assess the association of ED parameters on statin therapy with genetic markers of inflammation.

Methods: We include 97 T2D patients with first prescribed atorvastatin 10-20 mg: M/F 26/71; mean age 57 yr., genotyped for a complex of polymorphic markers (*PPARG2 Pro12Ala, TNFα G(308)A and G(238)A, LIPC C(-514)T, ACE I/D, SLCO1B1 Val174Ala*). For ED evaluation, we performed pulse wave analysis with reactive hyperemia by peripheral arterial tonometry before and after 12 month on statin therapy. The genotypes were identified using PCR in real time with the TaqMan probes. Statistic analysis was evaluated using the Mann-Whitney and Wilcoxon tests, p <0,05.
Results: We observed significant differences in amplitude of postocclusive wave (Apw) before and after 12 month of statin therapy. The % of Apw improvement didn't depend on age, diabetes duration, basal and reached levels of lipids and HbA1c but genotypes distribution of *TNF-a* gene markers *G(238)A* and *G(308)A. The* carries *GG G(238)A* and *GA G(308)A* patients had significantly greater Apw increase compared with *GA G(238)A* and *GG G(308)A* patients (+8,16 % vs. -0,93%, p=0,04; +44% vs. -4.4%, p=0,004, respectively). There was no statistically significant association between ED and other studied markers.
Conclusions: Genetic factors might play a significant role in ED development. Significant association of *TNF-α* gene polymorphism with ED in T2D suggests an important role of inflammation in the genesis of MVD.

EAS16-0873, DYSLIPIDEMIAS: ENDOTHELIAL DYSFUNCTION. BENEFICIAL EFFECT OF AZILSARTAN AND AMLODIPINE ON ENDOTHELIAL FUNCTION IN HYPERTENSIVE PATIENTS

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Objectives: Azilsartan (AZ) reportedly exerts a greater anti-hypertensive effect as compared to other angiotensin II receptor blockers (ARB); however, it remains unclear whether AZ improves endothelial function in hypertensive patients. We therefore performed a prospective, randomized crossover trial in which AZ (20 mg/day) and calcium channel blocker, amlodipine (AM, 5 mg/day), were compared in 24 hypertensive patients.

Methods: Endothelial function was evaluated by flow-mediated vasodilation (FMD) in brachial artery using ultrasonography.
Results: Both treatments for 3 months achieved comparable blood pressure (BP)-lowering both at office and home, in which were not different between AZ and AM. AZ significantly increased plasma renin activity (PRA) and decreased aldosterone concentration (PAC), indicating AZ did not provoke "aldosterone escape phenomenon". In contrast, AM did not affect PRA and PAC. Finally, FMD test revealed that both drugs significantly improved endothelial function; however, AZ appeared to be superior as compared to AM (baseline, AZ, AM; 3.5±3.1, 8.8±4.3, 7.5±6.3 %; p<0.0001 vs. AZ, p=0.02 vs. AM).
Conclusions: Despite the comparable BP-lowering effect between AZ and AM, AZ may have a greater beneficial property on endothelial function. Further studies assessing effects on markers for inflammation and oxidative stress will clarify the underlying mechanisms.

EAS16-0407, DYSLIPIDEMIAS: ENDOTHELIAL DYSFUNCTION. CIRCULATING PROGENITOR CELLS IN HYPERTENSIVE SUBJECTS: EFFECTIVENESS OF A TREATMENT WITH OLMESARTAN IN IMPROVING CELL NUMBER AND MIRS PROFILE BESIDES EXPECTED PHARMACOLOGICAL EFFECTS

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Objectives: CD34+ circulating progenitor cells (CD34+CPCs) are a population of multipotent spare cells able to delay the development of atherosclerosis and cardiovascular disease (CVD) in conditions of increased CV risk. Increased reactive oxygen species (ROS), a common feature of CV risk factors including hypertension, may be toxic for cells. MicroRNAs (miRs) 221 and 222 have been shown to participate in differentiation and proliferation of CD34+CPCs, inhibiting cell migration and homing; miR221/222 are increased and associated with cell number and ROS in CD34+CPCs from hypertensive patients without additional risk for CAD. Moreover, miR221/222 modulate different genes regulating angiogenesis and inflammation.The aim of the present study was to evaluate whether in hypertensives a treatment with olmesartan may modify the number of CD34+CPCs and the levels of miR221/222 and ROS.

Methods: We evaluated CD34+CPC number, intracellular miR221/222 and ROS levels, arterial stiffness and echocardiographic indices at baseline (T0) and after a six-months treatment with olmesartan, 20 mg/die (T1) in 57 hypertensives with no additional risk factor for CAD, and in 29 healthy controls (baseline); fibrinogen, CRP, glucose and lipid profile were also evaluated.
Results: At T1, systolic and diastolic blood pressure, ROS and miR221/222 were significantly decreased (all p <0.001) with respect to T0, and cell number was increased (p<0.001). CRP and fibrinogen levels also were reduced (p<0.001), as were arterial stiffness indices.
Conclusions: Olmesartan is effective in reducing miRs and ROS levels in CD34+CPCs from hypertensives, as well as in increasing CD34+CPC number, besides its expected pharmacological effects.

EAS16-0121, DYSLIPIDEMIAS: TREATMENT. EFFECTS OF DIFFERENT DOSES OF ATORVASTATIN ON PLATELET AGGREGATION, ENDOTHELIAL FUNCTION, LEVELS OF SEROTONIN, ANGIOTENSIN II, BLOOD PRESSURE, CEREBRAL BLOOD FLOW IN HYPERTENSIVE PATIENTS WITH HYPERLIPIDEMIA

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Objectives: To study the effects of atorvastatin (A) at different dose on platelet aggregation (PA), levels of end-metabolites of nitric oxide (NOx), endothelin-1 (ET-1), serotonin (S), angiotensin (AT) II, uric acid (UR), blood pressure (BP), and cerebral blood flow (CBF) in patients (pts) with arterial hypertension (AH) and hyperlipidemia (HLP).

Methods: The 34 pts were randomized into groupsI, which received A in doses of 10 mg or 40 mg, respectively, for 12 wks. The PA was studied by the Born G method after stimulation with ADP in concentration of 2,0 μM. 24-hour ambulatory BP monitoring was performed according to standard protocol and CBF were searched by Doppler ultrasonography.
Results: After 12 wks of therapy A the ADP-induced PA decreased at A 10 mg (-31,6%, p<0,01) and A 40 mg (-67,2%, p<0,01); the levels NOx increased by 15% (p<0.05) at A 10 mg and A 40 mg. Significantly decreased levels of ET-1 (-4,8%, p<0,05), S (-28,6%, p<0,05), ATII (-7,1%, p<0,05) and UR (-5,9%, p<0,05) were found only after therapy of A 40 mg. After12 wks therapy of A 40 mg average variability of systolic BP at the night significantly reduced. In pts taking the A in dose 10 and 40 mg after 12 wks there were some improvement of CBF in carotid artery: end-diastolic velocity increased by 10,5% (p<0,05), index of Stuart and pulsatility index decreased by 6% (p<0,05) and 8.4% (p<0.05), respectively.
Conclusions: A showed dose-depended effects in the inhibition of PA, increase in NO bioavailability, improvement of CBF in carotid artery. Only at intensive therapy of A 40 mg there were significantly decreased levels of ET-1, S, ATII, UR and BP.

EAS16-0123, DYSLIPIDEMIAS: TREATMENT. EFFECT OF STATIN ON FASTING BLOOD SUGAR LEVEL; COMPARISONS OF KINDS OF STATINS AND ASSOCIATED PARAMETERS

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