CANDIDATE GENE POLYMORPHISMS AND THEIR ASSOCIATION WITH PRECLINICAL ORGAN DAMAGE IN UNTREATED HYPERTENSION

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Authors: Emmanuel Androulakis, Dimitris Tousoulis, Nikolaos Papageorgiou, Evaggelos Chatzistamatiou, Antigoni Miliou, George Moustakas, George Hatzis, Costas Tsioufis, Kostas Toutouzas, Marietta Charakida, Ioannis Kallikazaros, Christodoulos Stefanadis, Hippocration Hospital, Athens, Greece

Background: The role of the renin-angiotensin-aldosterone system in the pathophysiology of target organ damage in hypertension is still under investigation. The angiotensinogen M235T and aldosterone synthase C-344T gene polymorphisms may be associated with vascular indices, renal function and left ventricular hypertrophy thus, we sought to investigate their potential associations with preclinical organ damage in untreated hypertension.

Methods: The study population consisted of 319 untreated hypertensive patients and 193 matched controls. In all participants, flow mediated dilation (FMD), pulse wave velocity (PWV), intima-media thickness (IMT), as well as the incidence and progression of retinopathy were evaluated. Left cardiac indices were assessed by echocardiography. Glomerular filtration rate (GFR) was estimated by Cockcroft-Gault formula.

Results: 235TT homozygotes compared with M-carriers had significantly lower FMD in controls (p=0.038) and higher PWV values in hypertensive patients (p=0.025). Regarding other vascular properties no other significant associations were observed. TT homozygosity showed a trend towards lower GFR compared with MM+MT genotype in hypertensive patients (99.9±30.2 vs 106.8±35.3 mL/min/1.73 m², p=0.067) but neither genotype was associated with increased creatinine. Notably, we observed higher values of IMT in -344TT homozygosity, in the group of hypertensives (781.6±33.5 vs 712.5±16.2 μm, p=0.039). This genotype also predicted higher prevalence of atherosclerotic plaques in total population (OR: 0.32; CI 0.12 to 0.84, p=0.020), and in hypertensives, though without reaching statistical significance (p=0.057). Moreover, -344TT hypertensives exhibited higher values of left ventricular mass index compared to C-allele carriers (p=0.020) and higher prevalence of concentric hypertrophy (p=0.001).

Conclusions: The present study suggests that aldosterone synthase variant may be a marker of subclinical atherosclerosis and may predict not only left ventricular hypertrophy but also concentric hypertrophy pattern, while angiotensinogen genotype may be a marker of arterial stiffness in untreated hypertension.