

Relation of RAS Gene Polymorphisms with Impaired Glucose Tolerance in Patient with End Stage Renal Failure

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ABSTRACT Renin angiotensin system (RAS) is a system that is the role of renal hemodynamics, blood pressure and fluid electrolyte balance regulation with progression to end-stage renal disease (ESRD). Angiotensin II (AII), the main mediator of this system, is thought to cause a functional impairment in insulin receptor and post receptor signaling pathways. Our aim; was to show the relation of angiotensin-converting enzyme (ACE), angiotensin II receptor type I (AT1 R) and angiotensinogen gene (AGT) polymorphisms, that cause genetic susceptibility to over activation of RAS, to glucose intolerance in patient with end stage renal failure. The study included one hundredth patients. Based on fasting plasma glucose values and oral glucose tolerance test second hours glucose values, patients were grouped as normal patients and patients with glucose intolerance [impaired fasting glycemia (IFG), impaired glucose tolerance (IGT) and diabetes mellitus (DM)]. Insulin resistance was calculated by HOMA-IR method and insulin sensitivity index was calculated by ISI-S method. ACE, AT1 R, AGT polymorphisms were detected by polymerase chain reaction (PCR) method. There was no statistically significant difference between distribution of ACE, AT1 R and AGT genotypes and glucose intolerance groups, and insulin resistance. There was a statistically significant difference between MM and TT genotypes and average insulin resistance values ($p < 0,04$). We have obtained results suggesting a relation between ACE gene D allele and glucose intolerance in patients with end stage renal failure ($p=0.06$). Moreover, a relation has been shown between AGT gene T allele and insulin resistance ($p=0.04$).