Low and Slow: A Pattern Recognition for Overdose with Beta and Calcium Channel Blockers

Sir,

A 70-year lady with a history of hypertension and coronary artery disease presented in the emergency department (ED) with the complaint of slow heart rate. She also reported a syncopal event one hour earlier and continued to feel slow. Her cardiologist increased the dose of metoprolol from 100 mg to 200 mg 5 days back. On arrival, she was slowly responding to the questions and her vital signs: heart rate of 30 beats/minute, blood pressure of 70/40 mmHg, respiratory rate of 18 breaths/min, oxygen saturation of 96% on room air and temperature of 36.5°C. She was resuscitated with intravenous fluids, atropine and temporary pacing. Hypoglycemia was corrected by dextrose administration and hyperkalemia was corrected by giving calcium gluconate and dextrose-insulin. Her heart rate raised to 60 beats per minutes and blood pressure to 130/80 mmHg. She was kept under observation and was discharged uneventfully after she remained stable for 24 hours.

On encountering low and slow hypotension and bradycardia patients in the ED, the emergency physicians should think of a pattern of a drug overdose. Both intentional and unintentional overdoses of these drugs are implicated; hence, awareness and early recognition and management are important. [1]

The key features to differentiate between CCB and BB overdoses are blood glucose and level of consciousness. Overdose with CCBs has intact mentation while overdose with BBs leads to altered mentation. CCB poisoned patients are more likely hyperglycemic while BB poisoned patients are euglycemic or hypoglycemic.[2,3]

Patients with a suspected CCB or BB overdose present with seizures in the ED. Benzodiazepines are the first line drug here with advancement to Phenobarbital or Propofol. If electrocardiogram shows a wide QRS complex, bicarbonate administration should be considered in the overdose seizing patients.[1-3]

The general approach includes fluids, decontamination, atropine with transcutaneous pacing, calcium, high-dose insulin, vasopressors, glucagon, lipid emulsion therapy and extra-corporeal membrane oxygenation.[1,2] Normal saline is usually used in bolus form for fluid administration. Activated charcoal is used for decontamination with a ratio of 10:1 (charcoal to drug). Atropine can be given with 0.5 mg IV dosage if hemodynamically significant bradycardia is present.[1-2] Transcutaneous pacing is not a successful intervention in patients with bradycardia; however, it can be attempted. In low and slow patients, it may induce dysrhythmias. Calcium gluconate 3 ampules or calcium chloride 1 ampule followed by infusion should be given. For BB and CCB poisoned patients, high dose insulin with a bolus of 1 unit/kg intravenous push with 2 ampules of D50W is administered followed by 1 unit/kg/hour insulin infusion to titrate.[2,3] It is recommended to give liberal dosing of insulin and the dosage can go upto 10 units/kg/hour.[3]

Serum glucose should be recorded every 15 minutes, as serum potassium. Early insulin treatment is recommended as insulin takes 45 minutes to start working. Inotropic support may be needed until insulin's action takes effect. Epinephrine is preferred in myocardial depression and norepinephrine is favoured in normal cardiac contractility assessed on point-of--care ultrasound. Glucagon is used as a last resort as it worsens hypotension and bradycardia. Lipid emulsion therapy is used in patients with an overdose of lipid-soluble drug such as CCBs with refractory shock.[2,3]

Early and prompt recognition of BB and CCB toxicity is important to guide therapy.

Timely involvement of the poison control centres is important. Gastrointestinal decontamination may be useful for patients presenting early or with extended-release ingestions. One can consider the 'kitchen sink' approach with a vriety of salvage therapies in the patient unresponsive to initial resuscitative efforts.[3,4]

It is important for an emergency physician to be aware of this complex management of BB and CCB overdose. There is a wide range of clinical presentations for these toxicities; however, low and slow patients make it easier to recognise the causes to initiate timely management.

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The author declared no competing interest.

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