Released July 1, 2014
Updated Nov. 1, 2015
Updated July 1, 2016

Calcium Channel Blocker Poisoning: Quick Treatment Tips

Calcium channel blocker poisoning involves complex physiological changes and significant case-to-case variability; it is best treated by a multidisciplinary team with appropriate knowledge and experience in cardiovascular critical care support and in poisoning treatment.

These tips are provided as general information, but each poisoned patient warrants an individualized treatment plan and careful risk/benefit consideration of all treatment interventions.

Phase 1: General Supportive Care

- GI Decontamination — consider options to prevent further drug absorption
- Positioning — reclined position allows gravity to assist brain perfusion with blood
- Intravascular Volume — administer intravenous 0.9% NaCl as warranted to optimize tissue perfusion and cardiac filling
- Calcium — supplementation may augment transport across dysfunctional calcium channels
  - 10% calcium gluconate may be given peripherally (typically 0.3 mL/kg, 30 mL max – repeat q5 min PRN)
  - 10% calcium chloride (typically 0.1 mL/kg, 10 mL max) may be given centrally
  - Optimum calcium targets are uncertain; we recommend a goal of [iCa] ~ 1.5 times normal
- Serum Electrolytes — K, Mg, Na, CO2, and other metabolic chemistries should be carefully controlled

Advanced Physiological Assessment

Calcium channel blocking drugs may cause diminished vital organ perfusion through cardiogenic shock via a weakened heart pump and/or through *distributive shock* via peripheral vasodilation. Echocardiography and invasive cardiovascular monitoring may assist in patient care in some cases.

Impaired Heart Pump

- Typical of verapamil
- Usually bradycardic
- Diminished cardiac contractility
  "Cold Shock"
  - cool extremities
  - sluggish capillary refill
  - low pulse pressure

Vasodilation

- Typical of amlodipine
- Sometimes tachycardic
- Hyperdynamic cardiac function
  "Warm Shock"
  - warm extremities
  - flash capillary refill
  - wide pulse pressure

Treatment of calcium channel blocker-mediated shock should be targeted toward the underlying physiological changes.

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24-hour hotline
1-800-222-1222

34th Street & Civic Center Boulevard, Philadelphia, PA 19104-4399
Administration: 215-590-2003 • Fax: 215-590-4419
poisoncontroladmin@email.chop.edu • chop.edu/poisoncontrol

Phase 2: Targeted Cardiovascular Support

A) Treatment of Cardiogenic Shock Due to Calcium Channel Blockade[a]

Goal = to improve cardiac output to maintain sufficient perfusion of vital organs.

- *High-dose insulin*[b] therapy can increase cardiac output without increasing myocardial oxygen demand.
  - Not FDA-approved for this indication
  - Appears most effective if initiated early in course of poisoning illness
  - Not a vasoconstrictor, so improvements in blood pressure may be modest
  - Success of therapy may be measured by assessment of perfusion
    - > Mentation
    - > Urine output
    - > Blood lactate
    - > Cardiac contractility assessed by ultrasonagraphy
- Standard **goal-directed** pharmacological inotropes and vasopressors, such as epinephrine, may be used if insulin therapy is insufficient. Some have also suggested a role for glucagon, but its benefit beyond that of standard inotropes and vasopressors is unclear.
- Some advocate a trial of *lipid emulsion*[b] *therapy* (not FDA-approved for indication) when insulin and other therapies seem to be failing.
- Extracorporeal life support (such as "ECMO") may be used to support cardiovascular function when all other therapies have failed, until the calcium channel blocker has been eliminated.

B) Treatment of Distributive Shock Due to Calcium Channel Blockade[a]

Goal = to effect peripheral vasoconstriction to maintain cerebral and coronary perfusion.

- Standard pharmacological inotropes and vasopressors, such as norepinephrine, may be titrated in **goal-directed** fashion to achieve optimal cardiac output and tissue perfusion.
- If standard therapies prove insufficient, *high-dose insulin*[b] therapy might be added to increase cardiac output.
  - Not a vasoconstrictor, so improvements in blood pressure may be modest
  - Success of therapy may be measured by assessment of perfusion
    - > Mentation
    - > Urine output
    - > Blood lactate
    - > Cardiac contractility assessed by ultrasonagraphy
- Some advocate a trial of *lipid emulsion*[b] *therapy* when standard therapies seem to be failing.
- Extracorporeal life support (such as "ECMO") may be used to support cardiovascular function when all other therapies have failed, until the calcium channel blocker has been eliminated.

Selected References for More In-Depth Information:

- Graudins et al. Calcium channel and beta-blocker overdose: antagonist antidotes and adjunct therapies. *Br J Clin Pharmacol*, doi: 10.1111/bcp.12763.
- Jang DH, et al. Toxin-induced cardiovascular failure. *Emerg Med Clin North Am.* 2014;32:79-102.
- St-Onge M, et al. Treatment for calcium channel blocker poisoning: a systematic review. *Clin Toxicol.* 2014;52:926-44.
- Levine M, et al. Critical care management of verapamil and diltiazem overdose with a focus on vasopressors: a 25-year experience at a single center. *Ann Emerg Med.* 2013;62:252-58.
- Engebretsen KM, et al. *High-dose insulin* therapy in beta-blocker and calcium channel-blocker poisoning. *Clin Toxicol.* 2011;49:277-83.

[a]Combined cardiac pump failure and vasoplegia are most typical of calcium channel blocker toxicity and multiple therapeutic pathways may need to be considered concurrently; again, the choice of therapies is best based upon the underlying physiology of shock.

[b]See The Poison Control Center's Tip Sheets for *high-dose insulin* therapy and *lipid emulsion therapy*.

The specialists and consulting toxicologists at The Poison Control Center may be able to provide more nuanced information to assist clinicians trying to make patient care decisions.