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CASE REPORT

Calcium channel blocker overdose in real world practice: A case report

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ABSTRACT:

Introduction: Calcium channel blocker (CCB) toxicity, particularly from dihydropyridine agents, like amlodipine, can cause life-threatening vasodilatory shock, bradycardia, and metabolic disturbances. In severe cases, standard resuscitative measures may be insufficient, necessitating advanced supportive and targeted therapies.

Case presentation: A 72-year-old male presented 9 h after intentional ingestion of 200 tablets (1000 mg) of amlodipine with hypotension, metabolic acidosis, and acute kidney injury, but without hyperglycemia. Initial management included intravenous fluid resuscitation, calcium gluconate, and vasopressors. Despite this, he developed refractory shock, acute respiratory failure, and cardiac arrhythmia, requiring mechanical ventilation and intensive hemodynamic support. High-dose insulin euglycemia therapy (HIE) and methylene blue were administered. Renal function gradually improved, and vasopressors were weaned, and the patient was successfully extubated and discharged.

Conclusions: This case highlights the complexity of managing severe amlodipine overdose, emphasizing the role of early vasopressors, calcium salt administration, and adjunctive therapies such as HIE and methylene blue. Prompt recognition and a multimodal approach are critical for improving outcomes in patients with CCB toxicity.

Keywords: Calcium channel blockers toxicity; High-dose insulin euglycemia therapy; GI decontamination; Methylene blue; Intravenous lipid emulsion; Intensive care unit

INTRODUCTION

Calcium channel blockers (CCBs) are commonly prescribed for cardiovascular conditions such as hypertension, supraventricular tachycardia, and angina. They are broadly categorized into dihydropyridines (e.g., amlodipine, nifedipine) and non-dihydropyridines (e.g., verapamil, diltiazem), based on their predominant action on vascular smooth muscle or cardiac myocytes, respectively (Table1).

CCBs exert their effects by inhibiting L-type voltage-gated calcium channels, reducing calcium influx required for vascular tone, cardiac contractility, and insulin secretion. These agents are lipophilic, highly protein-bound, and widely distributed (>2 L/kg), limiting the efficacy of extracorporeal removal methods like hemodialysis [1,2].

In overdose, pharmacologic selectivity is lost, leading to profound hypotension, bradycardia, and metabolic complications. Dihydropyridines may initially cause reflex tachycardia followed by myocardial depression [2]. Additionally, CCB toxicity can impair glucose metabolism, resulting in hyperglycemia and lactic acidosis [3-5]. Serum glucose levels have been proposed as a useful prognostic marker in severe CCB poisoning, potentially reflecting the degree of toxicity more reliably than hemodynamic parameters [2,6].

CASE PRESENTATION

A 72-year-old Thai male from Chiang Mai with a known history of essential hypertension treated with a daily dose of Amlodipine 10 mg. He presented to the emergency department following a deliberate overdose. Approximately 9 h prior to arrival, the patient ingested an estimated 200 tablets (2,000 mg) of Amlodipine. He subsequently developed fatigue and palpitations without chest pain, followed by progressive drowsiness. He remained verbally responsive. His neighbors summoned emergency medical services, and he was transported to the hospital.

On day 1 of admission, he was hypothermic with a body temperature of 35°C. He was hypotensive with blood pressure of 80/48 mmHg (mean arterial pressure [MAP] 59 mmHg) and bradycardic with a heart rate of 66 beats per minute (bpm). He was drowsy but arousable (GCS 14: E3V5M6). Oxygen saturation was 99% on 3 L/min of oxygen via nasal cannula, with a normal respiratory pattern.

Initial laboratory results revealed normal limits of complete blood count, serum glucose of 125 mg/dL, and the occurrence of acute kidney injury with elevated creatinine (2.63 mg/dL) and wide anion gap metabolic acidosis (bicarbonate 18 mmol/L and anion gap 17). Lactate was mildly elevated at 1.94 mmol/L.

The patient was managed with 1.5 liters of fluid resuscitation with 0.9% sodium chloride, norepinephrine infusion of 0.03 µg/kg/min initially, and intravenous calcium gluconate 10% solution (5 gm). After that, his blood pressure improved to 100/63 mmHg (MAP 75 mmHg) with a heart rate of 69 bpm. A diagnosis of calcium channel blocker (CCB) toxicity was made, with hypotension attributed to vasoplegia and hypovolemia. Acute kidney injury due to hemodynamic acute tubular necrosis was also suspected. He was transferred to the ICU for advanced monitoring and treatment.

KEY MESSAGES:

- Amlodipine overdose can lead to life-threatening vasodilatory shock, bradycardia, and organ dysfunction.
- Early, aggressive hemodynamic support with fluids and vasopressors is critical.
- Calcium salt administration and high-dose insulin euglycemia therapy (HIE) are key treatments to restore cardiac and vascular function.
- Methylene blue may be effective in managing refractory vasoplegia.
- Continuous monitoring and organ support, especially for renal and respiratory systems, are essential for recovery.

On the same day, high-dose insulin euglycemia therapy (HIE) was initiated using regular insulin 1,000 units in 100 mL of 0.9% NaCl at 3 mL/h (0.5 unit/kg/h) and 50% dextrose at 60 mL/h (0.5 g/kg/h). Capillary blood glucose was monitored every 15 min, and serum electrolytes were assessed every 4 h. Dopamine was additionally started to maintain perfusion.

Within hours, the patient developed dyspnea and hypotension (BP 70/40 mmHg, Central venous pressure [CVP] 7 mmHg) despite norepinephrine 2 µg/kg/min and dopamine 5.5 µg/kg/min. Electrocardiography (ECG) showed frequent premature ventricular contractions. Arterial blood gas (ABG) showed pH 7.27, HCO_3^- 13.6 mmol/L, and serum lactate 7 mmol/L. He was intubated and given calcium gluconate (3 gm) and 800 mL balanced crystalloid solution. Dopamine was discontinued due to arrhythmias, and adrenaline was started at 0.5 µg/kg/min instead for the purpose of refractory hypotension despite the high dose of norepinephrine administered.

On day 2 of admission, hemodynamic status was supported by norepinephrine 2 µg/kg/min and adrenaline 1.6 µg/kg/min. ABG showed worsening acidosis (pH 7.22), and the ionized calcium level was low (0.48 mmol/L, less than 1.05-1.3 mmol/L). Labs showed BUN 49 mg/dL, creatinine 4.43 mg/dL, potassium 2.5 mmol/L, bicarbonate 11 mmol/L, and anion gap 23. Calcium gluconate infusion and a single dose of methylene blue (1 mg/kg over 6 h) were administered.

Table 1. Classes of Calcium channel blockers and their mechanism [2,39].

CCBs Classes	Agents	Mechanism	
		Vasodilator effect	Cardiac contractility
Dihydropyridine CCBs	Amlodipine, Nifedipine, Nicardipine, Nimodipine	Potent	Less
	Manidipine, Lercanidipine	Potent & Long lasting	Less*
Non-dihydropyridine CCBs	Phenylalkylamines (Verapamil)	Potent	Potent
	Benzothiazepines (Diltiazem)	Less	Potent

*Antihypertensive effect through dual inhibition of both vascular L-type and sympathetic N-type calcium channels by attenuating sympathetic nerve activity and limiting norepinephrine release, it effectively mitigates reflex tachycardia commonly associated with peripheral vasodilation

Due to oliguria and volume overload (fluid balance +3,277 mL in 24 h with urine output 210 mL in 24 h), intravenous furosemide 250 mg was administered, increasing urine output to 6,564 mL in 24 h. HIE therapy was continued for 7 days.

Hemodynamics stabilized, and all vasopressors were weaned within 5 days, and he was successfully extubated the following week. Renal function improved with increasing urine output and declining creatinine. The patient was transferred to the general ward and subsequently discharged in stable condition.

DISCUSSION

This patient consumed an excessive quantity of amlodipine, totaling 200 tablets, which is considered CCBs overdose and intoxication, resulting in refractory vasodilatory shock, by which the accompanying poor vital organ perfusion included acute kidney failure and poor cerebral perfusion.

Management for a patient who is in a state of shock due to CCBs intoxication could be categorized into general management and specific management. General management included initial stabilization with airway, breathing, and circulation management and optimization of the hemodynamic profile in order to avoid prolonged hypotension or refractory shock and restore organ perfusion pressure with fluid resuscitation and early vasopressors [2].

To restore vascular tone and enhance myocardial contractility in cases of calcium channel blocker toxicity, initiation of intravenous vasopressor and/or inotropic therapy are often warranted. A retrospective study conducted by Michael Levine et al. reviewed 48 patients over the age of 14 who were admitted to an inpatient toxicology service for the management of verapamil or diltiazem overdose between 1987 and 2012. Vasopressor therapy was employed in 33 of the 48 patients (69%). Reported maximal infusion rates included epinephrine at 150 μg/min, dopamine at 100 μg/kg/min, dobutamine at 245 μg/kg/min, isoproterenol at 60 μg/min, phenylephrine at 250 μg/min, and norepinephrine at 100 μg/min. The concurrent use of multiple vasopressors was frequently observed. Notably, despite the administration of high-dose vasopressors, ischemic complications were uncommon and, when present, were typically pre-existing rather than a direct consequence of vasopressor therapy[7].

Due to the lack of comparison studies between vasopressor types, initial selection is based upon clinical presentation and types of shock [8-10]. The choice of these medications is informed by the underlying pathophysiological mechanisms and the specific type of shock, each of which significantly influences individualized patient management. In cases where vasodilation predominates, norepinephrine which is primarily an α1 adrenergic receptor agonist with additional β1 activity, is initially employed and may enhance myocardial contractility. Epinephrine exerts a potent β1 adrenergic effect that augments cardiac inotropy while concurrently stimulating the α1 adrenergic receptor. In this patient, norepinephrine was initiated as the first-line agent due to the predominant mechanism of calcium channel blocker toxicity being profound vasodilation. Given the additional concern for impaired myocardial contractility, intravenous epinephrine was subsequently administered to augment cardiac output.

The use of dopamine or other vasopressor agents as sole therapeutic interventions is generally not recommended due to their variable efficacy and inconsistent hemodynamic responses reported in multiple case series. In light of these findings, the expert workgroup advises against the routine use of dopamine as a primary agent in this clinical context [8,10,11].

Point of care ultrasonography, including echocardiograms, should be performed to evaluate myocardial contractility and cardiac output, which could imply cardiac function and help for assessment of fluid status. Intravenous atropine should be administered in case of bradycardia; some may require cardiac pacing [8,12].

In patients exhibiting signs of significant positive fluid balance, often as a consequence of high-volume intravenous administration as part of high-dose insulin euglycemic therapy (HIE) and concurrent supplementation with dextrose and potassium, along with complications related to acute kidney injury secondary to hemodynamic instability, the use of a loop diuretic such as furosemide may be considered to facilitate diuresis and support the maintenance of euvolemia [12]. However, if failure to maintain fluid balance and/or correction of severe metabolic disturbance occurs, renal replacement therapy may be initiated.

Associated complications, such as non-cardiogenic pulmonary edema (NCPE) and acute respiratory distress syndrome (ARDS) may be troublesome in some patients as results of capillary leakage.

Pulmonary edema has been reported in numerous cases of calcium channel blocker (CCB) overdose. In an early case described in 1985, a patient who had ingested a large quantity of nifedipine developed rapidly progressive dyspnea, accompanied by radiographic evidence consistent with pulmonary edema.

CCBs are known to exert significant effects on the pulmonary vasculature. In such cases, precapillary vasodilation has been proposed as a potential mechanism underlying the development of non cardiogenic pulmonary edema (NCPE) following CCB toxicity.

Therefore, oxygen supplementation and mechanical respiratory support with positive pressure ventilation may help reduce the work of breathing [13,14].

Specific management includes GI decontamination in case of ingestion within 1 h, by giving 1 gm/kg of activated charcoal to the patient with normal mental status who is able to protect their airway from aspiration. Activated charcoal effectively prevents the absorption of CCBs, but it is most effective up to 4 h after ingestion of sustained-release CCBs.

Gastric lavage is not routinely indicated, according to the study by Lapatto-Reiniluoto et al., who investigated the impact of GI decontamination by assessing plasma and urinary levels of verapamil following administration of either 25 grams of activated charcoal or gastric lavage. Their findings demonstrated a significant reduction in the mean plasma concentration with activated charcoal, but only a minimal reduction with gastric lavage. Later, in a 2013 joint position statement, the American Academy of

Clinical Toxicology and the European Association of Poisons Centers and Clinical Toxicologists highlighted a lack of robust data supporting its efficacy and recommended against gastric lavage in routine use [8,15-17] In our cases, the patient did not receive GI decontamination due to exposure time over 1 h and high risk of aspiration.

Initial treatment with calcium salt increased the extracellular calcium gradient, resulting in the influx of calcium into cardiac myocytes and vascular endothelial cells. This promotes cardiac contraction and increases vascular tone. It has been proposed that calcium salts counteract the competitive antagonism at cardiac conduction pathways [9].

Initial dosing of a 0.2 mL/kg bolus of 10% calcium chloride intravenously via central access, which may be repeated. This can be followed by a continuous infusion of 0.2-0.5 mL/kg/h. In patients without central venous access, 10% calcium gluconate may be administered at an initial dose of 0.6 mL/kg intravenously, with a continuous infusion of 0.6 -1.5 mL/kg/h or intermittent boluses [18]. Calcium infusion should be titrated, with serum ionized calcium level monitors every 1-2 h, targeting 1.5-2 times the upper normal reference value [2]. In this patient, 20 mL of 10% calcium gluconate was administered intravenously, followed by repeated doses and a continuous infusion.

Adjunctive therapy such as HIE therapy may exert an inotropic effect on cardiac myocytes in cases of profound shock with cardiac dysfunction or cardiogenic shock. Preclinical studies have demonstrated improved survival with HIE therapy compared to conventional agents such as glucagon and epinephrine [19]. HIE is typically initiated with a bolus of 1 U/kg, followed by a continuous infusion at 1–10 U/kg/h, with titration of 1-2 U/kg/h every 10 minutes to achieve adequate organ perfusion (maximum 10 units/kg/h). Concurrent administration of a concentrated dextrose infusion is recommended at the initiation of HIE to maintain euglycemia [3,8,19-22]. In this patient, he received an intravenous regular insulin infusion starting at 0.5 unit/kg/h together with a 50% dextrose solution intravenously. Capillary blood glucose was checked with target levels between 100-250 mg/dL.

Methylene blue, a selective inducible nitric oxide synthase (iNOS) inhibitor, may be considered as an additional treatment in cases of persistent shock despite high-dose vasopressor [23]. Amlodipine has been shown to activate endothelial nitric oxide synthase, resulting in vasodilation [24]. Methylene blue works by blocking the inducible form of nitric oxide synthase, promoting calcium release from the sarcoplasmic reticulum in vascular smooth muscle cells, and thereby increasing vascular tone (Figure 1)[25]. The recommended dose is 1-2 mg/kg administered intravenously over 5 min, followed by continuous infusion at 1 mg/kg/h. Peak effects are typically observed within 5 min [26,27]. The maximum recommended dosing is 7 mg/kg. Of note, methylene blue may induce methemoglobinemia [28] and hemolysis in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency, potentially further compromising tissue oxygenation. In this patient, a 50 mg bolus of methylene blue was given intravenously, followed by a continuous infusion at 1 mg/kg/h for 6 h.

Intravenous lipid emulsion (ILE) may be optional adjunctive therapy in case of refractory shock or cardiac arrest. according to limited studies. The mechanisms of ILE include providing energy and free fatty acids to voltage-gated calcium channels in cardiac myocytes, thereby augmenting cardiac contractility. It exerts its effect through a "lipid sink" phenomenon, sequestering lipophilic toxins away from target tissues and into an intravascular lipid phase. Given its role as a rescue therapy. ILE should be considered primarily in cases of refractory shock or when standard interventions have proven ineffective [10].

ILE administration has been associated with immediate complications such as pyrogenic reactions and fat overload syndrome, while delayed complications include acute lung injury, fat embolism, hemolytic anemia, and hyperlipidemia. [29,30]. The dosage regimen is 20% Intralipid at 1.5 mL/kg bolus dose over 2-3 min intravenously, which could be repeated in cases of hemodynamic instability. This is followed by an infusion of 0.25-0.5 mL/kg/min for 30-60 minutes [31]. If a favorable hemodynamic response is achieved, the infusion rate may be reduced to 0.025 mL/kg/min [32]. Some authors recommend a max-

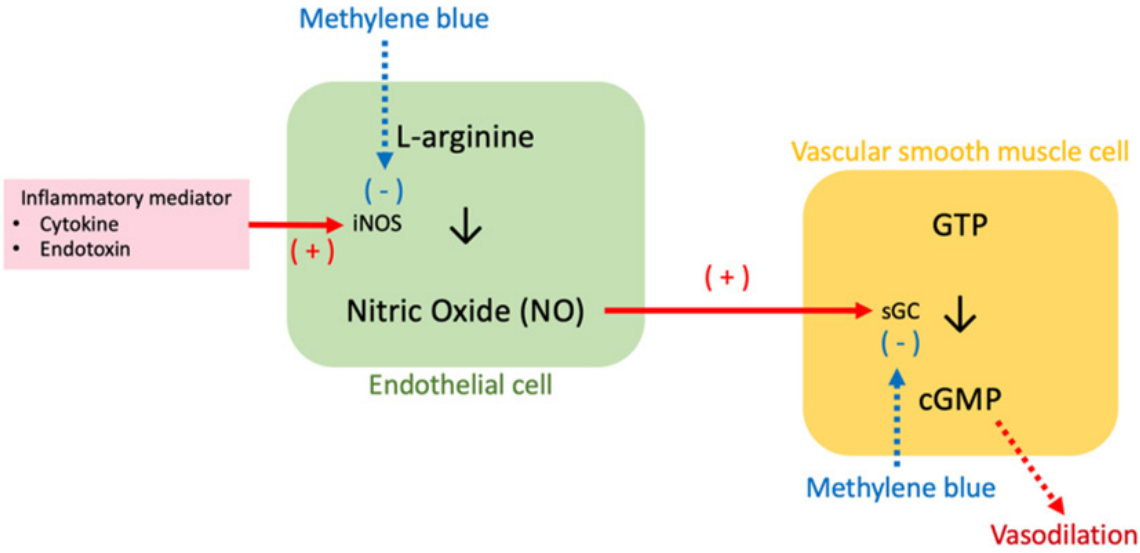

Figure 1. Mechanism of methylene blue [24,25].

Abbreviations: iNOs: Inducible Nitric Oxide Synthase; GTP: Guanosine triphosphate; cGMP: Cyclic guanosine monophosphate; sGC: Guanylate cyclases

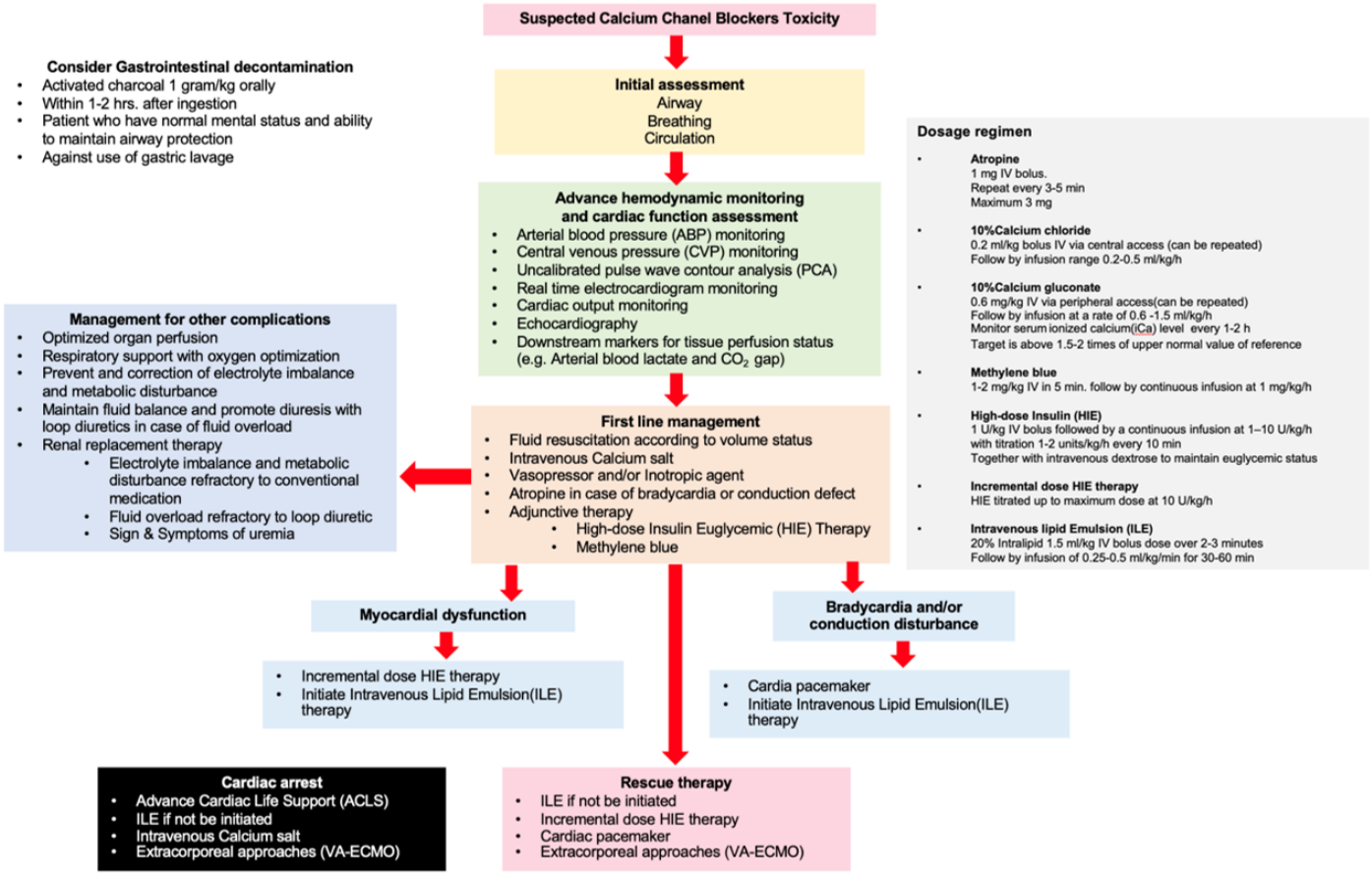

Figure 2. Recommended treatment for patients with CCBs intoxication [2,8,19].

imum cumulative dose of 10 mL/kg based on clinical experience and safety considerations [33].

Administration of 5 gm of hydroxocobalamin intravenously may be considered in case of refractory shock, as it acts as a nitric oxide scavenger that can attenuate vasoplegia [34]. Currently, there are no established guidelines for the use of hydroxocobalamin in the treatment of vasoplegia. However, preclinical studies in animal models have demonstrated its vasopressor effects, and in healthy human subjects, hydroxocobalamin has been associated with transient elevations in blood pressure [35].

Extracorporeal modalities may serve as bridging therapies during recovery in cases of acute kidney injury. The Extracorporeal in Poisoning (EXTRIP) Workgroup recommends against the use of extracorporeal treatments for CCBs overdose, as CCBs are highly protein-bound with a large volume of distribution, making them poorly dialyzable [36,37]. Extracorporeal membrane oxygenation (ECMO) may be beneficial in patients with cardiogenic or combined refractory shock. Veno-arterial (VA) ECMO is preferred for augmenting cardiac output, while veno-venous (VV) ECMO may be considered in cases of severe pulmonary edema or ARDS [38].

CONCLUSION

CCBs toxicity from ingestion of a large amount can lead to hypotension and bradycardia from reduced peripheral vascular resistance and decreased cardiac contractility, resulting in multiple complications from impaired organ perfusion. Management can be divided into general supportive care, aimed at maintaining hemodynamic stability and organ perfusion to prevent end-organ dysfunction by initiation of vasopressor and/or inotropic agent and intravenous fluid optimization, and specific treatments, which target the elimination of active metabolites and diminish CCBs effect, including calcium salt administration and adjunct therapies such as HIE and intravenous methylene blue (Figure 2). The choice of therapeutic modalities should be guided by clinical presentation and a careful assessment of potential adverse effects.

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AUTHORS' CONTRIBUTIONS

Adisak Chaisrima contributed to the conception and design of the case report, collected clinical data, performed the literature review, and drafted the initial manuscript; Kaweesak Chittawatanarat was involved in clinical insights and data interpretation and critically revised the manuscript for important intellectual content; Konlawij Trongtrakul supervised the overall project, contributed to manuscript drafting and editing, and ensured the accuracy and integrity of the case presentation and discussion.

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