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LETTER TO THE EDITOR

Further support for the early administration of lipid emulsion in the treatment of ropivacaine-induced central nervous system toxicity

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To the Editor:

In the June 2011 issue of the *Journal of Anesthesia*, Mizutani et al. [1] reported the treatment of ropivacaine-induced central nervous system (CNS) toxicity with intravenous (IV) 20% lipid emulsion, demonstrating significant reduction in unbound plasma ropivacaine accompanying neurological recovery. We would like to give further support for the use of lipid emulsion in this context by reporting a similar case. In our patient, CNS toxicity manifested as visual hallucinations and myoclonic movements of the head and neck. Treatment with 100 ml of IV 20% lipid emulsion, given as a single bolus over 1 min, resulted in instant resolution of symptoms and likely prevented further neurological or cardiovascular morbidity.

Our American Society of Anesthesiologists (ASA) physical status (PS)-1, 1.82-m, 72-kg, 19-year-old male patient received an ultrasound-guided supraclavicular brachial plexus block for ulnar medial collateral ligament repair. The brachial plexus was easily identified sonographically with a 13-6 MHz linear array Sonosite transducer (Bothell, WA, USA). After sedation with 2 mg of IV midazolam, a 21-gauge 90-mm Teleflex block needle (Research Triangle Park, NC, USA) was advanced to the "corner pocket" and 5 ml of 0.5% ropivacaine was injected after negative aspiration. Because local anesthetic spread was not visualized, the block needle was repositioned, and an additional 10 ml of ropivacaine was given, this time with good visualization of spread. Vital signs at this time were sinus rhythm (SR) with heart rate (HR) 58 beats per min (BPM), blood pressure 135/53 mmHg, and oxygen saturation of 99%. The patient suddenly turned his head back and forth repetitively toward the ultrasound machine. When asked where he was, he replied that he was playing in a baseball game. Vital signs escalated to HR 160 BPM (SR), blood pressure 190/89 mmHg, and oxygen saturation 99%. A single bolus of 100 ml (1.5 ml/kg) of Intralipid® 20% fat emulsion (Deerfield, IL, USA) was given over 1 min, per the recommended guidelines of both the Association of Anaesthetists of Great Britain and Ireland (AAGBI) [2] and Weinberg [3]. Supplemental oxygen was administered by face mask, and another 2 mg of IV midazolam was given after completion of the lipid bolus to raise the seizure threshold. There was no loss of consciousness or seizure activity. The patient regained complete orientation within 2 min as vital signs normalized. He remained asymptomatic afterward.

In our case, the first 5 ml of ropivacaine was likely injected intravascularly, resulting in a rapid rise in the CNS concentration of ropivacaine. Prompt administration of 20% lipid emulsion proved to be efficacious in rapidly reversing the CNS symptoms and may have prevented any cardiovascular involvement. Two other factors may have played a role in the patient's course: benzodiazepine administration and the natural drop in the plasma level of ropivacaine over time. However, the small dose of midazolam, coupled with its late administration, was unlikely to have halted the symptoms so drastically. Likewise, although the course of events in this case may be attributed to a drop in brain concentration of local anesthetic, the direct temporal relationship between the IV lipid administration and instantaneous recovery supports the notion that lipid was the major player. The reversal of CNS symptoms witnessed here lends further support for the early usage of 20% lipid emulsion, following the guidelines of the AAGBI or Weinberg, in the treatment of local anesthetic systemic toxicity.

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