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Research Article

A Comparative Study to Evaluate the Safety and Efficacy of Cilnidipine versus Amlodipine in Newly Diagnosed Essential Hypertensive Patients at a Tertiary Health Care Centre of Uttar Pradesh

Dr. Abhishek Kumar Singh[1], Dr. Anil Kumar[2], Dr. Niraj Kumar Chaudhari[3], Dr. Shilpa U. Vahikar[4]
[1]DOCTOR OF MEDICINE (MD) (PHARMACOLOGY)
[2]MD (Pharmacology), Associate Professor & Nodal Head, Department of Pharmacology, B.R.D. Medical College, Gorakhpur.
[3]MD (Medicine), Associate Professor, Department of Medicine, B.R.D. Medical College, Gorakhpur.
[4]MD (Pathology), Professor, Department of Pathology, B.R.D. Medical College, Gorakhpur.
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ABSTRACT
Introduction: Hypertension remains a major modifiable determinant of cardiovascular, cerebrovascular and renal morbidity, and sustained pharmacological therapy is frequently required to achieve adequate blood-pressure control. Amlodipine is an established long-acting L-type calcium-channel blocker, whereas cilnidipine inhibits both L- and N-type calcium channels and may provide additional attenuation of sympathetic activity. Comparative assessment of their antihypertensive efficacy and tolerability is clinically relevant, particularly because peripheral edema and other treatment-related adverse effects may influence long-term adherence.
Aim: To compare the safety and efficacy of cilnidipine versus amlodipine in newly diagnosed essential hypertensive patients.
Methodology: This prospective, randomized, open-label comparative clinical study was conducted in the Department of Pharmacology and Therapeutics in collaboration with the Department of Medicine, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, from September 2024 to September 2025. A total of 180 newly diagnosed Stage 1 or Stage 2 essential hypertensive patients were allocated in a 1:1 ratio by simple randomization. Ninety received cilnidipine 10 mg once daily and 90 received amlodipine 5 mg once daily. Clinical assessment was performed at baseline and at 1, 3, 6, 9 and 12 months. Blood pressure, heart rate, treatment compliance and adverse events were evaluated during follow-up.
Results: The treatment groups were comparable at baseline. Among participants completing 12-month follow-up, mean SBP reduction was −22.3 ± 10.1 mmHg with cilnidipine and −23.1 ± 9.8 mmHg with amlodipine (p=0.611), while DBP reduction was −14.3 ± 6.2 and −13.5 ± 6.0 mmHg, respectively (p=0.407). At 12 months, SBP was 130.4 ± 4.8 versus 130.1 ± 4.6 mmHg (p=0.686), while DBP was 81.5 ± 2.7 versus 82.8 ± 2.8 mmHg (p=0.003). Pedal edema occurred in 5.6% versus 16.7% (p=0.018). Overall adverse events occurred in 15.6% versus 26.7% (p=0.068), and drug-related events in 12.2% versus 23.3% (p=0.051). Heart-rate reduction was greater with cilnidipine (−5.9 ± 4.1 vs −0.8 ± 3.8 bpm; p<0.001).
Conclusion: Both agents provided substantial antihypertensive efficacy. Cilnidipine demonstrated a favourable tolerability and heart-rate profile, with a significantly lower frequency of pedal edema, while systolic blood-pressure reduction was similar between treatments.

Keywords: Cilnidipine, Amlodipine, Hypertension, Pedal Edema, Blood Pressure.

INTRODUCTION

Hypertension is among the most prevalent chronic non-communicable diseases and contributes substantially to cardiovascular, cerebrovascular and renal morbidity and mortality. Persistent elevation of arterial blood pressure promotes progressive vascular and target-organ injury, often remaining clinically silent until complications such as myocardial infarction, heart failure, stroke or chronic kidney disease become evident [1]. The global burden remains considerable, with inadequate awareness, treatment and control accounting for an important proportion of preventable cardiovascular disease [2]. India has experienced a progressive rise in hypertension in association with demographic transition, urbanization, changing dietary patterns, physical inactivity, obesity and increasing life expectancy. Epidemiological studies have

demonstrated a considerable prevalence in both urban and rural populations, establishing elevated blood pressure as a major non-communicable disease risk factor [3,4]. Essential hypertension constitutes the predominant form and arises through complex interactions among genetic susceptibility, ageing, excessive dietary sodium intake, obesity, insulin resistance, physical inactivity and sympathetic nervous system activation [5]. Appropriate lowering of arterial pressure is therefore central to prevention of long-term complications, and even moderate reductions in systolic and diastolic blood pressure can produce clinically meaningful cardiovascular benefit [6]. Pharmacological treatment becomes necessary when lifestyle modification alone fails to achieve adequate control. Among available antihypertensive classes, calcium-channel blockers have an established therapeutic role because of their potent vasodilatory activity, sustained efficacy and broad clinical applicability [7]. Amlodipine is a long-acting dihydropyridine that predominantly blocks L-type calcium channels and provides reliable once-daily blood-pressure reduction. Its widespread use is nevertheless accompanied by vasodilatory adverse effects, particularly peripheral edema, which may become troublesome during prolonged therapy. Cilnidipine is distinguished by combined L- and N-type calcium-channel inhibition. Vascular L-type blockade produces peripheral vasodilatation, whereas N-type inhibition at sympathetic nerve terminals attenuates norepinephrine release and sympathetic activity [8]. This dual pharmacological action may permit effective pressure reduction while limiting reflex sympathetic responses and excessive heart-rate elevation. Cilnidipine has therefore attracted interest as an alternative to conventional L-type calcium-channel blockade, particularly in patients susceptible to peripheral edema or heightened sympathetic activity [9]. Selection of long-term antihypertensive therapy should consider not only the magnitude of pressure reduction but also adverse-event burden, heart-rate response, compliance and treatment persistence. These factors assume particular importance in newly diagnosed patients, in whom the initial treatment choice may influence adherence over subsequent years. Direct comparative evaluation of cilnidipine and amlodipine therefore remains clinically relevant. The present study compared their safety and efficacy in newly diagnosed essential hypertensive patients attending a tertiary healthcare centre in Uttar Pradesh, with emphasis on blood-pressure reduction, adverse effects and the overall balance between efficacy and tolerability.

Aim

To compare the safety and efficacy of cilnidipine versus amlodipine in newly diagnosed essential hypertensive patients.

OBJECTIVES

1. To evaluate the reduction in blood pressure achieved with cilnidipine and amlodipine.
2. To assess the incidence and type of adverse effects associated with each drug.
3. To provide data that may inform treatment guidelines for hypertension.

MATERIALS AND METHODS

This prospective, randomized, open-label comparative clinical study was conducted in the Department of Pharmacology and Therapeutics in collaboration with the Department of Medicine, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, from September 2024 to September 2025. Newly diagnosed essential hypertensive patients attending the Medicine outpatient department constituted the study population. Hypertension was diagnosed on the basis of persistently elevated blood-pressure readings of ≥140/90 mmHg in accordance with JNC-8 criteria [10].

Sample size was estimated using the formula, with Z=1.96 for a 95% confidence interval, an anticipated hypertension prevalence of 11% and absolute precision of 5%. The calculated requirement was 151 participants; after allowance for anticipated attrition, 180 patients were enrolled.

Male and female patients aged >18 and <75 years with newly diagnosed Stage 1 or Stage 2 essential hypertension, attending the Medicine outpatient department and willing to provide written informed consent were eligible. Patients with secondary hypertension of renal, endocrine or other identifiable origin, pregnancy or lactation, known hypersensitivity to calcium-channel blockers, severe renal or hepatic dysfunction, previous antihypertensive treatment or unwillingness to consent were excluded.

After eligibility assessment and informed consent, demographic and clinical information including age, sex, body weight, family history of hypertension and relevant lifestyle characteristics was documented using a predesigned case-record form. Participants

were allocated in a 1:1 ratio by the simple randomization technique described in the study protocol. Ninety patients received cilnidipine 10 mg orally once daily in the morning and 90 received amlodipine 5 mg orally once daily. The available study records do not provide further information regarding the method of sequence generation or allocation concealment.

Clinical assessments were performed at baseline and at 1, 3, 6, 9 and 12 months. Blood pressure was measured in the seated position after at least 10 minutes of rest using a standard mercury sphygmomanometer by the auscultatory method. Three measurements were obtained from the right arm at one-minute intervals and their mean was recorded. Systolic blood pressure, diastolic blood pressure and pulse rate were monitored throughout follow-up.

Adverse events were actively assessed at every visit. Pedal edema was evaluated over the medial malleolus of both lower limbs, with edema involving either limb considered positive. Headache, dizziness, flushing, palpitations and other treatment-emergent symptoms were documented. Treatment compliance was assessed through patient self-report and pill count where feasible. The principal efficacy assessment was change in SBP and DBP, while safety assessment included the incidence and pattern of adverse drug reactions. Heart-rate response, treatment compliance and discontinuation contributed to the overall comparative evaluation.

A pilot study involving 10 patients was conducted before the main study to assess feasibility and standardize study procedures; these participants were excluded from the final analysis.

Continuous variables were expressed as mean ± standard deviation. Between-group continuous outcomes were evaluated using the independent-samples Student's t-test. Categorical variables were presented as frequencies and percentages and compared using Pearson's Chi-square test when expected frequencies were adequate; Fisher's exact test was used for sparse categorical outcomes. All tests were two-sided, and $p<0.05$ was considered statistically significant.

The 12-month change analysis included patients completing follow-up (cilnidipine n=82; amlodipine n=79). Safety outcomes were evaluated in the initially allocated populations (n=90 per group).

RESULTS

Table 1. Baseline Demographic and Clinical Profile of Study Participants

Characteristic	Cilnidipine (n=90)	Amlodipine (n=90)	p-value
Age (years), mean ± SD	52.4 ± 11.7	53.1 ± 10.9	0.678
Male	51 (56.7%)	50 (55.6%)	0.881
Female	39 (43.3%)	40 (44.4%)	—
BMI (kg/m²), mean ± SD	26.1 ± 3.8	25.8 ± 4.1	0.611
Baseline SBP (mmHg), mean ± SD	152.7 ± 12.4	153.2 ± 11.8	0.782
Baseline DBP (mmHg), mean ± SD	95.8 ± 7.2	96.3 ± 6.9	0.635
Baseline heart rate (bpm), mean ± SD	81.5 ± 8.3	80.9 ± 7.7	0.616
Stage 1 hypertension	56 (62.2%)	54 (60.0%)	0.760
Stage 2 hypertension	34 (37.8%)	36 (40.0%)	—
Type 2 diabetes mellitus	16 (17.8%)	17 (18.9%)	0.847
Dyslipidemia	21 (23.3%)	19 (21.1%)	0.720
Family history of hypertension	59 (65.6%)	61 (67.8%)	0.752
Current smoker	23 (25.6%)	21 (23.3%)	0.894*
Former smoker	14 (15.6%)	16 (17.8%)	—
Never smoked	53 (58.9%)	53 (58.9%)	—

*Overall comparison of smoking-status categories.

Table 2. Mean Reduction in Blood Pressure from Baseline to 12 Months among Participants Completing Follow-Up

Parameter	Cilnidipine (n=82), mean Δ ± SD	Amlodipine (n=79), mean Δ ± SD	p-value
Δ SBP (mmHg)	−22.3 ± 10.1	−23.1 ± 9.8	0.611

Δ DBP (mmHg)	−14.3 ± 6.2	−13.5 ± 6.0	0.407
Percentage reduction in SBP	14.6 ± 6.1%	15.1 ± 5.9%	0.598
Percentage reduction in DBP	14.9 ± 6.3%	14.0 ± 6.1%	0.358

Both treatment groups showed substantial reductions in SBP and DBP. None of the between-group differences in overall pressure reduction reached statistical significance.

Table 3. Incidence and Pattern of Adverse Events during Treatment

Adverse event	Cilnidipine (n=90)	Amlodipine (n=90)	p-value
Any adverse event	14 (15.6%)	24 (26.7%)	0.068
Drug-related adverse event	11 (12.2%)	21 (23.3%)	0.051
Pedal edema	5 (5.6%)	15 (16.7%)	0.018
Headache	4 (4.4%)	4 (4.4%)	1.000
Dizziness	3 (3.3%)	3 (3.3%)	1.000
Palpitations	2 (2.2%)	2 (2.2%)	1.000
Flushing	1 (1.1%)	3 (3.3%)	0.621†
Fatigue	2 (2.2%)	2 (2.2%)	1.000†
Gingival hyperplasia	0	1 (1.1%)	1.000†
Serious adverse event	1 (1.1%)	1 (1.1%)	1.000†

†Fisher's exact test used for sparse comparisons.

Pedal edema was significantly less frequent with cilnidipine. Overall and drug-related adverse events were numerically lower with cilnidipine but did not reach the conventional threshold for statistical significance.

Table 4. Comparative Summary of Major Efficacy and Safety Outcomes

Outcome	Cilnidipine	Amlodipine	p-value
Mean SBP at 12 months, mmHg (n=82 vs 79)	130.4 ± 4.8	130.1 ± 4.6	0.686
Mean DBP at 12 months, mmHg (n=82 vs 79)	81.5 ± 2.7	82.8 ± 2.8	0.003
BP control, n/N (%)	74/90 (82.2%)	71/90 (78.9%)	0.572
Pedal edema, n/N (%)	5/90 (5.6%)	15/90 (16.7%)	0.018
Drug-related AE, n/N (%)	11/90 (12.2%)	21/90 (23.3%)	0.051
AE-related discontinuation, n/N (%)	2/90 (2.2%)	6/90 (6.7%)	0.278†
Heart-rate reduction (bpm), mean ± SD	−5.9 ± 4.1	−0.8 ± 3.8	<0.001
Compliance (%), mean ± SD	94.2 ± 7.1	92.8 ± 8.3	0.226

†Fisher's exact test.

The BP-control value has been expressed as 74/90 (82.2%) because the thesis explicitly states that 74 cilnidipine-treated patients achieved control at 12 months.

A total of 180 patients were enrolled, with 90 participants assigned to each treatment arm. Mean age was 52.4 ± 11.7 years in the cilnidipine group and 53.1 ± 10.9 years in the amlodipine group (p=0.678). Men constituted 51 (56.7%) and 50 (55.6%), respectively. Mean BMI was 26.1 ± 3.8 versus 25.8 ± 4.1 kg/m² (p=0.611). Baseline SBP was 152.7 ± 12.4 versus 153.2 ± 11.8 mmHg (p=0.782), while DBP was 95.8 ± 7.2 versus 96.3 ± 6.9 mmHg (p=0.635). Heart rate was 81.5 ± 8.3 versus 80.9 ± 7.7 bpm (p=0.616). Stage 1 hypertension affected 62.2% and 60.0% of participants, respectively, whereas Stage 2 disease was present in 37.8% and 40.0%. No baseline characteristic differed significantly between the groups.

Among participants completing 12-month follow-up, SBP decreased by 22.3 ± 10.1 mmHg with cilnidipine and 23.1 ± 9.8 mmHg with amlodipine (p=0.611). Corresponding DBP reductions were 14.3 ± 6.2 and 13.5 ± 6.0 mmHg (p=0.407). Percentage changes in SBP and DBP were likewise similar.

Overall adverse events occurred in 14 (15.6%) versus 24 (26.7%) patients (p=0.068), and drug-related events in 11 (12.2%) versus 21 (23.3%) (p=0.051). Pedal edema was significantly less frequent with cilnidipine,

occurring in 5 (5.6%) patients compared with 15 (16.7%) receiving amlodipine (p=0.018).

At 12 months, mean SBP was 130.4 ± 4.8 versus 130.1 ± 4.6 mmHg (p=0.686), while DBP was 81.5 ± 2.7 versus 82.8 ± 2.8 mmHg (p=0.003). BP control was achieved in 74/90 (82.2%) versus 71/90 (78.9%) (p=0.572). Heart-rate reduction was greater with cilnidipine (−5.9 ± 4.1 vs −0.8 ± 3.8 bpm; p<0.001), whereas compliance remained high and similar between groups (94.2 ± 7.1% vs 92.8 ± 8.3%; p=0.226).

DISCUSSION

The present randomized comparative study assessed cilnidipine and amlodipine in newly diagnosed essential hypertension, with particular attention to antihypertensive efficacy and treatment safety. Both regimens produced substantial reductions in arterial pressure during follow-up. The principal distinction was not the magnitude of systolic blood-pressure lowering but rather selected aspects of tolerability and heart-rate response, particularly the lower occurrence of pedal edema among patients receiving cilnidipine.

The baseline demographic and clinical characteristics were well balanced between groups. Mean age was 52.4 ± 11.7 years with cilnidipine and 53.1 ± 10.9 years with amlodipine, while men represented 56.7% and 55.6% of the respective cohorts. Baseline SBP and DBP were also closely matched at 152.7 ± 12.4/95.8 ± 7.2 mmHg and 153.2 ± 11.8/96.3 ± 6.9 mmHg. Adake et al. [11] evaluated a similarly middle-aged hypertensive cohort with a modest male predominance. Kumar et al. [12] reported baseline systolic pressure close to that observed in the present population. Kabir et al. [13] likewise described a predominantly male hypertensive cohort with baseline SBP around 151 mmHg, while Shetty et al. [14] documented pretreatment systolic and diastolic values of approximately 150 and 94 mmHg. The demographic and haemodynamic profiles were therefore broadly similar to those reported in previous comparative studies and provided a balanced foundation for evaluation of treatment outcomes.

The primary efficacy analysis demonstrated meaningful pressure reduction with both agents. Mean SBP decreased by 22.3 ± 10.1 mmHg with cilnidipine and 23.1 ± 9.8 mmHg with amlodipine, without a material between-group difference. DBP decreased by 14.3 ± 6.2 and 13.5 ± 6.0 mmHg, respectively. Takei et al. [15] observed systolic reductions of approximately 21–23 mmHg with the two drugs, closely resembling the magnitude seen here. Dharapur and Patil [16] also documented substantial decreases in both systolic and diastolic pressure with either regimen. Chakraborty et al. [17], in a meta-analysis of prospective studies, found cilnidipine to provide effective pressure reduction of a magnitude similar to other calcium-channel blockers. Hoshide et al. [18] further demonstrated sustained ambulatory BP lowering with both cilnidipine and amlodipine. Collectively, these studies support a similar overall antihypertensive response with the two agents.

The safety assessment revealed a more distinct difference. Any adverse event occurred in 15.6% of cilnidipine-treated patients and 26.7% receiving amlodipine. Drug-related events affected 12.2% and 23.3%, respectively. Although these overall comparisons did not reach conventional statistical significance after recalculation, pedal edema occurred significantly less frequently with cilnidipine (5.6% vs 16.7%; p=0.018). Other adverse effects were uncommon and showed no meaningful between-group differences.

Dalal et al. [19] documented pedal edema in approximately 6.3% of patients treated with cilnidipine and 17.8% of those receiving amlodipine, closely paralleling the present pattern. Kafle et al. [20] observed improvement in amlodipine-associated edema after substitution with cilnidipine while maintaining satisfactory pressure control. Xu et al. [21], in a systematic review and meta-analysis, reported effective antihypertensive activity with a favourable tolerability profile for cilnidipine. Shetty et al. [22] similarly observed resolution of amlodipine-associated ankle edema after patients were changed to cilnidipine. Collectively, this evidence identifies the lower frequency of peripheral edema as a potentially important clinical advantage of cilnidipine.

The integrated 12-month assessment provided further distinction between the treatments. Mean SBP was virtually identical at 130.4 ± 4.8 mmHg with cilnidipine and 130.1 ± 4.6 mmHg with amlodipine, whereas DBP was modestly lower with cilnidipine (81.5 ± 2.7 vs 82.8 ± 2.8 mmHg; p=0.003). BP-control rates were 82.2% and 78.9%, respectively, without a statistically significant difference. Thus, overall pressure control remained similar despite differences in selected secondary outcomes.

Heart-rate response was more pronounced with cilnidipine. Mean heart rate decreased by 5.9 ± 4.1 bpm, compared with only 0.8 ± 3.8 bpm

with amlodipine. This observation is pharmacologically compatible with cilnidipine's additional N-type calcium-channel inhibition, which reduces sympathetic neurotransmitter release. The greater decline in heart rate therefore occurred without loss of antihypertensive efficacy.

Harlalka et al. [23] likewise reported effective pressure reduction with both agents while describing favourable heart-rate and edema outcomes with cilnidipine. NK et al. [24] observed satisfactory efficacy with either drug but fewer troublesome adverse effects among patients receiving cilnidipine. Osterloh [25] identified peripheral edema as an important component of the established amlodipine safety profile. Jadhav et al. [26] demonstrated sustained blood-pressure control with dihydropyridine calcium-channel blockers while emphasizing the importance of considering drug-specific tolerability in long-term therapy. These comparisons reinforce the similar overall BP control achieved with both treatments while highlighting clinically relevant differences in edema and autonomic response.

When efficacy and safety are considered together, the principal distinction between the two agents lies less in systolic BP reduction than in their broader therapeutic profiles. Both produced substantial pressure lowering and similar overall control rates. Cilnidipine was associated with less pedal edema and a greater decrease in heart rate, while treatment compliance remained high with either regimen. Its dual L- and N-type calcium-channel blockade provides a plausible pharmacological basis for attenuation of sympathetic activity without compromising antihypertensive effectiveness.

These differences are relevant in newly diagnosed hypertension because therapy is generally continued for prolonged periods, and even non-serious adverse effects may influence adherence or prompt treatment modification. Peripheral edema can be sufficiently troublesome to result in additional consultations, dosage alteration or substitution of therapy. An agent capable of sustaining effective pressure control with a lower burden of such symptoms may therefore have practical value in appropriately selected patients.

The present results support both amlodipine and cilnidipine as effective antihypertensive options. Amlodipine retained robust blood-pressure-lowering efficacy, whereas cilnidipine provided similar overall systolic control with a lower frequency of pedal edema and a more pronounced heart-rate response. Treatment selection should therefore remain individualized according to haemodynamic response, adverse-effect susceptibility, sympathetic activity and the importance of maintaining long-term adherence.

CONCLUSION

Both cilnidipine and amlodipine produced substantial antihypertensive responses in newly diagnosed essential hypertension. Overall systolic blood-pressure reduction and BP-control rates were similar. Cilnidipine was associated with significantly less pedal edema and a greater reduction in heart rate, while overall treatment compliance remained high with both regimens. Cilnidipine therefore represents an effective alternative when blood-pressure control must be balanced against peripheral edema and sympathetic cardiovascular responses.

Limitations

The single-centre, open-label design may limit generalizability and introduce reporting bias. The sample size was insufficient to evaluate uncommon adverse reactions, and 12 months of follow-up did not allow assessment of longer-term cardiovascular, renal or mortality outcomes. The available source also lacked detailed reporting of random-sequence generation and allocation concealment.

Recommendations

Both agents remain effective options for newly diagnosed essential hypertension. Cilnidipine may be particularly useful when peripheral edema or increased sympathetic activity is clinically relevant. Regular assessment of blood pressure, heart rate, compliance and adverse effects is advisable. Larger multicentric, double-blind randomized studies with longer follow-up are required to define comparative long-term cardiovascular outcomes.

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