

Local Anesthesia Toxicity and Lipid Rescue

Kim A. Noble, PhD, RN, CPAN

Regional anesthesia as a treatment modality for the control of surgical pain has been in practice since the late 1880s with the introduction of cocaine. The use of lipid emulsion therapy as an emerging treatment for the rare but life-threatening development of local anesthesia systemic toxicity (LAST) has been in the animal literature for approximately 20 years, and case reports have documented successful results with this treatment in humans. The perianesthesia nurse has a significant role in the assessment, communication with the anesthesia care provider, and the emergent management of the patient experiencing LAST. Using a fictitious case study of a patient with LAST, the conduction of sensory information will be reviewed. The pharmacologic characteristics of local anesthetics will be presented, and the evidence-based practice recommendations for the prevention, monitoring, and treatment will be provided.

Keywords: LAST, local anesthesia toxicity, lipid emulsion rescue.

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OBJECTIVES—(1. REVIEW THE action of local anesthetics in the body; (2). Describe the symptoms of local anesthetic systemic toxicity (LAST); (3). Discuss the treatment for LAST.

A significant challenge for perianesthesia nurses is the anticipation of potential complications while providing safe care for patients receiving emerging treatment modalities. Changes in surgical and anesthetic care are inevitable in the desire for improved patient outcomes, mandating the nurse is knowledgeable of the application of evidence-based practice changes to provide safe care. One such treatment modality is the provision of care for patients receiving local anesthesia (LA) and the rapid assessment and acute management of the rare life-threatening development of local

anesthetic systemic toxicity (LAST)¹ with the administration of lipid emulsion therapy.

LA toxicity was reported as early as 1880, shortly after the introduction of cocaine into clinical practice. The initial publication included descriptions of respiratory failure and seizures but failed to include related cardiac abnormalities common to LAST. These early publications led to an American Medical Association study in the 1920s to identify this complex disorder.² Despite the release of more fat soluble LA agents in the 1960s and 1970s, LAST remained a problem, prompting the release of a “Dear Doctor” warning for the use of bupivacaine in obstetric anesthesia.² The use of lipid infusion for the emergent management of LAST appeared in the animal literature in the 1990s; however, these findings have yet to be consistently applied to human research. The publication of case reports of the successful use of lipid rescue for the treatment of LAST has added clinical data as early as 2006.³ The American Society of Regional Anesthesia (ASRA) conducted a survey of national anesthesia practice and identified a lack of uniform approach to the identification and management of LAST. In 2010, the ASRA released an evidence-based practice

Kim A. Noble, PhD, RN, CPAN, and ASPAN Director for Research, is Assistant Professor, School of Nursing, Widener University, Chester, PA.

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Address correspondence to Kim A. Noble, Widener University, One University Place, Chester, PA 19013; e-mail address: kimmoble@verizon.net.

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advisory for the emergent treatment of LAST including the use of lipid emulsion therapy.²

Rapid recognition of the clinical manifestations of LAST is important but challenging because of the variability of symptom presentation, requiring diligent assessment, and monitoring by the perianesthesia nurse. Using a fictitious perianesthesia case study presentation, the mechanism of action and pharmacokinetics of LA will be discussed, followed by the pathophysiology and clinical manifestation of LAST. Finally, the rationale and recommended lipid emulsion therapy will be presented and applied to the post-LA patient care, with the identification of patient care priorities in the perianesthesia setting.

Perianesthesia Case Study

Michael Osborn (MO) was a 52-year-old American Society of Anesthesiology status II male undergoing a total knee replacement with general anesthesia and a planned femoral block for post-operative pain management to be given on completion of the procedure. MO was 5'6" tall, 180 pounds (80 kg), and had a past medical history of hypertension well controlled with lisinopril daily. MO presented to the preoperative unit for elective surgery in good spirits with normal vital signs and assessment data. He reported a baseline pain level of 7 on a verbal zero to 10 numeric pain scale (0 indicating no pain and a 10 indicating severe pain).

MO was prepared for his elective surgical procedure and transferred to the operating room (OR) table joking with the anesthesia and nursing staff. He was given 100% oxygen by mask and received intravenous midazolam 2 mg and fentanyl 50 mcg during the application of noninvasive monitoring. General anesthesia was induced using succinylcholine and a total of 200 mg of propofol. The operative case proceeded without complication with a blood loss of approximately 400 mL. MO was noted to have transient hypotension on induction and received approximately 600 mL of lactated ringers solution with complete resolution. Anesthetic maintenance was provided using sevoflurane and a single dose of vecuronium. A total of 6 mg of morphine sulfate was given in incremental doses over the final 40 minutes of the operative case and 4 mg of ondansetron was

administered. MO was reversed and extubated in the OR without incident and transported to the phase I postanesthesia care unit (PACU). MO's admission vital signs included BP 148/84, a normal sinus rhythm at 96 beats per minute, 24 shallow respirations per minute, and temperature of 96.4°F. He reported pain at 10 using a verbal 0 to 10 pain scale and was noted to be slightly restless. Handoff of care was completed with the anesthesia and OR providers, and MO was prepared for the administration of a femoral block for the treatment of his surgical pain. There were no abnormalities identified on head-to-toe admission assessment by the phase I PACU nurse.

MO received a total of 40 mL of local anesthetic for the femoral block using 20 mL of 0.5% of *bupivacaine* (Marcaine) and 20 mL of 1.5% of *mepivacaine* (ropivacaine). The femoral artery, vein, and nerve were identified using continuous ultrasound throughout the procedure. Injection was completed using a slow incremental injection technique with aspiration after each 5 mL injection. At no time was blood aspirated nor was an increase in pain or paresthesia reported. MO was awake and conversant throughout the procedure, requesting a drink.

MO remained awake with stable vital signs and verbalized the gradual improvement in his pain over the next 15 minutes. Suddenly, MO began yelling incoherently. He was noted to be in a slow wide complex ventricular rhythm with frequent multifocal premature ventricular beats on cardiac monitoring. Anesthesia was notified and the following vital signs obtained: BP 74/32, heart rate 44 and irregular, respirations 12 per minute, and shallow with a saturation of 86%. MO was placed on a 100% non-rebreather mask and his head of bed flattened. He became unresponsive and was noted to have ventricular fibrillation on cardiac monitoring. Advanced Cardiac Life Support (ACLS) was initiated using good quality cardiopulmonary resuscitation. Over the next 10 minutes, MO was defibrillated a total of 6 times and received epinephrine, vasopressin, and amiodarone as per ACLS protocol but remained in refractory ventricular fibrillation. A 20% lipid emulsion was ordered with an initial bolus of 120 mL given, followed by a continuous drip at 20 mL per hour via infusion pump. MO was noted to convert into a sinus rhythm and begin

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