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Case Report

Seizure following in-office lidocaine administration: a case report on local anesthetic systemic toxicity

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***Abstract*:** Local anesthetics have a broad application for minor and major surgeries, in outpatient and inpatient settings. Drug dosing, frequency, duration of action, and coadministration with other drugs, are some of many factors that must be considered for each patient, before drug administration. Like other medical treatments, the use of local anesthetics has potential complications, such as local anesthetic systemic toxicity (LAST). LAST primarily affects the cardiac and central nervous systems (CNS), seizures and cardiac arrest being some of the more time-sensitive symptoms requiring immediate treatment. Patients should be briefed on potential symptoms if LAST occurs and physicians should be aware of the warning signs, treatment, and prevention. In our case study, a 40-year-old, 51 kg woman was administered a lidocaine dosage of 760 mg in an outpatient setting. She presented to the emergency department with diffuse tremors, paresthesias of the mouth and face, spasticity, irritability, and a single generalized tonic–clonic seizure. The patient was successfully treated with Ativan along with lipid emulsion. We review this case and perform a literature review to identify key points in the use of local anesthetics. Healthcare providers should be trained in LAST treatment and prevention. Our case study therefore serves to reduce the frequency of LAST and other adverse outcomes associated with local anesthetic administration.

Plain language summary

Case of local anesthetic overdose that lead to a seizure

Local anesthetics are injectable medications that are widely used to prevent the sensation of pain during invasive or surgical procedures. These drugs block pain signals from travelling up nerve fibers to the brain. Because of their increasing use, it is important to keep in mind signs and symptoms of local anesthetic overdose. These symptoms can occur as soon as less than five minutes after administration. Symptoms primarily affect the central nervous and cardiovascular systems and include numbness, drowsiness, confusion, heart palpitations, slurred speech, ear-ringing or possibly cardiac arrest or seizure. These signs and symptoms should be monitored for and explained to the patient prior to patient discharge. In our paper, we report a case of an accidental local anesthetic overdose in plastic surgery clinic during a surgical procedure. The patient was inadvertently administered 2.1 times the recommended maximum dose. She complained of facial numbness and a metallic taste in her mouth that was not immediately reported to the clinic. About 90 minutes after the administration of lidocaine, a commonly used local anesthetic, the patient had a seizure while being evaluated in the emergency department. Appropriate treatments were administered, including benzodiazepines for the treatment of seizure, the administration of lipids to remove the anesthetic from the heart and brain, and the administration of oxygen. This case

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serves as an education tool for both patients and providers. Patients will be able to more clearly identify symptoms of local anesthetic, while providers will be able to more easily calculate the maximum allowable dose of a local anesthetic. With its increasing use in the outpatient setting, we hope that our case report will help reduce the incidence of local anesthetic overdose in the future.

Keywords: last, local anesthesia, seizure, toxicity

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Introduction

Local anesthetics are widely used to provide analgesia during invasive or surgical procedures. These drugs inhibit nerve conduction and propagation of pain signals up nerve fibers to the central nervous system (CNS). Local anesthetic administration in the perioperative setting decreases postoperative pain and recovery time and reduces the length of hospital stay.[1] They also have use at the bedside and in the outpatient setting for small procedures. Because of their increasing use, it is important to keep in mind the signs and symptoms of local anesthetic systemic toxicity (LAST).[2] In this case study, we discuss an inadvertent lidocaine overdose that led to a generalized tonic–clonic seizure. We review the most common anesthetic agents and their dosing, signs of local anesthetic toxicity, and general safety tips for administration. The reporting of this study conforms to the CARE statement and checklist.[3]

Case presentation

A 40-year-old, 51 kg woman with a past medical history of hypothyroidism presented to our outpatient office with the complaint of excess tissue from her incisions, bilaterally, after a recent panniculectomy. The decision was made to proceed with a bilateral excision under local anesthesia in the office. Of note, other medications included the following: levothyroxine, oxycodone–acetaminophen, and nystatin. Her allergies included Keflex and nickel and was noted to have a tachycardic response to procaine during her initial panniculectomy in March 2023.

In the office, the patient's vitals were within normal range pre- and post-procedure. Immediately prior to her procedure, her blood pressure was 136/83 mmHg, heart rate 64 beats per minute, 18 respirations per minute, with an O_2 saturation of 100%. Her weight at that time was 51 kg. The patient had no prior tobacco or illicit drug use. At the start of the procedure at 09:30, 10cc of 2% lidocaine with epinephrine was injected into each excess skin flap. After 20–30 min, the patient was tested for anesthesia by clamping the excess tissue present bilaterally, with forceps with teeth, to which she reported pain. Consequently, each excess skin flap was injected with another 9cc, and anesthesia was achieved. Hence, a total of 38cc was injected into the patient. Each excess skin flap was then successfully excised and closed with 3-0 Vicryl and 4-0 Monocryl sutures, and covered with dermabond. Immediately post-procedure, it was determined that the patient inadvertently received an excess dose of lidocaine. With this noted, the patient was questioned for symptoms of lidocaine toxicity including numbness, tingling, palpitations, dizziness, metallic taste, and tinnitus, in which she denied all symptoms. She was advised to remain in the clinic for monitoring. At 11:00 the same day, about 1.5 h post-procedure, the patient continued to deny all symptoms and was medically cleared to leave the clinic. Proper education on warning signs and symptoms was discussed prior to her leaving. Interestingly, upon questioning the patient after the resolution of this case, she noted that she did feel paresthesias of the mouth and face immediately post-procedure but she did not report this to the office staff, as she misinterpreted her symptoms and needed to go home to care for her children. The patient returned to the clinic at about 12:00 with worsening tremors and continued paresthesia of the face and mouth. She was immediately transferred to the emergency department.

In the emergency department, the patient presented with diffuse tremors, intermittent spasticity, and irritability, secondary to lidocaine toxicity. Vitals were taken at 12:31 showing tachycardia from 120 to 140 beats per minute, blood pressure 144/88, and O_2 saturation 96% on room air. The workup also included a comprehensive metabolic panel, complete blood count, troponin, CK, methemoglobin level, and an EKG. EKG was done at 12:40 (roughly 3 h post-injection) showing sinus tachycardia with a ventricular rate of 120 bpm. The neurologic exam was otherwise intact and her abdominal exam was unremarkable. The patient was given supplemental oxygen, and IV 20% lipid emulsion therapy (Intralipid) was started for cardiac and CNS protection at 12:45. 20% Intralipid was administered first as a bolus of 100 mL over 5 min, followed by an infusion of 250 mL at 100 mL per hour. 1 mg of IV Ativan was administered at the same time as the Intralipid bolus. At 12:50–12:55 (roughly 3 h after the accidental overdose and 5–10 min after administration of the Intralipid bolus and Ativan), the patient was observed to have a generalized tonic–clonic seizure and became unresponsive to verbal and painful stimuli. The patient was given a nonrebreather facemask and maintained saturations over 85%. 4 mg IV Ativan was immediately administered and the seizure abated shortly thereafter. She did not have tongue biting or urinary incontinence. Her airway was protected throughout her seizure, and she remained hemodynamically stable. The patient was admitted to the telemetry unit in stable condition for 6 h of observation and diagnosed with local anesthesia systemic toxicity. Five hours into her observation period, the patient left against medical advice but was back to her normal baseline.

Discussion

Common local anesthetics

Local anesthetics bolster multimodal analgesia by potentially improving the quality of recovery, decreasing opioid exposure, decreasing postoperative nausea and vomiting, improving patient satisfaction, decreasing the length of hospital stay, and reducing the risk of chronic postsurgical pain.[4] Toxicity is influenced by formulation, anatomic site of administration, pharmacokinetic profile, and whether a vasoconstrictor is added. Table 1 is a summary of some of the most used local anesthetics used in a peri-procedural setting.

We used Table 1 and our patient's weight (51.76 kg) to calculate her maximum recommended dosage, for 2% lidocaine with epinephrine (20 mg/mL). This was done by multiplying her body weight by 7 mg/kg This value came out to be a total allowable dose of 362.32 mg of lidocaine, or 18.2cc. Her administered dosage of 38cc of 2% lidocaine is the equivalent of 760 mg; hence, her administered dosage was 2.10 times her maximum recommended amount, with an excess of 397.68 mg (19.88cc).

Warning signs and symptoms

The warning signs and symptoms for LAST are usually fairly rapid, occurring as soon as less than 5 min post-administration. For about 18% of individuals, prodromal symptoms can occur, including circumoral numbness, neurological issues (i.e., drowsiness, auditory disturbances, confusion), and a metallic taste in the mouth.[2] LAST primarily affects the CNS and cardiovascular systems because these two systems are particularly intolerant of anaerobic metabolism and subsequent inhibited oxidative phosphorylation. Of those with cardiac and/or CNS symptoms, 35% of patients experienced both CNS and cardiac symptoms, 24% experienced isolated cardiac symptoms, and 32% experienced isolated CNS symptoms.[7] It is important to note that not all cases of LAST have rapid-onset signs and symptoms. There have been cases described in the literature of late-onset LAST, suggesting that symptoms may present after 3–4 h.[8]

CNS effects include those listed as prodromal symptoms in addition to agitation, seizures, and loss of consciousness. Cardiovascular symptoms secondary to LAST include arrhythmias (i.e., bradycardia, tachycardia, ventricular tachycardia, ventricular fibrillation), conduction abnormalities (i.e., wide QRS complex, ST-segment changes), chest pain, dyspnea, blood pressure changes (i.e., hypotension, hypertension).[2]

Local anesthetic-specific adverse effects

Each local anesthetic has its adverse effects. Liposomal bupivacaine, or Exparel, is a common local anesthetic that poses a unique risk for toxicity due to its pharmacologic mechanism. Exparel uses a DepoFoam drug delivery to encapsulate bupivacaine, which slowly releases the anesthetic. This provides pain relief up to 72 h, whereas

Table 1. Commonly used local anesthetics, their maximum recommended dosages, duration of action, clinical uses, and the color of their respective vial labels.[4]

Local anesthetic	Maximum recommended dose (mg)	Maximum recommended dose (mg/kg)	Duration of action (min)	Indications	Comments	Color of cartridge band
Amino amides						
Lidocaine	200 500 w/epi	4.5 7 w/epi	30–120 120–240 w/epi	Wound infiltration, epidural, intrathecal, nerve blockade, IV Regional Anesthesia (Bier Block)	Tumescent solution: 28 mg/kg without liposuction and 45 mg/kg with liposuction[5,6]	Lidocaine HCl 2% Lidocaine HCl 2% with epi 1:50,000 Lidocaine HCl 2% with epi 1:100,000
Bupivacaine	175 (400 over 24 h)	2 2.5 w/epi	120–175 180–480 w/epi	Wound infiltration, epidural, intrathecal, ophthalmic, nerve blockade	Maximum dosing for liposomal Bupivacaine (Exparel) 266 mg (20 mL)[6]	Bupivacaine 0.5% with epi 1:200,000
Ropivacaine	225 (800 over 24 h)	3	120–140 140–180 w/epi	wound Infiltration, nerve blockade, epidural, intrathecal, continuous wound infusion	Avoid concurrent use with drugs that cause methemoglobinemia[2]	
Mepivacaine	400 550 w/epi	4.5 7 w/epi	45–90 120 w/epi	Wound Infiltration, epidural, intrathecal, nerve blockade		Mepivacaine HCl 3% Mepivacaine HCl 2% with levonordephrin 1:20,000
Levobupivacaine	150	2		Wound infiltration, nerve blockade, ophthalmic, epidural, intrathecal	S-isomer of Bupivacaine	
Amino esters						
Procaine	1000	7 10 w/epi	20–30 30–45 w/epi	Wound infiltration, epidural, intrathecal, nerve blockade		
2-Chloroprocaine	800 1000 w/epi	10–12 14 w/epi	30–60 60–90 w/epi	Wound infiltration, epidural, intrathecal, nerve blockade		
Tetracaine	Topical skin: 7 g/24 h Topical Children: 2 g/24 h Topical Mucous Membranes: 20 mg/dose Wound infiltration: 3 mg/kg per dose	1.5	120–180	Topical use, mucous membranes, wound infiltration		
Cocaine		1.5		Fiberoptic endotracheal intubation, topical anesthesia for surgery on the ear, nose, and throat		

bupivacaine alone lasts only 2–3 h. Due to this mechanism, caution should be taken when co-administering other non-liposomal local anesthetics, as this can cause rapid release of bupivacaine from the liposome and subsequent toxicity.[9] By itself, bupivacaine increases the risk of nausea and increasing length of recovery in a patient, especially in comparison to lidocaine.[7] However, when indicated for narcotic analgesia, liposomal bupivacaine is superior to the short-acting effects of lidocaine, when treating postoperative pain.[10]

Treatment

The objective of treating pharmacotoxicity is to reduce the anesthetic concentration in the body's tissue. Before this can be achieved, airway and resuscitative management must be addressed, followed by early infusion of a lipid emulsion solution. This is meant to prevent the mechanisms that potentiate LAST: hypercarbia, acidosis, and hypoxia.[7] Hypercarbia potentiates LAST by increasing cerebral blood flow to the brain, so preventing the occurrence of hypercarbia prevents local anesthetic delivery to the brain. Hypoxia is prevented and reduced via 100% oxygen ventilation.

Lipid emulsion. 20% intravenous lipid emulsion is strongly recommended in patients with LAST. The literature has shown that an initial 1.5 mL/kg over 2–3 min is recommended, followed by an infusion of 0.25 mL/kg/min 10 min later if necessary.[1,7,9] The use of repeat boluses of the initial 1.5 mL/kg may be recommended over the titrated infusion, to achieve the maximum emulsion dose. Furthermore, the initial combination of 1.5 mL/kg followed by the 0.25 mL/kg can be repeated up to two times before increasing the infusion dose to 0.5 L/kg/min.[10] It is important to record the dosing to avoid surpassing the recommended dose of 10–12 mL/kg, over the first 30 min.[2,7,9] In patients refractory to lipid emulsion treatment, cardiac bypass and or extracorporeal membrane oxygenation may be utilized.

The effectiveness seen with intravenous lipid emulsion is attributable to two mechanisms. First, current literature describes an indirect effect known as the "lipid shuttle." This is the emulsion's molecular ability to bind to the fat-soluble drugs in the tissues, specifically in the brain and heart. This decreases the local anesthetic concentration in these tissues, bringing it into the plasma and forming a lipid depot, secondary to binding to the lipid emulsion mix.[7] The lipid depot redistributes the drug to other areas of the body, including the muscle, adipose tissue, and liver where it is more easily excreted.[11] The second mechanism proposed in the literature describes lipid emulsion therapy's direct effects. It acts as a fatty acid substrate to the myocardium to provide positive inotropic support and increase cardiac output and oxygenation of the body's tissues.[7,9] In addition, lipid emulsion inhibits the release of nitric oxide, thereby increasing left ventricular systolic pressure.[11] It has also been shown that the use of long-chain triacylglycerides is more effective and with less adverse effects than medium-chain triacylglycerides, the latter of which can increase systemic vascular resistance and decrease cardiac contractility. It should be acknowledged that the lipid emulsion has some adverse effects: hyperthermia, allergic reaction, pancreatitis, hypercoagulabiity, and or elevated liver function values.[9]

Cardiac treatment. To treat cardiac symptoms, specifically advanced life support (ALS), a smaller dose of intravenous epinephrine is administered. In LAST patients, a dose of 1 ug/kg epinephrine is the recommended dosing for addressing ALS cardiac measures.[1,2] Epinephrine is usually administered 1 mg every 3–5 min, but in LAST patients, this can be arrhythmogenic in the patient, possibly interfering with cardiac resuscitation. The use of vasopressin may also be administered in the setting of ALS to restore circulation and hemodynamic stability. However, in LAST patients, vasopressin usage is contraindicated to avoid excessive vasoconstriction in these patients, preventing efforts to reduce the effects of LAST.[7] Here, calcium channel blockers and beta blockers are avoided due to the risk of excess cardiac depression.[2] After resolving cardiac complications in a LAST patient, a patient should be observed for greater than 6 h to ensure continued and long-term hemodynamic stability.[7] Cardiac toxicity secondary to LAST is primarily responsible for death in LAST patients; hence, continued monitoring includes routine measurements of blood pressure, electrocardiogram, and pulse oximetry values.[2,12]

Central nervous system treatment. Seizures are the most common feature of LAST and can worsen hypoxia and acidosis in the patient. This is

because local anesthetics can inhibit a particular potassium channel, thereby increasing neuronal excitability.[12] To treat seizures, benzodiazepines may be used due to their cardio-depressant effects, such as thiopentone and propofol.[10] Midazolam is another type of benzodiazepine that has been used to treat emergency seizures.[13] Ketamine is a dissociative anesthetic that has been shown to delay the onset of seizures and decrease future seizure risk in mice studies; however, this is not effective in any human studies.[14] In fact, ketamine is known to decrease the seizure threshold and should be used cautiously when co-administering local anesthesia. After resolving CNS symptoms in a LAST patient, continue to observe them for greater than 2 h to ensure long-term cognitive and neuromuscular function and stability.[10]

Prevention

There are guidelines provided by the Association of Anesthetics in the United Kingdom and the American Society of Regional Anesthesia and Pain Medicine. The most recent outstanding change in preventing LAST has been with ultrasound, to help identify unidentified intravascular injections. This reduces the risk of LAST fourfold by avoiding mistakenly giving a patient more local anesthetic than has already been administered to them.[7]

Anesthetic properties. Each local anesthetic has different properties such as lipid solubility, onset and duration of action, dosing, and indications. When considering dosing for a given patient, factors outside of the patient's weight should be carefully considered, that is, the use of ED95, the dose required to achieve the desired effect in 95% of the population.[12] In addition, dosing via incremental injections coupled with frequent aspiration can prevent LAST. The use of a pharmacological marker such as 2.5–5 µg/mL of epinephrine may help detect intravascular injection of local anesthetic in a patient.[2]

Injection techniques. The site of injection also plays a role in the dosing and avoiding LAST in the patient. There is a particularly high risk of LAST in paravertebral, intercostal, and intrapleural blocks. Furthermore, there is a greater occurrence of LAST in upper limb blocks than in lower limb blocks. When a local anesthetic is administered via a continuous catheter, there is an increased risk of LAST than when administered as a single shot. This is due to possible accumulation of the local anesthetic in the tissues with continuous catheter administration, especially in individuals with less muscle mass. If local anesthetic is applied and a procedure is performed while the patient is awake, this can increase the likelihood that healthcare providers can detect the neurological signs of LAST, but it does not necessarily decrease the risk of LAST.[7]

Patient factors. Beginning with patients' ages, the elderly and infants have lower lean mass and as was touched on previously, this increases the risk of LAST. This is because skeletal muscle acts as a neutral local anesthetic storage reservoir. The elderly population often has more comorbidities that affect drug metabolism, as will be explained next. Infants have immature hepatic cytochrome P450 in infants and lower levels of the alpha1 acid glycoprotein.[2,7] Cytochrome P450 and the alpha1 acid glycoprotein play a role in metabolizing the local anesthetics and helping the body excrete them, so infants are unable to do this as efficiently as adults. The action of acute phase alpha1 acid glycoprotein helps reduce anesthetic levels in free plasma. The use of various medications can also increase the risk of LAST, such as beta-blockers, digoxin, cytochrome P450 inhibitors, and calcium antagonists.[2]

Metabolic diseases and comorbidities affect how patients' bodies respond and mechanistically metabolize drugs. Such diseases, which have greater prevalence in women than men, include diabetes, mitochondrial diseases, carnitine deficiency, and malnourishment.[2,7] Malnourishment reduces plasma protein levels. As noted earlier, local anesthetics particularly affect the CNS and cardiovascular systems. Patients with reduced cardiac contractility and or conduction abnormalities secondary to cardiac disease can experience an increase in the peak plasma concentration of the local anesthetic, as well as increase its post-intravascular absorption in the body.[7] On the other hand, increased cardiac output and vascular diffusion can increase absorption of local anesthetics, subsequently increasing the risk of developing LAST.[2] As a result, ischemia can result in their patients because anesthetics impair mitochondrial metabolism and adenosine triphosphate production, further exacerbating their cardiac disease and prolonging the effects of LAST. Patients with hepatic and or renal disease are also at increased risk for developing LAST.[7]

Due to decreased and or impaired hepatic blood flow, these patients have decreased levels of hepatic enzymes to metabolize the local anesthetic. These patients may have uremia and acidosis, increasing the concentration of free local anesthetic.

Pregnant women have decreased levels of alpha1 acid glycoproteins and reduced volume of epidural and subarachnoid space.[7] Moreover, they have a larger volume of distribution secondary to the fetus, so this decreases the amount of active (unbound) levels of local anesthetic; as such, these two factors counteract one another.[1] Lipid solubility has to be considered in relation to the placenta, which has higher lipid solubility. Here, low molecular weight substances can more easily cross the placenta. Also, the fetal environment is acidotic, trapping the local anesthetic and preventing it from returning to maternal circulation and being excreted from the mother's body. It is recommended to use the minimal effective local anesthetic dose in these individuals.[10]

Conclusion

LAST can leave patients with neurological and/or cardiac sequelae, possibly resulting in death. Warning signs such as circumoral numbness, drowsiness, confusion, heart palpitations, and so forth, should be monitored for and explained to the patient prior to patient discharge. When indicated, common LAST treatment involves one or more boluses of 20% lipid emulsion, ventilation to reduce and/or prevent hypercarbia, ALS with post-monitoring to establish hemodynamic stability, benzodiazepines for seizures, neurological monitoring to ensure cognitive stability and baseline neuromuscular function.

Our patient experienced LAST symptoms (diffuse tremors; paresthesias of the face and mouth; intermittent spasticity; single generalized tonic–clonic seizure) secondary to lidocaine toxicity. She was treated accordingly with 20% lipid emulsion IV, 100% O_2 nasal cannula, Ativan IV, and benzodiazepines, with no further complications.

This paper focused on commonly used local anesthetics. Here, the type of drug used, the anatomic site of administration, injection technique coadministration with a vasoconstrictor, and its pharmacokinetic profile are significant in the occurrence of LAST. In addition, patient factors (i.e., age; comorbidities; percentage of lean muscle mass; cytochrome P450 and alpha1 acid glycoprotein activity; cardiac contractility; and pregnancy) must be considered to further adjust drug dosing. With these factors in mind, the adverse outcomes associated with LAST can be prevented and/or reduced.

Declarations

Ethics approval and consent to participate

Our study did not require board ethical approval because it is a single case study and no identifying information was included.

Consent for publication

The patient in this study was given a copy of the manuscript and provided us with written informed consent to publish.

Author contributions

Deana Chan: Investigation; Methodology; Writing – original draft; Writing – review & editing.

Christopher Wood: Conceptualization; Investigation; Project administration; Supervision; Writing – original draft; Writing – review & editing.

Andre Rafizadeh: Conceptualization; Investigation; Supervision; Validation; Writing – review & editing.

Michael Nagai: Conceptualization; Methodology; Project administration; Supervision; Validation; Writing – review & editing.

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