.0$  vs  $58 \pm 7.2$  yrs;  $M \pm SD$ , height:  $168 \pm 6.7$  vs  $168 \pm 6.0$  cm and body weight:  $78 \pm 14$  vs  $77 \pm 12$  kg). PDB patients presented a significantly higher PWVcf ( $9.8 \pm 1.8$  vs  $7.7 \pm 1.5$  m/s,  $p = 0.008$ ) and a trend toward higher central PP ( $48 \pm 19$  vs  $45 \pm 10$  mmHg,  $p = 0.60$ ). The difference in PWVcf was confirmed after adjustment for age, gender, heart rate and central mean pressure ( $9.6 \pm 0.54$  vs  $7.18 \pm 0.53$  m/s  $M \pm SE$ ;  $p = 0.006$ ).

**Conclusions:** PDB patients presented evidence of higher arterial stiffness as compared with control subjects. These results support the hypothesis

that PDB is a systemic condition associated with increased cardiovascular risk.

#### 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. Applanation tonometry (Sphygmocor®) was used to determine aortic (carotid to femoral) PWV. Brachial artery endothelium-dependent flow-mediated dilation (FMD), and endothelium-independent dilation by 25  $\mu$ g sublingual glycerol trinitrate (GTN) were assessed by high resolution ultrasound and computerized edge detection system.

In DM+ PWV was higher ( $9.9 \pm 1.8$  vs  $8.4 \pm 1.4$  m/s,  $p = 0.0001$ ) and FMD was lower ( $3.1 \pm 1.7$  vs  $6.3 \pm 3.5$ ,  $p < 0.0001$ ) than DM-. In DM+, PWV was related to BMI ( $r = 0.47$ ,  $p = 0.0007$ ), triglycerides ( $r = 0.54$ ,  $p = 0.00006$ ), systolic BP ( $r = 0.54$ ,  $p = 0.00006$ ) and FMD ( $r = -0.47$ ;  $p = 0.0005$ ). In multivariate analysis, only FMD ( $r^2 = 0.10$ ,  $p = 0.003$ ), systolic BP ( $r^2 = 0.17$ ,  $p = 0.002$ ), BMI ( $r^2 = 0.22$ ,  $p = 0.009$ ) resulted significant independent predictors of PWV. On the contrary in DM-, PWV was related to age ( $r^2 = 0.35$ ,  $p = 0.01$ ), systolic BP ( $r^2 = 0.45$ ,  $p = 0.001$ ), but not to FMD. In multivariate analysis, only SBP ( $p = 0.02$ ) was an independent predictor of PWV ( $r^2 = 0.23$ ).

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  $\approx 10$  minutes of cycling exercise (60-70% of age-predicted maximal heart rate) in 77 untreated subjects with a hypertensive response to exercise (HRE) (aged  $54 \pm 8$  years) and 61 patients with hypertension (HT) receiving therapy (aged  $62 \pm 8$  years). All subjects underwent 24 hour ambulatory BP monitoring (24hrABPM) and MH was defined as clinic systolic BP (SBP)  $< 140$  mmHg and 24hrABPM SBP  $\geq 130$  mmHg.

**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 ( $127 \pm 9$  vs.  $120 \pm 9$  mmHg;  $p < 0.05$ ). After correction for resting SBP, MH subjects had significantly higher brachial ( $187 \pm 22$  vs.  $168 \pm 15$  mmHg;  $p < 0.05$ ) and central SBP ( $154 \pm 17$  vs.  $141 \pm 12$  mmHg;  $p < 0.05$ ) 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 ( $\beta = 0.35$ ;  $p < 0.001$ ), and if  $> 190$  mmHg, 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.