Lipid rescue for bupivacaine toxicity during cardiovascular procedures

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Introduction

Bupivacaine toxicity is a recognized complication of procedures done under local anesthetic infiltration. While local anesthetic toxicity is rare, it is potentially catastrophic and life-threatening.[1] A 20% lipid emulsion has been used to resuscitate patients after bupivacaine overdose or inadvertent intravascular injection.[2-7] While the use of lipid emulsion for local anesthetic toxicity has been reported extensively in the anesthesia literature,[8] it has not yet been reported in the cardiology literature. We report a case of local anesthetic toxicity resulting in pulseless electrical activity during an electrophysiology procedure that was successfully treated by infusion of 20% lipid emulsion.

Case Report

A 28-year old male from El Salvador (1.58 m, 55.8 kg) with no significant past medical history presented to a community hospital with a 3-month history of worsening shortness of breath, dyspnea on exertion, fatigue, nausea and vomiting. The patient denied chest pain, fevers, chills, recent febrile illness, sick contacts or palpitations.

The patient was found to be in sinus rhythm with complete heart block (3rd degree AV block) and a junctional escape rhythm of 30 bpm. He was then transferred to our institution where he underwent emergent placement of a right internal jugular transvenous pacing wire for temporary pacing. Further evaluation showed the patient to have a nonischemic cardiomyopathy with an echocardiogram demonstrating a left ventricular ejection fraction of 12% with severe global hypokinesis.

The patient was scheduled for a biventricular implantable cardioverter defibrillator (ICD) implant. In the electrophysiology suite, the patient had standard monitors placed and oxygen was delivered at 3 L/min via nasal cannula. Intravenous cefazolin 1 g was given as prophylaxis, with fentanyl 12.5 μg and midazolam 2 mg administered intravenously. During this period the patient was comfortable, awake and able to communicate.

During the procedure, the patient received a total of 50 cc of local anesthetic injection comprised of a mixture of 2% lidocaine and 0.5% bupivacaine to the left subpectoral region to facilitate left axillary venous access and construction of a subpectoral pocket for the device. The heart rate and blood pressure remained stable during injection of local anesthetic. Soon after obtaining axillary venous access, the patient complained of dizziness but with no changes in blood pressure, heart rate or pulse oximetry. Several minutes later he exhibited generalized seizure activity with severe tonic-clonic activity. Initially, midazolam 1 mg IV and then lorazepam 2 mg IV was used to treat the seizure while a bag valve mask was used to support ventilation. The patient remained hemodynamically stable during this episode. Urgent anesthesiology consultation was requested and the patient was intubated without complication. Subsequently, the patient was noted to have pulseless electrical activity and cardiopulmonary resuscitation was immediately started. Advanced cardiac life support protocol was initiated during which the patient received a total of 4 mg epinephrine, 40 U of vasopressin, 4 mg atropine, normal saline bolus and 200 meq sodium bicarbonate. An echocardiogram demonstrated no pericardial effusion. Bupivacaine toxicity was suspected as the cause for the cardiac arrest and the patient was given 2 units of 20% lipid emulsion. During the second dose of 20% lipid emulsion infusion, the patient regained his pulse and became hemodynamically stable. The pocket was closed after rinsing with antibiotics. A CT scan of the brain and a neurology consultation were unrevealing. Several hours after intubation and cardiac arrest the patient was successfully extubated.

Approximately one week later, the patient had successful placement of a biventricular ICD without complication. No bupivacaine was used. Several days later the patient was discharged home.

Discussion

The anesthesiology community has had extensive experience with local anesthetics as well as their complications.[1,8,9] As a result, the use of lipid emulsion to rescue patients with local anesthetic toxicity is well known to anesthesiologists but perhaps less to cardiologists.[2,3,5] The proposed mechanism of intralipids action is the "lipid sink" binding. In this proposed mechanism, the lipids "bind" the lipophilic bupivacaine and reduce tissue content. The first reported use of lipid therapy in patients was in 2006, with the successful rescue of patients undergoing regional blocks who failed to respond to conventional cardiopulmonary resuscitation after showing signs of local anesthetic toxicity. The patients were successfully resuscitated after treatment with lipid emulsion.[10,11] Weinberg *et al.* demonstrated that intravenous lipid emulsion therapy increases resistance to, and enhances resuscitation of, rats and dogs exposed to local anesthetic overdoses.[12-14] Importantly, while lipids have been shown to be helpful in treat-

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ing lipophilic local anesthetics such as bupivacaine, levobupivicaine, ropivicaine, and mepivicaine, they do not treat toxicity from the more hydrophilic local anesthetics such as lidocaine.

While no serum levels were drawn during the resuscitation in this case, we are confident the events were secondary to local anesthetic toxicity due to the timing, the clinical presentation consistent with local anesthetic toxicity (dizziness, hypotension, seizure activity, and arrhythmia)[8] and the immediate resuscitation following administration of 20% lipid emulsion. It is well documented that bupivacaine toxicity first manifests as central nervous system disorders (tinnitus, a metallic taste in the mouth, dizziness, seizures). Cardiovascular signs follow the neurologic signs and include bradycardia, dysrhythmias and, in severe cases, asystole.[8]

This patient received 50 cc of a combination of 2% lidocaine and 0.5% bupivacaine (in a 4:3 ratio) yielding a total dose of 570 mg of lidocaine and 107 mg of bupivicaine. In this size patient, that yields a dose of 10 mg/kg of lidocaine plus 1.9 mg/kg of bupavicaine. The maximum local anesthetic dose for lidocaine is 4.5 mg/kg and the maximum dose for bupivacaine is 2.5 mg/kg. Therefore, this patient received a toxic dose of lidocaine plus a dose of bupivacaine approaching the upper limit.

In summary, this case represents the first reported case of local anesthetic cardiotoxicity with successful reversal using lipid emulsion in the electrophysiology laboratory, a site that frequently utilizes local anesthetics. Bupivacaine toxicity and the use of lipid emulsion as rescue therapy should be considered in all cases in which symptoms consistent with local anesthetic toxicity occur. Furthermore, all clinical sites where local anesthetics are routinely used should have 20% lipid emulsion readily available and personnel should be educated regarding this medication. Published evidence to date suggests that lipid rescue for presumed local anesthetic toxicity should be considered in prolonged cardiopulmonary resuscitation when there is suspicion of local anesthetic toxicity.

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