LIPID PEROXYDATION IS NOT INCREASED IN NEVER TREATED HYPERTENSION

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To compare the levels of urinary isoprostanes (UIP), a marker of lipid peroxidation expressing the degree of oxidative stress, in hypertensive and normotensive patients.

Thirty mild to moderate never treated hypertensive patients (HT) (clinical SBP > 140 mmHg and/or DBP > 90 mmHg) (age = 53±12 years, sex ratio = 1) (Mean clinic BP = 152/95 mmHg, duration of HT = 41±59 months) were included. Three of the 30 patients (10%) had white coat hypertension. Thirty control subjects were matched for age and sex. All the subjects had an ambulatory BP recording over 24 hours, a carotid intima-media thickness measurement by ultrasound, an echo-cardiography, a measurement of the carotid to femoral pulse wave velocity (PWV), and a determination of glucose concentration, serum lipids and urinary isoprostanes.

The two groups were similar apart for BP, by definition, and the PWV which was greater in the HT group (10.4±2.2 vs 9.1±2.2 m/s, p<0.05). The levels of UIP were no different between the groups (69±36 in HT vs 75±34 pmol/mmol of creatinine in control subjects). No correlation was found between the levels of UIP and the BP and cardiovascular parameters.

Many experimental studies have shown the importance of the effect of oxidative stress on vascular remodelling and the rise in BP. In our study, lipid peroxidation as evaluated by the level of UIP is not increased in humans with never treated mild to moderate essential HT. These results can be partly explained by the relatively short duration and the moderate levels of HT in our population.

Key Words: Urinary isoprostanes, Lipid peroxidation, Arterial wall

BIOCHEMICAL AND HEMODYNAMIC DETERMINANTS OF C-REACTIVE PROTEIN AS AN INFLAMMATORY MARKER OF ENDOTHELIAL DYSFUNCTION. THE IMPACT OF SYSTOLIC HYPERTENSION

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Background: C-reactive protein (CRP) is a known factor of systemic inflammation and is currently conceived as a cardiovascular risk factor in the short and long term.

Aims: To know the biochemical and hemodynamic determinants of CRP. To assess its correlation with Endothelial Dysfunction (ED).

Material and Methods: Study Population: N=131 patients from a cardiovascular risk population, 70 female, 61 male, aged 21-82 years (57 +/-14), 52 dyslipemic (LDLc > 160mg/dl), 84 hypertensive (WHO Criteria), 50 type 2 diabetics (WHO Criteria) and 31 active smokers.

Protocol: Biochemical parameters: basal glycemia, HbA1c, total cholesterol, cLDL, cHDL, triglycerides, and lipoprotein a (Lpa).

Hemodynamic parameters: Blood Pressure Holter (Spacelabs 90210): Four periods were assessed: 1) Global 24h Period ; 2) Daytime Period (9-22h); 3) Nighttime period (22-6h) and 4) Critical Period (6-9h). ENDOTHELIAL DYSFUNCTION: endothelium-dependent vasodilatation (EDV), by Flow Mediated Vasodilatation (FMV) and endothelium-independent vasodilatation (EIV), by sublingual nitroglycerin, were assessed by brachial artery doppler ultrasonography (DUS) in N=58.

CRP: Behring Nephelometer II.

Statistical Analysis: T-Student, Chi-square.

Results: 1- 18% of patients had CRP serum levels higher or equal to 5 mg/l. 2- Such levels were positively correlated with : basal glycemia (143.22 +/-17.71 vs 122.07 +/- 5.93; p<0.02), HbA1c (6.66 +/- 0.44 vs 5.76 +/- 0.16; p<0.039), Lpa (35.57 +/- 8.92 vs 24.75 +/- 2.55; (Mean +/- SEM); p<0.015).

75% of patients with high levels of CRP had average systolic arterial blood pressure levels higher or equal to 125 mmHg versus 47% with systolic blood pressure levels <125 (p<0.040). 3- A statistically significant association was found between high levels of CRP and endothelial dysfunction (22.8% ED and CRP>5 vs 4.3% no ED and CRP>5; p<0.05).

Conclusions: 1- Basal glycemia, HbA1c and Lpa, but not total cholesterol, cLDL nor triglycerides are biochemical factors of CRP levels elevation. 2- The degree of systolic arterial blood pressure levels could be the determinant hemodynamic factor. 3- ED associated with high CRP levels justified by itself the high risk for acute events.

Key Words: C-Reactive Protein, Endothelial Dysfunction

OBESITY AND VASCULAR WALL INFLAMMATION. EFFECTS OF WEIGHT LOSS AND WEIGHT CYCLING

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Obesity represents an important cardiovascular risk able to promote the onset and development of vascular inflammation. We evaluated the effects of body weight changes on circulating levels of VCAM-1, ICAM-1, E-selectin, P-selectin, CD40L, CRP and 8-iso-PGF2α, as indexes of vascular wall inflammation in obese patients. Fortyeight low risk obese patients were assigned to a 16 wk period of intense caloric restriction (800-1200 Kcal/die) and then to a 22 month period of weight maintaining diet. At baseline all the assessed parameters were significantly higher (p<0.0001) in obese patients than in a group of 38 nonobese subjects. Caloric restriction significantly reduced initial BMI, WHR, skinfold thickness, fasting insulin levels and all the indexes of atherogenic and thrombotic status in obese patients. Changes in BMI showed a trend to be directly correlated with changes in adhesion molecules, CD40L, CRP and 8-iso-PGF2α levels(Table).

During the follow-up period 20 patients maintained weight loss (weight loss maintainers) while 28 patients recovered their initial body weight (weight cyclers). In weight loss maintainers all the assessed parameters did not further change during the follow up period. By contrast, in weight cyclers adhesion molecules, CD40L, CRP and 8-iso-PGF2α showed a trend to achieve the baseline value. This trend was mostly evident in the cases of E-selectin (109.3+/-22.1, ns vs baseline), CD40L (3.2+/-0.4, ns vs baseline) and CRP (3.1+/-0.6, ns vs baseline). Our data clearly demonstrated that weight loss downregulated endothelial activation and vascular inflammation while body weight recovery abolished these vasoprotective effects.

Key Words: obesity, endothelium, atherosclerosis