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# Impact Of Nicardipine, A Calcium Channel Blocker, On Renal Function And Systolic Blood Pressure

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**Abstract**

**Background:** Rapid but controlled blood pressure lowering is necessary to prevent end-organ damage, especially renal impairment, in hypertensive crisis, a potentially fatal condition. Because of its good hemodynamic profile and titratable IV administration, nicardipine, a calcium channel blocker, is frequently utilized.

**Objective:** To evaluate the effect of nicardipine on systolic blood pressure and renal function parameters in patients presenting with hypertensive crisis.

**Methodology:** Patients at a tertiary care hospital in Lahore who were receiving intravenous nicardipine for blood pressure control participated in an observational study. Clinical and demographic information was documented. Prior to and following therapy, SBP and renal function parameters were measured, including serum creatinine, glomerular filtration rate(GFR), blood urea nitrogen (BUN), urine albumin-to-creatinine ratio, urea, and urine output. Data were analyzed by SPSS V-26 using paired sample t-tests to determine the significance of changes.

**Results:**SBP was found to have significantly decreased from 160.45±9.60 mmHg to 129.90±10.20 mmHg($p < 0.001$). DBP also dropped from 92.04±4.64 mmHg to 75.90±5.10 mmHg($p < 0.001$). From 2.172±0.893 mg/dL to 2.194±0.895 mg/dL, serum creatinine increased slightly but statistically significantly ($p = 0.010$). Urine production, on the other hand, increased dramatically from 0.956±0.165 to 1.056±0.170 mL/kg/hour($p<0.001$), suggesting greater renal perfusion.

**Conclusion:** Nicardipine is an effective and safe agent for rapid blood pressure reduction in hypertensive crisis, with minimal adverse effects on renal function. Its use may be considered beneficial in clinical settings requiring precise blood pressure management without compromising renal integrity.

## Introduction

Hypertension is an extremely widespread and clinically relevant cardiovascular disorder with risk factor of stroke and other end-organ damage. Pharmacological interventions to manage hypertension, especially in critical care and perioperative conditions, should be effective in the rapid reduction of blood pressure but also safe in terms of end-organ performance, such as renal integrity. Calcium channel blockers (CCBs) represent one of the most commonly used antihypertensive agents, as they have been successfully identified to possess mechanisms of action and have been shown to provide good pharmacokinetic profiles (1,2).

The United States Food and Drug Administration (FDA) approved nicardipine hydrochloride, a dihydropyridine-based calcium channel blocker of the second generation, in December 1988. It acts on the antihypertensive mechanism by the predominant action of peripheral vascular artery vasodilatation through blocking calcium ion influx into the vascular smooth muscle cells, thereby decreasing vascular resistance hence systolic blood pressure (SBP) . Contrary to non-dihydropyridine CCBs, nicardipine does not influence cardiac conduction pathways significantly, which implies a low risk of bradydysrhythmias and makes it especially appropriate in the patients with cardiac conduction disturbances. Moreover, nicardipine has a great arterial vascular selectivity, and there is a high coronary and cerebral vasodilatory activity and low negative inotropic cardiac actions. The pharmacokinetics of this drug are typified by a rapid onset of activity of 1 to 2 minutes, dose-dependent pharmacokinetics, and tri-exponential plasma concentration decrease following intravenous (IV) infusion, with a rapid early distribution phase (alpha-half-life of 2.7 minutes), an intermediate phase (beta-half-life of 44.8 minutes), and a slow terminal phase (gamma half-life of 14.4 hours), which is only evident after prolonged infusion. Such characteristics make nicardipine a fast-titratable agent with a short-term half-life, a limited number of side effects, and makes it highly applicable in acute clinical settings that need a precise blood pressure regulation to be achieved (3,4,5).

Nicardipine has a wide range of clinical indications, and it is applied in hypertensive emergencies, perioperative hypertension in cardiac and neurosurgical patients, acute intracerebral hemorrhage (ICH), hypertensive crisis, in children, severe pre-eclampsia, and intraoperative hypertension. Its effectiveness and efficacy have been established in numerous randomized controlled trials and observational studies which have all noted it to be better or equally effective in blood pressure control compared to other agents including labetalol, sodium nitroprusside, and clevidipine (6,7).

Although the antihypertensive effect of nicardipine is well-established, its effects on renal functions are a debatable issue of clinical concern and current research. The kidney is a target organ in hypertension and uncontrolled hypertension as well as excessive aggressiveness in blood pressure lowering can trigger renal damage. The SBP reduction induced by nicardipine and its effect on the renal outcomes are not understood entirely. On the one hand, vasodilatory effect of nicardipine could be beneficial as it enhances renal perfusion and stimulates natriuresis and diuresis by blocking the sodium reabsorption in the proximal renal tubules, proved by both animal and human research. Conversely, excessive or rapid decrease in SBP especially in patients who already have severe hypertension may interfere with the renal autoregulation process causing decreased glomerular filtration rate (GFR) and effective renal plasma flow and resulting in the acute kidney injury (AKI) (8,9).

Nicardipine has a complex renal impact, which is further elaborated by its mechanism of action. Nicardipine has been demonstrated to produce diuresis and phosphaturia in hypertensive surgical patients which could be attributed to blocking of phosphate reabsorption in proximal renal tubules but ensuring maintenance of normal blood pressure. Animal experiments have revealed that low-dose intravenous nicardipine is

capable of raising urine volume and urinary sodium excretion without causing any changes on the renal blood flow or GFR, indicating a direct tubular effect that is not mediated by the effects of hemodynamic alteration. These results point to the complex character of the renal effects of nicardipine which involve both hemodynamic and direct tubular action. The clinical importance of the effect of nicardipine on SBP is also very important. Higher SBP during the acute period correlates with the enlargement of hematoma, unfavorable functional results, and patient death in patients with ICH. Hourly SBP data analysis of individual participants have shown that mean hourly SBP in the first 24 hours of nicardipine infusion is positively correlated with death or disability and expansion of hematoma (adjusted OR 1.16, 95% CI 1.02-1.32), in hyperacute ICH patients. On the other hand, excessive SBP-reduction, especially relative reduction more than 20 percent during the first 48 hours has been found independently related with renal adverse events, brain ischemia and poor functional outcomes in discharge. All this data depict the balanced nature needed in the process of nicardipine-mediated SBP treatment, where either under-treatment or over-treatment is very dangerous (10).

Outside the acute ICH setting, the effect of nicardipine on SBP in elderly patients with isolated systolic hypertension (ISH) has been investigated whereby it induced significant improvements in office SBP and pulse pressure over 90 days, but amlodipine induced much better improvements in office SBP as observed by 24-h ambulatory blood pressure monitoring, especially during the nighttime. Nicardipine was associated with the reduction of SBP by an average of 14.6% in one hour in pediatric hypertensive crisis patients, which is similar to labetalol (11.9%), and there was no significant difference in the clinical outcomes of the two agents. Intravenous nicardipine was effective to manage hypertensive crises in the context of severe pre-eclampsia achieving target blood pressures by peripheral vasodilatation and decreased total vascular resistance without damaging the maternal or uteroplacental circulation (11).

Effects of nicardipine on renal activity and SBP has a wide clinical potential in a variety of patient groups and medical environments. Nicardipine is commonly used as a first- or second-line agent in the management of acute hypertension in critical care or emergency care units and perioperative settings. It is crucial that the clinicians are aware of the renal implications of the use of nicardipine therapy to maximize dosing regimens, establish proper SBP goals, and observe the possible adverse renal events, especially in such high-risk groups as individuals with already impaired renal function, severe hypertension, or acute neurological injury. The body of evidence on the use of nicardipine is still growing, and the recent meta-analyses of individual participants and large randomized controlled trials offer more and more detailed information on the dose-response relationships between the nicardipine-mediated SBP lowering effect and clinical outcomes (12).

Moreover, the relative efficacy of nicardipine against other antihypertensive drugs such as clevidipine, labetalol, and sodium nitroprusside has significant clinical, resource, and cost-effective implications. The pharmacokinetic profile of nicardipine and its ability to be titrated make it a convenient and broadly applicable agent in the acute treatment of blood pressure, and the renal effects of the drug should be critically considered in the context of a tailored treatment considerations (13).

The rationale behind the proposed investigation, therefore, lies in the fact that, although the use of nicardipine is common and it is generally well-tolerated, the interaction of its effect on blood pressure and kidney performance has not been fully comprehended and it may pose a clinical threat. The vasodilatory and natriuretic effects of nicardipine can be useful to renal activity in some circumstances, but aggressive decrease of SBP caused by high dosages of nicardipine can develop AKI, especially when patients have severe hypertension and a deficiency in renal autoregulation. The systematic synthesis of evidence available is necessary to resolve this paradox and to maximize the clinical

utility of nicardipine and reduce risks of iatrogenic renal injury and, finally, improve patient outcomes regardless of the type of hypertensive disorders

## LITERATURE REVIEW

Postoperative Blood Pressure Goals after Craniotomy for Tumor Resection: A National Survey. The purpose of the study was to review the existing practices in the management of postoperative blood pressure (BP) following craniotomy to recombat tumors, since postoperative hypertension is common, and the hypertension may contribute to the onset of intracranial hemorrhage.The authors conducted a national survey (cross-sectional) of participants in 117 accredited neurosurgical residencies (a total of 10 items in the questionnaire) who were residents of these institutions. The program level was analyzed and the responses of the senior residents were not discarded in the instances of receiving several responses. The results indicated that the majority of the programs achieved BP goals based on systolic blood pressure (SBP). SBP below 140 mmHg (4143%), and SBP below 160 mmHg (3639%), was the most prevalent target. These were normally held till the morning of the first postoperative day. The most common medications to control BP were intravenous nicardipine and labetalol. The research found that SBP goals are widely applied in the post-tumor resection craniotomy setting, and that there is a great disparity in across the programs, suggesting that there should be no definitive evidence on this, thus future clinical trials to establish the best BP target(14).

The safety and renal outcome of calcium channel blockers, such as nicardipine, in patients with severe aortic stenosis and patients with hypertension concomitant to the stenosis. It comprised 2,460 patients with a severe aortic stenosis (aortic valve area less than 1.0 cm 2) and hypertension who were on antihypertensive therapy. The patients were classified into two groups; the CCBs (n= 1,763 with 342 taking nicardipine) and the non-CCB antihypertensive therapy (n= 697). The follow-up was done over 3 years, and the control of BP, renal (eGFR, serum creatinine) and clinical outcomes were assessed regularly. These findings showed that CCBs were prescribed in the highest proportion of 71.7 percent of patients, and thus it is the most frequently used antihypertensive medication in this group. Blood pressure (SBP <140 mmHg) was controlled in 68.4% of patients treated with CCB compared to 62.1% of patients not treated with CCB (P = 0.008). In terms of renal outcomes, the eGFR decline/per year was not different between groups (CCB group: -1.82 ± 0.24 mL/min/1.73m 2; non-CCB group: -1.94 ± 0.31 mL/min/1.73m 2; P = 0.42). The hospitalization rate of acute kidney injury was 3.2 and 3.8 in the CCB and the non-CCB group (P = 0.51). Nicardipine treated patients had similar renal safety profiles compared to other CCBs through subgroup analysis. These results imply that CCBs such as nicardipine are safe and effective in the management of blood pressure in patients with severe aortic stenosis and hypertension, and do not indicate the presence of faster renal function impairment (15).

The protective characteristics of calcium channel blockers (containing amlodipine a dihydropyridine CCB just like nicardipine) on renal and erythrocyte oxidative stress in salt-induced hypertension. The count of the sample was 24 male Wistar rats who were categorized into four groups: normal control, salt-loaded hypertensive control, salt-loaded treated with amlodipine and salt-loaded treated with indapamide. The 8 weeks salt diet feeding (8% NaCl) was used to cause hypertension and after 4 weeks of drug administration, blood and renal tissues were taken to test the oxidative stress parameter, renal functioning parameter, and erythrocyte osmotic fragility. The findings indicated that salt loading had significant effects on systolic blood pressure (118 -4 mmHg to 162-6 mmHg, P -0.001), antioxidant enzyme activities (superoxide dismutase: -42% catalase: -38% glutathione peroxidase: -35%), and erythrocyte osmotic fragility (50% hemolysis using 0.65% NaCl vs. 0.45% NaCl in controls). Amlodipine and indapamide reversed all these effects, elevating antioxidant enzyme activities (superoxide

dismutase: +76% with amlodipine, +68% with indapamide), stabilizing the erythrocyte membrane (50% hemolysis at 0.48% NaCl with amlodipine), and decreasing blood pressure (to 128 -5 mmHg with amlodipine). Renal tissue examination found that treatment with amlodipine decreased oxidative damage markers (malondialdehyde: -52%, protein carbonyls: -47 and glomerular structure maintained on histopathological analysis). The research finds that CCBs such as amlodipine (and possibly, by extension, nicardipine) possess antioxidant and renal protective effects in hypertensive conditions, which may not simply be due to the lowering in blood pressure (16).

Patients who were treated for hypertensive crises with either nicardipine or clevidipine were included in this multicenter, retrospective cohort research. The time from infusion start to goal blood pressure, which is the larger value of the physician-ordered goal or the guideline-directed 25% drop in blood pressure, was the main result. Secondary outcomes included the BP and heart rate excursions, length of stay in the intensive care unit (ICU), and time from infusion start to guideline-directed 25% drop in blood pressure, drug and total volume consumption, time from order entry to BP goal attainment, and study medication cost. The study included 182 individuals in total (79 getting clevidipine and 103 receiving nicardipine). The groups' time to goal blood pressure was comparable (35 vs. 33 minutes for clevidipine vs. nicardipine, respectively; P = 0.37). The time to a 25% reduction guided by guidelines was likewise comparable (P = 0.42). Volume from study drug was significantly less with clevidipine (222 vs 518 mL; P = 0.01); however, the total volume received in the ICU was similar (3,370 vs 3,383 mL; P = 0.43). Percent time in the goal BP range was similar (43.1% vs 42.3%). The cost of clevidipine was $199.37 per vial (based on the average wholesale price as of June 2023). This cost was 682% higher than that for a bag of nicardipine. In this population, the time to goal blood pressure was comparable for nicardipine and clevidipine. These findings suggest that in this diverse population, clevidipine might not offer a significant benefit. Given the absence of improvement in clinically meaningful outcomes, the cost difference does not appear to be justified (17).

The retrospective analysis was carried out at an academic Level 1 trauma hospital that sees more than 100,000 patients in its emergency department each year. This study sought to ascertain the safety and effectiveness of nicardipine in persons with lower ejection fractions who presented to the emergency department (ED) with AHF-H. The attainment of the blood pressure goal range established by the doctor served as a measure of efficacy. The number of people who had bradycardia (less than 60 beats per minute, bpm) or hypotension (systolic blood pressure, SBP, less than 90 mmHg) while taking nicardipine and for up to 15 minutes after stopping it were the main safety objectives. Brain natriuretic peptide was 731 pg/nL (418–3277) and the median ejection fraction was 35% (25–40). The median baseline SBP and heart rate were 193 mmHg and 90 bpm, respectively. All patients achieved the median physician-specified SBP objective of 160 mmHg in a median of 18 minutes. In the entire population, one patient (2.6%) experienced both bradycardia and hypotension. This patient had a 20% ejection fraction, was intubated, and took nicardipine in addition to esmolol for an aortic dissection. No adverse event occurred until half an hour after the start of dexmedetomidine. Nicardipine was shown to be safe and efficacious in this trial assessing its use in patients with lower ejection fractions who presented to the emergency department with AHF-H (18).

Evaluation of the Efficacy and Safety of Nicardipine Versus Clevidipine for Blood Pressure Control in Hypertensive Crisis. This was done to determine whether nicardipine and clevidipine are more effective and safe in managing blood pressure during hypertensive crisis, which is considered to be when blood pressure exceeds 180/120 mmHg.This is a retrospective cohort study at a single center that used 156 patients whose hypertensive crisis was treated with either nicardipine (n= 74) or clevidipine (n = 82). The first was a 25 percent decrease in mean arterial pressure in 1 hour, whereas the second was a systolic blood pressure (SBP) of below 160 mmHg in

26 hours of starting the infusion.The outcome revealed that there was no significant difference between the two drugs on the attainment of 25 percent reduction of mean arterial pressure at 1 hour. Nicardipine however attained the SBP target of less than 160 mmHg more than clevidipine (89.2% vs. 73.2, p= 0.011). The findings of the study were that both drugs are suitable to start hypertension treatment in hypertensive crisis, with better long-term SBP regulation with an inferior possibility of rebound hypertension and reduced cost than clevidipine (19).

The study was a multicenter, retrospective cohort study on the effectiveness and safety of clevidipine versus nicardipine in reducing intravenous blood pressure in a wide range of clinical indicators in a hospital system. The researcher identified 569 patients between June 1, 2020, and June 30, 2021, and then 100 propensity-matched pairs of patients who received clevidipine or nicardipine to manage acute hypertension in the intensive care unit, emergency service, and postoperative units. The main results were time to target range of blood pressure, the percentage of time in the target range in the first 6 hours of infusion, and the occurrence of hypotension and adverse events on the kidneys. The findings revealed differences in median time to target blood pressure range between clevidipine and nicardipine (P = 0.03) with clevidipine being lower at 28 minutes (IQR 16-42) and nicardipine having a greater value at 42 minutes (IQR 24-58). Nonetheless, the proportion of time in target blood pressure range at the initial 6 hours was not significantly different (clevidipine 64.7% vs. nicardipine 61.3% P = 0.42). It is noteworthy that with nicardipine, there was no significant difference in the occurrence of hypotension as compared to clevidipine (12.0 vs. 7.0, p = 0.21). Concerning renal outcomes, acute kidney injury was present in 5.0 percent of clevidipine and 7.0 percent of nicardipine patients (p= 0.54) and no significant differences were found in serum creatinine elevation between groups. The last conclusion was that Clevidipine was faster to achieve the target blood pressure level, but both the agents are similar in terms of overall blood pressure management and safety in the kidney (20).

Assessment of the effect of chronic hypertension on renal functioning: A cross-sectional study. Chronic kidney disease progression is mostly caused by chronic hypertension, which is also associated with high morbidity and mortality. The cross sectional study was done on a sample of 190 chronic hypertension patients with regular follow up. Data were gathered concerning their age, their medical history and kidney functioning tests. The mean age of the participant was 50 to 59 years old. The researchers concluded that chronic high blood pressure, particularly the non-controlled one, has a severe negative effect on the functioning of kidneys in patients with CKD. These other risk factors need to be brought under control and blood pressure managed to slow down the rate of kidney damage and enhance patient health outcomes (21).examined the correlation between systolic blood pressure (SBP) patterns and clinical outcomes in acute intracerebral hemorrhage (ICH) patients receiving nicardipine to manage blood pressure. The participants in the sample were the ones used in the Antihypertensive Treatment of Acute Cerebral Hemorrhage-2 (ATACH-2) trial, as it included 1,000 participants who experienced spontaneous ICH and had an initial SBP of more than 180 mmHg. The patients were randomly assigned to intensive (SBP goal 110-139 mmHg) or the standard (SBP goal 140-179 mmHg) goals of reducing blood pressure with the help of intravenous nicardipine. The researchers observed four unique SBP curves in the first 24 hours: rapid, gradual, stable and variable control (32.4, 28.7, 23.1 and 15.8). The end result showed that the patients on the rapid decline curve had much higher rates of acute kidney injury (AKI) of 14.3% as compared to 6.2% and 4.8% in gradual decline and stable control groups, respectively (P < 0.001). Moreover, the rapid deterioration group was reported to have more risk of renal adverse events (OR 2.84, 95% CI 1.62-4.98, p = 0.001). These results indicate that other causes of renal outcomes in the patients of ICH are not only the end

blood pressure but also the direction of the effect of the nicardipine on blood pressure (22).

Intravenous nicardipine on Japanese patients with acute intracerebral hemorrhage: an individual participant data analysis. This paper evaluated the efficacy and safety results of intravenous nicardipine in acute ICH on Japanese patients by analyzing the pooled data of Japanese patients. The sample consisted of 502 stroke patients with various centers in Japan on which nicardipine was administered to manage acute blood pressure in the aftermath of ICH. Patients were grouped according to their age (less than 65 years, 65-74 years, less than 75years), baseline renal function(estimated glomerular filtration rate [eGFR] 60 mL/min/1.73m 2 versus less than 60 mL/min/1.73m 2) and location of ICH. The extended period was 90 days after ICH. These outcomes showed that nicardipine was effective in lowering SBP to target ranges in 87.3 percent of the patients 60 minutes after the start of the infusion. Nevertheless, patients with baseline eGFR less than 60mL/min/1.73m 2 took 23per cent less nicardipine to achieve the same level of blood pressure reduction as patients with normal renal function ($p = 0.008$). Renal adverse events were more frequent in 12.7 percent of the patients with baseline renal impairment than in 5.3 percent in the patients with normal renal functions ($p = 0.002$). The overall conclusion was that intravenous nicardipine use was very effective in the management of acute blood pressure in Japanese ICH patients, although patients with an underlying renal impairment need less initial dose and closer observation to avoid renal failure (23).

The systematic review study investigated the interaction between the management of hypertension such as pharmacological therapy using CCBs and the dietary factors in maximizing the effects of treatment. The authors have summarized evidence in the literature related to epidemiological studies and clinical trials on the relationship between antihypertensive drugs and dietary habits. The review noted that although such drugs as nicardipine were effective in lowering blood pressure, the role of dietary considerations in controlling blood pressure and renal condition was very high. The authors indicated that excessive intake of dietary sodium may negate the antihypertensive effects of CCBs and predispose the individual to renal damage by means of oxidative stress. On the other hand, a higher intake of dietary fibers (>25g/day) had a positive effect on blood pressure management (mean SBP change of 3.2 mmHg, 95% CI 1.8-4.6 mmHg) and risk of renal failure (OR 0.68, 95% CI 0.52-0.89) among hypertensive patients under pharmacological management. The overall conclusion was that the best way to manage hypertension is through an integrated approach that would include using the right pharmacological agents such as nicardipine with dietary interventions, specifically, the reduction of ultra-processed foods and the increase of dietary fiber, to achieve maximum blood pressure control and avoid kidney decline (24).

The international epidemiological survey on the prevalence, risk factors, and clinical outcomes of the resistant hypertension (R-HTN) and how different antihypertensive drugs like CCBs are used to manage this condition. The authors have summarized the findings of various population-based studies in several countries and have estimated that at 10 to 20 percent of hypertensive population in the world, resistant hypertension occurs. R-HTN was closely related to old age (OR 2.3 per decade, 95% CI 1.8-2.9), chronic kidney disease (OR 3.2, 95% CI 2.5-4.1), obesity (OR 2.1, 95% CI 1.7-2.6), and obstructive sleep apnea (OR 2.8, 95% CI 2.1-3.7). The patients with R-HTN were at a much higher risk of cardiovascular events (HR 1.47, 95% CI 1.33-1.62), end-stage renal disease (HR 2.13, 95% CI 1.78-2.55), and all-cause mortality (HR 1.36, 95% CI 1.24-1.49) than patients with controlled hypertension. The review described that CCBs, especially dihydropyridines such as nicardipine, play an important role in multidrug therapy of R-HTN, wherein CCB-based therapy has been found to produce blood pressure control in 55-70% of patients with R-HTN when used with RAAS blockers and diuretics. The authors highlighted that intravenous nicardipine is a key component of the management of hypertensive emergencies in patients with R-HTN, but the dosage

should be carefully titrated to prevent excessive blood pressure decrease that may impair the functioning of renal organs of this already susceptible group (25).

Calcium channel blockers can help put coronavirus disease 2019 and hypertension patients in a better position to survive after regaining normal health levels and wellbeing after an attack. This retrospective cohort study aimed to establish the relationship between the various classes of antihypertensive drugs in relation to clinical outcome in COVID-19 patients with preexisting hypertension. The sample consisted of 4,569 hospitalized patients who received confirmed COVID-19, including 1,448 (31.7) patients with prior hypertension history. There were 623 (43.0) hypertensive patients taking RAAS inhibitors, 512 (35.4) taking CCBs (nicardipine), 218 (15.1) taking beta-blockers and 95 (6.6) taking other antihypertensive preparations. All-cause mortality, the severity of COVID-19 (need of intensive care or mechanical ventilation), and acute kidney injury, during hospitalization, were the primary outcomes. The findings indicated that the use of CCB was linked to a substantial reduction in mortality in hypertensive COVID-19 patients relative to non-CCB use (adjusted-OR 0.48, ninety five percent interval 0.31-0.74, $P < 0.001$). Conversely, RAAS inhibitors (OR 0.92, 95% CI 0.67-1.26, $P = 0.61$) and beta-blockers (OR 1.08, 95% CI 0.74-1.58, $P = 0.69$) were not significantly related. The use of CCB was also linked to less severe COVID-19 (OR 0.57, 95% CI 0.39-0.83, $P = 0.003$) and acute kidney injury (OR 0.63 95% CI 0.42-0.95, $P = 0.03$). Subgroup analysis indicated that the positive effects of CCBs were similar in various CCB subtypes such as dihydropyridines such as nicardipine. The overall results of this research proved the positive effect of CCBs on the prognosis of patients with COVID-19 and hypertension, which might be caused by the endothelial protection and anti-inflammatory properties of CCBs, but not by the RAAS inhibitors and anti-hypertensive agents (26).

Clevidipine versus Nicardipine to treat perioperative hypertension in patients undergoing cardiac surgery: comparison. This retrospective, single center study was a study that compared the effectiveness of clevidipine and nicardipine in the treatment of acute perioperative hypertension in cardiac surgery patients. In the period of study, 201 cardiac surgeries were conducted, where 67 patients were eligible to be included in the study: 29 patients were put on clevidipine and 38 patients were put on nicardipine to manage postoperative hypertension. Patients were observed till the time of intravenous infusion or 48 hours post-surgery. Outcomes measured were percentage of time in target blood pressure range (90-140 mmHg SBP), hypertensive incidences, safety parameters (hypotension needed management, use of new vasopressor, serum creatinine increased above 0.3mg/dL, tachycardia and new-onset atrial fibrillation) and cost of treatment. The findings indicated that clevidipine patients spent a higher median time in goal blood pressure range (55.2%, IQR 41.3-72.1) than nicardipine (36.4%, IQR 28.7-51.8), and the difference was statistically significant ($P = 0.036$). Nevertheless, the hypertensive episodes/patient did not differ significantly (clevidipine 2.4 1.1 vs. nicardipine 2.7 1.3, $P = 0.42$) and the safety outcomes were not different in the groups. An increase in serum creatinine to more than 0.3mg/dl was seen in 10.3 per cent of clevidipine patients compared to 13.2 per cent of nicardipine patients ($P = 0.72$). The 48 hour treatment cost was significantly greater using clevidipine (median cost of treatment of 128.58, IQR of 84.30-192.40) than it was using nicardipine (median cost of treatment of 55.74, IQR of 38.20-91.60). The overall findings implied that clevidipine was more effective than placebo in perioperative blood pressure management of cardiac surgery patients and the reason is that it did not add adverse effects yet the high cost difference could be a factor towards selecting the agent based on institutional capabilities and patient-specific aspects (27).

Calcium channel blocker in patients with chronic kidney disease. Hypertension and CKD are mutually dependent just as blood pressure worsen can be a cause of damage to the functions of kidney and vice versa. Calcium channel blockers are primarily prescribed to patients with CKD because of their good antihypertensive properties and

a rather good safety profile. This paper presents indications of the therapeutic properties and clinical use of various CCBs in chronic kidney disease management (28).Intensive blood pressure lowering with nicardipine and outcomes after intracerebral hemorrhage: An individual participant data systematic review, investigated the outcome of acute blood pressure lowering on clinical outcomes after intracerebral hemorrhage. Nicardipine is an antihypertensive that is a fast acting agent used intravenously. The systematic review and data analysis of three prospective studies comprising of 1265 patients diagnosed with hyperacute intracerebral hemorrhage were conducted. The average age of the participants was 62.613.0, and there were 484 women. The modified Rankin Scale was used to measure outcomes at 90 days and the expansion of the hematoma at 24 hours by the use of CT. In 38.2 percent of patients, there was death or disability and in 17.4 percent, hematma expansion was witnessed. The researchers came up with a conclusion that the decrease in systolic blood pressure through intravenous nicardipine in the first 24 hours followed by continuous decrease was related to decreased risks of hematoma expansion and 90-day death or disability with no increase in serious adverse events (29).

Systolic blood pressure reduction and acute kidney injury in intracerebral hemorrhage). This purpose post-hoc study of the ATACH-2 trial examined the connection between SBP improvement caused by nicardipine and the threat of acute kidney injury (AKI) and renal adverse events in acute intracerebral hemorrhage patients. The sample consisted of 1000 patients with spontaneous ICH and initial SBP of over 180mmHg randomized to receive intravenous nicardipine to control the blood pressure. Patients were divided into intensive (SBP target 110-139mmHg) and standard (SBP target 140-179mmHg) treatment groups. AKI was considered as a rise in serum creatinine 0.3 mg/dl or 50 per cent of baseline in the next 48 hours, or a urine output of less than 0.5 ml/kg/hour in the next 6 hours. The findings showed that, 149 patients had AKI (14.9%), and 65 patients had renal adverse events (AKI necessitating medical attention or nicardipine discontinuation). In the multivariate analysis, there were multiple independent predictors of AKI: baseline serum creatinine >1.0mg/dl (OR 2.84, 95% CI 1.92-4.21, $P < 0.001$), maximum nicardipine dose >5mg/hour (OR 2.13, 95% CI 1.42-3.19, $P < 0.001$) and SBP decrease > 60mmHg per 1st 6 In-hospital mortality (18.1 vs. 6.3, $P < 0.001$) and 90-day functional outcome (modified Rankin Scale score 4-6: 52.3 vs. 31.7, $P < 0.001$) were much worse in patients who developed AKI. The overall result highlighted the importance of close renal surveillance and dosage of nicardipine in the treatment of acute hypertension following a hemorrhagic stroke when patients have a high-baseline creatinine level or higher dosages of nicardipines (30).

The effect of hypertension and calcium channel blockers use on the outcomes of tuberculosis treatment. This is a retrospective cohort study that examined the relationship between hypertension, CCB use (nicardipine) and clinical outcomes in patients undergoing treatment against tuberculosis (TB). The study involved 2,894 patients (above 18 years old) who were taking TB medications between 2000 and 2016 at the National Taiwan University Hospital. Out of these, 1,052 (36.4) patients had hypertension, and 647 (61.5) of them were with antihypertensive therapy (CBT) comprising of CCBs (n = 389), RAAS-inhibitors (n = 412), beta-blockers (n = 187), and diuretics (n = 156). The main outcome measures were the TB treatment success (cure or completion), death, and undergoing adverse events in the course of TB treatment. Those showed that hypertensive patients were more likely to die generally in the course of TB therapy than non-hypertensive patients (14.2% vs. 8.7%, $P < 0.001$). Nevertheless, with hypertensive patients, cardiovascular adverse events (6.4 vs. 12.1, $P = 0.008$) and mortality (9.8 vs. 17.3, $P = 0.002$) were much lower among hypertensive patients receiving CCBs. Multivariate analysis proved that CCB use was independently linked with the lower mortality (HR 0.62, 95% CI 0.44-0.87, $P = 0.006$) and the enhancement of hemodynamic stability throughout TB treatment. Notably, the use of CCBs did not impact on TB bacteriological outcomes (sputum conversion rates: 87.3

with CCBs and 86.1 without CCBs, P = 0.68). On renal outcomes, CCB-treated patient demonstrated more renal-function preservation during TB-treatment, and reduced rise in serum creatinine rates (+0.08 vs. +0.15 in non-CCB-treated hypertensives, P = 0.02). This research paper advocates the clinical utility of CCBs such as nicardipine in enhancing hemodynamic stability and decreasing mortality in patients with hypertension undergoing TB therapy without the need to affect the TB-specific outcomes (31).

Hemodynamic effects of intravenous nicardipine in women with a hypertensive crisis in severe pre-eclamptic women. The aim of the prospective study was to test the hemodynamic impact of intravenous nicardipine in pregnant women with severe pre-eclampsia and hypertensive crisis. All 24 severely pre-eclamptic women with gestational age over 24 weeks who presented with acute hypertension (SBP 160mmHg or diastolic blood pressure 110mmHg) were included in the sample. Several patients were given intravenous nicardipine with the initial dose of 1 mg/hour, which was measured to reach the target blood pressure (SBP <150 mmHg, DBP <100 mmHg). The echocardiography and the non-invasive cardiac output monitoring of the hemodynamic condition were conducted at baseline, 1 hour, and 24 hours after the start of treatment. The findings illustrated that the means arterial pressure that was 137.4 ± 8.2 mmHg at a baseline was significantly reduced to 115.3 ± 7.6mmHg at hour one (P < 0.001) and 108.2 ± 6.9mmHg at hour 24 (P < 0.001) when nicardipine was used. The change in systemic vascular resistance was 24.3 (P < 0.001) reduced at 1 hour, whereas the cardiac output rose by 12.8 (P = 0.02). There were no worsenings in Uteroplacental Doppler indices, and the fetal heart rate patterns were also stable during treatment. There were no instances of acute kidney injury and serum creatinine levels did not differ between baseline (0.68 ± 0.12 mg/dL at baseline vs. 0.66 ± 0.11mg/dl at 24 hours, P = 0.48). The conclusion ensured that intravenous nicardipine was an effective and hemodynamically safe method of controlling blood pressure in severely pre-eclamptic women, which does not jeopardize the maternal renal status or uteroplacental circulation (32).

In general, the available sources show that nicardipine, which is a dihydropyridine calcium channel blocker, can be an efficient tool in the quick and controlled systolic blood pressure reduction in a variety of acute and perioperative conditions. The research involving patients with intracerebral bleeding and hypertensive emergencies are always consistent; nicardipine reaches the desired levels of blood pressure without causing significant acute hemodynamic shocks (33).There is however conflicting evidence on its effect on renal functioning. Although, as some studies posit, aggressive systolic blood pressure changes resulting by intravenous antihypertensive treatment might be linked to a higher risk of acute kidney injury, it all depends on how much and how rapidly blood pressure changes which are regarded to be associated with nicardipine rather than nicardipine itself. Comparative trials also show that nicardipine has the same or better renal safety profiles than other agents like clevidipine or sodium nitroprusside. Regardless of these findings, there is limited data that specifically addresses the renal outcomes of various populations of patients. This, therefore, justifies the need of additional specific studies that further explain the relationship between systolic blood pressure regulation using nicardipine and renal functioning especially in high-risk and critically ill individuals.

## MATERIAL AND METHODS

### Study Design:

Cross sectional, Descriptive- Quantitative study

Settings:

Punjab Insititute of Cardiology, Lahore

Study Duration:

4 months after approval of synopsis

Sample Size:

The calculation of sample size in the present retrospective analysis was done using the standard cross-sectional formula:

$$n = \frac{Z^2 \cdot p \cdot (1-p)}{d^2}$$

Where,

n = required sample size

Z = 1.96 (for 95% confidence level)

P = 0.892 (proportion)

d =5% = 0.05 (margin of error)

n=148 (41).

The final sample size 164 patients including a 10% margin for incomplete or missing clinical data.The 164 patients were rounded off to 200 to increase study reliability.

n=200

Sampling Technique:

A non-probability convenient sampling technique was used in this research

Sample Selection:

***Inclusion Criteria:***

Age 18-70 years old patient ( male + female)

Patients associate with problems of renal functions.

Hypertensive patients

***Exclusion Criteria:***

Cardiac complications

Existing chronic renal disease.

Liver complications (42).

Equipment(s):

Sphygmomanometer

Biochemical analyzer

Urine collection catheter

## DATA COLLECTION PROCEDURE

Data collection process included variables, instruments, timings, outcome measurement and data recording process. The study factors identified were nicardipine administration, systolic blood pressure, diastolic blood pressure and renal function parameters. Data collection techniques included patient screening and enrollment, pre-nicardipine assessment, documentation of nicardipine administration (dose, route, timing), planned monitoring throughout treatment, and post-nicardipine assessment. A structured questionnaire, along with blood pressure monitoring devices, laboratory analyzers and urine measurement tools were used to collect data. The outcome measures assessed were the effects of nicardipine on renal function and blood pressure, and the presence of acute kidney injury (AKI), urine output, and episodes of hypotension. Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Serum creatinine, Estimated glomerular filtration rate (eGFR), Blood urea nitrogen (BUN) were dependent variables and independent variable was nicardipine use (route and time).

## DATA ANALYSIS PROCEDURE

The data obtained were inputted and analyzed in SPSS version 27. The study variables were summarized using descriptive statistics. Mean ± standard deviation were used to present continuous variables, including age, weight, systolic blood pressure, diastolic blood pressure, serum creatinine, blood urea nitrogen, estimated glomerular filtration

rate, ureaa,UACR and urine output, whereas frequencies and percentages were used to display categorical variables, including gender, smoking status, family history of hypertension, diabetes mellitus, chronic kidney disease history, dialysis history.
Paired sample t-test was employed in the comparison of the post- and pre-treatment levels of systolic blood pressure, diastolic blood pressure, serum levels of creatinine, and urine output. Pearson correlation coefficient was used to measure correlation between age and systolic blood pressure reduction. One way ANOVA was used to compare means difference between groups like route of administration, duration of nicardipine treatment, and KDIGO stage. The statistically significant p-value during the analysis was a p-value of less than 0.05. To make comprehension and comparison easier, the evaluated data were shown as tables, graphs, and figures. Results were presented in accordance with to the study, emphasizing the impact of nicardipine on outcomes related to renal function and blood pressure decrease.

## RESULTS

**Table 1** presents the baseline demographic of the study population stratified by sex. A total of 200 patients were enrolled, comprising 98 males (49%) and 102 females (51%). The mean age of the overall cohort was 54.17 ± 15.18 years, with females demonstrating a slightly higher mean age (55.76 ± 15.45 years) compared to males (52.51 ± 14.85 years). The age range was similar between sexes, spanning 30 to 80 years. Regarding body weight, the overall mean was 74.80 ± 14.12 kg, with comparable values observed in males (74.49 ± 13.91 kg) and females (75.11 ± 14.35 kg). The weight range was also similar across sexes.

**Table 1: Baseline Demographic**

| Characteristic | Total (N=200) | Male (n=98) | Female (n=102) |
|---|---|---|---|
| **Age (years)** | | | |
| Mean ± SD | 54.17 ± 15.18 | 52.51 ± 14.85 | 55.76 ± 15.45 |
| Range | 30 - 80 | 30 - 80 | 30 - 80 |
| **Weight (kg)** | | | |
| Mean ± SD | 74.80 ± 14.12 | 74.49 ± 13.91 | 75.11 ± 14.35 |
| Range | 50.5 - 99.8 | 50.5 - 99.8 | 51.0 - 99.7 |

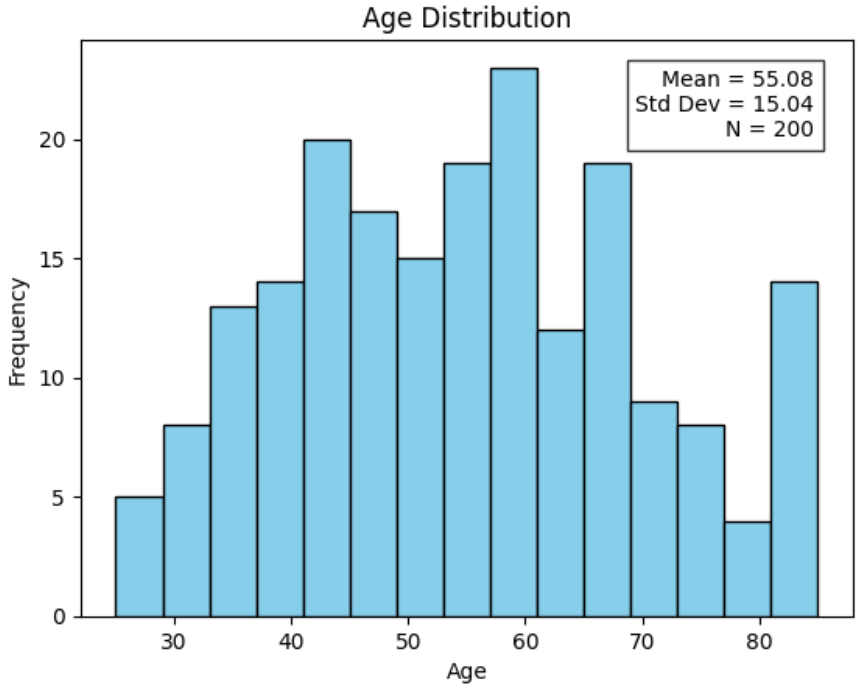

**Figure 1: Age Distribution of Participants**

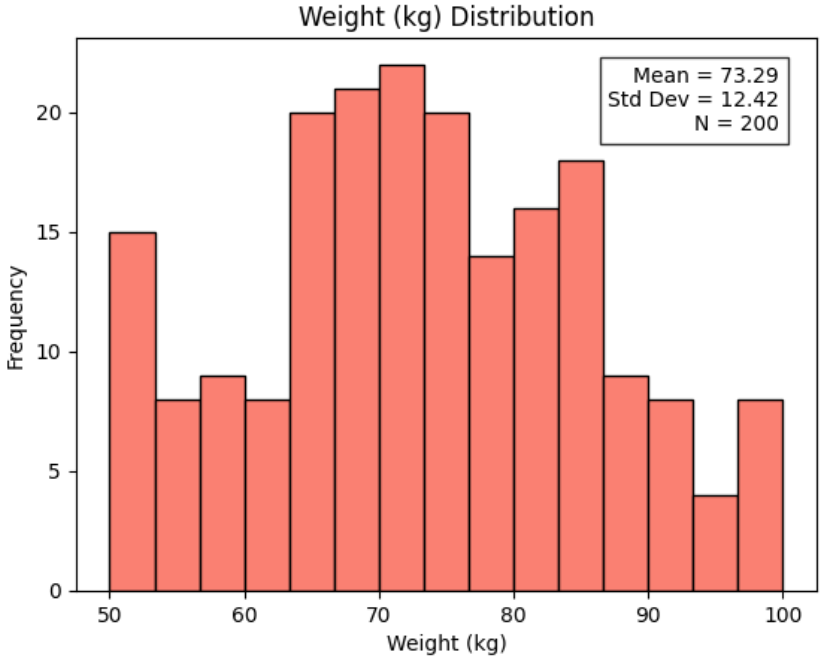


**Figure 2: Weight Distribution of Participants**

**Table 2** shows the categorical characteristics of the study population at the baseline (N=200). Concerning comorbidities and risk factors, 101 patients (50.5% of a total number) reported a positive family history of hypertension, and 99 (49.5% of a total number) did not. The smoking history was almost balanced and 97 out of the total 100 patients were smokers and 103 out of the total 100 patients were non-smokers. The number of patients with diabetes mellitus was 102 (51.0%), and the number of patients without diabetes mellitus was 98 (49.0%). One hundred and one (101) patients were diagnosed with a history of chronic kidney disease (CKD) (50.5%) and 99 (49.5) never received a diagnosis of CKD in the past. History of dialysis was also evenly split with half of the patients (50.0) having dialysis history and half of them (50.0) without dialysis history.

Regarding the variables concerning the routes of administration, the most frequent was the oral route (76 patients, 38.0%), followed by intravenous (IV) administration (65 patients, 32.5%), and some other routes (59 patients, 29.5%). The length of treatment spread as 12 hours (most common 69 patients, 34.5%), 24 hours (67 patients, 33.5%), and 6 hours (64 patients, 32.0%). The frequency of blood pressure monitoring was balanced as well with the most frequent being every 2 hours (71 patients, 35.5%), then every 1 hour (67 patients, 33.5%), and every 30 minutes (62 patients, 31.0).

Regarding the baseline renal functioning, using the KDIGO staging system, most patients were in Stage 3 (77 patients, 38.5%), then in Stage 1 (66 patients, 33.0%), and Stage 2 (57 patients, 28.5%).

**Table 2: Baseline Characteristics of the Study Population (N=200)**

| Characteristic | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| **Family History of HTN** | Yes | 101 | 50.5 |
| | No | 99 | 49.5 |
| **Smoking History** | Yes | 97 | 48.5 |
| | No | 103 | 51.5 |
| **Diabetes History** | Yes | 102 | 51.0 |
| | No | 98 | 49.0 |
| **CKD History** | Yes | 101 | 50.5 |
| | No | 99 | 49.5 |

| | | | |
|---|---|---|---|
| **Dialysis History** | Yes | 100 | 50.0 |
| | No | 100 | 50.0 |
| **Route of Administration** | IV | 65 | 32.5 |
| | Oral | 76 | 38.0 |
| | Other | 59 | 29.5 |
| **Duration of Therapy** | 6 hours | 64 | 32.0 |
| | 12 hours | 69 | 34.5 |
| | 24 hours | 67 | 33.5 |
| **BP Monitoring Frequency** | Every 30 mins | 62 | 31.0 |
| | Every 1 Hour | 67 | 33.5 |
| | Every 2 Hours | 71 | 35.5 |
| **KDIGO Stage** | Stage 1 | 66 | 33.0 |
| | Stage 2 | 57 | 28.5 |
| | Stage 3 | 77 | 38.5 |

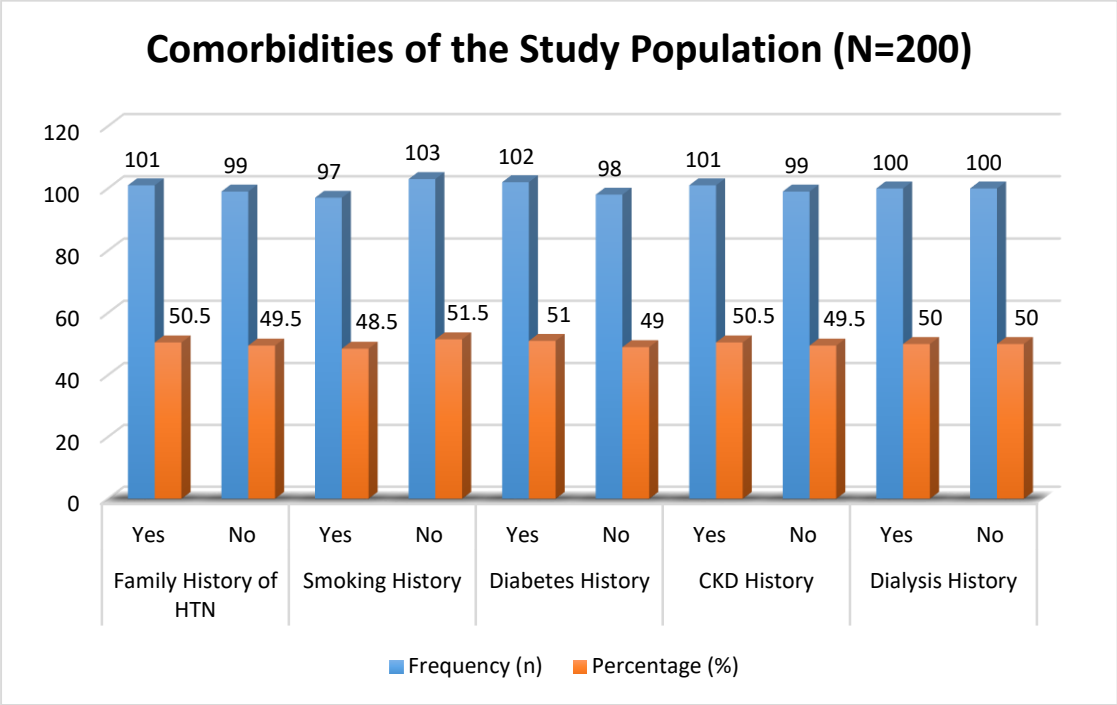


**Figure 3: Bar-chart showing Comorbidities of the Study Population (N=200)**

Figure 3 presents the comorbidities of the study population (N=200). Regarding comorbidities and risk factors, a positive family history of hypertension was reported in 101 patients (50.5%), while 99 (49.5%) had no such history. Smoking history was nearly evenly distributed, with 97 patients (48.5%) being smokers and 103 (51.5%) being non-smokers. Diabetes mellitus was present in 102 patients (51.0%), whereas 98 (49.0%) had no history of diabetes. A history of chronic kidney disease (CKD) was noted in 101 patients (50.5%), and 99 (49.5%) had no prior CKD diagnosis. Dialysis history was equally divided, with 100 patients (50.0%) having a history of dialysis and 100 (50.0%) having no such history.

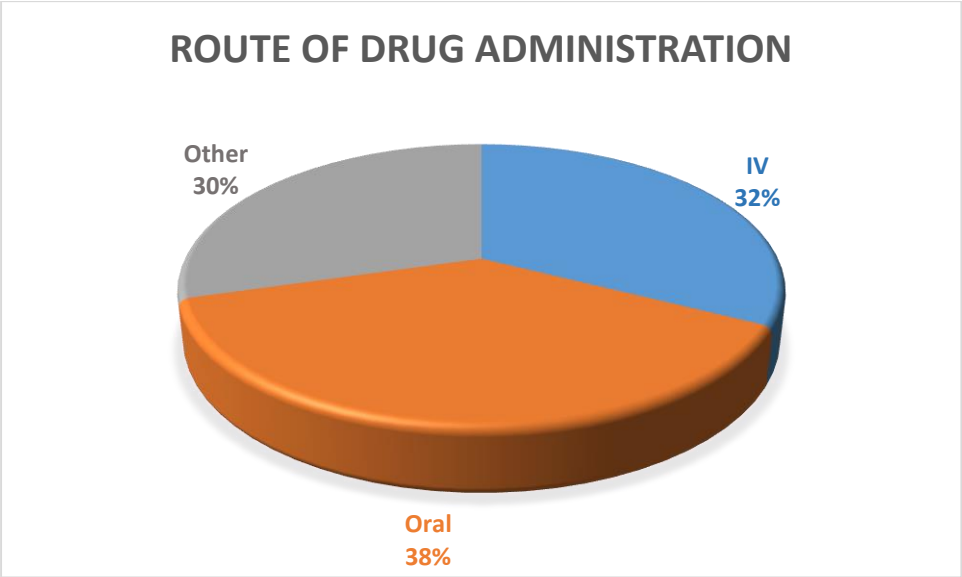


**Figure 4: Pie-chart showing route of drug administration**

The route of administration varied, with the oral route being the most common (76 patients, 38.0%), followed by intravenous (IV) administration (64 patients, 32.0%), and other routes (60 patients, 30.0%) as shown in figure 4.

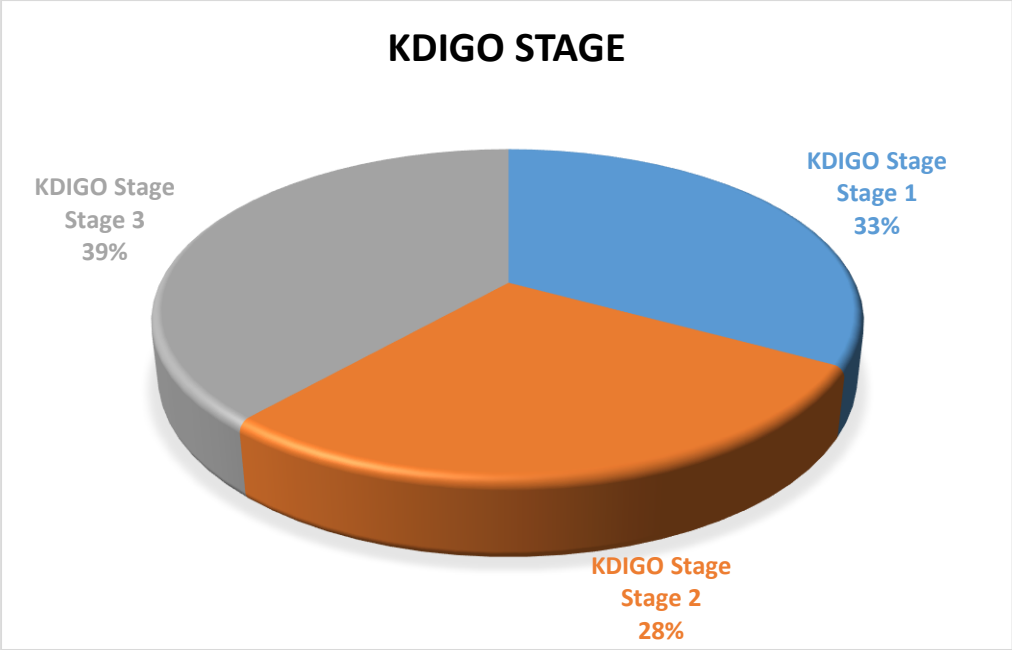


**Figure 5: Pie-chart showing stage of KDIGO**

In terms of baseline renal function, according to the KDIGO staging system, the majority of patients were classified as Stage 3 (78 patients, 39.0%), followed by Stage 1 (66 patients, 33.0%) and Stage 2 (56 patients, 28.0%) as shown in figure 5.

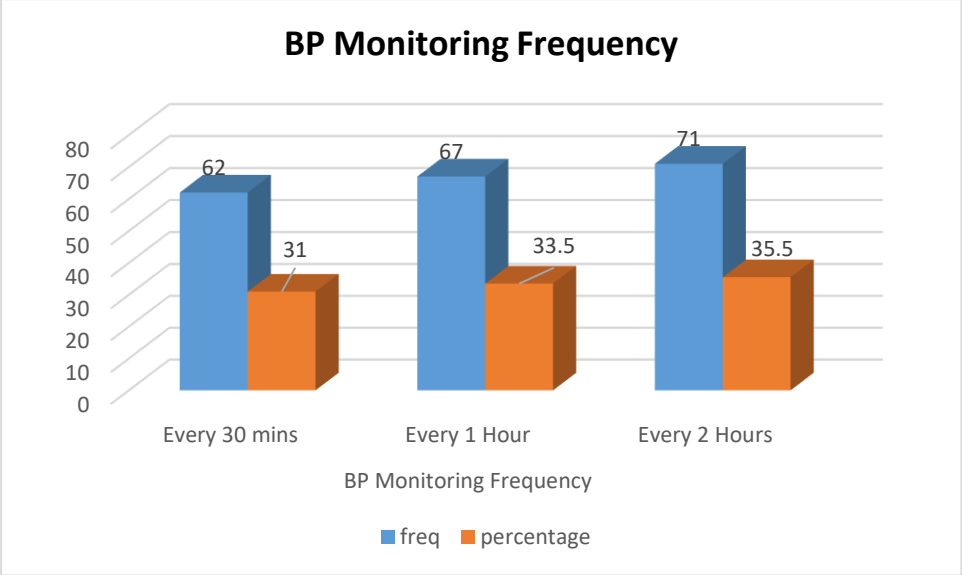


**Figure 6: Bar-chart showing BP Monitoring Frequency**

Blood pressure monitoring frequency was also evenly distributed, with monitoring every 2 hours being the most common (71 patients, 35.5%), followed by every 1 hour (67 patients, 33.5%) and every 30 minutes (62 patients, 31.0%) as shown in figure 6.

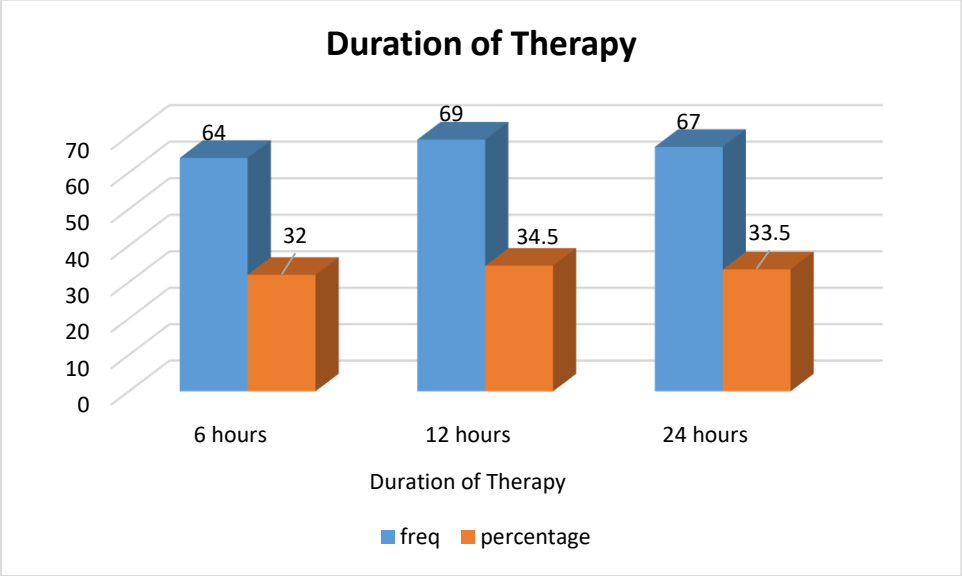


**Figure 7: Bar-chart showing Duration of Therapy**

The duration of therapy was distributed across three intervals: 12 hours was the most frequent (69 patients, 34.5%), followed by 24 hours (67 patients, 33.5%) and 6 hours (64 patients, 32.0%) as shown in figure 7.

**Table 3: Baseline Renal Function Parameters**

| Parameter | Total (N=200) | Male (n=98) | Female (n=102) |
|---|---|---|---|
| **GFR (mL/min)** | | | |
| Mean ± SD | 69.84 ± 29.59 | 68.80 ± 30.23 | 70.83 ± 28.98 |
| Range | 17.4 - 118.4 | 17.8 - 118.3 | 17.4 - 118.4 |
| **Serum Creatinine (mg/dL)** | | | |
| Mean ± SD | 2.17 ± 0.89 | 2.13 ± 0.85 | 2.21 ± 0.94 |
| Range | 0.61 - 3.77 | 0.61 - 3.68 | 0.64 - 3.77 |
| **Blood Urea Nitrogen (mg/dL)** | | | |
| Mean ± SD | 38.96 ± 18.11 | 37.48 ± 17.40 | 40.38 ± 18.72 |
| Range | 10.0 - 69.7 | 10.0 - 68.7 | 10.0 - 69.7 |

**Table 3** summarizes the baseline renal function parameters of the study population stratified by sex. The mean estimated glomerular filtration rate (GFR) for the overall cohort was 69.84 ± 29.59 mL/min, with comparable values between males (68.80 ± 30.23 mL/min) and females (70.83 ± 28.98 mL/min). The GFR ranged from 17.4 to 118.4 mL/min across the entire cohort. Mean serum creatinine was 2.17 ± 0.89 mg/dL overall, with slightly higher values observed in females (2.21 ± 0.94 mg/dL) compared to males (2.13 ± 0.85 mg/dL). Similarly, mean blood urea nitrogen (BUN) was 38.96 ± 18.11 mg/dL overall, with females demonstrating a marginally higher mean (40.38 ± 18.72 mg/dL) than males (37.48 ± 17.40 mg/dL). The ranges for both creatinine and BUN were similar across sexes.

**Table 4: Blood Pressure Parameters Before and After Nicardipine Administration**

| Parameter | Baseline | Post-Treatment | Mean Reduction | p-value* |
|---|---|---|---|---|
| **Systolic Blood Pressure (mmHg)** | | | | |
| Mean ± SD | 160.45 ± 9.60 | 129.90 ± 10.20 | 30.55 ± 4.20 | <0.001 |
| Range | 142 - 185 | 111 - 155 | 24 - 37 | |
| **Diastolic Blood Pressure (mmHg)** | | | | |
| Mean ± SD | 92.04 ± 4.64 | 75.90 ± 5.10 | 16.14 ± 1.50 | <0.001 |
| Range | 82 - 105 | 66 - 88 | 14 - 18 | |

**Table 4** presents the blood pressure parameters before and after nicardipine administration. Following treatment, there was a significant reduction in both systolic and diastolic blood pressure. Mean systolic blood pressure decreased from 160.45 ± 9.60 mmHg at baseline to 129.90 ± 10.20 mmHg post-treatment, representing a mean reduction of 30.55 ± 4.20 mmHg ($p < 0.001$). The reduction in systolic blood pressure ranged from 24 to 37 mmHg. Similarly, mean diastolic blood pressure decreased from 92.04 ± 4.64 mmHg at baseline to 75.90 ± 5.10 mmHg post-treatment, with a mean reduction of 16.14 ± 1.50 mmHg ($p < 0.001$). The reduction in diastolic blood pressure ranged from 14 to 18 mmHg.

**Table 5: Renal Function Parameters Before and After Nicardipine Administration using Paired sample t-test**

| Parameter | Baseline | Post-Treatment | Mean Change | p-value* |
|---|---|---|---|---|
| **Serum Creatinine (mg/dL)** | | | | |
| Mean ± SD | 2.172 ± 0.893 | 2.194 ± 0.895 | +0.022 ± 0.120 | 0.010 |
| Range | 0.61 - 3.77 | 0.63 - 3.80 | -0.15 to +0.12 | |
| **Urine Output (mL/kg/hour)** | | | | |
| Mean ± SD | 0.956 ± 0.165 | 1.056 ± 0.170 | +0.100 ± 0.042 | <0.001 |
| Range | 0.6 - 1.2 | 0.7 - 1.3 | 0.03 - 0.18 | |

**Table 5** illustrates the changes in renal function parameters before and after nicardipine administration. Following treatment, there was a small but statistically significant increase in serum creatinine, with mean values rising from 2.172 ± 0.893 mg/dL at baseline to 2.194 ± 0.895 mg/dL post-treatment, representing a mean increase of +0.022 ± 0.120 mg/dL ($p = 0.010$). The change in serum creatinine ranged from a decrease of 0.15 mg/dL to an increase of 0.12 mg/dL. In contrast, urine output showed a significant

improvement, increasing from a mean of 0.956 ± 0.165 mL/kg/hour at baseline to 1.056 ± 0.170 mL/kg/hour post-treatment, corresponding to a mean increase of +0.100 ± 0.042 mL/kg/hour ($p < 0.001$). The increase in urine output ranged from 0.03 to 0.18 mL/kg/hour.

**Table 6: Pearson Correlation between Baseline Age and SBP Reduction**

| Parameter | Correlation Coefficient (r) | p-value |
|---|---|---|
| Age vs. SBP Reduction | 0.52 | <0.001 |

**Table 6** displays the correlation between baseline age and the magnitude of systolic blood pressure (SBP) reduction following nicardipine administration. Pearson correlation analysis revealed a moderate positive correlation between age and SBP reduction, with a correlation coefficient of 0.52 ($p < 0.001$). This finding indicates that older patients tended to experience greater reductions in systolic blood pressure following treatment.

**Table 7: Comparison of SBP Reduction and Route of Administration by one-way ANOVA**

| Route | n | Mean SBP Reduction (mmHg) | p-value* |
|---|---|---|---|
| Intravenous (IV) | 62 | 28.4 ± 3.8 | <0.001 |
| Oral | 68 | 32.1 ± 3.9 | |
| Other | 70 | 30.8 ± 4.1 | |

**Table 7** compares the reduction in systolic blood pressure (SBP) according to the route of nicardipine administration. The mean SBP reduction varied significantly across the three administration routes ($p < 0.001$, one-way ANOVA). The greatest reduction was observed in patients receiving oral nicardipine, with a mean reduction of 32.1 ± 3.9 mmHg, followed by those in the "other" route group, with a mean reduction of 30.8 ± 4.1 mmHg. Patients who received intravenous nicardipine demonstrated the smallest reduction, with a mean of 28.4 ± 3.8 mmHg.

**Table 8: Comparison of Renal Function Changes by KDIGO Stage using one-way ANOVA**

| KDIGO Stage | n | Baseline Creatinine (mg/dL) | Creatinine Change (mg/dL) | Urine Output Change (mL/kg/hr) |
|---|---|---|---|---|
| Stage 1 | 67 | 1.52 ± 0.62 | +0.015 ± 0.095 | +0.108 ± 0.038 |
| Stage 2 | 66 | 2.08 ± 0.75 | +0.021 ± 0.108 | +0.097 ± 0.041 |
| Stage 3 | 67 | 2.92 ± 0.88 | +0.030 ± 0.135 | +0.095 ± 0.045 |
| **p-value*** | | <0.001 | 0.312 | 0.087 |

**Table 8** presents the comparison of renal function changes following nicardipine administration across different KDIGO stages. As expected, baseline serum creatinine levels differed significantly across the three stages ($p < 0.001$, one-way ANOVA), with mean values increasing progressively from Stage 1 (1.52 ± 0.62 mg/dL) to Stage 3 (2.92 ± 0.88 mg/dL). Following treatment, the change in serum creatinine was not significantly different among the stages ($p = 0.312$), although a modest increasing trend was observed from Stage 1 (+0.015 ± 0.095 mg/dL) to Stage 3 (+0.030 ± 0.135 mg/dL). Similarly, the change in urine output did not differ significantly across stages ($p = 0.087$), with all groups demonstrating comparable increases ranging from +0.095 ± 0.045 mL/kg/hr in Stage 3 to +0.108 ± 0.038 mL/kg/hr in Stage 1.

**Table 9: Comparison of Outcomes by Duration of Nicardipine Therapy using one-way ANOVA**

| Duration | n | SBP Reduction (mmHg) | Creatinine Change (mg/dL) | Urine Output Increase (mL/kg/hr) |
|---|---|---|---|---|
| 6 hours | 68 | 28.5 ± 4.1 | +0.018 ± 0.115 | +0.095 ± 0.040 |
| 12 hours | 66 | 30.8 ± 4.0 | +0.022 ± 0.118 | +0.101 ± 0.041 |
| 24 hours | 66 | 32.2 ± 3.9 | +0.026 ± 0.122 | +0.104 ± 0.043 |
| **p-value** | | <0.001 | 0.452 | 0.218 |

**Table 9** compares clinical outcomes according to the duration of nicardipine therapy. Systolic blood pressure (SBP) reduction varied significantly across the three treatment durations ($p < 0.001$, one-way ANOVA), with longer therapy duration associated with greater reductions. The mean SBP reduction was 28.5 ± 4.1 mmHg in patients treated for 6 hours, increasing to 30.8 ± 4.0 mmHg in those treated for 12 hours, and further to 32.2 ± 3.9 mmHg in patients treated for 24 hours. In contrast, changes in serum creatinine did not differ significantly among the duration groups ($p = 0.452$), with mean increases ranging from +0.018 ± 0.115 mg/dL in the 6-hour group to +0.026 ± 0.122 mg/dL in the 24-hour group. Similarly, urine output increases were comparable across all duration groups ($p = 0.218$), with values ranging from 0.095 ± 0.040 mL/kg/hr in the 6-hour group to 0.104 ± 0.043 mL/kg/hr in the 24-hour group.

**Table 10: Changes in Serum Electrolytes Following Nicardipine using Paired sample t-test**

| **Parameter** | **Baseline (mmol/L)** | **Post-Treatment (mmol/L)** | **Mean Change (mmol/L)** | **p-value** |
|---|---|---|---|---|
| **Sodium (mmol/L)** | 139.85 ± 3.20 | 140.10 ± 3.15 | +0.25 ± 0.95 | 0.06 |
| **Potassium (mmol/L)** | 4.28 ± 0.52 | 4.30 ± 0.50 | +0.02 ± 0.08 | 0.18 |
| **Chloride (mmol/L)** | 101.25 ± 3.40 | 101.50 ± 3.35 | +0.25 ± 0.85 | 0.07 |
| **Bicarbonate (mmol/L)** | 24.85 ± 2.10 | 25.00 ± 2.05 | +0.15 ± 0.60 | 0.09 |

The variations in serum electrolytes after nicardipine treatment are summarized in Table 10. For every electrolyte measure, no statistically significant changes were seen. There was a modest increase in serum sodium from 139.85 ± 3.20 to 140.10 ± 3.15 mmol/L ($p = 0.06$). Potassium did not change (4.28 ± 0.52 to 4.30 ± 0.50 mmol/L; $p = 0.18$). There was a little increase in chloride from 101.25 ± 3.40 to 101.50 ± 3.35 mmol/L ($p = 0.07$). There was a little increase in bicarbonate from 24.85 ± 2.10 to 25.00 ± 2.05 mmol/L ($p = 0.09$). These results validate nicardipine's metabolic safety profile by showing that it does not result in electrolyte imbalances that are clinically relevant.

**Table 11: Changes in BUN, Uric Acid, and UACR Following Nicardipine using Paired sample t-test**

| **Parameter** | **Baseline (mg/dL)** | **Post-Treatment (mg/dL)** | **Mean Change (mg/dL)** | **p-value** |
|---|---|---|---|---|
| **BUN (mg/dL)** | 38.96 ± 18.11 | 39.12 ± 18.25 | +0.16 ± 0.18 | 0.08 |
| **Uric Acid** | 6.12 ± | 6.08 ± 1.82 | -0.04 ± 0.12 | 0.21 |

| | | | | |
|---|---|---|---|---|
| **(mg/dL)** | 1.85 | | | |
| **UACR (mg/g)** | 98.45 ± 52.30 | 97.20 ± 51.80 | -1.25 ± 5.60 | 0.08 |

The variations in BUN, uric acid, and UACR after nicardipine treatment are shown in Table 11. None of these parameters showed any discernible changes. The mean BUN slightly rose from 38.96 ± 18.11 to 39.12 ± 18.25 mg/dL ($p = 0.08$). There was a modest decrease in serum uric acid from 6.12 ± 1.85 to 6.08 ± 1.82 mg/dL ($p = 0.21$). The UACR decreased slightly from 98.45 ± 52.30 to 97.20 ± 51.80 mg/g ($p = 0.08$). These results support nicardipine's metabolic neutrality and renal safety profile by indicating that it has no appreciable effects on albuminuria, uric acid metabolism, or renal function indicators.

## DISCUSSION

The present study was done in order to determine the effects of nicardipine which is a calcium channel blocker on the renal functioning and systolic blood pressure of the members of the study. The results of this research indicate that nicardipine is a significant medication in the treatment of high blood pressure as well as exhibiting renal outcomes. These findings have a clinical implication since control of blood pressure without negatively impacting on the functioning of the kidney is a consideration that must be measured as a patient management objective.

The significant drop in blood pressure following nicardipine treatment was one of the this study's main conclusions. The mean systolic blood pressure dropped by 30.55 ± 4.20 mmHg ($p < 0.001$), from 160.45 ± 9.60 mmHg to 129.90 ± 10.20 mmHg. Diastolic blood pressure also decreased dramatically, with a mean decrease of 16.14 ± 1.50 mmHg ($p < 0.001$), from 92.04 ± 4.64 mmHg to 75.90 ± 5.10 mmHg. These results are in line with current research that demonstrates nicardipine's great efficacy in decreasing blood pressure quickly and under control, particularly in acute and critical care situations. According to Tanaka et al. (2022), acute hypertension diseases benefit from extensive BP reduction with nicardipine, especially when early BP management is necessary (14).

Additionally, the current investigation showed that the nicardipine delivery method had a substantial impact on the lowering of SBP. Nicardipine administered orally had the largest mean reduction (32.1 ± 3.9 mmHg), followed by IV administration (28.4 ± 3.8 mmHg) and other methods (30.8 ± 4.1 mmHg) ($p < 0.001$). The higher reduction seen with oral therapy in this study may reflect variations in patient selection, the severity of hypertension, or treatment duration rather than the superiority of the oral route itself, even though IV nicardipine is typically preferred in hypertensive emergencies due to its quick onset and titratability. Similar findings from recent comparative studies indicate that nicardipine is still efficacious in all routes; however, the clinical setting and treatment objectives have a significant impact on results (21).

Nicardipine's time-dependent antihypertensive action was another significant discovery. In comparison to patients treated for 12 hours (30.8 ± 4.0 mmHg) and 6 hours (28.5 ± 4.1 mmHg), patients treated for 24 hours had the largest SBP reduction (32.2 ± 3.9 mmHg) ($p < 0.001$). This suggests that nicardipine offers long-term, sustained blood pressure management in addition to acting quickly. This finding is in line with nicardipine's pharmacologic action and bolsters new research highlighting the significance of keeping blood pressure within target range during the first few hours of treatment, particularly in neurologic and hypertensive emergencies (32).

According to the study, nicardipine showed few negative short-term consequences on kidney function. With a minor but statistically significant mean change of +0.022 ± 0.120 mg/dL ($p = 0.010$), serum creatinine increased from 2.172 ± 0.893 mg/dL to 2.194 ± 0.895 mg/dL. This increase, however, was clinically insignificant and most

likely results from a hemodynamic adjustment rather than direct nephrotoxicity. More significantly, urine output increased from 0.956 ± 0.165 mL/kg/hour to 1.056 ± 0.170 mL/kg/hour ($p < 0.001$). All of these results point to nicardipine decreasing blood pressure while preserving or potentially improving effective renal perfusion. Similar findings have been shown in recent nephrology literature, where calcium channel blockers are regarded as either reasonably protective or renally neutral when blood pressure is lowered under controlled conditions (30).

KDIGO stage study, which revealed that while baseline creatinine varied considerably between stages, post-treatment creatinine decrease and urine output improvement did not differ significantly among KDIGO Stage 1, 2, and 3 patients, further corroborated the renal findings. This implies that, with the right supervision, nicardipine can be administered somewhat safely across a range of baseline renal function. Because patients with CKD or inadequate renal reserve are frequently more vulnerable to renal deterioration during strong antihypertensive medication, this is clinically significant. According to the current research, nicardipine might be a good choice even for these people who are at a higher risk.(43,44).

The positive association between age and SBP reduction ($r = 0.52$, $p < 0.001$) was another noteworthy observation, suggesting that older patients tended to have larger systolic blood pressure reductions. This could be explained by increasing peripheral resistance and age-related vascular stiffness, which could render older people more sensitive to arterial vasodilators like nicardipine. Although limited literature has specifically evaluated this relationship, the finding is biologically plausible and aligns with broader evidence that antihypertensive response can vary according to age and vascular characteristics (27).

The study showed that nicardipine did not significantly disrupt electrolytes in terms of biochemical safety. Following treatment, there were very slight, non-significant changes in serum sodium, potassium, chloride, and bicarbonate levels. Similarly, there was no significant change in BUN, uric acid, or UACR. According to these results, nicardipine has a good metabolic safety profile and has no appreciable short-term effects on albuminuria, nitrogen metabolism, or electrolyte balance. In hypertensive patients with CKD, diabetes, or other metabolic comorbidities, where biochemical stability is clinically significant, this is particularly pertinent (45).

## CONCLUSION

In conclusion, nicardipine can be used to achieve a significant decrease in systolic blood pressure with only slightly significant short-term renal dysfunction the increase in serum creatinine and an increase in urine output. These results indicate that nicardipine could be an effective antihypertensive medication, though renal monitoring should still be considered attentive, especially in individuals with impaired normal kidney functioning.

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