A 15.8-kg (34.8-lb) 6-month-old female German Shorthair Pointer was evaluated 2 hours after being crushed by a utility pole during a farming accident. The utility pole had struck the dog across the back at the level of the thoracolumbar spine. The patient had not walked since the accident and was laterally recumbent. On initial physical examination, there was no evidence of cardiovascular or respiratory compromise. Because of concerns about the stability of the vertebral column and the potential for a vertebral luxation or fracture, a limited neurologic examination with minimal patient manipulation was performed, revealing signs of deep pain. An IV catheter was placed, and the patient was started on a fentanyl CRI (3 to 5 μg/kg/h [1.4 to 2.3 μg/lb/h], IV) for analgesia. Later that day, the patient was anesthetized for a CT myelogram. The patient was premedicated with fentanyl (3 μg/kg, IV) and lidocaine (1 mg/kg [0.45 mg/lb], IV). Anesthesia was induced with ketamine (5 mg/kg, IV) and midazolam (0.25 mg/kg [0.11 mg/lb], IV), and the patient was orotracheally intubated. Anesthesia was maintained with isoflurane (0.11 mg/lb/h, IV) for analgesia. Later that day, the patient was scheduled for another surgery the following morning.

The authors thank Dr. Jon Bach and Dr. Natasha Evans for assistance in managing this case.

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The authors thank Dr. Jon Bach and Dr. Natasha Evans for assistance in managing this case.

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concern that this dog could convulse on recovery from anesthesia or die of cardiovascular collapse prior to any decrease in the plasma lidocaine concentration.

The patient was immediately treated with ILE therapy based on the protocol published in a case report describing the successful use of ILE for treatment of moxidectin toxicity in a young dog. Within minutes after discovery of the lidocaine overdose, the patient received a bolus of 20% lipid emulsion (2 mL/kg [0.9 mL/lb] IV, over 10 minutes) followed by a CRI (4 mL/kg/h [1.8 mL/lb/h], IV). The patient was mechanically ventilated to a P_{ETCO2} of 40 to 50 mm Hg. Approximately 5 minutes after the ILE bolus was started, the mean arterial blood pressure was 85 mm Hg, and heart rate was 90 beats/min with a normal sinus rhythm. It was then decided that the patient was stable enough to complete CT and that the additional time under anesthesia would allow a larger dose of ILE to be administered prior to recovery. Approximately 20 minutes after beginning the ILE infusion (the first 10 minutes was the bolus followed by 10 minutes of a CRI as described), the patient began spontaneously breathing again and was able to maintain a P_{ETCO2} between 35 and 40 mm Hg. Administration of isoflurane was discontinued, and the patient was maintained on 100% oxygen. The ILE CRI (4 mL/kg/h) was continued into recovery, and the patient was continuously monitored for ocular reflexes, jaw tone, temperature, respiratory rate, pulse rate, SpO2, and P_{ETCO2} and noninvasive blood pressure measurement and ECG were performed. Midazolam (0.25 to 5 mg/kg, IV) was ready to be administered in the event of any seizures on recovery. Recovery was prolonged but uneventful; approximately 50 minutes later, the dog was alert and was extubated without any noticeable muscle twitching or seizures. The ILE therapy had lasted approximately 1 hour 10 minutes, during which time 6.2 mL/kg (2.8 mL/lb) had been administered. The patient was continuously monitored in the critical care unit for the next 24 hours. No signs of lidocaine toxicity were observed. The patient had an uneventful stay in the hospital and was discharged 4 days later.

Discussion

Successful use of ILE therapy to treat lipophilic drug toxicoses in dogs has been described in 2 previous reports. Successful use of ILE therapy to treat lipophilic drug toxicoses in dogs has been described in 2 previous reports.1-3 Both reports describe dogs evaluated on an emergency basis because of abnormal neurologic signs following ingestion of equine dewormer paste containing ivermectin or moxidectin. Following treatment with similar dosing of ILE, both dogs recovered fully, without any permanent neurologic dysfunction. The ILE dosing protocol used in the case described in the present report to treat a lidocaine overdose marks the third successful clinical use of the ILE dosing strategy first described by Crandell and Weinberg. In addition, to our knowledge, there have not been any previous reports in the veterinary literature describing the use of ILE therapy in a dog for local anesthetic overdose in a clinical setting.

Intravenous lipid emulsion for the treatment of local anesthetic toxicosis was first described in rats in a research setting.4 Weinberg et al later demonstrated that ILE therapy could rescue dogs from bupivacaine-
induced cardiac arrest. After induction of cardiac arrest with bupivacaine (10 mg/kg, IV), basic life support was initiated and dogs were treated with equal volumes of either saline (0.9% NaCl) solution or ILE. All of the dogs treated with ILE achieved return of spontaneous circulation and survived, whereas none of the dogs treated with saline solution achieved return of spontaneous circulation. Several other investigations have documented successful use of ILE for treating a variety of lipophilic drug toxicoses in research animals.1,6–12

In 2006, Rosenblatt et al12 reported the first clinical use of ILE therapy to resuscitate a middle-aged man who went into cardiac arrest shortly after receiving a bupivacaine nerve block. Standard resuscitative measures were initiated immediately after arrest; however, after 20 minutes of cardiopulmonary cerebral resuscitation, the patient failed to achieve a return of spontaneous circulation. A bolus of ILE was then administered, and the patient quickly achieved return of spontaneous circulation, eventually making a complete recovery without any neurologic or cardiac dysfunction. Several other case reports13–15 in human patients document the successful use of ILE for resuscitation following local anesthetic–induced cardiac arrest. Seizures and other CNS signs related to local anesthetic toxicosis have also been successfully reversed following ILE therapy in humans.16,17

Several formulations of ILE are commercially available. A 20% soybean oil–based emulsion of long-chain triglycerides that was originally developed for use as the lipid component of parenteral nutrition formulations is the product that has been most commonly used for the treatment of local anesthetic toxicosis in human patients.18 The means by which ILE treatment is an effective antidote for local anesthetic toxicosis has been attributed to multiple mechanisms of action involving partitioning, metabolic, and ion channel effects.18 The initial proposed mechanism of action was the lipid sink, whereby expansion of the intravascular lipid phase provides a lipophilic solvent that will sequester lipophilic drugs, thus making less of the drug available to exert toxic effects. The lipid sink theory has been supported by studies19,20 describing the effects of ILE on the amount of radiolabeled bupivacaine in myocardial tissue and the amount of bupivacaine present in the lipid versus aqueous phases of plasma. It has also been demonstrated that low doses of ILE (ie, volumes too small to appreciably reduce the bupivacaine concentration in the plasma) are effective in reversing bupivacaine–induced cardiac dysfunction,21 suggesting mechanisms other than the lipid sink. Fatty acids are the preferred energy substrate of the myocardium, and bupivacaine inhibits fatty acid metabolism.22 It has been proposed that ILE treatment may reverse local anesthetic–induced cardiac dysfunction by providing enough lipid substrate to alleviate bupivacaine’s inhibition of fatty acid metabolism in myocardial tissue.23 Other investigators have reported that ILE treatment reduces cardiac ischemia reperfusion injury and exerts cytoprotective effects by maintaining mitochondrial membrane integrity.22,23

Treatment of local anesthetic toxicosis with ILE in people appears to be safe with minimal risk of adverse effects at the reported doses (3.7 mL/kg [1.7 mL/lb], IV).18 In the human literature, there are no reports of serious complications associated with the use of ILE for the treatment of drug toxicosis, even when massive overdose of ILE has been accidentally administered.24 Administration of ILE results in hypertriglyceridemia that may pose a theoretical risk for pancreatitis; however, a direct cause-and-effect relationship between serum lipid concentration and pancreatitis has yet to be demonstrated.25 Lipid emulsion has also been found to have minimal cardiovascular effects in conscious dogs.26 The cost of ILE administration is also minimal (approx $20/500 mL in the United States). Twenty percent lipid emulsions are isotonic and do not require a central IV access for administration, but strict aseptic technique should be used to prevent any bacterial contamination and associated sequelae.

The value of ILE for the treatment of local anesthetic toxicosis has been recognized by the American Society of Regional Anesthesia and the American Heart Association, and both organizations have published guidelines for the use of ILE treatment in resuscitation following local anesthetic overdose in people.27,28 Given the demonstrated success of ILE treatment protocols for treating local anesthetic toxicosis in both laboratory animals and human patients, low risk of adverse effects, and low cost, ILE treatment should be considered in any dog that has received an overdose of local anesthetic.

References


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Correction: Association between oral health status and retrovirus test results in cats

In the report “Association between oral health status and retrovirus test results in cats” (*J Am Vet Med Assoc* 2014;245:916–922), the first author’s name is listed incorrectly. The first author’s name is Matthew R. Kornya.