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Original Article

Anti-proteinuric Effect of Ramipril and Cilnidipine in Diabetic Nephropathy - A Comparative Study

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ABSTRACT

Background: Reduction of proteinuria leads to reduced risk of adverse renal and cardiovascular events. Angiotensin-converting enzyme (ACE) inhibitors have been established to be first-line drugs in preventing the development and retarding the progress of diabetic nephropathy by reducing proteinuria. Apart from this Cilnidipine a dihydropyridine derivative dual L/N type calcium channel blocker also has effect on reducing proteinuria by mechanism that differs from that of ACEI. **Objective:** To compare the anti-proteinuric effect of Cilnidipine and Ramipril. **Methods & Materials:** This randomized control experimental study was conducted in the Department of Nephrology, Bangabandhu Sheikh Mujib Medical University, Dhaka for the duration of 10 months. Diabetic Nephropathy Patients were taken as the study population. Total 93 subjects were enrolled as study sample with a view to determine the presence of proteinuria in Diabetic Nephropathy patients. Informed written consent was taken from all study population. Data were obtained in preformed data collection sheet and analyzed by SPSS program version 26. **Results:** Mean age of the study population was in Group A was 45.9±13.5 years and 50.1±12.1 in Group B. Male female distribution in two groups were matched. At enrolment the mean systolic blood pressure recorded of the patients was 132.6±6.1 mmHg and the mean diastolic blood pressure 82.8±6.8 mmHg, after addition of Ramipril and Cilnidipine there was a reduction of blood pressure at 3 and 6 months. The mean UTP level in Group A at the start of study was 4.87±1.85 gm and in Group B 5.35±1.78 gm. At the end of 3 months, the mean UTP was 4.47±1.65 in Group A and 4.70±1.66 in Group B. The mean UTP reduction from baseline at the end of 6 months in Group A was 2.88±1.25 while in Group B it was 3.78±1.48 ($p < 0.001$). The mean uACR level in Group A at the start of study was 2223.4±163.8 gm and in Group B 2821.9±159.4 gm. At the end of 3 months, the mean uACR was 1964.6±142.6 mg/gm in Group A and 2415.6±144.0 mg/gm in Group B. The mean uACR reduction from baseline at the end of 6 months in Group A was 1190.5±98.7 mg/gm while in Group B it was 1828.9±116.9 mg/gm (p=0.004). **Conclusion:** We conclude that in diabetic population, Ramipril has better effect on proteinuria reduction than Cilnidipine.

Keywords: *Diabetic nephropathy, Ramipril, Cilnidipine, Proteinuria, Urinary albumin-to-creatinine ratio.*

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INTRODUCTION

Diabetes mellitus (DM) is a leading cause of death and disability World-wide [1]. Its global prevalence is about 8% in 2011 and is predicted to rise to 10% by 2030 (IDF. 2012). In Bangladesh, a recent meta-analysis showed that the prevalence of diabetes among adults had increased substantially, from4% in 1995 to 2000 and 5% in 2001 to 2005 to 9% in 2006 to 2010 [2]. Overall, the prevalence of diabetes in Bangladesh is 9.7% (aged 35 years or more). Diabetic nephropathy, which damage kidney, can lead to the chronic renal failure, eventually to death. There are four types of lesions described in diabetic nephropathy: diabetic glomerulosclerosis, vascular lesions, diabetic pyelonephritis and necrotizing renal papillitis and tubular lesions or Armanni-Ebstein lesion. However, the classical definition of diabetic nephropathy is somewhat different characterized with a progressive rise in urine albumin excretion, coupled with increasing blood pressure, leading to declining glomerular filtration and eventually end stage kidney failure. Recently, links have been observed between nephropathy and cardiovascular disease [3]. Diabetic nephropathy have led to the inclusion of premature cardiovascular disease, cardiovascular risk increasing in parallel with albuminuria [4]. Diabetic nephropathy is now the commonest cause of ESRF worldwide and is acknowledged as an independent risk factor for cardiovascular disease. Today, diabetic renal disease describes not only diabetic nephropathy but also atheroembolic disease, ischemic nephropathy, and interstitial fibrosis that occurs as a direct result of diabetes [5]. Micro albuminuria precedes overt proteinuria by several years, a potentially reversible state [6]. Recently importance of micro albuminuria has been raised because its appearance in diabetics predicts development of macro albuminuria and coronary artery disease. Progression of diabetic nephropathy in diabetic individuals with micro albuminuria has been reported to be retarded by good glycemic control. In addition, tight blood pressure control also has been noted to reduce micro albuminuria, particularly with the use of ACEI [7,4]. Current treatment strategies are based on inhibition of the renin–angiotensin–aldosterone system (RAAS), using angiotensin- converting enzyme inhibitors (ACEI) or angiotensin II receptor blockers (ARB), with the dual aim of managing hypertension and obtaining remission of proteinuria. In patients with micro albuminuria, ACEI or ARB treatment significantly retards the progression of proteinuria and slows the rate of loss of renal function [8]. Remission of proteinuria has been reported in up to 50% of patients with type 2 diabetes and microalbuminuria, and is associated with a reduction in the incidence of cardiovascular morbidity and mortality Most L-type calcium channel blockers dilate only renal afferent arterioles and are prone to causing glomerular hypertension [9]. It has been reported that Cilnidipine, a L/N-type calcium channel blocker, inhibits sympathetic nervous activity by blocking N-type calcium channels and dilating renal afferent and efferent arterioles equally, showing a renoprotective effect, compared with other calcium channel blockers, this drug reduces urinary albumin and protein excretion to a greater extent [10,11]. It has a

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urinary protein-lowering effect equivalent to ACEIs and its combination with an ARB further reduces albuminuria. The aim of this study is to assess and compares the anti proteinuric effect of Cilnidipine along with Ramipril [12].

OBJECTIVES

General Objective

- To compare the anti-proteinuric effect of Ramipril and Cilnidipine.

Specific Objectives

- To assess the anti-proteinuric effect of Ramipril.
- To assess the anti-proteinuric effect of Cilnidipine.
- To determine the urinary protein level of the study subjects.
- To determine the urinary albumin-to-creatinine ratio (uACR) of the study subjects.
- To measure the HbA1c level of the study subjects.

METHODS & MATERIALS

This was an interventional study. The study was conducted in the Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. The study was conducted from September 2019 to June 2020. The study population consisted of adult patients with diabetic nephropathy attending the outpatient and inpatient departments of Nephrology, BSMMU. A total of 93 patients were included in the study.

Inclusion Criteria: Adult patients aged ≥18 years with diabetic nephropathy, proteinuria (uACR ≥30 mg/g), eGFR ≥60 mL/min/1.73 m², HbA1c ≤7%, and those who provided informed written consent were included in the study.

Exclusion Criteria: Patients with eGFR ≤60 mL/min/1.73 m², those previously or currently receiving antiproteinuric drugs, and patients with blood pressure ≥140/90 mmHg were excluded from the study.

Study Procedure

Eligible adult patients with diabetic nephropathy were screened and enrolled after fulfilling the predefined inclusion and exclusion criteria and providing written informed consent. Participants were randomized into two groups using a computer-generated random number table. Group A received Ramipril 5 mg once daily, while Group B received Cilnidipine 10 mg once daily for 6 months. Concomitant antidiabetic and antihypertensive medications were continued as required to maintain HbA1c <7% and blood pressure <140/90 mmHg. Clinical and laboratory parameters, including urinary total protein and uACR, were assessed at baseline, after 3 months, and after 6 months. Participants were followed regularly to evaluate treatment efficacy, adherence, and adverse drug reactions, with telephone contact maintained to minimize loss to follow-up.

Data Collection

Relevant data were collected using a preformed data collection sheet. Additional information was obtained from the patients' history sheets, clinical examinations, and laboratory investigation reports.

Data Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 26. Quantitative variables were expressed as mean ± standard deviation (SD), and statistical significance was assessed using Student's unpaired *t*-test. Qualitative variables were expressed as frequency and percentage, and the Chi-square test was used to assess statistical significance. The results were presented in tables, figures, and diagrams.

Ethical Considerations

Prior to commencement of the study, the research protocol was approved by the Ethical Review Committee of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. The aims, objectives, procedures, potential risks, and benefits of the study were explained to the participants in an easily understandable local language. Written informed consent was obtained from each participant before enrollment. Participants were assured that all information and records would be kept confidential and that the collected data would be used solely for research purposes.

RESULTS

The mean age of the patients was 45.9 ± 13.5 years in Group A and 50.1 ± 12.1 years in Group B. There was no statistically significant difference in mean age between the two groups (p=0.073). In Group A, 60.4% of the patients were male and 39.6% were female, whereas in Group B, 55.6% were male and 44.4% were female. The distribution of sex was comparable between the two groups (p=0.635). Regarding BMI, the majority of patients were overweight in both groups, accounting for 54.2% in Group A and 48.9% in Group B. The mean BMI was 25.2 ± 2.98 kg/m² in Group A and 24.8 ± 3.4 kg/m² in Group B, with no statistically significant difference between the groups (p=0.453). At baseline, the mean systolic blood pressure was 132.6 ± 6.1 mmHg in Group A and 134.2 ± 5.2 mmHg in Group B. At 3 months, it was 129.8 ± 4.6 mmHg and 130.3 ± 5.1 mmHg, respectively, while at 6 months it was 125.7 ± 5.1 mmHg and 123.0 ± 6.2 mmHg, respectively. No statistically significant difference was observed in mean systolic blood pressure between the two groups at baseline or during follow-up. Similarly, the mean diastolic blood pressure was 82.8 ± 6.8 mmHg in Group A and 83.9 ± 6.3 mmHg in Group B at baseline, which decreased to 80.9 ± 4.5 mmHg and 81.8 ± 4.5 mmHg at 3 months and to 80.1 ± 3.1 mmHg and 79.8 ± 3.3 mmHg at 6 months, respectively. No statistically significant difference was observed between the groups at any follow-up interval. The mean HbA1c level at baseline was 7.04 ± 0.24% in Group A and 6.97 ± 0.25% in Group B. At 3 months, the mean HbA1c level was 7.00 ± 0.22% in Group A and 6.94 ± 0.23% in Group B, while at 6 months it was 6.93 ± 0.26% and 6.82 ± 0.27%, respectively. No statistically significant difference was observed in mean HbA1c levels between the two groups at baseline or during follow-up (p>0.05). The mean serum creatinine level at baseline was 1.03 ± 0.11 mg/dL in Group A and 1.01 ± 0.09 mg/dL in Group B. At 3 months, the mean serum creatinine level was 0.98 ± 0.21 mg/dL in Group A and 0.94 ± 0.08 mg/dL in Group B, while at 6 months it was 0.93 ± 0.20 mg/dL and 0.91 ± 0.11 mg/dL, respectively. No statistically significant difference was observed in mean serum creatinine levels between the two groups at baseline, 3 months, or 6 months. The mean UTP level at baseline was 4.87 ± 1.85 g in Group A and 5.35 ± 1.78 g in Group B. After 3 months of treatment, the mean UTP level decreased to 4.47 ± 1.65 g in Group A and 4.70 ± 1.66 g in Group B. At the end of 6 months, the mean reduction in UTP from baseline was 2.88 ± 1.25 g in Group A and 3.78 ± 1.48 g in Group B. The difference in UTP reduction between the two groups was statistically significant (p<0.001).The mean uACR level at baseline was 2223.4 ± 163.8 mg/g in Group A and 2821.9 ± 159.4 mg/g in Group B. At the end of 3 months, the mean uACR decreased to 1964.6 ± 142.6 mg/g in Group A and 2415.6 ± 144.0 mg/g in Group B. At the end of 6 months, the mean reduction in uACR from baseline was 1190.5 ± 98.7 mg/g in Group A and 1828.9 ± 116.9 mg/g in Group B. The reduction in uACR was statistically significant between the two groups at 6 months (p=0.004). Regarding adverse effects, in the Ramipril group, nausea/vomiting was reported in 2 (4.2%) patients, hypotension in 1 (2.1%) patient, hyperkalaemia in 2 (4.2%) patients, and cough in 2 (4.2%) patients. In the Cilnidipine group, tachycardia and peripheral edema were reported in 2 (4.4%) patients each, while nausea/vomiting and hypotension were reported in 1 (2.2%) patient each. Overall, the reported adverse effects were infrequent in both groups during the 6-month follow-up period.

Table I
Demographic Characteristics of the Patients in Two Groups (*n*=93).

Demographic Characteristics	Group A (n=48) No. (%)	Group B (n=45) No. (%)	p-value
Age group (years)			
31–40	11 (22.9)	10 (22.2)	0.073
41–50	12 (25.0)	8 (17.8)	
51–60	13 (27.1)	13 (28.9)	
61–70	12 (25.0)	14 (31.1)	
Mean ± SD	45.9 ± 13.5	50.1 ± 12.1	
Sex			
Male	29 (60.4)	25 (55.6)	0.635
Female	19 (39.6)	20 (44.4)	
Ratio	1.5:1	1.3:1	
BMI (kg/m²)			
Underweight (<18.5)	4 (8.3)	4 (8.9)	0.453
Normal (18.5–24.9)	18 (37.5)	19 (42.2)	
Overweight (25.0–29.9)	26 (54.2)	22 (48.9)	
Mean ± SD	25.2 ± 2.98	24.8 ± 3.4	

Data were expressed as frequency, percentage, and mean ± SD. Unpaired Student's t-test was used for quantitative variables and Chi-square test for qualitative variables. GroupA:Patients administered Ramipril 5 mg daily. Group B: Patients administered Cilnidipine 10 mg daily.

Table II
Blood Pressure Between the Two Groups at Baseline and Different Follow-up Intervals (*n*=93).

Blood Pressure	Group A (n=48) Mean ± SD	Group B (n=45) Mean ± SD	p-value
SBP (mmHg)			
At baseline	132.6 ± 6.1	134.2 ± 5.2	0.178
At 3 months	129.8 ± 4.6	130.3 ± 5.1	0.620
At 6 months	125.7 ± 5.1	123.0 ± 6.2	0.109
DBP (mmHg)			
At baseline	82.8 ± 6.8	83.9 ± 6.3	0.684
At 3 months	80.9 ± 4.5	81.8 ± 4.5	0.259
At 6 months	80.1 ± 3.1	79.8 ± 3.3	0.147

Unpaired Student's t-test. Group A: Patients administered Ramipril 5 mg daily. Group B: Patients administered Cilnidipine 10 mg daily.

Table III
HbA1c Between the Two Groups at Baseline and Different Follow-up Intervals (*n*=93).

HbA1c	Group A (n=48) Mean ± SD	Group B (n=45) Mean ± SD	p-value
At baseline	7.04 ± 0.24	6.97 ± 0.25	0.117
At 3 months	7.00 ± 0.22	6.94 ± 0.23	0.170
At 6 months	6.93 ± 0.26	6.82 ± 0.27	0.088

Unpaired Student's t-test. Group A: Patients administered Ramipril 5 mg daily. Group B: Patients administered Cilnidipine 10 mg daily.

Table IV
Serum Creatinine Between the Two Groups at Baseline and Different Follow-up Intervals (*n*=93).

Serum Creatinine	Group A (n=48) Mean ± SD	Group B (n=45) Mean ± SD	p-value
At baseline	1.03 ± 0.11	1.01 ± 0.09	0.341
At 3 months	0.98 ± 0.21	0.94 ± 0.08	0.233
At 6 months	0.93 ± 0.20	0.91 ± 0.11	0.554

Unpaired Student's t-test. Group A: Patients administered Ramipril 5 mg daily. Group B: Patients administered Cilnidipine 10 mg daily.

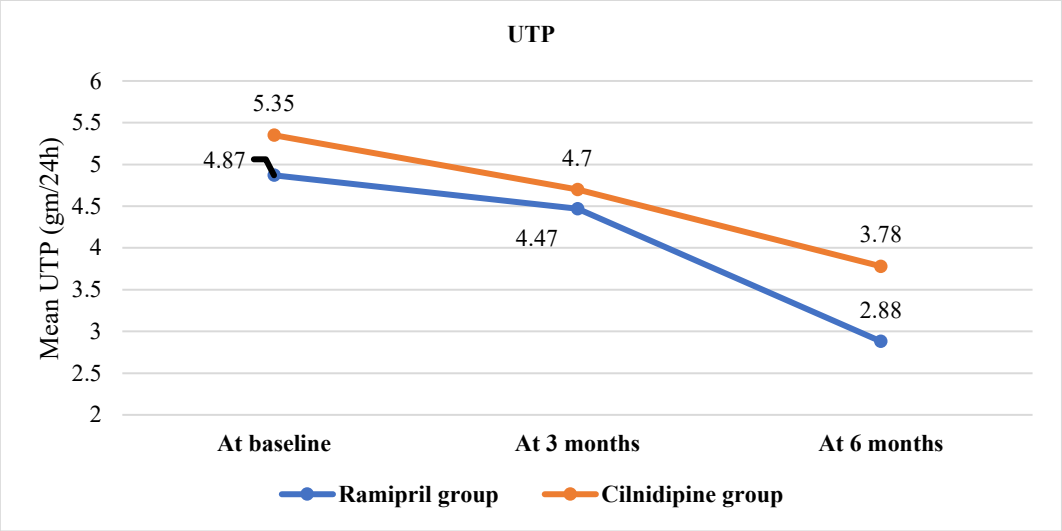


Figure 1 Line diagram showing the mean changes of UTP at 3 months and 6 months.

Table V
uACR Between the Two Groups at Baseline and Different Follow-up Intervals (*n*=93).

uACR	Group A (n=48) Mean ± SD	Group B (n=45) Mean ± SD	p-value
At baseline	2223.4 ± 163.8	2821.9 ± 159.4	0.077
At 3 months	1964.6 ± 142.6	2415.6 ± 144.0	0.133
At 6 months	1190.5 ± 98.7	1828.9 ± 116.9	0.004*

**Unpaired Student's t-test; p<0.05 considered statistically significant. Group A: Patients administered Ramipril 5 mg daily. Group B: Patients administered Cilnidipine 10 mg daily.*

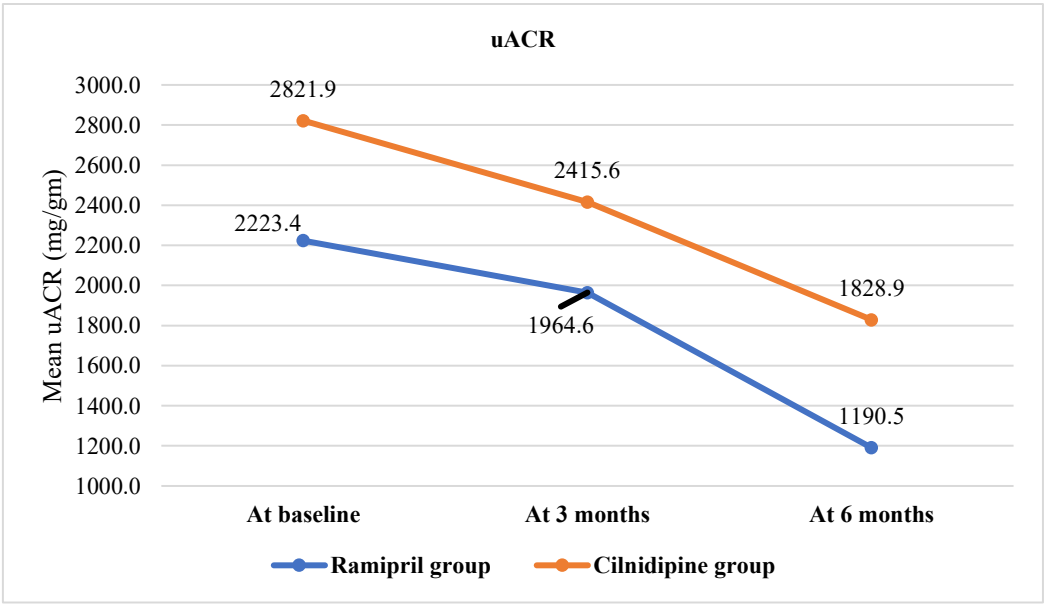


Figure 2 Line diagram showing the mean changes of uACR at the end of 3rd month and 6th month.

Table VI
Distribution of Patients by Adverse Effects in Group A (*n*=48).

Adverse Effects	Frequency	Percentage
Nausea/vomiting	2	4.2
Hypotension	1	2.1
Hyperkalaemia	2	4.2
Cough	2	4.2

Group A: Patients administered Ramipril 5 mg daily.

Table VII
Distribution of Patients by Adverse Effects in Group B (*n*=45).

Adverse Effects	Frequency	Percentage
Nausea/vomiting	1	2.2
Hypotension	1	2.2
Tachycardia	2	4.4
Peripheral edema	2	4.4

Group B: Patients administered Cilnidipine 10 mg daily.

DISCUSSION

This study compares the anti-proteinuric effect of Ramipril (Group A) and Cilnidipine (Group B). The mean age of the study population in Group A was 45.9±13.5 years and 50.1±12.1 in Group B. No significant difference of mean age between two groups. Similar mean age was observed in another study by Hwang et al. (2017) which included patients with a mean age of 61±8.5 years [13]. This might be because most of the patients enrolled in the study were diagnosed as having diabetic nephropathy developing mostly 10-15 years following the onset of diabetes, also a number of epidemiological studies have shown an association between increasing age and incidence of nephropathy [14]. There are a number of other studies that have a mean age which is much similar to current study like one study conducted by Malleshappa (2013) had a mean age of 44.9±13.4 years [15]. In present study in Group A, 60.4% patients were male and 39.6% were female. In Group B 55.6% patients were male and 44.4% were female. No significant difference of male and female distribution between two groups. Number of male patients observed in the study were higher that is in accordance to a number of studies like the one conducted by Malleshappa (2013) consisting of 35 males compared to only 25 females [15]. Another study by Tanaka et al. (2010) also consisted of 53 male and 24 female subjects [16]. The difference in the prevalence of diabetic nephropathy among males and females has a very varying picture, a number of studies have observed the effects of gender on diabetic kidney disease but unfortunately these results are always contradictory, maybe due to the sample size which is small, differences in study population or methodology. In the present study at enrolment the mean systolic blood pressure of the patients was 132.6±6.1mmHg and the mean diastolic blood pressure was 82.8±6.8 mmHg, after addition of Ramipril and Cilnidipine there was a reduction of blood pressure at 3 and 6 months. Sharma et al. (2020) reported mean systolic blood pressure recorded of the patients was 147.76±21.34mmHg and the mean diastolic blood pressure was 82.96±13.23 mmHg at baseline [17]. After addition of Cilnidipine there was a reduction of 2 mmHg in the systolic blood pressure which was similar to current study. Hence it can be inferred that the anti-proteinuric effect of Cilnidipine is completely independent of its anti-hypertensive effect. Globally diabetes mellitus has emerged as the commonest cause of end stage renal disease. It is the etiological factor in 20–40% of all ESRD patients. Microalbuminuria (30–300 mg albumin/24 h) is a well-known predictor of poor renal outcomes in patients with type 2 diabetes [18,19]. Micro albuminuric patients with type 2 diabetes if left untreated, almost invariably progress to macro albuminuria and overt diabetic nephropathy. Although there are various classes of drugs to treat proteinuria, we compared Group A (Ramipril 5 mg) with Group B (Cilnidipine 10 mg) to find out whether both have similar efficacy or one is better than another in reduction of proteinuria. In our study, (Group A) was more effective in reducing proteinuria than Cilnidipine (Group B). In Group A proteinuria was reduced by 40.8%, while in Group B there was reduction of 29.3%, which was significant. In accordance, Singh et al. (2015) reported in a study, combination regimen of Enalapril and Cilnidipine was more effective in reducing proteinuria than Enalapril alone [6]. In Enalapril group proteinuria was reduced 25.68%, while in Cilnidipine there was reduction of 54.88%, which was significant. Katayama et al. (2006) also conducted a similar trial in patients with type II diabetes with proteinuria [20]. The patients were randomized into two groups to receive either Valsartan (an ARB) or Valsartan plus Cilnidipine for 1-year. After 1-year, proteinuria was found to have decreased more in the Valsartan plus Cilnidipine group (−44 ± 11%) than in the Valsartan group (−9 ± 7%) (Katayama et al., 2006) [20]. The antiproteinuric action of ACE inhibitor and its superiority over L-type CCB is supported by a trial conducted by Remuzzi et al. (2006) over hypertensive patients with type-2 diabetes and normal urinary albumin excretion rate [21]. The study showed that diabetic nephropathy can be prevented by ACE inhibitor therapy but combination of ACE inhibitor with non-dihydropyridine calcium channel blocker (verapamil) does not provide any additional protective effect over kidney against the development of microalbuminuria. Hatta et al. (2012) also observed that in chronic kidney disease patients, Cilnidipine has antihypertensive effects equivalent to Amlodipine, but proteinuria was reduced by shifting from Amlodipine to Cilnidipine [22]. Kojima et al. (2004) also did a study in 28 proteinuric hypertensive patients. One group was kept on Amlodipine and other on Cilnidipine [23]. The Amlodipine group showed a significant increase in proteinuria, while the increase was suppressed in the Cilnidipine group. Tanaka (2010) conducted his trial in over 25 diabetic patients with hypertension who were on treatment with CCB other than Cilnidipine [16]. Medication was changed to Cilnidipine, and there was a significant decrease in urinary albumin/creatinine ratio after 3 months of the new treatment. Zaman and Kumari (2013) also compared the effects of Cilnidipine and Amlodipine over BP, heart rate, proteinuria and lipid profile in hypertensive patients [24]. They found that both drugs significantly reduced BP. There was decrease in pulse rate, urinary protein excretion and serum triglyceride in diabetic patients in Cilnidipine group. Our study focused on proteinuria and its levels observed after therapeutic intervention with antiproteinuric agents as determinant for progression of diabetic kidney disease. We also see the effect on serum creatinine level in between groups. The results of our study indicate greater reduction in level of proteinuria by giving Ramipril than Cilnidipine. The Ramipril leads to greater reduction in proteinuria as compared to Cilnidipine. Mathiesen et al. (1999) reported ACE inhibitors are established agents to reduce proteinuria in diabetic nephropathy [25].

CONCLUSION

We conclude that in diabetic nephropathy population, both Ramipril and Cilnidipine have antiproteinuric effect. Although Ramipril is more effective in proteinuria reduction than Cilnidipine. In our study we found no significant change in serum creatinine level in between two groups. In the present study, we demonstrate that the anti-proteinuric effect of both Ramipril and Cilnidipine is sustained for six months during continued treatment in diabetic nephropathy patients. Hence, Cilnidipine can also be used as an alternative antiproteinuric medication in diabetic nephropathy where Ramipril cannot be given.

LIMITATIONS

The main drawback of our study was that it was a single center, open-labeled randomized trial conducted over a small number of patients. Duration of the study was also short to comment on its renal benefits.

RECOMMENDATION

A large scale, multicenter, double-blind clinical trial involving a larger number of patients for a longer duration will be needed in future.

REFERENCES

1. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, Abraham J, Adair T, Aggarwal R, Ahn SY, AlMazroa MA. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. The lancet. 2012 Dec 15;380(9859):2095-128.
2. Saquib N, Saquib J, Ahmed T, Khanam MA, Cullen MR. Cardiovascular diseases and type 2 diabetes in Bangladesh: a systematic review and meta-analysis of studies between 1995 and 2010. BMC public health. 2012 Jun 13;12(1):434. DOI: https://doi.org/10.1186/1471-2458-12-434.
3. Vallianou NG, Mitesh S, Gkogkou A, Geladari E. Chronic kidney disease and cardiovascular disease: is there any relationship?. Current cardiology reviews. 2019 Feb 1;15(1):55-63. DOI: https://doi.org/10.2174/1573403x14666180711124825.
4. Ritz E, Rychlík I, Locatelli F, Halimi S. End-stage renal failure in type 2 diabetes: a medical catastrophe of worldwide dimensions. American journal of kidney diseases. 1999 Nov 1;34(5):795-808. DOI: https://doi.org/10.1016/s0272-6386(99)70035-1.
5. López-Novoa JM, Rodríguez-Peña AB, Ortiz A, Martínez-Salgado C, López Hernández FJ. Etiopathology of chronic tubular, glomerular and renovascular nephropathies: clinical implications. Journal of translational medicine. 2011 Jan 20;9(1):13. DOI: https://doi.org/10.1186/1479-5876-9-13.
6. Singh A, Satchell SC. Microalbuminuria: causes and implications. Pediatric Nephrology. 2011 Nov;26(11):1957-65. DOI: https://doi.org/10.1007/s00467-011-1777-1.
7. Vijan S, Hayward RA. Treatment of hypertension in type 2 diabetes mellitus: blood pressure goals, choice of agents, and setting priorities in diabetes care. Annals of internal medicine. 2003 Apr 1;138(7):593-602. DOI: https://doi.org/10.7326/0003-4819-138-7-200304010-00018.
8. De Zeeuw D, Remuzzi G, Parving HH, Keane WF, Zhang Z, Shahinfar S, Snapinn S, Cooper ME, Mitch WE, Brenner BM. Albuminuria, a therapeutic target for cardiovascular protection in type 2 diabetic patients with nephropathy. Circulation. 2004 Aug 24;110(8):921-7. DOI: https://doi.org/10.1161/01.cir.0000139860.33974.28.
9. MacIsaac RJ, Jerums G. Diabetic kidney disease with and without albuminuria. Current opinion in nephrology and hypertension. 2011 May 1;20(3):246-57. DOI: https://doi.org/10.1097/mnh.0b013e3283456546.
10. Konno Y, Kimura K. Vasodilatory effect of cilnidipine, an L-type and N-type calcium channel blocker, on rat kidney glomerular arterioles. International Heart Journal. 2008;49(6):723-32. DOI: https://doi.org/10.1536/ihj.49.723.
11. Konda T, Enomoto A, Matsushita J, Takahara A, Moriyama T. The N-and L-type calcium channel blocker cilnidipine suppresses renal injury in dahl rats fed a high-sucrose diet, an experimental model of metabolic syndrome. Nephron Physiology. 2005 Sep 1;101(1):p1-3. DOI: https://doi.org/10.1159/000085713.
12. Rose GW, Kanno Y, Ikebukuro H, Kaneko M, Kaneko K, Kanno T, Ishida Y, Suzuki H. Cilnidipine is as effective as benazepril for control of blood pressure and proteinuria in hypertensive patients with benign nephrosclerosis. Hypertension Research. 2001;24(4):377-83. DOI: https://doi.org/10.1291/hypres.24.377.
13. Hwang YC, Yoon KH, Cha BS, Lee KW, Jang HC, Min KW, Chung CH, Lee MK. Reduction in microalbuminuria by calcium channel blockers in patients with type 2 diabetes mellitus and hypertension—A randomized, open-label, active-controlled, superiority, parallel-group clinical trial. International Journal of Clinical Practice. 2017 Sep;71(9):e12987. DOI: https://doi.org/10.1111/ijcp.12987.
14. Varma PP. Prevalence of chronic kidney disease in India-Where are we heading?. Indian journal of nephrology. 2015 May;25(3):133. DOI: https://doi.org/10.4103/0971-4065.148304.
15. Malleshappa P. Cilnidipine effectively reduces low-grade albuminuria in hypertensive chronic kidney disease patients. Dialisis Y Trasplante. 2013 Jan 1;34(1):2-6. DOI: https://doi.org/10.1016/j.dialis.2012.04.001.
16. Tanaka M. The L/N-type calcium channel blocker, Cilnidipine, reduces heart rate and albuminuria in patients with type 2 diabetes. Journal of International medical research. 2010 Apr;38(2):602-10. DOI: https://doi.org/10.1177/147323001003800222.
17. Sharma T, Tausif A, Ahmad S. Evaluating the antiproteinuric efficacy of cilnidipine in diabetic kidney disease. Int J Basic Clin Pharmacol. 2019;24(9):129. DOI: https://doi.org/10.18203/2319-2003.ijbcp20195774.
18. Keane WF, Brenner BM, De Zeeuw D, Grunfeld JP, McGill J, Mitch WE, Ribeiro AB, Shahinfar S, Simpson RL, Snapinn SM, Toto R. The risk of developing end-stage renal disease in patients with type 2 diabetes and nephropathy: the RENAAL study. Kidney international. 2003 Apr 1;63(4):1499-507. DOI: https://doi.org/10.1046/j.1523-1755.2003.00885.x.
19. Anavekar NS, Gans DJ, Berl T, Rohde RD, Cooper W, Bhaumik A, Hunsicker LG, Rouleau JL, Lewis JB, Rosendorff C, Porush JG. Predictors of cardiovascular events in patients with type 2 diabetic nephropathy and hypertension: a case for albuminuria. Kidney International. 2004 Nov 1;66:S50-5. DOI: https://doi.org/10.1111/j.1523-1755.2004.09213.x.
20. Katayama K, Nomura S, Ishikawa H, Murata T, Koyabu S, Nakano T. Comparison between valsartan and valsartan plus cilnidipine in type II diabetics with normo-and microalbuminuria. Kidney international. 2006 Jul 1;70(1):151-6. DOI: https://doi.org/10.1038/sj.ki.5000349 https://doi.org/10.1038/sj.ki.5000349.
21. Remuzzi G, Macia M, Ruggenenti P. Prevention and treatment of diabetic renal disease in type 2 diabetes: the BENEDICT study. Journal of the American Society of Nephrology. 2006 Apr 1;17(4_suppl_2):S90-7. DOI: https://doi.org/10.1681/asn.2005121324.
22. Hatta T, Takeda K, Shiotsu Y, Sugishita C, Adachi T, Kimura T, Sonomura K, Kusaba T, Kishimioto N, Narumiya H, Tanda S. Switching to an L/N-type calcium channel blocker shows renoprotective effects in patients with chronic kidney disease: the Kyoto Cilnidipine Study. Journal of International Medical Research. 2012 Aug;40(4):1417-28. DOI: https://doi.org/10.1177/147323001204000420.
23. Kojima S, Shida M, Yokoyama H. Comparison between cilnidipine and amlodipine besilate with respect to proteinuria in hypertensive patients with renal diseases. Hypertension Research. 2004;27(6):379-85. DOI: https://doi.org/10.1291/hypres.27.379.
24. Zaman ZA, Kumari V. Comparison of the effects of amlodipine and cilnidipine on blood pressure, heart rate, proteinuria and lipid profile in hypertensive patients. Clinical Pharmacology, 2(2), 160. DOI: https://doi.org/10.5455/2319-2003.ijbcp20130308.
25. Mathiesen ER, Hommel E, Hansen HP, Smidt UM, Parving HH. Randomised controlled trial of long-term efficacy of captopril on preservation of kidney function in normotensive patients with insulin dependent diabetes and microalbuminuria. Bmj. 1999 Jul 3;319(7201):24-5. DOI: https://doi.org/10.1136/bmj.319.7201.24