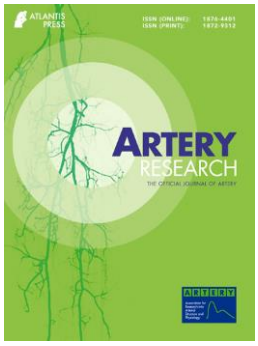

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6.7: FIRST EVIDENCE OF PULSATILE PRESSURE INTERACTION BETWEEN THE MACRO-VASCULATURE AND MICRO-VASCULATURE: PROOF-OF-CONCEPT BY ASSOCIATION WITH KIDNEY DYSFUNCTION AMONG PATIENTS WITH TYPE 2 DIABETES

Rachel Climie, Dean Picone, Sarah Blackwood, Ahmad Qasem, Stephen Rattigan, James Sharman

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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±12.7mmHg. PP>60mmHg was observed in 18,1%.Group with PP>60 mmHg was characterized by higher HbA1c (9.8±1.8vs8.4±2.0%) and stiffness gradient (1.4±0.4vs1.2±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>10m/s was observed in 27.2% of patients. Groups with PWV above and below 10m/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±11.6vs54.0±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

Rachel Climie [2], Dean Picone [1], Sarah Blackwood [1], Ahmad Qasem [3], Stephen Rattigan [1], James Sharman [1]
[1]*Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia*
[2]*Baker IDI Heart and Diabetes Institute, Melbourne, Australia*
[3]*University of New South Wales, Sydney, Australia*

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 (AIx) 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 AIx and AP were significantly related to micro-vasculature AIx (r=0.428. p=0.005 and r=0.545, p=0.004 respectively). Micro-vascular (but not macro-vascular) AIx 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

Davide Agnoletti [1], Filippo Valbusa [1], Ari Lieber [2], Guido Arcaro [1], Michel Safar [2], Jacques Blacher [2]
[1]*Department of Internal Medicine, Sacro Cuore Hospital, Negrar, Italy*
[2]*University of Paris Descartes, Centre de Diagnostic, Hotel-Dieu Hospital, Paris, France*

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 [AIx] 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-R2=0.14), gender (partial-R2=0.27), heart rate (partial-R2=0.26), angiotensin blockers treatement (partial-R2=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

Liv Vernstroem Hald, Anne-Mette Hvas, Erik Lerkevang Grove, Esben Laugesen, Kristian Loekke Funck, Per Loegstrup Poulsen
Aarhus University Hospital, Aarhus, Denmark

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 vasodilatation 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

Kwame Yeboah, Daniel A. Antwi, Ben Gyan
University of Ghana, Accra, Ghana

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