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ORIGINAL ARTICLE

Comparative Antiproteinuric Efficacy of Ramipril and Cilnidipine in Diabetic Nephropathy: A Randomized Controlled Trial

Iklash Uddin Choudhury[1], Abu Ahmed Golam Akbar[2], Sayful Hasan[3], Ishtiaqe Bakht[4], Shahana Reza[5], Alahi Khandaker[6*]

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*Corresponding Author

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ABSTRACT

Background: *Diabetic nephropathy is a leading cause of end-stage kidney disease worldwide, with proteinuria serving as a key modifiable risk factor. Renin-angiotensin system blockade remains the standard antiproteinuric therapy, though alternative agents like cilnidipine may offer additional benefits through dual calcium channel blockade.* ***Objective:*** *To compare the antiproteinuric efficacy and safety of ramipril versus cilnidipine in adult patients with diabetic nephropathy.* ***Methods & Materials:*** *This interventional study was conducted at Bangabandhu Sheikh Mujib Medical University, Dhaka, from September 2019 to June 2020. Ninety-three patients with diabetic nephropathy (uACR ≥30 mg/g, eGFR ≥60 ml/min/1.73m^2, HbA1C ≤7%) were randomized to receive ramipril 5mg daily (n=48) or cilnidipine 10mg daily (n=45) for six months. We assessed urinary albumin-to-creatinine ratio, urinary total protein, blood pressure, serum creatinine, HbA1c, and adverse effects at baseline, 3 months, and 6 months. Data were analyzed using SPSS v26.* ***Results:*** *Baseline characteristics were comparable between groups. At six months, urinary albumin-to-creatinine ratio reduction was significantly greater in the cilnidipine group (1828.9±116.9 mg/g vs. 1190.5±98.7 mg/g, p=0.004). Urinary total protein reduction also favored cilnidipine (3.78±1.48 gm vs. 2.88±1.25 gm, p<0.001). Blood pressure, serum creatinine, and HbA1c showed no significant intergroup differences. Adverse effects were mild and comparable between groups.* ***Conclusion:*** *Cilnidipine demonstrates superior antiproteinuric efficacy compared with ramipril in patients with diabetic nephropathy, with a comparable safety profile. Cilnidipine may represent a valuable alternative for proteinuria reduction, particularly in patient's intolerant to renin-angiotensin system blockers.*

Keywords: *Antiproteinuric, Cilnidipine, Diabetic nephropathy, Proteinuria, Ramipril*

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1. *Medical Officer, 250 Bedded General Hospital, Moulvibazar, Bangladesh*
2. *Assistant Professor, Department of Nephrology, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh*
3. *Assistant Professor, Department of Nephrology, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh*
4. *Dialysis Medical Officer, Sylhet MAG Osmani Medical College Hospital, Sylhet, Bangladesh*
5. *Medical Officer, 250 bedded General Hospital, Chittagong, Bangladesh*
6. *Health Policy Analyst, Bangladesh Center for Health Studies, Dhaka, Bangladesh (ORCID: 0000-0002-6551-1309)*

INTRODUCTION

Diabetic nephropathy (DN) is a clinical syndrome occurring in approximately 20% to 30% of patients with diabetes mellitus and remains the leading cause of end-stage kidney disease worldwide [1]. The condition is characterized by persistent microalbuminuria, which subsequently progresses to macroalbuminuria, and is accompanied by a progressive decline in estimated glomerular filtration rate, ultimately leading to renal failure [2]. Epidemiological data from recent large-scale surveys demonstrate that the prevalence of diabetic kidney disease among hospitalized patients with diabetes is approximately 39.9%, with albuminuria present in 36.6% of cases [3]. In community-based populations, the prevalence has been reported at 25.4%, with albuminuria accounting for 22.0% of cases [4]. These figures underscore the substantial public health burden posed by DN and the urgent need for effective therapeutic strategies to retard its progression. The pathophysiology of DN is multifactorial, involving complex interactions between hemodynamic, metabolic, and inflammatory pathways [1]. Hyperglycemia initiates a cascade of deleterious effects, including activation of the renin-angiotensin-aldosterone system, oxidative stress, and advanced glycation end-product formation, all of which contribute to glomerular hypertension, podocyte injury, and progressive proteinuria [5]. Proteinuria itself is not merely a marker of renal damage but an active mediator of tubulointerstitial fibrosis, making its reduction a critical therapeutic target [6]. Among the earliest clinically detectable abnormalities in DN is microalbuminuria, which, if left untreated, progresses to overt proteinuria and is strongly predictive of adverse renal and cardiovascular outcomes [7]. Renin-angiotensin system blockade with angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers has long been established as the cornerstone of antiproteinuric therapy in DN [8]. These agents reduce intraglomerular pressure by preferentially dilating the efferent arteriole, thereby decreasing urinary albumin excretion and slowing the decline in renal function [9]. Ramipril, a widely used ACE inhibitor, has demonstrated consistent antiproteinuric efficacy in patients with DN, with studies reporting a reduction

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in urinary albumin-to-creatinine ratio of 30% or more in approximately 50% of treated patients [10]. Despite this well-documented benefit, a substantial proportion of patients continue to exhibit persistent proteinuria despite maximal RAAS blockade, necessitating exploration of additional therapeutic options [11]. Calcium channel blockers have emerged as potential adjunctive agents for proteinuria reduction, though their antiproteinuric effects vary considerably depending on the specific agent employed [12]. Traditional L-type calcium channel blockers, such as amlodipine, predominantly dilate afferent arterioles and may not consistently reduce intraglomerular pressure, limiting their antiproteinuric efficacy [11]. In contrast, cilnidipine is a unique fourth-generation dihydropyridine calcium channel blocker that blocks both L-type and N-type calcium channels [13]. The N-type calcium channel blockade inhibits sympathetic neurotransmitter release from nerve terminals supplying the renal vasculature, leading to dilation of both afferent and efferent arterioles, a reduction in intraglomerular pressure, and consequently, a decrease in urinary protein excretion [13]. This dual mechanism of action distinguishes cilnidipine from conventional L-type calcium channel blockers and suggests a potentially superior renoprotective profile [13]. Clinical evidence supporting the antiproteinuric efficacy of cilnidipine has been accumulating. Studies have demonstrated that cilnidipine reduces urinary protein-to-creatinine ratio and improves kidney function in patients with diabetic kidney disease [14]. Furthermore, when added to ACE inhibitor therapy, cilnidipine has shown additive antiproteinuric effects, with one study reporting a significant reduction in microalbuminuria with combination therapy compared to ACE inhibitor alone [15]. However, the comparative efficacy of cilnidipine versus ACE inhibitors as monotherapy in DN has not been extensively investigated. Meta-analyses have suggested that the antiproteinuric efficacy of calcium channel blockers may be less than that of ACE inhibitors after short-term treatment, though both classes appear equally effective in reducing the progression from microalbuminuria to macroalbuminuria in the long term [12]. The SAKURA trial, a prospective randomized study comparing cilnidipine with amlodipine in renin-angiotensin system inhibitor-treated patients, found no significant difference in renoprotective effects between the two agents, though the study was not designed to compare cilnidipine against an ACE inhibitor [16]. Given the limited comparative data between cilnidipine and ramipril as monotherapy in patients with DN, this study was undertaken to compare the antiproteinuric efficacy and safety of cilnidipine versus ramipril in adult patients with diabetic nephropathy. We hypothesized that cilnidipine would demonstrate non-inferior antiproteinuric efficacy compared to ramipril, with a favorable safety profile.

METHODS & MATERIALS

This interventional study was conducted at the Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from September 2019 to June 2020. Adult patients with diabetic nephropathy attending the outpatient and inpatient departments of Nephrology were screened for eligibility. A total of 93 patients were enrolled and randomly allocated into two groups: 48 patients received ramipril 5 mg daily (Group A) and 45 patients received cilnidipine 10 mg daily (Group B). The sample size was calculated using the two-sample mean test formula (Rosner B. Fundamentals of Biostatistics, 2015) [17].

Inclusion criteria: Patients aged 18 years or older with diabetic nephropathy, defined as urinary albumin-to-creatinine ratio ≥30 mg/g, estimated glomerular filtration rate ≥60 ml/min/1.73m^2, and glycated hemoglobin ≤7%, were included. Only those who provided informed written consent were enrolled.

Exclusion criteria: Patients with eGFR <60 ml/min/1.73m^2, those previously or currently taking antiproteinuric drugs, and those with blood pressure ≥140/90 mmHg at baseline were excluded from the study.

Study procedure: Eligible patients were thoroughly counseled regarding the study protocol, including drug efficacy, safety, cost, and potential side effects. After obtaining informed written consent, patients were randomized using a random number table generated by a statistician. Group A received ramipril 5 mg daily, while Group B received cilnidipine 10 mg daily for six months. Both groups received standard antidiabetic medications to maintain HbA1c <7% and antihypertensive agents to maintain blood pressure <140/90 mmHg. Clinical and laboratory parameters, including urinary albumin-to-creatinine ratio, urinary total protein, serum creatinine, and HbA1c, were recorded at baseline, 3 months, and 6 months. Microalbuminuria was defined as urinary albumin excretion of 30 to 300 mg/day, while macroalbuminuria was defined as albumin excretion exceeding 300 mg/day [18]. Hypertension was defined according to JNC 8 criteria [19]. Regular follow-up was maintained through phone contact to minimize dropout and monitor adverse drug reactions.

Data analysis: Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0. Quantitative variables were expressed as mean ± standard deviation, and intergroup comparisons were performed using the unpaired Student's t-test. Qualitative variables were expressed as frequencies and percentages, with comparisons made using the Chi-square test. A p-value of <0.05 was considered statistically significant.

RESULT

A total of 93 patients with diabetic nephropathy were randomized into two groups: Group A (ramipril 5 mg daily, n=48) and Group B (cilnidipine 10 mg daily, n=45). All patients completed the six-month follow-up. Baseline demographic and clinical characteristics were comparable between groups (*Table I*). The mean age was 45.9 ± 13.5 years in Group A and 50.1 ± 12.1 years in Group B (p=0.073). Male predominance was observed in both groups (60.4% vs. 55.6%). BMI distribution was similar, with mean values of 25.2 ± 2.98 kg/m^2 and 24.8 ± 3.4 kg/m^2, respectively (p=0.453).

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Table I: Demographic characteristics of the patients in two groups (*n*=93)

Characteristics	Group A (n=48) n (%)	Group B (n=45) n (%)	p-value
Age group (years)			
31-40	11 (22.9)	10 (22.2)	
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	0.073
Sex			
Male	29 (60.4)	25 (55.6)	0.635
Female	19 (39.6)	20 (44.4)	
BMI (kg/m^2)			
Underweight (<18.5)	4 (8.3)	4 (8.9)	
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	0.453

Values are presented as frequency (percentage) or mean ± standard deviation. Continuous data were compared using the unpaired Student's t-test; categorical data were compared using the Chi-square test. A p-value < 0.05 was considered statistically significant. Group A: Ramipril 5 mg daily. Group B: Cilnidipine 10 mg daily.

Blood pressure parameters showed no significant differences between groups at any time point (*Table II*). At six months, systolic blood pressure was 125.7 ± 5.1 mmHg in Group A and 123.0 ± 6.2 mmHg in Group B (p=0.109); diastolic blood pressure was 80.1 ± 3.1 mmHg and 79.8 ± 3.3 mmHg, respectively (p=0.147).

Table II: Blood Pressure between 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	0.62
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

Values are presented as mean ± standard deviation. All continuous variables were compared using the unpaired Student's t-test. A p-value < 0.05 was considered statistically significant. Group A: Ramipril 5 mg daily. Group B: Cilnidipine 10 mg daily.

Glycemic control, measured by HbA1c, was well maintained and comparable between groups throughout the study (*Table III*). At six months, HbA1c was 6.93 ± 0.26% in Group A and 6.82 ± 0.27% in Group B (p=0.088). Serum creatinine levels remained stable, with no significant intergroup differences at any time point.

Table III: A1c between 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	0.17
At 6 months	6.93 ± 0.26	6.82 ± 0.27	0.088

Values are presented as mean ± standard deviation. All continuous variables were compared using the unpaired Student's t-test. A p-value < 0.05 was considered statistically significant. Group A: Ramipril 5 mg daily. Group B: Cilnidipine 10 mg daily.

The antiproteinuric efficacy of both drugs was assessed through urinary total protein (UTP) and urinary albumin-to-creatinine ratio (uACR). At six months, the mean UTP reduction from baseline was significantly greater in Group B (3.78 ± 1.48 gm) compared to Group A (2.88 ± 1.25 gm), with a p-value <0.001 (*Figure 1*).

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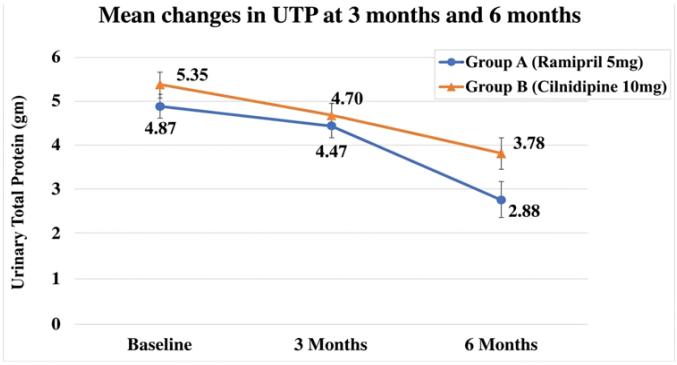


Figure 1: Line diagram showing the mean changes in UTP at 3 months and 6 months

Figure 1 shows the progressive decline in urinary total protein levels in both groups over the six-month study period. The cilnidipine group (Group B) demonstrates a steeper decline Urinary albumin-to-creatinine ratio showed a significant difference between groups at final follow-up (Table IV). At six months, mean uACR was significantly lower in Group B (1828.9 ± 116.9 mg/g) compared to Group A (1190.5 ± 98.7 mg/g), with a p-value of 0.004 (*Figure 2*).

Table IV: uACR between two groups at baseline and different follow-up intervals (*n*=93)

uACR (mg/g)	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*

*Values are presented as mean ± standard deviation. All continuous variables were compared using the unpaired Student's t-test. A p-value < 0.05 was considered statistically significant. *Significant. Group A: Ramipril 5 mg daily. Group B: Cilnidipine 10 mg daily.*

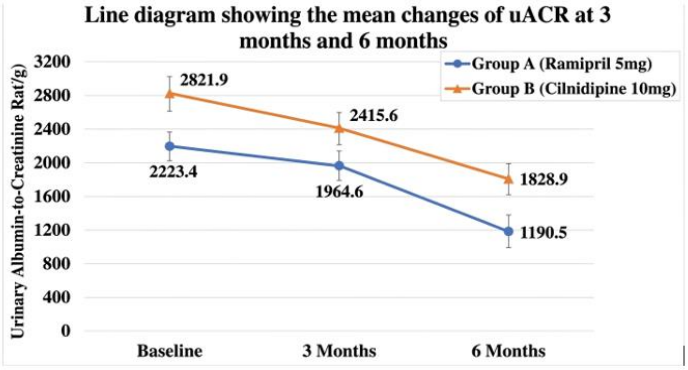


Figure 2: Line diagram showing the mean changes in uACR at 3 months and 6 months

Figure 2 demonstrates the serial changes in urinary albumin-to-creatinine ratio over the six-month study period. Both groups show a progressive reduction in uACR, with the cilnidipine group (Group B) exhibiting a significantly greater reduction at six months compared to the ramipril group (Group A), highlighting the superior antiproteinuric effect of cilnidipine. Adverse effects were mild and manageable in both groups. In Group A, nausea/vomiting occurred in 2 patients (4.2%), hypotension in 1 (2.1%), hyperkalemia in 2 (4.2%), and cough in 2 (4.2%). In Group B, nausea/vomiting occurred in 1 (2.2%), hypotension in 1 (2.2%), tachycardia in 2 (4.4%), and peripheral edema in 2 (4.4%). All adverse effects resolved with symptomatic management.

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DISCUSSION

The present study was undertaken to compare the antiproteinuric efficacy and safety of ramipril versus cilnidipine in adult patients with diabetic nephropathy. Our findings demonstrate that cilnidipine 10 mg daily is significantly more effective than ramipril 5 mg daily in reducing urinary protein excretion over a six-month treatment period, while both agents exhibit comparable blood pressure-lowering effects and safety profiles. These results contribute important comparative evidence to the clinical management of diabetic nephropathy, particularly for patients who may not tolerate or adequately respond to angiotensin-converting enzyme inhibitors. The most striking finding of this study was the superior reduction in urinary albumin-to-creatinine ratio achieved with cilnidipine compared to ramipril at six months (1828.9 ± 116.9 mg/g versus 1190.5 ± 98.7 mg/g, p=0.004). This difference represents a substantially greater antiproteinuric effect favoring cilnidipine, despite similar blood pressure control between groups. The reduction in urinary total protein was also significantly greater in the cilnidipine group (3.78 ± 1.48 gm versus 2.88 ± 1.25 gm, p<0.001). These findings align with previous studies demonstrating the antiproteinuric potential of cilnidipine in diabetic populations. A prospective randomized trial reported that the addition of cilnidipine to an angiotensin-converting enzyme inhibitor resulted in a significant reduction in microalbuminuria, confirming an additive effect beyond renin-angiotensin system blockade alone [20]. Another interventional study evaluating cilnidipine in diabetic kidney disease observed a significant reduction in urinary protein-to-creatinine ratio at 12 weeks, along with improvement in renal function parameters [21]. The superior antiproteinuric efficacy of cilnidipine observed in our study can be attributed to its unique dual L-type and N-type calcium channel blocking action [22]. Unlike conventional L-type calcium channel blockers such as amlodipine, which predominantly dilate afferent arterioles and may increase intraglomerular pressure, cilnidipine blocks N-type calcium channels in sympathetic nerve terminals supplying the renal vasculature. This action leads to dilation of both afferent and efferent arterioles, reduction in intraglomerular pressure, and consequently, decreased urinary protein excretion [20]. In diabetic patients, enhanced sympathetic nervous activity results in constricted efferent arterioles and elevated intraglomerular pressure, making the sympatholytic effect of cilnidipine particularly relevant [20]. This mechanistic distinction explains why cilnidipine, but not L-type calcium channel blockers, has demonstrated consistent antiproteinuric effects in renin-angiotensin system inhibitor-treated patients [23]. Blood pressure control was comparable between the two groups throughout the study, with no significant differences in systolic or diastolic blood pressure at any time point. This observation is clinically important as it indicates that the superior antiproteinuric effect of cilnidipine is independent of its blood pressure-lowering action. Previous comparative studies have similarly demonstrated that cilnidipine and amlodipine exert equivalent antihypertensive effects, yet cilnidipine produces a greater reduction in urinary protein excretion [24]. The absence of significant blood pressure differences between groups in our study strengthens the argument that the antiproteinuric advantage of cilnidipine arises from its unique intrarenal hemodynamic effects rather than systemic blood pressure reduction alone. Glycemic control, as measured by HbA1c, was well maintained and comparable between groups throughout the study period, with no significant intergroup differences at any time point. This finding is consistent with the understanding that neither ramipril nor cilnidipine directly affects glycemic parameters, and it confirms that the observed differences in antiproteinuric efficacy were not confounded by variations in glycemic control. Serum creatinine levels remained stable in both groups, with no significant changes from baseline or intergroup differences, indicating that neither agent adversely affected renal function over the six-month follow-up period. This safety profile is reassuring and consistent with published data showing nonsignificant changes in serum creatinine and estimated glomerular filtration rate with both agents [25]. The adverse effect profiles of both drugs were mild, manageable, and consistent with their known pharmacological properties. In the ramipril group, cough occurred in 4.2% of patients, a well-recognized side effect of angiotensin-converting enzyme inhibitors attributed to bradykinin accumulation [26]. Hyperkalemia was observed in 4.2% of ramipril-treated patients, which is consistent with the known effect of renin-angiotensin system blockade on potassium homeostasis [25]. In the cilnidipine group, peripheral edema occurred in 4.4% of patients, which is notably lower than the rates typically reported with L-type calcium channel blockers such as amlodipine [27]. This favorable tolerability profile may be attributable to cilnidipine's dual L/N-type blocking action, which reduces reflex sympathetic activation and minimizes the arteriolar vasodilation that contributes to peripheral edema [22]. Tachycardia occurred in 4.4% of cilnidipine-treated patients, consistent with the mild sympatholytic effect of N-type calcium channel blockade.

LIMITATIONS

The study is limited by its modest sample size, relatively short six-month follow-up period, open-label design, and use of fixed drug doses without titration, which may not reflect optimal clinical practice. The absence of long-term renal outcome data and biomarkers of renal injury beyond proteinuria further restricts the generalizability of findings.

CONCLUSION

Cilnidipine 10 mg daily demonstrates superior antiproteinuric efficacy compared to ramipril 5 mg daily in patients with diabetic nephropathy, with comparable blood pressure control and safety profiles over six months. The unique dual L/N-type calcium channel blocking action of cilnidipine, reducing intraglomerular pressure through dual arteriolar dilation, likely underlies this advantage. Cilnidipine may represent a valuable alternative for proteinuria reduction, particularly in patients intolerant to angiotensin-converting enzyme inhibitors.

RECOMMENDATION

Future prospective randomized controlled trials with larger sample sizes, longer follow-up periods, and dose-titration protocols are recommended to confirm these findings and determine whether the superior antiproteinuric effect of cilnidipine translates into long-term preservation of renal function and reduced cardiovascular morbidity.

CONFLICT OF INTEREST

The authors declare no conflicts of interest. No funding was received for this study. The corresponding author had full access to all data and final responsibility for the decision to submit for publication.

AI DECLARATION

Artificial intelligence tools contributed marginally to manuscript preparation and development. All core scientific content was human-verified.

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