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P6.19: GENDER DIFFERENCES OF ARTERIAL STIFFNESS AND CENTRAL BLOOD PRESSURE IN PATIENTS WITH ARTERIAL HYPERTENSION AND THE INFLUENCE OF MENOPAUSE

Valéria Costa-Hong, Henrique Muela, Allan Sales, Luiz Bortolotto*

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to cardiovascular risk factors remain unclear. We attempted to compare the contribution of cardiovascular risk factors to the variance in cIMT and WLR. **Methods:** Noninvasive measurements of cIMT and WLR were made with high-resolution ultrasonography in 5,983 subjects. They were male aged 40 to 55 and female aged 50 to 65 without previous cardiovascular events, participating in the Lithuanian High Cardiovascular Risk primary prevention program. We performed a multiple linear regression on cIMT and WLR incorporating traditional and less traditional cardiovascular risk factors.

Results: Mean left and right cIMT was 0.66 ± 0.12 mm and 0.65 ± 0.11 mm respectively, whereas mean WLR was 0.092 ± 0.015 . We found that cardiovascular risk factors could explain 8.9% of left cIMT and 8.3% of right cIMT. Strikingly, traditional and less traditional factors (namely age, male sex, LDL/HDL ratio, mean arterial blood pressure and triglyceride) contributed to a significantly larger proportion of WLR variance, amounting to 14.2%.

Conclusions: Adjustment for carotid lumen diameter in analyses evaluating common carotid artery intima-media thickness should be considered. The precise role of WLR as an ultrasound marker of subclinical atherosclerosis remains a topic of interest for future research.

P6.18

CARDIOVASCULAR TARGET ORGAN DAMAGE IN PREMENOPAUSAL SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS AND IN CONTROLS; ARE THERE ANY DIFFERENCES?

A. Paini^{1,*}, L. Andreoli², M. Salvetti¹, F. Dall'Ara², S. Piantoni², C. Donini¹, C. Agabiti Rosei¹, F. Bertacchini¹, D. Stassaldi¹, E. Agabiti Rosei¹, A. Tincani², M. L. Muijsan¹

¹Internal Medicine, University of Brescia, Italy

²Rheumatology, University of Brescia, Italy

Background: In patients with Systemic lupus erythematosus (SLE) a greater prevalence of structural and functional cardiovascular (CV) alterations has been described, possibly explaining the higher incidence of CV events, as compared to subjects matched for age and sex.

Aim of this study was to analyze the presence of target organ damage in premenopausal women with SLE and in controls matched not only for demographic characteristics but also for other cardiovascular risk factors.

Subjects and methods: 4 patients with SLE clinically stable (SLEDAI score 2.5 ± 1.5) (age 32 ± 7 years, range 19-44) and 34 controls matched for sex, age, BMI, clinic blood pressure (BP) and antihypertensive treatment (if present), underwent 24 hours BP monitoring, echocardiography with tissue Doppler analysis (TDI) for the evaluation of left ventricular (LV) structure and of systolic and diastolic function, carotid ultrasound for intima-media thickness (IMT), carotid distensibility measurement, and pulse wave velocity measurement (PWV).

Results: By definition no difference was observed for age, sex, BMI and clinic BP values and a similar Framingham risk score was observed between SLE and controls (1.3 ± 2.7 vs 1.5 ± 2.3 , $p = ns$). No significant differences were observed for all echocardiographic parameters except LV longitudinal systolic function (Sm), an early index of LV systolic dysfunction (see Table). Carotid IMT and distensibility, as well as PWV and the prevalence of an abnormal aortic stiffness were both similar in the two groups. At the logistic analysis, PWV was independently associated with LV mass in controls and with the steroid weekly dose in SLE patients.

Conclusions: In patients with SLE and low activity index of the disease we did not observe significant vascular alterations as compare to controls with similar cardiovascular risk. The early LV systolic impairment observed in this group of patients needs confirmation in larger cohorts.

P6.19

GENDER DIFFERENCES OF ARTERIAL STIFFNESS AND CENTRAL BLOOD PRESSURE IN PATIENTS WITH ARTERIAL HYPERTENSION AND THE INFLUENCE OF MENOPAUSE

Valéria Costa-Hong¹, Henrique Muela¹, Allan Sales², Luiz Bortolotto^{1,*}

¹Heart Institute (Incor), University of São Paulo Medical School, Hypertension Unit, São Paulo, São Paulo, Brazil

²Heart Institute (Incor), University of São Paulo Medical School, Cardiovascular Rehabilitation and Exercise Physiology Unit, São Paulo, São Paulo, Brazil

Introduction: In general population women seems to have greater arterial stiffness and central blood pressure (BP), measured by augmentation index (Alx), than men, but in hypertension this condition is poorly studied.

Objectives: To evaluate differences of central BP and arterial stiffness between men and women with arterial hypertension and the influence of postmenopausal status.

Methods: We studied 128 subjects with stage 1-3 arterial hypertension, mean age 51.1 ± 11 years, 48% males, BP = $141 \pm 24 / 87 \pm 13$ mmHg, hypertension duration 10.1 ± 8 years. Noninvasive central aortic BP and wave form characteristics (Alx and Alx corrected by heart rate of 75 bpm – Alx75) were synthesized from radial arterial pressure waves (applanation tonometry) by SphygmoCor®. Brachial BP was obtained by an automatic device (OMRON®).

Results: Brachial systolic BP was higher (145 ± 26 vs. 136 ± 20 mmHg, $p = 0.04$) and, weight (76.2 ± 14 vs. 86.4 ± 13.2 , $p < 0.01$) and height (1.59 ± 0.06 vs. 1.72 ± 0.08 , $p < 0.01$) were lower in females than in males. Central systolic BP (137 ± 30 vs 125 ± 23 mmHg, $p < 0.01$), Alx (32.7 ± 9.8 vs 19.5 ± 11.7 , $p < 0.01$), Alx75 (29.6 ± 6.9 vs 18.0 ± 9.3 , $p < 0.01$) were higher in females, even after adjustments for weight, height and systolic BP. Postmenopausal status was present in 70% of females and mean age of menopause beginning was 47.8 years. Women at postmenopausal status older than 48 years showed worse Alx (35.3 ± 9.4) than younger women (26.8 ± 8.9) and also than men (23.2 ± 12.4) at same age ($p < 0.05$).

Conclusions: Hypertensive females have higher brachial and central systolic BP than hypertensive males. Arterial stiffness is higher in hypertensive females than in men, at all ages, and in postmenopausal status is worse than in fertile period.

P6.20

ROLE OF ALTERED VASCULAR REACTIVITY IN THE PATHOPHYSIOLOGY OF ACUTE MOUNTAIN SICKNESS

Rosa Maria Bruno^{1,*}, Guido Giardini³, Sandro Malacrida², Bruna Catuzzo³, Sabina Armenia⁴, Lorenzo Ghiadoni⁴, Raffaele Brustia⁵, Paolo Laveder², Paolo Salvi⁶, Emmanuel Cauchy⁷, Lorenza Pratali¹

¹Institute of Clinical Physiology, CNR, Pisa, Italy

²University of Padua, Padua, Italy

³Valle d'Aosta Regional Hospital, Aosta, Italy

⁴University of Pisa, Pisa, Italy

⁵Hôpital Saint-Antoine, Paris, France

⁶Istituto Auxologico Italiano, Milan, Italy

⁷IFREMMONT, Chamonix, France

Purpose: The aim of this study is to explore the physiological vascular adaptation to exposure to high altitude and to test the hypothesis that its impairment might play a role in the pathophysiology of acute mountain sickness (AMS).

Methods: 34 healthy volunteers (age 38 ± 11 years, 13 women) were studied at the sea-level and after passive ascent to 3842 m (Aiguille du Midi, France). Blood pressure (BP), O₂ saturation (SO₂), endothelial function (flow-mediated dilation, FMD), carotid distensibility coefficient (DC), carotid-femoral pulse wave velocity (PWV), peak systolic velocity in the middle cerebral artery (MCA-PSV) were performed at sea level (T0) and after 4-h hypobaric hypoxia (T1). AMS was defined as a Lake-Louise Score > 5 after 24-h hypobaric hypoxia (T2).

Results: At T2 12 individuals developed AMS (AMS+). AMS+ had a greater SO₂ worsening at T1 as compared to AMS- (AMS+: 97.2 ± 1.2 to 79.3 ± 5.8 %; AMS-: 97.3 ± 1.2 to 83.1 ± 5.7 %, $p = 0.03$), with similar heart rate increase and unchanged BP. FMD was significantly reduced in AMS+ (5.75 ± 3.01 to 3.27 ± 1.87 %, $p = 0.04$), but not in AMS- (4.74 ± 2.47 to 4.02 ± 2.36 %). Mean carotid diameter was increased at T1 in both groups. DC tended to be increased in AMS- but not in AMS+, while PWV was unchanged. MCA-PSV was increased in AMS-, but not in AMS+.

Conclusions: In healthy asymptomatic individuals exposed to high altitude, conduit artery endothelial function is preserved in the cerebral district vasodilatation, increased elasticity and blood flow occurs. This compensatory response is early blunted in AMS+, before symptoms onset, thus suggesting a pathogenetic role.

P7.1

INCREASED PLATELET REACTIVITY IS RESPONSIBLE OF MODIFICATIONS OF THROMBIN GENERATION IN PATIENTS WITH UNCONTROLLED ARTERIAL HYPERTENSION

Jeremy Lagrange^{1,*}, Yvonne Weihert¹, Susanne Karbach^{1,2}, Philip Wenzel^{1,2}

¹Center for Thrombosis and Hemostasis, Mainz, Germany

²Department of Medicine 2 University Medical Center, Mainz, Germany

Background: Hypertensive crisis is an extreme phenotype of increased blood pressure that can lead to organs failure and thrombotic complications. Recently, we were able to show an angiotensin II driven FXI-thrombin amplification loop leading to vascular injury in experimental hypertension.