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Case Report

Accidental Intrathecal Injection of Tranexamic Acid during Emergency Cesarean Section: A Case Report

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Abstract: ***Introduction*:** Accidental intrathecal injection of tranexamic acid (TXA) is a rare but potentially fatal medication error responsible for severe neurotoxicity. It most often results from drug ampoule confusion during spinal anesthesia. ***Case Presentation*:** We report the case of a 40-year-old ASA II woman admitted for semi-urgent cesarean section under spinal anesthesia. Due to a medication error, 150 mg of tranexamic acid was inadvertently injected intrathecally after confusion with hyperbaric bupivacaine. General anesthesia was immediately induced to allow fetal extraction. Approximately forty-five minutes after the accidental injection, the patient developed generalized tonic–clonic seizures refractory to midazolam. Management consisted of a combination of thiopental, midazolam, a volatile anesthetic agent, and cerebrospinal fluid lavage–drainage. The clinical outcome was favorable, with a normal neurological examination one month later. ***Conclusion*:** This case highlights the severity of accidental intrathecal tranexamic acid injection and emphasizes the need for strict preventive measures to avoid medication errors in anesthesia.

Keywords: Tranexamic Acid, Intrathecal, Medication Error, Caesarean Section, Anesthesia.

INTRODUCTION

Medication-related adverse events are preventable incidents resulting from drug interactions or errors occurring at any stage of the medication-use process, including prescription, dispensing, administration, and monitoring [1, 2]. Although the operating room is generally considered a highly controlled and safe environment, it remains a setting where medication errors may occur. These incidents may result either from adverse reactions to correctly administered drugs or from catastrophic complications related to administration via an incorrect route. Accidental intrathecal administration of tranexamic acid, a drug strictly contraindicated for neuraxial use, represents one of the most severe forms of medication error. This complication is rare but potentially fatal and continues to be reported worldwide [3]. We describe the case of a 40-year-old woman who experienced accidental intrathecal injection of tranexamic acid during caesarean section. We analyze the contributing factors, therapeutic management, clinical course, and preventive strategies.

CASE PRESENTATION

A 40-year-old multiparous woman with no significant medical history was admitted to the operating room for cesarean section indicated for prolonged pregnancy with an unfavorable Bishop score. Preoperative assessment revealed normal neurological, hemodynamic, and respiratory findings. Obstetric examination showed active fetal movements, uterine contractions, and normal fetal heart rate monitoring. Laboratory tests revealed anemia with hemoglobin level of 9.9 g/dL, without other abnormalities. The patient was classified as ASA II and deemed suitable for spinal anesthesia, antibiotic prophylaxis, and intravenous administration of tranexamic acid. Upon arrival in the operating room, vital signs were stable. The anesthesia tray contained hyperbaric bupivacaine, fentanyl, tranexamic acid, cefuroxime, and emergency drugs (ephedrine, atropine, adrenaline). Separate trays contained hypnotics, opioids, and neuromuscular blocking agents for general anesthesia. After aseptic preparation, the anesthetist requested fentanyl and hyperbaric bupivacaine. Due to ampoule confusion, fentanyl and tranexamic acid were handed over. The

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anesthetist drew 0.5 mL of fentanyl and 1.5 mL of tranexamic acid, corresponding to 25 µg of fentanyl and 150 mg of tranexamic acid. Lumbar puncture was performed at the L4–L5 interspace, and the mixture was injected into the subarachnoid space. Shortly after injection, the patient complained of severe perineal and anal pain, initially attributed to uterine contractions. While preparing intravenous tranexamic acid, the anesthetist noticed that the tranexamic acid ampoule had already been opened and partially used, whereas the bupivacaine ampoule remained unopened. The error was immediately recognized. General anesthesia was promptly induced, and emergency fetal extraction was performed. A male neonate was delivered approximately one minute after surgical incision, with an Apgar score of 8 at one minute. Anesthesia was maintained with fentanyl, atracurium, and sevoflurane at 1.2% MAC. Intraoperative hemodynamic parameters remained stable, and no clinically apparent seizures were observed. At the end of surgery, sevoflurane was discontinued, and the patient remained intubated and was transferred to the intensive care unit. Mechanical ventilation was initiated with continuous sedation using fentanyl and midazolam. Approximately ten minutes after ICU admission (45 minutes after the accidental intrathecal injection) the patient developed generalized tonic–clonic seizures despite ongoing sedation and two boluses of 10 mg midazolam. Seizures ceased after administration of thiopental (5 mg/kg bolus followed by continuous infusion at 4 mg/kg/h) combined with continuous midazolam. However, despite high doses of both agents, the patient remained awake with a Ramsay score of 1/6. An anesthesia ventilator was used to deliver sevoflurane in the ICU in combination with intravenous agents. Electrocardiography showed no arrhythmias. The patient was then positioned in lateral decubitus and underwent cerebrospinal fluid lavage–drainage. An epidural catheter was inserted into the subarachnoid space at the L3–L4 level, while a spinal needle was introduced at L5–S1 for passive CSF drainage. Isotonic saline was infused through the catheter using a syringe pump, with a total volume of 50 mL administered over one hour. Vital signs were continuously monitored throughout the procedure. After one hour, both devices were removed, and the patient was returned to the supine position. Sevoflurane was continued for six hours, then discontinued. Mechanical ventilation and intravenous sedation were maintained for 24 hours. On day two, sedation was stopped. Three hours later, the patient gradually regained consciousness, demonstrated effective spontaneous breathing, and remained hemodynamically stable. She was extubated and placed on supplemental oxygen with continuous neurological monitoring. The patient reported heaviness in both lower limbs without sensory deficits, moderate headache (5/10), and surgical site pain. No abnormal movements were observed. On day three, assisted ambulation was resumed without recurrence of seizures. On day four, neurological, hemodynamic, and respiratory examinations were normal, and the patient was transferred to the maternity ward. On day six, she was ambulating independently with no motor deficits. Neurological examination remained normal, and she was discharged home. Follow-up at one month revealed no neurological abnormalities.

DISCUSSION

Tranexamic acid is a synthetic antifibrinolytic agent, structurally analogous to lysine, which exerts its effect by inhibiting the binding of plasminogen and plasmin to fibrin, thereby stabilizing the clot and reducing bleeding [4]. It is widely used in surgical practice for the prevention and treatment of hemorrhage. However, tranexamic acid is strictly contraindicated for intrathecal or epidural administration because of the major risk of severe neurotoxicity [5]. The neurotoxic effects of tranexamic acid are mainly related to antagonism of inhibitory glycinergic and GABA-A receptors at both spinal and supraspinal levels. This antagonism leads to marked neuronal hyperexcitability, which clinically manifests as early and often refractory seizures, myoclonus, muscle rigidity, and severe agitation [6]. When tranexamic acid is injected into the subarachnoid space, even small volumes result in supratoxic concentrations within the cerebrospinal fluid. This is explained by the direct exposure of neural tissue to the drug and the absence of initial metabolic degradation, leading to sustained high local concentrations [7]. The resulting central nervous system hyperexcitability can trigger massive catecholamine release, which contributes to the development of associated cardiovascular complications, including arrhythmias and hypertension [5]. Despite the extreme severity of this adverse event, accidental intrathecal administration of tranexamic acid continues to be reported worldwide. Between 1988 and 2018, twenty-one cases were documented in the literature [3]. Although this event is considered rare, its recurrence highlights persistent vulnerabilities in medication safety systems. One of the principal causes identified is the visual similarity between tranexamic acid and local anesthetic ampoules, particularly bupivacaine. Another critical contributing factor is the inappropriate presence of tranexamic acid on the spinal anesthesia tray, which unnecessarily increases the risk of drug confusion. In addition, human factors such as fatigue, time pressure, cognitive overload, and reduced vigilance play a significant role. In the present case, the absence of loud verbal confirmation of the drug name during preparation and administration was an additional contributory factor. Several preventive strategies have been proposed to reduce the risk of such catastrophic errors. First, tranexamic acid should be strictly excluded from spinal and epidural anesthesia trays. Second, a mandatory double-checking process should be implemented before any intrathecal injection, involving two qualified healthcare professionals and loud verbal confirmation of the drug name and concentration. Clear labeling and standardized organization of anesthesia workspaces are also essential components of effective prevention strategies. In the present case, generalized tonic–clonic

seizures occurred approximately forty-five minutes after accidental intrathecal injection. This delay is notably longer than that reported by Harby, who described seizure onset approximately two minutes after injection [8]. This difference may be explained by two main factors. First, the dose administered in our patient (150 mg) was significantly lower than the 400 mg reported in the case by Harby [8]. Second, general anesthesia was instituted shortly after recognition of the error to allow urgent fetal extraction, prior to the onset of seizures. During general anesthesia, the patient received hypnotic agents, including propofol, as well as the volatile anesthetic sevoflurane. Both agents are well known for their potentiating effects on GABAergic neurotransmission [9 ; 10]. Their pharmacological action may have transiently counteracted the antagonistic effects of tranexamic acid on inhibitory receptors, thereby delaying the clinical expression of neurotoxicity. In addition, atracurium, by blocking neuromuscular transmission, may have masked overt convulsive activity during the intraoperative period [11]. Once the effects of these anesthetic agents diminished, generalized seizures became clinically apparent. The seizures observed in our patient were refractory to repeated boluses of midazolam, which is consistent with findings reported in other similar cases [8 ; 12]. The limited efficacy of midazolam can be explained by its mechanism of action. Benzodiazepines do not directly activate GABA-A receptors; rather, they enhance the effect of endogenous GABA by increasing receptor affinity. When GABA-A receptors are antagonized by tranexamic acid, the pharmacological effect of benzodiazepines is therefore significantly reduced [13]. Seizure control was achieved after the addition of thiopental, a barbiturate with a distinct mechanism of action. Thiopental binds to a different site on the GABA-A receptor than benzodiazepines and, importantly, is capable of directly opening the chloride channel even in the absence of GABA [14 ; 15]. This results in a marked influx of chloride ions into neurons, leading to neuronal hyperpolarization and suppression of seizure activity. Despite this combined therapy, the patient remained awake with a Ramsay sedation score of 1/6. This paradoxical clinical state may be explained by disinhibition of excitatory neuronal pathways, particularly glutamatergic circuits, resulting in persistent imbalance between inhibitory and excitatory neurotransmission [6]. The addition of a volatile anesthetic agent was therefore required to achieve adequate hypnosis and allow effective mechanical ventilation. Volatile anesthetics exert their central nervous system depressant effects through multiple mechanisms, including opening of potassium channels, leading to neuronal hyperpolarization, and inhibition of calcium channels, which reduces synaptic excitability. These effects contribute to global central nervous system depression even in the presence of partial GABA-A receptor blockade [16]. Cerebrospinal fluid lavage represents an emergency therapeutic strategy described after accidental intrathecal injection of tranexamic acid [8-17]. The rationale of this approach is to dilute and eliminate the drug from the subarachnoid space as rapidly as possible, thereby reducing local neurotoxic concentrations. This reduction in exposure may decrease seizure severity and potentially improve neurological outcomes [17]. However, considerable variability exists among reported techniques, particularly regarding the volume of cerebrospinal fluid exchanged and the duration of the procedure. Some authors have described continuous lavage using external ventricular drainage combined with lumbar drainage [17], while others have reported lavage using two lumbar needles inserted below the L3 level, with intermittent infusion and passive drainage [8]. These techniques are not without risk, as they may increase the likelihood of infection and induce potentially harmful fluctuations in intracranial pressure. Given these limitations, prevention remains the most effective and reliable strategy to reduce morbidity and mortality associated with accidental intrathecal tranexamic acid injection. Simple, low-cost preventive measures—including strict separation of medications, clear labeling, standardized anesthesia trays, mandatory double-checking, and systematic visual and verbal verification before any neuraxial injection—are essential and should be actively promoted.

CONCLUSION

Accidental intrathecal injection of tranexamic acid is a rare but potentially fatal medication error. It is responsible for severe neurotoxicity and refractory seizures. This case illustrates the importance of early, multimodal treatment combining intravenous hypnotics and volatile anesthetic agents. Cerebrospinal fluid lavage–drainage may be considered, although techniques remain poorly standardized. Prevention remains the most effective strategy and relies on optimal organization, systematic double-checking, and strict visual and verbal verification before any neuraxial drug administration.

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