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CALCIUM CHANNEL BLOCKER POISONING: DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN EMERGENCY MEDICINE

Lela Asambadze[1], Ekaterine Kuparashvili[1], Eka Kurdadze[1, 2]

[1]First University Clinic, TSMU, Tbilisi, Georgia

[2]Anesthesiology, Reanimatology and Toxicology Department, TSMU, Tbilisi, Georgia

Background. Calcium channel blocker (CCB) overdose causes refractory vasodilatory shock, hyperglycemia and metabolic acidosis. Unreliable history and resemblance to other emergencies often delay recognition.

Case presentation. A 31-year-old woman was transferred from a regional hospital with suspected acute pancreatitis and hypotension. Initial history suggested accidental ingestion of two amlodipine tablets after alcohol; later, ingestion of a large quantity of amlodipine and an amlodipine-containing combination antihypertensive was disclosed, with suspected intentional overdose. On admission she was alert but pale, with BP 60/36 mmHg and SpO2 89% on room air. Tests showed pH 7.34, base excess –7.2, anion gap 20.4, lactate 3.9 mmol/L, glucose 305 mg/dL, ionized calcium 0.65 mmol/L and creatinine 327.5 μmol/L; lipase (14 U/L), CRP (<5 mg/L) and pancreatic imaging were normal. Ejection fraction was preserved (52–55%).

Management and course. In the ICU, she received crystalloid infusion therapy, norepinephrine, phenylephrine and dobutamine, calcium gluconate, sodium bicarbonate and high-dose insulin euglycemia therapy (1 IU/kg/h) with dextrose and potassium. As shock was vasodilatory rather than cardiogenic, dobutamine was withdrawn and epinephrine added to high-dose norepinephrine. Dialysis was not used (high volume of distribution, protein binding). The course was complicated by capillary leak with generalized edema, pleural effusions up to 700–800 mL (right) and 500–600 mL (left) with respiratory failure requiring non-invasive ventilation and thoracentesis (800 mL), and drug-induced hypoglycemia. Lactate normalized, vasopressors were weaned and creatinine reached 55 μmol/L. Psychiatric evaluation: acute stress reaction, no active suicidal risk. She was discharged on day 9.

Discussion. Recognition was hampered by a minimizing history, a pancreatitis-like presentation (excluded by normal lipase and imaging) and delayed profound vasodilation consistent with amlodipine's prolonged kinetics. Hyperglycemia, reflecting impaired insulin release, and

preserved ejection fraction were key clues that guided early insulin therapy and vasopressor choice. Prolonged vasodilatory shock likely caused functional endothelial glycocalyx disruption and capillary leak, resulting in generalized edema and pleural and peritoneal fluid accumulation that complicated fluid management. Pleural fluid showed no microbial growth, arguing against an infectious effusion. As leukocytosis is common with corticosteroids and in poisoning, CRP (<5 mg/L on admission, 51 mg/L on day 3) and pulmonary infiltrates supported empirical antibacterial therapy for pneumonia.

Conclusion. CCB poisoning should be suspected in unexplained vasodilatory shock with hyperglycemia and preserved ejection fraction, even when the history suggests trivial ingestion. Early high-dose insulin euglycemia therapy, mechanism-based vasopressor selection and vigilance for capillary leak and pleural effusions are essential for a good outcome.

Keywords: calcium channel blockers; amlodipine; vasodilatory shock; high-dose insulin therapy; poisoning.