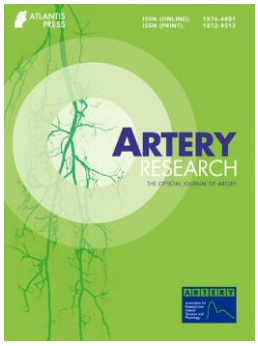

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P4.07: DIFFERENT IMPACT OF HYPERTENSION AND TYPE 2 DIABETES ON AORTIC, CAROTID AND PERIPHERAL VASCULAR STIFFNESS

R.M. Bruno, E. Bianchini, L. Landini, G. Cartoni, F. Stea, G. Penno, S. Del Prato, S. Taddei, L. Ghiadoni

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Conclusion: Higher apolipoprotein B/A-I and total /HDL cholesterol ratios are independent predictors of increased arterial stiffness in never-treated hypertensives and predict increased arterial stiffness better than LDL.

P4.03
WOMEN WITH SYSTEMIC SCLEROSIS HAVE WORSE ENDOTHELIAL FUNCTION AS COMPARED TO WOMEN WITH RHEUMATOID ARTHRITIS AND SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: The endothelial function has been repeatedly shown to be damaged in rheumatoid arthtritis (RA), systemic lupus erythematosus (SLE) and systemic sclerosis (SSc) patients. However, it is uncertain which of diseases has the greatest influence on endothelial function.
Methods: We studied 60 women with RA (aged 40.47 ± 9.93 years) with disease activity (DAS28) 5.47 ± 0.94, 30 women with SLE (aged 37.33 ± 9.22 years), disease activity index (SLEDAI) 18.40 ± 8.17, organ damage index (SLICC) 1.27 ± 1.53 and 16 women with diffuse cutaneus SSc (aged 47.94 ± 10.46 years). Blood tests included serum lipid profile, glucose and high-sensitivity CRP (hsCRP) measurement. The endothelium-dependent flow-mediated dilatation (FMD) test in a brachial artery was performed by the ultrasound system (Logiq 700, General Electric).
Results: Direct comparison of FMD in RA, SLE and SSc groups has shown that there was overall difference between groups ($p = 0.033$). This result was confirmed by application of linear models with adjustment for other confounding factors. FMD significantly differed in SSc and RA groups (5.69 ± 5.10 % vs. 8.37 ± 3.84 %; $p = 0.021$; $\beta = -0.2$) as well as in SSc and SLE groups (5.69 ± 5.10 % vs. 9.25 ± 5.15 %; $p = 0.021$; $\beta = -0.2$).
Conclusion: Results of our study have shown that FMD is lower in SSc group as compared to RA and SLE groups, thus the SS does greater damage on endothelial function as compared with RA and SLE.

P4.04
IMPAIRED ABDOMINAL AORTIC WALL INTEGRITY IN ELDERLY MEN CARRYING THE ANGIOTENSIN-CONVERTING ENZYME D-ALLELE

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Objective: A genetic polymorphism in the angiotensin-converting enzyme (ACE) gene (ACE I/D polymorphism) has been associated with abdominal aortic aneurysm. A link between aortic aneurysm and aortic stiffness has been suggested. However, no study has previously investigated the relationship between ACE and abdominal aortic wall integrity. The aim of this study was to explore the links between ACE I/D polymorphism, circulating ACE, and abdominal aortic wall integrity determined by abdominal aortic wall stiffness.
Methods: The study population consisted of 406 subjects (212 men and 194 women), 70-88 years. Diastolic lumen diameter, pulsatile diameter change and intima-media thickness were measured and used to calculate the cross-sectional compliance (CC), distensibility coefficient (DC), stiffness β and wall stress. ACE genotype was determined by PCR followed by gel electrophoresis, and circulating ACE level was measured using ELISA.
Results: Male carriers of the ACE D allele had a lower distensibility coefficient than II carriers (ID/DD 8.09 vs. II 10.38, $p=0.017$). Multiple regression analyses adjusting for confounding factors showed significant associations between the ACE D-allele and increased stiffness β as well as reduced CC. No significant association between abdominal aortic stiffness and the ACE D-allele was found in women.
Conclusion: This study showed, for the first time, a gender-specific association between the ACE-D allele and abdominal aortic wall mechanics, with men carrying the ACE D-allele having stiffer abdominal aortas compared to II carriers. Increased abdominal aortic stiffness indicates impaired vessel wall integrity, which along with other local predisposing factors, may increase the risk of aneurysm formation.

P4.05
DERANGED ARTERIAL WALL REMODELLING IN CENTRAL ARTERIES OF PATIENTS WITH ABDOMINAL AORTIC ANEURYSMS – A REASON FOR THE HIGH COMORBIDITY/MORTALITY IN CARDIOVASCULAR DISEASE?

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Objectives: Patients with abdominal aortic aneurysm (AAA) have a high cardiovascular comorbidity besides the risk of aneurysmal rupture, the reasons being largely unknown. The aim of this study was to determine other possible vascular defects in the arterial system besides aneurysmal disease that might be of relevance for the increased comorbidity.
Methods: 23 male AAA-patients and 20 age-matched controls (C) were examined with tonometry using the Sphygmocore system, to determine aortic and brachial PWV. The relation between local common carotid artery (CCA) pressure and lumen diameter (LD) as well as IMT determined by ultrasound was studied as a measure of remodelling capacity of the carotid wall.
Results: AAA:s had higher aoPWV than C (11,2±2 vs 9,9±2 m/s, $p=0.03$). No difference in bPWV was seen. An increased LD in CCA was found in the AAA:s (7,5±1,3 vs 6,5±0,7 mm respectively, $p<0,001$). In C there was a positive correlation between local pulse pressure (LPP) and CCA IMT ($r=0,49$, $p<0,001$), which was not seen in the AAA ($r=0,03$, NS). The expected negative correlation between LPP and LD/IMT ($r=-0,44$, $p=0,004$) indicating a remodelling response in the CCA was found in C but not in AAA ($r=-0,25$, NS).
Conclusion: A changed aortic wall structure in central elastic arteries with high PWV in patients with AAA is seen. Further, the remodelling of the common carotid artery in response to local pulse pressure is defect. This form a background explaining the high cardiovascular comorbidity besides the risk of aneurysmal rupture in these patients.

P4.06
ENDOTHELIAL VASOMOTOR FUNCTION AND ARTERIAL STIFFNESS IN YOUNG MEN WITH ARTERIAL HYPERTENSION, GRADE I, AND THEIR RELATION WITH RED BLOOD CELL DISTRIBUTION WIDTH

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Endothelial dysfunction and arterial stiffness increasing are negative prognostic markers in arterial hypertension. Red blood cell distribution width (RDW) was shown as a new negative prognostic marker in some cardiovascular diseases.
Objective: To study the relation between endothelial function and RDW, arterial stiffness and RDW in young men with arterial hypertension.
Materials and Methods: 54 men, 29,9±0,9 ys (M±SE), with arterial hypertension, Grade I were examined. Flow-mediated vasodilatation (FMD) was assessed by Vivid 7 (GE), arterial stiffness by brachial-ankle pulse wave velocity (PWV) (Vasera VS-1000, Fukuda Denshi), RDW by Cell Din 3500 (Abbot).
Results: FMD ranged from 18,4% to -1,8 % (6,8 ±0,7%), PWV from 9,7 m/s to 16,3 m/s (12,1 ±0,2 m/s), RDW from 12,3% to 16,2 % (14,2 ±0,1%). Endothelial dysfunction was detected in 46% patients, increasing the arterial stiffness in 61% and elevated RDW in 37%. Patients with elevated RDW had lower FMD (4,6±0,4 % vs 8,0±0,4 %, $p=0,01$), more prevalence of endothelial dysfunction (70 % vs 33 %, $p=0,01$) and higher values of PWV (12,6±0,3m/s vs 11,9±0,2 m/s, $p=0,04$) in comparison with patients with normal RDW. RDW correlated with FMD: $r = -0,4$, $p=0,01$. Chance of endothelial dysfunction in young men with arterial hypertension and elevated RDW is more in 5 times.
Conclusion: Endothelial dysfunction, increased arterial stiffness and elevated RDW have high prevalence in young men with arterial hypertension. Elevated RDW is accociated with endothelial dysfunction and increased arterial stiffness and may be considered as a perspective prognostic marker in arterial hypertension.

P4.07
DIFFERENT IMPACT OF HYPERTENSION AND TYPE 2 DIABETES ON AORTIC, CAROTID AND PERIPHERAL VASCULAR STIFFNESS

R. M. Bruno [1], E. Bianchini [2], L. Landini [1], G. Cartoni [1], F. Stea [1], G. Penno [1], S. Del Prato [1], S. Taddei [1], L. Ghiadoni [1]
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Aim of the study was to evaluate the impact of diabetes, hypertension, and their combination on aortic, carotid and peripheral stiffening.

139 subjects (31 normotensives-NT, 38 hypertensives-HT, 24 diabetic normotensives-DMNT, and 46 diabetic hypertensives-DMHT) were enrolled. Applanation tonometry (Sphygmocor) was used to measure aortic (carotid to femoral) and peripheral (carotid to radial) pulse wave velocity (aPWV and pPWV respectively). Common carotid stiffness (CS) was determined using the real-time computerized echo-tracking system "Carotid Studio".

pPWV was similar in the four groups. aPWV significantly increased from NT (7.3±1.1m/s) to HT (8.2±1.4 m/s) and DMNT (8.3±1.3 m/s), reaching the highest value in the DMHT group (10.8±1.9 m/s). CS was similar in NT (6.1±0.8m/s) and DMNT (6.4±1.1 m/s), and significantly increased in HT and DMHT (6.7±1.2m/s, 7.4±1.2m/s).

In a model adjusted for age, gender and diabetes, the presence of hypertension carried a 5.6-fold (confidence interval-95% 1.9-16.5) increased risk of having aPWV above the median value of 8.5 m/s, and a 4.5-fold (1.7-11.9) increased risk of having CS (above median value of 6.6 m/s). The presence of diabetes carried a 7.2-fold (2.8-18.9) increased risk of having increased aPWV, while the analysis was not significant for carotid stiffness (OR 2.1, 0.9-4.7).

In conclusion, increased aPWV is present in both diabetic and hypertensive patients, and the combination of the two risk factors induces an even greater aortic stiffening. While hypertension is characterized by vascular stiffening at both the aortic and carotid level, carotid stiffening in diabetic patients appears to be mostly dependent on the concomitant presence of hypertension.

P4.08
AUGMENTATION INDEX AND AORTIC DISEASE IN MARFAN-LIKE SYNDROMES

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The augmentation index (AIx@HR75) and central pulse pressure (CPP) can be measured noninvasively with applanation tonometry (APT). In this observational study, we investigated the relationship between AIx@HR75, CPP and aortic disease in Marfan-like syndromes.

Methods: We performed APT in 78 consecutive patients in whom classic Marfan syndrome (MFS) had been excluded (46 men and 32 women aged 34 ±13 years). These patients comprised 9 persons with MFS-like habitus, 6 with a bicuspid aortic valve (BAV), 5 with MASS phenotype, 3 with vascular type of Ehlers–Danlos syndrome (EDS), 3 with familial thoracic aortic aneurysm, 2 with Loeys–Dietz syndrome (LDS), 1with mitral valve prolapse syndrome, 1 with familial ectopia lentis,and 48 persons with Marfan-like features but no defined syndrome. During 20 ± 18 months after APT, we observed progression of aortic diameters in 15 patients, and aortic surgery or aortic dissection in 3 individuals.

Results: All 11 patients with Marfan-like syndromes and progression of aortic disease exhibited AIx@HR75 ≥11%, including 8 individuals with aortic diameters ≤95th percentile of normal at baseline. Similarly, all 7 individuals without any defined syndrome but progression of aortic diameters exhibited AIx@HR75 >11%, including 6 individuals with aortic diameters ≤95th percentile at the time of APT. Aortic disease did not evolve at AIx@HR75 <11%. CPP is also related to disease progression.

Conclusions: Aortic disease evolution relates to AIx@HR75 and CPP in Marfanlike syndromes. Larger studies will be required to establish APT for prediction of aortic disease evolution in Marfan-like syndromes.

P4.09
THE INCREASED TRANSMURAL PRESSURE CHANGES A PATTERN OF DILATION REACTION OF MAGISTRAL ARTERIES

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Objective: In our previous investigations it was shown that dilatation of magistral arteries (MA) after arterial occlusion (AO) occurs only after the restoration of free blood flow and manifests as an increment of blood flow in the beginning of reactive hyperemia (RH). The aim of this study was to examine whether the increased transmural pressure ($P_{transm.}$) can modulate dilatation reaction of MA after AO.

Design and methods: The investigation was performed on 12 healthy subjects aged between 28-40 yrs. Dilation of MA was appraised from changes in blood flow (I) and haemodynamic resistance (R) in the forearm during RH in two different situations - after 7 min. AO and after 7 min. AO with previous venous congestion (VC). Forearm I was determined by venous occlusion plethysmography. R was calculated from data on I and mean arterial pressure. Systemic arterial pressure was determined auscultatory.

Results: VC before AO of 7 min. abolished the increment of blood flow during RH - maximal I and minimal R was observed just after restoration of free blood flow and normalisation rate of I and R was more rapid than that after 7 min. AO. Maximal I was greater after 7 min. AO with previous VC than that after 7 min. AO (42±1,4 and 33±1,6 ml/100cm^3/min., correspondingly) and minimal R was smaller after 7 min. AO with previous VC than that after 7 min. AO (2,25±0,1 and 2,7±0,2 PRU_{100}, correspondingly).

Conclusion: The increase of $P_{transm.}$ changes a patern of dilatation reaction of forearm magistral arteries.

P4.10
INFLAMMATION IS NOT RELATED TO ABDOMINAL AORTIC ANEURYSM DIAMETER

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Background: Chronic inflammation is a hallmark characteristic in the pathophysiology of abdominal aortic aneurysms (AAA). In this study we sought to prospectively investigate whether inflammation was related to aortic diameter. Given that diameter has been shown to be the best predictor of AAA expansion rate, we hypothesised that larger aneurysms would have the greatest degree of inflammation.

Methods: 34 patients with AAA were imaged using fluoro-deoxyglucose positron emission tomography (FDG-PET) with CT co-registration. Uptake of FDG, which directly reflects metabolic activity and is shown to correlate with macrophage activity, was quantified as a tissue to background ratio (TBR). A TBR was calculated for the entire aorta and for the ascending, descending, abdominal and aneurysmal segments. TBR values were correlated with AAA diameter as measured on CT images. Inflammatory biomarkers, including hsCRP were also measured.

Results: The mean aneurysm diameter was 44mm (SD+/-9mm). There was no correlation between aortic diameter and TBR within the aneurymal sac ($r=0.021$, $p=0.93$). The AAA diameter also did not correlate with abdominal aortic TBR ($r=0.144$, $p=0.54$) or the total aortic TBR ($r=0.058$, $p=0.844$). There was a modest but statistically non-significant correlation between AAA diameter and hsCRP ($r=0.424$, $p= 0.12$).

Conclusions: There was no relationship between FDG uptake, a surrogate of inflammation, and AAA diameter. However, this may indicate that inflammation is a potential predictor of AAA growth rate independently of AAA diameter.

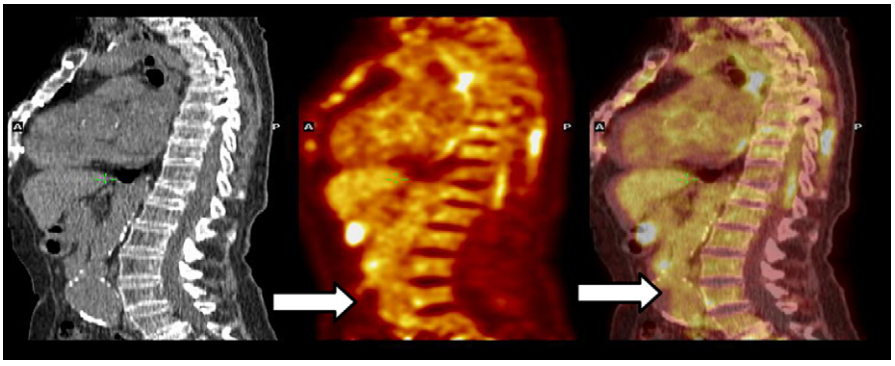

Figure 1 Image of CT, PET and co-registered image showing inflamed AAA.