13.01: C-REACTIVE PROTEIN LEVELS ARE GRADUALLY ASSOCIATED WITH ADIPONECTIN AND ARTERIAL STIFFNESS IN NEWLY DIAGNOSED UNTREATED ESSENTIAL HYPERTENSIVE SUBJECTS: A UNIFYING APPROACH TO ATHEROSCLEROSIS


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13.01 C-REACTIVE PROTEIN LEVELS ARE GRADUALLY ASSOCIATED WITH ADIPONECTIN AND ARTERIAL STIFFNESS IN NEWLY DIAGNOSED UNTREATED ESSENTIAL HYPERTENSION SUBJECTS: A UNIFYING APPROACH TO ATHEROSCLEROSIS

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Purpose: To examined the plausible correlations between hs-CRP levels, adiponectin and arterial stiffness in essential hypertensive patients.

Methods: In 148 newly diagnosed untreated non-diabetic essential hypertensive patients [96 men, mean age = 49 years, office BP = 150/97 mmHg], aortic stiffness was evaluated on the basis of carotid to femoral pulse wave velocity (PWV), by means of a computerized method (Complior SP). Venous blood samples were drawn for estimation of lipid profile and hs-CRP and adiponectin levels. All subjects according to hs-CRP values were divided into group A (hs-CRP < 1.29 mg/l), group B (hs-CRP = 1.3–2.39 mg/l) and group C (hs-CRP > 2.39 mg/l).

Results: Patients in group A (n = 51) compared to subjects in group B (n = 45) and C (n = 52) had lower office systolic BP and left ventricular mass index (r = 0.32, p < 0.005 for all cases), while groups did not differ regarding lipid levels (p = NS). In the entire population, hs-CRP was positively associated with body mass index (r = 0.34, p < 0.001). In the whole study group, the product moment correlation coefficient between hs-CRP and PWV was 0.28 (p = 0.02). Furthermore, patients in group C exhibited lower levels of adiponectin compared to group B and A (7.0 ± 4.0 vs 8.9 ± 5.1 vs 9.4 ± 4.9 μg/ml, respectively; p < 0.05 for all cases) and more augmented PWV values (8.6 ± 1.6 vs 8.2 ± 0.9 vs 7.8 ± 1.2 m/s, p < 0.05 for all cases). Analysis of covariance revealed that adiponectin and PWV values remained different between groups after adjustment for confounding factors (p = 0.05).

Conclusions: Low-grade inflammation is associated in a graded fashion with proatherogenic processes linked with hyperadaptonectinemia and arterial stiffening, even in the early stages of essential hypertension.

13.02 EXPOSURE TO URBAN AIR POLLUTANTS ALTERS ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS

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Exposure to urban air pollution, ultrafine particles or gas, is associated with acute cardiovascular mortality and morbidity. We investigated the effects of ambient air pollution on endothelial function in 40 healthy Caucasian men, previously described in the KLK study (JCI 2005), who spontaneously breathed ambient air pollution in Paris.

Endothelial function was measured by the % of brachial artery dilatation (dDr) after a hand warming test (from 28ºC to 44ºC) and endothelium-independent (FDV) by the % of brachial artery dilatation (dDr) during a sublingual nitroglycerin challenge (0.9 mg). A random, population-based sample of 250 subjects aged 25 to 64 years. Two frequent polymorphisms, A1166C of AGTR1 and T 786C of eNOS, were assessed in a random, population-based sample of 250 subjects aged 25 to 64 years. Pulse wave velocity was measured in the aorta (APWV, between carotid and femoral arteries) and on the lower extremity (peripheral pulse wave velocity, PPWV, between femoral and tibials posterior/dorsalis pedis arteries). Both polymorphisms were significantly associated with PPWV: 12.4 ± 0.7, 13.8 ± 0.2, 15.2 ± 2.7 m/s for AA, AC and CC genotypes of AGTR1, respectively, p = 0.01 for trend; 12.3 ± 0.8, 13.4 ± 1.0, 15.1 ± 1.6 m/s for TT, TC and CC genotypes of eNOS, respectively, p < 0.05). The combined effect of the polymorphisms was further studied. Subjects with 3-4 mutant alleles (heterozygous + homozygous or homozygous + homozygous, n = 35) had significantly increased PPWV (17.9 ± 2.4 m/s) than those with no mutant allele (12.4 ± 1.2 m/s) or 1-2 alleles (12.3 ± 0.5 m/s, p < 0.007 for difference). These associations remained highly significant in multiple regression models with adjustment on potential confounders. The polymorphisms did not influence APWV or blood pressure. In conclusion, both AGTR1 and eNOS gene polymorphisms are associated with increased stiffness of peripheral muscular-type large arteries and their effect is synergistic. This finding reflects an interaction between the renin-angiotensin and nitric oxide systems in their effect on arterial properties.

13.03 SYNERGISTIC EFFECT OF ANGIOTENSIN II TYPE 1 RECEPTOR AND ENDOTHELIAL NITRIC OXIDE SYNTHASE GENE POLYMORPHISMS ON ARTERIAL STIFFNESS

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Angiotensin II and nitric oxide play an important role in the function of arterial system. We wondered whether the mutations of angiotensin II type 1 receptor (AGTR1) and endothelial nitric oxide synthase (eNOS) genes are associated with increased arterial stiffness. Two frequent polymorphisms, A1166C of AGTR1 and T786C of eNOS, were assessed in a random, population-based sample of 250 subjects aged 25 to 64 years. Pulse wave velocity was measured in the aorta (APWV, between carotid and femoral arteries) and on the lower extremity (peripheral pulse wave velocity, PPWV, between femoral and tibialis posterior/dorsalis pedis arteries). Both polymorphisms were significantly associated with PPWV: 12.4 ± 0.7, 13.8 ± 0.2, 15.2 ± 2.7 m/s for AA, AC and CC genotypes of AGTR1, respectively, p = 0.01 for trend; 12.3 ± 0.8, 13.4 ± 1.0, 15.1 ± 1.6 m/s for TT, TC and CC genotypes of eNOS, respectively, p < 0.05). The combined effect of the polymorphisms was further studied. Subjects with 3-4 mutant alleles (heterozygous + homozygous or homozygous + homozygous, n = 35) had significantly increased PPWV (17.9 ± 2.4 m/s) than those with no mutant allele (12.4 ± 1.2 m/s) or 1-2 alleles (12.3 ± 0.5 m/s, p < 0.007 for difference). These associations remained highly significant in multiple regression models with adjustment on potential confounders. The polymorphisms did not influence APWV or blood pressure. In conclusion, both AGTR1 and eNOS gene polymorphisms are associated with increased stiffness of peripheral muscular-type large arteries and their effect is synergistic. This finding reflects an interaction between the renin-angiotensin and nitric oxide systems in their effect on arterial properties.