Dopamine and renalase in type 2 diabetic patients with and without diabetic nephropathy

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Introduction: Type 2 diabetes cause diabetic nephropathy which is a common cause of end stage renal disease. Dopamine is involved in the regulation of blood pressure. Renalase metabolizes circulating catecholamines and regulates blood pressure.

Aim: Assessing the relationship between Dopamine and Renalase in type 2 diabetic patients with and without diabetic nephropathy.

Subjects & Methods: This study was conducted on 80 subjects. Group 1:10 healthy volunteers as controls, group 2:60 type 2 diabetic patients with normal or increased albumin excretion rate (AER) and group 3:10 type 2 diabetic patients on maintenance hemodialysis (HD). To all studied subjects thorough clinical assessment and laboratory investigations included: Estimation of serum levels of fasting glucose (FSG), urea, creatinine, calcium and phosphorus. Estimation of urinary albumin/creatinine ratio (ACR) to assess AER and estimation of plasma Dopamine and serum Renalase were done by ELISA.

Results: There were no significant differences in the mean Dopamine levels between the three studied groups. Renalase level was significantly higher in HD patients compared to controls and other diabetic patients. Diabetic patients with increased AER had significantly higher systolic blood pressure, serum creatinine and Renalase levels. Diabetic patients with increased serum creatinine ≥1.5 mg/dl had significantly higher systolic and diastolic blood pressures. They also had significantly higher AER, FSG, Dopamine and Renalase levels. ACR was positively correlated with duration of diabetes, systolic and diastolic blood pressure and serum creatinine. Renalase was positively correlated with diastolic blood pressure, ACR, serum creatinine, phosphorus and Dopamine.

Conclusion: Serum levels of Renalase are increased in type 2 diabetic patients with renal dysfunction. Renalase levels may be increased to compensate the increase in Dopamine level. The higher Renalase level in HD patients may be due to much lower Renalase clearance, higher production or slower degradation in these patients.

Biochemical approaches for diabetic management

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Diabetes mellitus is the most prevalent metabolic syndrome world-wide, characterized by hyperglycemia (increase in glucose level) resulting in various short-term metabolic changes in lipid and protein metabolism and long-term irreversible vascular changes. When proteins are exposed to elevated levels of glucose a series of non-enzymatic chemical reactions occur that lead to the gradual build-up of advanced glycation end products (AGEs) in body tissues that cause various complications in the body. Hyperglycemia, affects eyes (cataract), blood vessels (atherosclerosis), nerves (neuropathy), kidney (nephropathy) and cause impaired wound healing. Postprandial hyperglycemia is an independent risk factor for cardiovascular diseases. Non-enzymatic models for anti-glycation i.e., BSA-MG and enzymatic model α-glucosidase inhibition will be discussed. In glycation, reactive intermediate methyl glyoxal (MG) binds with amino acid more easily than its carbohydrate precursor. Serum albumin, 80% of blood protein, is more prone to non-enzymatic glycation. Inhibition of protein glycation due to hyperglycemia is therefore an important and attractive approach towards the prevention and management of late diabetic complications. Alpha-glucosidase is an enzyme responsible for the conversion of complex carbohydrates to glucose. Keeping this in view, our group is working for investigation of novel anti-glycating agents. Based on virtual screening results, we have synthesized several classes of compounds and evaluated them for their in vitro and in vivo α-glucosidase inhibitory potential and methyl glyoxal binding potential. All these interesting results will be discussed in detail during talk.