

normal and 15 patients. Although there were no significance difference in AoD between the 2 groups ($9.8 \pm 1.8\%$ vs $6.7 \pm 1.8\%$, $p = 0.08$ in controls and hypertensives respectively) a significant difference was in aortic segmental ($p < 0.01$ for each segment) and averaged LS (averaged LS was $-26.3 \pm 6.9\%$ vs $-20.4 \pm 2.5\%$, $p = 0.007$ in controls and pts respectively). A significant negative correlation between AoD and averaged aortic LS ($r = -0.495$, $p = 0.01$) was found.

Conclusions: Our preliminary results suggest that essential hypertension is associated with a profound change in aortic wall mechanical properties. Speckle tracking promises new insights into aortic mechanics and can be evaluated in the daily clinical practice.

PULSE WAVE VELOCITY PROGRESSION OVER A 3.7 YEARS FOLLOW-UP: FOCUS ON URIC ACID

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Objective: The role of Uric Acid (UA) on the arterial stiffness progression in prospective studies has been evaluated only in three studies. Our aim was to evaluate the role of UA as a possible determinants of the Pulse Wave Velocity (PWV) progression over a 3.7 ± 0.5 years follow-up period in hypertensive subjects.

Design and method: We enrolled 431 consecutive hypertensive outpatients 18–80 aged, followed by the Hypertension Unit of St. Gerardo Hospital (Monza, Italy). At baseline anamnestic, Blood Pressure (BP) and laboratory data as well as PWV were assessed. PWV was performed again at follow-up. We analysed data separately for gender with hyperuricemia defined as a UA > 6 mg/dL for women and > 7 mg/dL for men

Results: Baseline age was 53.2 ± 13.1 years, 43% were female, Systolic and Diastolic BP $141.8/86.8 \pm 17.5/10.8$ mmHg, UA 5.2 ± 1.4 mg/dL and PWV 8.5 ± 2.0 m/s. At follow-up, despite better BP values ($132.5/78.8 \pm 17.4/10.7$ mmHg, $p < 0.001$), PWV increases to 9.15 ± 2.3 m/s ($p < 0.001$) with mean deltaPWV of $+0.56 \pm 2.2$ m/s. 66 patients were hyperuricemic (15%) and when compared to normouricemic were older (58.3 ± 11.6 vs 52.3 ± 13.1 , $p < 0.001$) with superimposable baseline and follow-up BP with a higher PWV baseline and follow-up (9.1 ± 2.7 vs 8.4 ± 1.8 and 9.9 ± 3.2 vs 9.0 ± 2.1 , $p < 0.05$). Despite this, similar deltaPWV were found (0.8 ± 3.4 vs 0.5 ± 1.9 , $p = ns$). Hyperuricemic female (6.8%, 12 subjects) were older (63.1 ± 10.6 vs 53.1 ± 13.4 , $p < 0.001$), with similar baseline and follow-up BP and, despite similar baseline PWV, a higher follow-up PWV (11.1 ± 2.5 vs 8.7 ± 2.2 , $p < 0.001$) and deltaPWV (2.1 ± 3.5 vs 0.5 ± 1.9 , $p = 0.01$). In males no differences were showed in arterial stiffness in hyperuricemic (21%, 54 subjects). UA correlate with age, sex and baseline and follow-up PWV ($r = 0.13$ and 0.19) in the whole population but not with deltaPWV. In females UA significantly correlate with age, follow-up PWV ($r = 0.31$) and deltaPWV ($r = 0.26$). Contrariwise in male no significant correlation were seen. At multivariate analysis UA were not a significant predictor of arterial stiffness, also for gender analysis.

Conclusions: in HT, arterial stiffness showed some sign of correlation with UA particularly in women. Despite this it were not a significant predictor of arterial stiffness and its progression. A strong limitation of the present work is the low number of hyperuricemic female subjects.

REFERENCE VALUES OF DIFFERENT PARAMETERS OF VASCULAR STRUCTURE AND FUNCTION IN CAUCASIAN POPULATION WITHOUT CARDIOVASCULAR DISEASES. EVA STUDY

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Objective: To describe the mean values of different parameters of vascular structure and function, evolution with age and differences by gender in the general population without cardiovascular diseases.

Design and method: Cross-sectional study in a general population cohort without cardiovascular diseases. Scope and Subjects: 360 subjects aged between 35 and 75 years old (51% female) were included, selected by random sampling stratified by age groups (35, 45, 55, 65 and 75 years) and gender using the base of Health Card of 4 urban Health Centers.

Using ultrasonography, we measured the thickness of the intima media of the carotid artery (IMT). With the VaSera device, the Cardio Ankle Vascular Index

(CAVI) and the pulse wave velocity ankle arm (aaPWV) were measured. With the Sphygmocor we measured the pulse wave velocity femoral carotid (cfPWV).

Results: The mean values were: age 56.8 ± 14.9 years (males = 57.1 ± 14.4 and women = 56.6 ± 15.4 , $p = 0.730$). GIM = 0.688 ± 0.114 mm (males = 0.708 ± 0.119 mm and females = 0.661 ± 0.108 mm, $p = 0.020$), CAVI = 8.19 ± 1.45 (males = 8.39 ± 1.44 and females = 8.02 ± 1.45 , $p = 0.015$), aaPWV = 13.18 ± 2.81 m/sec (males = 13.46 ± 2.52 m/sec and women = 12.93 ± 3.02 m/sec, $p = 0.070$) and cfPWV = 6.63 ± 2.04 m/sec (males = 6.97 ± 2.15 m/sec and females = 6.34 ± 1.91 m/sec, $p = 0.004$).

For each year that the age increases, an increase of the IMT of 0.006 mm ($y = 0.364$ mm + $(0.006$ mm * age)), in males 0.006 ($y = 0.365$ mm + $(0.006$ mm * age)) and in women ($y = 0.363$ mm + $(0.005$ mm * age)) An increase in CAVI of 0.070 ($y = 4.212 + (0.070$ * age)), in males 0.071 ($y = 4.340 + (0.071$ * age)) and in women 0.069 ($y = 4.113 + (0.069$ * age)) An increase in aaPWV of 0.139 m/sec ($y = 5.276$ m/sec + $(0.139$ m/sec * age)), in males 0.117 ($y = 6.785$ m/sec + $(0.117$ mm * age)) and in women 0.155 ($y = 4.140$ m/sec + $(0.155$ m/sec * age)) and an increase in cfPWV of 0.088 m/sec ($y = 1.655$ m/sec + $(0.088$ m/sec * age)), in males 1.458 ($y = 1.458$ m/sec + $(0.097$ m/sec * age)) and in women 0.081 ($y = 1.784$ m/sec + $(0.081$ m/sec * age))

Conclusions: All the parameters of structure and function analyzed, except for aaPWV, show average values and a greater annual increase in males comparing with females.

SODIUM-SENSITIVE BLOOD PRESSURE RESPONSE IN TYPE 1 DIABETES IS ACCOMPANIED BY IMPEDED SKIN LYMPHANGIOGENESIS

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Objective: Studies showed that sodium can be non-osmotically stored within the skin. In response to high sodium diet (HSD), skin sodium content increases and macrophages are attracted, inducing lymphangiogenesis. Disruption of this system has been shown to lead to sodium-sensitive hypertension. This study investigates the effects of HSD on skin lymphatic and blood capillaries as well as blood pressure (BP) in type 1 diabetic patients (DM1).

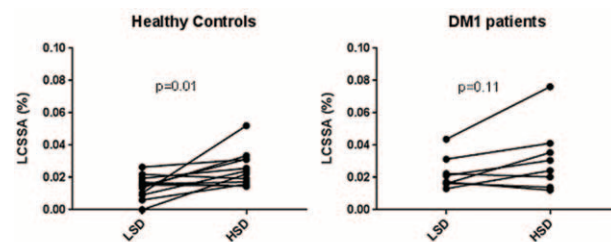


Fig 1A. Lymphatic cross sectional surface area (LCSSA) expressed as a percentage of the histological slice. LSD = low sodium diet, HSD = high sodium diet.

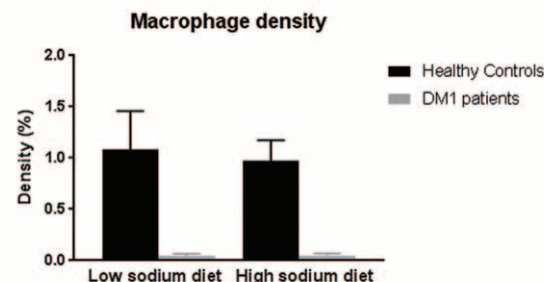


Fig 1B. Macrophage density in healthy controls and DM1 patients after low and high sodium diet, expressed as the percentage of the histological slice that is positively stained with CD68. Data are presented as median with interquartile range.

Design and method: We performed a randomized crossover study in males with DM1 and healthy controls. All subjects pursued an 8-day low sodium diet (LSD: < 50 mmol Na+/day) and HSD (> 200 mmol Na+/day). Diet order was randomized and time in-between diets was 1–2 weeks. After each diet, BP measurements and skin biopsies were obtained. Macrophages (CD68), vascular en-