Journal of Language and Health
Volume 6 No 2, June 2025
e-ISSN 2722-3965; p-ISSN 2722-0311
http://jurnal.globalhealthsciencegroup.com/index.php/JLH

THERAPEUTIC PITFALLS OF NICARDIPINE (CALCIUM CHANNEL BLOCKER) IN HYPERTENSIVE EMERGENCY WITH LOW VENTRICULAR EJECTION FRACTION: A CASE OF REFRACTORY CARDIOGENIC SHOCK

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ABSTRACT
Nicardipine, a member of the dihydropyridine class of calcium channel blockers, is frequently employed for acute blood pressure control in hypertensive emergencies due to its potent and rapid vasodilatory effects. However, its use in patients with compromised cardiac function particularly those with significantly diminished left ventricular ejection fraction (LVEF) warrants caution, given its potential to depress myocardial contractility. This report presents the clinical course of a 72-year-old female patient with long-standing coronary artery disease and chronically reduced LVEF who was admitted in hypertensive crisis accompanied by respiratory failure. This study seeks to explore and understand the effects of intravenous nicardipine on vulnerable patients whose hearts are already weakend particulary those with severaly reduced left ventricular ejection fraction (LVEF). She was administered intravenous nicardipine at a conservative starting dose of 2 mg/hour. Within six hours, the blood pressure dropped precipitously from 252/138 mmHg to 88/46 mmHg. Despite prompt discontinuation of the drug and initiation of vasopressor support with norepinephrine and dobutamine, the patient's condition rapidly worsened, progressing to cardiogenic shock and culminating in cardiac arrest on the sixth day in the intensive care unit. This case exemplifies the potential dangers of administering vasodilatory agents like nicardipine to patients with severely limited myocardial reserve. The convergence of systemic vasodilation, reduced inotropic support, and poor compensatory response likely precipitated the hemodynamic collapse.

Kata kunci: CCB; cardiogenic shock; low EF; nicardipine; vasodilator toxicity

INTRODUCTION
Hypertensive emergencies reflect a severe form of cardiovascular decompensation requiring immediate therapeutic measures to promptly lower dangerously high blood pressure and prevent damage to vital organs. In such scenarios, intravenous antihypertensive drugs are often preferred due to their ability to deliver swift and controlled reductions in vascular tone. Among the pharmacologic options, nicardipine, a calcium channel blocker of the dihydropyridine subclass is commonly selected because of its rapid onset, ease of dose adjustment, and targeted vasodilatory effect on arterial musculature. Nonetheless, administering this agent in patients with compromised cardiac performance warrants a high degree of caution (Ibarra F Jr, 2024) .

Calcium channel blockers (CCBs) are therapeutically classified into two principal types: dihydropyridines and non-dihydropyridines. The former group primarily targets vascular smooth muscle located in peripheral circulation, whereas the latter demonstrates greater selectivity for cardiac muscle cells. On a molecular level, CCBs exert their action by blocking the alpha-1 subunit of L-type voltage-gated calcium channels. This blockade restricts the intracellular movement of calcium ions, which is a crucial process for initiating smooth muscle contraction in the myocardium, blood vessels, brain, kidneys, and gastrointestinal system(Koroki T, 2022). Inhibiting this calcium influx promotes relaxation of

smooth muscle fibers, thereby inducing vasodilation and lowering blood pressure (McKeever RG, 2025). Dihydropyridines like Nicardipine are known for their ability to significantly decrease systemic vascular resistance (SVR) and act swiftly, making them a widely preferred choice in the management of hypertensive emergencies(Hilal-Dandan, 2022).

Patients with chronic cardiac conditions, including coronary artery disease and reduced left ventricular ejection fraction (LVEF), are particularly vulnerable to rapid shifts in vascular tone. The administration of vasodilators like nicardipine can impose abrupt hemodynamic changes, which may not be well tolerated in those with diminished myocardial reserve. In such settings, the heart may be unable to increase stroke volume in response to decreased afterload, leading to inadequate tissue perfusion. When compensatory mechanisms fail, patients may rapidly progress to a state of cardiogenic shock a condition characterized by sustained hypotension and insufficient cardiac output (Ibarra F Jr, 2024). This case report describes the clinical trajectory of a 72-year-old female patient with a documented history of coronary artery disease and pre-existing left ventricular systolic impairment, who arrived with critically high blood pressure accompanied by respiratory compromise. As an initial intervention, she was started on intravenous nicardipine at a lower-than-standard infusion rate (Bress AP, 2024). Nonetheless, this cautious dosing strategy was followed by a rapid and profound decline in blood pressure, prompting the immediate initiation of vasopressor and inotropic support. Despite sustained hemodynamic management over the ensuing days, the patient's condition failed to stabilize, ultimately progressing to full cardiovascular collapse.

In addition to their role as vasodilators, Nicardipine also has other mechanisms of action. Hypotension does not primarily occur due to vasodilation, but also as a combination of negative inotropic and chronotropic effects on the myocardium. This highlights the need for careful safety considerations in the use of CCBs especially Nicardipine, despite their significant benefits in lowering blood pressure (Bauer M., 2017). Calcium channel blocker toxicity has the potential to cause critical complications. In severe cases, it may manifest as dangerously slow heart rate, profound hypotension, and even cardiogenic shock. Given these risks, it is essential for clinicians to remain vigilant about the possibility of drug-induced harm and exercise strict care in dosing and monitoring when managing patients receiving agents like Nicardipine. *This study seeks to explore and understand the effects of intravenous nicardipine on vulnerable patients whose hearts are already weakend particulary those with severaly reduced left ventricular ejection fraction (LVEF)(Andrusaitis JG, 2024).*

METHOD

This case report presents a retrospective review of an elderly patient admitted to the intensive care unit (ICU) with a hypertensive emergency and acute respiratory failure. The patient's medical background included coronary artery disease and reduced left ventricular ejection fraction, both of which placed her at significant risk for hemodynamic instability. Clinical data were drawn from ICU records, including admission details, progress notes, medication administration logs, and nursing observations. Specific information such as presenting symptoms, baseline hemodynamic values, nicardipine usage, ventilator settings, neurological evaluations, and the patient’s clinical trajectory were documented. The timeline of care was reconstructed using bedside monitoring sheets and nursing documentation to ensure chronological accuracy. The purpose of this approach is to present a clinically relevant and ethically sound narrative that highlights key therapeutic decisions, potential causal relationships, and patient outcomes. The insights gained are intended to support greater clinical awareness and guide future management strategies for similarly vulnerable patient populations.

CASE REPORT

A 72-year-old female with a history of low ventricular ejection fraction (EF) and chronic coronary arterial disease (CAD) presented with altered consciousness due to hypoxia from respiratory failure, accompanied by hypertensive emergency and acute lung oedema (ALO). On arrival, the Glasgow coma

scale (GCS) was E2M2V2, vital signs included a temperature of 36°C, BP 252/138 mmHg, HR 120, RR 8 (gasping), and O2 saturation of 35% on room air. Physical examination revealed respiratory muscle retraction, bilateral lung ronchi, and elevated Jugular Venous Pressure (JVP). Electrocardiogram (ECG) showed sinus rhythm with a ventricular rate of 118 bpm, T-wave inversion in leads II, III,aVF, V5-V6, Q waves in lead V2 (QRS: 80 ms, QTC: 400 ms) (Figure 1).

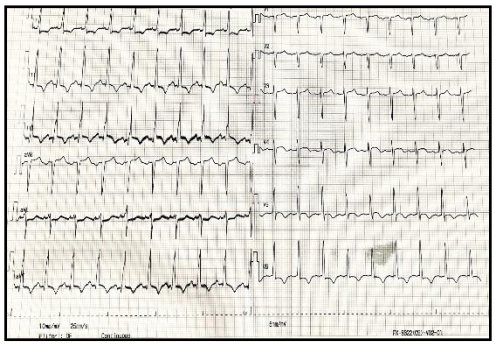

Figure 1. Initial electrocardiogram

Echocardiogram showed a left ventricular ejection fraction (LVEF) of 41%, left ventricular hypertrophy, tricuspid annular plane systolic excursion (TAPSE) 2.4 cm, All heart valves are operating normally, showing no signs of stenosis (narrowing), regurgitation (leakage), or any other structural or functional abnormalities. All of the laboratory results were normal (including complete blood count and renal function), except the blood gas analysis showed respiratory acidosis (pH: 7.23 {Normal 7.37-7.45}, CO2: 51.00 mmHg {Normal 35.00-45.00}, HCO3: 21.40 mmol/L{Normal 21.00-25.00}). Thyroid stimulating hormone sensitive (TSHs) was 0.163 uIU/mL (Normal 0.270-4.200). Chest X-ray showed pulmonary edema (Figure 2) and CT scan revealed minimal infarction at the anterior corner of the right lateral ventricle due to severe hypoxia (Figure 3). Her home medications included furosemide 40 mg per oral (PO) once a day (QD), clopidogrel 75 mg PO QD, aspirin 80 mg PO QD, ramipril 2,5 mg PO QD, atorvastatin 20 mg PO QD, bisoprolol 2,5 mg PO QD. She did not take the medications routinely as prescribed.

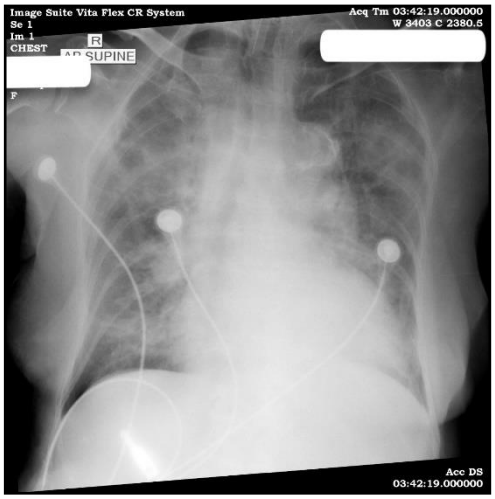

Figure 2. Initial Chest X-Ray

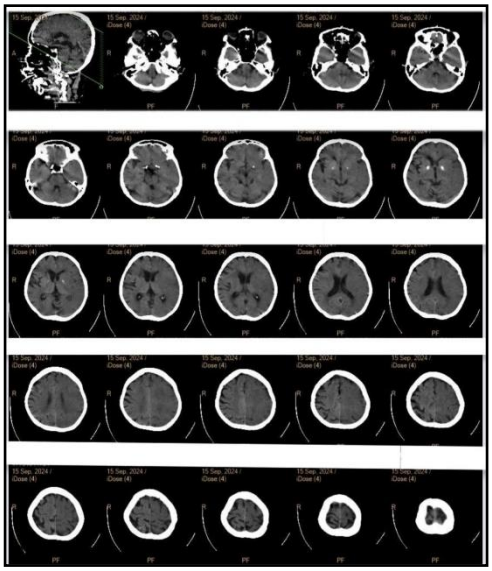

Figure 3. Brain CT

On arrival at the emergency department (ED), the patient was severely ill-appearing, unresponsive (GCS E2M2V2), and acute respiratory failure. Emergent intubation was prosperously completed using 7.0-m.m endotracheal tube (ETT). She was escorted by the sign of emergency hypertension and began receiving nicardipine drip treatment at a rate of 2 mg per hour. After six hours of nicardipine administration, the patient's blood pressure decreased dramatically from 252/138 mmHg to 88/46 mmHg. Along with the decrease in heart rate from 120 beats per minute, regular, and strongly palpable, to 98 beats per minute, regular, but weakly palpable, the extremities felt moist, and capillary refill time (CRT) was prolonged. Patient was transferred to the Intensive Care Unit (ICU). The patient's antihypertensive medication was discontinued, and resuscitation with fluids and norepinephrine (NE) was initiated to manage cardiogenic shock. By the following day, the patient's condition became hemodynamically unstable, prompting the addition of dobutamine (Figure 4). After the administration of two inotropic agents within 24 hours, blood pressure was successfully maintained at normal and stable levels, although the Glasgow Coma Scale (GCS) did not show any improvement. The process of weaning off cardiac inotropic support was initiated on the fifth day of care. Unfortunately, during the weaning process, blood pressure suddenly decreased. On the sixth day of ICU care, the patient experienced cardiac arrest. In accordance with the family's wishes, the physician adhered to the Do Not Resuscitate (DNR) order.

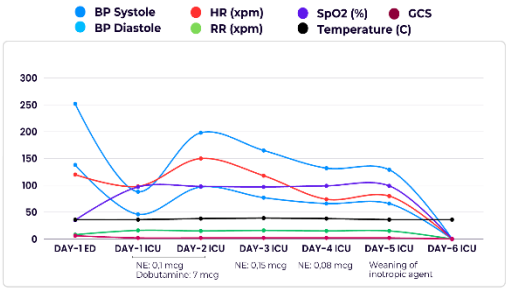

Figure 4: Vital Sign Trend in ICU

RESULTS AND DISCUSSION

Nicardipine, a calcium channel blocker (CCB), is widely employed in managing hypertension, particularly during hypertensive crises. These crises are defined by a sudden and significant increase in blood pressure, typically exceeding 180/110-120 mmHg, as outlined by the American Heart Association in 2017. They are classified into two types: hypertensive emergency, which involves acute target organ damage, and hypertensive urgency, which does not (Mancia G, 2023). Treatment in these situations requires careful blood pressure reduction, delivered orally or intravenously, but must be done cautiously

to avoid inducing hypoperfusion. Hypertension-mediated organ damage (HMOD) refers to the structural and functional deterioration of organs such as the kidneys, heart, and brain due to persistent high blood pressure (Mancia G, 2023).

When hypertension is not properly controlled, it can lead to complications such as hypertensive heart disease (HHD), which may eventually progress to heart failure with reduced left ventricular ejection fraction (LVEF)(Bakris GL, 2018). The normal LVEF range is generally between 52% and 74%, as stated by the American Society of Echocardiography and the European Association of Cardiovascular Imaging(Kosaraju A, 2025). Increased systemic vascular resistance (SVR), a hallmark of hypertension, causes the heart muscle cells to enlarge. This hypertrophy disrupts coronary blood flow and can lead to subendocardial ischemia.(Mancia G, 2023) Over time, it also promotes collagen accumulation, resulting in cardiac fibrosis. Both of these changes are central to the development of heart failure symptoms, such as shortness of breath (Bakris GL, 2018). Coronary artery disease can further aggravate heart failure and worsen clinical outcomes.

One of the risks associated with nicardipine use is a sharp drop in blood pressure, which can result in hypotension. Vasodilators are essential in managing heart failure because they decrease left ventricular filling pressures and improve stroke volume by reducing SVR(Butt AK, 2022), which helps lower myocardial oxygen demand. However, while calcium channel blockers are effective vasodilators, they are also known for their negative inotropic properties, which can impair cardiac performance in susceptible patients(Bauer M., 2017). Nicardipine reduces the strength of heart muscle contractions by limiting calcium entry into cardiac cells. This action can further weaken heart function, particularly in individuals with preexisting heart failure and low LVEF. In such cases, while SVR reduction can lower afterload, the heart may not be able to respond adequately to the drop in blood pressure. The combination of vasodilation and decreased contractility may lead to systemic hypoperfusion and exacerbate circulatory shock (Hiraiwa H, 2024). In the case described, treatment involved using a vasodilator to manage SVR while simultaneously supporting cardiac output and blood pressure with inotropic therapy(Butt AK, 2022).

Intravenous nicardipine is frequently used in hypertensive emergencies because of its fast-acting nature and adjustable dosing, allowing tight control over vascular resistance. However, in patients with significantly weakened heart function, especially those with reduced left ventricular ejection fraction (LVEF), its benefits can be outweighed by the risk of destabilizing the cardiovascular system (Jones KE, 2024). This case highlights a 72-year-old woman with existing coronary artery disease and a low LVEF who suffered severe circulatory collapse shortly after receiving nicardipine during a hypertensive crisis. On admission, her blood pressure was critically high at 252 over 138 mmHg. Nicardipine was administered at a low initial dose of 2 mg per hour. Within six hours, her blood pressure dropped dramatically to 88 over 46 mmHg. This sudden decline was followed by reduced peripheral perfusion, delayed capillary refill, and visible signs of clinical shock. These developments strongly suggest that the vasodilatory effects of nicardipine exceeded what her failing heart could handle, leading to cardiovascular collapse.

In patients with systolic dysfunction, vasodilators can lower preload and afterload to a degree the heart is unable to compensate for. Although a reduction in systemic vascular resistance might seem advantageous, the heart's diminished pumping ability often fails to maintain sufficient stroke volume, resulting in dangerously low perfusion pressures. Nicardipine can also reduce the heart's contractile strength, which further complicates the situation in vulnerable individuals. The patient quickly showed signs of clinical deterioration, including a thready pulse, altered mental status, and hypotension, which pointed to early cardiogenic shock. The treatment team responded by halting the nicardipine infusion and starting norepinephrine to restore vascular tone. When this failed to produce a sufficient response,

dobutamine was added to improve the heart's contractile output. Although these interventions temporarily stabilized her condition, they did not prevent continued decline.

This scenario demonstrates the importance of close and continuous monitoring. The transition from hypertensive crisis to full shock occurred within a few hours. In patients with minimal cardiac reserve, such rapid decompensation is not uncommon. Tracking urine output, mental alertness, lactate levels, and frequent blood pressure readings can help clinicians catch early signs of medication intolerance. The combination of norepinephrine and dobutamine is a standard approach when treating cardiogenic shock. Norepinephrine counters vasodilation by tightening blood vessels, while dobutamine supports the heart's pumping ability. Despite this, the patient's mean arterial pressure remained below target, pointing to the severity of her cardiac impairment.

Individuals with coronary artery disease and low LVEF have little tolerance for abrupt shifts in vascular load. Blood flow to the heart muscle itself depends heavily on stable diastolic pressure and overall cardiac output. When resistance drops and pressure falls, the body may struggle to deliver oxygen to vital organs, and even high-dose inotropes may be insufficient to reverse the process once shock sets in. The patient was also placed on pressure-controlled mechanical ventilation to maintain oxygen levels. While this supported her respiratory status, it may have inadvertently worsened cardiovascular function. Positive pressure in the chest can reduce venous return and preload, which can be harmful when combined with drug-induced vasodilation.

In the following days, there was no meaningful neurological recovery, and her blood pressure remained low despite continued support. ICU notes consistently described signs of inadequate blood flow to the brain and other organs. Despite intensive efforts, she never regained adequate circulation or consciousness. This pattern of deterioration reflects what is often seen in advanced heart failure. Once oxygen delivery falls below a critical threshold, a cascade of metabolic problems follows, including lactic acidosis and tissue hypoxia. As this progresses, the body becomes less responsive to medication, and organ recovery becomes less likely. Even after stopping nicardipine and administering multiple vasoactive agents, the patient failed to recover. Her dependence on continuous drug support, paired with ongoing neurological dysfunction, led the care team and family to agree on a Do Not Resuscitate order. This reflected a realistic assessment of the situation and a compassionate decision based on her prognosis. The repeated episodes of hypotension despite aggressive treatment confirmed that the patient was in refractory cardiogenic shock. Each time the team attempted to reduce the doses of norepinephrine or dobutamine, her blood pressure fell again, showing that her heart could not maintain output on its own.
This case highlights the critical need to tailor blood pressure management strategies in patients with low ejection fractions. Nicardipine, though effective, may not be safe in individuals with severely reduced cardiac function. Other medications with less pronounced vasodilatory or negative inotropic effects might be more appropriate.

Importantly, the timeline of the patient's deterioration aligns closely with the initiation of nicardipine. Hypotension developed within hours of infusion, followed by escalating need for circulatory support. While direct causation can't be proven without more invasive data, the clinical sequence strongly supports the role of nicardipine in triggering this crisis. As her ICU stay progressed, there was no meaningful improvement in blood pressure or neurological status. The team ultimately shifted the focus of care from aggressive treatment to comfort, in line with the patient's condition and wishes of the family. This experience illustrates the dangers of using strong vasodilators in patients with known or suspected heart failure. Even low doses can precipitate serious complications when the heart lacks the reserve to respond. Structured clinical pathways and careful drug selection are essential when managing hypertension in this population.
By the final days of care, the patient remained dependent on vasopressors and showed no neurological improvement. Every attempt to reduce drug support led to another hypotensive event, reinforcing the

heart's inability to function unaided. This clinical pattern is typical of severe cardiogenic shock. Despite the use of mechanical ventilation, sedation, and all appropriate ICU interventions, the patient's condition continued to decline. It is likely that the initial hypotensive episode caused irreversible damage to multiple organ systems, especially the heart and brain. Further diagnostic work was not pursued, consistent with the palliative goals of care. This case calls attention to the importance of using vasodilators cautiously in elderly patients with cardiac comorbidities. Even a conservative starting dose of 2 mg per hour of nicardipine resulted in major instability. This suggests that standard protocols should be adapted to individual patient risk factors.

Finally, the case underscores the need for a team-based approach in intensive care. Coordinated efforts among physicians, pharmacists, nurses, and specialists are vital to detect early signs of medication intolerance and respond appropriately. Understanding the unique vulnerability of each patient remains central to safe and effective care.

CONCLUSION

Nicardipine is widely used in hypertensive emergencies due to its rapid onset and easily titratable vasodilatory effects. However, its increasing use carries a risk of accidental or iatrogenic overdose, particularly when administered without appropriate hemodynamic monitoring. In some cases, even therapeutic doses can lead to severe adverse effects such as cardiogenic shock. This risk is heightened in patients with limited cardiac reserve, such as those with significantly reduced ejection fraction or underlying coronary artery disease. The absence of a standardized intravenous dosing protocol further complicates its safe administration, underscoring the importance of individualized dosing and close patient monitoring. Drug accumulation from inadvertent overdosing can quickly escalate into toxicity and circulatory collapse. The case presented illustrates the clinical challenge of using nicardipine in patients with compromised cardiac function. A dramatic blood pressure drop following administration in a patient with low ejection fraction highlights the delicate balance required when managing blood pressure in such individuals. The rapid progression from hypertensive crisis to cardiogenic shock exemplifies the dangers vasodilators can pose in fragile hearts. Therefore, comprehensive cardiac evaluation is essential before initiating intravenous antihypertensive therapy in high-risk patients to prevent life-threatening complications.

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