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EH (either newly diagnosed with high BP values or longer lasting), emphasizing on the need of early and effective glycemetic control.

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MARKER OF TYPE VI COLLAGEN FORMATION (PRO-C6) IS ASSOCIATED WITH HIGHER ARTERIAL STIFFNESS IN TYPE 1 DIABETES

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Aim: Arterial stiffening reflects fragmentation and loss of elastin fibers and accumulation of stiffer collagen fibers in the media of large arteries. We evaluated associations between carotid-femoral pulse wave velocity (cfPWV) and a marker of collagen type VI formation (Pro-C6) and a marker of collagen type III degradation (C3M) in type 1 diabetes.

Methods: Serum and urinary level of Pro-C6 and C3M was measured with ELISA in 634 patients with type 1 diabetes. cfPWV was assessed by the SphygmoCor device. We applied unadjusted and adjusted linear regression analyses. Adjustment included sex, age, mean arterial pressure, LDL cholesterol, smoking, HbA_{1c}, eGFR and urinary albumin excretion rate. To adjust for urine output levels, the urinary markers were normalized for urinary creatinine.

Results: Of the 634 patients, 349 (55%) were male, mean \pm SD age was 54.6 ± 12.6 years and cfPWV 10.4 ± 3.3 m/s.

Higher serum and urinary level of Pro-C6 was associated with higher cfPWV in unadjusted models ($p \leq 0.039$), after adjustment only higher serum level remained significantly associated with higher cfPWV (β estimate per doubling: 0.47 ± 0.21 ; $p = 0.028$).

Lower urinary level of C3M was associated with higher cfPWV in the unadjusted model ($p < 0.001$), but significance was lost after adjustment ($p = 0.44$). Higher serum level of C3M was associated with higher cfPWV in the unadjusted model ($p = 0.002$), but significance was lost after adjustment ($p = 0.34$).

Conclusion: In type 1 diabetes, higher serum levels of Pro-C6, a marker of collagen type VI formation, was associated with increased arterial stiffness. This observation could introduce a new target for therapeutic intervention.

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VASCULAR STRUCTURE AND FUNCTION IN RELATION TO WEIGHT EXCESS AND BLOOD PRESSURE IN A SAMPLE OF OBESE CHILDREN

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Objective: To investigate the influence of weight excess and blood pressure (BP) on vascular structure and function in a sample of obese children.

Methods: We measured office and ambulatory BP (ABPM), carotid intima-media thickness (cIMT), endothelial function by the Flow Mediated Dilation (FMD) technique, carotid distensibility (cDC) (ultrasonograph: LogiQ P5 Pro; software: Multimedia Video Engine II, DSP Lab, Pisa CNR, Italy) (1) and stiffness index (SI) by photoplethysmography (Pulce Trace PT1000, MicroMedical Ltd, UK) (2) in overweight and obese children (BMI > 90th percentile for sex and age).

Results: Seventy children with weight excess were enrolled (age 11.5 ± 2.4 years; female n: 30). cDC showed inverse correlation with BMI and waist circumference ($r_{\text{spearman}} = -0.403$ and $r_s = -0.346$, respectively), 24h-SBP ($r_s = -0.449$) and nighttime-SBP ($r_s = -0.490$). SI inversely correlated with BMI ($r_s = -0.359$) and waist/height ratio ($r_s = -0.303$). When comparing normotensive and hypertensive children, as defined on the basis of the

ABPM, no significant differences in vascular tests were found, although cDC tended to be lower in hypertensive. In normotensive subjects (n: 53) cIMT directly correlated with nighttime-SBP and DBP. Most of the correlations remained significant after adjustment for age, sex, BMI and BP.

Conclusions: These data suggest that arterial elasticity is negatively affected by weight excess and 24h-BP levels even in childhood. In the normotensive subgroup it is detectable an effect of BP also on arterial structure.

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CORRELATION OF SOLUBLE RECEPTOR FOR ADVANCED GLYCATION END PRODUCTS AND S100-A1 ON ARTERIAL STIFFNESS IN NORMOTENSIVE PATIENTS WITH DIABETES

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Background: Accumulation of advanced glycation end products (AGEs) are involved in several pathophysiological processes in the vessel wall, that may cause premature atherosclerosis and arterial stiffening (1). A soluble form of RAGE (sRAGE), which is a splice variant of full-length RAGE has been considered to be protective against diseases originating from RAGE activation since sRAGE can bind and sequester RAGE ligands and reduce RAGE activation (2). S100A1 is the most abundant calcium-binding protein in myocardial tissue and is a major determinant of cardiac function. This circulating ligand of the RAGE is known as a pro-inflammatory factor in diabetes. Aberrant expression levels of S100A1 surfaced as molecular key defects, driving the pathogenesis of cardiovascular diseases (3).

Objective: This study was design to explore the relationship between serum levels of sRAGE and S100A1 on arterial stiffness in non-hypertensive patients with diabetes.

Methods: Using a cross-sectional design, a total of 20 non-hypertensive patients with diabetes were recruited. A fasting blood sample, medical history and arterial stiffness parameters were collected.

Results: In bivariate analysis, sRAGE positively correlated with time evolution of diabetes ($r = 0.503$, $p < 0.024$) and negatively correlated with systolic ($r = -0.457$, $p = 0.043$) and diastolic blood pressure ($r = -0.527$, $p = 0.017$). S100A1 positively correlated with creatinine ($r = 0.724$, $p < 0.000$) and negatively correlated with peripheral and central hemodynamics, including augmentation index ($r = -0.469$, $p = 0.037$), as shown in Table 2.