

Case Presentation

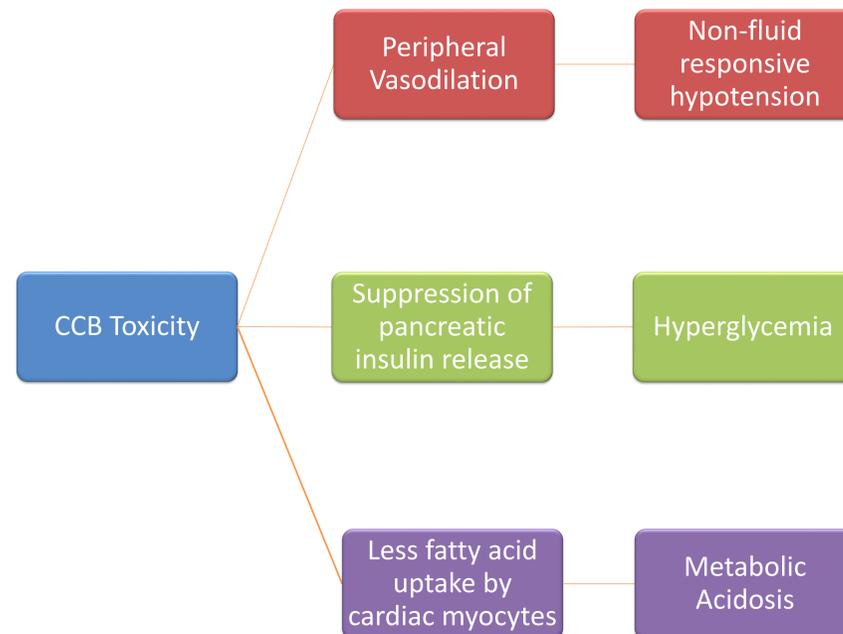
Brief History:

- Previously healthy 12 year old female presents with:
 - 2 minute witnessed, syncopal event
 - 1 week of nausea, crampy abdominal pain
- Day 2 of menses, first menstrual period began 2 months ago
- Denies drug use, suicidal or homicidal ideation, she identifies as bisexual, which family knows
- This morning felt nauseated, passed out, caught by mother. No head trauma or seizure-like activity

Vitals/Physical Exam:

- Afebrile, tachycardic to 109, hypotensive to 84/43
- Neurologically intact. Mild tenderness over left scapula and epigastrium

Figure 1: Pathophysiology and the associated clinical manifestations of calcium channel blocker (CCB) toxicity



Discussion

- CCBs among the most commonly prescribed medications
 - Among the most lethal when overdosed
 - Amlodipine has a half-life of 30-50 hours
- CCB intoxication is a challenging diagnosis
 - The clinician can only rely on signs and symptoms
 - No rapid blood level test available
 - Urine toxicology only screens for certain drugs and medications. Know your institution's test.
- CCBs cause peripheral vasodilatation, suppress pancreatic insulin release, decrease fatty acid uptake by cardiac myocytes, and at toxic levels can lead to profound non-fluid responsive hypotension, hyperglycemia, and acidosis (see Figure 1).

Figure 3: Symptoms of CCB toxicity and their treatments

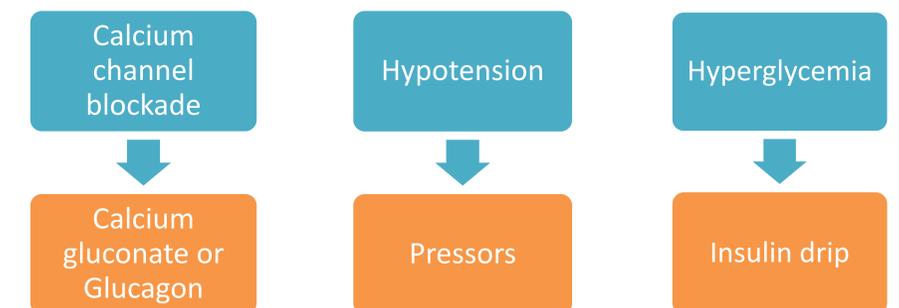
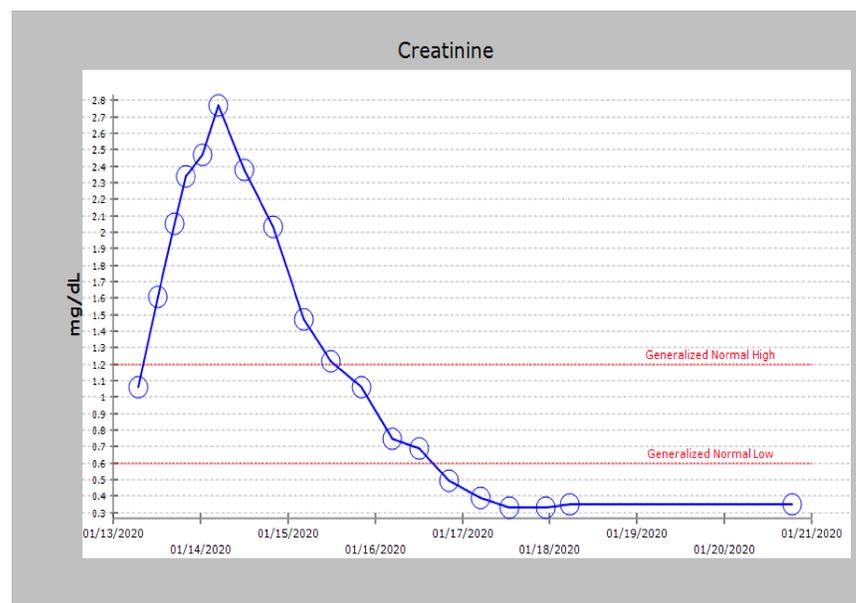


Figure 2: Patient's rising creatinine level, as a reflection of hypotension leading to decreased renal perfusion, eventually normalizes over her hospital course.



Conclusions

- Given the prevalence of CCBs in the community and the lethality of the medication when consumed in excess, either intentionally or accidentally, pediatricians must quickly recognize the classic signs of CCB intoxication
 - Refractory hypotension, hyperglycemia, normal mentation despite the degree of hypotension
- Understanding the pathophysiology (Figure 1) leads to early recognition and proper management (Figure 3).
- Earlier identification of CCB intoxication can:
 - Be life-saving
 - Decrease hospital length of stay
 - Lead to earlier psychiatric intervention

Clinical Course and Outcome

84/43 100 Emergency Department: No orthostasis. Point of care tests: Glucose 221, Lactate 32.4, Hemoglobin 13.9. Three normal saline boluses were given.

73/39 101 Labs at presentation: Leukocytosis with neutrophil predominance, Bicarbonate 19, Creatinine 1.04. Ceftriaxone, Vancomycin started for sepsis concern.

75/42 99 Initial diagnostics: Normal Chest x-ray, EKG, Echocardiogram. Blood cultures negative. Dopamine started given no response to boluses.

77/37 105 Negative pregnancy test, urinalysis, urine toxicology screen. Epinephrine added. Extremities cold and mottled. Norepinephrine added.

75/41 102 Admitted to PICU to start insulin drip for hyperglycemia and fourth pressor, Vasopressin.

105/72 78 Over five days, her blood pressure stabilized (Figure 2) and pressors were weaned. She was transferred to the general ward with no source for her profound shock.

Toxicology was consulted: Calcium Channel Blocker overdose likely. The patient disclosed ingesting 16 of her mother's pills in a suicide attempt. She was transferred to a psychiatric unit.