ASSESSMENT OF INFLUENCING FACTORS IN THE DEVELOPMENT OF TARGET-ORGAN DAMAGES IN ADOLESCENCE

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The causative role of the endothel dysfunction and the imbalance between nitric oxide (NOx) and endothelin (ET-1) system in the development of hypertension has been known already for decades. We compared a hypertensive (n=67) and a healthy controll (n=58) adolescent group (age: 16.5±1.1 vs 16.8±0.7 years; gender: female/male: 28/39 vs. 29/29). Left ventricular mass index (LVMI) and intima mdia thickness (IMT) in the hypertensive group was higher than that in the control group (107±32.4 vs. 91.1±25.2 g/m^2; p<0.001 and 0.54±0.11 vs, 0.48±0.1 cm; p<0.001). The plasma NOx concentration decreases (27.7±13.7 µmol/ml vs. 35.8±7.0 µmol/ml; p<0.05) and ET-1 concentration increases (3.1±3.9 vs. 1.1±1.1; p<0.01) in adolescent hypertension. We found a significant negative correlation between plasma levels of NOx and ET-1 (r= -0.29; p=0.003). Blood pressure correlated with NOx negatively, while a positive correlation could be detected between plasma ET-1 concentrations and BP values. We demonstrated a significant positive relationship between IMT and plasma concentration of ET-1 (r=0.26; p=0.006). We have shown that middle cerebral artery velocities in adolescent hypertensives are higher than that of healthy teenagers (74.5±22.4 vs. 62.9±15.7; p<0.001). Cerebrovascular reactivity values (as assessed by breath holding and hyperventilations tests) did not differ between hypertensives and normotensives. Although both NOx and ET-1 influenced resting cerebral blood flow velocity (r=-0.24; p<0.001; and r=0.27, p=0.004), it did not have any effect on cerebrovascular reactivity. No relationship was found between severity of target-organ damages and ACE gene polymorphism in adolescent hypertension.