

EFFECT OF LISINOPRIL ON MICROALBUMINURIA

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The purpose of this study was to evaluate the effect of Lisinopril on microalbuminuria in non- insulin dependent diabetic hypertensive patients. One hundred and twenty patients were divided into two groups. Group-A was given placebo of Lisinopril and Group-B was given Lisinopril in a fixed dose of 10 mg once daily for two weeks. Comparison of microalbuminuria before and after the treatment showed a decrease in microalbuminuria level in patients treated with Lisinopril.

INTRODUCTION

In diabetic hypertensive patients suffering from diabetic nephropathy, early antihypertensive treatment can improve the course of albumin excretion and renal function as it is found that advanced diabetic nephropathy is frequently associated with heavy proteinuria.

In hypertensive diabetic patients with nephropathy, the use of ACE inhibitors may protect the renal function through its action on internal haemodynamics¹. Proteinuria in diabetics is aggravated by systemic hypertension or hyperglycemia, it can be alleviated by maintaining long term strict control of these conditions².

It has been found that elevation in transcapillary hydraulic pressure is mainly responsible for proteinuria. In a study on Lisinopril and Nifedipine in non-insulin dependent diabetic patients, it was found that both drugs induced a reduction in the albumin excretion rate although Lisinopril appeared to be better tolerated than Nifedipine³. In the present study we noted the effect of Lisinopril on microalbuminuria in non-insulin dependent diabetes mellitus patients.

MATERIAL AND METHODS

One hundred and twenty outdoor patients of both sexes, ages between 35-60 years, visiting the diabetic clinic of JPMC. Karachi, were included in this study. Patients selected were having systolic blood pressure range of 160-200 mm Hg and diastolic blood pressure range of 92-114 mm Hg. Their fasting blood glucose level ranged from 140-200 mg/dl. All the patients were advised to take fixed diet during the study period and avoid from overeating. They were advised to continue their normal activities. Patients having gastrointestinal tract, hepatic and ischaemic disorders were not included in the study.

The subjects were prepared by withdrawal of all antihypertensive drugs for two weeks. Their 24 hours urinary microalbumin level on day-0 was determined as base line reading. Patients were divided into two groups.

Group-A: Control

Consisted of 60 mild to moderate hypertensive non-insulin dependent diabetes mellitus patients. They were given glibenclamide 5 mg once daily with placebo of Lisinopril for two weeks.

Group-B:

Consisted of 60 milds to moderate hypertensive non-insulin dependent diabetes mellitus patients, and they were given glibenclamide 5 mg once daily and Lisinopril 10 mg once daily for two weeks. After 14 days, 24-hour urinary albumin level was again determined. Urinary microalbumin level was estimated by colorimetric test "Pyrogallol Red" by using microalbumin estimation kit prepared by Reactivos Spin React, SA. The reaction between the protein and Pyrogallol Molibidate forms a Red complex. The colour is directly proportional to the albumin concentration. Statistical analysis was performed by means of students "t" test.

RESULTS

Group-A:

Consisted of mild to moderate diabetic hypertensive patients. They were given placebo of Lisinopril for two weeks. Their 24 hours urinary microalbumin level on day-0 was 153.16±4.80 and on day-14 it was 165.70±4.96 mg/24 hours.

Table-1: Effect of Lisinopril On Microalbuminuria.

Drug Regimen	0-week (mg/24 hours)	2-weeks (mg/24 hours)
Placebo	153.16 ± 4.80	165.70±4.96
Lisinopril	155.70±4.89	130.10±4.14

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Group-B:

Consisted of patients who were given Lisinopril 10 mg once a day for two weeks. Their 24 hours urinary microalbumin level on day-0 was 155.70 ± 4.89 and on day 14 it was 130.10 ± 4.14 .

Table-1 shows the mean 24-hours urinary microalbumin values at the base line and after two weeks of medication.

DISCUSSION

In the present study it was found that Lisinopril reduced the 24 hours urinary microalbumin level, while without Lisinopril the urinary microalbumin level was increased. Lisinopril reduced the 24 hours urinary microalbumin level approximately 25 mg/24 hours. This reduction in 24 hours urinary microalbumin may be due to reduction in glomerular filtration. Although there are three determinants, glomerular capillary permeability, transcapillary hydraulic pressure and capillary plasma flow rate¹. In diabetic nephropathy, transcapillary hydraulic pressure and glomerular plasma flow rate are enhanced i.e. hyper-filtration is caused by metabolic changes⁵. Lisinopril was observed in a study on uninephrectomized rats and was found that it significantly reduced the blood pressure, completely prevented proteinuria and renal function deterioration⁶.

In the present study Lisinopril decreased the level of microalbuminuria, which is beneficial for patients, our results are also supported by some recent studies such as, treatment of hypertension with ACE inhibitors in diabetic patients reduce the microalbuminuria and slows down the progression of nephropathy compared with agents that do not maintain declines in proteinuria⁷.

In another study it was concluded that Lisinopril has a significantly more beneficial effect on urinary albumin excretion than Nifedipine, despite similar effects on both blood pressure and glycaemic control in type-111 diabetic patients with hypertension⁸.

In this study it is concluded that the use of Lisinopril in type-11 diabetic hypertensive patients, produces marked decrease in microalbuminuria. Therefore, it is suggested that this drug is highly beneficial if given to NIDDM hypertensive patients.

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