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EFFECT OF ANGIOTENSIN II RECEPTOR BLOCKER ON EXTRA CELLULAR SIGNAL REGULATED KINASE (ERK) PATHWAY AND NADPH OXIDASE 4 IN DIABETIC NEPHROPATHY IN RAT MODEL

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Introduction: Diabetic nephropathy is the most common renal complication of diabetes mellitus and a leading cause of kidney failure worldwide. Several factors have been engaged in the pathogenesis, of which the extracellular regulated kinase (ERK) is a critical player being activated via different stimuli as angiotensin II and ROS. NADPH oxidase 4(NOX 4) is a major contributor for ROS production in the kidney through angiotensin system and result in kidney damage via direct effect or through activation of signalling pathway that play role in the pathogenesis.

Objectives: To evaluate the effect of angiotensin II receptor blocker on ERK 1/2 and NOX4 mRNA expression, protein concentration and ROS production in diabetic nephropathy rat model.

Methods: 60 rats were divided into 3 groups: group I (n=20) valsartan treated STZ induced diabetic rats, group II (n=20) vehicle treated STZ induced diabetic rats and group III (n=20 vehicle treated control rats. Diabetes mellitus was induced in rats with single intra-peritoneal streptozotocin injection of 65mg/kg body weight. Diabetic nephropathy was confirmed by altered kidney function then rats were treated with valsartan 30 mg/kg/day for 4weeks, after 4 week of treatment blood were collected for chemistry analysis, then rats were anesthetized sacrificed and kidney tissues were excised for the detection of NOX4 and ERK1/2 gene expression level by RT-PCR, and NOX4 and pERK1/2 protein concentration by ELISA and malonaldhyde (MDA) concentration for different group.

Results: In diabetic rats valsartan treatment significantly attenuate albuminuria (p=0.001), improve overall kidney function parameters and glomerulosclerosis. Expression of NOX4 gene and ERK1 gene were significantly lowered with treatment (p=0.001) and as well as NOX4 and pERK1/2 protein concentration, and MDA concentration.

Conclusion: Valsartan inhibit progression of kidney damage occur in diabetic kidney through down regulation of ERK pathway, reduction in NOX4 expression level and amelioration of oxidative damage.

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