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Transforming growth factor-beta 1 in diabetic nephropathy

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In diabetic nephropathy the extent of matrix accumulation in both glomeruli and the interstitium correlates strongly with the degree of renal insufficiency and proteinuria. Factors responsible for the deposition and accumulation of extra cellular matrix material within the kidney are therefore of considerable interest. Such factors include the potent fibrotic cytokine TGF-beta. We measured serum TGF-beta1 in patients with various stages of diabetic nephropathy, and correlated its level with different biochemical parameters. The study was conducted on: Group I: 30 patients with diabetic nephropathy (Subgroup IA: 20 patients with microalbuminuria; Subgroup IB: 10 patients with overt nephropathy), Group II: 19 diabetic patients without nephropathy (positive control), Group III: 20 healthy volunteers (negative control). Serum creatinine, Fasting and postprandial blood glucose, Fasting serum cholesterol, Glycated haemoglobin (HbA1c), Microalbumin estimation in urine, Serum TGF-beta1 estimation were done for all the studied groups. Our results showed a statistically significantly higher serum TGF-beta1 level in patients with diabetic nephropathy versus diabetic patients without nephropathy (mean +/- SD, 47.66 +/- 21.92 and 27.07 +/- 15.46 respectively) (P<0.001). Also in patients with diabetic nephropathy versus healthy controls (mean +/- SD, 47.66 +/- 21.29 and 27.05 +/- 8.95 respectively) (P<0.001). While serum TGF-beta1 concentrations were almost similar in diabetic patients without nephropathy and in healthy controls. Serum TGF-beta1 was statistically significantly higher in patients with overt nephropathy versus patients with microalbuminuria (mean+/-SD, 73.5 +/- 2.41 and 34.9 +/- 12.41) (P<0.001). Serum TGF-beta1 was significantly positively correlated with albumin excretion rate, fasting and postprandial blood glucose levels, serum cholesterol and HbA1c, these correlations were only found in diabetic patients with nephropathy but not in those without nephropathy or the control group. (r=0.86, P<0.001, r=0.444, P<0.05, r=0.375, P<0.05, r=0.532, P <0.01, r=0.696, P<0.001 respectively. HbA1c was found to be predictor of 68% of changes of serum TGF-beta1 (P<0.001) and serum cholesterol was predictor of 73% of changes of serum TGF-beta1 concentration (P<0.01). In conclusion, our results suggest that TGF-beta1 may play a key role in the development and progression of diabetic nephropathy. Accordingly, it may be also directly implicated in the functional deterioration of the kidney functions seen in patients with diabetic nephropathy, therefore beside proper glycemic control, strategies aiming at antagonizing TGF- beta1 for example by the use of specific antibodies or a specific inhibitor of TGF-beta1 may help to prevent the development or attenuate the progression of nephropathy in diabetic patients.