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Original Research Article

Cilnidipine antihypertensive efficacy, pleiotropic benefits and experiences by cardiologists: findings from a digital questionnaire-based study

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ABSTRACT

Background: Cilnidipine, a fourth-generation calcium channel blocker with dual L- and N-type action, offers distinct benefits in hypertension, though varied cardiologist awareness and prescribing may limit optimal use.
Methods: A cross-sectional survey was conducted among 416 practicing cardiologists across India between 17 April and 15 July 2025 to assess knowledge, perceptions, and prescribing preferences for cilnidipine in essential hypertension. A validated 20-item self-administered multiple-choice questionnaire captured mechanistic understanding and clinical perspectives following e-consent. Items addressed comparative advantages over other calcium channel blockers, effects on target organs, therapeutic applications (including combinations and patient subgroups), and metabolic benefits in metabolic syndrome. Responses were collected electronically and analyzed using descriptive statistics to summarize awareness and prescribing trends.
Results: Most cardiologists (85.1%) identified cilnidipine's dual L- and N-type blockade as its key differentiator from conventional CCBs. Over half recognized its superior nocturnal BP control (56.3%), reduced BP variability via autonomic stabilization (52.6%), and lowering of mean arterial pressure without reflex tachycardia (54.6%). A substantial proportion (64.2%) acknowledged reno-protective benefits over amlodipine, while 55.8% favored its use in elderly and resistant hypertensive patients. Notably, 61.0% recognized improved insulin sensitivity in metabolic syndrome. However, knowledge gaps remained regarding its effects on the RAAS and endothelial function.
Conclusions: The survey reveals strong cardiologist awareness of cilnidipine's sympatholytic, reno-protective, and hemodynamic benefits, alongside partial misconceptions regarding RAAS and endothelial effects. These findings underscore the need for ongoing clinical education and support cilnidipine's role as a well-tolerated, organ-protective antihypertensive.

Keywords: Cilnidipine, Hypertension, Calcium channel blocker

INTRODUCTION

Hypertension remains the most prevalent modifiable risk factors for cardiovascular morbidity and mortality worldwide, demanding lifelong pharmacologic intervention in most patients.[1]

Among the antihypertensive therapies, calcium channel blockers (CCBs) remain a cornerstone due to their potent vasodilatory effects and favourable metabolic profile.[2] While CCBs operate through a shared mechanism of inhibiting calcium influx, the pharmacological effects they produce vary significantly across different subclasses.[3] The first-generation dihydropyridine CCBs, such as nifedipine and amlodipine, are often limited by adverse

effects including reflex tachycardia and peripheral edema that can compromise long-term adherence.[4-6] The availability of fourth-generation highly lipophilic dihydropyridines, has introduced a new level of therapeutic ease, offering sustained efficacy, minimized adverse effects, and broad clinical utility, particularly in managing myocardial ischemia and potentially in congestive heart failure.[3] A meta-analysis stated that its high lipophilicity contributes to sustained efficacy and reduced blood pressure variability compared to other CCBs. Emerging calcium channel blockers that exhibit sustained activity and target both T-type and N-type calcium channels may offer superior therapeutic benefits compared to classical agents, potentially broadening their clinical applications.[7,8]

Amlodipine remains the most prescribed CCB for managing hypertension. While both amlodipine and cilnidipine demonstrate comparable efficacy in lowering blood pressure, cilnidipine by targeting both N-type and L-type calcium channels is associated with a reduced incidence of pedal edema relative to amlodipine, which selectively blocks only L-type channels.[9,10]

Cilnidipine, a fourth-generation dihydropyridine CCB, offers a unique advantage by blocking both L-type and N-type calcium channels.[3] This mechanism not only lowers blood pressure effectively but also attenuates sympathetic nerve activity, thereby reducing the risk of tachycardia and pedal edema compared with conventional agents.[11] Furthermore, cilnidipine has shown renoprotective and cardioprotective benefits, particularly in patients with diabetes and proteinuria.[12] The cardioprotective action of cilnidipine has been analyzed in a rabbit model of myocardial infarction, in which cilnidipine decreased the myocardial interstitial norepinephrine levels during ischemia and reperfusion periods, leading to reduction of the myocardial infarct size and incidence of ventricular premature beats.[13] Although cilnidipine awareness among Cardiologists is considerable, particularly in Indian clinical settings where Cardiologist surveys show its wide preference due to favourable tolerability and reno-cardioprotective profile, its adoption still varies by region and practice context.[14,15] This variability may stem from gaps in clinical knowledge or divergent prescribing preferences, which could impede the optimal incorporation of cilnidipine into hypertension management. To address this issue, the present survey seeks to assess Cardiologists' familiarity with, attitudes toward, and prescribing patterns related to cilnidipine, with particular focus on its perceived benefits over traditional CCBs.

METHODS

A cross-sectional, multiple-response questionnaire–based survey was conducted among 416 practicing Cardiologists across India from 17 April 2025 to 15 July 2025 to evaluate Cardiologists' knowledge and prescribing preferences regarding cilnidipine in hypertension management. An electronic consent was taken from 416 cardiologists, located at major cities across Indian states to provide data.

The survey questionnaire comprised 20 structured questions designed to capture information through a secured online platform on cardiologist awareness of cilnidipine's pharmacological profile, comparative efficacy and safety, preferred clinical scenarios for its use, and factors influencing its selection in routine practice. It also explored alignment with hypertension treatment guidelines and identified potential knowledge gaps that may impact optimal prescribing behaviour.

The questionnaire was developed by the study team based on predefined clinical objectives and a review of relevant literature to assess cardiologists' perspectives and routine clinical practices. It was designed as a structured, self-administered survey instrument. No previously validated scale or formal scoring system was employed, as the objective of the study was to descriptively capture expert opinions and practice patterns rather than to quantify outcomes using standardized metrics. The study did not involve direct patient recruitment, patient interviews, or collection of individual patient-level data. The responses reflected the cardiologists' clinical experience and routine practice patterns. As no specific patients were enrolled in the study, demographic details of individual patients were not collected or analysed.

Data were analysed using descriptive statistics. Categorical variables were expressed as percentages to summarize their distribution, and the frequency of occurrence along with the corresponding percentage was used to represent each variable. To visualize the distribution of categorical variables, graphs and pie charts were created using Microsoft Excel 365 (version 22502, March 11).

RESULTS

Cardiovascular effects of cilnidipine

The hemodynamic and antihypertensive effects of Cilnidipine were evaluated through questions exploring its hemodynamic benefits, superiority in nocturnal blood pressure control, preference in elderly hypertensive patients, ability to stabilize blood pressure variability, influence on sympathetic inhibition, and potential for cardiovascular risk reduction. The survey revealed that most cardiologists recognized cilnidipine's additional N-type calcium channel blockade as its key distinction from amlodipine (85.1%), whereas only a few associated this difference with blockade of P/Q-type (5.8%), T-type (5.5%), or R-type (3.6%) channels.

In terms of clinical outcomes, cardiologists most frequently identified reduction in nocturnal blood pressure spikes (56.3%) as the area where cilnidipine outperforms amlodipine, followed by improvement in left ventricular ejection fraction (26.7%), reduction in total cholesterol

(9.9%), and enhanced vasoconstrictive response (7.2%). When asked about preferred patient groups, elderly hypertensive patients were considered most suitable for cilnidipine, primarily because it lowers blood pressure without triggering sympathetic activation (55.8%). Other cited advantages included its natriuretic effect (17.3%), greater impact on systolic than diastolic pressure (13.7%), and shorter half-life minimizing drug accumulation (13.2%) (Figure 1). Stabilization of autonomic tone was the most recognized mechanism (52.6%) for blood pressure variability as reported by cardiologists, though some respondents mentioned episodic vasodilation leading to increased BPV (23.1%), or reported no notable effect (18.0%). With respect to sympathetic modulation, the reduction in mean arterial pressure without an accompanying rise in heart rate was identified as the principal antihypertensive action (54.6%) of cilnidipine, while baroreceptor-mediated bradycardia (25.0%), inhibition of renin secretion (12.3%), and enhanced vasoconstrictive response to catecholamines (8.2%) were also reported as contributory mechanisms.

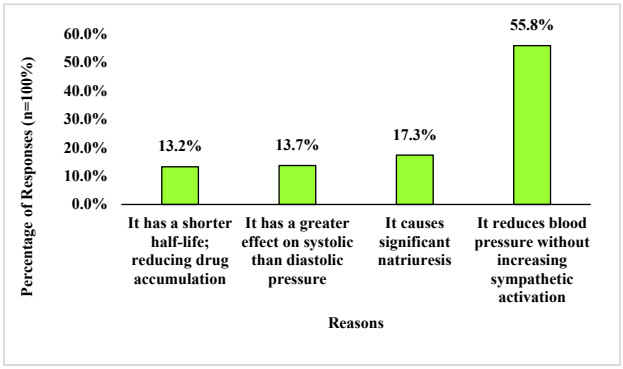

Figure 1: Reasons for preferring cilnidipine in elderly hypertensive patients.

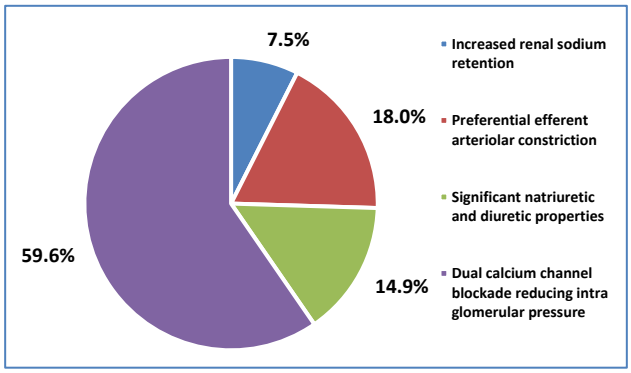

Figure 2: Reasons for cilnidipine use in hypertensive patients with proteinuria.

Renal protection with cilnidipine (kidney function and proteinuria)

Cardiologists highlighted cilnidipine's nephroprotective role through questions assessing its ability to reduce proteinuria, decrease intraglomerular pressure, and modulate renin-angiotensin-aldosterone system (RAAS) activity in hypertensive patients with renal involvement.

Overall, most cardiologists (64.2%) reported that switching to cilnidipine in patients with chronic kidney disease (CKD) and hypertension is most likely to reduce glomerular pressure and proteinuria, while 59.6% indicated that its dual calcium channel blockade makes it particularly useful in proteinuric kidney disease by lowering intraglomerular pressure (Figure 2). Regarding its mechanism of action, 44.0% of cardiologists believed that cilnidipine indirectly suppresses the RAAS through reduced sympathetic activation. Smaller proportions of respondents attributed its effects to preferential efferent arteriolar constriction (18.0%), natriuretic and diuretic properties (14.9%), direct inhibition of aldosterone production (9.1%), or reported no change in proteinuria (7.2%). A minority anticipated less favourable effects, such as increased urinary protein excretion (19.7%), increased angiotensin II synthesis (28.1%), or enhanced afferent arteriolar constriction (8.9%).

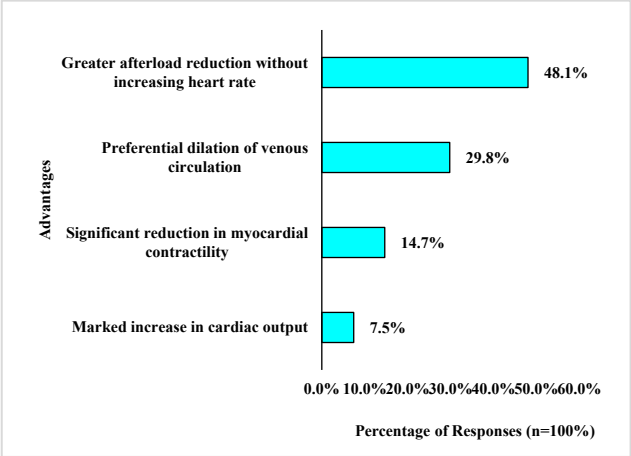

Figure 3: Hemodynamic advantages of cilnidipine compared to dihydropyridine CCBs.

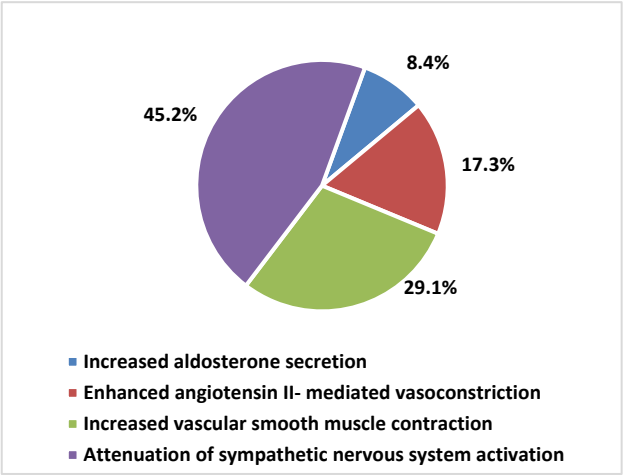

Figure 4: Direct consequences of cilnidipine's N-type calcium channel blockade.

Vascular and neurohumoral effects (endothelium and sympathetic modulation)

The vascular and neurohumoral effects of cilnidipine were explored through questions assessing its additional N-type calcium channel blockade, suppression of sympathetic overactivity, improvement in endothelial function, prevention of pedal edema through reduced sympathetic activation, and long-term benefits such as reduced arterial stiffness and improved vascular compliance.

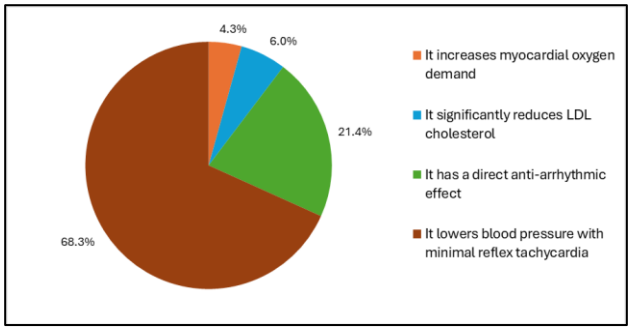

Figure 5: Cilnidipine vs. dihydropyridine CCBs: cardiovascular risk reduction advantages.

Most cardiologists found cilnidipine's main hemodynamic advantage to be greater afterload reduction without raising heart rate (48.1%), plus venous dilation (29.8%) and lowered myocardial contractility (14.7%) (Figure 3). Its primary action is seen as N-type calcium channel blockade, suppressing sympathetic outflow (51.4%) and reducing activation (45.2%). Cilnidipine's N-type blockade mainly attenuates sympathetic activation (45.2%), with smaller effects on vascular contraction (29.1%), angiotensin II vasoconstriction (17.3%), and aldosterone secretion (8.4%) (Figure 4).

Cilnidipine reportedly improves endothelial function by reducing oxidative stress (56.5%) and aids long-term hypertension control through less arterial stiffness and better vascular compliance (59.1%). Compared to amlodipine, it helps prevent pedal edema (44%), lowers systemic venous resistance (20.7%), and limits bradycardia (11.3%). Additional noted benefits include efficacy in severe bradycardia, acute heart failure, orthostatic hypotension, negative inotropic effect, natriuresis, and heart rate reduction. Overall, cilnidipine is considered a superior choice to amlodipine due to its combined cardiovascular benefits.

Therapeutic applications and combination strategies (clinical use)

The therapeutic applications and combination strategies of cilnidipine were evaluated through questions examining its role with the combination medication in resistant hypertension, suitability in patients with autonomic dysfunction and exaggerated sympathetic response, recognized clinical advantages such as reduction in central aortic pressure and proteinuria, and its potential for cardiovascular risk reduction by lowering blood pressure with minimal reflex tachycardia.

Most cardiologists consider cilnidipine highly effective and versatile in hypertension management: 55.8% reported that combining it with ACE inhibitors or ARBs provides the best blood pressure control in resistant cases, while smaller proportions favored combinations with beta-blockers (29.3%), loop diuretics (9.1%), or amlodipine (5.8%). For patients with newly diagnosed hypertension and autonomic dysfunction, 85.6% identified cilnidipine as the most rational choice due to its sympatholytic properties, compared with much lower preferences for amlodipine (5.3%), hydrochlorothiazide (4.8%), or diltiazem (4.3%). Cardiologists also highlighted its advantages and limitations: 68.3% recognized its ability to lower blood pressure with minimal reflex tachycardia as a key benefit over conventional dihydropyridine CCBs, while smaller groups emphasized anti-arrhythmic effects (21.4%) or LDL cholesterol reduction (6.0%), and 4.3% noted increased myocardial oxygen demand (Figure 5). Conversely, notable proportions of cardiologists did not consider certain effects such as reduction in central aortic pressure (30.3%), increased renin secretion (29.3%), attenuation of sympathetic overactivity (23.8%), or reduction in proteinuria (16.6%) as recognized clinical benefits of cilnidipine.

Metabolic benefits in hypertension with metabolic syndrome (pancreatic function)

The metabolic benefits of cilnidipine in patients with hypertension and metabolic syndrome were assessed through questions focusing on its ability to improve insulin sensitivity, highlighting its favourable impact on pancreatic function and overall metabolic regulation.

Most cardiologists (52.2%) viewed improved insulin sensitivity as the key beneficial effect of cilnidipine for managing hypertension in patients with metabolic syndrome, while smaller proportions noted its effects on sodium and water retention (24.3%), stimulation of beta-cell insulin secretion (17.1%), and a potential increase in fasting plasma glucose levels (6.5%).

DISCUSSION

This survey assessed the cardiologists' awareness and prescribing preferences for cilnidipine in treatment of hypertension and evaluated how the perceived benefits by cardiologists influence their choice in clinical practice. Cilnidipine is unique among dihydropyridine CCBs because it blocks both L-type and N-type calcium channels. The blockade of N-type channels inhibits sympathetic neurotransmission, reducing heart rate variability and vasoconstrictive tone.[16] In this survey, a substantial proportion of respondents (85.1%) correctly identified the dual blockade of L- and N-type calcium channels as the key differentiating feature of cilnidipine,

reflecting strong awareness of its unique pharmacological profile. Additionally, in elderly hypertensive patients, 55.8% of cardiologists preferred cilnidipine over conventional CCBs because it effectively lowers blood pressure with anti-sympathetic activity.[17] Furthermore, 56.3% of cardiologists acknowledged that cilnidipine offers superior control of nocturnal blood pressure surges compared to amlodipine. These findings are consistent with that of the comparative study of cilnidipine vs amlodipine demonstrating improved night time BP stability, autonomic regulation and sympathetic modulation with cilnidipine.[18] Blood pressure variability (BPV) is associated with arterial stiffness, which can impair baroreceptor sensitivity a key factor influencing BPV. In a comparative study involving 60 Indian patients treated with either amlodipine or cilnidipine, cilnidipine demonstrated greater efficacy in reducing arterial stiffness, thus implying its beneficial effects in reducing BPV.[19] Over half of the cardiologists (52.6%) recognized that cilnidipine reduces blood pressure variability (BPV) by stabilizing autonomic tone. Most of the cardiologists (54.6%) reported the observations align with existing clinical data showing that cilnidipine reduces blood pressure without inducing reflex tachycardia, owing to its suppression of sympathetic overactivity via N-type calcium channel blockade.[20]

The impact of dihydropyridine CCBs on proteinuria and renal function differs depending on the specific subclass of channel blockade. Compared to amlodipine, a 12-month regimen of cilnidipine an N-type CCB administered alongside other antihypertensive agents, attenuated the progression of proteinuria and led to a modest decline in glomerular filtration rate.[21] Consistent with these findings, results from the current survey reveal that a majority (64.2%) of cardiologists believe switching to cilnidipine in patients with chronic kidney disease (CKD) and hypertension is likely to reduce glomerular pressure and proteinuria, identifying this as the primary therapeutic benefit. Furthermore, 59.6% of respondents reported that cilnidipine is particularly beneficial in hypertensive patients with proteinuric kidney disease due to its dual L- and N-type calcium channel blockade, which contributes to a reduction in intraglomerular pressure. These perceptions among cardiologists align with published evidence supporting cilnidipine's reno-protective effects. Previous studies have shown that although cilnidipine had no significant effect on plasma renin activity or plasma angiotensin II levels, amlodipine markedly increased these parameters compared with the vehicle group. Moreover, cilnidipine was associated with significantly lower plasma aldosterone levels and reduced renal cortical tissue norepinephrine concentrations, suggesting an attenuated sympathetic response.[22] Findings from the current survey align with these mechanistic observations, with 44.0% of cardiologists attributing cilnidipine's RAAS effect to indirect suppression via reduced sympathetic activation. Meanwhile, 28.1% suggested possible enhancement of angiotensin II synthesis, 18.8% reported no significant RAAS impact, and 9.1% cited direct inhibition of aldosterone production.

Cilnidipine exhibits a stronger inhibitory effect on N-type calcium channels compared to other calcium channel blockers. *In vitro* study by Nap et al have shown that cilnidipine effectively reduces norepinephrine release from sympathetic nerve terminals.[23] In the current survey, 45.2% of cardiologists identified attenuation of sympathetic nervous system activation as the direct consequence of cilnidipine's N-type calcium channel blockade. Additionally, 51.4% recognized that cilnidipine suppresses sympathetic outflow via N-type channel blockade, while smaller proportions attributed it to increased norepinephrine release (20.2%), presynaptic L-type blockade (17.3%), or no autonomic effect (11.1%). The survey also concluded that patients with increased sympathetic overactivity benefit the most from the cilnidipine therapy. Cilnidipine has been shown to exert beneficial effects on endothelial function through multiple mechanisms. A study by Fan et al demonstrated that cilnidipine relaxes human arteries via calcium channel antagonism and by enhancing endothelial nitric oxide synthase (eNOS) activity, thereby increasing nitric oxide (NO) production in the human internal thoracic artery.[24] In alignment with these findings, results from the current survey indicate that cardiologists (56.5%) recognize cilnidipine's endothelial benefits, with the majority reporting that it improves endothelial dysfunction by reducing oxidative stress. Together, these mechanisms enhanced NO bioavailability and attenuation of oxidative stress underscore cilnidipine's favorable impact on vascular endothelial health. A clinical investigation assessed the therapeutic impact of cilnidipine in 27 patients diagnosed with essential hypertension who developed ankle edema as an adverse effect of amlodipine. The study reported complete resolution of edema in all participants following the transition to cilnidipine, without any significant deterioration in blood pressure control or the emergence of tachycardia. These findings highlight cilnidipine as a well-tolerated and effective alternative antihypertensive agent for individuals experiencing amlodipine-induced peripheral edema.[25] In the survey, 44.0% of cardiologists attributed amlodipine-induced pedal edema to increased capillary hydrostatic pressure, whereas cilnidipine prevents sympathetic overactivation; smaller proportions cited venodilation (24.0%), systemic venous resistance (20.7%), or bradycardia (11.3%) as differentiating factors.

The use of combination therapy with a CCB and an angiotensin II receptor blocker (ARB) has been steadily increasing. Cilnidipine, when combined with an ARB, effectively controls blood pressure without significant adverse effects and additionally helps reduce elevated heart rate, a potential cardiovascular risk factor.[26] In the current survey, the majority (55.8%) of cardiologists reported that cilnidipine combined with ACE inhibitors or ARBs provides superior blood pressure control in resistant hypertension. Smaller proportions favoured combinations

with beta-blockers (29.3%), loop diuretics (9.1%), or amlodipine (5.8%).

Cilnidipine not only lowers blood pressure but also exerts beneficial metabolic effects by promoting glucose metabolism. This property is particularly important in patients with hypertension who also have insulin resistance, a common feature of metabolic syndrome and obesity. By improving insulin sensitivity, cilnidipine helps reduce hyperinsulinemia and associated cardiovascular risks, making it a valuable therapeutic option for hypertensive patients with metabolic disorders.[11,27] In the present survey, the majority (52.2%) of cardiologists identified improvement in insulin sensitivity as the key benefit of cilnidipine in such patients. Smaller proportions cited promotion of sodium and water retention (24.3%), stimulation of β-cell insulin secretion (17.1%), and increased fasting plasma glucose (6.5%) as its potential effects.

The present survey has several notable strengths. It included a large and diverse cohort of 416 cardiologists across India, providing comprehensive insights into real-world clinical perceptions and prescribing practices related to cilnidipine. The structured questionnaire was designed to capture both mechanistic understanding and therapeutic reasoning, enabling a focused evaluation of cardiologist awareness in alignment with current scientific evidence. Moreover, the findings bridge the gap between pharmacological data and clinical practice, offering valuable direction for educational and therapeutic strategies in hypertension management. However, the study also has inherent limitations typical of survey-based research. The responses were self-reported and may be influenced by recall or response bias, reflecting perceptions rather than verified prescribing behavior. Being cross-sectional, the survey represents knowledge and attitudes at a single point in time and does not assess causality or changes over time. Additionally, the voluntary nature of participation may introduce selection bias toward cardiologists already familiar with cilnidipine, while the absence of correlation with actual prescription or patient outcome data limits external generalizability. Despite these constraints, the survey provides meaningful insights that can guide further clinical education and research on evidence-based hypertension therapy.

CONCLUSION

This survey provides real-world insights into Cardiologists' awareness and prescribing preferences for Cilnidipine in hypertension management. Most respondents recognized its dual L- and N-type calcium channel blockade and related benefits, including sympathetic inhibition, blood pressure stability, and renal protection. While overall awareness was high, some misconceptions regarding its effects on RAAS and endothelial function remain, indicating the need for continued education. The findings reflect Cilnidipine's growing acceptance as a comprehensive antihypertensive with both hemodynamic and organ-protective advantages.

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