

An Accidental Intrathecal Injection of Tranexamic Acid: A Never Miss Event

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Abstract

Several factors have been identified as contributing to medical errors such as labels, appearance, and location of ampoules. In this paper, inadvertent intrathecal injection of tranexamic acid has resulted from similarities in appearance between tranexamic acid and heavy bupivacaine 0.5% ampoules. The patient experienced severe itching in the gluteal region followed by generalised myoclonic seizures after accidental intrathecal administration of tranexamic acid.

Keywords: Tranexamic Acid, Intrathecal, Seizures

Introduction

Tranexamic acid is an antifibrinolytic that is used in a variety of hemorrhagic conditions to control bleeding, including post-partum hemorrhage. It works by preventing the breakdown of fibrin, thus promoting clotting. When given intrathecally instead of a local anesthetic, tranexamic acid Injection is a potent neurotoxin with a mortality rate of around 50%. Survivors of intraspinal tranexamic acid often experience seizures, permanent neurological Injury, and paraplegia [1]. There are several reports of serious medication errors due to mix-ups between tranexamic acid and bupivacaine during regional anesthesia.

Case Description

We report a case of a forty- five years old women, ASA 1, who was posted for correction of bilateral Haglund deformity. She had no previous history of convulsions or seizures. Her preoperative vitals and investigations were within normal limits. Spinal anesthesia was performed with the patient in the sitting position at the L4-5 interspace using a 25-gauge Quincke's spinal needle. Injection Bupivacaine 0.5% (10 mg) 2 mL was Injected intrathecally. Immediately after drug administration, when she was placed in the supine position, she complained of severe itching in her gluteal region and she was irritable. Her arterial BP raised to 160/100 mmHg, and HR to 120/ min. Inje Avil 45 mg iv, Inje dexamethasone 8 mg iv and Inje hydrocortisone 200 mg iv were given in suspicion of allergic reaction. The patient then developed myoclonic seizures. Inje midazolam 1 mg iv given and the procedure was converted to general anaesthesia with Inje fentanyl 100 mcg iv, Inje propofol 100 mg iv, Inje vecuronium 6 mg iv. The airway was secured with endotracheal tube. The depth was maintained with O₂, N₂O and isoflurane. She was put on controlled mechanical ventilation and her vital parameters were within normal limits throughout the surgery. Accidental intrathecal Injection of the wrong drug was suspected and a used ampoule of tranexamic acid was found in the trash can.

Reversal of neuromuscular blockade was done with Inje neostigmine 3 mg iv and Inje glycopyrrolate 0.6 mg iv. This time again the patient started having myoclonic seizures and irregular breathing pattern. She was observed for sometimes and it was decided to keep her on mechanical ventilation in ICU for the time till she regains consciousness with adequate breathing patterns. A CT scan was done which showed only mild cerebral edema.

Neurological consultation was done and Inje sodium valproate 100 mg 12-hourly, Inje dexamethasone 8 mg every 8 hourly advised. On 3rd day in ICU, after her good respiratory efforts and consciousness, she was extubated. She was kept for another 24 hrs for observation and then shifted to the ward.

Discussion

Many factors have been identified as contributing to medication errors which include labels, appearance and location of ampoules, syringes, inattention, poor communication, carelessness and fatigue on the part of the anesthesiologist [2]. In this case, drug error-induced polymyoclonus occurred due to Injection of tranexamic acid for spinal anesthesia, as two different ampoules had a similar appearance. This illustrates the importance of double-checking of drug ampoules to reduce such errors.

A very less known information is available regarding the effect of direct intrathecal administration of tranexamic acid in humans. Wong *et al* reported the first case of inadvertent intrathecal Injection of 75 mg tranexamic acid in an 18-year-old man scheduled for appendectomy [3]. He developed clonic convulsions that progressed to a generalized seizure, which was treated with iv diazepam, and the patient recovered without any sequelae. De leede *et al* have reported a case of a 68-year-old man who accidentally received an intrathecal Injection of 50 mg tranexamic acid. Immediately after the Injection, he developed status epilepticus, eventually complicated with hypotonic paresis of all four limbs, which resolved but resulted in residual bilateral peroneal palsy [4]. Yeh *et al.* reported that seizures and refractory ventricular fibrillation after accidental intrathecal administration of 500 mg tranexamic acid were associated with fatal outcome [5]. The exact mechanism by which tranexamic acid induces seizures or ventricular fibrillation is unknown. However, there are reports of neurotoxicity in animal studies, i.e., when applied topically to the cerebral cortex in animals, this drug

is known to produce seizures [6]. Myoclonus could arise from cerebral cortex, brain stem, spinal cord, peripheral nerve and spinal roots. Some authors have linked it to cerebellar dysfunction. In an experimental study, the drug caused intracranial and systemic hypertension and epilepsies. Very high doses of drugs would cause massive sympathetic discharge, as evidenced by initial hypertensive response and subsequent ventricular arrhythmias reported in some patients [5].

In our case, the patient reported severe itching and myoclonus developed immediately after the intrathecal Injection of 200 mg tranexamic acid, which was quite a large dose compared with those in former case reports. The explanation for severe itching could be irritation of nerve roots caused by inadvertent or expired drug administration [6]. In our case, these factors were ruled as the ampoule was rechecked for expiry and wrong drug.

Tranexamic acid -induced seizures result either from direct cerebral ischemia secondary to decrease in the cerebral blood flow or blockage of inhibitory cortical-amino butyric acid (GABA)-A receptors. Because GABA-A receptors govern opening of chloride channels resulting in neuronal hyperpolarization and reduced excitability, blockage by tranexamic acid results in lowering of depolarization threshold and enhanced excitotoxicity [7,8]. anticonvulsants like phenytoin sometimes does not help much in such polyclonus, therefore, sodium valproate is suggested [2].

Fatal catastrophies after incidental intrathecal Injection of penicillin, gallamine and vincristine have been treated with anticonvulsant and spinal fluid lavage [8].

We speculate that treatment of tranexamic acid induced convulsions should include administration of anticonvulsants, intensive haemodynamic monitoring and, possibly, CSF lavage. The benefits of CSF lavage are that they remove and dilute the Injected drug thus limiting the possibility of neurological damage [9].

In most of the reports [2,3]. general anesthesia was given after spinal anesthesia as there was no sensory and motor blockade with spinal anesthesia. In these cases, convulsions appeared after reversal of muscle relaxants. Therefore, intrathecal drug remained in the CSF for 2–3 hrs and treatment was started later.

To conclude, this case report alarms the anesthesiologist about the possibility of having myoclonus following intrathecal bupivacaine administration, for which he may be blamed for either faulty technique or inadvertent drug administration. Therefore, double checking of the drug ampoule before giving subarachnoid block can avoid such complications.

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