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ABSTRACTS

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P13 Physiopathology 7

P13.01 THE IMPACT OF ACUTE SYSTEMIC INFLAMMATION ON ARTERIAL FUNCTION OF PATIENTS WITH STABLE ANGINA PECTORIS: ADDING FUEL TO THE FIRE WITHIN?

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Purpose: According to recent evidence, acute inflammation is associated with transiently impaired vascular function and aortic compliance in healthy adults. The relationship between acute inflammatory stimuli and arterial stiffness in patients with stable angina pectoris (SAP) has not been investigated yet.

Methods: We studied the effect of an acute inflammatory stimulus on wave reflections and aortic stiffness, in 13 patients with SAP (mean age 49.4 years, 13 men) and in a control group consisting of 13 healthy adults (mean age 47.2 years, 7 men). We used Salmonella Typhi vaccine to induce an acute, mild, transient and systemic inflammatory response in both groups. cfPWV and Alx were measured at baseline and 8h after vaccination.

Results: Inflammation led to a decrease in Alx, 8h after vaccination in both groups (from 26.2% to 20% in SAP patients, $P<0.05$, from 31.2% to 22.9% in healthy adults, $P<0.05$). cfPWV increased in the healthy group after 8h (from 6.70m/sec to 6.96m/sec, $P<0.05$), however in the SAP group there was no change in aortic stiffness 8h after vaccination (from 8.33m/sec to 8.01m/sec, $P=ns$).

Conclusions: Acute systemic inflammation has an effect on wave reflection in both healthy and SAP patients, however there is a significant increase of large artery stiffness only in the healthy group, indicating a different behaviour of large arteries in the SAP group, probably due to the atherosclerotic burden present on their arteries. This finding is particularly important for determining possible links between inflammation and arterial function in the setting of coronary heart disease.

P13.02 VENTRICULO-VASCULAR COUPLING IS IMPAIRED IN PATIENTS WITH TYPE-II-DIABETES MELLITUS AND RESISTANT HYPERTENSION

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Characteristic	Controlled hypertension	Resistant hypertension	P	Adjusted P
Sex (male/female)	15/9	27/7		
Age (years)	62±10	64±9	0.49	
Body mass index (kg/m ²)	34±7	35±5	0.311	0.205
Length of disease (years)	10.4±6.3	14±7	0.023	0.028
Pulse pressure (mmHg)	50±8	66±9	<0.0001	<0.0001
Mean arterial pressure (mmHg)	88±4	96±7	<0.0001	<0.0001
Heart rate (bpm)	74±11	71±13	0.312	0.238
Pulse wave velocity (m/s)	9.7±3	12.1±5	0.042	0.385
Characteristic impedance	0.07±0.03	0.1±0.1	0.031	0.045
EA (mmHg/ml)	1.63±0.5	1.86±0.6	0.124	0.057
EMAX (mmHg/ml)	2.7±1	2.1±0.9	0.023	0.077
EA/EMAX	0.7±0.3	1.1±0.5	0.003	0.005
KLV (mmHg/ml)	0.16±0.1	0.3±0.2	0.015	0.04
Ejection fraction (%)	55±10	45±11	<0.0001	0.001
E/É	9±3	11±4	0.006	0.007

Objective: To examine if ventriculo-vascular coupling (VVC) is impaired in patients with resistant hypertension (RH) and type-II-diabetes mellitus.

Methods: We included 87 patients. RH was defined according to guidelines from the American Heart Association.

Echocardiography was performed using GE Vivid 7 and pulse wave analysis using Sphygmocor. All examinations were done under standardized conditions. All analyses were done blinded offline using Echopac and customized software.

VVC was estimated from stroke volume, end systolic pressure and -volume using the formula E_A/E_{MAX} . Left ventricular chamber stiffness (K_{LV}) was estimated from mitral valve deceleration time (MV dect) using the formula $70(MV \text{ dect}-20)^2$.

All statistical analyses were adjusted for sex, age, length of disease and heart rate using multiple linear regression.

Results: 34 patients had RH and 24 had controlled hypertension (CH) leaving 29 with uncontrolled hypertension. See table 1 for patient characteristics. Patients were comparable with regards to age and BMI. Pulse pressure, mean arterial pressure and length of disease varied significantly between patients with RH and CH.

Patients with RH had higher E_A/E_{MAX} ($P=0.005$) and lower EF ($P=0.001$). They also had higher K_{LV} ($P=0.04$) and E/É ($P=0.007$) as well as higher characteristic impedance ($P=0.045$). Pulse wave velocity was not statistically significantly higher when adjusted for covariates ($P=0.385$).

Conclusion: Patients with resistant hypertension have dysfunctional VVC most likely due to stiffening of the left ventricle, which could be due to increased afterload and stiffness of the arterial system.