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### **P4.51: NONINVASIVE EVALUATION OF STRUCTURAL AND FUNCTIONAL CHANGES IN THE HEART AND LARGE ARTERIES IN PATIENTS WITH PRIMARY ALDOSTERONISM**

A. Paini, M. Salvetti, F. Bertacchini, C. Agabiti Rosei, C. Aggiusti, D. Stassaldi, G. Rubagotti, A. Comaglio, F. Mattavelli, R. Germano, E. Agabiti Rosei, M.L. Muiesan

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bisoprolol alone. **PARICIPANTS:** 50 patients with stable angina and COPD were included (88% men, mean age  $62.8 \pm 7.2$  years). All patients received bisoprolol, the dose of which was titrated until the clinical signs of intolerance. Then, the patients were randomized into two groups: patients of the first group continued to take bisoprolol and patients of the second group were added ivabradine (5–15 mg).

**Results:** Combination therapy resulted in further decrease of heart rate (HR) to an average of  $62 \pm 4$  bpm over 6 month of follow-up. This was associated with additional decrease of the number of angina attacks (by  $4.7 \pm 4.4$  per week vs.  $2.5 \pm 4.7$ ,  $p < 0.05$ ) and increase of quality of life ( $p < 0.05$ ). Only in combination therapy group there was also the decrease of broncholytics consumption (from  $2.9 \pm 3.2$  to  $1.9 \pm 2.7$  per week,  $p < 0.05$ ). Pulse wave velocity (PWV) estimated by Arteriograph (TensioClinic «Meditech») decrease significant in both groups, but no difference delta PWV between groups.

**Conclusion:** The combination of tolerable doses of bisoprolol and ivabradine is safe and allows to achieve adequate HR decrease. This is associated with maximal antianginal effect, decrease in the need for broncholytic therapy, improvement of the quality of life compared with the treatment with bisoprolol alone. We did not find the influences of ivabradine on the arterial stiffness.

#### P4.51

##### NONINVASIVE EVALUATION OF STRUCTURAL AND FUNCTIONAL CHANGES IN THE HEART AND LARGE ARTERIES IN PATIENTS WITH PRIMARY ALDOSTERONISM

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**Background** Only few studies, in small groups of patients, have evaluated large arteries alterations in primary aldosteronism (PA). In addition, the assessment of cardiac and vascular involvement has not been simultaneously performed.

**Aim** of the study was to evaluate cardiac, vascular and renal complications in patients with PA.

**Patients and Methods:** 70 patients with PA (age  $49 \pm 6$  years, 27 F) and 70 essential hypertensives (EH) matched for age, sex, BMI and BP, underwent laboratory examinations, cardiac and carotid ultrasound and PWV.

**Results:** no differences in age, sex, BMI, BP and HR were observed. Cholesterol was lower in PA ( $195 \pm 37$  vs  $215 \pm 33$  mg/dl,  $p < 0.05$ ); no difference in tryglicerides, glucose and uric acid was observed. In PA eGFR(MDRD) was greater in comparison to EH ( $96 \pm 22$  vs  $89 \pm 17$  mL/min/1.73m<sup>2</sup>,  $p = 0.05$ ). LV mass index (LVMI) and relative wall thickness (RWT) were significantly greater in PA vs EH (LVMI  $42 \pm 13$  vs  $34 \pm 7$  g/m<sup>2</sup>,  $p < 0.001$  and RWT  $0.34 \pm 0.08$  vs  $0.31 \pm 0.04$ ,  $p < 0.001$ ). At TDI Emvel was significantly lower, and Evel/Em vel was significantly higher in PA ( $9.5 \pm 2.9$  vs  $10.5 \pm 2.7$ ,  $p < 0.05$  and  $9.2 \pm 4.4$  vs  $7.6 \pm 2.7$ ,  $p < 0.05$ , respectively). No difference was observed in carotid prevalence of thickening or plaques and in IMT (Meanmax  $1.02 \pm 0.25$  vs  $0.98 \pm 0.18$ ,  $p$  ns, CBMMax  $1.05 \pm 0.26$  vs  $1.01 \pm 0.16$ ,  $p$  ns), as well as in PWV ( $10.8 \pm 2.1$  vs  $10.8 \pm 1.6$  m/sec,  $p$  ns).

**Conclusions:** In this large group of patients with PA a significant increase in LVMI and concentric geometry, associated with a worse diastolic function were observed. Opposite to previous findings, no difference in aortic stiffness and carotid structure was observed.

#### P4.52

##### CORONARY ARTERY DISEASE AND STROKE IN TYPE 2 DIABETIC PATIENTS: POSSIBLE ROLE OF A RAISED CENTRAL PULSE PRESSURE

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**Background:** Whether large artery static and dynamic properties are different in type 2 diabetics with coronary or cerebrovascular disease (DM-CVD) as compared to patients free of clinical complications (DM) is debated. **Aim:** To evaluate common carotid (CCA) IMT, local and aortic stiffness in DM-CVD as compared to DM and controls (CTRL).

**Methods:** thirty-five nondiabetic normotensive CTRL, 130 diabetics without complications (DM) and 25 diabetics with CAD and/or previous stroke/TIA (DM-CVD) (Tab 1) underwent radiofrequency (RF)-based ultrasound (QIMT® and QAS®, Esaote) of CCA, that provides automatic measurement of far-wall IMT, local stiffness ( $\beta$ -index) and contour wave analysis yielding local pulse pressure (PPc). Carotid-femoral pulse-wave velocity (PWV) was also measured (Complior).

**Results:** Adjusting for sex, age and smoking habit, CTRL and diabetics differed significantly ( $p < 0.05$ ) in PWV ( $9.0 \pm 1.9$  vs.  $11.4 \pm 2.5$  m/s), CCA IMT ( $619 \pm 146$  vs.  $736 \pm 169$   $\mu$ m) and PPc ( $37 \pm 9$  vs.  $46 \pm 14$  mmHg), but not in  $\beta$ -index ( $10.1 \pm 2.9$  vs.  $12.5 \pm 4.9$ ;  $p = 0.23$ ). Within diabetics, after adjustment for confounders, DM-CVD showed higher ( $p$  at least 0.05) PPc but not CCA IMT,  $\beta$ -index and brachial PP (PPb) than DM (Tab 2).

Table 1.

Groups	Age	BMI	Glycemia	SBPb	PPb
CTRL	$56 \pm 8^*$	$26 \pm 4^*$	$88 \pm 10^*$	$123 \pm 12^*$	$46 \pm 8^*$
DM	$62 \pm 8$	$29 \pm 5$	$146 \pm 42$	$136 \pm 18$	$57 \pm 16$
DM-CVD	$66 \pm 7$	$29 \pm 4$	$138 \pm 42$	$136 \pm 23$	$62 \pm 18$

Table 2.

Groups	PWV	IMT	$\beta$ -index	SBPc	PPc
CTRL	$9.0 \pm 1.9^*$	$619 \pm 146^*$	$10.1 \pm 2.9^*$	$115 \pm 12^*$	$37 \pm 9^*$
DM	$11.3 \pm 2.4$	$726 \pm 173$	$12.4 \pm 4.3$	$123 \pm 16$	$44 \pm 13^*$
DM-CVD	$12.0 \pm 3.5$	$794 \pm 129$	$14.6 \pm 6.8$	$127 \pm 19$	$52 \pm 16$

\*:  $p < 0.05$  in CTRL vs DM and DM-CVD; °:  $p < 0.05$  in DM vs DM-CVD

**Conclusions:** central PP appears to be the only structural/functional large artery measure significantly different in diabetic patients with or without cardiovascular complications, despite similar glucose levels and BMI. Contour wave analysis by ultrasound provides additional information beyond IMT and local stiffness indices.

#### P4.53

##### IMMEDIATE EFFECTS OF SUBMAXIMAL EFFORT ON PULSE WAVE VELOCITY IN PATIENTS WITH MARFAN SYNDROME

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Marfan syndrome (MS) is a dominant autosomal disease caused by mutations in chromosome 15, the locus controlling fibrillin 1 synthesis, and may exhibit skeletal, ocular, cardiovascular, and other manifestations. Pulse wave velocity (PWV) is used to measure arterial elasticity and stiffness and is related to the elastic properties of the vascular wall. Since the practice of exercise is limited in MS patients, it was of interest to analyze the acute effect of submaximal exercise on aortic distensibility using PWV and other hemodynamic variables in patients with MS with either mild or no aortic dilatation. PWV and physiological variables were evaluated before and after submaximal exercise in 33 patients with MS and 18 controls. PWV was  $8.51 \pm 0.58$  at rest and  $9.10 \pm 0.63$  m/s at the end of exercise ( $P = 0.002$ ) in the group with MS and  $8.07 \pm 0.35$  and  $8.98 \pm 0.56$  m/s in the control group, respectively ( $P = 0.004$ ). Comparative group analysis regarding PWV at rest and at the end of exercise revealed no statistically significant differences. The same was true for the group that used  $\beta$ -blockers and the one that did not. The final heart rate was 10% higher in the control group than in the MS group ( $P = 0.01$ ). Final systolic arterial pressure was higher in the control group ( $P = 0.02$ ). PWV in MS patients with mild or no aortic dilatation did not differ from the control group after submaximal effort.