

## Metabolic Aspects and Mechanisms

### 9.23 The Metabolic Syndrome Early Exacerbates the Cerebral Vasodilatory Reactivity and the Microcirculatory Damage In Grade 1 Hypertensives

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**Introduction.** Hypertension, often associated with metabolic disorders, may impair the cerebral and peripheral blood perfusion.

**Aim.** To discern the impact of the metabolic syndrome (MetS) upon the cerebral vasodilating reserve and the macro- and micro-vascular damage at the early stage of hypertension.

**Methods.** By office and ambulatory blood pressure measurements, blood chemistry and anthropometry (ATPIII), 16 normotensive (NTN), 46 hypertensive, 22 with out (HTN) and 24 with MetS (HTN-MetS), untreated males underwent supra-aortic and trans-cranial echocolor-doppler. The carotid intima-media thickness (IMT) and the breath holding index (BHI=as percent change of blood flow velocity at the of the medial cerebral artery following the apnoea), served as indices of vascular damage and cerebral vasodilating reactivity. By video capillaroscopy of standardized areas of periungueal, phalangeal and forearm skin of the non-dominant arm, the basal capillarity (CAP), was calculated. By venous congestion, capillarity was maximized (CVC) and the secondary capillary recruitment (GAIN), as functional and structural indices of microcirculatory damage, were obtained.

**Results.** Data are reported in the table. Pearson analyses, adjusted for age, smoke and SBP/DBP, showed a significant association between BHI and CAP (0.3 37\*\*) and CVC (0.408\*\*)

Paz/var	SBP/DBP	IMT	BHI	CAP	CVC
GAIN					
NTN	1.25±2/76±2	0.67±0.04	1.63±0.12	41.6±2.26	46.2±1.9
5.4 ± 1.7					
HTN	147±2/90±1***	0.72±0.04	1.28±0.09**	35.1±0.7**	42.1±1.1*
6.9 ± 0.7					
HTN-MetS	148±3/86±2***	0.71±0.04	1.01±0.09***^^	32.3±0.8***^	
37.6±1.2***^	5.6±0.7				

\*\*\*p<.001 vs HTN, ^p<.05, ^^p<.01, ^^p<.001 vs HTN-MetS.

**Conclusions.** The findings show that MetS might exacerbate the structural microvascular damage and the cerebral vasodilating reserve in grade-1 hypertensive patients, before the onset of the preclinical macro-vascular damage.