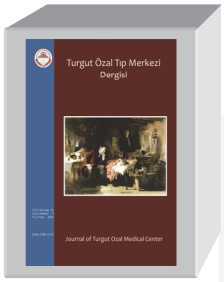

EDİTÖRE MEKTUP/LETTER TO THE EDITOR
J Turgut Ozal Med Cent 2014;21(3):245-6

Journal Of Turgut Ozal Medical Center
www.jtomc.org

Convulsion Due to High Dose Lidocaine Administration

Yüksek Doz Lidokaine Bağlı Konvülziyon

Yusuf Ziya Çolak

Kangal State Hospital, Department of Anaestesiology and Reanimation, Sivas, Turkey

Dear Editor,

General anaesthesia requires the successive administration of a number of drugs, each of which have many side effects and are classified as high-risk drugs by hospital pharmacists, by experienced staff only. One of these drugs, lidocaine, is often used in suppressing pressor responses during intubation due to its antiarrhythmic and local anaesthetic properties. However, there are many publications covering systemic toxic reactions, particularly central nervous system (CNS) toxicity, that develop following lidocaine application (1). In this paper, we intend to present a CNS toxicity case, which we believe, developed after lidocaine administration during the induction of anaesthesia.

A 21 year-old male patient (weight: 65 kg; height: 174 cm) was taken to the operating room as an elective, urgent case by the orthopaedic clinic for an incision in his right hand. His medical history and physical examination did not show any abnormalities apart from drinking alcohol and smoking. The laboratory test results were Hb 12.2 g/dL, hematocrit: 35.6%, and Plt: 162,000/mm3. Assuming that the operation would take a long time, the general anaesthesia procedure was started for the patient. During the operation, we monitored the heart rate (HR), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), peripheral oxygen saturation (SpO2), and peripheral body temperature (fever). We started to infuse 0.9% NaCl through the already existing intravenous catheter (iv). At this point, we noted the following data: SAB: 130 mmHg, DBP 75 mmHg, HR: 88 beats per min-1, SpO2 98%, and body temperature: 36.2°C. The induction of the general anaesthesia started with lidocaine. Following the application of 5 cc lidocaine iv bolus, the patient told us that he did not feel alright and that he had dizziness and nausea. Meanwhile, the patient lost consciousness and started to have a generalised tonic-clonic seizure. Upon this, we applied 100 mg sodium thiopental iv bolus. The patient's seizure soon terminated: SAB: 150 mmHg, DBP: 95 mmHg, HR: 114 beats per min-1, SpO2: 94%, and body temperature: 36.4°C. We added thiopental sodium up to 325 mg so as to be 5 mg/kg. We applied 100 mcg of fentanyl and 40 mg of rocuronium. Following the air-mask ventilation of 80% O_2 and 20% air and having achieved completion muscle relaxation, we intubated the patient with a number 7,5 ETT. Maintenance of anaesthesia was achieved by a mixture of sevoflurane 2.5%, N_2O 50%, and O_2 50%. Examining the lidocaine bulbs that had been used at the beginning of the anaesthesia, it was noticed that they were 10% lidocaine bulbs and the patients had actually undergone a total of 500 mg of lidocaine iv. The rest of the anaesthesia and surgery were uneventful. After a 3 hour-and-45-minute surgical operation, we applied 30mg of tramadol and 1g of paracetamol iv infusion for postoperative analgesia. After the anaesthesia, the patient awakened without any problems. Since the vital signs and neurological examination findings were normal in the recovery room, the patient was sent to the service. After the neurology consultation, we applied EEG and MRI for the brain and then checked the values of routine biochemistry tests, complete blood count, TSH, free T3, free T4, magnesium and calcium levels, all which were within normal ranges. Because the patients had not being using any medication prior to the anaesthesia induction, we concluded that the patient's condition during the operation was because of lidocaine. Having no other loss of consciousness, seizures, or medical issues, the patient was discharged within 48 hours in a healthy state. Systemic side effects are common when high doses of local anaesthetics are applied intravenously or when they are absorbed in high doses. The absorption rate depends on tissue vascularization, site of administration, the volume and concentration of the local anaesthetics, addition of vasoconstrictors, and concomitant diseases (2,3). Systemic effects of local anaesthetics are usually seen in cardiovascular systems (CVS) and central nervous systems (CNS). Passing the blood brain barrier easily, local anaesthetics cause medullary depression. Initially, they start with numbness around the mouth and tongue, dizziness, sedation, disorientation, visual and hearing impairment (nystagmus, tinnitus), nausea, and vomiting. These are followed by restlessness, nervousness, tremors, and muscle twitching. Eventually, convulsions, loss of consciousness, apnea, coma, and cardiovascular collapse take place (2). The treatment of CNS toxicity includes ensuring adequate ventilation and oxygenation and controlling convulsions by thiopental sodium or benzodiazepine administration (4).

A large number of researches on the effects lidocaine have reported that lidocaine, which is said to be more toxic on CNS, may therefore cause CNS toxicity while it does not effect CVS (5,6). In the case at hand, we only experienced CNS toxicity and no CVS issues. As a matter of fact, we even observed increase in tension arterial and HR values. This increase was attributed to the seizure activity. In their experimental study on the application of lidocaine iv, Feldman et al. (7) have demonstrated that CVS is seven times more resistant to lidocaine induced toxicity than CNS. There are, however, publications that claim that no side effects had been observed despite high doses of lidocaine, while some studies report smaller amounts of CNS toxicity (7,8).

In this patient, immediately after his description of nausea, the patient lost consciousness and had a generalised tonic-clonic seizure. The intrusion of high doses of lidocaine iv into CNS may have quickly lead to loss of consciousness and convulsions before we were able to observe the initial signs of anaesthesia. At the end, I would like to remind anaesthesiologists, who work with high-risk drugs, that they may not always encounter normal toxicity conditions.

REFERENCES

1. Chiang YY, Tsengk KF, Lih YW, Tsai TC, Liu CT, Leung HK. Lidocaine induced CNS toxicity-a case report. Acta Anesthesiol Sin 1996;34:243-6.
2. Covino BG, Wildsmith JAV. Clinical pharmacology of local anesthetic agents. In: Cousins MJ, Bridenbaugh OP,eds. Neural Blockade, 2 nd edition. Philadelphia: Lippincott Raven; 1998. p.97-128.
3. Faccenda KA, Finucane BT. Complications of regional anaesthesia incidence and prevention. Drug Saf 2001;24:413-42.
4. Naguib M, Magboul MM, Samarkandi AH, Attia M. Adverse effects and drug interactions associated with local and regional anaesthesia. Drug Saf 1998;8:221-50.
5. Reynolds F. Adverse effects of local anaesthetics. Br J Anaesth 1987;59:78-95.
6. Rutten AJ, Noncarrow C. Hemodynamic and central nervous system effects of intravenous bolus doses of lidocaine, bupivacaine and ropivacaine in sheep. Anesth Analg 1998;69:291-9.
7. Feldman HS, Arthur GR, Covino BG. Comparative systemic toxicity of convulsant and supraconvulsant doses of intravenous ropivacaine, bupivacaine and lidocaine in the conscious dog. Anesth Analg 1989;69:794-6.
8. Berde CB, Stricharte GR. Local anesthetics. In: Miller RD,ed. Anesthesia. 5 th edition. Philadelphia: Churchill Livingstone Inc; 2000. p. 491-521.

Received/Başvuru: 10.02.2014, Accepted/Kabul: 10.03.2014

Correspondence/İletişim

Yusuf Ziya ÇOLAK
Kangal State Hospital, Department of Anaestesiology and Reanimation, SİVAS, TURKEY
E-mail: yzcolak@gmail.com

For citing/Atıf için

Colak YZ. Convulsion due to high dose lidocaine administration. J Turgut Ozal Med Cent 2014;21:245-6
DOI: 10.7247/jtomc.2014.1779