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Efficacy of urinary n-acetyl β - D-Glucosaminidase in detecting renal tubular damage: An early consequence in Type 2 diabetes mellitus leading to Diabetic nephropathy

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Progression of diabetic nephropathy is strongly associated with irreversible loss of renal function, which remains undiagnosed till the appearance of microalbuminuria, decrease in creatinine clearance and/or increase in serum creatinine, observed at the late stage 2 of diabetic nephropathy. Proximal tubular damage plays a central role in pathogenesis of Type 2 diabetic nephropathy (T2DM) and the site specific enzymes located in renal tubules may be the useful prognostic markers. Thus, this study was aimed to evaluate the prognostic accuracy of urinary excretion of N-acetyl β -D-glucosaminidase (NAG), which is localized in the lysosomes of renal tubules for the onset of T2DM. The study was conducted on the total of 491 eligible participants including 76 healthy controls, 194 T2DM patients with duration 0-5 yrs, 5-10 yrs, 10-15 yrs and 15-20 yrs of diabetes, 71 microalbuminuric patients, 100 diabetic nephropathy patients and 50 non-diabetic nephropathy patients. Fasting glucose, serum fructosamine, HbA1C, urinary microalbumin, serum creatinine, estimated glomerular filtration rate (eGFR), serum-NAG and urinary-NAG were estimated. The urinary excretion of NAG was compared with other well established markers like microalbuminuria, eGFR, serum creatinine, which are routinely used for assessment of diabetic nephropathy. Urinary-NAG excretion was increased by 8 and 12 folds in Type 2 diabetic patients of 10-15 yrs and 15-20 yrs of diabetes duration respectively without showing microalbuminuria. The 16 and 18 fold increased urinary-NAG was observed in microalbuminuric and diabetic nephropathy patients respectively, which has not shown any alteration in non-diabetic nephropathy patients. A cutoff value of 3 U/L of urinary NAG has demonstrated a sensitivity of 96.1% and a specificity of 100% to discriminating healthy controls from patients with diabetes duration 10-15 yrs (AUC 1.000), 15-20 yrs of diabetes (AUC 0.999), microalbuminuria (AUC 0.999) and diabetic nephropathy (AUC 1.000). Excretion of urinary-NAG was gradually increased with the duration of diabetes and appeared much before the microalbuminuria and increased serum creatinine. Thus, urinary-NAG may be used as a potential site specific early tubular damage marker leading to diabetic nephropathy.

Biography

Kiran Kalia is a Professor since 1998 at School of Biosciences, Sardar Patel University, Vallabh Vidyanagar, and joined this university 1986 after obtaining her Ph.D from Industrial Toxicology Research Centre – Lucknow. She was a visiting professor at School of Health Sciences, Purdue University, USA in 2006 under DBT overseas programme 2006-07.

Kalia has to her credit 32 years of teaching and research experience, published over 65 publications in various national and international peer review journals and over 100 presentations at various national and international symposiums and conferences and 2 chapters in book, published by International Publishers.

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