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P185: CARDIO-ANKLE VASCULAR INDEX AND PLASMA LEVELS OF LEPTIN AND ADIPONECTIN IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Methods: Our study evaluated global risk factors for coronary heart disease (CHD), structure and function of precerebral arteries in 41 HD subjects and 41 matched controls. HD subjects were divided into groups by the United Huntington disease rating scale (presymptomatic-PHD, early-EHD, mid-stage-MHD and late-LHD). CHD risk factors assessment and Doppler examination of precerebral arteries were performed, including measurements of the carotid artery intima-media thickness (IMT), and parameters indicating local carotid artery distensibility (stiffness index β , pulse wave velocity, pressure strain elasticity module and carotid artery compliance).

Results: In the HD and controls we identified a comparable number of non-obstructive plaques (<50% lumen narrowing). No obstructive plaques (>50% lumen narrowing) were found in patients or controls. There was significantly increased IMT in MHD patients. In PHD and EHD the parameters of arterial stiffness were significantly higher and the carotid artery compliance was significantly lower.

Conclusions: Our results reveal functional vascular pathology in PHD, EHD, and MHD. Precerebral arteries dysfunction in HD therefore appears to be mostly functional and in agreement with autonomic nervous system dysfunction in HD.

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INCREASED ARTERIAL STIFFNESS IS ASSOCIATED WITH POORER LEFT VENTRICULAR STRUCTURE AND FUNCTION IN ADOLESCENCE

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Introduction: Increased arterial stiffness (AS) in adults causes increased left ventricular (LV) afterload, putting additional strain on the heart. Long-term, this can lead to an adverse cardiovascular phenotype and AS has been found to be a determinant of CVD, independent of traditional cardiovascular risk factors. However, limited evidence exists for this association in children and adolescents.

Methods: 1625 young adults (age 17y; 46% male) from the Avon Longitudinal Study of Parents and Children (ALSPAC), a UK based birth cohort, underwent echocardiography and carotid-to-femoral pulse wave velocity (PWV) measures. Linear regression was used to investigate associations between PWV and LV structure and function, including LV mass, relative wall thickness (RWT), left atrial diameter (LAD), mitral inflow (E:A), midwall fractional shortening (MFS) and tissue Doppler peak systolic velocity (s').

Results: Elevated PWV was associated with increased LV mass and RWT and inversely associated with E:A and MFS (Table 1). Adjustment for age and sex attenuated the association with LV mass. Further adjustment for body mass index (BMI), systolic blood pressure (SBP), alcohol, smoking and socioeconomic status (SES) attenuated the association with RWT, whilst the associations with E:A and MFS remained.

Table 1.

	Unadjusted		Age and sex adjusted		Age, sex, BMI, SBP, alcohol, smoking, SES adjusted	
	Coefficient $\pm$ SE	P value	Coefficient $\pm$ SE	P value	Coefficient $\pm$ SE	P value
LV mass ^{2.7} (g/m ^{2.7})	0.55 $\pm$ 0.21	0.009	-0.066 $\pm$ 0.225	0.768	-0.123 $\pm$ 0.225	0.584
RWT	0.007 $\pm$ 0.002	<0.001	0.008 $\pm$ 0.002	<0.001	0.005 $\pm$ 0.00	0.069
LAD (cm)	-0.004 $\pm$ 0.015	0.784	-0.017 $\pm$ 0.015	0.268	-0.010 $\pm$ 0.016	0.516
E:A	-0.054 $\pm$ 0.014	<0.001	-0.073 $\pm$ 0.015	<0.001	-0.067 $\pm$ 0.019	<0.001
MFS (%)	-0.40 $\pm$ 0.079	<0.001	-0.246 $\pm$ 0.085	0.004	-0.232 $\pm$ 0.1	0.022
s' (cm/s)	0.078 $\pm$ 0.05	0.138	0.004 $\pm$ 0.057	0.937	-0.038 $\pm$ 0.067	0.567

Conclusion: Increased AS is already associated with poorer measures of LV structure and function in adolescence. Adjustment for potential confounders did not substantially attenuate these associations with LV function.

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INCREASED ARTERIAL STIFFNESS IS ASSOCIATED WITH HIGH INFLAMMATORY ACTIVITY IN RHEUMATOID ARTHRITIS

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Background: Patients with rheumatoid arthritis (RA) have a high cardiovascular (CV) risk. Relationships between inflammation and arterial stiffness (AS) in patients with RA are not well understood.

Aim: To evaluate parameters of AS and their associations with inflammation in patients with RA.

Methods: 62 patients with RA without known CVD were examined (73% females, age 58.5 $\pm$ 15.4 years, 13% smokers, 61% with AH). Median duration of RA was 8 years (IQR 3–17).

Median hsCRP 12.1 mg/dl (IQR 2.2; 23.4), median rheumatoid factor (RF) 32.5 IU/ml (IQR 8.3; 173 IU/ml). All patients received disease-modifying antirheumatic drugs.

Median duration of AH 6.1 years (IQR 0–10 years). Parameters of AS were assessed by applanation tonometry. Cardio-ankle vascular index (CAVI) and vascular age were measured by VaSera 1500. PWV > 10.0 m/s and CAVI > 9.0 were considered as AS increase. $p < 0.05$ was considered significant.

Results: Mean PWV was 9.3 $\pm$ 3.2 m/s. PWV > 10 m/s was observed in 32.3% patients, CAVI > 9.0 in 25.8%. Patients with PWV > 10 m/s were older (69.8 $\pm$ 8.5 vs 53.2 $\pm$ 15.1 years), had higher BMI (29.3 $\pm$ 6.5 vs 24.7 $\pm$ 4.8 kg/m²), duration of AH (median 11.5 [IQR 5.5; 17] vs 0 [IQR 0; 5] years), higher SBP levels (144 $\pm$ 20 vs 123 $\pm$ 14 mmHg), higher levels of hs-CRP (median 22 [IQR 13.3; 60] vs 6.7 [IQR 1.6; 17.2] mg/dl), higher CAVI (9.5 $\pm$ 1.1 vs 7.6 $\pm$ 1.4), vascular age (71 $\pm$ 8.4 vs 53.4 $\pm$ 17.5 years). There were positive correlations between PWV and age ($r = 0.7$), BMI ($r = 0.4$), SBP ($r = 0.6$), hs-CRP ($r = 0.3$), vascular age ($r = 0.6$). Multiple regression analysis confirmed that AH duration ($\beta = 0.2$, $p = 0.03$), SBP ($\beta = 0.6$, $p < 0.0001$) and hs-CRP ($\beta = 0.3$, $p = 0.00009$) were independent predictors of AS increase.

Conclusion: Elevation of hsCRP as well as other traditional risk factors is an independent predictor of PWV increase in patients with RA.

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CARDIO-ANKLE VASCULAR INDEX AND PLASMA LEVELS OF LEPTIN AND ADIPONECTIN IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Objective: Systemic lupus erythematosus (SLE) is a chronic inflammatory disease associated with vascular derangement [1]. Leptin and adiponectin

are adipokines with immunomodulatory and vascular functions [2]. We studied the association between arterial stiffness and plasma leptin and adiponectin levels in SLE patients in Ghana.

Methods: In a case control design involving 80 SLE patients and 90 non-SLE controls, arterial stiffness was assessed by cardio-ankle vascular index

(CAVI) and heart-ankle pulse wave velocity (haPWV) using Vasera 1500N. Circulating levels of leptin, adiponectin, insulin and C-reactive protein (CRP) were measured by ELISA.

Results: Compared to non-SLE controls, SLE patients had higher levels of CAVI (7.3 ± 1.1 vs 6.1 ± 1 , $p < 0.001$), haPWV (7.7 ± 1.3 vs 6.5 ± 0.8 m/s, $p = < 0.001$), insulin [76.8 (45.9–184.8) vs 39.8 (22.9–86.3) pmol/ml, $p = 0.007$], leptin [856.1 (364.8–1509.3) vs 426.7 (426.8 (84.7–1178.7) ng/ml, $p = 0.039$], adiponectin [1.1 (0.8–2.3) vs 1.6 (1.3–2.6) ng/ml, $p = 0.039$] and CRP [1.6 (0.8–2.2) vs 0.9 (0.6–1.2) mg/ml, $p = 0.021$]. In a partial correlation analysis with adjustment for age and BMI, CAVI was associated with leptin ($r = 0.21$, $p = 0.031$), CRP ($r = 2.9$, $p < 0.001$) and insulin ($r = 0.18$, $p = 0.04$), but not adiponectin ($r = -0.15$, $p = 0.068$).

Conclusion: In our study population, SLE patients have higher arterial stiffness, associated with low-grade inflammation and deranged circulating adipokine levels.

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IMPACT OF OBESITY ON VASCULAR STRUCTURE AND FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS

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Background: Cardiovascular disease is a leading cause of disease progression and death in multiple sclerosis (MS). Obesity has a negative impact on vascular structure and function, but whether this contributes to worse vascular function similarly in individuals with MS and controls is unknown.

Aim: To investigate the impact of obesity on vascular function and structure in a group with MS.

Methods: In a sample of $n = 133$ participants (MS: $n = 89$, control $n = 44$), height and weight were measured to calculate BMI. After a 10 minute rest in the supine position, resting heart rate (HR) and brachial blood pressure (BP) were collected. Augmentation index (AIX), HR normalized AIX (AIX@HR75) and pulse wave velocity (PWV) and subendocardial viability ratio (SEVR) were measured with applanation tonometry.

Carotid intima-media thickness (IMT) and beta-stiffness (beta) were measured with carotid ultrasound, and Forearm Blood Flow (FBF Baseline, Peak and Area Under the Curve (AUC)) was measured with strain gauge plethysmography. Data were analyzed with multiple linear regression analyses with group, sex, BMI and GroupxBMI as independent variables.

Results: Higher BMI correlated with higher HR and PWV in both groups. In the MS group however, a higher BMI was also correlated with worse outcomes on the SEVR, FBF Baseline, Peak and AUC.

Conclusions: Having a higher BMI contributes even more to a worse vascular profile in MS patients than in controls, suggesting that reducing overweight and obesity in the MS population will benefit their vascular structure and function.

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IN SEVERE AORTIC STENOSIS, DECREASED SYSTEMIC VASCULAR RESISTANCE IS ASSOCIATED WITH A LARGER, THICKER WALLED VENTRICLE EXCEPT FOR THE SEPTUM

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Background: The ventricle in aortic stenosis (AS) is influenced by both valvular and vascular factors. The importance of afterload on left ventricular (LV) remodeling is not completely understood. Traditional imaging techniques which rely on geometric assumptions may not assess regional remodeling accurately.

Aim: To understand the influence of systemic vascular resistance (SVR), systemic arterial compliance (SAC), valvulo-arterial impedance (Zva) on LV remodeling using a cardiac atlas technique.

Methods: 109 patients with symptomatic severe AS awaiting surgical valve replacement (age 69 ± 10 y, 60% male, aortic valve area 0.7 ± 0.3 cm², mean gradient 48 ± 15 mmHg) underwent comprehensive clinical, echocardiographic and cardiovascular magnetic resonance (CMR) examinations. SVR, SAC and Zva were calculated as previously published (1). CMR LV short axis steady-state free precession cine images were segmented and co-registered using a cardiac atlas technique (2). Data were extracted and analysed using mass-univariate 3D regression modeling adjusted for age, sex, and height and accounting for multiple testing, presented as standardized β .

Results: Lower SVR correlated with increased wall thickness and larger cavity volume. SVR related changes were more prominent in the lateral wall ($\beta -0.3$ to -0.6 , $p = 0.04$), with no discernable influence on the septum (Figure 1). With lower SVR, LV cavity enlargement was directed away from the septum ($\beta -0.17$ to -0.56 , $p = 0.002$). There was no influence of SAC or Zva on 3D parameters.

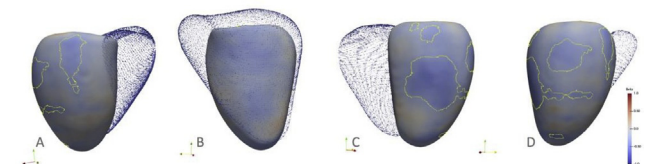


Figure 1 Mass-univariate 3D regression maps relating wall thickness and systemic vascular resistance over the left ventricle, adjusted for age, sex and height. Yellow line encloses areas with p values < 0.05 . There is a negative correlation between SVR and wall thickness, sparing the septum (B). $SVR = (80 * \text{Mean Arterial Pressure (MAP)}) / \text{Cardiac Output}$.

Conclusion: In severe AS there is an association between lower SVR and a larger, thicker walled ventricle except for the septum. The use of a cardiac atlas in aortic stenosis may offer new insights into regional LV remodeling.

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Outcome variables	Standardized beta ¹				Adjusted R ²
	Group	Sex	BMI	Grp x BMI	
HR rest	1.081*	0.005	0.520*	-0.944	0.11
AIX	-0.019	-0.523*	0.040	0.045	0.26
AIX@HR75	0.341	-0.536*	0.235	-0.270	0.32
SEVR	-1.292*	0.175*	-0.565*	1.090*	0.22
PWVc	0.001	-0.003	0.321*	0.120	0.12
PWVc/MAP	0.324	-0.126	0.278	-0.122	0.11
IMT	0.715	0.164	0.385	-0.511	0.12
FBF Baseline	0.432	0.070	0.326*	-1.090*	0.33
FBF Peak	0.580	0.318*	0.152	-1.035*	0.35
FBF AUC	0.746	0.230*	0.316	-1.174*	0.21

¹Group (0 = control, 1 = multiple sclerosis), Sex (1 = Female, 2 = Male).

* ($p < 0.05$).