Original Research Paper

Volume - 16 | Issue - 08 | August - 2026 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Emergency Medicine

DELIBERATE SELF-HARM–INDUCED MIXED POLYPHARMACY WITH VASOPRESSOR-DEPENDENT DRUG-INDUCED SHOCK AND RECURRENT BRADYCARDIA MANAGED WITH EARLY MULTIMODAL GASTROINTESTINAL DECONTAMINATION: A CASE REPORT

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ABSTRACT Intentional mixed polypharmacy overdose is a life-threatening toxicological emergency characterized by overlapping toxidromes, delayed drug absorption, and unpredictable haemodynamic deterioration. Combined ingestion of beta-blockers, calcium channel blockers, centrally acting alpha-2 agonists, iron preparations, and paracetamol-containing medications presents significant diagnostic and therapeutic challenges requiring prompt recognition and aggressive multidisciplinary management. We report the case of a 41-year-old female who presented approximately two hours after deliberate ingestion of multiple medications, including atenolol, nifedipine extended-release, clonidine, ferrous sulfate, paracetamol, spironolactone, dicyclomine, ofloxacin, cefixime, amoxicillin-clavulanate, norethisterone, furosemide, and a chlorpheniramine-containing cold preparation. On arrival, she was drowsy but arousable with hypotension (80/50 mmHg). Despite immediate resuscitation, she developed vasopressor-dependent drug-induced shock requiring intensive care support for approximately 48 hours. During hospitalization, multiple episodes of symptomatic bradycardia occurred, all responding to intravenous atropine. Early gastrointestinal decontamination consisted of gastric lavage, activated charcoal, and whole bowel irrigation using polyethylene glycol-electrolyte solution (PegLac® 40 g diluted in 250 mL normal saline administered via a Ryle's tube every six hours). Additional management included repeated glucagon and calcium gluconate therapy, intravenous N-acetylcysteine, vasopressor and inotropic support, electrolyte correction, and comprehensive intensive care management. The patient recovered completely without permanent hepatic, renal, neurological, or structural cardiac complications and was discharged following psychiatric evaluation. **Conclusion:** Severe mixed polypharmacy overdose can rapidly progress to life-threatening shock despite initially preserved clinical parameters. Early gastrointestinal decontamination, toxidrome-directed therapy, and aggressive intensive care support are crucial for achieving favourable outcomes.

KEYWORDS : Polypharmacy; Drug overdose; Shock; Whole bowel irrigation.

INTRODUCTION

Intentional drug overdose is a common cause of emergency department visits and remains a significant contributor to poisoning-related morbidity and mortality worldwide. Mixed polypharmacy overdose presents a unique clinical challenge because ingestion of multiple medications with different pharmacological actions produces overlapping toxidromes, making diagnosis and management considerably more difficult than single-agent poisoning. Early recognition of life-threatening toxicity and timely institution of supportive care are essential to reduce complications and improve survival.

Cardioactive medications, particularly beta-blockers, calcium channel blockers, and centrally acting alpha-2 agonists, are among the most potentially lethal drugs encountered in overdose. Beta-blockers reduce myocardial contractility and heart rate, while calcium channel blockers cause profound vasodilatation and myocardial depression. Clonidine further contributes to hypotension, bradycardia, and central nervous system depression by suppressing sympathetic outflow. Simultaneous ingestion of these agents can produce severe mixed cardiogenic and vasodilatory shock requiring prolonged intensive care support.

Management becomes even more challenging when sustained-release preparations are involved because delayed gastrointestinal absorption may result in progressive haemodynamic deterioration several hours after ingestion. Iron-containing preparations further increase the risk of gastrointestinal injury and systemic toxicity. Although whole bowel irrigation is not routinely indicated for all poisoning cases, it remains an important therapeutic option in carefully selected patients presenting early after ingestion of sustained-release formulations or iron-containing medications.

We report a rare case of severe mixed polypharmacy overdose complicated by prolonged vasopressor-dependent drug-induced shock and recurrent atropine-responsive bradycardia that was successfully managed with early multimodal gastrointestinal decontamination and comprehensive critical care support. This case highlights the importance of anticipating delayed cardiovascular deterioration and demonstrates the value of prompt, multidisciplinary management in severe poisoning.

CASE REPORT

A 41-year-old female was brought to the Emergency Department approximately two hours after an alleged deliberate ingestion of multiple medications in a suicide attempt. According to her relatives, the exact quantity of tablets consumed could not be determined. The reportedly ingested medications included atenolol, nifedipine extended-release (30 mg), clonidine (100 µg), paracetamol (500 mg

and 650 mg formulations), ferrous sulfate, spironolactone (50 mg), dicyclomine, ofloxacin, cefixime, amoxicillin-clavulanate, norethisterone, furosemide, and a chlorpheniramine-containing cold preparation.

On arrival, the patient was drowsy but arousable to verbal commands with a Glasgow Coma Scale (GCS) score of 14/15 (E4V4M6). Her pupils were equal and reactive to light. Initial vital signs revealed blood pressure 80/50 mmHg, pulse rate 74 beats/minute, respiratory rate 20 breaths/minute, oxygen saturation 98% on room air, and a random blood glucose of 182 mg/dL. Cardiovascular examination revealed normal heart sounds without murmurs, respiratory examination demonstrated bilateral equal air entry without added sounds, abdominal examination was unremarkable, and neurological examination showed no focal motor or sensory deficits.

Based on the history of ingestion of multiple cardioactive medications together with severe hypotension, mixed drug-induced cardiodepressant and vasodilatory shock was suspected. Immediate resuscitation was initiated according to Advanced Cardiac Life Support principles. Two large-bore intravenous cannulas were secured, blood samples were obtained for baseline laboratory investigations, and continuous electrocardiographic and haemodynamic monitoring was commenced. The patient was admitted to the intensive care unit for close observation because of the anticipated risk of delayed toxicity from sustained-release medications.

Considering the early presentation and reported ingestion of sustained-release nifedipine together with ferrous sulfate, aggressive gastrointestinal decontamination was undertaken. Gastric lavage was performed, followed by administration of activated charcoal. Whole bowel irrigation was subsequently initiated through a Ryle's tube using polyethylene glycol-electrolyte solution (PegLac® sachet, 40 g diluted in 250 mL normal saline) administered every 6 hours. A total of six doses were administered with the objective of reducing continued gastrointestinal absorption of sustained-release and iron-containing preparations.

Despite adequate crystalloid resuscitation, the patient remained hypotensive and progressed to vasopressor-dependent drug-induced shock. High dose noradrenaline and dopamine infusion was started and subsequently titrated according to haemodynamic response. Vasopressor and inotropic support was required for approximately 48 hours before gradual haemodynamic stabilization was achieved. Continuous invasive arterial blood pressure monitoring and serial arterial blood gas analyses were performed throughout the critical phase of illness. During the ICU stay, the patient developed multiple episodes of symptomatic bradycardia, each accompanied by worsening hypotension. Every episode responded promptly to intravenous atropine, restoring heart rate and haemodynamic stability without the need for temporary transvenous pacing. Because combined beta-blocker and calcium channel blocker toxicity was strongly suspected, repeated doses of intravenous glucagon and calcium gluconate were administered as targeted toxicological therapy. Intravenous N-acetylcysteine was also initiated according to the standard treatment protocol because the exact quantity of paracetamol ingested could not be reliably determined.

Supportive management included intravenous electrolyte replacement for severe hypokalaemia, proton pump inhibitor therapy, antiemetics, maintenance intravenous fluids, thromboprophylaxis, stress ulcer prophylaxis, and meticulous neurological monitoring. The patient received multidisciplinary care involving specialists from Emergency Medicine, Critical Care, Cardiology, Gastroenterology, Nephrology, Psychiatry, and Clinical Toxicology.

Investigations

Initial laboratory investigations demonstrated transient leukocytosis ($16.08 \times 10^3/\mu L$) and significant hypokalaemia (2.60 mmol/L), which further decreased to 2.17 mmol/L before correction with intravenous potassium supplementation. Arterial blood gas analysis showed mild metabolic acidosis with elevated serum lactate, consistent with tissue hypoperfusion secondary to shock.

Peak serum iron concentration reached 365.9 µg/dL, confirming significant iron exposure. However, the patient did not develop severe systemic iron toxicity, hepatic failure, gastrointestinal haemorrhage, or refractory metabolic acidosis. Renal and hepatic function remained preserved throughout hospitalization.

Serial electrocardiograms demonstrated sinus rhythm without high-grade atrioventricular block or malignant ventricular arrhythmias. Serial cardiac troponin-I levels remained negative. Transthoracic echocardiography showed normal cardiac chamber dimensions with preserved biventricular systolic function, confirming that the persistent hypotension was toxin-mediated rather than secondary to structural cardiac disease.

Table 1. Serial Laboratory Investigations

Table 1. Laboratory Trends During Hospitalization

Parameter	At Admission	Worst / Peak Value	At Discharge
Hemoglobin (g/dL)	13.3	12.0	12.2
Total Leukocyte Count ($\times 10^3/\mu L$)	16.08	16.08	10.26
Platelet Count ($\times 10^3/\mu L$)	400	263	294
INR	1.10	1.24	1.18
Serum Creatinine (mg/dL)	0.8	0.9	0.6
Sodium (mmol/L)	136.5	144.6	138
Potassium (mmol/L)	2.60	2.17	4.03
Serum Lactate (mmol/L)	2.51	3.50	1.00
Serum Iron (µg/dL)	106.7	365.9	179.3
AST (U/L)	16	14	12
ALT (U/L)	12	14	11
Troponin-I	Negative	Negative	Negative

DISCUSSION

Mixed polypharmacy overdose is associated with significant morbidity and mortality because simultaneous ingestion of drugs with different mechanisms of action produces complex and often unpredictable clinical manifestations. Cardioactive drugs such as beta-blockers, calcium channel blockers, and clonidine are particularly dangerous when ingested together because they produce additive myocardial depression, vasodilatation, and suppression of sympathetic activity, resulting in profound hypotension and circulatory collapse. Early recognition and prompt supportive management are therefore essential to improve survival.

In the present case, the patient developed vasopressor-dependent drug-induced shock requiring continuous haemodynamic support for approximately 48 hours. The persistent hypotension was likely multifactorial, resulting from the combined effects of atenolol-induced negative chronotropy and inotropy, nifedipine-induced peripheral vasodilatation with myocardial depression, and clonidine-induced suppression of central sympathetic outflow. The synergistic action of these medications resulted in prolonged cardiovascular instability despite early resuscitative measures.

Table 2. Timeline of Clinical Events

Table 2. Timeline of Key Clinical Events

Time (Approx.)	Event
0 hour	Ingestion of multiple medications
~2 hours	Presentation to Emergency Department
~2–3 hours	Gastric lavage, activated charcoal and whole bowel irrigation initiated
First 48 hours	Vasopressor support and 6 episodes of bradycardia treated with atropine
Day 3	Weaned off vasopressors, shifted out of ICU
Day 6	Discharge after psychiatric assessment

An important clinical feature was the occurrence of multiple episodes of symptomatic bradycardia, all of which responded to intravenous atropine. Although atropine has variable efficacy in severe beta-blocker toxicity, it remains an appropriate first-line treatment for symptomatic bradycardia. The favourable response in our patient may have been due to the predominant vagotonic effects of clonidine and the absence of irreversible myocardial conduction abnormalities. Temporary transvenous pacing or extracorporeal circulatory support was therefore not required.

Another notable aspect of this case was the early institution of multimodal gastrointestinal decontamination. Because the patient presented within two hours of ingestion and had consumed sustained-release nifedipine together with ferrous sulfate, gastric lavage and activated charcoal were followed by whole bowel irrigation using polyethylene glycol-electrolyte solution (PegLac® 40 g diluted in 250 mL normal saline administered via a Ryle's tube every six hours; total six doses). Whole bowel irrigation is not routinely recommended for all poisoning cases; however, current toxicology guidelines support its selective use following ingestion of sustained-release formulations and iron-containing preparations in appropriately selected patients. Early initiation of this therapy may have reduced continued gastrointestinal drug absorption and contributed to the favourable outcome observed in our patient.

Targeted pharmacological therapy further contributed to successful management. Repeated intravenous glucagon was administered because of suspected beta-blocker toxicity, while intravenous calcium gluconate was given to improve myocardial contractility and vascular tone in suspected calcium channel blocker overdose. N-acetylcysteine was administered because the exact quantity of paracetamol ingested was unknown, thereby minimizing the risk of delayed hepatotoxicity. Despite a peak serum iron concentration of 365.9 µg/dL, the patient did not develop severe systemic iron toxicity, suggesting that early gastrointestinal decontamination and aggressive supportive care may have limited further absorption.

Previous case reports describing combined beta-blocker and calcium channel blocker overdose have reported prolonged hypotension requiring vasopressor therapy, high-dose insulin euglycaemic therapy, lipid emulsion therapy, temporary pacing, or extracorporeal membrane oxygenation. In contrast, our patient recovered completely with early gastrointestinal decontamination, repeated glucagon and calcium therapy, vasopressor support, and meticulous intensive care management, without requiring advanced mechanical circulatory support. This favourable outcome emphasizes the importance of prompt recognition, early intensive care admission, and individualized toxidrome-directed therapy.

This case also highlights the value of a multidisciplinary approach, involving emergency physicians, intensivists, cardiologists, gastroenterologists, psychiatrists, nursing staff, and clinical laboratory services. Early coordination between specialties facilitated rapid diagnosis, continuous haemodynamic monitoring, timely therapeutic interventions, and comprehensive post-recovery psychiatric care.

CONCLUSION

Mixed polypharmacy overdose involving beta-blockers, calcium channel blockers, clonidine, iron preparations, and paracetamol can rapidly progress to life-threatening vasopressor-dependent drug-induced shock despite initially preserved clinical parameters. Early recognition, prompt multimodal gastrointestinal decontamination, toxidrome-directed pharmacological therapy, and aggressive intensive care management can result in complete recovery without permanent organ dysfunction. Clinicians should anticipate delayed cardiovascular deterioration following sustained-release drug ingestion and institute prolonged monitoring in all high-risk patients.

LEARNING POINTS

- Mixed polypharmacy overdose may initially appear deceptively stable before progressing to severe haemodynamic collapse.
- Combined beta-blocker, calcium channel blocker, and clonidine toxicity can produce prolonged vasopressor-dependent shock requiring intensive care management.
- Whole bowel irrigation using polyethylene glycol-electrolyte solution should be considered in selected patients presenting early after ingestion of sustained-release and iron-containing preparations.
- Early toxidrome-directed therapy, including atropine, glucagon, calcium supplementation, and appropriate supportive care, can prevent progression to irreversible cardiovascular collapse.
- A multidisciplinary approach, together with mandatory psychiatric evaluation after recovery, is essential for optimal patient outcomes.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical information. Every effort has been made to maintain patient confidentiality by removing all identifying information.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

SOURCE OF FUNDING

No financial support or external funding was received for this work.

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