THE IMPACT OF AN ALDOSTERONE SYNTHASE (CYP11B2) POLYMORPHISM ON VASCULAR FUNCTION AND INFLAMMATORY BIOMARKERS IN ESSENTIAL HYPERTENSION

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Background: Evidence suggests that a polymorphism (C-344T) in the promoter of the aldosterone synthase gene is associated with increased constitutive aldosterone production, which is involved in vascular injury and elevation of blood pressure. However, the impact of this polymorphism in aforementioned parameters is unknown. Therefore, the aim of the study was to investigate the potential role of this polymorphism on vascular function and inflammation in essential hypertension (EH).

Methods: Our population consisted of 303 untreated essential hypertensive patients stage I-II, and 193 controls. The gene mutation frequency was determined using polymerase chain reaction-restriction fragment length polymorphism technique. Aortic stiffness was evaluated, on the basis of carotid to femoral pulse wave velocity (c-f PWV) by means of a computerized method. Brachial artery flow-mediated dilatation (FMD) has been used to assess endothelial dysfunction. The concentrations of intracellular cell adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) in the serum were measured by the ELISA method.

Results: The CYP11B2/C-344T genotype distribution in patients with EH (CC = 26.6%, CT = 52.2%, TT = 21.5%) differed from genotype distribution in controls (CC = 33.1%, CT = 50.8%, TT = 16.0%), and the TT genotype was associated with EH (RR: 0.62; 95% CI 0.400-0.951; P = 0.03). There was no significant difference of PWV between C-allele carriers and TT homozygous both in controls (7.5±1.32 vs 7.3±0.8 m/s, p=NS) and in EH population (8.6±1.5 vs 8.8±1.7 m/s, p=NS). Similarly, -344C allele carriage compared with TT genotype in hypertensive patients, was not associated with increased levels of FMD (4.445±3.3 vs 4.813 ±3.0 p=NS) nor with ICAM-1 and VCAM-1 concentrations (298.9±53.2 vs 311.2±80.7 ng/mL, 854.3±202.5 vs 838.8±167.1 ng/mL, p=NS in both cases), respectively.

Conclusions: These data illustrate that T allele homozygosity of the C-344T polymorphism might be a risk marker for essential hypertension. However, this polymorphism is unrelated to vascular properties and inflammatory mechanisms in hypertension.