

AN ALLELIC VARIANT OF NA⁺/CA⁺⁺ EXCHANGER PROMOTER REGION, A2689C, PLAYS A ROLE IN DIASTOLIC BLOOD PRESSURE RESPONSE TO β -BLOCKERS AND DIHYDROPYRIDINE CA⁺⁺ ANTAGONISTS TREATMENT IN HYPERTENSIVE PATIENTS

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BACKGROUND: Systolic and diastolic blood pressure (BP) levels are regulated by an interplay of genetic and environmental factors. Recently, it has been suggested that Na⁺/Ca⁺⁺ exchanger protein (NCX-1) may exert a role in the regulation of BP.

AIM OF THE STUDY: To investigate the potential effect of an allelic variant A2689C within NCX-1 promoter region on systolic (SBP) and diastolic (DBP) values in response to pharmacological treatment in a cohort of hypertensive patients.

METHODS: 316 patients (183 male, 133 female, 58±0.6 years old) were enrolled in the study and anamnestic, clinical and biochemical data collected. Genomic DNA was extracted from peripheral blood and conventional RFLP technique was used to detect the two allelic variants at A2689C locus. Patients were stratified according to the respective genotype. Data are expressed as mean±SEM and differences among groups were tested by ANOVA.

RESULTS: No difference was observed among AA (n=222), AC (n=85) and CC (n=9) patients in regard to age, length of hypertensive disease, incidence of common cardiovascular risk factors and target organ damage. At diagnosis SBP/DBP values (mmHg) tended to be higher in CC patients 166±3/106±4 as compared to AA, 159±1/99±1 and AC 164±3/100±2 (all p=ns). During pharmacological treatment CC patients showed higher DBP levels: CC 82±11 vs AC 76±6 and AA 78±8 (p=0.03). This difference in DBP was abolished in CC patient treated with β -blockers (78±3, p=0.03) or dihydropyridine Ca⁺⁺ antagonists (76±1, p=0.04). Pharmacological treatment with diuretics, ACE-I and non-dihydropyridine Ca⁺⁺ antagonists, was not associated to any significant change in DBP in CC patients.

CONCLUSIONS: Our results strongly suggest that allelic variants in NCX-1 gene might be associated to blood pressure response to selective pharmacological treatment more than to the regulation of SBP and DBP levels in hypertensive patients.