Severe Amlodipine Overdose Presenting with Refractory Hypotension: A Case Report

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***Abstract*—Amlodipine overdose is a life-threatening condition that can lead to severe vasodilation, refractory hypotension, and multi-organ dysfunction. Prompt diagnosis and aggressive management are essential to improve outcomes. We report a case of a 29-year-old female who presented with deliberate self-harm following ingestion of approximately 60 tablets of amlodipine (5mg) corresponding to a total dose of about 300 mg. The patient was admitted with symptoms of dizziness, throat pain, chills, and profound hypotension. On admission, vital signs revealed hypotension (BP 70/50 mmHg), bradycardia (HR 62 bpm initially), and oxygen saturation of 85% on room air. Initial laboratory investigations showed leukocytosis, mild anemia, metabolic abnormalities, and reduced ionized calcium levels.**

The patient required intensive care management including gastric lavage, intravenous fluids, vasopressor support with norepinephrine, calcium gluconate administration, and high-dose insulin therapy with glucose supplementation. Due to persistent hemodynamic instability, mechanical ventilation and multiple sessions of plasmapheresis were performed. Further supportive management included electrolyte correction, antibiotics, and close monitoring of organ function. Imaging studies including chest radiography and echocardiography showed no significant cardiac abnormalities with preserved ejection fraction.

With aggressive supportive care, the patient gradually improved clinically and was successfully extubated. She remained hemodynamically stable and was discharged with appropriate follow-up advice. This case highlights the importance of early recognition and multidisciplinary management in severe amlodipine poisoning.

***Index Terms*—Amlodipine toxicity, Anemia, Hypotension, High-dose insulin therapy, Plasmapheresis, Hyperglycemia.**

I. INTRODUCTION

Calcium channel blockers (CCBs) are widely used in clinical practice for the treatment of cardiovascular conditions such as hypertension, angina, and certain cardiac rhythm disorders. These agents act by inhibiting voltage-dependent calcium channels, which decreases the entry of calcium ions into vascular smooth muscle and cardiac cells, resulting in vasodilation and reduced myocardial contractility.[1] Among the calcium channel blockers, amlodipine is a long-acting dihydropyridine agent frequently prescribed due to its therapeutic efficacy and generally good safety profile. Nevertheless, excessive ingestion of amlodipine may result in significant toxicity, manifested by marked peripheral vasodilation, severe hypotension, and reflex bradycardia, which can further progress to circulatory shock and multiple organ dysfunction.[2]

Toxicity due to calcium channel blockers constitutes a notable proportion of cardiovascular drug overdoses and is linked with considerable morbidity and mortality. In severe cases, patients may present with complications such as persistent hypotension, bradycardia, metabolic abnormalities, pulmonary edema and acute renal failure, which can make clinical management difficult even in intensive care units.[3] Because amlodipine has a relatively long elimination half-life, the toxic manifestations following overdose may persist for an extended duration. Therefore, patients often require intensive supportive management and continuous monitoring. Prompt identification of toxicity and early therapeutic intervention are crucial for improving clinical outcomes.[4]

In patients who develop refractory shock and do not respond adequately to standard therapies such as vasopressors, calcium supplementation, and high-dose

insulin therapy, extracorporeal membrane oxygenation (ECMO) can be utilized as a rescue treatment. This technique offers temporary cardiopulmonary support, maintaining circulation and oxygenation while allowing sufficient time for the elimination of the toxic drug and recovery of myocardial function in severe poisoning cases.[5]

Beyond the typical clinical features, calcium channel blocker toxicity can disrupt pancreatic β-cell activity, resulting in diminished insulin release and consequent hyperglycemia, which is often considered an indicator of severe toxicity. In addition, reduced intracellular calcium availability adversely affects myocardial metabolic processes, thereby impairing cardiac function and exacerbating hemodynamic compromise.[6] The pharmacokinetic characteristics of amlodipine, particularly its extensive protein binding and large volume of distribution, further pose challenges in management, as they reduce the efficacy of conventional elimination methods such as hemodialysis.[7] Consequently, treatment primarily relies on supportive care and frequently necessitates a multimodal therapeutic approach, including interventions like lipid emulsion therapy and extracorporeal life support in critically ill patients. Understanding these underlying pathophysiological mechanisms is crucial for timely and targeted management, ultimately improving patient outcomes in severe cases of calcium channel blocker overdose.[3]

II. CASE PRESENTAION

A 29-year-old female presented to the emergency department with a history of deliberate self-harm following ingestion of approximately 60 tablets of amlodipine 5 mg at her residence. She complained of dizziness, throat pain, and chills prior to hospital admission.

On arrival, the patient was hemodynamically unstable with blood pressure of 70/50 mmHg, heart rate of 62 beats/min, GRBS was 517mg/dl, Liver enzymes got elevated and oxygen saturation of 85% on room air. Due to severe hypotension and cardiovascular instability, she was immediately managed with gastric lavage, intravenous fluids, and vasopressor support with norepinephrine. Further compromised airway reflexes, and the patient required endotracheal intubation and ventilatory support due to worsening hemodynamic status. Initial biochemical investigations revealed electrolyte imbalance and metabolic disturbances. She was transferred to the intensive care unit for further management and continuous monitoring.

Date	Hb (g/dL) (11.5–15)	PCV (%) (36–46)	WBC (×10³/µL) (4–10)	Platelets (×10³/µL) (150–600)	MPV (fL) (6–10)	CREATININE mg/dl (0.6-1.1)	GRBS mg/dl (70-140)
24/01	11.1 ↓	33.9 ↓	23.39 ↑	328	10.9 ↑		517↑
25/01	9.5 ↓	29 ↓	16.14 ↑	246	10.7 ↑	0.67	528↑
26/01	9.9 ↓	31 ↓	14.3 ↑	171	12.3 ↑	1.63	206↑
27/01	7.8 ↓	34.1 ↓	17.20 ↑	142 ↓	12.3 ↑	1.55	194↑
28/01	7.1↓	34.2↓	16.72 ↑	262	11.2 ↑	1.12	190↑
30/01	10.4 ↓	34.2 ↓	10.8 ↑	209	10.3 ↑		106
31/01	11.4 ↓	34.2 ↓	10.30 ↑	229	10.5 ↑		100
01/02	11.8	36.5	9.94	229	10.0		112

Table1: Lab parameters during hospitalization.

During the course of hospitalization, the patient developed progressive anemia, which was suspected to be secondary to medication exposure. The patient had previously received methylene blue therapy as part of the management for refractory vasodilatory shock associated with severe amlodipine poisoning. Post administration of methylene blue the patient developed greenish-blue discoloration of urine, as shown in Image1.Following this treatment, a decline in hemoglobin levels was noted on serial hematological investigations with hemoglobin decreasing from 11.1 g/dL on admission to 7.1 g/dL. Peripheral smear showed normocytic normochromic anemia with neutrophilic leukocytosis as mentioned in table1, and a Direct Coombs test was negative, suggesting non-immune hemolysis, possibly related to

methylene blue therapy administered for refractory vasodilatory shock. The patient received packed red blood cell transfusion and supportive care, after which hemoglobin levels improved. Due to persistent hemodynamic instability from severe amlodipine toxicity, she required endotracheal intubation and mechanical ventilation and was successfully extubated.

Additionally, two sessions of therapeutic plasma exchange (PLEX) were performed as part of advanced supportive management, leading to gradual clinical stabilization. The patient was managed with supportive treatment, including packed red blood cell (PRBC) transfusion, and hematology consultation was obtained. Hemoglobin levels gradually improved following supportive management, and no further complications related to anemia were observed during the hospital stay. Following intensive care management, the patient demonstrated gradual clinical improvement with stabilization of blood pressure and laboratory parameters. She was successfully extubated after improvement in respiratory and hemodynamic status

Ultrasonographic evaluation of the abdomen and pelvis demonstrated bilateral mild pleural effusion with basal lung collapse showed in the image2 and trace free fluid in the pelvic cavity. These findings are commonly observed in critically ill patients experiencing severe circulatory shock. In the context of amlodipine toxicity, profound vasodilation and systemic inflammatory responses can increase vascular permeability, resulting in capillary leak and third-space fluid accumulation. Additionally, aggressive fluid resuscitation administered during the management of refractory hypotension may contribute to pleural and peritoneal fluid collections. Basal lung collapse or atelectasis may occur secondary to compression of the lower lung segments by pleural effusion and reduced lung expansion in mechanically ventilated Follow up radiography showing resolution in image3. Although these findings were mild, they indicated systemic fluid redistribution associated with severe toxicity and intensive supportive therapy. With stabilization of hemodynamics and appropriate clinical management, these radiological abnormalities are often reversible and typically improve as the patient's overall condition recovers. The patient remained stable and was discharged.

Image1: Greenish-blue discoloration of urine

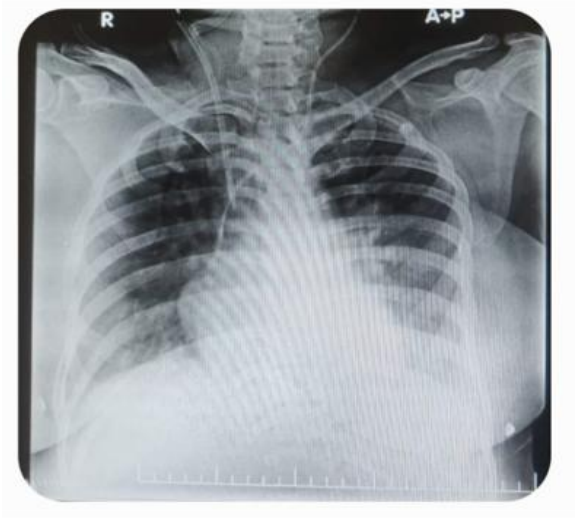

Image2: Bilateral mild pleural effusion with basal lung collapse

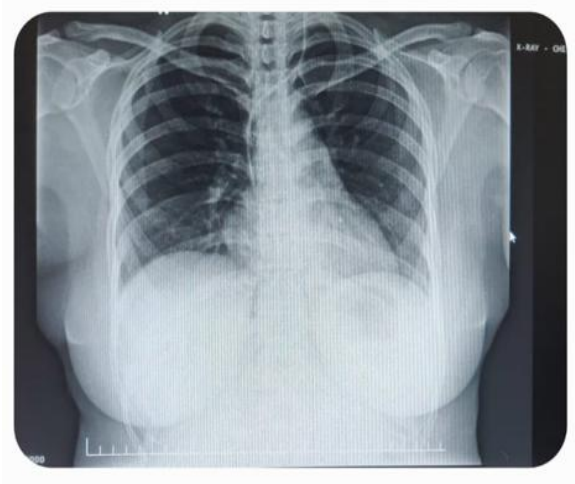

Image3: Follow up radiography showing resolution.

III. DISSCUSSION

Amlodipine overdose can result in severe vasodilatory shock and cardiovascular instability requiring intensive care management. In this case, the patient presented with dizziness and vomiting after consuming approximately 60 tablets of amlodipine (5 mg each). On admission, she had profound hypotension and bradycardia, requiring immediate fluid resuscitation, norepinephrine support, gastric lavage, intravenous calcium, high-dose insulin therapy, and methylene blue. Due to respiratory distress, mechanical ventilation was also required.[3]

Investigations revealed severe anaemia, neutrophilic leucocytosis, transient metabolic and renal disturbances, and elevated markers of tissue injury. Imaging showed bilateral mild pleural effusion with basal lung collapse, trace pelvic free fluid, and mild bilateral hydronephrosis, likely related to systemic inflammation, capillary leak, and aggressive fluid therapy during shock management.

With prompt multidisciplinary treatment and close monitoring, the patient gradually recovered and was discharged in stable condition.

IV. CONCLUSION

Amlodipine overdose is a serious medical emergency that can cause severe hypotension, circulatory collapse, and multiple organ complications due to its prolonged action. In this case, the patient developed refractory shock after consuming a large quantity of amlodipine and required intensive care treatment, including gastric decontamination, intravenous fluids, vasopressors, calcium therapy, high-dose insulin therapy, and ventilatory support. Additional interventions such as methylene blue and therapeutic plasma exchange were needed because of persistent hemodynamic instability. The patient later developed anemia, which was treated with blood transfusion and supportive care, resulting in gradual recovery. This case highlights the importance of early aggressive supportive management, and a multidisciplinary approach in improving outcomes in severe calcium channel blocker toxicity.

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