Still in Shock: A terrifying tale of refractory hypotension
Connor Mitrovich1 DO, Joshua Weiss1 MD, and Namrata Ahuja2 MD
1Department of Pediatrics, 2Division of Hospitalist Medicine, Children’s Hospital Los Angeles

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

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).

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

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

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

Figure 3: Symptoms of CCB toxicity and their treatments