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LIFE-THREATENING AMLODIPINE SHOCK REVERSED WITHIN HOURS: A CASE DEMONSTRATING THE HYPERINSULEMIC EUGLYCEMIC THERAPY

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ABSTRACT

Amlodipine, a dihydropyridine calcium channel blocker, is primarily prescribed for managing essential hypertension and angina pectoris. An overdose of calcium channel blockers can lead to severe hypotension and shock. We report a case of a 45-year-old male who was brought to the emergency department with altered mental status and shock after ingesting 200 mg of amlodipine. The patient's circulatory shock was managed with inotropes and high-dose insulin therapy can restore myocardial metabolism and save lives.

KEYWORDS: Calcium channel blockers, Amlodipine, Dihydropyridine, Hypotension, high-dose insulin euglycemia therapy.

INTRODUCTION

Amlodipine is a dihydropyridine calcium channel blocker. Its extended half-life of 30 to 50 hours, large volume of distribution, slower and prolonged action lasting up to 72 hours, minimal negative inotropic effect, and once-daily dosing make it a preferred choice over verapamil or nifedipine. CASE REPORT A 45-year-old male was admitted 12 hr after consuming 40 tablets of amlodipine (5 mg each, total 200mg). Upon admission, the patient exhibited altered mental status. He had a history of hypertension for three years and was on treatment. He had experienced a stroke three years prior and was on antiplatelet therapy. There was no history of diabetes mellitus or cardiac disease. Upon arrival, the patient was drowsy and unresponsive to commands. There was no pallor, cyanosis, clubbing, jaundice, or pedal edema. Vitals: Pulse – 60/min, regular, low volume, BP – 80/60 mm Hg, SPO2 – 98% in room air. CVS – S1S2+ heard, no murmur, RS – B/L NVBS+, P/A – Soft, no organomegaly, no free fluid. CNS – Residual left hemiparesis. Investigations: The patient was treated with a saline bolus, Inj Calcium gluconate, and Inj Noradrenaline infusion. The patient did not respond to noradrenaline. High-dose insulin therapy was initiated for 24 hours. BP improved, and noradrenaline was weaned and stopped 6 hours later. Total count 10800 cells/cumm, Hb- 12 g%, Platelets- 2.1 lacs, Sugar- 100 mg/dl, urea- 32 mg/dl, creatinine 2 mg/dl, Sodium 139 mEq/l, potassium- 3.6 mEq/l, Total bilirubin -1.1 mg/dl, SGOT 138 IU/L, SGPT 38 IU/L.

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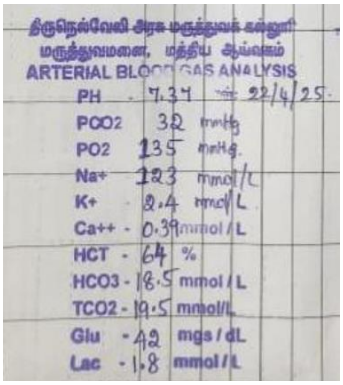

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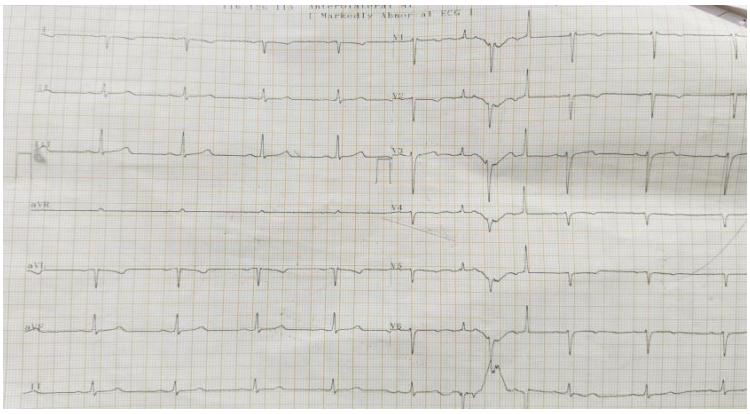

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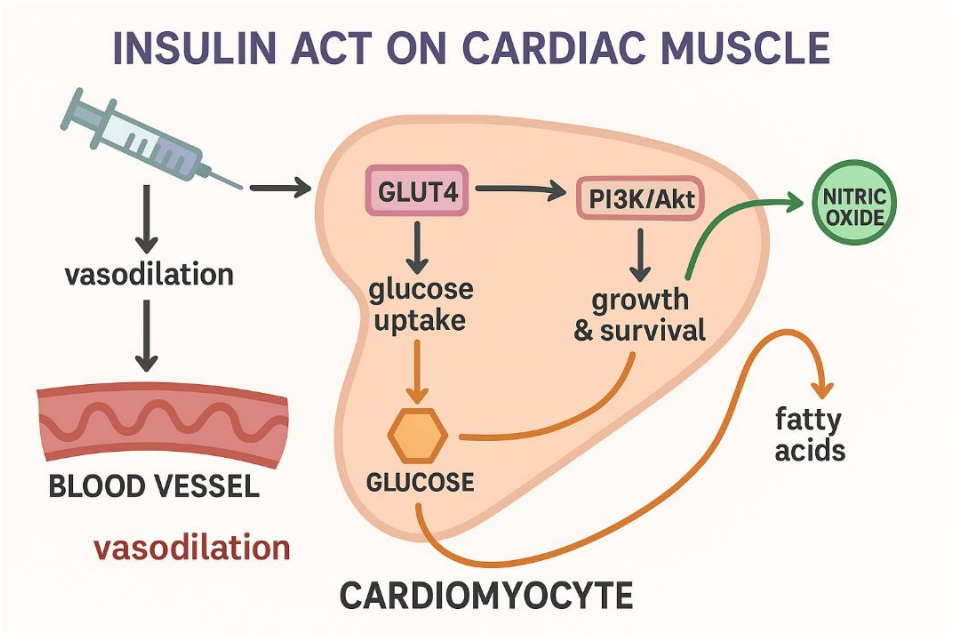

DISCUSSION

Amlodipine toxicity represents a challenging clinical emergency due to its profound vasodilatory effects, associated metabolic derangements, and unpredictable dose–response profile. In our patient, ingestion of 200 mg of amlodipine resulted in significant hypotension and altered sensorium within hours, highlighting that even moderate overdoses may produce severe hemodynamic compromise.

High-dose insulin euglycemia therapy (HIET)0.5unit/kg bolus followed by 1unit/kg/hr infusion played a central role in this patient's rapid stabilization. High-dose insulin therapy improves cardiac performance by rapidly increasing glucose uptake to supply efficient energy to the myocardium, providing a strong non-adrenergic positive inotropic effect without raising oxygen demand, enhancing nitric-oxide–mediated vasodilation to improve systemic and coronary perfusion, and optimizing intracellular calcium handling to further strengthen contractions This aligns with the established understanding that calcium channel blocker toxicity induces hypoinsulinemia and disrupts myocardial carbohydrate utilization, leading to impaired cardiac contractility (1) The prompt improvement in blood pressure following initiation of HIET in our case strongly supports the proposed mechanism of improved myocardial glucose uptake and enhanced inotropy.

Our findings compare favorably with existing literature. In a beta blocker and calcium channel bloker overdose scenario involving intralipid therapy, significant complications such as severe lipemia, laboratory interference, and difficulties with continuous renal replacement therapy were encountered (2).By contrast, our patient was successfully managed without intralipid, thereby avoiding issues related to fat overload, sampling errors, and filter clotting. This reinforces the advantage of HIET as a cleaner, complication-free therapeutic strategy, particularly in isolated amlodipine toxicity.

Comparison with the landmark massive overdose case from the New England Journal of Medicine reveals additional insights. In that report, a 1000 mg amlodipine

ingestion required a highly complex, multi-modal approach including multiple vasopressors, glucagon, pacing, whole-bowel irrigation, and prolonged HIET, with vasopressor weaning achieved over several days (3). In contrast, our case demonstrates that early initiation of HIET alone, combined with a short course of a single vasopressor, led to rapid hemodynamic recovery with noradrenaline discontinued within six hours. This rapid response underscores the importance of early metabolic support and suggests that timely HIET initiation may reduce the need for escalation to advanced adjunctive therapies.

Our management also aligns with guideline-based recommendations for HIET dosing and monitoring detailed in current toxicology reviews (3). By adhering to principles of early insulin bolus administration, continuous infusion, and vigilant monitoring of glucose and potassium, we observed a predictable and favorable response consistent with evidence-based practice. This reinforces HIET as a cornerstone therapy in calcium channel blocker overdose and supports the growing consensus that it should not be delayed until vasopressor therapy fails.

Overall, this case contributes to the expanding body of evidence favoring HIET as an effective, physiologically sound, and relatively complication-free therapy for amlodipine poisoning. It highlights several important clinical messages: (3)moderate amlodipine overdoses may still cause severe shock; (4)early HIET can produce rapid and sustained hemodynamic improvement; (5) avoidance of intralipid may prevent treatment-related complications; and (4) early recognition and timely initiation of metabolic support are critical to optimizing outcomes.

CONCLUSION

Toxicity from calcium channel blockers leads to severe, unresponsive circulatory shock, cardiovascular instability, and metabolic acidosis that may not improve with high doses of vasopressor agents. In such cases, treatments like HIET, glucagon, methylene blue, and calcium chloride can be effective. Other treatment options include extracorporeal membrane oxygenation and plasmapheresis. Prompt treatment with high-dose insulin therapy can restore myocardial metabolism and save lives.

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