The Journal of Emergency Medicine, Vol. 50, No. 1, pp. 47–50, 2016

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2015.07.035

Selected Topics: Toxicology

LIDOCAINE-INDUCED CARDIAC ARREST IN THE EMERGENCY DEPARTMENT: EFFECTIVENESS OF LIPID THERAPY

Kevin James Tierney, MD, Tiffany Murano, MD, and Brenda Natal, MD

Department of Emergency Medicine, New Jersey Medical School, Newark, New Jersey
Corresponding Address: Kevin James Tierney, MD, Department of Emergency Medicine, New Jersey Medical School, 30 Bergen St, ADMC 1110, Newark, NJ 07103

☐ **Abstract—Background: Local anesthetics are commonly used in the emergency department (ED). Overdoses can lead to disastrous complications including cardiac toxicity and arrest. Recognition of local anesthetic systemic toxicity (LAST) is important; however, prevention is even more critical. Knowledge of proper lidocaine dosage can prevent LAST. LAST may be effectively treated with lipid emulsion therapy. Although the mechanism is not well understood, its use may have a profound impact on morbidity and mortality. Case Report: Fifty milliliters of 2% lidocaine was infiltrated for local anesthesia in a 35-year-old woman during the incision and drainage of a labial abscess. Following the procedure, the patient complained of vomiting, with rapid progression to an altered mental state and seizure requiring endotracheal intubation for airway protection. Suspecting lidocaine toxicity, intralipids were ordered. While waiting for the intralipids, the patient decompensated and suffered pulseless electrical activity (PEA) cardiac arrest. A 100-mL bolus of 20% intralipids was administered 3 minutes into the resuscitation, after which return of spontaneous circulation occurred. The intralipid bolus was then followed by a continuous infusion of 0.25 mL/kg/minute, for an infusion dose of 930 mL. Despite a complicated hospital course, the patient was discharged home neurologically intact. Why Should an Emergency Physician Be Aware of This?: We believe this patient's cardiovascular collapse was secondary to an iatrogenic overdose of lidocaine. This is one of the first cases to support the efficacy of intravenous lipids in the treatment of LAST in humans in the ED.** © 2016 Elsevier Inc.

☐ **Keywords—LAST; local anesthetic toxicity; lipid rescue therapy; toxicology**

INTRODUCTION

Lipid emulsion therapy has been used in the treatment of cardiovascular collapse caused by toxic doses of local anesthetics. Although the evidence for the benefit of lipid therapy is mostly from animal studies, several anesthesiology case reports discuss evidence supporting the use of lipids as a potentially life-saving antidote for local anesthetic systemic toxicity (LAST) in humans (1–3). We add to that evidence by reporting the successful emergency department (ED) use of a 20% lipid infusion to resuscitate a 35-year-old patient from cardiovascular collapse after the unintentional administration of a toxic dose of lidocaine after incision and drainage of a labial abscess.

CASE REPORT

A 35-year-old woman presented to the ED with a 4-day history of increasing pain and swelling over the left labia. She denied any associated fevers, chills, vaginal discharge, bleeding, or drainage from the labia. Her medical history was significant for coronary artery disease, diabetes, and stage 3 chronic kidney disease. Her medications included amlodipine, metolazone, torsemide, atorvastatin, nitroglycerin, carvedilol, aspirin, and neutral

RECEIVED: 28 February 2015; FINAL SUBMISSION RECEIVED: 26 June 2015;
ACCEPTED: 25 July 2015

protamine hagedorn insulin. She had no history of surgery. She reported an allergy to penicillin, described as a rash. She denied any alcohol, tobacco, or illicit drug use. On arrival to the ED, the patient was in no acute distress. Her blood pressure was 116/82 mm Hg, her heart rate was 90 beats/minute, her respiratory rate was 20 breaths/minute, her temperature was 36.8°C, and she weighed 62 kg. Her glucose was 222 mg/dL, and her urine pregnancy test was negative. Her physical examination was remarkable for a tender and fluctuant left labia majora, measuring 4 cm × 4 cm with no involvement of the Bartholin gland. The patient was diagnosed with a left labial abscess. She received 2 tablets of oxycodone/acetaminophen 5 mg/325 mg for pain control, and the gynecology (GYN) service was consulted for further evaluation and management.

The patient was seen by the GYN consult service in the ED approximately 4 hours later, at which time she reported recurrent pain and was given 8 mg of morphine intramuscularly before incision and drainage of the abscess. An incision and drainage of the labial abscess was performed with local anesthesia using lidocaine 2% without epinephrine; a large amount of purulent material was drained and the abscess was packed. About 15 minutes postprocedure, the patient complained of not feeling well, and began to vomit nonbloody, nonbilious fluid. She received a 4-mg dose of ondansetron in tablet form, but 5 minutes later the nurse reported that the patient was "not acting right" and was vomiting again. On reassessment, the patient was no longer responsive to voice, was flailing her arms and legs, and had lip smacking, consistent with seizure activity. She also continued to vomit nonbilious, nonbloody fluid. The decision was made to emergently intubate the patient for airway protection. The patient underwent rapid sequence induction for intubation using intravenous rocuronium 50 mg and etomidate 20 mg and was successfully intubated. Successful intubation was immediately confirmed by direct visualization of passage of the endotracheal tube through the vocal cords, auscultation of absent sounds over the stomach, the presence of bilateral breath sounds, and positive colorimetric capnography. It was unclear why the patient had decompensated; the electronic medical record (EMR) and medications administered in the ED were reviewed.

In the EMR, the administration and dosages of the oxycodone/acetaminophen, morphine, and ondansetron were verified with the nurse; however, the amount of lidocaine administered was unclear. The EMR showed that a 60-mL vial of lidocaine 2% without epinephrine was given to the GYN consultant. After discussion with the GYN consultant, it was reported to the ED team that 50 mL of lidocaine 2% (1000 mg) was administered using the ring block technique with reported aspiration between each injection without return of blood. This dosage was >3 times the maximum recommended dose. Knowing that lipid infusion may have a beneficial role in cases of local anesthetic toxicity, the lipid emulsion was ordered and the Poison Control Center (PCC) was consulted. The PCC agreed that this was a potential lethal dose of lidocaine and recommended the emergent administration of intralipids, starting with a 100-mL intravenous bolus of 20% intralipids, followed by an intravenous infusion of 0.25 mL/kg/minute of 20% intralipid until cardiac stability was restored.

Before arrival of the intralipids, the patient went into a pulseless electrical activity (PEA) arrest. Cardiopulmonary resuscitation (CPR) was initiated, and the PEA advanced cardiac life support (ACLS) algorithm followed. High-quality CPR was instituted, and the patient received epinephrine 1 mg. Three minutes into the resuscitation, the 20% intralipids arrived and the 100-mL bolus was given. About 1 minute after infusion of the intralipid bolus, there was restoration of a perfusing rhythm of sinus tachycardia with a blood pressure of 100/60 mm Hg. The patient was placed on the intralipid infusion and was admitted to the medical intensive care unit (ICU). She received a total infusion dose of 930 mL of intralipids.

The patient had a complicated hospital course, including pneumonia, hyperkalemia requiring 1 session of hemodialysis, and a thrombus in the atrial appendage requiring anticoagulation. She was discharged home on hospital day 7, neurologically intact. At follow-up 1 week later, the patient was doing well and complained only of mild chest pain.

DISCUSSION

Lethal local anesthetic overdoses are uncommon in the literature. In 2013, there were 1454 exposures to lidocaine, along with 4092 single exposures to other unknown local or topical anesthetics reported to U.S. Poison Control Centers (4). Of the 1218 fatalities reported to Poison Control Centers in 2013, only 5 were caused by local anesthetics (4). The most common reason for local anesthetic overdoses are inadvertent injection of a therapeutic dose into a blood vessel, repeated uses of therapeutic doses, and unintentional administration of a toxic dose (5).

The diagnosis of LAST is clinical, because serum levels are not immediately available at most institutions and would not help guide treatment decisions. However, a serum level could be sent, if LAST is suspected, so that it may later be confirmed with laboratory evidence. Unfortunately, we did not send a serum lidocaine level. One of the earliest clinical manifestations of LAST is neurologic symptoms, and most specifically seizure, as seen in our patient. This often precedes cardiovascular