
**Objective:** To test the hypothesis that polymorphism of angiotensin converting enzyme (ACE) gene I/D polymorphism is linked to the atheromatous renal artery stenosis.

**Design and methods:** ACE gene polymorphism was determined using PCR method in 40 Caucasian patients with angiographically confirmed renal artery stenosis compared to 73 control subjects matched for age and gender from the same ethnic group. Presence of other risk factors (cholesterol, triglycerides, serum glucose, smoking) was also analyzed as well as the severity of hypertension. History of cardiovascular or cerebrovascular disorders was also analyzed.

**Results:** The distribution of ACE gene polymorphism was II (32.5%), ID (40%) 16 DD 11 (27.5%); and controls II 18 (24.8%), ID 31 (42.4%), DD 24 (32.8%) (p < 0.05). Therefore, the frequency of D allele was not significantly different from the control group (p > 0.05). We failed to find significant difference in blood pressure values between particular genotypes (p > 0.05). There were also no differences in fasting serum glucose, cholesterol and triglyceride levels between patients with DD genotype and others (p > 0.05). Also no association was found between ACE gene polymorphism and ECG findings and fundus examination (p > 0.05). However, although not statistically significant (p > 0.05), duration of hypertension was the shortest in patients with DD genotype (7.7 ± 3.8 vs. 11.3 ± 2.7 in II and vs. 9.6 ± 1.8 in ID).

**Conclusion:** According to our results ACE gene polymorphism does not seem to have a predisposing role in the development of atheromatous renal artery stenosis. However, the observed shorter duration of hypertension in patients with DD genotype could implicate that it might have some influence in the progression of this disease.

Key Words: ACE gene polymorphism; renal artery stenosis

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**ASSESSMENT OF INTERSTITIAL FLUID ANGIOTENSIN II CONCENTRATION IN THE RAT HEART BY MICRODIALYSIS**

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As a model to study the relative contributions of local tissue synthesis vs. systemic delivery of angiotensin II (Ang II), the microdialysis technique was used to estimate cardiac interstitial fluid (ISF) levels of Ang II in the intact beating rat heart in situ. A dialysis probe (10 mm length, 0.5 mm OD) was inserted into the anterior myocardial wall (apex to base) of normal open-chest, pentobarbital-anesthetized rats. Krebs solution with 0.5% BSA was perfused at 2 μL/min for 30–60 min periods and samples were collected on ice in tubes containing EDTA. Blood samples were collected in EDTA prior to and following each period. Ang II was measured by radioimmunoassay following extraction of dialysates or plasma on C18 SepPak cartridges. Further analysis of immunoreactive (ir) Ang II was performed by reversed-phase high performance liquid chromatography (rpHPLC) using a C18 column eluted isocratically with 22% acetonitrile in water. In initial studies (n = 5) irAng II levels averaged 72 ± 53 (mean ± SD) pg/ml in cardiac ISF and 51 ± 4 pg/ml in plasma. By rpHPLC analysis the irAng II in plasma and ISF had retention times (9.5 min) corresponding to that of au-