PP – 07

Diltiazem induced vasoplegic shock managed with high dose pressors and methylene blue

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Objective: Diltiazem overdose is associated with cardiac conduction abnormalities, negative inotropic effects and peripheral vasodilatation. We present a case of diltiazem ingestion with a clinical picture dominated by predominantly vasoplegic shock.

Case Report: A 57 year old male presented to the Emergency department by ambulance complaining of postural dizziness, and feeling unwell. On presentation he was hypotensive with blood pressure 90/45 mmHg, heart rate 85 beats per minute, sluggish capillary refill, respiratory rate 24 beats per minute and a venous blood gas showed a lactic acidosis with a lactate level of 5.4 mmol/L. Intravenous crystalloids were administered however he continued to be hypotensive. A CT abdomen was ordered as he complained of abdominal and back pain, following which a surgical consult was requested as mesenteric ischaemic was thought to be a possible cause of his condition. During this the patient revealed that he had ingested 6 x 180 mg diltiazem earlier in the day. He was commenced on peripheral inotrope infusions and transferred to the intensive care unit. Bedside ECHO in ICU showed a hyperkinetic left ventricle and a pulmonary artery catheter was inserted which revealed a cardiac output of 6.4 L/minutes (RR 4 – 8), a cardiac index of 3.4 L/min/m^2 (RR 2.5 – 4) and a systemic vascular resistance of 415 dynes/sec/cm^5 (RR 770 – 1500). Concentrated noradrenaline was commenced and was quickly titrated to maximum doses whereupon vasopressin was added in. Calcium Chloride infusion was also begun titrated to an ionized calcium of 2.0 mmol/L. Following consultation with the regional toxicology service a 2 mg/kg bolus of methylene blue was administered followed by an infusion of 0.5 mg/kg/hour. This led to a modest improvement in systemic vascular resistance over the next 24 hours during which the patient's noradrenaline requirements decreased however he continued to deteriorate clinically. He was commenced on non invasive ventilation and subsequently intubated 48 hours post admission for progressive hypoxia. He was commenced on continuous renal replacement therapy for a rising creatinine which had climbed to 410 micromol/L (RR 60-100) having been within normal limits on admission. He required CRRT for another 8 days when he was also extubated. He was discharged to a mental health facility 24 days after initial presentation.

Conclusion: This case was unusual in that vasoplegic shock was the predominant clinical manifestation of toxicity. Methylene blue was added when standard measures appeared inadequate and was associated with modest though not dramatic change in clinical condition.