emDocs

The EM Educator Series

The EM Educator Series: Calcium Channel Blocker Overdose

Author: Alex Koyfman, MD (@EMHighAK) // Edited by: Brit Long, MD (@long_brit) and Manpreet Singh, MD (@MprizzleER)

Case 1:
A 62-year-old male presents from home. He has a history of hypertension. He thinks he made a mistake and took too much of one of his blood pressure medication. He is bradycardic and hypotensive.

Case 2:
A patient is brought in by EMS with weakness and altered mental status. Empty pill bottles were found at the scene. As you are assessing the patient, he suddenly becomes bradycardic and hypotensive.

Case 3:
A 18-year-old female presents with intentional overdose, but she is not sure what she took. She is bradycardic and hypotensive.

Questions for Learners:

1. What is the differential diagnosis for hypotension and bradycardia
2. What are clinical presentations of patient status post CCB overdose?
3. What does management include? ABCs, IV fluids, calcium, vasopressors, high dose insulin therapy?
4. Does lipid emulsion and/or ECMO have a role?
5. Is there any utility in atropine, glucagon, transcutaneous or transvenous pacing?
6. Who can be discharged? Admitted to telemetry? ICU?

Suggested Resources:

- Articles
  - EM@3AM - CCB Overdose
  - First10EM - CCB Overdose
  - Emergency Medicine Cases
  - EMCrit
  - CoreEM - HIET
- Journal Articles
  - Crit Care Med - Expert Consensus Recommendations
  - EM Clinics NA - Toxin-Induced CV Failure

Answers for Learners:

1. What is the differential diagnosis for hypotension and bradycardia

Non-toxicological causes:

- MI with cardiogenic shock
- Hyperkalemia
- Myxedema coma
- Spinal cord injury
- Hypothermia

Toxicological causes:

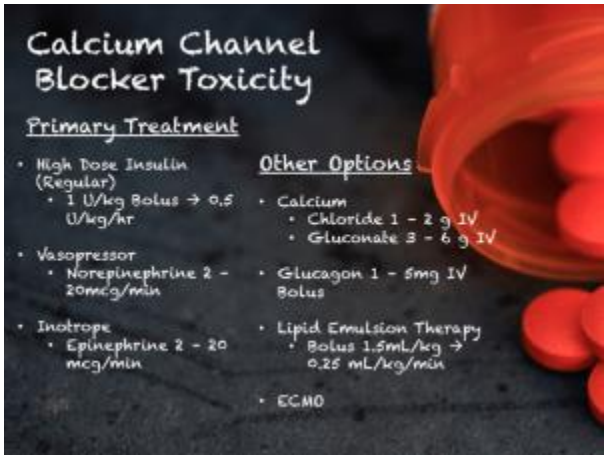
- Calcium channel blockers
- Beta-blockers
- Digoxin
- Opiates
- Alpha-2 antagonists (e.g., clonidine)
- Sodium channel blockers (e.g., TCA, carbamazepine, flexeril, antipsychotics, propranolol, cocaine)

2. What are clinical presentations of patient status post CCB overdose?

Pathophysiology and Clinical Presentation: Calcium is vital to a number of physiologic processes – an understanding of which yields insight regarding patient presentation and subsequent treatment:[2]

- Cardiac:
    - Sinoatrial nodal and atrioventricular nodal tissue: slow inward calcium channels (and to a lesser extent, sodium channels) responsible for action potential generation.
    - Cardiac myocyte: calcium entry by L-type or voltage-gated calcium channels => calcium induced calcium release from intracellular organelles => excitation-contraction coupling.
- Vascular:
    - Calcium required for the maintenance of vascular smooth muscle tone.
- Metabolic:
    - Shock state induced by cardiac calcium insufficiency => increased free fatty acid metabolism and liver glycogenolysis.
    - Pancreatic β-islet cells: require calcium for insulin release. Insulin required for glucose uptake (cell specific: skeletal muscle, etc.). Lack of insulin => Krebs cycle shift to anaerobic metabolism (pyruvate production).
- Presentation: bradycardia, conduction abnormalities, hypotension, possible shock, hyperglycemia, acidosis. (Note: patients with dihydropyridine CCB toxicity frequently present with reflex tachycardia – see below.)

3. What does management include? ABCs, IV fluids, calcium, vasopressors, high dose insulin therapy?



Approach to the Patient with a Beta Blocker or Calcium Channel Blocker Overdose

Apply monitors, obtain IV access, check blood glucose and obtain an EKG. Consider co-ingestions. Involve your local poison centre early.

↓ Observe for signs of toxicity

- **Decontamination**
  - Activated charcoal if patient protecting airway and based on timing of ingestion and formulation of drug (i.e., < 1 hour post-ingestion or longer if slow-release/enteric coated)
  - Dose charcoal based on a ratio of 1:10 of drug ingested to charcoal

↓ Hypotension and bradycardia

- **Fluids** for hypotension
- **Atropine/Transcutaneous Pacing** if bradycardia present
- **Calcium**
  - Calcium gluconate (peripheral IV), or calcium chloride (central line)
- **Pressors** (in patients with hemodynamic instability, until insulin infusion takes effect)
  - Epinepherine if poor cardiac contractility on bedside ultrasound
  - Norepinepherine if normal cardiac contractility on beside ultrasound

↓

High Dose Insulin Therapy:
- Bolus 1 U/kg of regular insulin by IV push with 2 amps of D50W
- Infusion of 1U/kg/h of regular insulin with dextrose infusion if blood glucose < 11. Titrate insulin infusion up every 15 minutes by 0.25-0.50U/kg/h until adequate cardiac contractility.
- Monitor blood glucose every 15 minutes until stable, then monitor hourly. Check potassium frequently.

↓ Refractory shock or peri-arrest

- **Lipid Emulsion Therapy**
  - 1.5mL/kg of 20% lipid emulsion as a bolus, repeated up to two times as needed until clinically stable. Then an infusion of 0.25mL/kg/min for 30-60 minutes is given.
- **ECMO**

4. Does lipid emulsion and/or ECMO have a role?

Toxicity refractory to initial treatment:

- Incremental increase in HIET up to 10U/kg/hr.
- Lipid emulsion therapy: 1.5 mL/kg of 20% lipid emulsion administered as a bolus; repeat up to two times as needed to achieve clinical stability.
  - Followed by infusion of 0.25mL/kg/min for 30-60 min.
- In the absence of myocardial contractility dysfunction, consider pacemaker if bradycardia or high grade AV block.
- Consult for venoarterial extracorporeal membrane oxygenation (VA-ECMO) if available.

Lipid emulsion therapy (intralipid) is a management option for patients who have overdosed on a lipid soluble drug (e.g., lidocaine/bupivacaine, calcium channel blockers, amitriptyline, seroquel, buproprion) who are in refractory shock or peri-arrest.

There are downsides to lipid therapy including complications such as pancreatitis and pulmonary fat emboli. Electrolytes, blood gases etc. cannot be measured in lipemic serum.

Intralipid treatment should be reserved for lipophilic drug poisoning with:

1. Hypotension or
2. Dysrhythmias causing hemodynamic instability (not responsive to sodium bicarbonate or lidocaine) or
3. Seizures unresponsive to usual treatments

There is no role for lipid emulsion therapy

- as prophylaxis
- in isolated altered mental status or coma
- as 1st line therapy

How do you give lipid emulsion therapy?

Draw up 100mL from a 500mL bag of lipid emulsion and give as an IV bolus, then run the remaining 400mL over 30mins.

5. Is there any utility in atropine, glucagon, transcutaneous or transvenous pacing?

Atropine

Consider a trial of 0.5mg IV atropine in patients with bradycardia.

What about cardiac pacing? In the overdose patient, transcutaneous pacing is unlikely to be successful, but may be attempted. If transcutaneous pacing is unsuccessful, it is generally agreed that transvenous pacing should be avoided in the patient with a slow and low poisoning as it may precipitate dysrhythmias in the overdose patient with an 'irritable' heart.

Glucagon

Glucagon may be considered as a last resort. Our experts do *not* recommend the routine use of glucagon in beta blocker overdoses. It can worsen hypotension and bradycardia as well as cause vomiting which increases the risk of aspiration.

6. Who can be discharged? Admitted to telemetry? ICU?

Symptomatic patients required ICU-level care. All asymptomatic patients in whom concern for ingestion of SR formulations exist = admission for 24 hour monitoring.