



Present study was carried out in 33 patients of diabetic nephropathy, who were attending diabetic clinic regularly at M.L.B. Medical College, Jhansi. Out of 33, 16 patients were included in captopril group and remaining 17 in lisinopril group.

#### Captopril Group

Sixteen patients were included in captopril group, 11 were males and 5 females. Maximum number of cases belonged to age group 41-50 years (37.5%). Out of 16 patients, 10 (62.5%) were having non-insuline dependent Diabetes Mellitus and rest had Insulin dependent Diabetes Mellitus.

#### Lisinopril Group

Seventeen cases were included in lisinopril group, out of which, 10 were males and 7 females. Maximum number of cases were in age group of 51-60 years (29.5%). Out of 17 patients, 9 (52.9%) were having noninsulin dependent Diabetes Mellitus, while 8 (47.1%) had Insulin dependent Diabetes Mellitus.

Maximum duration of Diabetes Mellitus in captopril group was 1-5 years in 9 (56.3%) patients, while in lisinopril group, the duration was same in 8 (47.1%) patients. All of these patients were having significant proteinuria despite short duration of diabetes mellitus.

Bjorck Staffan et al (1985) studied 15 patients with insulin dependent diabetes. All patients had diabetic nephropathy and mean duration of diabetes was 22 years. All of them were given captopril and frusemide in combination.

In present study, such short duration of diabetes mellitus could be because of late diagnosis. In Bundelkhand region most of the patients are illiterate and socio-economically poor and they are not aware of the symptoms of Diabetes Mellitus.

#### EFFECT ON BLOOD PRESSURE

##### Captopril Group

In NIDDM, mean systolic blood pressure at 0 month was  $149.8 \pm 26.5$  mm Hg which came down to  $133.6 \pm 20.0$  mm Hg after two months. This change is insignificant ( $p > 0.05$ ). Similarly the diastolic blood pressure fell from initial  $82.4 \pm 13.0$  mm Hg to  $76.2 \pm 13.0$  mm Hg after 2 months, again this decrease is insignificant ( $p > 0.05$ ). It is due to the fact that in present study most of the cases were normotensive while only 2 patients were having mild hypertension.

Similarly it was not observe any significant decrease in systolic and diastolic blood pressure in IDDM patients, who were given captopril. Again it is because there was no hypertensive patient in this group and the dose of captopril used was too low to cause hypotension.

### Lisinopril Group

In NIDDM patients the mean systolic blood pressure fell from  $145.6 \pm 20.1$  mm Hg to  $126.9 \pm 15.2$  mm Hg. This change is insignificant ( $p > 0.05$ ) while mean diastolic blood pressure fell from initial  $90 \pm 14.4$  to  $78.4 \pm 9.3$  mm Hg after 2 months but this decrease is significant ( $p < 0.05$ ). It may be due to the fact that 33% patients in this group were having mild to moderate hypertension.

Similarly in IDDM patients, the mean systolic blood pressure fell from  $132.0 \pm 33.4$  to  $112.0 \pm 19.1$  mm Hg and this change is insignificant ( $p > 0.05$ ) while mean diastolic blood pressure fell from  $84.0 \pm 19.8$  to  $68 \pm 14.2$  mm Hg, but this change is significant ( $p < 0.05$ ). In this group also, 2 patients had mild hypertension and another patient had severe hypertension.

### EFFECT OF PROTEINURIA

In present study, 16 patients were included in captopril group. Out of 16 patients, 10 were related to NIDDM. In these patients initially mean urinary protein excretion was  $139 \pm 92.4$  mg/l which fell down to  $128 \pm 101.7$  mg/l. This difference is statistically insignificant ( $p > 0.05$ ).

In lisinopril group, 17 patients were included out of which, 9 patients were related to NIDDM. In

these patients, initially mean urinary protein was  $201.1 \pm 40.7$  mg/l. After 2 months therapy the mean excretion was only  $158 \pm 82$  mg/l. The difference is insignificant ( $p > 0.05$ ).

These drugs were also given to patients related to IDDM group in which 6 patients were treated with captopril while 8 patients with lisinopril. Similarly in these both groups, protein excretion was reduced but difference is statistically insignificant ( $p > 0.05$ ).

On comparing the captopril group to lisinopril group according to their efficacy in reducing proteinuria, the difference is insignificant ( $p > 0.05$ ). With present study it can be said that there is no difference between captopril and lisinopril regarding effect on proteinuria in diabetic nephropathy.

According to Bjorck Staffan et al (1986), 14 patients of diabetic nephropathy were treated with captopril for 2 years. The mean urinary protein excretion was  $2.9(2.0)$  gm/24 hours before and  $2.8(1.9)$  gm/24 hours after treatment. Protein excretion was reduced in 10 of the 14 patients but this decreased protein excretion was statistically insignificant.

According to Yoshio Taguma et al (1985) 10 azotemic diabetic with heavy proteinuria were treated with captopril (37.5 mg/day) for 2 months. Urinary protein decreased promptly within two weeks from  $10.6 \pm 2.2$  to

6.1±1.4 gm/day. This decrease of protein excretion was statistically significant ( $p < 0.01$ ).

In present study, the effect of captopril and lisinopril on mean urinary protein excretion was not significant but proteinuria was reduced in most of the patients. But Taguma et al reported that treatment with captopril reduced proteinuria in diabetic nephropathy. The patients in their study, however, different from present study. They were much older and had more severe proteinuria, congestive heart failure was common among their patients but was present in none of our patients. Differences between the patients may therefore explain the different effects of captopril on proteinuria.

#### EFFECT ON RENAL FUNCTION

##### Blood Urea :

In present study 10 patients of NIDDM were treated with captopril and 9 patients with lisinopril. In captopril group, initially the mean blood urea was 33.5±13.6 mg/dl which fell down to 30.9±10.7 mg/dl after 2 months' treatment. This difference is statistically insignificant ( $p > 0.05$ ), but in patients with lisinopril group the mean value of blood urea was increased from 26.4±5.5 to 30.4±5.2 mg/dl and again this difference is insignificant ( $p > 0.05$ ).

Similarly 6 patients of IDDM were treated with captopril and 8 patients with lisinopril (Table XXII &

XXIII) but difference of values of both groups after 2 months treatment were statistically insignificant ( $p > 0.05$ ).

On comparing the results of captopril and lisinopril as mentioned above, in NIDDM patients, the levels of blood urea slightly increased in lisinopril group while in case of captopril the level of blood urea decreased but in IDDM patients the values of blood urea decreased in both groups.

Statistical comparison of both drugs shows that effects of captopril and lisinopril are the same ( $p > 0.05$ ) on blood urea. The insignificant effect on blood urea could be because most of the values were within normal range (Table XIX, XX, XXII and XIII).

#### SERUM CREATININE

Same patients were also investigated for serum creatinine before and after 2 months of therapy. In NIDDM patients of captopril group, the mean serum creatinine decreased from  $1.1 \pm 0.2$  to  $1.0 \pm 0.2$  mg/dl but in NIDDM patients of lisinopril group the values were same before and after 2 months of therapy. The changes were <sup>not</sup> statistically significant ( $p > 0.05$ ).

Similarly IDDM patients of captopril group were also investigated and serum creatinine decreased from  $1.4 \pm 0.6$  to  $1.2 \pm 0.4$  mg/dl but in IDDM patients of lisinopril group, the mean values decreased from  $1.3 \pm 0.4$  to  $1.2 \pm 0.3$  mg/dl. Again these changes are statistically

insignificant ( $p > 0.05$ ). Statistical insignificant effect on serum creatinine is because all initial findings of serum creatinine were within normal limits.

According to Bjorck Staffan et al (1986) 13 patients were treated with captopril and frusemide in combination for 2 years. All patients had diabetic nephropathy. The mean serum creatinine concentration was 2.1 mg/dl before treatment and 2.5 mg/dl at the end of the follow up period.

According to Siphae Lee et al (1990) 11 patients of diabetic nephropathy were treated with captopril for 24 months and serum creatinine increased from  $1.72 \pm 0.43$  mg/dl ( $N = 11$ ) to  $3.45 \pm 0.67$  mg/dl ( $N = 6$ ), this increase was statistically significant ( $p < 0.01$ ).

#### GLOMERULAR FILTRATION RATE

In present study, GFR was calculated in 10 patients of NIDDM who were treated with captopril while 9 patients of NIDDM were treated with lisinopril. In captopril group, initially the mean GFR was  $68.9 \pm 13.2$  ml/min and it increased to  $73.0 \pm 14.5$  it increased to  $73.0 \pm 14.5$  ml/min after 2 months of therapy. This difference is insignificant ( $p > 0.05$ ), and in patients of lisinopril group the GFR increased from  $69 \pm 25.6$  to  $70.3 \pm 21.2$  ml/min which is again insignificant ( $p > 0.05$ ).



Similarly 6 patients of IDDM were treated with captopril and 8 patients with lisinopril and results are shown in table XXXIV and XXXV but difference of values of both groups after 2 months were statistically insignificant ( $p > 0.05$ ).

On comparing, results show that effect of captopril and lisinopril on renal function are almost same, and it was maintained through out the study period.

According to Mogensen et al (1982), Poring et al (1983), Bjorck Staffan et al (1986), Eva Hommel (1986), Parving (1988) and Sphae Lee et al (1990) the effect of angiotensin converting enzyme inhibitors on renal function was almost same and maintained through out the study period. ACE inhibitors delayed the renal failure which is much similar to present study.

-----