Refractory amlodipine overdose managed with venoarterial ECMO

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Introduction: Calcium channel blocker (CCB) overdose is a leading cause of cardiovascular drug-related mortality, typically presenting with refractory hypotension, bradycardia, and metabolic acidosis. Standard management includes calcium, high-dose insulin euglycemia therapy (HIET), and vasopressors, while extracorporeal membrane oxygenation (ECMO) serves as rescue therapy in refractory cardiogenic shock. A systematic review reported an 84.6% survival rate with ECMO in CCB overdose, supporting AHA recommendations for VA-ECMO when maximal medical therapy fails. We report a case of severe amlodipine-induced vasoplegic shock successfully managed with VA-ECMO.

Case Presentation: A 37-year-old woman with hypertension and hypothyroidism presented 15 hours after ingesting 220 mg of amlodipine and 6 g of paracetamol in a suicide attempt. Despite aggressive fluid resuscitation, HIET, vasopressors, glucagon, and intralipid therapy, she remained hypotensive and acidotic. She progressed to refractory vasoplegic shock with pulmonary edema requiring mechanical ventilation. Serial blood gases showed worsening acidosis and rising lactate levels. Given persistent hemodynamic instability despite maximal therapy, femoral–femoral VA-ECMO was initiated at the bedside. Hemodynamics rapidly improved, allowing decannulation after three days and subsequent recovery with an uncomplicated discharge.

Discussion: This case underscores the dual pathophysiology of CCB overdose, where amlodipine toxicity produces both vasoplegia and cardiac depression, resulting in mixed cardiogenic-distributive shock. Dihydropyridines cause peripheral vasodilation at therapeutic doses, but significant overdose impairs myocardial contractility. ECMO serves as a bridge to recovery by maintaining organ perfusion during drug clearance. The patient's rapid stabilization after ECMO initiation aligns with literature showing reversal of vasoplegia within 48–72 hours. Her persistent tachycardia reflected predominant vasodilation, consistent with amlodipine toxicity. Although HIET remains first-line therapy, its vasodilatory effect may limit efficacy in profound vasoplegic states. Intralipid therapy provided minimal benefit in this case, reinforcing ECMO as the preferred salvage option in refractory presentations.

Conclusion: Early recognition of refractory shock and timely initiation of VA-ECMO are key to favorable outcomes in severe amlodipine overdose. Prompt multidisciplinary coordination and early ECMO team involvement can prevent irreversible organ damage and improve survival in complex toxicologic emergencies.

Keywords: CCB overdose, ECMO, Amlodipine, dihydropyridine.

List of abbreviation

CCB	Calcium Channel Blocker
HIET	High-Dose insulin Euglycemia Therapy
ECMO	Extracorporeal Membrane Oxygenation
VA-ECMO	Venoarterial Extracorporeal Membrane Oxygenation

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