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Renalase and Dopamine study in chronic renal failure patients

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Introduction: The human kidney releases a monoamine oxidase, renalase, which was discovered in 2005, to the blood stream to regulate the blood pressure. Renalase decreases systemic pressure by metabolizing the circulating catecholamines. Hypertension is highly prevalent in patients with diabetic nephropathy which is one of the leading causes (about 80%) of chronic kidney disease and end-stage kidney disease. When considered in isolation, hypertension and diabetes are associated with increased risk of the development of cardiovascular and renal complications. It is recognized that sympathetic nervous activation and stimulation of the rennin-angiotensin-aldosterone are involved. The dopaminergic and rennin-angiotensin systems interact to regulate the blood pressure. The vasodilator, Dopamine, counteracts angiotensin receptors in the paracrine regulation of renal sodium transport. Levels of renalase that metabolize catecholamines are decreased in chronic kidney disease and the plasma concentration of renalase is markedly reduced in patients with ESRD. Chronic kidney disease is often characterized by the presence of sympathetic hyperactivity, which contribute to the development of other forms of organ damage independent of its effect on blood pressure. It is associated with heart failure, arrhythmias and atherogenesis. Decrease renalase level plays an important role in cardiovascular pathology. Chronic kidney disease leads to an 18-fold increase in cardiovascular complications not fully explained by traditional risk factors. Preventing the progression of renal failure and reducing cardiovascular risk of uraemic patients are major challenges for nephrologists. Interference with sympathetic over activity may provide a new therapeutic avenue to follow in clinical medicine.

Aim: To assess the relationship between Dopamine and Renalase in Egyptian type-2 diabetic patients in the presence and absence of diabetic nephropathy.

Subjects & Methods: 80 subjects were divided in three groups as follow: Group-1: 10 control healthy volunteers, Group-2: 60 type-2 diabetic patients and Group-3: Type-2 diabetic patients on maintenance hemodialysis.

Results: Significant increase in blood pressure, both systolic and diastolic in diabetic patients and diabetic patients on maintenance hemodialysis as compared to controls. No significant change in Dopamine level in between the studied groups. No significant change in Renalase in type-2 diabetic patients but significant increase in renalase level in diabetic patients on maintenance hemodialysis as compared to controls (p=0.000) also to diabetic patients (p=0.004). There was significant correlation between Renalase and Dopamine (r=0.261, p=0.022) and Renalase and diastolic blood pressure (r=0.243, p=0.041) in diabetic patients.

Conclusion: Renalase is an attractive replacement therapeutic modality in hypertensive type-2 diabetic patients in order to prolong the interval between early chronic and end-stage renal failure.

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