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P2.12: THE INTERPLAY OF ENDOTHELIAL FUNCTION, INFLAMMATORY AND OXIDATIVE STATUS IN HIV INFECTION. DOES ANTIRETROVIRAL THERAPY PLAY A ROLE?

P. Xaplanteris, C. Vlachopoulos, E. Mariolis, E. Sambatakou, D. Terentes-Printzios, N. Ioakeimidis, D. Kardara, A. Synodinos, C. Stefanadis

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endothelial function, arterial stiffness, and renal vasodilating response to glyceryl trinitrate (GTN), a new parameter of renal vascular damage, in lean, normotensive patients with OSA.

Methods: 17 lean normotensive patients with moderate-severe OSA (AHI 31±19), and 21 matched healthy controls were recruited. Renal resistive index (RI) was obtained by Duplex ultrasound at baseline and after sublingual GTN (25 µg), evaluating renal vasodilation as percent RI change. Endothelium-dependent (flow-mediated-dilation, FMD) and -independent (response to GTN) vasodilation in the brachial artery was assessed by computerized edge detection system. Arterial stiffness was assessed as carotid-femoral pulse wave velocity (PWV).

Results: OSAS patients and controls presented similar RI (0.61 vs 0.59, $p=ns$), but impaired renal vasodilation to GTN ($-5.7\pm6.2\%$ vs $-10.3\pm4.6\%$, $p<0.05$). FMD was reduced ($4.1\pm2.5\%$ vs $6.2\pm3.1\%$, $p<0.05$), while endothelial-independent brachial artery vasodilation was preserved. PWV was not different between OSAS and controls (7.9 ± 1.5 vs 7.7 ± 1.4 m/s, $p=ns$).

Conclusions: Even in the absence of hypertension and obesity, OSAS is characterized by endothelial dysfunction and impaired renal vasodilating capacity. Thus, OSAS could predispose per se to vascular and renal damage.

P2.09

DAMAGE ACCRUAL IS ASSOCIATED WITH ENDOTHELIAL FUNCTION DETRIMENT: A PROSPECTIVE COHORT STUDY

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Introduction: Our aim was to study endothelial function in a cohort of systemic lupus erythematosus (SLE) patients prospectively followed at our unit and to investigate its relation with disease activity and damage over time.

Materials and Methods: 38 female SLE patients without overt cardiovascular involvement were enrolled (age 35.8 ± 8 ys), followed-up for a mean of 4.45 ± 1.5 years. Clinical history, traditional cardiovascular risk factors, and laboratory parameters were recorded. Active disease was defined as ECLAM global score >2 ; SLICC/ACR-DI was used for scoring disease damage. FMD was assessed in the brachial artery by high-resolution ultrasound and computerized edge detection system (Quipu s.r.l., Pisa, Italy). FMD assessment was performed at study entry and was repeated in a subgroup of 21 patients at the end of follow-up.

Results: At enrollment, 18 patients presented active disease; mean FMD was $7.9\pm3.1\%$ with no differences between active (8.7 ± 1) and inactive group (7.9 ± 0.8 ; $p=0.53$), even after correction for age and disease duration. Baseline FMD tended to correlate with disease duration ($p=0.06$), and was similar in the patients with final poor outcome - death ($n=3$) or damage accrual ($n=12$) - compared to the others. In the follow-up, FMD showed a significant decline over time (from 8.0 ± 3.2 to 5.9 ± 3.3 , $P=0.04$) while endothelial-independent dilation did not (from 9.2 ± 3.5 to 8.6 ± 4.9 ; $p=0.63$). The decline was not different between active and inactive group; however, patients with poor outcome ($n=7$) showed a greater worsening in FMD over time (-4.1% vs -2.0%).

Conclusions: This study shows that, in SLE patients, disease duration rather than disease activity appears to influence endothelial function. Furthermore, damage accrual is associated with progressive detriment in endothelial function, with preserved response to glyceryl trinitrate.

P2.10

MECHANISTIC INSIGHTS INTO THE RELATIONSHIP BETWEEN WAVE REFLECTION AND RETINAL ARTERY FLOW PULSATILITY

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Background: Increased arterial stiffness is associated with a reduced buffering capacity of the large arteries, therefore predisposing the microcirculation to increased flow and pressure pulsatility. Previous data from our group have illustrated a positive relationship between aortic pulse wave velocity and an inverse relationship between wave reflection and retinal artery flow pulsatility. Therefore, the aim of this study was to investigate the macrovascular haemodynamic mechanisms involved in retinal artery flow pulsatility, by manipulation of wave reflection by Glycerol Trinitrate Nitrate administration.

Methods: Nine individuals, aged 63 ± 6 years and free from CV acting medication participated in this study. Augmentation index (AIx) was recorded using the Sphygmocor system (Atcor) as a measure of wave reflection. Pulsatility index (PI), a measure of retinal artery flow pulsatility was recorded using

doppler ultrasound (GE) and both peripheral and central blood pressure were measured using the Mobilograph system (IEM). All vascular haemodynamic measurements were recorded simultaneously at baseline and then again at 1, 3, 5, 10 and 15 minutes post GTN administration.

Results: The relationship between AIx and PI change from baseline were significantly different at 3 and 5 minutes ($P=0.02$ and $P=0.03$, respectively). See figure 1.

Conclusion: This study illustrates a direct inverse relationship between AIx and retinal artery flow pulsatility, suggesting a direct link between large artery haemodynamics and pulsatile flow in the microvasculature.

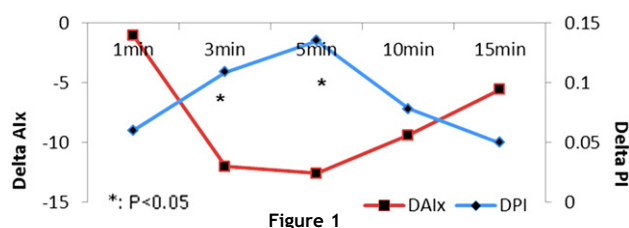


Figure 1

P2.11

IMAGING OF RETINAL ARTERIOLAR WALL IN VIVO IN HUMANS

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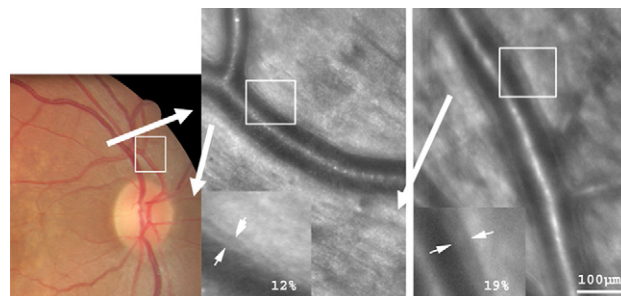
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Purpose: Adaptive optics (AO) is an opto-electronic technique improving lateral resolution of fundus images. Here we report a novel application of AO imaging, namely the visualization of the arteriolar wall in the human retina.

Methods: This study was done in compliance with French ethical regulations. AO fundus imaging was performed using a prototypic camera (RTX1, ImagerieEye, Orsay, France) in a cohort of healthy subjects and in patients affected by arterial hypertension. The camera uses infrared light, and the total acquisition time is less than 1 minute. The wall-to-lumen ratio (WLR) was measured in an arteriolar segment approximately 500 microns from the disc.

Results: 12 healthy subjects, 2 hypertensive patients and 1 patient affected by branch retinal vein occlusion were examined. The wall-to-lumen ratio varied from 11 to 15% in healthy eyes, and was 18% and 19% in the two hypertensive subjects. In the subject that had branch retinal vein occlusion, irregular thickening of the vessel wall was found.



Conclusions: We provide here the first in vivo images of the arteriolar wall in humans. This technique may be useful for the quantitative assessment of microvascular damage in aged and/or hypertensive patients.

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P2.12

THE INTERPLAY OF ENDOTHELIAL FUNCTION, INFLAMMATORY AND OXIDATIVE STATUS IN HIV INFECTION. DOES ANTIRETROVIRAL THERAPY PLAY A ROLE?

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Purpose: HIV infection is linked to heightened cardiovascular risk; this is partly mediated through endothelial dysfunction. We investigated the interplay of endothelial function with markers of inflammatory and oxidation in two groups of HIV infected patients: on ART and naïve to ART.

Methods: We recruited 47 HIV infected patients (46 male, mean age 35±10 years, mean CD4 count: 579±271). All were free from overt cardiovascular disease. 31 patients were naïve to ART; 17 were on ART. FMD of the brachial artery was used as an index of endothelial function. Markers of inflammatory (CRP, IL6), oxidative (ADMA) and functional (CD4, viral load) status were measured. Between groups comparisons (ART vs naïve patients) were performed using the Mann-Whitney U test.

Results: Endothelial function and inflammatory markers did not differ across groups. ART group had higher ADMA levels, lower viral load and higher CD4 count, thereby a favorable oxidative/functional status compared to naïves.

	Naïve (n=31)	ART (n=17)	P value
FMD (%)	4.2 (2.3, 7.5)	5.2 (3.1, 8.2)	0.740
CRP (mg/dL)	1.37 (0.98, 2.79)	1.05 (0.28, 3.32)	0.249
IL6 (pg/mL)	1.65 (0.83, 3.39)	1.00 (0.77, 1.65)	0.258
ADMA (μmol/L)	0.63 (0.25, 0.79)	0.84 (0.78, 1.06)	0.006
Viral load	19419 (8144, 54025)	50 (50, 50)	0.001
CD4 count	450 (378, 627)	635 (510, 779)	0.011

Conclusion: In HIV infection, ART does not change endothelial function; nevertheless it is linked to a favorable oxidative and functional status.

P2.13

ALTERED MICROVASCULAR RESPONSES TO ANGIOTENSIN II INFUSION INDICATING ENDOTHELIAL DYSFUNCTION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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Purpose: Angiotensin II (ANG) is implicated in the development of cardiovascular disease. We examined vascular responses to ANG in subjects with familial hypercholesterolemia (FH), a group at high cardiovascular risk.

Methods: The effects of ANG (3h iv infusion, 10 g/kg/min) on brachial blood pressure and forearm skin microvascular function were studied in 8 female and 8 male FH (mean age 43±8 ys) and in 16 matched healthy controls. Skin microcirculation was studied by laser Doppler fluxmetry during rest and local heating of the skin to 44°C (microvascular hyperaemia). Microvascular resistance was assessed by mean arterial pressure/microvascular hyperaemia, and macrovascular reactivity by blood pressure changes. Measurements were performed before, at 1 and 3h of ANG, and 1h after stopping infusion. Mean values ±SD.

Results: Baseline systolic blood pressure was higher in FH (127±14 vs 115±12 mm Hg; p=0.02), while pressure responses to ANG were similar in both groups (eg +24±10 vs +21±7 mm Hg, ANG 3 h). There were no baseline differences in microvascular hyperaemia or resistance between the groups. However, during and after ANG microvascular hyperaemia was impaired (p=0.01; eg 126±95 vs 184±102 units, ANG 3 h), and microvascular resistance higher (p=0.01; eg 1.9±0.9 vs 0.9±0.8 mm Hg/units; ANG, 3 h) in FH. Saline infusion verified stability of the experimental design (n=8).

Conclusions: Despite similar blood pressure responses to ANG in FH and controls, microvascular dilatation capacity was impaired in FH, indicating

endothelial dysfunction. These findings and increased microvascular resistance may lead to hypertension and cardiovascular complications in FH.

P2.14

RELATION OF HEART RATE VARIABILITY TO ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS – DEPENDENCE UPON CARDIAC CYCLE LENGTH

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In various diseased states reduced cardiac vagal modulation (CVM) is accompanied by impaired endothelial function. It is unclear whether CVM and endothelial function undergo dysregulation independently, or these systems affect each other negatively as a consequence of the disease process. Since the respective physiology is unclear, we aimed to investigate if such relationship between CVM and endothelial function exists in healthy subjects.

46 young males were studied. From 10 minute long ECG recordings mean RR interval (RRI) and time and frequency domain vagal heart rate variability (HRV) indices (SDNN; RMSSD; pNN50 and HF, respectively) were determined. HRV indices were used to define CVM. Endothelial function was assessed by measuring brachial artery flow mediated dilation (FMD). Hyperemic, diastolic shear rate was used to normalize FMD (nFMD).

RRI was related to most HRV indices, but not to FMD. On the other hand, all HRV indices correlated significantly and positively with FMD across subjects (r = 0.49, p<0.01 for HF–nFMD). After adjusting for potential confounders, RMSSD and HF remained significantly associated with nFMD. When subjects were dichotomized according to median RRI, the HRV–nFMD relations lost significance at higher (RRI>910 ms), and gained further significance at lower (RRI<910 ms) heart rates (r = 0.57, p<0.01 for HF–nFMD).

Our data demonstrate that vagal HRV indices are related to FMD across healthy male subjects. Although RRI is not related to FMD, the HRV–FMD relation is dependent upon RRI. The underlying mechanism may involve centrally released endothelial mediators, which enhance CVM through vasculo-neural communication.

P3 – Interventional studies 1

P3.01

A COMPARISON OF ARTERIAL FUNCTION OF HIV INFECTED (TREATED AND NEVER-TREATED) AND UNINFECTED BLACK SOUTH AFRICANS AFTER FIVE YEARS: PURE STUDY

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The prevention and treatment of hypertension is marginalized in South Africa by the overwhelming prevalence of HIV. HIV-1 infection and the treatment thereof paradoxically affect cardiovascular risk factors and may add to the cardiovascular risk of these individuals.

We aimed to compare the 5 year cardiovascular changes of black South Africans who were (a) HIV-1 infected without treatment, (b) HIV-1 infected with antiretroviral therapy and (c), uninfected. In this study 164 uninfected and 145 HIV-1 infected (77 never-treated and 68 treated) participants were followed-up after a 5 year period. Two hundred and ninety one participants were lost to follow-up, 11 were newly HIV infected, 39 died and 241 did not partake. The cardiovascular and anthropometric variables were assessed and the percentage change determined. Follow-up analysis (cross-sectional) showed a lower IMT (p<0.01), central systolic blood pressure (p<0.01) and augmentation index (p=0.03) in the HIV infected compared to the HIV uninfected participants. After 5 years (2005-2010) the treated HIV-1 infected participants showed an increase in pulse pressure (p=0.03) and no change in pulse wave velocity, whilst a decrease (p=0.02) was encountered in the never-treated HIV infected participants. No difference in the % change was seen between the treated and uninfected participants.

In conclusion, the cardiovascular profile of the treated HIV-1 infected Africans show signs of an early development of arterial dysfunction over 5 years which is not seen in the never-treated participants. How the