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6.10: PERIPHERAL SENSORY NEUROPATHY AND VASCULAR ANGIOGENIC FACTORS IN TYPE 2 DIABETES PATIENTS IN GHANA

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(PP)>60 mmHg, PWV>10 m/s. Stiffness gradient was assessed by CF-PWV/CR-PWV ratio, with values>1 indicating the stiffness mismatch. $p<0.05$ was considered significant.

Results: Mean PP was 47.6 ± 12.7 mmHg. PP>60 mmHg was observed in 18.1%. Group with PP>60 mmHg was characterized by higher HbA1c (9.8 ± 1.8 vs 8.4 ± 2.0 %) and stiffness gradient (1.4 ± 0.4 vs 1.2 ± 0.1) $p<0.05$ for trend. Mean CR-PWV was 7.7 ± 1.2 m/s, mean CF-PWV was 10.3 ± 2.0 m/s. CF-PWV>10 m/s was observed in 27.2% of patients. Groups with PWV above and below 10 m/s were similar by age, gender, metabolic risk factors and haemodynamic parameters. Mean stiffness gradient was 1.3 ± 0.4 , gradient >1 was observed in 92.7%. Patients with high stiffness gradient were older (63.3 ± 11.6 vs 54.0 ± 10.2 , $p<0.05$). All other parameters were similar.

Conclusion: Patients with AH and type 2DM are characterized by aortic-brachial stiffness mismatch. Thus it can be used as early marker of vascular ageing in this patients' population.

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6.7

FIRST EVIDENCE OF PULSATILE PRESSURE INTERACTION BETWEEN THE MACRO-VASCULATURE AND MICRO-VASCULATURE: PROOF-OF-CONCEPT BY ASSOCIATION WITH KIDNEY DYSFUNCTION AMONG PATIENTS WITH TYPE 2 DIABETES

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Objectives: It is widely thought that excess pulsatile pressure energy from increased stiffness of large central arteries (macro-vasculature) is transmitted to capillary networks (micro-vasculature) and causes end-organ damage (i.e. kidneys). However, this hypothesis has never been tested, and we sought to achieve this by examining people with increased macro-vascular stiffness (patients with type 2 diabetes T2DM) compared with non-diabetic controls.

Methods: Among 13 T2DM (68 ± 6 years) and 15 controls (58 ± 11 years) macro-vascular function was measured by aortic stiffness and radial artery waveforms by tonometry. Forearm micro-vascular waveforms were simultaneously measured via low power laser Doppler flowmetry, with augmentation index (Alx) and augmented pressure (AP) derived on all waveforms. Kidney function was assessed by estimated glomerular filtration rate (eGFR).

Results: Aortic stiffness was higher among T2DM (9.3 ± 2.5 vs 7.5 ± 1.4 m/s, $p=0.046$). There was an obvious pulsatile micro-vascular waveform, with qualitative features similar to radial waveforms. Macro-vasculature Alx and AP were significantly related to micro-vasculature Alx ($r=0.428$, $p=0.005$ and $r=0.545$, $p=0.004$ respectively). Micro-vascular (but not macro-vascular) Alx was associated with eGFR in T2DM ($r=-0.632$, $p=0.037$).

Conclusions: This is the first in-human evidence of pulsatile pressure interaction between the macro-vasculature and micro-vasculature, and provides potential explanation for accelerated kidney dysfunction.

6.8

THE RELATIONSHIP BETWEEN DIASTOLIC FUNCTION AND CENTRAL HEMODYNAMICS IN DIABETIC HYPERTENSIVE PATIENTS

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Background: Diabetic hypertensives patients present different hemodynamic pattern than only hypertensive patients. We aimed to investigate

the relationship between the diastolic function and the pulse pressure amplification (PPA), an index combining both arterial stiffness and wave reflexion, in diabetic hypertensives subjects compared to hypertensive subjects.

Methods: We examined 123 patients admitted to the one day hospital of the Hotel-Dieu Hospital (Paris, France) for cardiovascular risk assessment. Anthropometric, laboratory and clinical measurements were collected. Hemodynamic parameters (central blood pressure, aortic pulse wave velocity [PWV], augmentation index [Alx] and PPA) were measured using applanation tonometry. Standard ultrasound echocardiography was performed.

Results: Diabetic hypertensive subjects ($n=44$) were older than hypertensive subjects ($n=79$) (mean age[SD] $64[9]$ vs $56[14]$, $p<0.05$), and they presented similar cardiovascular risk factors frequencies. Gender was equally distributed. The diastolic function, assessed by the E/E' ratio was significantly positively correlated with PWV in total population ($r=0.19$, $p=0.03$), with no differences between the two groups. At the contrary, E/E' ratio was not correlated with PPA in total population, but it was significantly and negatively correlated with PPA only in the diabetic group (p for interaction 0.007 , $r=-0.35$, $p=0.02$). The multiregression analysis (containing all the confounding variables) in this group revealed as significant (p value<0.05) determinants of PPA: the diastolic function (partial- $R^2=0.14$), gender (partial- $R^2=0.27$), heart rate (partial- $R^2=0.26$), angiotensin blockers treatment (partial- $R^2=0.13$).

Conclusion: We confirmed that diabetic hypertensive patients have different hemodynamic behaviour than hypertensive non-diabetic patients. The results suggest that the mechanisms linking diastolic function with PPA are more prominent in diabetic patients.

6.9

ANTIPLATELET AND VASCULAR EFFECTS OF ASPIRIN IN HEALTHY PERSONS AND PATIENTS WITH TYPE 2 DIABETES

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Background: Treatment with aspirin is a cornerstone in the secondary prevention of cardiovascular disease (CVD) in diabetes, whereas its place in primary prevention remains controversial.

The effect of once-daily aspirin on platelet aggregation is unclear in patients with diabetes. Furthermore, the effects of aspirin on endothelial-dependent vasodilation and arterial stiffness, both important predictors of CVD, needs to be clarified.

Our aim is to investigate both the acute and the chronic effects of aspirin on platelet aggregation, endothelial-dependent vasodilation and arterial stiffness during 24 hours in patients with type 2 diabetes without CVD and in healthy controls.

Method: Based on power calculations, we will include 21 patients with type 2 diabetes and 21 sex and age-matched controls. Platelet aggregation is measured by impedance aggregometry, whereas arterial stiffness (carotid-femoral pulse wave velocity) is assessed by applanation tonometry. Endothelial-dependent vasodilation is assessed by peripheral arterial tonometry.

Outcome variables will be obtained at baseline and 1 hour after administration of aspirin. Participants are then treated for 6 days with once-daily aspirin and measurements are performed again 24 hours and 1 hour after aspirin intake.

Results: Preliminary results will be ready for presentation at the congress.

Perspective: This study provides new insight into whether once-daily dosing of aspirin is sufficient for effective platelet inhibition during 24 hours in patients with type 2 diabetes without CVD. Furthermore, this study will clarify if aspirin has positive effects on endothelial-dependent vasodilation and arterial stiffness and if these effects are obtained effectively using a standard once-daily regimen of aspirin.

6.10

PERIPHERAL SENSORY NEUROPATHY AND VASCULAR ANGIOGENIC FACTORS IN TYPE 2 DIABETES PATIENTS IN GHANA

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Background: Impaired angiogenesis may be amongst the possible mechanism underlining the development of peripheral sensory neuropathy (PSN) in type

2 diabetes (T2DM) patients.(1) Angiogenesis is regulated by circulating vascular growth factor, notably, angiopoietin (Ang)-1, Ang-2 and vascular endothelial growth factor (VEGF).(2) We studied the relationship between PNS and circulating vascular growth factors, Ang-1, Ang-2 and VEGF in T2DM patients.

Method: PNS was assessed by vibration perception threshold (VPT) using Horwell's neurothesiometer, and serum levels of Ang-1, Ang-2 and VEGF were also measured by Elisa in 107 T2DM patients and 93 nondiabetes subjects (controls). PNS was defined as VPT>25V.

Results: The overall prevalence of PNS was 11.2% higher in T2DM patients (10.1% vs. 1.1%, $p=0.012$) than controls. T2DM patients had higher mean VPT (12.1 ± 7.8 vs. 7.3 ± 3.8 V, $p<0.001$) than controls. Compared to those without PNS, PNS patients had lower Ang-2 levels [0.4 ($0.2 - 0.8$) vs. 0.8 ($0.4 - 1.1$) nmol/l, $p=0.03$] and higher VEGF levels [120 ($60.8 - 254.4$ vs. 59.4 ($17.2 - 146.8$), $p=0.037$], but no difference in Ang-1 levels. VPT was associated, positively with VEGF levels ($r=0.22$, $p=0.003$), and negatively with Ang-1 ($r=-0.17$, $p=0.024$), but not with Ang-2.

Discussion: Diabetes is associated with high prevalence of PNS and elevation of circulating vascular growth factors. PNS patients had imbalanced levels of circulating vascular growth factors, which may indicate impaired angiogenesis.

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7.1

THE DETRIMENTAL EFFECTS OF LIVE FIREFIGHTING ON ARTERIAL FUNCTION IN FIREFIGHTERS

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Background: Aging is associated with increased arterial stiffness and wave reflection, which is predictive of all-cause cardiovascular (CV) mortality (1-3). Firefighters have the highest cardiovascular mortality of any occupational group (4). High levels of heat stress, physical exertion, and elevated arterial stiffness (5) during/following firefighting provide a susceptible milieu for CV events.

Purpose: To describe the differential effects of age following live firefighting on cardiac, arterial function and wave reflection.

Methods: Firefighters aged 18-37yrs ($n=18$, YA) or 38-55yrs ($n=17$, MA) participated in a staged 12-minute live firefighting scenario. Blood pressures (BP), pulse wave analysis, pulse wave velocity (PWV) and hemodynamic measurements were obtained at rest, immediate and 30 minutes post-firefighting using an automated ambulatory blood pressure monitor (Mobil-O-Graph, I.E.M, Germany).

Results: YA increased heart rate and PWV more than MA in response to live firefighting ($p<0.01$). YA also decreased systemic arterial compliance ($p<0.01$) immediately post-firefighting more compared to MA, which returned to baseline values at 30-minutes. MA had higher PWV, total vascular resistance, and diastolic BP than YA ($p<0.01$). Systolic BP, pulse pressure, and reflective magnitude increased immediately post-firefighting for YA ($p<0.01$) but not in MA ($p>0.05$).

Conclusions: Young and MA firefighters exhibit differential cardiovascular responses to live firefighting. Although MA had higher PWV, diastolic BP and higher peripheral resistance they exhibited attenuated changes following live firefighting. Thus, arterial and hemodynamic parameters in younger firefighters appeared to change in a direction associated with increased risk to a greater degree than observed in older firefighters.

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7.2

POOR SLEEP QUALITY RELATED TO WORSE VASCULAR FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS

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Background: Poor sleep quality has been related to poor vascular function and higher risk of cardiovascular disease in the general population. Persons with multiple sclerosis (MS) exhibit a high cardiovascular risk, and report poor sleep quality. To date, the association between sleep quality and vascular health in MS has not been investigated. Objective: To investigate differences in vascular health between good and poor quality sleepers with MS. Methods: After a 10 minute rest in the supine position, resting heart rate (HR) and brachial blood pressure (BP) were collected. Aortic SBP, augmentation index (AIX), HR normalized AIX (AIX@HR75), subendocardial viability ratio (SEVR), end systolic pressure (ESP), and central pulse wave velocity (PWV) were measured with applanation tonometry in individuals with MS ($n=49$). Carotid intima-media thickness (IMT) and beta-stiffness were measured with carotid ultrasound, and peak forearm blood flow (FBF Peak) was measured with strain gauge plethysmography. Sleep quality was measured with the Pittsburgh Sleep Quality Index (>5 was categorized as poor sleep quality). Age was used as a covariate.

Results: AIX@HR75 and SEVR were different between groups, even with age as a covariate, suggesting higher vascular risk for the poor quality sleepers with MS.

Conclusions: This study shows that within the MS population, poor quality sleepers have a higher cardiovascular risk than good quality sleepers. Whether poor sleep raises their cardiovascular risk more than in the general population is an area of future investigation.

	Good quality sleepers with MS (n=23)	Poor quality sleepers with MS (n=26)	Effect of sleep quality	Effect of age		
	Mean (SD)	Mean (SD)	p	Partial p	Partial Eta ²	Partial Eta ²
N Female (%)	18 (75%)	22 (82%)				
PSQI score	2.9 (1.5)	9.7 (3.1)				
Age	44 ±13	52 ±10				
BMI	26.9 ±5.4	28.1 ± 5.6	.582	0.007	.584	0.007
HR	64 ±10	65 ± 8	.136	0.048	.014*	0.125
SBP	116 ±14	122 ±14	.384	0.017	.025*	0.105
DBP	72 ±9	74 ±10	.457	0.012	.559	0.007
MAP	86 ± 10	90 ±11	.403	0.015	.175	0.040
Aortic SBP	106 ±14	114 ±14	.223	0.032	.001**	0.200
AIX	21.1 ±13.7	30.0 ± 9.0	.082	0.064	<.001**	0.282
AIX@HR75	15.1 ± 12.0	25.5 ± 7.6	.007**	0.150	.001**	0.211
SEVR	158 ± 21	147 ±18	.026*	0.103	.160	0.042
ESP	96 ±12	103 ± 14	.213	0.034	.007**	0.148
PWVc	6.7 ± 1.4	7.5 ± 1.4	.200	0.036	.007**	0.146
PWVc/MAP	0.078 ± 0.014	0.084 ± 0.016	.376	0.017	.063	0.073
IMT	0.51 ± 0.11	0.53 ± 0.12	.571	0.007	<.001**	0.342
Beta	7.3 ± 2.3	8.1 ± 2.7	.785	0.002	.002**	0.193
FBF Peak	17.8 ± 4.4	17.2 ± 6.6	.988	0.000	.185	0.038

* $p<0.05$ ** $p<0.01$