



# HEMOADSORPTION AS A MANAGEMENT STRATEGY IN AMLODIPINE INTOXICATION: CASE REPORT

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## Background:

Calcium channel blockers (CCB) are easily accessible and widely used drugs for managing hypertension and rhythm disorders<sup>[1]</sup>. In 2020, 6132 cases were reported in the United States<sup>[2]</sup>. The mortality rate approaches 2% at thirty days<sup>[3]</sup>. Amlodipine has an affinity for peripheral vascular smooth muscle cells, leading to vasodilation, hypotension, myocardial depression, hyperglycemia and reduced cardiac glucose utilization<sup>[3,4]</sup>. There is no gold standard for the management of acute poisoning, with limited literature support<sup>[5]</sup>. As it is a highly protein-bound drug, HA is likely to play a role in the therapeutic management of acute poisoning.

## Case Report:

16-year-old female, was admitted to the ER due to ingestion of 500 mg of amlodipine 5 hours before; BP 60/38 mmHg, HR 124x', O2Sat 95%. Lab: metabolic acidemia, AG 19, lactate 5.2 mg/dL, Glucose 252 mg/dL. Detoxification general treatment was initiated (gastric lavage, activated charcoal, and norepinephrine), admitted to the ICU, initial echocardiographic assessment exhibited a Cardiac Output of 1.76 L/min, systolic volume 17 ml. Notably, no evidence of diastolic dysfunction or right ventricular failure was observed. Timeline of key events during ICU stay is depicted in Figure 1. Complementary therapies included intravenous calcium, lipids, and insulin. 7 hours post-intoxication, a decision was made to implement Jafron HA 230 cartridge, 3 sessions, 6 hours each one, Qb: 300 ml/min.

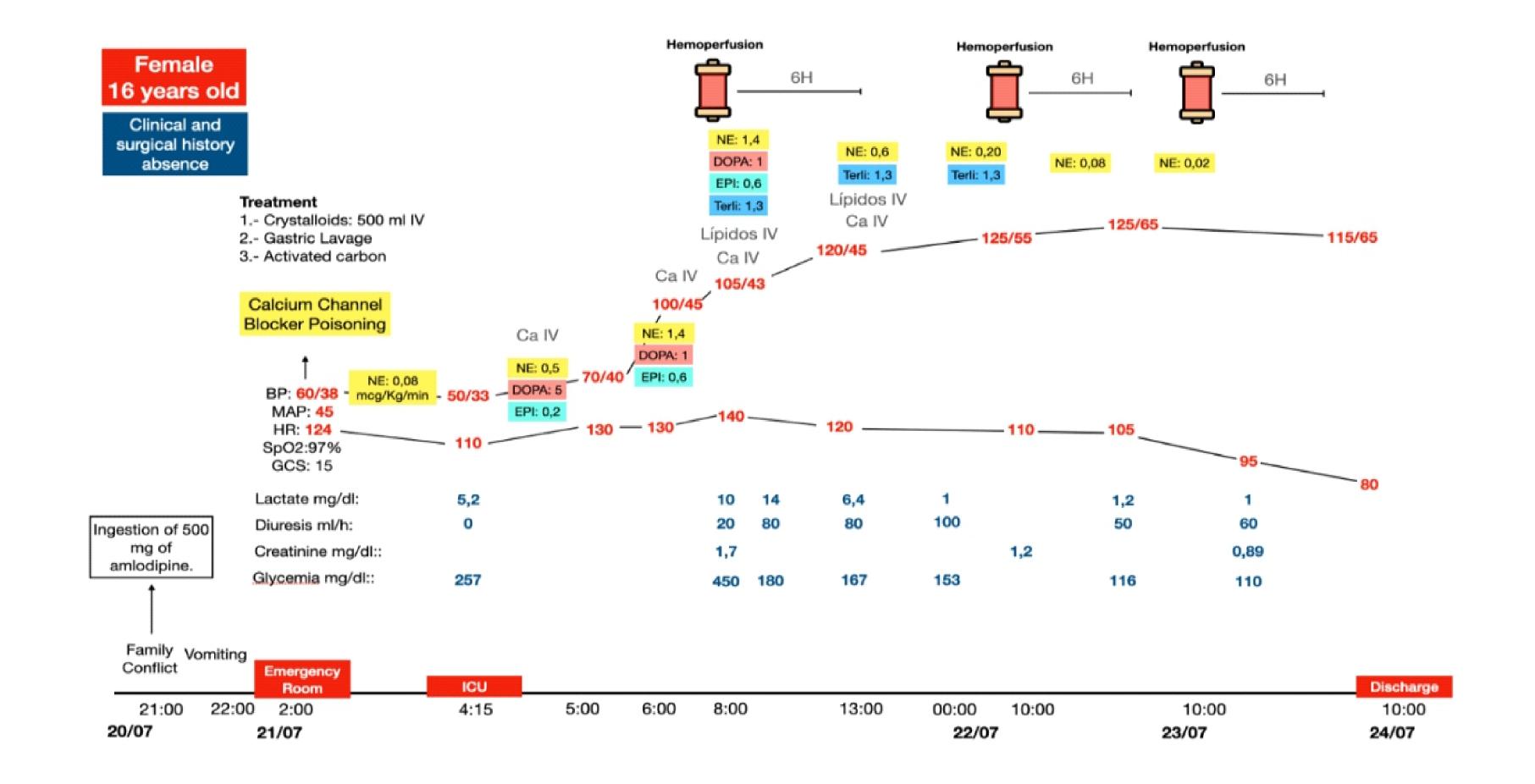
Results and Discussion: Following the HA initial session, we achieved the discontinuation of vasoactive and inotropic drugs. Myocardial contractility was recovered 8 hours later, the withdrawal of vasoactive agents became feasible, and she was discharged from the ICU.

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## Table and Figures: Fig. 1

Timeline of key events during ICU stay.



#### Conclusion:

There is limited evidence regarding the impact of HA in CCB poisonings. However, the lipophilic nature coupled with high protein binding justifies the rationality of its use. A potential benefit with shortened ICU stay, less dependence on vasoactive drugs, and survival that should be evaluated in further studies.

#### Reference:

<sup>[1]</sup> Unger T, et al. 2020 International society of hypertension. Global hypertension practice guidelines. Vol. 38, Journal of Hypertension. Lippincott Williams and Wilkins; 2020. p. 982–1004. [2] Gummin DD, et al. 2020 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 38th Annual Report. Clin Toxicol (Phila). 2021 Dec 1;59(12):1282–501.

<sup>[3]</sup> Christensen MB, et al. Outcomes following calcium channel blocker exposures reported to a poison information center. BMC Pharmacol Toxicol. 2018 Nov 27;19(1). [4] Graudins A, Lee HM, Druda D. Calcium channel antagonist and beta-blocker overdose: Antidotes and adjunct therapies. Br J Clin Pharmacol. 2016 Mar 1;81(3):453–61.

<sup>[5]</sup> Alshaya OA, et al. Calcium Channel Blocker Toxicity: A Practical Approach. Vol. 15, Journal of Multidisciplinary Healthcare. Dove Medical Press Ltd; 2022. p. 1851–62.