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Case Review

A 42-Year-Old Woman with a Beta Blocker Overdose

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An emergency phone call was made by a family member to 911. Ground emergency medical services were dispatched to an unresponsive woman, and upon their arrival they found a 42-year-old woman lying in bed with agonal respirations. Next to her were empty bottles of lorazepam and propranolol. At the time, it was unclear how many tablets of each she had ingested. The patient was unarousable with painful stimuli. She had a Glasgow Coma Scale score of 3 (eye: 1, verbal: 1, and motor: 1). The paramedics attempted to intubate the patient without success. Subsequently, they placed an oropharyngeal airway and supplemented her respirations with bag-mask ventilations at 15 L oxygen. They established intravenous (IV) access in the right antecubital fossa, and the patient was placed on a cardiac monitor. While traveling to the emergency department, the patient became pulseless and apneic. Cardiopulmonary resuscitation (CPR) and advanced cardiac life support (ACLS) were initiated. The patient received an epinephrine 1-mg IV push. Return of spontaneous circulation was achieved soon after. Upon arrival to the rural emergency department, the patient was intubated with a 7.0-mm endotracheal tube on the first attempt. After the patient was somewhat stabilized, the health care team activated a local air medical program to transfer the patient to a regional medical center that offered cardiology and critical care services.

Upon arrival of the flight team, the patient was being ventilated by the respiratory therapist with a bag-valve mask. The patient had been placed on the cardiac monitor with pulse oximetry, and IV access had been attained with 2 IV lines. She had received a total of a .9% normal saline bolus of 1500 mL. The emergency physician had administered calcium chloride 10 mL intravenously, and a dopamine drip was initiated. This was quickly titrated up to 20 μg/kg/min. Her vital signs were heart rate (HR) of 50, blood pressure of 78/46 mm Hg, respiratory rate of 16 with artificial ventilation, and oxygen saturation of 98%. The critical care flight team began to transfer the patient to their equipment. In addition to the pulse oximetry, they connected the end-tidal carbon dioxide ($ETCO_2$) detector with a reading of 35 mm H_2O. The patient did not require any sedation, and they documented the Glasgow Coma Scale score as 3T (intubated). The patient had skin that was cool with delayed capillary refill at 4 seconds. After a brief discussion with the sending physician, they agreed to initiate an additional norepinephrine infusion at 30 μg/min. Lastly, the team also administered glucagon 5 mg intravenously in an effort to reverse the potential effects of the beta blocker. The transport ventilator was set to assist control mode with volume control. The rate was 16, the fraction of inspired oxygen was 100%, the tidal volume was 300 mL, and the positive end-expiratory pressure was 5 cm H_2O. The patient was packaged and loaded into the aircraft for transport.

During the course of the flight, the patient became hemodynamically unstable with a drop in her blood pressure to 66/48 mm Hg. IV fluids were already being administered rapidly via a pressure bag. The flight team discussed the choice of vasopressors. They elected to discontinue the dopamine drip and start an epinephrine drip with a rate of 5 μg/min. Initially, this seemed to be effective. However, approximately 10 minutes later, a blood pressure could not be obtained. The patient lost peripheral pulses, and CPR was initiated by the critical care paramedic. The critical care nurse administered epinephrine 1 mg intravenously, and return of spontaneous circulation was achieved. The rate of the epinephrine drip was increased to 10 μg/min. Her HR was 51, blood pressure was 62/46, pulse oximetry was 93%, and $ETCO_2$ was 42 mm H_2O. The critical care team placed pacer pads on the patient. Because of the dropping oxygen saturations, the positive end-expiratory pressure was increased to 8 cm H_2O. $ETCO_2$ began to drop to 23 mm Hg. The critical care team assessed peripheral perfusion and found her to have a weak pulse. They initiated external pacing using a rate of 60 beats/min and obtained electrical and mechanical capture at 70 mA. They adjusted the HR to 70 beats/min, and her vital signs improved with an HR of 70, a blood pressure of 88/60 mm Hg, and an $ETCO_2$ of 30 mm H_2O.

Upon arrival to the receiving facility, the flight crew was met by the emergency department team. The receiving physician contacted poison control, and, based on their recommendations, aggressive therapy was initiated. The patient was placed onto the hospital equipment. Multiple physicians were at the bedside including cardiology and critical care. A femoral central line was quickly established, and a radial arterial line was also placed for accurate blood pressure monitoring. An electrocardiogram was obtained and showed wide complex bradycardia (Fig. 1).

The patient then lost peripheral pulses again, and ACLS was again initiated with the patient receiving CPR with protocolized dosing of epinephrine as indicated. Poison control recommended the initiation of

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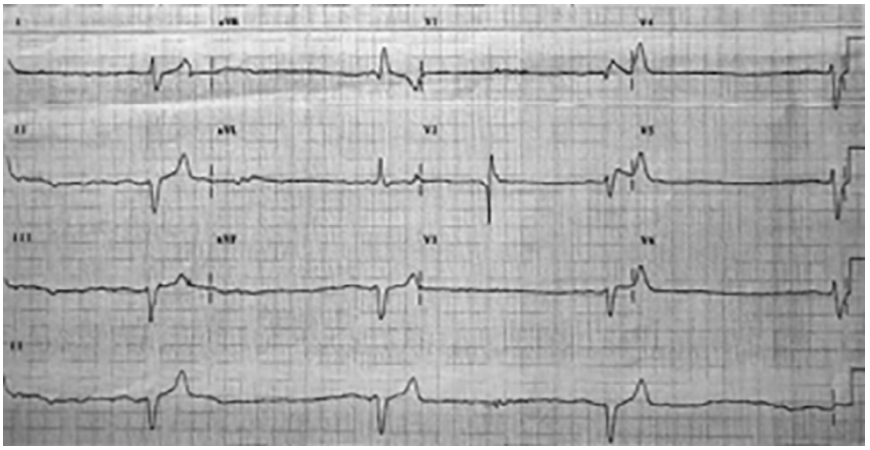

Figure 1. Electrocardiographic wide complex bradycardia identified in the receiving emergency department.

sodium bicarbonate, glucagon, and high-dose insulin infusions for therapy. Ten milligrams of glucagon was intravenously administered followed by a glucagon infusion at 5 mg/h. Additionally, a variety of infusions were initiated concurrently including a sodium bicarbonate infusion at 100 mg/h; a norepinephrine infusion of 30 μg/min was continued as well as an epinephrine infusion at 10 μg/min. Lastly, high-dose insulin therapy was also initiated. The patient received insulin, 70 units per hour intravenously. Dextrose 10% in .9% normal saline at 100 mL/h was initiated to ensure that the serum glucose did not drop precipitously with the high-dose insulin therapy. The patient had a systolic blood pressure of 100 mm Hg after the initiation of these therapies. The cardiologist was concerned because of the refractory nature of her presentation, and he recommended concurrent transvenous pacing. He placed an internal jugular cordis catheter and successfully inserted the transvenous pacemaker under fluoroscopy. Transvenous pacing was initiated successfully (Fig. 2).

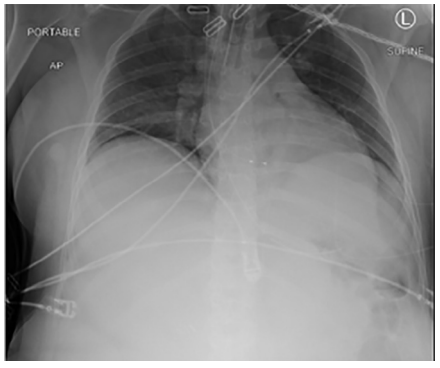

Figure 2. A chest radiograph with a temporary pacemaker wire and an endotracheal tube in the appropriate positions.

Because the patient was about to be transferred to the critical care unit, she lost peripheral pulses, and CPR was initiated again. The critical care attending was at the bedside and recommended lipid emulsion therapy as a last attempt at medical therapy. The clinical pharmacist assisted with the administration of this bolus and infusion. At this point, the cardiologist noted that the transvenous pacemaker was no longer able to capture electrically. ACLS was continued for 20 minutes. Multiple conversations were had with the patient's family about her poor prognosis. They agreed to withhold life-sustaining efforts, and the patient died 2 and half hours after her first contact with emergency medical services.

Pathophysiology

Understanding the pathophysiology of a beta blocker overdose first requires a discussion about the role of beta-adrenergic receptors in the healthy individual. Catecholamines, such as epinephrine and norepinephrine, stimulate beta-adrenergic receptors and are the primary signals in the "fight or flight" response. There are 3 separate types of beta receptors. The first are those found on the myocardium. These beta 1 receptors increase the chronicity (rate) and inotropy (strength of contraction) of the cardiac tissue when stimulated. The second, beta 2 receptors, are found on smooth muscle. Agonists of the beta 2 receptors cause smooth muscle relaxation, vasodilation, and bronchodilation.[1,2] The third, beta 3 receptors, play a role in lipolysis of adipose tissue.[2] The pervasive nature of beta receptors allows beta blocking medications to be an effective treatment for a variety of health ailments including congestive heart failure, coronary artery disease, supraventricular tachycardia, migraines, anxiety, and hyperthyroidism.[1]

The medical therapy of using beta blockade is useful in patients with heart disease because it decreases the contractile force of the heart and heart rate through its control of calcium flow. Calcium plays a pivotal role in both the contraction of myocytes (muscle cells) and the propagation of the electrical signal through the myocardium via beta 1 receptors. Calcium floods into myocytes as a result of beta 1 receptor stimulation by catecholamines. This, in turn, increases the intracellular calcium levels, leading to the release of additional calcium from the sarcoplasmic reticulum into the same intracellular space. The rapid increase in calcium levels inside the myocytes leads to contraction. The conduction of electricity of the heart begins with depolarization of the primary pacemaker site, the sinoatrial node. The electricity then travels through subsequent electrical pathways of the heart including the atrioventricular (AV) node, the bundle of His, and lastly the ventricles. The pacemaker capacity and conduction through this pathway are similarly dependent on calcium flux and are controlled by beta 1 receptor stimulation.[1,3]

An overdose of beta blocker medications generally presents as an exaggerated picture of the desired therapeutic effects of decreased blood pressure and heart rate. The hallmarks of cardiotoxicity in beta blocker overdose are bradydysrhythmias, hypotension, and cardiogenic shock.[1] Initially, patients may have an asymptomatic presentation. However, decompensation may occur quickly, leading to rapid cardiovascular collapse and death.[3] The onset of clinical manifestations of beta blocker overdose usually begins within 2 to 6 hours.[4] There is less evidence regarding extended-release beta blocker formulations, which may lead to a later onset of signs and symptoms.[3] The cardiovascular toxicity from beta blockers is the result of 3 primary

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