

# **CALCIUM CHANNEL BLOCKERS OVERDOSE**

*(Last updated October 2020; Reviewer: Alzbeta Glancova, MD; Aysun Tekin, MD; Ognjen Gajic, MD)*

**PRESENTING COMPLAINT:** Lightheadedness, syncope, chest pain.

## **FINDINGS**

- **A** Check airway
- **B** ↓/↑/N RR
- **C** ↓ BP, ↓ HR
- **D** Altered variable (V,P,U,D)\*
- **E** Flushing, diaphoresis, abdominal pain and distention
- **U<sub>PC</sub>** pulmonary edema, LV dysfunction
- **L<sub>PC</sub>** ↑ glucose, ↓ calcium, ↓ potassium, ↑ lactate, ABG: ↓ pH, ↓ HCO<sub>3</sub>, ↓ CO<sub>2</sub>

\*V (verbal), P (Pain), U (unconsciousness), D (delirious)

L<sub>PC</sub> (point-of-care labs), U<sub>PC</sub> (point-of-care ultrasound)

## **OTHER HISTORY**

### **Signs & Symptoms:**

- Negative inotropic & chronotropic effects with systemic vasodilatation leading to hypotension & bradycardia.
- Progression to altered mental status and cardiovascular collapse in severe cases.
- Non-dihydropyridines (e.g., diltiazem, verapamil) have more profound conduction and contractility effects compared to dihydropyridines (e.g., nifedipine, amlodipine), which are more potent vasodilators.
- Abdominal pain, distention, and altered sensorium may also develop.

## **DIFFERENTIAL DIAGNOSIS**

- Beta blocker, digoxin, and/or clonidine overdose
- Narcotic or sedative/hypnotic intoxication
- Inferior myocardial infarction
- Sepsis
- Anaphylaxis

## **OTHER INVESTIGATIONS**

### **Monitoring:**

- ECG:
  - prolonged PR interval due to SA and AV nodal blockade
- Continuous cardiac monitoring
- Serial neurologic examinations

**Additional tests:** serum/urine toxicology screening, glucose (hyperglycemia common), electrolytes, BUN, creatinine, bicarbonate (metabolic acidosis), cardiac biomarkers.

### **Imaging:**

- CXR for respiratory symptoms (pulmonary edema)
- CT brain for altered level of consciousness.
- Abdominal imaging (to rule out bowel obstruction)

## **THERAPEUTIC INTERVENTIONS**

### **Initial stabilization:**

- Admit to ICU bed,
- Airway stabilization
- Fluid resuscitation,
- Give atropine (in symptomatic bradycardia), glucagon , and consider calcium salt bolus.
- High dose insulin and glucose
- Gastric lavage, activated charcoal or whole-bowel irrigation for extended-release formulations or if presentation within 1-2 hours of ingestion.
- Correct electrolyte/acid-base disturbances.
- Consider vasopressors, including dopamine, dobutamine, norepinephrine (1<sup>st</sup> choice), or epinephrine if initial efforts fail.

### **CCB-specific antidotes:**

- Calcium salts (bolus or infusion),
- Insulin and glucose (bolus followed by infusion),
- Glucagon (bolus followed by infusion),
- Lipid emulsions, and/or amrinone.

### **Procedures:**

- Gastric lavage (within 1-2 hours of ingestion),
- Whole bowel irrigation (consider if ingested non-dihydropyridine, even in asymptomatic patients),
- Consider arterial line and central venous access,
- Cardiac pacing and/or balloon pump may be considered in refractory cases.

### **Contact / Cosult:**

- local poison control center and toxicologist

## MANAGEMENT AFTER STABILIZATION

### Follow-Up:

- Observe in ICU for minimum of 12 hours or 24 hours for extended release formulations,
- Monitor glucose, potassium at regular intervals while on insulin infusion.

### Further Treatment:

- Consider additional CCB-specific antidotes if initial therapies fail to improve hemodynamics.

### Manage Complications:

- Mechanical ventilation for respiratory failure and airway protection in the setting of neurologic deterioration.

## CAUTIONS

- Avoid vagal stimulation (*e.g.*, gastric lavage) in pts with bradycardia or heart block.
- Calcium salts contraindicated in the setting of concomitant digoxin toxicity.
- GI decontamination contraindicated in the setting of ileus.
- Caution with excess fluid administration due to propensity to develop pulmonary edema.

## **5) REFERENCES & ACKNOWLEDGMENT**

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