Brief Communication

2 year followup of patients with diabetes mellitus nephropathy showing albuminuria reversal following angiotensin converting enzyme inhibitors

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ABSTRACT

Introduction: Two-year follow-up of patients with diabetes mellitus (DM) nephropathy shows albuminuria reversal following angiotensin converting enzyme (ACE) inhibitors. **Aim:** To study about a clinical profile of 2-year follow-up of patients with DM nephropathy showing albuminuria reversal following ACE inhibitors. **Materials and Methods:** Twenty patients were taken up for study with duly informed consent and suggested for glycemic profile with HbA1C. Baseline renal function, urine microscopy, albuminuria, and other microvascular complications such as neuropathy and retinopathy. These patients were followed up for a period of 2 years with every month follow-up and monthly dose titration of ACE inhibitors, enalapril (Quote: Dr. M. K. Mani), to a maximum tolerable dose and checked after 1 week for raise in creatinine and potassium. **Inclusion Criteria:** Twenty patients, who have attended a secondary level diabetic clinic with diabetic nephropathy and are on regular follow-up for 2 years, were selected. **Exclusion Criteria:** Sick patients requiring parenteral feeds, IV antibiotics, co-morbid conditions such as autonomic gastroparesis and diabetic foot infections, type 1 diabetes and other known kidney disease, chronic kidney disease on dialysis are excluded from the study. **Expected Result:** Reversal of albuminuria.**Conclusion:** Enalapril is a safe, cheaper ACE inhibitors and the good dose titration coupled with early screening for DM nephropathy really help in halting the progression of chronic kidney disease from DM nephropathy.

Key words: Diabetes mellitus nephropathy, albumin reversal, angiotensin converting enzyme inhibitors

Introduction

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Aim

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Materials and Methods

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Twenty patients, who have attended a secondary level diabetic clinic with diabetic nephropathy and are on regular follow-up for 2 years, were selected.

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Expected Result

Reversal of albuminuria

Conclusion

Enalapril is a safe, cheaper ACE inhibitors and the good dose titration coupled with early screening for DM nephropathy really help in halting the progression of chronic kidney disease (CKD) from DM nephropathy.

Brief Communication

Diabetes (types 1 and 2) constitutes the commonest current cause of established renal failure in the industrialized world. Type 2 diabetes is rapidly increasing in prevalence and, although often a disease of middle to old age, is occurring more frequently in younger age groups as global obesity increases. In about one-third of patients, renal involvement can occur usually after 10-20 years of diabetes (diabetic nephropathy is small vessel complications, such as sensorimotor/autonomic neuropathy, retinopathy, and small arteriolar disease). The first sign of renal involvement by diabetic nephropathy is microalbuminuria (MAU). Early treatment with ACEI and ARB, to lower blood pressure and reduce MAU, can abort the otherwise inevitable progression to overt proteinuria and renal functional decline. It is of the greatest importance that all patients with diabetes have annual urine testing for the presence of MAU, and have meticulous attention paid to overall metabolic control, dyslipidemia, and blood pressure. Cardiovascular disease is very common in all diabetics, the more so in those with any degree of renal disease.

Association of diabetic nephropathy with other complications of diabetes

The prognosis for diabetic patients with any degree of diabetic nephropathy is much poorer than for individuals without nephropathy. The risk of cardiovascular disease (CVD) and of other microvascular complications is greatly increased. Indeed, diabetic nephropathy may be a vascular disease.

Cardiovascular disease

In T1DM, the relative risk of CVD is 1.2-fold in microalbuminuric and 10-fold higher in proteinuric than normoalbuminuric patients. The cumulative incidence of CVD by the age of 40 years is 43% in patients with T1DM with diabetic nephropathy, compared to 7% in patients without diabetic nephropathy, with a 10-fold increased risk of coronary heart disease and stroke. In ESRD, the risk is even higher. In T2DM, with MAU, the risk is increased two- to four-fold and with proteinuria nine-fold. Once serum creatinine is out of normal range, cardiovascular risk increases exponentially. Survival with ESRD is very limited: 20-25% of individuals with T2DM die in the 1st year of dialysis, and almost all are died within 4-5 years.

Hypertension

Hypertension is an invariable accompaniment of persistent proteinuria and end stage renal disease (ESRD). The excess prevalence of hypertension in T1DM is confined to patients with nephropathy, individuals with normal urinary albumin excretion (UAE) having a prevalence of hypertension similar to the non-diabetic population. This suggests that hypertension is an integral part of diabetic nephropathy, perhaps arising from the same underlying mechanisms. In support of this, BP rises very early in the development of nephropathy. Accordingly, patients with UAE in the high normal range, who are at increased risk of progression to MAU, have a higher blood pressure (BP) than those with lower UAE. Changes in BP are very subtle at this stage and may only be documented on 24-h BP monitoring, perhaps as reduced dipping in nocturnal diastolic BP.

Other microvascular complications

Patients with nephropathy are also much more likely to have other microvascular complications. In fact, the absence of other microvascular disease should make one question the diagnosis of diabetic nephropathy as the cause for the renal disease. Significant retinopathy is almost invariably present in patients with T1DM with MAU or more severe renal disease. In patients with T2DM, the relationship is less clear-cut. Those with classic nephropathy and progressively increasing UAE usually have significant retinopathy. In those with non-classic disease, with non-progressive low levels of MAU, retinopathy may be absent.

Peripheral neuropathy is also more common in patients with diabetes and renal disease, associating with both albuminuria and declining glomerular infiltration rate (GFR). Autonomic neuropathy, perhaps relating to loss of nocturnal BP dipping, occurs frequently.

We have time tested ACE inhibitor being well known to arrest the progress of diabetic nephropathy in increasing

dosage. But in clinical practice because of various reasons like compliance with patient and physician, it is not carried out. Nephropathy is a known microalbumin complication of diabetes with a characteristic by lesion of glomerulosclerosis with progressive damage to the filtering mechanism of the glomerulus resulting in albuminuria. Albuminuria begets in albuminuria which further deranges the glomerulus progressing to complete damage to the nephrons and CKD. This progression of nephropathy to CKD can be reasonably arrested by prevention of albumin excretion in excess by drugs. That is ACE inhibitors but in increasing doses.

We have enrolled patients diagnosed with nephropathy documented by MAU, albuminuria, retinopathy with neuropathy evident by quantitative tuning fork assessment. The ACE inhibitor of Choice was Enalapril (Ref: Dr. Mani), titratable to a maximum dose as the drug was cheaper than other ACE inhibitors and no major recent adverse event profile with good patient counseling and after detailed explanation. We were able to follow them up with titratable dose of 5 mg every month. Monitoring with urea, creatinine, and potassium were checked the following week.

All investigation were carried once in our laboratory which undergoes to external quality assessment programs from CMC, Erba, EQAS. Blood urea done by ureas method creatitine done by potassium by electrolysis method in our 20 patient followed up for 2 years one patient complaints of relevant folliculitis abscess.

All the patient showed albuminuria reversal maintaining at a good GFR and were tolerating the maximum dose.[1-3]

References

1. Unnikrishnan RI, Rema M, Pradeepa R, Deepa M, Shanthirani CS, Deepa R, *et al*. Prevalence and risk factors of diabetic nephropathy in an urban South Indian population: The Chennai Urban Rural Epidemiology Study (CURES 45). Diabetes Care 2007;30:2019-24.
2. Damsgaard EM, Mogensen CE. Microalbuminuria in elderly hyperglycaemic patients and controls. Diabet Med 1986;3:430-5.
3. Nelson RG, Knowler WC, Pettitt DJ, Hanson RL, Bennett PH. Incidence and determinants of elevated urinary albumin excretion in Pima Indians with NIDDM. Diabetes Care 1995;18:182-7.

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