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### **P60: INFLUENCE OF ANGER ON ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH RECENT MYOCARDIAL INFARCTION**

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**Conclusions:** Arterial stiffness is a useful and feasible parameter to be measured in community pharmacies. It allows for a holistic service, adding to other CV risk predictors already available.

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#### P60

##### INFLUENCE OF ANGER ON ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH RECENT MYOCARDIAL INFARCTION

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**Background:** The literature demonstrates that anger is associated with cardiovascular disease, but the underlying physiological mechanisms remain undefined. Endothelial dysfunction, present in atherosclerosis, has also been associated with anger.

**Purpose:** To examine the association between anger and endothelial function measured by flow-mediated dilatation (FMD) of the brachial artery.

**Methods:** Patients were assessed during hospitalization after acute myocardial infarction answered the Spielberger Trait-State Anger inventory (STAXI). After discharge, patients were submitted to ultrasound of the brachial artery, the FMD technique, which was calculated by the maximum percentual of change in the diameter of the brachial artery from baseline to peak of dilation after deflation of the cuff.

**Results:** The study included 90 patients, 86% caucasian, with  $57 \pm 10$  years old, 73% male, 48% smokers, 57% with hypertension, 32% with dyslipidemia, 23% with diabetes, and 21% with a family history of arterial disease coronary artery disease. The mean dilation of this group was  $6.70 \pm 4.64$ . The presence of endothelial dysfunction was evaluated by the percentage of arterial dilation below 8.0%. In the multivariate analysis, only the anger reaction was associated with endothelial dysfunction. At each point of anger reaction increases 31% the chance of endothelial dysfunction ( $p = 0.008$ ).

**Conclusions:** In this sample of infarcted patients with anger score below average, the anger reaction is related to endothelial dysfunction.

#### P61

##### ARTERIAL STIFFNESS IS ASSOCIATED WITH AORTIC VALVE CALCIFICATIONS

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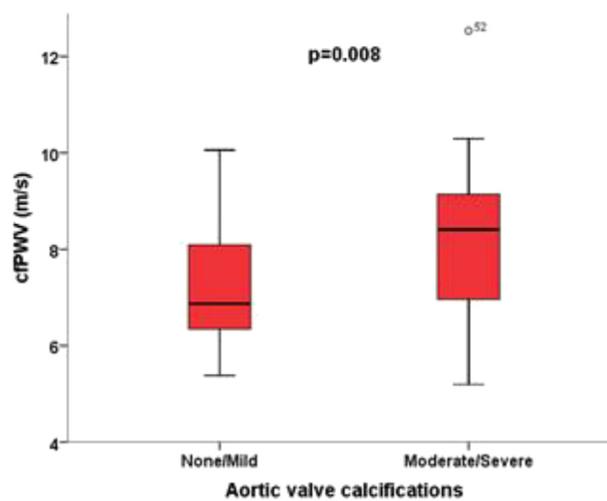
**Purpose/Background/Objective:** Arterial stiffness and aortic hemodynamics are independent predictors of adverse cardiovascular events. Indications for transcatheter Aortic Valve Implantation (TAVI) are increasing in number and Aortic Valve Calcifications (AVC) are an important prognostic factor of TAVI. We sought to investigate the associations between AVC and aortic vascular function/hemodynamics.

**Methods:** Fifty-two high-risk patients (mean age  $80.4 \pm 8.5$  years, 27 male) with severe symptomatic aortic stenosis undergoing TAVI were included. Arterial stiffness was estimated through carotid-femoral pulse wave velocity (cfPWV) and brachial-ankle pulse wave velocity (baPWV). Aortic hemodynamics were also measured. Measurements were conducted prior to the implantation and at discharge. In all patients, a native and contrast-enhanced

multislice cardiac computed tomography were performed pre-interventionally. AVC were then graded semi-quantitatively.

**Results:** Group 1 (subjects with none/mild AVC,  $n = 29$ ) did not significantly differ on age, gender and body-mass index compared to group 2 (subjects with moderate/severe AVC,  $n = 23$ ). From the traditional cardiovascular risk factors, only hypertension ( $p = 0.008$ ), coronary artery disease ( $p = 0.016$ ), atrial fibrillation ( $p = 0.075$ ) and insulin-dependent diabetes mellitus ( $p = 0.068$ ) were more statistically or showed a significant trend to be more prevalent in group 2. Group 2 had significantly higher both cfPWV and baPWV ( $8.3 \pm 1.7$  vs  $7.2 \pm 1.2$  m/s and  $1750 \pm 484$  cm/s vs.  $2101 \pm 590$  cm/s with  $p = 0.008$  and  $p = 0.022$  respectively) compared to Group 1. (Figure) There was no difference in wave reflections indices between the two groups.

**Conclusions:** Our study shows that in patients with aortic stenosis there is a correlation between an increase in aortic stiffness and damage of aortic valvular leaflets as well as calcifications.



#### P62

##### DAPAGLIFLOZIN PRESERVES RENAL VASODILATING CAPACITY IN HYPERTENSIVE PATIENTS WITH TYPE 2 DIABETES

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**Aim:** Mechanisms through which SGLT-2 inhibitors achieve cardiovascular and renal protection are still unknown. We investigated whether dapagliflozin modulates Na and water balance and systemic and renal vascular parameters like endothelial function, arterial stiffness and renal vasodilating capacity.

**Methods:** 40 type2-diabetic hypertensive patients were studied at baseline (V0) and after four weeks (V1) of dapagliflozin 10 mg (Dapa,  $N = 20$ ) or hydrochlorothiazide 12,5 mg (HCT,  $N = 20$ ), collecting blood and urinary samples for routine analyses, plasma renin activity, aldosterone, catecholamines and 24 hour-urinary electrolytes. Flow-mediated dilation of the brachial artery (FMD), baseline (RI) and dynamic renal resistive index (DRIN), carotid-femoral pulse-wave velocity (PWV) and Augmentation Index (AIx) were also measured. **Results:** Both Dapa and HCT marginally lowered systolic and diastolic BP values and did not change blood fasting glucose. Serum magnesium concentration significantly rose in Dapa group (from  $1.88 \pm 0.27$  to  $2.01 \pm 0.22$  mg/dl,  $p = 0.02$  for time\*treatment interaction), while magnesuria was unchanged. 24h diuresis and glycosuria and osmolar clearance increased in Dapa ( $p < 0.001$ ), with no changes in sodiuria and creatinine clearance. Dapa induced also a rise in aldosterone ( $p = 0.02$ ). Nor DAPA neither HCT modified FMD, AIx and PWV. Interestingly, in Dapa group DRIN remained unmodified, while tended to increase in HCT group ( $p = 0.05$ ).

**Conclusions:** 4-week Dapa treatment did not significantly influence BP, glucose and systemic indices of vascular function. However, in comparison to HCT, renal vasodilating capacity was preserved after Dapa, indicating a selective effect on renal vascular function, which may act as nephroprotective mechanism. Furthermore, the increase in serum magnesium might contribute to cardiovascular protection.