REVIEW ARTICLE

Consensus Statements from the Nephrologists Alliance on Calcium Channel Blockers with Special Emphasis on Cilnidipine and Its Role in the Management of Hypertension in Chronic Kidney Disease

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Abstract

Hypertension is a prevalent health issue that significantly elevates the risks of cardiovascular and renal complications and results in 12.8% deaths worldwide. A steering group of three Indian nephrologists reviewed the relevance of existing literature related to calcium channel blockers (CCBs) with specific emphasis on a fourth-generation CCB, cilnidipine, in managing hypertension and chronic kidney disease (CKD). To provide recommendations for successfully controlling hypertension in CKD patients, a panel of 46 eminent Indian nephrologists was assembled for a discussion. The panelists discussed the literature evidence and provided their valuable insights on the use of cilnidipine and other CCBs in achieving blood pressure (BP) targets and improving cardiovascular events and kidney functions in regular clinical practices. The discussion areas included (i) antihypertensive effect of CCBs in patients with hypertension and CKD (9 statements) and (ii) renoprotective effect of CCBs in hypertensive CKD patients (5 statements) to vote on the total 14 nine-point Likert scale statements. The topics included standard BP targets, choice of drug classes, use of monotherapy/combination therapy based on individual risk factors, and the cardioprotective and renoprotective roles of various types of CCBs. The cardioprotective and renoprotective effects of L-/N-type CCB cilnidipine were extensively discussed. The consensus recommendations that have been developed could provide guidance in a real-world setting while prescribing antihypertensive therapy to patients with CKD, with or without diabetes. In conclusion, different types of CCBs should be used with proper knowledge of their pharmacological, antihypertensive, and organ-protective effects, tailored to the patient's pathophysiology.

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Introduction

In recent times, hypertension has become a major concern for public health, owing to the increasing incidence and the resultant 12.8% death rate worldwide.[1] Hypertension prevalence in India has increased from 11.3% in 2015–2016 to the current prevalence of 22.6%.[2] The rising burden of hypertension, coupled with limited screening and awareness are attributable to 13.24% prevalence of chronic kidney disease (CKD) in India.[3] Hypertension, being a modifiable risk factor for cardiovascular disease (CVD), can be managed to prevent the inevitable adverse outcomes, especially in CKD patients.[4]

Blood pressure (BP) monitoring at all CKD stages is recommended for slowing the progression of end-stage kidney disease (ESKD).[4] The 2024 Kidney Disease Improving Global Outcomes (KDIGO) guideline suggested setting the target systolic BP (SBP) to <120 mm Hg in adults with high BP and CKD.[5] The 2025 Indian Society of Hypertension (InSH) guideline recommended BP goal for most CKD patients as <130/80 mm Hg; however, SBP <120 mm Hg could be targeted if well-tolerated.[6] The 2025 American College of Cardiology/American Heart Association (ACC/AHA) guideline recommended <130/80 mm Hg in CKD patients, while the 2023 European Society of Hypertension (ESH) guideline and 2016 Ministry of Health and Family Welfare, Government of India, suggested <140/90 mm Hg BP goal.[4,7,8] A target of <130/80 mm Hg may be aimed in patients with urinary albumin/creatinine ratio (UACR) ≥300 mg/g, high cardiovascular (CV) risk, or kidney transplant.[4]

Combination therapy with two or more antihypertensives, usually a renin–angiotensin system (RAS) blocker plus a calcium channel blocker (CCB) or thiazide/thiazide-like diuretic, is standard for reaching target BP in patients whose estimated glomerular filtration rate (eGFR) is ≥45 mL/min/1.73 m^2.[4] The 2023 ESH and 2024 KDIGO guidelines recommended an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) for CKD patients with moderate (UACR 30–300 mg/g) or severe (UACR >300 mg/g) albuminuria.[4,5] A dihydropyridine (DHP)-CCB or an ARB is recommended as the first-line treatment for adult kidney transplant recipients including elderly and diabetic patients by the 2021 KDIGO guideline.[9] A DHP-CCB could be considered as an alternative to ACEi or ARB for achieving BP target in the absence of albuminuria.[5] The traditional DHP-CCBs (amlodipine and nifedipine) block voltage-dependent L-type calcium channels, while the novel CCBs mostly block L-/N-type (cilnidipine), L-/T-type (azelnidipine, efonidipine and mibefradil), and L-/T-/N-type (benidipine) channels.[10] Besides BP management, these CCBs overall exert organ-protective actions, induce vasodilation, and reduce the development of congestive heart failure, angina, and renal complications.[11,12] Recently, the renoprotective effects of cilnidipine have garnered more attention.[11] However, disparity exists related to the CCB use in individuals with CKD, due to limited evidence on the side effects and uncertainty in their effect on predialysis SBP, diastolic BP (DBP), and CV death.[13] In order to limit this disagreement on management of hypertension-associated complications and highlight the current practices, a group of eminent Indian nephrologists discussed and provided valuable insights on the cardioprotective and renoprotective effects of CCBs, particularly cilnidipine (monotherapy/combination therapy) in CKD patients to reach an appropriate consensus.

Materials and Methods

Google Scholar and PubMed databases were searched for research and review articles, and clinical guidelines related to the role of CCBs in managing hypertension and CKD, with particular attention to the cardioprotective and renoprotective benefits of cilnidipine. Combinations of the keywords "albuminuria", "angiotensin-converting enzyme inhibitor", "angiotensin receptor blocker", "blood pressure", "calcium channel blockers", "cardiovascular disease", "chronic kidney disease", "cilnidipine", "creatinine", "hypertension" and "proteinuria" were used. A steering group comprising three senior members (nephrologists) has screened and reviewed the existing literature and formulated a total of 14 statements pertaining to the relevant evidence.

Forty-six eminent nephrologists across India were invited to participate in the meeting based on their clinical experience in hypertension management in patients with CKD. Three nephrologists of the steering group presented the evidence on "Antihypertensive effect of CCBs in patients with hypertension and CKD" and "Renoprotective effect of CCBs in patients with hypertension and CKD." With reference to the guidelines and studies presented, the assembled panelists discussed and added their experience of regular practice. The 14 statements were discussed and voted on by the panelists on a nine-point Likert scale (1–4: degrees of disagreement, 5: neutral opinion, 6–9: increasing degree of agreement). Score 1 on the Likert scale was considered as strong disagreement, and 9 as strong agreement. The percentage of agreement and median Likert scale score were analyzed using the SPSS software (SPSS, V 28.0, USA).

Results

A total of 14 consensus statements (Table 1) were discussed in the meeting. The level of agreement on each statement via consensus voting has been provided in Figure 1. The statements are discussed with rationale in the following section.

Discussion

Part I: Antihypertensive Effect of CCBs in Patients with Hypertension and CKD

Statement 1: To maximize treatment benefits, target BP achievements should take precedence over choosing specific antihypertensive medication.

A median Likert score of 8 and 80.0% agreement was recorded for the statement. Although comorbidities like proteinuria may influence drug selection, the panelists pointed out that achieving BP target should be the primary objective for getting maximum benefits on renal and CV systems. The 2025 InSH guidelines recommended BP-lowering precedence over choosing among drug classes to maximize the treatment benefits.[6] A study has shown that prioritizing BP targets helped in lowering CV events and all-cause death.[14]

Another consideration of the panel was that usually BP is inadequately controlled with ACEis or ARBs; hence, a CCB should be

Table 1: Consensus statements

Discussion area	*No.*	*Statement*	*Neutral (%)*	*Agree (%)*	*Strongly agree (%)*	*Disagree (%)*	*Strongly disagree (%)*	*Median Likert scale score*
Antihypertensive effect of CCBs in patients with hypertension and CKD	1	To maximize treatment benefits, target BP achievements should take precedence over choosing specific antihypertensive medication	13.3	80.0	33.3	6.7	4.4	8
	2	Patients with stage 1 hypertension (BP 130–139/80–89 mm Hg) without proteinuria can be initiated with monotherapy (CCBs, ACEis, thiazide-like diuretics) in background of lifestyle modification	4.4	93.3	42.2	2.2	0	8
	3	In patients with stage 2 hypertension (BP ≥140/90 mm Hg), preferably a dual drug combination should be initiated as the first-line treatment according to the patient's individual risk factors	4.5	95.5	40.9	0	0	8
	4	Hypertension patients with CKD can be treated with a target SBP of <120 mm Hg, when tolerated	2.2	95.6	40.0	2.2	0	8
	5	Cilnidipine effectively reduces 10–15 mm Hg SBP in patients with hypertension and CKD	0	93.3	26.7	6.7	4.4	8
	6	Cilnidipine reduces nocturnal BP, improves the dipping status, and has beneficial effects on morning and nocturnal hypertension	4.4	95.6	24.4	0	0	8
	7	Cilnidipine does not cause reflex tachycardia (improves baroreflex sensitivity, decreases sympathetic activity)	4.4	93.3	33.3	2.2	2.2	8
	8	Cilnidipine has been shown to offer smooth and sustained 24-hour BP control (home BP monitoring, clinic BP monitoring, and ambulatory BP monitoring)	9.1	88.6	11.4	2.3	0	8
	9	Cilnidipine, along with telmisartan, is a preferred choice of combination therapy in patients with hypertension and diabetic nephropathy and other proteinuric nephropathies	0	100.0	48.8	0	0	8
Renoprotective effect of CCBs in patients with hypertension and CKD	10	A combination of an RAS blocker with a CCB or a diuretic is recommended as initial therapy for the treatment of hypertension in CKD	0	97.7	38.6	2.3	0	8
	11	L-/N-type CCBs combined with RAS blockers provide a greater reduction in proteinuria and improvement in kidney function	0	97.7	45.5	2.3	2.3	8
	12	For reducing urine albumin/protein excretion in hypertensive patients with CKD, L-/N-type CCB is more effective than L-type CCB. L-/N-type CCB act by decreasing the intraglomerular pressure, dilating the afferent and efferent arterioles and provide reno-protective benefits beyond blood pressure control	6.8	93.2	50.0	0	0	8.5
	13	Cilnidipine provides renoprotection because it has antioxidative properties, inhibits renal RAAS, and suppresses podocyte injury	17.8	73.3	20.0	8.9	4.4	7
	14	Cilnidipine is one of the preferred CCBs for managing hypertension in patients with T2D for renoprotection	11.1	64.4	17.8	24.4	13.3	7

ACEi, angiotensin-converting enzyme inhibitor; BP, blood pressure; CCB, calcium channel blocker; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; RAAS, renin–angiotensin–aldosterone system; RAS, renin–angiotensin system; SBP, systolic blood pressure; T2D, type 2 diabetes; UPCR, urinary protein-to-creatinine ratio. A score >6 on the 9-point Likert scale was interpreted as indicating a high degree of agreement with the statement

Statement	Agree (%)
Cilnidipine, along with telmisartan, is a preferred choice of combination therapy in patients with hypertension and diabetic nephropathy and other proteinuric nephropathies.	100.0
A combination of renin-angiotensin system blocker with a CCB or a diuretic is recommended as initial therapy for treatment of hypertension in CKD	97.7
L-/N-type CCBs combined with renin-angiotensin system blockers provide greater reduction in proteinuria and improvement in kidney function.	97.7
Hypertension patients with CKD can be treated with a target systolic BP of <120 mm Hg, when tolerated	95.6
Cilnidipine reduces nocturnal BP, improves the dipping status and has beneficial effects on morning and nocturnal hypertension.	95.6
In patients with stage 2 hypertension (BP≥140/90 mm Hg), preferably a dual drug combination should be initiated as the first-line treatment according to the patient's individual risk factors.	95.5
Patients with stage 1 hypertension (BP 130–139/80–89 mm Hg) without proteinuria can be initiated with monotherapy (CCBs, angiotensin-converting enzyme inhibitors, thiazide-like diuretics) in background of lifestyle modification.	93.3
Cilnidipine effectively reduces 10–15 mm Hg SBP in patients with hypertension and CKD.	93.3
Cilnidipine does not cause reflex tachycardia (improves baroreflex sensitivity, decreases sympathetic activity).	93.3
For reducing urine albumin/protein excretion in hypertensive patients with CKD, L-/N-type CCB is more effective than L-type CCB. L-/N-type CCB act by decreasing the intraglomerular pressure, dilating the afferent and efferent arterioles and provide reno-protective benefits beyond blood pressure control.	93.2
Cilnidipine has been shown to offer smooth and sustained 24-hour BP control (home BP monitoring, clinic BP monitoring and ambulatory BP monitoring)	88.6
To maximize treatment benefits, target BP achievements should take precedence over choosing specific antihypertensive medication.	80.0
Cilnidipine provides renoprotection because it has antioxidative properties, inhibits renal renin-angiotensin-aldosterone system and suppresses podocyte injury	73.3
Cilnidipine is one of the preferred CCBs for managing hypertension in patients with type 2 diabetes for renoprotection.	64.4

Fig. 1: Representation of panel agreement on the statements (BP, blood pressure; CCB, calcium channel blocker; CKD, chronic kidney disease); SBP, systolic BP

introduced for BP reduction rather than its proteinuric effects. The 2021 World Health Organization (WHO), 2022 ACC/AHA/ESH and 2023 ESH guidelines recommended an ACEi or ARB with CCB or diuretic for hypertension management in patients with impaired kidney functions.[4,15,16] The ACCOMPLISH trial found superiority of the ACEi-DHP-CCB combination to the ACEi-thiazide diuretic combination for reducing CV events in hypertensive patients.[17]

- Achieving the BP goal should be prioritized over selecting specific antihypertensive medication.
- In patients unresponsive to ACEi or ARB monotherapy, the addition of CCB should be considered.

Statement 2: Patients with stage 1 hypertension (BP 130–139/80–89 mm Hg) without proteinuria can be initiated with monotherapy (CCBs, ACEis, thiazide-like diuretics) in the background of lifestyle modification.

Lifestyle modifications (low-sodium diet, increased vegetable and fruit intake, weight reduction, abstention from alcoholism, and smoking cessation) enhance the effects of antihypertensive agents and reduce CV risks.[6,18] The 2025 InSH guidelines suggested monotherapy either with a CCB, ACEi, or thiazide-like diuretic along with lifestyle modification for low-risk stage 1 hypertension patients (BP 130–139/80–89 mm Hg). Because of antiproteinuric effects, ACEi/ARB is preferred in patients with proteinuria or microalbuminuria; thiazide/ thiazide-like diuretic is preferred when additional BP control is required, and loop diuretic in cases of volume overload.[6] The 2025 ACC/AHA guideline suggested ACEi or ARB for managing hypertension in patients with CKD to decrease CVD and slow kidney disease progression.[8] The 2019 Japanese Society of Hypertension (JSH) guideline recommended RAS blockers, CCBs, or thiazide-type diuretics for controlling BP in the absence of proteinuria.[19] A meta-analysis supported the beneficial effect of CCB or other monotherapy in managing hypertension and preventing CV complications.[20] After reviewing available evidence followed by the discussion, the panel agreed upon this statement (93.3%, median score 8) of using monotherapy with lifestyle modifications in stage 1 hypertension. The panel also suggested considering the age while recommending antihypertensives, as older patients (>50 years) have a superior response to CCBs and diuretics than ACEis and ARBs.[6]

- Monotherapy may be started in stage 1 hypertensive patients.
- Age should be a determinant while recommending antihypertensive drugs.

Statement 3: In patients with stage 2 hypertension (BP ≥140/90 mm Hg), preferably a dual drug combination should be initiated as the first-line treatment according to the patient's individual risk factors.

In stage 2 hypertension patients, the 2025 ACC/AHA guideline preferred initiating antihypertensive therapy with a single-pill combination of two drug classes to improve the BP control and adherence.[8] Consistent with the guideline recommendations,[4,6,7,21] the panel majorly agreed (95.5%, median score: 8) upon using dual therapy with an ACEi or ARB in combination with a CCB or thiazide-like diuretic to obtain a desirable effect on the individual risk factors. This perspective is in agreement with the OSCAR study that reported reduced BP and CV complications with ARB-CCB combination in patients with hypertension and CKD.[22] The TACTICAL2 study

has shown that hypertensive patients on ARB with cilnidipine (L-/N-type CCB) had better renoprotection due to a reduction in urinary albumin excretion than those on amlodipine (L-type CCB).[23] Dual therapy (ACEi + CCB) in stage 2 hypertension results in 27% higher BP control, compared to ACEi, ARB, CCB, diuretic, or beta-blocker monotherapy.[24] The panel thus endorsed the guideline-recommended BP-lowering pharmacotherapy.

- A combination of an RAS blocker and a CCB or a diuretic is preferable for stage 2 hypertensives with comorbidities.

Statement 4: Hypertension patients with CKD can be treated with a target SBP of <120 mm Hg, when tolerated.

The KDIGO guidelines recommended a target BP of <120 mm Hg in adults with high BP and CKD.[5,9] The SPRINT study also supported the SBP target of <120 mm Hg in patients with high CV risk but without diabetes for reducing CV events and death.[25] The 2021 National Institute for Health and Care Excellence (NICE) guideline recommended proteinuria-dependent BP targets: 120–139/<90 mm Hg for UACR <70 mg/mmol and 120–129/<80 mm Hg for UACR ≥70 mg/mmol.[26] The same BP target does not apply to kidney transplant recipients or dialysis patients.[9,27] According to the 2024 ESC guidelines, the target SBP must be 120–129 mm Hg in patients with CKD (eGFR over 30 mL/min/1.73 m^2).[27] Well-tolerated treatment is recommended for achieving BP target in young adults with high UACR (>300 mg/g) and CV risk,[6] as well as in elderly patients (target <140/<90 for below 80 years and <150/<90 mm Hg in more than 80 years).[7] Although the majority agreed upon the statement (95.6%) with a median score of 8, the InSH consensus guideline (2025) suggested avoiding BP target excessively <120/70 mm Hg in CKD patients as it may affect the renal perfusion.[6]

- Many guidelines suggest that therapy should be individualized.

Statement 5: Cilnidipine effectively reduces 10–15 mm Hg SBP in patients with hypertension and CKD.

The 2024 KDIGO guideline recommended ACEi or ARB as the first-line treatment for maintaining BP in patients with albuminuria, or else, in addition to RAS blockers, a DHP-CCB or diuretic can be considered to attain BP targets.[5] The 2019 JSH guideline suggested RAS blockers, CCBs, or thiazide-type diuretics if proteinuria is absent.[19] The panel reviewed studies on the efficacy of L-/N-type CCB cilnidipine for reducing BP in CKD. A meta-analysis recommended cilnidipine monotherapy/combination therapy as the novel first-line CCB for reducing BP and pulse rate (PR), based on the findings of 24 randomized and nonrandomized clinical trials.[11] L-type CCB amlodipine lowers BP similarly to cilnidipine, but results in increased PR.[28] A fall in clinical, morning, and evening SBP by 19.6, 17.0, and 13.6 mm Hg, respectively, after 12 weeks of cilnidipine therapy, as reported by the ACHIEVE-ONE study,[29] further proved the efficacy of cilnidipine, resulting in 93.3% agreement (median score: 8).

- Cilnidipine as monotherapy or combination therapy effectively reduces BP.

Statement 6: Cilnidipine reduces nocturnal BP, improves the dipping status, and has beneficial effects on morning and nocturnal hypertension.

The statement is supported by the 2022 Research Society for the Study of Diabetes in India (RSSDI) guideline, according to which cilnidipine offers antihypertensive and CV benefits due to unique sympatholytic activity and N-type calcium channel blockade.[30] Cilnidipine suppresses high sympathetic activity through blocking the neuronal N-type channels,[29] therefore, lessens the morning and office BPs as well as PR and the hyperactive sympathetic nerve-caused white-coat effect in patients with morning hypertension.[31] Ambulatory BP monitoring (ABPM) after 24-week treatment with cilnidipine showed significantly lowered daytime SBP in hypertensive patients with CKD, compared to amlodipine, azelnidipine, benidipine, manidipine, or nifedipine treatment.[32] Cilnidipine reduces the morning SBP, especially in patients with higher morning BP and PR than the patients with lower PR (<70 bpm) (ACHIEVE-ONE study).[29] Further analysis of the ACHIEVE-ONE study indicated that cilnidipine restores abnormal nocturnal dipping patterns (dippers, extreme dippers, nondippers, or risers) to a normal dipping pattern.[33] The HOPE-Combi survey demonstrated the sympatholytic effects of cilnidipine combined with ARB (valsartan) in effectively controlling morning home systolic BP, pulse pressure, and pulse rate over 12 months, particularly in uncontrolled hypertension patients with sympathetic hyperactivity.[34] Majority of the panelists (95.6%) agreed with a median score of 8, and additionally pointed the necessity of ABPM in CKD patients. The same has been recommended by the 2021 KDIGO guideline.[9]

- Cilnidipine lessens the hyperactive sympathetic nerve-caused white-coat effect and manages morning and nocturnal BP.
- ABPM is recommended for complementing standardized office BP and managing high BP.

Statement 7: Cilnidipine does not cause reflex tachycardia (improves baroreflex sensitivity, decreases sympathetic activity).

The 2025 InSH consensus guideline recommended cilnidipine for its lower tendency to cause edema and tachycardia.[6] The 2019 JSH guideline stated that cilnidipine may diminish sympathetic nerve activity by suppressing the release of noradrenaline from sympathetic nerve terminals.[19] This property of cilnidipine, along with partial suppression of pathological cardiac remodeling, is responsible for a decrease in nighttime PR in patients with kidney disease. Nighttime PR was reported to be increased in these patients after 24 weeks of treatment with amlodipine, nifedipine, azelnidipine, benidipine, or manidipine.[32] In patients with unmanageable hypertension and sympathetic hyperactivity, cilnidipine significantly reduces morning home PR.[34] A similar reduction in PR was noted by some other studies.[35,36] 93.3% of panelists agreed that they have observed no reflex tachycardia with cilnidipine (median score: 8).

- Cilnidipine reduces BP without causing reflex tachycardia.

Statement 8: Cilnidipine has been shown to offer smooth and sustained 24-hour BP control (home BP monitoring, clinic BP monitoring, and ABPM).

Better safety and tolerability, as well as cardioprotection and renoprotection with cilnidipine, make it preferable over other CCBs.[30] Cilnidipine was reported to significantly reduce 24-hour BP after 24 weeks of treatment compared to the other CCBs such as amlodipine, azelnidipine, benidipine, manidipine, and nifedipine.[32] Although 88.6% panelists agreed upon the statement (median score: 8), some had differences in opinion. They discussed that cilnidipine has to be prescribed twice daily in clinical practice for sustained 24-hour BP control, owing to its short duration of action. Indeed, the START ABPM study concluded that, particularly during the 18- to 24-hour postdose period or critical early morning hours, ARB (telmisartan 40 mg once-daily) is more effective in providing sustained 24-hour BP control over 10 or 20 mg once-daily cilnidipine.[37]

The panelists also added that the equivalence ratio of cilnidipine is approximately half that of amlodipine, and switching from 5 mg amlodipine requires 10 mg cilnidipine. A randomized study reported a decrease in 24-hour BP with once-daily cilnidipine and amlodipine, but a greater reduction in 24-hour PR with cilnidipine. The study increased the cilnidipine dose from 10 to 20 mg when BP remained high (≥140/90 mm Hg), while amlodipine was increased from an initial 2.5 mg to an additional 2.5 mg.[35] The panel suggested modifying cilnidipine dose when required in a real-world setting for sustained 24-hour BP control.

- Dose adjustment of cilnidipine is essential for controlling 24-hour BP.
- Safety, tolerability, cardioprotective, and renoprotective effects of cilnidipine make it preferable over other CCBs.

Statement 9: Cilnidipine, along with telmisartan, is a preferred choice of combination therapy in patients with hypertension and diabetic nephropathy and other proteinuric nephropathies.

A cross-sectional questionnaire-based survey highlighted a high (73%) preference for using cilnidipine-telmisartan combination among Indian clinicians in managing BP (63.2%), renal comorbidities (91%), and diabetic hypertensive patients (76%).[38] An Indian randomized trial on stage 2 hypertensive patients reported a difference in SBP and DBP by −0.48 and −0.77 mm Hg, respectively, with cilnidipine + telmisartan vs efonidipine + telmisartan treatment.[39] Another trial recorded similar effects of cilnidipine and benidipine as combination therapy with an ARB (telmisartan/olmesartan) in reducing BP and proteinuria in patients with hypertension and CKD.[40] Calcium channel blocker, particularly cilnidipine in combination with ARB, is preferred by the RSSDI (2022) for managing hypertension in diabetic patients.[30] For patients with hypertension and CKD, both 2021 KDIGO and 2020 Canada's hypertension guidelines recommended initial therapy with an ACEi or ARB.[9,41] The panel also encouraged the first-line choice of pharmacotherapy with an ACEi or ARB in CKD patients exhibiting very high albuminuria. All the panelists agreed (median score: 8) on the efficacy of cilnidipine along with an ARB in patients with hypertension and kidney disease. Indeed, cilnidipine and its combination presented favorable tolerability and suitability in Indian settings.[42]

- Treatment with ACEi/ARB should be the first-line therapy in patients with CKD and high albuminuria.
- Cilnidipine-telmisartan combination for hypertension with renal impairment can be prescribed to control BP.

Part II: Renoprotective Effect of CCB in Patients with Hypertension and CKD

Statement 10: A combination of RAS blocker with a CCB or a diuretic is recommended as initial therapy for treatment of hypertension in CKD.

A large physiological model in a virtual population demonstrated the potential risk of CCB monotherapy in African-American CKD patients and suggested combining CCB therapy with an RAS blocker for CKD treatment.[43] A two-drug combination, preferably a CCB or thiazide/ thiazide-like diuretic with an RAS blocker, is recommended in hypertensive patients.[4,6,15,16,21] An Indian survey evaluated the clinician's preferences and found that the combination of cilnidipine and ARB (91.33%) is majorly used in managing hypertension in patients with both kidney disease and diabetes, followed by amlodipine (7.2%), benidipine (0.8%) and nifedipine (0.67%).[38] A nationwide cohort study from Sweden reported better kidney outcomes with diuretics when given on top of an RAS blocker than CCB in patients with moderate-to-advanced CKD; however, cardioprotection was similar with both combinations.[44] The panel, with 97.7% agreement (median score 8), supported initial therapy with RAS blocker in combination with a CCB or a diuretic according to the guidelines. However, patient-specific factors (e.g., age, electrolytes) should be considered before initiating the therapy.

- Initial therapy with an RAS blocker, combined with CCB or diuretic, must be used in hypertensives with CKD, only after considering patient-specific factors.

Statement 11: L-/N-type CCBs combined with RAS blockers provide greater reduction in proteinuria and improvement in kidney function.

A meta-analysis assessing the efficacy of L-/N-type (cilnidipine) and L/T-type (azelnidipine, efonidipine or benidipine) CCBs combined with RAS blocker, reported significantly reduced albuminuria, proteinuria and serum creatinine, and improved kidney function in patients with hypertension and proteinuria.[12] Cilnidipine as add on to ARB exerts similar antiproteinuric effect as benidipine in reducing urinary protein excretion in hypertensive patients with CKD.[40] Cilnidipine also resulted in higher reduction in SBP and spot urinary protein/creatinine ratio (UPCR) than efonidipine in patients with CKD, when administered with RAS blocker.[45] The CARTER study exhibited a greater decrease in UPCR with cilnidipine + RAS blocker than amlodipine + RAS blocker in hypertensive patients with CKD.[46]

Expert perspectives and practices in Indian settings indicated a high preference for the cilnidipine-ARB combination because of its beneficial effects on BP and renal safety profile.[38] On the basis of the available evidences as well as personal observations, 97.7% (median score: 8) panelists agreed with the statement.

- Combination therapy of cilnidipine with an RAS blocker exerts favorable effect in reducing BP and proteinuria.

Statement 12: For reducing urine albumin/protein excretion in hypertensive patients with CKD, L-/N-type CCB is more effective than L-type CCB. L-/N-type CCB act by decreasing the intraglomerular pressure, dilating the afferent and efferent arterioles and provide reno-protective benefits beyond blood pressure control.

L-/N-type CCB cilnidipine and L-/T-/N-type CCB benidipine have comparable effects in decreasing urinary protein excretion in hypertensive patients with CKD.[40] Cilnidipine is superior to L-type CCBs in providing kidney protection through amelioration of glomerular microcirculation,[47] and significantly decreased proteinuria[48] and urine albumin excretion.[49] The reason for the decrease in UPCR with cilnidipine is the dual blockage of L-/N-type calcium channel-led afferent and efferent arteriole dilations. This, in turn, reduced the glomerular pressure and diabetic nephropathy progression in patients with diabetic kidney disease.[50] The mechanism of reducing intraglomerular pressure and improving the kidney function with cilnidipine[47,48,50] is presented in Figure 2.

Switch from amlodipine to cilnidipine resulted in reduced UACR and uric acid excretion in patients with hypertension and CKD, as evident in the J-CIRCLE study.[51] On the other hand, switch from cilnidipine to amlodipine significantly increased urinary albumin excretion in patients with T2D of the CLEARED study.[52] The CARTER study found that cilnidipine (in combination with an RAS blocker) decreased the progression of proteinuria in hypertensive patients, compared to amlodipine.[46] 93.2% of panelists agreed (median score 8.5) that L-/N-type CCB is more effective than L-type CCB in reducing proteinuria.

- Cilnidipine has dual L-/N-type calcium channel blocking property that helps in decreasing proteinuria.
- Cilnidipine, by reducing intraglomerular pressure, may provide better kidney protection than L-type CCBs.

Statement 13: Cilnidipine provides renoprotection because it has antioxidative properties, inhibits renal renin-angiotensin-aldosterone system (RAAS), and suppresses podocyte injury.

The highly specialized epithelial cells on the glomerular filtration barrier, the podocytes, act as an early indicator of CKD, as their injury or dysfunction leads to proteinuria and glomerulosclerosis.[53] Cilnidipine enhances podocyte protection and antiproteinuric effect probably by inhibiting the cycle of RAS and oxidative stress in the kidney, thereby reducing angiotensin II, the mediator of podocyte damage.[54] Moreover, cilnidipine notably reduces uric acid, compared to azelnidipine, the concentration of which is responsible for poor urinary albumin excretion and renoprotection.[55] The TACTICAL trial demonstrated that cilnidipine resulted in significant reductions in 8-hydroxy-2′-deoxyguanosine levels and the urinary liver-type fatty acid-binding protein-to-creatinine ratio, compared to amlodipine in hypertensive

patients, indicating less oxidative stress and tubular damage. The study demonstrated the renoprotective effect of cilnidipine through a significant reduction in UACR.[36] 73.3% panelists agreed, while 17.8% were neutral about the statement (median score: 7). A collective agreement on the inhibition of renal RAAS by cilnidipine could not be drawn. Many panelists pointed out that inhibition of RAAS would result in hyperkalemia, but that is generally not observed with cilnidipine in clinical practice. However, the panelists agreed upon the antioxidative and renoprotective properties of cilnidipine. The possible involvement of cilnidipine in providing renoprotection[36,54] is presented in Figure 3.

➤ The cycle of RAS and oxidative stress is inhibited by cilnidipine, which reduces angiotensin II and thus prevents podocyte injury and CKD progression.

Statement 14: Cilnidipine is one of the preferred CCBs for managing hypertension in patients with T2D for renoprotection.

A retrospective cohort study reported similar effects of cilnidipine, amlodipine, nifedipine, azelnidipine, and benidipine in improving renal function (change in eGFR and serum creatinine) in hypertensive patients with T2D.[56] A better efficacy of cilnidipine over azelnidipine in reducing albuminuria[57] and UACR[55] was reported in individuals diagnosed with T2D, which helped in slowing the progression of diabetic nephropathy. The 2022 RSSDI guideline recommended cilnidipine as the preferred CCB for its cardioprotective and renoprotective effects and better safety profile in diabetic hypertensive patients,[30] also agreed by 64.4% panelists (median score of 7). In India, cilnidipine monotherapy (91.2%) or combination therapy (76%) is widely used for diabetic hypertensive patients with renal comorbidities, as it reduces BP and provides cardioprotective and vasoprotective effects.[38] A comparatively better safety and efficacy of cilnidipine than olmesartan in reducing albuminuria and incidence of adverse reactions (hyperkalemia, dizziness and dry cough) have been reported in patients with hypertension and T2D.[58] Amlodipine is a time-tested, cost-effective L-type CCB, but cilnidipine is a better alternative for patients with sympathetic overactivity, proteinuria and pedal edema.[28]

Some evidences highlighting cardioprotective and renoprotective effects of cilnidipine in hypertensive patients are summarized in Table 2.

The study has its own set of limitations. The consensus reflects expert opinion in India and may not fully extrapolate to other populations. Additionally, variability in cilnidipine dosing and lack of randomized comparative trials on long-term cilnidipine data are the other noted limitations.

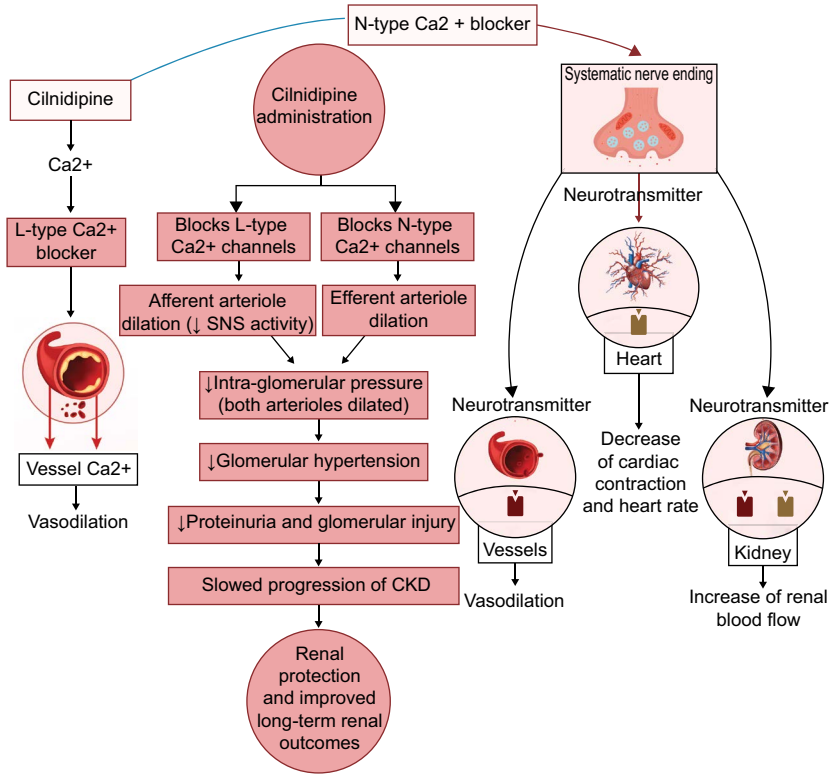

Fig. 2: Mechanism of cilnidipine in providing renoprotection and cardioprotection

Conclusion

Valuable insights on the cardioprotective and renoprotective role of CCBs in hypertensive patients with CKD have been provided through this consensus recommendation, particularly emphasizing the antihypertensive effect of L-/N-type CCB cilnidipine. The consensus could be helpful for shaping the knowledge of Indian clinicians. In conclusion, the CCBs should be used with a thorough understanding of their antihypertensive and organ-protective effects to ensure optimal therapeutic outcomes and minimal adverse effects in patients with hypertension and CKD.

Author Contribution

Conceptualization, data curation, formal analysis, methodology, supervision, validation, visualization, writing—original draft, and writing—review and editing: All authors. Project administration: SJ. Resources and software: SJ and OCS.

Conflict of Interest

Dr Sanjay Jain and Dr Onkar C Swami are full-time employees of Alembic Pharmaceuticals Ltd., which actively markets Cilnidipine.

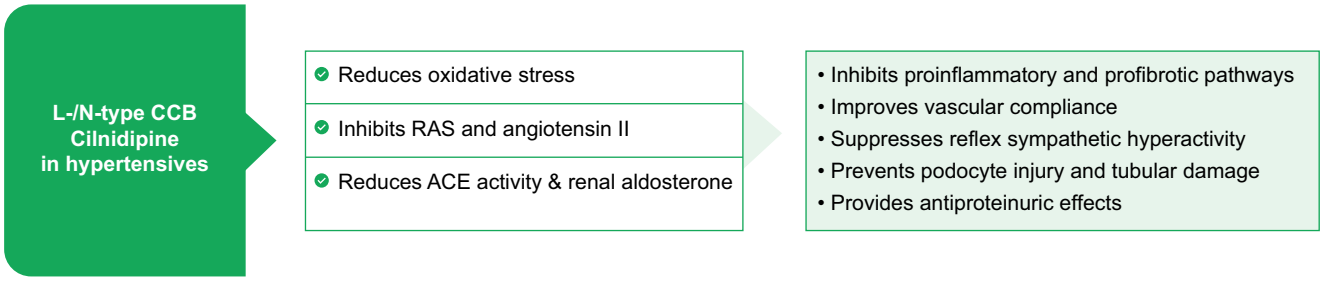

Fig. 3: Role of cilnidipine in overall renoprotection (ACE, angiotensin-converting enzyme; CCB, calcium channel blocker; RAS, renin–angiotensin system)

Table 2: Cardioprotective and renoprotective effects of cilnidipine

Study type	*Study population*	*Treatment*	*Renal/cardiac effects*
Multicenter, prospective, randomized, open-label clinical trial (TACTICAL2)[23]	Patients with hypertension (*n* = 25) receiving an RAS blocker	Cilnidipine vs amlodipine	Amlodipine significantly increased the levels of plasma angiotensin I and II Cilnidipine significantly reduced UACR Cilnidipine provides beneficial effects by increasing the ratio of angiotensin I and II
Open-label, parallel design randomized controlled study[28]	Patients with stage 1 hypertension (*n* = 63)	Cilnidipine vs amlodipine	Cilnidipine significantly decreased PR, while amlodipine increased PR Incidence of pedal edema was noted with amlodipine
Open-label, randomized study[35]	Patients with hypertension (*n* = 110)	Once-daily morning administration of cilnidipine vs amlodipine	In the amlodipine group: Significantly higher nighttime PR than pretreatment Higher 24-hour and daytime PR than pretreatment In cilnidipine group: No significant change in pre- and post-treatment PR Significantly decreased 24-hour PR, along with daytime and nighttime PR than amlodipine No reflex tachycardia
Randomized, open-labeled clinical trial (TACTICAL)[36]	Patients with hypertension (*n* = 35), receiving RAS blocker	Cilnidipine vs amlodipine as add-on therapy	Significantly reduced UACR with cilnidipine Cilnidipine provided renoprotection through its antioxidative properties
Multicenter, open-labeled, randomized study (CARTER)[46]	Patients with advanced CKD and hypertension (*n* = 339), receiving a RAS blocker	Cilnidipine vs amlodipine as add-on therapy	Significantly decreased UPCR with cilnidipine Cilnidipine exerted a greater antiproteinuric effect
Interventional study[50]	Patients with DKD (*n* = 50)	Cilnidipine vs amlodipine	12 weeks cilnidipine treatment: Significantly reduced UPCR Significantly reduced serum creatinine Significantly increased eGFR
Multicenter study (J-CIRCLE)[51]	Patients with hypertension and CKD, UACR ≥30 mg/g (*n* = 70)	Amlodipine was switched to cilnidipine	Cilnidipine exerted hypotensive activity, similar to amlodipine, and improved albuminuria Cilnidipine suppressed excessive uric acid formation
Multicenter, open-label, nonrandomized crossover study (CLEARED)[52]	Patients with T2DM, normoalbuminuria and microalbuminuria (*n* = 90), receiving CCB treatment for 6 months	Switched from cilnidipine to an L-type CCB or vice versa for 6 months	Switching from L-type CCB to cilnidipine significantly reduced urinary albumin excretion Switch from cilnidipine to L-type CCB led to a nonsignificant change

bpm, beats/minute; CARTER, Cilnidipine vs Amlodipine Randomized Trial for Evaluation in Renal Disease; CCB, calcium channel blocker; CLEARED, Cilnidipine vs L-type CCBs, Evaluation of Antihypertensive and Renoprotective Effects in Diabetic Patients; DBP, diastolic blood pressure; DKD, diabetic kidney disease; eGFR, estimated glomerular filtration rate; J-CIRCLE, Johoku-Cilnidipine Trial of Renal Function and Blood Pressure for Clinical Evaluation; PR, pulse rate; RCT, randomized controlled trial; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus; TACTICAL, Tokushima Antioxidation Clinical Trial in Hypertensives, Cilnidipine vs Amlodipine; UACR, urine albumin-to-creatinine ratio; UPCR, urinary protein-to-creatinine ratio

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