Case Report

SAGE Open Medical Case Reports

SAGE Open Medical Case Reports
Volume 11: 1–3

DOI: 10.1177/2050313X231153776
journals.sagepub.com/home/sco
SAGE

Lipid emulsion–induced recovery from unconsciousness caused by lidocaine toxicity: A case report

Miyeong Park[1], Ho Kyung Yu[1], Soo Hee Lee[1,2,3], Seong-Ho Ok[1,2,3], Sangcheol Yoon[2] and Ju-Tae Sohn[2,3]

Abstract

Lipid emulsion is used to treat systemic toxicity caused by local anesthetics. In addition, lipid emulsion was reported to be effective in ameliorating cardiovascular depression evoked by non-local anesthetic drug toxicity with high lipid solubility. A 47-year-old woman underwent local anesthetic infiltration with 40 mL of 2% lidocaine (20 and 20 mL) to remove a mass in the upper back. After operation, she experienced convulsions and loss of consciousness due to lidocaine toxicity. Midazolam followed by lipid emulsion was administered to treat central nervous system symptoms including unconsciousness and decreased Glasgow Coma Scale. The patient recovered from unconsciousness and presented improved Glasgow Coma Scale after lipid emulsion administration, and then fully recovered from local anesthetic systemic toxicity. This case suggests that early lipid emulsion treatment, before further progression of local anesthetic systemic toxicity, provides an enhanced recovery from unconsciousness and decreased Glasgow Coma Scale due to lidocaine toxicity.

Keywords

Lidocaine, lipid emulsion, unconsciousness, toxicity, Glasgow Coma Scale, local anesthetic systemic toxicity

Date received: 13 September 2022; accepted: 11 January 2023

Introduction

Local anesthetics (LAs) are widely used to provide analgesia during various operations. If administered in an excessive dosage or at an incorrect anatomic location, LA can cause localized and systemic toxicity.[1] In addition, even when a normal dose of LA is administered, local anesthetic systemic toxicity (LAST) can develop in patients with predisposing factors, such as extreme age, mitochondrial disease, and carnitine deficiency.[1,2] In general, central nervous system toxicity of LA occurs at lower concentrations than cardiovascular toxicity, which can be a precursor to cardiovascular complications of LAs.[3,4] Recently, intravenous lipid emulsion (LE) has been widely used as a therapeutic agent with advanced cardiovascular life support treatment in LAST.[5] We report a case of treatment with midazolam followed by LE, which contributed to an enhanced recovery from unconsciousness and a decreased Glasgow Coma Scale (GCS) caused by lidocaine toxicity.[6] Ethical approval was waived by the institutional review board of Changwon Gyeongsang National University Hospital (GNUCH 2022-08-026).

Case report

A 47-year-old woman (64 kg, 164 cm, American Society of Anesthesiologist Physical Status: 1) was admitted for excision of a mass in her upper back under local anesthesia. She had no underlying diseases or any recent history of medication. She had never experienced seizures, and preoperative physical and laboratory examinations were unremarkable. When the patient arrived at the operating room without

[1]Department of Anesthesiology and Pain Medicine, Gyeongsang National University Changwon Hospital, Changwon-si, Republic of Korea
[2]Department of Anesthesiology and Pain Medicine, Gyeongsang National University Hospital, Gyeongsang National University College of Medicine, Jinju-si, Republic of Korea
[3]Institute of Health Sciences, Gyeongsang National University, Jinju-si, Republic of Korea

Corresponding Author:
Ju-Tae Sohn, Department of Anesthesiology and Pain Medicine, Gyeongsang National University Hospital, Gyeongsang National University College of Medicine, 79 Gangnam-ro, Jinju-si 52727, Republic of Korea.
Email: jtsohn@gnu.ac.kr

premedication, basic anesthetic monitors, such as blood pressure monitoring and pulse oximetry, were applied, and intravenous access (18 gauge) was secured. Vital signs (systolic and diastolic blood pressure, 130/90 mm Hg; heart rate, 75 beats/min; respiratory rate, 20 breaths/min; and body temperature, 36.4°C) were stable and were maintained until the end of surgery. The patient was placed in the prone position to expose the preoperatively estimated mass (approximately $5 \times 5\,cm^2$) in upper back. Surgery was initiated under local infiltration anesthesia using an initial 20 mL of 2% lidocaine. The patient experienced pain mid-surgery, and an additional 20 mL of 2% lidocaine was infiltrated around the mass during the 40 min operation. Finally, a $7 \times 6 \times 4\,cm^3$ encapsulated lipoma was removed without complications, and vital signs were stable during the excision. The operator noticed that the patient was experiencing convulsions during disinfection and dressing of the wound. Thereafter, the patient experienced generalized tonic-clonic seizures and became unconscious. She was placed in the supine position, and her airway was secured by an anesthesiologist using a head tilt and jaw thrust maneuver. She was transferred to the recovery room thereafter; vital signs were as follows: systolic and diastolic blood pressure, 184/90 mm Hg; heart rate, 88 beats/min; respiration rate, 20 beats/min; and body temperature, 36.4°C. On neurological examination, the patient could not respond to commands and presented a decreased GCS (1/2/1). However, the neurologist did not detect any neurological signs that were suggestive of an intracranial lesion, and the generalized seizures subsequently subsided. Midazolam (2 mg) was intravenously administered, and the patient was closely observed; 5 L/min of oxygen via a face mask was supplied. Fifteen minutes after midazolam infusion, she was unable to recover consciousness, but her vital signs were stable. A 20% LE with 100% long-chain fatty acids (MG TNA®-Peri injection, MG human health, Jincheon-gun, Republic of Korea) was administered intravenously with a volume of 169 mL (1.5 mL/kg followed by 0.25 mL/kg/min for 4.5 min). Plasma lidocaine concentration was not measured in the patient. She recovered consciousness with a clearly improved GCS (4/5/5) 20 min after LE administration. Her neurologic and hemodynamic status was stable for 30 min after LE infusion; therefore, she was transferred to the general ward and closely observed. She was discharged without any complications 1 day after the surgery and did not show any adverse effects on the follow-up visit.

Discussion

The predisposing factors for systemic toxicity caused by LAs include patient-related and patient-unrelated risk factors.[3] The patient-related predisposing factors include extreme age (infant or the elderly), underlying disease, low muscle mass, low plasma binding protein, and mitochondrial and metabolic disease.[3] Patient-unrelated predisposing factors include the type of LA (risk of cardiotoxicity: bupivacaine > levobupivacaine > ropivacaine > lidocaine), amount of LAs, and vascularity of the area undergoing LA injection.[3,4] The maximal recommended dose of lidocaine without and with epinephrine is 4.5 and 7 mg/kg, respectively.[3] The maximal recommended dose of lidocaine in this patient was 288 (4.5 mg/kg × 64 kg); however, the patient received a higher dose, approximately 2.7 times (800 mg) the recommended dose, which produced central nervous system symptoms, including seizures and unconsciousness. LAST can produce prodromal symptoms, which include perioral numbness, dysarthria, drowsiness, and tinnitus.[3] Subsequently, central nervous system complications such as convulsion and unconsciousness can occur, followed by cardiovascular symptoms, including cardiac arrhythmia, hypotension, and QRS widening, which eventually leads to cardiac arrest.[3] Up to 50% of cases associated with LAST occur by non-anesthesiologists.[1] In light of this case, the following recommendations are helpful to prevent LAST: (1) education regarding maximal recommended dose of LAs for non-anesthesiologists, (2) using lower concentration of LAs and adjustment of LA dosage based on lean body mass, (3) usage of epinephrine as an intravascular injection marker, and (4) sufficient waiting time to achieve adequate anesthesia from local infiltration of LA to the beginning of operation.[1] Currently, LE, which is mainly used for parenteral nutrition in the critical care unit, is used to treat LAST.[5] In lipid shuttle, a widely accepted underlying mechanism of LE treatment, LEs absorb lipid-soluble LA lidocaine (Log P: 2.44) from the heart and brain, after which lidocaine is transported to the liver and muscles for detoxification and redistribution.[7] The early administration of LE alone for systemic toxicity caused by bupivacaine and ropivacaine was reported to treat prodromal and central nervous system symptoms, such as perioral numbness, agitation, restlessness, dysarthria, and dizziness.[8,9] In addition, a combined treatment with LE and benzodiazepine in a patient with only neurologic symptoms due to ropivacaine toxicity was reported to ameliorate the symptoms.[10,11] Furthermore, a prospective clinical study reported that LE shortened recovery time for isoflurane anesthesia and increased GCS in drug toxicity caused by non-LA.[12,13] The administration of midazolam did not allow recovery of consciousness in this patient, but LE induced complete consciousness recovery 20 min after administration. As midazolam, with a 1.5–3 h elimination half-life, leads to sedation and increased seizure threshold, and LE additionally improved GCS from 4 to 14, the complete recovery of consciousness observed in this patient can be ascribed to LE administration, when considering previous reports.[7,10–14] The recommended LE dosing regimen for LAST from the Association of Anesthetists of Great Britain and Ireland is as follows: 1.5 mL/kg bolus administration of 20% LE followed by

0.25 mL/kg/min of 20% LE.[15] The following measures should be performed to prevent LAST[3,4]: (1) assessment of predisposing factors, (2) adequate monitoring, (3) ultrasound-guided nerve block, (4) not exceeding the maximally recommended dose of the LA, (5) administration of several divided doses, (6) aspiration of the injection site, (7) LE preparation, and (8) use of a LA with lowest cardiac toxicity.

Conclusion

In conclusion, this case suggests that early LE treatment, before further progression of LAST, contributes to an enhanced recovery from unconsciousness and decreased GCS caused by lidocaine toxicity.

Author contributions

Conceptualization: Park M, Ok SH, and Sohn J-T
Data curation: Park M, and Ok SH
Forma analysis and investigation: Park M, and Lee SH
Project administration: Sohn J-T and Ok SH
Resource: Yu HK, and Yoon S
Supervision and validation: Sohn J-T
Writing original draft: Sohn J-T and Ok SH
Writing review and editing: Park M, Yu HK, Lee SH, Ok SH, Yoon S, Sohn J-T

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics approval

Ethical approval was waived by the institutional review board of Changwon Gyeongsang National University Hospital (GNUCH 2022-08-026).

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

ORCID iD

Ju-Tae Sohn https://orcid.org/0000-0003-0102-5800

References

1. Neal JM, Barrington MJ, Fettiplace MR, et al. The third American Society of Regional Anesthesia and Pain Medicine practice advisory on local anesthetic systemic toxicity: executive summary 2017. *Reg Anesth Pain Med* 2018; 43(2): 113–123.
2. Weinberg GL, Laurito CE, Geldner P, et al. Malignant ventricular dysrhythmias in a patient with isovaleric acidemia receiving general and local anesthesia for suction lipectomy. *J Clin Anesth* 1997; 9(8): 668–670.
3. Gitman M, Fettiplace MR, Weinberg GL, et al. Local anesthetic systemic toxicity: a narrative literature review and clinical update on prevention, diagnosis, and management. *Plast Reconstr Surg* 2019; 144(3): 783–795.
4. Groban L and Dolinski SY. Differences in cardiac toxicity among ropivacaine, levobupivacaine, bupivacaine, and lidocaine. *Tech Reg Anesth Pain Manag* 2001; 5: 48–55.
5. Ok SH, Hong JM, Lee SH, et al. Lipid emulsion for treating local anesthetic systemic toxicity. *Int J Med Sci* 2018; 15(7): 713–722.
6. Teasdale G and Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974; 2: 81–84.
7. Fettiplace MR and Weinberg G. The mechanisms underlying lipid resuscitation therapy. *Reg Anesth Pain Med* 2018; 43(2): 138–149.
8. Spence AG. Lipid reversal of central nervous system symptoms of bupivacaine toxicity. *Anesthesiology* 2007; 107(3): 516–517.
9. Lee SH, Park M, Ok SH, et al. Early lipid emulsion treatment of central nervous system symptoms induced by ropivacaine toxicity: a case report. *Am J Ther* 2020; 28: e736–e738.
10. Nguyen VH and White JL. Further support for the early administration of lipid emulsion in the treatment of ropivacaine-induced central nervous system toxicity. *J Anesth* 2012; 26(3): 479–480.
11. Widfeldt N and Kolmodin L. CNS symptoms of ropivacaine in bloodless field disappeared with intralipid. *Lakartidningen* 2014; 111: 742–743.
12. Li Q, Yang D, Liu J, et al. Intravenous lipid emulsion improves recovery time and quality from isoflurane anaesthesia: a double-blind clinical trial. *Basic Clin Pharmacol Toxicol* 2014; 115(2): 222–228.
13. Taftachi F, Sanaei-Zadeh H, Sepehrian B, et al. Lipid emulsion improves Glasgow coma scale and decreases blood glucose level in the setting of acute non-local anesthetic drug poisoning—a randomized controlled trial. *Eur Rev Med Pharmacol Sci* 2012; 16(Suppl. 1): 38–42.
14. Prommer E. Midazolam: an essential palliative care drug. *Palliat Care Soc Pract* 2020; 14: 2632352419895527.
15. AAGBI safety, http://p-h-c.com.au/doc/Local_Anaesthesia_Toxicity.pdf (accessed 8 August 2022).