



Overdose Aftermath



Michael Matchett, BSc; and Rob Green, BSc, MD, DABEM, FRCPC

Jason's case

Jason, 46, presents to the ED after ingesting a handful of pills. He states that he broke up with his girlfriend earlier that day and has ingested alcohol periodically since. He has a history of hypertension, erectile dysfunction and is a smoker. His medications include sildenafil and verapamil.

Examination

A medical examination notes the following:

- BP: 150/80 mmHg
- Heart rate: 49 bpm
- Respiratory rate: 16 breaths per minute

Jason is somewhat drowsy, but his O₂ saturations are 96% on room air. Ten minutes after the evaluation, Jason is found to be:

- hypotensive (BP is 88/30 mmHg),
- bradycardic (heart rate is 42 bpm) and
- at a decreased level of consciousness (Glasgow coma score 10).

Questions

1. What are the priorities in the management of Jason?
2. What is the differential diagnosis of a bradycardic patient who has ingested a toxin?
3. What calcium channel blocker (CCB) overdose (OD) is most common and how does it present?
4. What medications can be used in the management of an unstable CCB OD?
5. What other therapies are available?
6. What is the role of glucose-insulin infusions in CCB ODs?

Read on to learn more...

Questions & Answers

1. What are the priorities in the management of Jason?

All critically ill patients should be managed by systematically assessing their airway, breathing and circulation. The patient should be placed in an acute care area of the ED and continuous O₂ saturation and cardiac monitors should be put into place. Peripheral IV catheters should be inserted immediately and central venous access should be obtained when appropriate for fluid and pharmacologic intervention. The patient should be monitored for a decline in level of consciousness (LOC) during the initial resuscitation. If necessary, endotracheal intubation should be performed early to ensure airway patency and adequate ventilation. Serum glucose levels should also be assessed as soon as possible and opiate antagonist administration (naloxone) should be considered in the event of a co-ingestion of an opioid analgesic.

All patients with suspected calcium channel blocker (CCB) overdose (OD) require aggressive care and admission to a monitored setting.

2. What is the differential diagnosis of a bradycardic patient who has ingested a toxin?

The differential diagnosis of a patient presenting with bradycardia following a toxin ingestion should include:

- β -blocker ingestion
- CCB ingestion
- Cardiac glycoside ingestion (e.g., digoxin)
- Other antidysrhythmics (Types IA and IC)
- Clonidine ingestion
- Opioid ingestion
- Organophosphate ingestion
- Barbiturate ingestion

All patients with a suspected CCB OD require aggressive care and admission to a monitored setting.

3. What CCB OD is most common and does it present?

The most common CCB OD is verapamil. Many of the clinical manifestations of a verapamil OD are extensions of the therapeutic effect of the drug. Patients may present with marked hypotension, which is often secondary to a reduction in cardiac output and peripheral vasodilation. Furthermore, a decreased cardiac output may result in a decreased LOC on presentation. Bradycardia is often a clinical feature of a verapamil OD, caused by the depression of the conducting systems of the heart. Lactic acidosis may be present secondary to systemic hypoperfusion, also as a result of decreased cardiac output. Hyperglycemia may be present secondary to the inhibition of calcium-dependent insulin secretion.

4. What medications can be used in the management of an unstable CCB OD?

The following is a list of possible medications to use in the management of an unstable CCB OD:

- **Fluid bolus:** crystalloid (20 ml/kg of normal saline or Ringer's lactate) fluid resuscitation is recommended as the initial treatment for hypotensive patients
- **Atropine:** atropine administration (0.5 mg to 1 mg, up to 3 mg for adults and 0.02 mg/kg for children, minimum 0.1 mg) may transiently increase heart rate and is a reasonable initial management option
- **Calcium salts:** calcium gluconate or calcium chloride (10 ml of 10%) may be administered through peripheral venous access and repeated as necessary if the patient's vital signs improve
- **Adrenergic agents:** patients who do not respond adequately to calcium salt infusion may be given adrenergic agents, such as dopamine (5 µg/kg to 20 µg/kg per minute) or norepinephrine (0.5 µg/kg to 3.0 µg/kg per minute). Generally, these agents should be considered when the patient's BP does not adequately respond to initial treatment

Mr. Matchett is a Second Year Medical Student, Dalhousie Medical School, Halifax, Nova Scotia.

Dr. Green is an Associate Professor, Dalhousie University and Emergency Physician and Intensivist, Queen Elizabeth II Health Sciences Centre, Halifax, Nova Scotia.

Fluid bolus, atropine, calcium salts, adrenergic agents and glucagon are all possible medications to use in the management of an unstable CCB OD.


- **Glucagon:** glucagon should be administered initially as an IV bolus of 0.05 mg/kg, which may be repeated every 10 minutes (as needed) to induce a response. Following a response, an IV infusion of 1 mg to 10 mg per hour may be considered. The adverse effects of glucagon are hyperglycemia and vomiting

5. *What other therapies are available?*

Other therapies include decontamination and hemodynamic support. For decontamination, activated charcoal (1g/kg) may be considered if the ingestion has occurred less than two hours prior to presentation.

For cases of CCB ODs which are unresponsive to pharmacologic therapy, mechanical hemodynamic support, such as an intra-aortic balloon pump or cardiopulmonary bypass, have been used to support patients.

6. *What is the role of glucose-insulin infusions in CCB ODs?*

Recent evidence suggests that high-dose insulin infusions are beneficial in a CCB OD which are unresponsive to other modalities. The administration of an insulin bolus of 1 unit/kg, accompanied with 50 cc of 50% glucose, should be considered. Subsequent infusions of insulin at 1.0 unit/kg/hour with a 10% dextrose in water (D10W) infusion at 200 mL to 300 mL per hour should be maintained until calcium-dependent myocardial suppression is minimized. Serum glucose and serum potassium levels should also be closely monitored throughout treatment. High dose insulin infusions should be considered early in the management of severe OD. 

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