

causes EC hypertrophy, remodeling, leading to microvessel lumen deformation, narrowing and obstruction of them, causing CMC hypoperfusion. Long-term hibernation, caused by HT, leads to EC and CMC apoptosis causing HF. Onset of AMI in patients with hypertensive cardiomyopathy except necrosis leads to the progression of long-term hibernation and development of short-term hibernation of CMC and vascular endothelium. The latter occurs mainly due to PO and CS triggering cellular apoptosis or secondary necrosis. Stunned cardiomyocytes often transform in to hibernating cells. Accumulation of glycogen granules in CMC and EC was the most sensitive marker of cellular hibernation and dysfunction.

Conclusion: Hypertension causes CMC and vascular endothelium hypertrophy, their structural and functional heterogeneity, hibernation and apoptosis leading to the development of HF. Development of AMI except necrosis of CMC cause progressive myocardial hibernation, activates CMC and EC apoptosis and exacerbate HF. Appropriate pharmacotherapy of HT may partly prevent CMC and EC remodeling, hibernation and HF, while invasive treatment is vital in saving of hibernated myocardium in AMI.

PP.41.388 RETINOPATHY IN DIFFERENT HYPERTENSION PHENOTYPES, ANOTHER EXAMPLE OF TARGET ORGAN DAMAGE IN MASKED HYPERTENSIVES

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Objective: Longitudinal epidemiological studies have shown that hypertensive patients with retinal arteriolar alterations are at increased risk for cardiovascular events. However, the extent of retinal microvascular changes in naïve, never-treated patients with recent appearance of hypertension has been substantially understudied. In addition, the lack of relevant data regarding other hypertension phenotypes, masked (MHT) and white-coat hypertensives (WCH) is notable, and constitutes the aim of our study.

Design and Method: Ambulatory blood pressure was measured using the oscillometric Spacelabs 90,207 device. Retinal vascular calibers were estimated using a computer-based program for retinal photographs taken with a non-mydiatric digital fundus camera (NIDEK AFC-230/210). Central retinal artery (CRAE) and vein (CRVE) equivalents, as well as arterio-venous ratio (AVR) were calculated to estimate retinal abnormalities.

Results: We studied 99 newly diagnosed hypertensive patients, (71 with hypertension (HT), mean age 43.9 ± 11.1, 16 with MHT, mean age 40.1 ± 11.5 and 12 with WCH, mean age 49.2 ± 13.4) and 30 normotensive individuals (mean age 43.5 ± 11.5), without other comorbidities. Comparison of the retinal vascular parameters is depicted in table 1. Patients with HT as well as MHT had both lower CRAE and AVR, indicating narrower retinal vessels. CRAE and AVR were strongly and negatively associated with mean, systolic and diastolic daytime, nighttime, and 24-h blood pressure, even after adjustment for other factor in multivariate analysis.

Conclusion: Subtle retinal microvascular alterations are observed in patients with HT and also MHT, in contrast to patients with WCH, who have retinal findings resembling normotensive subjects. These changes, easily assessed by retinal photography, may be indicative of or mediating the differences in cardiovascular mortality in those groups.

Table 1. Central retinal arteriolar (CRAE) and venular (CRVE) caliber equivalents and arteriovenous ratio (AVR) according to blood pressure status groups

	CRAE	CRVE	AVR
HT (n = 71)	87.1 ± 10.4**	119.7 ± 15.0	0.734 ± 0.099**
MHT (n = 16)	87.3 ± 9.5*	126.3 ± 11.4	0.694 ± 0.072***
WCH (n = 12)	94.4 ± 8.8	121.0 ± 15.9	0.790 ± 0.117
NORMOTENSIVES (n = 30)	95.5 ± 10.3***	117.7 ± 14.3	0.816 ± 0.087****
p value	0.001	0.287	<0.001

*p < 0.05, **p < 0.001, ***p < 0.001.

PP.41.389 LEVELS OF COLLAGEN TYPE IV DERIVED PEPTIDES IN SERA OF CHILDREN WITH OBESITY AND ESSENTIAL HYPERTENSION

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Objective: Elastin and collagen are main proteins of vascular wall connective tissue. An important factor in development of vascular wall alterations is degradation of basement membrane's major protein – collagen type IV. The aims of our study were to: (1) Measure levels of collagen type IV derived peptides (CIVDP) in sera of obese children with essential hypertension; (2) To compare serum CIVDP levels of obese children with essential hypertension with CIVDP levels in obese children without essential hypertension and healthy controls.

Design and Method: The study population consisted of 113 children divided into three groups as follows: obese children with elevated blood pressure (n = 43) (Group 1); obese children with normal blood pressure (n = 32) (Group 2); and control group of healthy children (n = 38) (Group 3). Sandwich version of an enzyme-linked immunosorbent assay (ELISA) for detection of CIVDP was used.

Results: Children with obesity and AH (Group 1) showed statistically significantly higher levels of CIVDP (380 ± 16 ng/ml) in comparison with Group 2 (290 ± 58 ng/ml) and controls (270 ± 39 ng/ml) (p < 0.05). There were nonsignificant differences in serum CIVDP levels (290 ± 58 vs. 270 ± 39 ng/ml) between obese children without AH and healthy controls (p > 0.05). Twenty percent of children from Group 1 were positive for CIVDP. The results indicated both the obese boys and girls with AH to have increased mean levels of total cholesterol (7.2 ± 1.3 and 7.9 ± 1.5 mmol/l) in comparison with the obese boys and girls without AH (6.4 ± 1.5 and 6.9 ± 1.5 mmol/l; p < 0.01). Mean levels recorded in obese children without AH were relatively higher in comparison with healthy controls (p < 0.01). Interestingly, obese girls from Group 1 showed lower mean value of HDL-C (0.92 ± 0.24 mmol/l) than either obese boys from the same group (1.12 ± 0.18) or controls (1.31 ± 0.10) (p < 0.05).

Conclusion: Our data suggest existence of an association between changes in levels of serum CIVDP, obesity and essential hypertension in children. Determination of serum CIVDP levels may be a useful method for monitoring of development of arterial hypertension in obese children.

PP.41.390 FRAME COUNT RESERVE AND SLOW CORONARY FLOW FOR THE EVALUATION OF MICROVASCULAR ANGINA

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Objective: Previous studies suggested that the degree of frame count reserve (FCR) and slow coronary flow (SCF) were related with microvascular dysfunction. We investigated the clinical implication of FCR and SCF for the evaluation of microvascular angina.

Methods: We included consecutive 77 patients with the complaint of chest pain showed normal coronary angiography. Basal TIMI frame count (TFC) was obtained from left anterior descending artery. Intracoronary nitroprusside (15 µg) was infused to induce hyperemia, and repeated angiogram was performed after 30 s. FCR were calculated by dividing basal TFC by hyperemic TFC. SCF was defined as being present when TFC was more than 28. All patients underwent a treadmill test without medication after angiography.

Results: After the treadmill test, patients were divided into a microvascular angina group (40 patients) and a control group (37 patients). Hyperemic TFC was significantly higher in the microvascular angina group (10.9 ± 4.7) than in the control group (9.0 ± 3.5, p < 0.05). FCR were similar in both groups (2.0 ± 1.0 and 2.1 ± 0.9, microvascular angina and control group, respectively). Patients with SCF had a significantly higher incidence of microvascular angina (78.5%) than patients with a normal coronary flow (46.0%, p < 0.05). We defined a positive nitroprusside stress test result as the presence of SCF or an FCR < 1.7, and patients with a positive result had more microvascular angina (26/38 patients) than patients with negative result (14/39 patients, p < 0.01).

Conclusion: The presence of SCF or FCR < 1.7 was found to have a diagnostic value for microvascular angina.