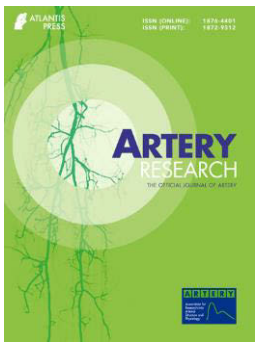

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P.015: ARE NITRIC OXIDE SYNTHASE AND CYCLOOXYGENASE PRODUCTS INVOLVED IN ACETYLCHOLINE VASODILATING EFFECTS "IN VIVO"?

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P.013
LONGITUDINAL STUDY OF VASCULAR MARKERS OF PREMATURE ATHEROSCLEROSIS AND METABOLIC CORRELATES IN HIV-INFECTED CHILDREN

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Purpose: To determine the presence of vascular markers of premature atherosclerosis and metabolic correlates in a prospectively followed cohort of antiretroviral treated HIV-infected children.
Methods: Vascular assessment included: carotid intima-media thickness and brachial artery reactivity using vascular ultrasound; peripheral pulse wave velocity (PWV) using photoplethysmography; central PWV, arterial stiffness and impedance indices using an Echo-Doppler method; and only at follow-up, augmentation index and PWV by applanation tonometry. Disease markers, oral glucose tolerance, fasting lipid profiles and abdominal fat (single slice CT scan) were also determined.
Results: Twenty children were assessed at baseline (median age 12.6 [range 8.5–18.5] years; 50% female) and follow-up 21–25 months later. All were on combination antiretroviral therapy at baseline, but 5 were off therapy at follow-up with fewer receiving protease inhibitors. Resting systolic blood pressure and pulse pressure increased significantly over the study period (both $p < 0.0001$), as did elastic modulus, stiffness index and input impedance ($p = 0.0018$, $p = 0.0004$, $p = 0.0082$, respectively). PWV measures by the different methods were not shown to correlate significantly. Dyslipidemia and abnormal glucose metabolism were present in 14 and 2 at baseline, and in 7 and 0 at follow-up, respectively. Visceral, subcutaneous and total abdominal fat content increased over time, but not significantly so.
Conclusions: An increase in measures of large arterial wall stiffness was observed over time. The reduction in dyslipidemia at follow-up may be related to fewer children receiving protease inhibitors. The potential risk of premature atherosclerosis in HIV-infected children on anti-retroviral therapy warrants long-term monitoring of metabolic profiles and vascular function.

P.014
VASCULAR MARKERS OF PREMATURE ATHEROSCLEROSIS IN PAEDIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS AND DISEASE, THERAPY, METABOLIC AND INFLAMMATORY CORRELATES

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Purpose: Controversy exists as to whether the dyslipidemia and premature atherosclerosis which occurs in Systemic Lupus Erythematosus (SLE) is more due to disease activity or the therapy. We sought to determine the presence and correlates of vascular markers of premature atherosclerosis in paediatric SLE.
Methods: Vascular assessment included: carotid intima-media thickness and brachial artery reactivity using vascular ultrasound; peripheral pulse wave velocity (PWV) using photoplethysmography; central PWV, arterial stiffness and impedance indices using an Echo-Doppler method. These vascular indices were converted to z-scores from normal population data, and tested for normality with single sample t-tests. Augmentation index and PWV, using applanation tonometry, were only assessed in the latter half of the cohort. Disease activity scores, disease duration, cumulative prednisone dose, other medication use and fasting lipid, glycemic and inflammatory profiles were also determined and Pearson correlations performed.
Results: Seventy paediatric SLE were assessed (median age 15.7 [7.3–18.7] years and diseased duration 1.4 [0.1–11.0] years, 80% females). The mean z-score adjusted data was significantly increased from normal for central PWV (+1.1, $p < 0.0001$), input (+0.3, $p < 0.04$) and characteristic impedance (+0.8, $p < 0.0001$). Correlations were found between aortic elastic modulus and higher fasting glucose (R 0.34, $p < 0.007$), and between stiffness index and homocysteine levels (R 0.38; $p < 0.008$).
Conclusions: In treated paediatric SLE of relatively short duration, dyslipidemia, abnormal glycemic and inflammatory profiles were relatively common. Some stiffness measures were increased and others correlated with known atherosclerotic risk factors. These patients warrant long-term monitoring of vascular function and traditional and non-traditional risk factors.

P.015
ARE NITRIC OXIDE SYNTHASE AND CYCLOOXYGENASE PRODUCTS INVOLVED IN ACETYLCHOLINE VASODILATING EFFECTS "IN VIVO"?

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It is current understanding in the literature that acetylcholine (ACh) vasodilating effect is endothelium-dependent. Also, different endothelial substances such as NO, EDHF and prostanoids can mediate ACh-induced vasorelaxation in different vessels. Although ACh-induced relaxation of isolated rat aorta rings is mainly related with NO release it is also dependent on prostanoids because pretreatment with indomethacin shifted the ACh curve to the right, reducing maximal effect (Vizioli et al., J Smooth Muscle Res, 41: 271–281, 2005). Consequently, one should expect that nitric oxide synthase (NOS) and cyclooxygenase (COX) inhibition should affect ACh-induced vasodilation "in vivo". To test this hypothesis we studied the effect of i.v. pretreatment with L-NAME (20 mg/kg), indomethacin (5 mg/kg) or its combination on the response to i.v. infusion of ACh in urethane-anesthetized rats. ACh infusion caused progressive blood pressure (BP) fall up to –40 mmHg, which was completely abolished by homatropine methylbromide (1 mg/kg). Although L-NAME significantly increased baseline BP (~50 mmHg) indicating NOS blockade, the response to ACh was not significantly affected. Indomethacin shifted the ACh curve to the left, suggesting that COX blockade potentiates the response to ACh "in vivo", as opposed to what was observed in isolated aortic rings. After the combined NOS and COX inhibition, the hypotensive response to ACh infusion was not significantly different from that observed prior to pretreatment. The present results disagree with those reported in isolated vessels and raise doubts on the mechanism actually involved in the hypotensive response to ACh "in vivo".

P.016
INCREASED ARTERIAL STIFFNESS IN YOUNG PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Chronic inflammation may impair arterial function and lead to an increase of their stiffness and risk of developing early atherosclerosis.
Aim of the study was to assess whether rheumatoid arthritis (RA) and high level of C-reactive protein can influence systemic arterial stiffness and aortic pulse wave velocity (PWV) in patients with RA.
Methods: We studied 53 RA patients (age 40.1±9.8 years) with moderate and high disease activity (DAS28 3.21–7.05) and 55 controls (age 39.7±8.1 years). Blood test included serum lipid profile, glucose and high-sensitivity CRP (hsCRP) measurement. The carotid-radial PWV and augmentation index (AIx) were assessed noninvasively by applanation tonometry (Sphygmocor v.7.01, AtCor Medical).
Results: In RA patients the adjusted for heart rate AIx (21.3±13.3% vs. 12.7±13.2%; $p < 0.001$) and hsCRP (31.32±40.29 mg/l vs. 1.58±3.36 mg/l; $p < 0.001$) were significantly higher as compared to the controls. Multivariate regression analysis revealed that RA is significant predictor of increased PWV adjusted for mean blood pressure ($p < 0.001$). In RA patients and control group correlations were not found between hsCRP and AIx ($r = -0.044$; $p = 0.752$ vs. $r = 0.215$; $p = 0.121$) as well as between hsCRP and PWV ($r = -0.076$; $p = 0.589$ vs. $r = -0.014$; $p = 0.921$).
Conclusion: RA is associated with the increase of aortic and systemic arterial stiffness. Elevation of serum hsCRP is not related to the increase of arterial stiffness neither in RA patients nor in controls.

P.017
THE ROLE OF THE CORONARY MICROCIRCULATION IN DETERMINING BLOOD FLOW

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Background: The coronary flow velocity profile is strikingly different from that of the proximal aorta, even though they are only a few centimetres apart and have almost identical pressure waveforms. We use wave intensity analysis to help explain this phenomenon, and to explore the importance of the coronary microcirculation in the regulation of coronary blood flow.
Method and Results: In 18 subjects (mean age 54 years, 12 female) we measured simultaneous pressure and Doppler velocity using intra-arterial wires in the proximal left main stem, left anterior descending, circumflex artery and proximal aorta. Wave intensity analysis was used to separate the