CASE REPORT

Tonic clonic convulsions induced by low dose intrathecal bupivacaine and intraurethral lidocaine gel

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ABSTRACT

Local anesthetics are commonly used as analgesic and anesthetics. The systemic toxicity of local anesthetics involves CNS and cardiovascular system. Grand mal seizure, as a critical event may occur during toxicity due to high doses, rapid entry of local anesthetic into the systemic circulation and erroneous injection of local anesthetics through the intravenous route. We would like to present a case that experienced tonic clonic convulsion in spite of administration of low dose intrathecal bupivacaine and intraurethral lidocaine gel.

Key words: Local anesthetic; Tonic clonic seizure; Bupivacaine; Lidocaine

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INTRODUCTION

Local anesthetics may result in systemic toxicity that involves central nervous or cardiovascular system. The causes of toxicity are multifactorial. There are some recognized risks including drug doses, patient factors, choice of anesthetic technique and pharmacokinetics.[1] Local anesthetic systemic toxicity generally begins with central nervous system excitation followed by inhibition. At the same time, it causes dysrhythmias and reduced systemic vascular resistance.[1] Grand mal seizure can be triggered by drugs used in spinal or epidural anesthesia or marked overdose injection due to inadvertent intravascular absorption.[2] There is no report of tonic clonic seizure following intrathecal administration of low dose of bupivacaine. We present a patient who developed a generalized tonic clonic seizure after intrathecal administration of low dose of bupivacaine and intraurethral usage of lidocaine2% gel simultaneously.

CASE REPORT

A 41-year old male (75 kg, 172 cm, ASA class 1) was scheduled to undergo a planned transurethral ureterolithotripsy (TUL) for treatment of urolithiasis. He had no medical problems except a history of asthma. The preoperative hemogram and biochemistry data were within normal limits. Since our routine protocol for TUL surgery involves spinal anesthesia using low dose of bupivacaine 0.5%, this protocol was performed under standard monitoring. Intrathecal bupivacaine 0.5% (10 mg Hyperbaric Marcaine™) without any adjuvant was administered in space L4-L5 via quincke type spinal needle 27G. A Cathejell with lidocaine 2% (each contains 200 mg lidocaine) was applied through urethra for easy insertion of endourethral devices. Sufficient sensory block was achieved. The urologic procedure was completed uneventfully. The patient was hemodynamically stable throughout the surgery. Approximately 40 min after administration

of intrathecal bupivacaine and lidocaine gel, suddenly the patient began to experience tonic clonic seizure. There were no initial symptoms and signs like dizziness, visual or auditory disturbances, tinnitus, disorientation.[3] The heart rate dropped from 89 to 45 bpm rapidly. There was no significant alteration in blood pressure or SpO_2. Prolonged PR interval was detected on the electrocardiogram (ECG) without any changes in QRS or ST intervals. The patient was controlled by administration of diazepam 10 mg and midazolam 5 mg. Oxygen was delivered via mask and atropine 1 mg administered IV. The patient was not intubated because he was not full stomach, the seizure not repeated again and there was no respiratory arrest. Blood sample was immediately sent for calcium and blood glucose. He was prescribed a loading dose of phenytoin in operating room. The patient was admitted in ICU and maintenance dose of phenytoin started. The neurologic consultation, electroencephalography (EEG) and brain computed tomography (CT) were obtained. Serum calcium, glucose, brain CT and follow up EEGs were normal. He was re-examined next day and no abnormal neurologic findings were seen. The patient was discharged and followed up for two months. There was no seizure during this period. Meanwhile, our patient signed the consent form relating to this publication.

DISCUSSION

The frequency of severe neurological complications using spinal local anesthetics has been found to be approximately 0.4/10000 by Moen etal.[2] The causes of the neurologic complications induced by local anesthetics are high doses administration, diffusion of the drug towards the cephalic region and rapid absorption into the blood circulation.

G. F. Rousseau found that plasma lidocaine concentration after the instillation of lidocaine gel 220 mg into the uterine cavity for uterine balloon thermal ablation was 272 ng / ml at 15 min after insertion, that was well below the level to cause local anesthetic toxicity.[4]

Based on Brosh-Nissimov study, toxic levels of lidocaine were seen in patients following administration of lidocaine gel or topical lidocaine for painful oral lesions or bronchoscopy respectively.[3] Literature documents that administration of lidocaine and bupivacaine simultaneously in rats results in systemic toxicity additively.[5] Probably this was the cause of convulsions in our patient. In our case, a low dose of intrathecal bupivacaine (10 mg) and intraurethral lidocaine gel (200 mg) were used simultaneously. Considering the time of convulsion, no pervious history of seizure, normal lab data and normal EEG, we concluded that systemic toxicity of local anesthetics was responsible for this tonic clonic convulsion as two different local anesthetics had additive or synergistic effects for reducing convulsive threshold.

Drawing blood sample to measure plasma concentration of local anesthetics was not considered since low doses of local anesthetics were administered. There is one study that has reported a case of normal arterial plasma concentration of ropivacaine causing grand mal seizure after epidural injection.[6]

In conclusion, regarding additive and synergistic effects of drugs, the use of several types of local anesthetics simultaneously should not be assumed completely harmless. Clinicians should consider the diagnosis of CNS toxicity when dizziness, mental obtundation, acute onset of confusion and seizures occur following administration of any different kinds of local anesthetics. It must also be kept in mind that usage of low dose of intrathecal bupivacaine with other types of local anesthetics administered peripherally may result in tonic clonic seizure. This case serves as a reminder of a possible systemic toxicity of local anesthetics used in urological surgery

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Authors' contribution: All author managed the case and took part in the preparation of the manuscript

convulsion with low dose local anesthetics

REFERENCES

1. El-Boghdadly K, Chin KJ. Local anesthetic systemic toxicity: Continuing Professional Development. Can J Anaesth. 2016 Mar;63(3):330-49. doi: 10.1007/s12630-015-0564-z. [PubMed]
2. Akil E, Varol S, Guzel A, Gocmez C. Status epilepticus induced by intrathecal bupivacaine use: A case report. J Clin Exp Invest. 2014;5(1):108-111. doi: 10.5799/ahinjs.01.2014.01.0371 [Free full text]
3. Brosh-Nissimov T, Ingbir M, Weintal I, Fried M, Porat R. Central nervous system toxicity following topical skin application of lidocaine. Eur J Clin Pharmacol. 2004 Nov;60(9):683-4. [PubMed]
4. Rousseau G, Oram M, Barrington J, Priston M, Swart M. Plasma lidocaine concentrations following insertion of 2% lidocaine gel into the uterine cavity after uterine balloon thermal ablation. Br J Anaesth. 2002 Dec;89(6):846-8. [PubMed] [Free full text]
5. Mets B, Janicki PK, James MF, Erskine R, Sasman B. Lidocaine and Bupivacaine Cardiorespiratory Toxicity Is Additive: A Study in Rats. Anesth Analg. 1992 Oct;75(4):611-4. [PubMed]
6. Iwama H. A case of normal ropivacaine concentration causing grand mal seizure after epidural injection. Eur J Anaesthesiol. 2005 Apr;22(4):322-3. [PubMed]

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My Most Memorable Patient

The patient with an absent gall bladder

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It was 1993 that we started Laparoscopic cholecystectomies at KRL hospital Islamabad. The patients showed great enthusiasm to accept this new modality of treatment. Among them was a beautician running a beauty salon in a nearby residential sector. She had had a long history of biliary symptoms; had been advised by doctors to have her gall bladder (GB) removed, but was reluctant to have it done due to expected big abdominal scar it would leave.

An upper abdominal ultrasound report stated '*contracted GB with no stones*'. The radiologist advised a repeat ultrasound after proper fasting the following week. Repeat report showed '*contracted GB with no stones*'. Hence an oral cholecystogram was requested, the report of which showed '*non-functioning GB*'.

After explaining the pros & cons of the surgery she agreed to have a lap chole and was put for surgery on the next available operating list. After abdominal insufflation with CO_2 we tried to locate the GB. We struggled for approximately half an hour with blunt dissection of whatever we thought could be a GB but failed to identify the structure positively. Ultimately we had to proceed with open cholecystectomy. During this time our anesthetist casually passed a remark that it may be 'a case of congenital absence of GB'. It drew my attention to the fact that it might well be the case and I started to think on those lines.

When we opened the abdomen I found to my horror that the structure we were dissecting for a gall bladder was actually the *porta hepatis*. Luckily we didn't cut or damage anything in that area. We started to look for the possible GB sites in the abdomen for a case of congenital absence of GB but failed to identify any such structure. Therefore, we closed the abdomen.

Her postop recovery was quite smooth and uneventful, but she was very disappointed to see the big cut in her abdomen. For some reason her symptoms of biliary colic resolved and when I last saw her a few years later, she was asymptomatic. The only question she asked me and our female theatre staff whenever they went to her salon for her services was, "If my gall bladder was absent from birth why did you have to open my tummy". I am still looking for an answer to her question.