emDocs

The EM Educator Series

The EM Educator Series: Beta-Blocker Overdose

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Case 1: A 45-year-old male is brought in by EMS for weakness and hypotension. He is also bradycardic and hypoglycemic en route.

Case 2: A 38-year-old female is brought in by family for recurrent seizures at home after intentional overdose. Her husband brought in an empty pill bottle, which appears to be propranolol.

Questions for Learners:

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

Suggested Resources:

- Articles
  - CoreEM – HIET
  - EM@3AM
  - Emergency Medicine Cases Low and Slow Poisoning
  - LIFTL - Beta Blocker Overdose
  - REBEL EM - Cardiotoxic Drugs
  - WikEM
- Journal Articles
  - High dose insulin therapy and lipid emulsion for cardiotoxic drugs
  - 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 beta blocker overdose? How is propranolol different from other beta blockers in overdose?**

Clinical Presentation: Bradycardia (most common initial sign), hypotension, altered mental status, respiratory arrest, dysrhythmias (atrioventricular block (AV) (beta-blockers capable of fast sodium channel blockade), or ventricular fibrillation. Hypoglycemia is more common in the pediatric population. Seizures are often witnessed following toxic propranolol exposure.

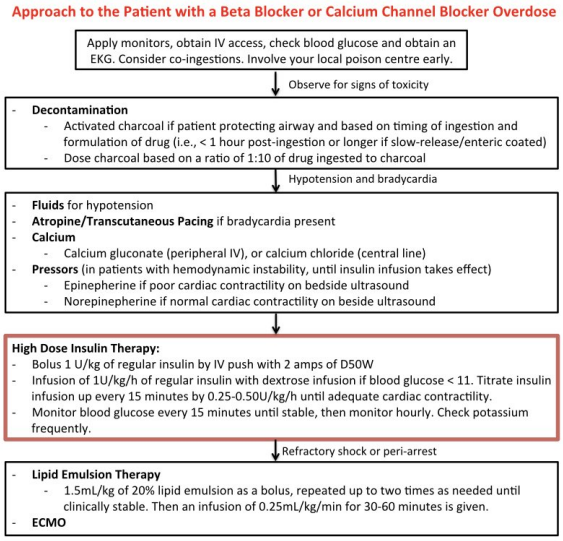
- Note: Acebutolol, oxprenolol, and pindolol exhibit intrinsic sympathomimetic activity => may manifest as sinus or ventricular tachycardia.
- Labetolol: significant exposure likely to result in hypotension and distributive shock (α1 blockade).

Propranolol is highly lipophilic ➔ CNS penetration and toxicity secondary to sodium channel blockade = toxic exposures associated with the highest fatality rate amongst beta-blockers.

3. **How does beta blocker overdose differ from calcium channel blocker overdose?**

There are two key features that may help differentiate a calcium channel blocker overdose from a beta blocker overdose: **Blood glucose** and **level of consciousness**. Calcium channel blocker overdoses tend to cause *hyperglycemia*, compared to a normal-to-low blood glucose in beta blocker overdoses. Beta blocker overdoses tend to cause a depressed level of consciousness, and calcium channel blocker overdoses, a normal level of consciousness.

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



- Initiate fluid resuscitation.
- Consider vasopressors (epinephrine and norepinephrine first line) for refractory hypotension to fluids.
- **Atropine** advised for symptomatic bradycardia:
    - Adult dose: 0.5-1 mg IV bolus. Pediatric dose: 0.02 mg/kg IV bolus (min 0.1 mg, max 3 mg).
- Refractory hypotension: consider **calcium** administration (final common pathway for stimulation of beta-andrenergic receptors = increase in intracellular calcium concentration; beta-blockade limits this process).
    - Adult dose: Calcium gluconate 10%, 3 g (30 mL) IV bolus. Calcium chloride 10%, 1 g (10 mL) IV bolus. Pediatric dose: Calcium gluconate 10%, 60 mg/kg (0.6 mL/kg (max: adult dose). Calcium chloride 10%, 20 mg/kg (0.2 mL/kg) (max: adult dose).
- **High-Dose Insulin Euglycemia Therapy (HIET)**: Insulin is an inotrope thought to optimize the use of carbohydrates by cardiac myocytes and modulate intracellular calcium.
    - Adult and pediatric dosing: 1 U/kg/hr titrated to 2 U/kg/hr every 10 minutes until a max of 10 U/kg/hr is reached.
        - Recommended: Administer a bolus of dextrose prior to insulin infusion (0.5 g/kg IV) if blood glucose < 200 mg/dL.
        - Initiate a dextrose drip (D25 or D50).

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

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.

→ If a patient is refractory to all of the above measures => consider extracorporeal membrane oxygenation (ECMO). ECMO is compatible with HIET and ILE.

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

Atropine advised for symptomatic bradycardia:

- Adult dose: 0.5-1 mg IV bolus. Pediatric dose: 0.02 mg/kg IV bolus (min 0.1 mg, max 3 mg).

Glucagon: Displays inotropic and chronotropic effects which function independently of beta-adrenergic stimulation; counteracts beta-blocker induced hypoglycemia => utilized as a bridge to high-dose insulin therapy.

- Adult dose: 5-10 mg IV bolus. Pediatric dose: 0.05-0.1mg/kg (max: adult dose).
    - Effects are transient: half-life approximately 20 minutes.

Refractory bradycardia: consider **transcutaneous** or **transvenous** pacing. (Note: Unlikely to improve perfusion if stroke volume is significantly reduced. Recommend bedside echo prior to initiation).

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

Monitor patients carefully for hypoglycemia (glucose q15 mins for first hour then q1h), hypokalemia and volume overload.

- Symptomatic patients require ICU level care.
- A patient who remains asymptomatic for 6 hours following an alleged toxic ingestion of a normal-release preparation: refer for psychiatric evaluation vs. home with parental guidance (pediatric).1
- Asymptomatic patients reporting ingestion of sustained release formulations: admit for 24-hour monitoring.