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P158: ASSOCIATION BETWEEN AMBULATORY ARTERIAL STIFFNESS INDEX, MARKERS OF BLOOD PRESSURE VARIABILITY AND INDICES OF SUBCLINICAL VASCULAR DAMAGE IN OBESE CHILDREN

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Table 2. Correlation of sRAGE with metabolic, hemodynamic and arterial stiffening variables.

Variables	r Spearman	p-value
T2DM time evolution	-0.203	0.309
HbA1c	-0.082	0.780
Creatinine	0.724	0.000*
Systolic Blood pressure	-0.487	0.029*
Diastolic Blood pressure	-0.456	0.043*
Pulse pressure	-0.476	0.034*
Arterial Mean Pressure	-0.437	0.054*
Central Systolic Blood	-0.452	0.045*
Pressure		
Central Diastolic Blood	-0.448	0.047*
pressure		
Aortic Pulse Wave velocity	-0.361	0.118
Central pulse pressure	-0.035	0.041*
Aortic Augmentation Index	-0.469	0.037*

Abreviation: sRAGE, soluble receptor for advanced glycation end products: T2DM. type 2 diabetes mellitus

Conclusion: This study shows a significant correlation of serum sRAGE and S100-A1 on peripheral and central hemodynamics in non-hypertensive diabetic patients.

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DURATION OF DIABETES MELLITUS IS A SIGNIFICANT PREDICTOR OF ARTERIAL STIFFNESS IN PATIENTS WITH ARTERIAL HYPERTENSION AND TYPE 2 DIABETES MELLITUS

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Background: Diabetic complications increase with disease duration but little is known about the relationships between aortic stiffness and diabetes duration

Aim: to assess associations of diabetes mellitus (DM) duration and parameters of arterial stiffness in patients with arterial hypertension (AH) and T2DM.

Methods: 90 patients with AH and T2DM were included (39%males, mean age63,8 \pm 11,6 years, 44%smokers). Mean office BP was 146 \pm 23/86 \pm 10 mmHg. All patients received combined AHT, target BP < 140/85 mmHg was achieved in 52,7% of patients. Median duration of DM was 8,5 years (IQR 2; 13 years), mean glucose was 8,0 \pm 2,4 mmol/l, mean HbA1c 9,2 \pm 2,0%. BP was measured with a validated oscillometric device. Parameters of arterial stiffness were assessed by applanation tonometry, cardio- ankle vascular index (CAVI) and vascular age were measured (VaSera 1500). p < 0,05 was considered significant.

Results: Mean central BP was $132\pm18/79\pm12$ mmHg, mean cfPWV-10,5 \pm 2,4 m/s, mean R- CAVI-8,8 \pm 1,9, L-CAVI-8,9 \pm 1,8. Further analysis was performed in subgroups according to tertiles of DM duration (G1 < 4 years (n = 31), G2-4·10 years (n = 30), G3 > 10 years (n = 29)). Patients in G3 were older (69,5 \pm 11,1 vs 62,1 \pm 11,2 vs 60,0 \pm 10,8 years), had higher vascular age (73,8 \pm 9,0 vs 68,6 \pm 11,8 vs 64,5 \pm 13,4 years) and R-CAVI (9,3 \pm 1,9 vs 9,0 \pm 1,8 vs 8,1 \pm 1,9); p < 0,05 for trend. Patients from G3 and G2 had the highest level of cfPWV compared to G1 (11,0 \pm 2,0 and 11,4 \pm 2,4 vs 9,1 \pm 2,4 m/s, p = 0,0009). There were significant correlations between duration of DM and age (r = 0,35), vascular age (r = 0,30), creatinine (r = 0,23), cfPWV (r = 0,34), and R-CAVI (r = 0,3). Only age and DM duration were predictors of PWV increase (β = 0,3, p = 0,02 and β = 0,2, p = 0,04, respectively).

Conclusions: In diabetic patients, aortic stiffness is strictly correlated with diabetes duration, independently of blood pressure level and diabetes control

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RETINAL ARTERIOLAR FUNCTION, ENDOTHELIAL DYSFUNCTION AND ARTERIAL STIFFNESS IN PATIENTS WITH TYPE 2 DIABETES

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Background: Crosstalk between large and small arteries has been suggested to partake in the microvascular complication development in patients with type 2 diabetes mellitus (T2DM). Yet, data are scarce.

In the present study, we aimed to elucidate the crosstalk between large and small arteries in T2DM.

Methods: Twenty patients with T2DM and 20 sex- and age matched controls were included. Arterial stiffness was assessed by carotid-femoral Pulse Wave Velocity (cfPWV) using the SphygmoCor. Endothelial function was assessed using EndoPAT. Retinal blood supply regulation was examined by retinal arteriolar diameter change during i) exposure to flickering lights, ii) isometric exercise (hand-weight lifting), and iii) a combined stimulus of i) + ii) using the Retinal Vessel Analyzer (RVA).

Results: T2DM patients had higher cfPWV than controls $(9.3\pm1.8\,\text{m/s})$ vs. $8.3\pm2.2\,\text{m/s}$, p=.049). No group difference was observed in endothelial function $(0.71\pm0.30\,\text{vs.}\,0.81\pm0.30,\,p=.32)$ or in response to intervention with flicker, exercise or the combination (all p>0.05). Endothelial function was associated with mean arteriolar diameter change for the combination intervention (Beta = 0.033 [0.0013; 0.064], p=.042) in patients and controls. No association was observed between cfPWV and retinal arteriolar %-diameter change in patients or controls.

Conclusion: Peripheral endothelial function was associated with retinal arteriolar diameter change. Our findings may indicate a contribution of macro-microvascular crosstalk in diabetes complication development.

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ASSOCIATION BETWEEN AMBULATORY ARTERIAL STIFFNESS INDEX, MARKERS OF BLOOD PRESSURE VARIABILITY AND INDICES OF SUBCLINICAL VASCULAR DAMAGE IN OBESE CHILDREN

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Objective: Ambulatory Arterial Stiffness Index (AASI) and symmetric AASI (sAASI) have been proposed as indices of arterial stiffness obtained by 24-hour ambulatory blood pressure monitoring (ABPM). ABPM allows the analysis of indices of BP variability like day and night SD, BP dipping, weighted 24-h SD (wSD), average real variability (ARV). Aim of the present study was to address the relationship between these indices and other markers of vascular subclinical damage in children.

Design and Method: 45 obese children were included. Children underwent vascular measurements, including: (i) office and 24-hour ambulatory BP; (ii) brachial flow-mediated dilatation (FMD), carotid intima media thickness (CIMT), and distensibility (cDC); (iii) systemic arterial stiffness (SIDVP). From ABPM we calculate AASI, sAASI, ARV, SD, SD, systolic and diastolic dipping and wSD.

Results: ARV showed a significant correlation with SIDVP (r=0.379; p=0.023). AASI but not sAASI correlated with FMD (r=0.361; p=0.031). In the population divided in hypertensive (n=11)/normotensive (n=34), ARV was associated with SIDVP only in normotensive (r=0.446; p=0.015). In normotensive, z score-BMI was correlated with both sAASI and wSD (respectively 0.340; p=0.049 and 0.423; p=0.014), wSD correlated with FMD (r=0.384; p=0.048); in hypertensive children, ARV

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correlated with FMD (r = 0.828; p = 0.011; rspearman = 0.738; p = 0.037). No indices of BP variability correlated with cIMT or cDC.

Conclusions: BP variability, in particular ARV, shows a correlation with systemic but not local vascular stiffness in a sample of obese children, suggesting a relation between daily BP variability and arterial elastic properties. Further studies, especially perspective ones, are needed to clarify the pathophysiological significance of these relations.

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ASSOCIATION BETWEEN PULSE WAVE VELOCITY AND APNEA-HYOPPNEA INDEX IN PATIENTS WITH TYPE 2 DIABETES AND OBSTRUCTIVE SLEEP APNEA

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Introduction: Obstructive sleep apnea (OSA) is associated with increased cardiovascular (CV) risk. OSA is highly prevalent among patients with type-2 diabetes (T2D).

Patients with T2D have increased risk of cardiovascular events, and have an increased aortic stiffness.

Continuous Positive Airway Pressure (CPAP) treatment reduces severity of OSA, but whether it reduces CV risk remains unclear. One randomized trial with CPAP intervention and pulse wave velocity (PWV) as endpoint has shown a significant reduction in PWV after four months, in non-diabetic patients. The effect on patients with diabetes remains unknown.

Aim: Investigate the effects of CPAP treatment on PWV in patients with T2D and newly diagnosed OSA. Furthermore, investigate the relationship between PWV and severity of OSA.

Method: A randomized, controlled, multicenter study. 70 patients with T2D and newly diagnosed OSA randomized to: CPAP treatment or a control group. Data will be collected at baseline, 4 and 12 weeks. PWV was measured using SphygmoCor (AtCor Medical, Sydney, Australia) and AHI measured using ApneaLink (ResMed, Poway, CA, USA). Relationship between PWV and AHI was evaluated at baseline.

Results: Baseline data from the first 21 patients showed mean age 63 years (± 8.1), mean systolic blood pressure (BP) was 134 (± 12.5) mmHg, mean AHI was 30.2 (± 12.4) and mean PWV was 11.6 (± 1.9) m/s.

AHI was associated with PWV in multivariate analysis with adjustment for age and systolic BP, beta-coefficient 0.08, p=0.029.

Conclusion: At baseline PWV and AHI were correlated. Progression of the study will reveal if CPAP treatment can lower PWV in this cohort.

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VASCULAR ABNORMALITIES AND HAEMODYNAMIC PATTERN IN OBESE YOUNG ADULTS

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Background: Obesity is linked to a higher prevalence of risk factors, metabolic and inflammatory pathways conducting to increased vascular disease and CV risk.

Objective: To assess vascular disarrangements and haemodynamic patterns in obese young subjects (O) compared with matched lean (L) controls, using non invasive methods.

Methods: From the database of our Non Invasive Vascular Lab with 3964 first evaluated patients, we performed a case control study with 363 sujects, 268 obese and 95 lean, age and sex matched controls. We measured IMT, Plaque analysis, PWV, Endothelial Function (EF) and arterial stiffness (CAP and Aix) (AS) using an oscillometric device (Arteriograph, Tensiomed. Hungary®) and

non invasive haemodynamic evaluation using impedance cardiography (Z Logic Exxer \circledR).

Results: Age (O 42.5 + 5; L 43.5 + 11) and sex % (O 80.6%; L 78%) were matched. BMI (O 33.5 + 3.3 L 25 + 1.1 Kg/m²), waist (O110.4 + 7.5; L 91.2 + 6.1 cm) and BP (SBP O 139.8 + 16.8; L119 + 8.8 and DBP O 89 + 3.9; L 74.3 + 8 mmHg) were higher in O (p < 0.001).CV Risk Factors in O: HTN 68% DLP 59.7% SMKG 24.2% DBT2 7.8% SED 72.4%. The % of abnormalities in IMT (O/L: 65.8/25.3%), Plaques (75.6/38.9%), EF (57.5/33.7%) and PWV (41.4/17.9%) were higher in O (p < 0.001). Central and Peripheral PP were higher in O but not Aix. CI was significantly lower and PVRI and Thoracic Fluid content higher in O.

Conclusion: Young obese patients present a higher prevalence of vascular disarrangements either structural and functional and a haemodynamic pattern of high peripheral resistance with volume expansion that may explain the role of this condition as a CV risk factor.

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ROLES OF ANGIOPOIETINS 1 AND 2 ON ARTERIAL FUNCTION DURING A TREATMENT TRIAL IN PEOPLE WITH OR AT RISK OF DIABETES

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Background/ Objective: Vascular growth factors angiopoietin-1 (Ang1) and -2 (Ang2) regulate vascular permeability and inflammation, Ang2 likely as Ang1's selective antagonist. Their role before or in type 2 diabetes (pre-&T2D) is unknown. We hypothesised that higher circulating Ang1 and lower Ang2 (=lower Ang2/1 ratios) would be linked to increased arterial stiffness and its change over the trial, independent of blood pressure (BP).

Methods: ELISA assays were performed from 60 participants with all time-points of plasma samples from 'VaSera', a trial of single-centre, double-blind, parallel, randomised, controlled, 2x2 factorial design. Interventions were spironolactone and beetroot juice, a NO³⁻ donor, with doxazosin and placebo juice respectively to control for BP change (Δ) over the trial. Vascular measurements were aortic pulse wave velocity (aPWV), cardiac-ankle pulse wave velocity (CAVI), analysed by multiple regression adjusted for baseline BP and Δ BP over 6 months.

Results: Baseline Ang1 was positively while higher baseline Ang2 was negatively associated with baseline aoPWV at $(\beta=0.37,\ p=0.01;\ \beta=-0.27,\ p=0.047,$ respectively), independent of BP, BMI and DM status, so baseline r=-0.45 for the Ang2:1 ratio with aPWV, and r=0.39 for $\Delta aPWV$ over the trial. Higher baseline Ang1 independently predicted decreased aoPWV over 6 months $(\beta=-0.44\text{m/sec}\ per\ ng/ml,\ p=0.006).$ Angiopoietin concentrations were not associated with CAVI or BP.

Conclusions: Angiopoietins were related to baseline aPWV, independently of BP, and to Δa PWV over the trial, also independent of BP change, but were unrelated to CAVI or BP. Monitoring and manipulating Angiopoietins may help arterial health in pre- α T2DM.

Poster Session II — Special Populations P182

ARTERIES IN PATIENTS WITH HUNTINGTON'S DISEASE

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Background: Huntington's disease (HD) is a neurodegenerative disorder leading to the progressive death of neurons in various brain regions. Although it is a disease of the central nervous system (CNS), mortality surveys indicate that heart disease is one of the mayor causes of death in HD patients. The mechanisms of cardiac pathophysiology of the disease remain unknown. It might be a consequence of altered activity of autonomic nervous system as part of the CNS.