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Neurotoxicity and Status Epilepticus After Inadvertent Intrathecal Tranexamic Acid: A Case Report

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Abstract

Inadvertent intrathecal administration of tranexamic acid (TXA) is a rare but potentially fatal medication error that causes acute neurotoxicity, thought to be mediated by antagonism of gamma-aminobutyric acid type A (GABA-A) and glycine receptors. No treatment guidelines specific to intrathecal TXA toxicity exist. A man in his early 30s received an estimated 280 mg of TXA intrathecally instead of bupivacaine during spinal anaesthesia for ankle fracture fixation because of ampoule misidentification. No block developed; he reported severe back and gluteal pain and then developed myoclonus and generalised tonic-clonic convulsions that did not respond to midazolam. General anaesthesia was induced, phenytoin and levetiracetam were loaded, and fracture fixation was completed. Recurrent convulsions at the end of surgery, consistent with convulsive status epilepticus, were treated with diazepam, propofol and magnesium sulphate, after which no further convulsive activity was observed; because neuromuscular blockade had been given and electroencephalography (EEG) was unavailable, nonconvulsive seizures could not be excluded. Cerebrospinal fluid (CSF) lavage was then performed. Brain computed tomography on day one showed no acute abnormality. Consciousness remained impaired for several days: he was comatose on day three, still ventilated on day four, extubated and oriented by the morning of day five, and discharged home on day eight. A routine EEG at 11 weeks and brain magnetic resonance imaging at about three and a half months were normal, and at three months he had a normal neurological examination with no further clinically recognised seizures while taking levetiracetam. The contribution of individual interventions cannot be determined from a single case. The principal lesson is preventive: TXA must be stored and handled separately from neuraxial drugs, with independent pre-administration checks.

Categories: Neurology, Quality Improvement, Anesthesiology
Keywords: csf lavage, drug error, intrathecal neurotoxicity, magnesium sulphate, patient safety, status epilepticus, tranexamic acid, wrong-route error

Introduction

Tranexamic acid (TXA) is a synthetic antifibrinolytic agent widely used to reduce bleeding in surgery and trauma. While safe when administered intravenously or orally, accidental intrathecal administration is a rare but devastating complication. Neurotoxicity has been described across a range of inadvertently administered intrathecal doses, and no safe threshold has been established because the evidence consists of case reports. Reported features include severe back and gluteal pain, myoclonus, agitation, and generalised tonic-clonic seizures that may progress rapidly to status epilepticus [1-5].

TXA neurotoxicity is thought to relate to antagonism of inhibitory neurotransmitter receptors, particularly gamma-aminobutyric acid type A (GABA-A) and glycine receptors, resulting in neuronal hyperexcitability [4,6]. When given intravenously, TXA penetrates the central nervous system only to a limited extent, whereas intrathecal injection exposes the spinal cord and brain directly to high drug concentrations [4,6]. Fatal outcomes have been reported, particularly with refractory seizures and cardiac arrhythmias [1,2,5]. There are no established treatment guidelines specific to intrathecal TXA toxicity, and acute management in published reports varies considerably.

Magnesium sulphate, an N-methyl-D-aspartate (NMDA) receptor antagonist, has been proposed as an adjunct for refractory seizures and has been reported in a case of refractory status epilepticus after intrathecal TXA [7]. Cerebrospinal fluid (CSF) lavage, using different techniques, has been described as a means of reducing intrathecal drug concentration after neurotoxic drug error [3,8,9].

We report a patient who developed convulsive status epilepticus and prolonged impaired consciousness after inadvertent intrathecal TXA, was managed by a multidisciplinary team including anaesthesia, neurology, and intensive care, and made a clinical neurological recovery. The case illustrates two failed safety barriers (storage of look-alike ampoules in the same tray with a single check, and incision without verification of the spinal block), the decision to complete surgery after the error was recognised, and recovery after several days

of impaired consciousness.

Case Presentation

Patient and clinical background

A male patient in his early thirties with no significant past medical or neurological history was admitted for open reduction and internal fixation of a traumatic left ankle fracture. His weight was 70 kg. No relevant family or psychosocial history was identified. There were no prior neurological conditions or previous relevant interventions.

Medication error and initial presentation

During spinal anaesthesia, an estimated 280 mg of TXA was inadvertently injected intrathecally instead of the intended 12 mg of bupivacaine because of ampoule misidentification, a recognised mechanism for this error [2,10]. The drug was confirmed by checking the used vial after the error was recognised, and the injected dose was estimated by the anaesthesiologist. The TXA and bupivacaine ampoules were stored in the same tray in the operating theatre and were similar in size and label appearance (Figure *1*); the spinal drug was drawn up by the anaesthesiologist and checked only by his assistant (a single check). No sensory or motor block developed.

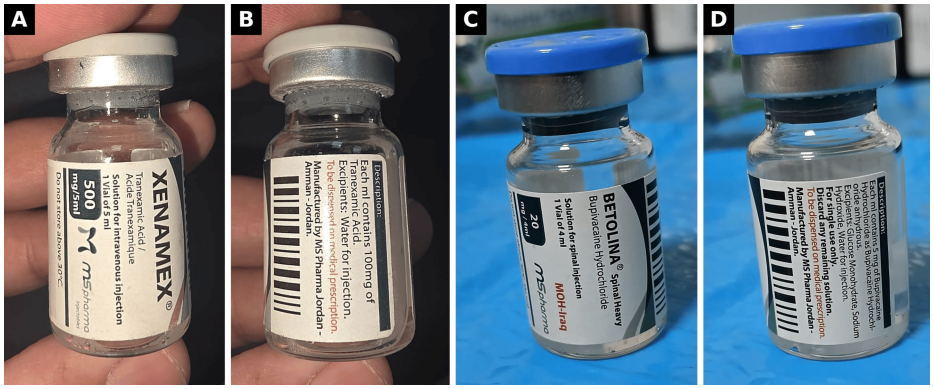


FIGURE 1: Vials of tranexamic acid and spinal bupivacaine stored in the same operating theatre tray

(A) Front and (B) back labels of the tranexamic acid vial (500 mg/5 mL, solution for intravenous injection). (C) Front and (D) back labels of the hyperbaric bupivacaine vial (20 mg/4 mL, solution for spinal injection). Both products come from the same manufacturer and have a similar vial size and shape, label layout and back-label format; the caps differ in colour (white versus blue). Photographs show vials of the same products, taken after the event.

Block adequacy was not assessed by formal sensory or motor testing before incision. The incision site had been infiltrated subcutaneously with levobupivacaine 50 mg in a total volume of 20 mL, and the team expected the spinal block to become effective as surgery began. This omitted an independent safety check that might have revealed the error before incision. After the skin incision, the patient reported severe back and gluteal pain, and the absence of any block raised suspicion of a medication error, which was confirmed by review of the used ampoules.

The patient became agitated, developed myoclonus, and progressed to generalised tonic-clonic seizures. Intravenous midazolam (5 mg) was administered immediately but did not terminate seizure activity. Surgery was halted. The skin incision had been made and the operative field was open, but internal fixation had not yet commenced.

Intraoperative decision-making

The operating surgeon, anaesthesiologist and neurologist discussed management at the operating table. General anaesthesia was induced for airway protection and seizure control; at that point the patient was haemodynamically and respiratorily stable. With the incision made and general anaesthesia established, the treating team judged that completing the fracture fixation was preferable to temporary closure and deferral of surgery. This was a judgement made in the circumstances; whether it was safer than deferral cannot be determined, and completion of fixation (approximately 45 minutes) preceded CSF lavage, which was performed after surgery.

This decision is reported as the treating team's judgement in this specific situation and is not offered as

guidance.

During the approximately 45-minute operative period, under general anaesthesia and neuromuscular blockade, no convulsive activity was observed while phenytoin and levetiracetam loading doses were given; because the patient was paralysed and electroencephalogram (EEG) monitoring was not available, nonconvulsive seizure activity during this period cannot be excluded. At the end of surgery, recurrent generalised tonic-clonic convulsions were observed. Table *1* summarises key events from error recognition to follow-up; intraoperative times are approximate and were reconstructed retrospectively from the clinical record and the treating team's recollection.

Time point	Event
Day 0 (02/2025), pre-operative	Patient admitted with traumatic left ankle fracture; spinal anaesthesia planned after patient discussion
T = 0 min	Spinal anaesthesia commenced; estimated 280 mg TXA inadvertently injected intrathecally instead of 12 mg bupivacaine (ampoule misidentification; drug confirmed from the used vial)
T = 0-3 min	No sensory or motor block develops
T = 3-5 min	Skin incision after subcutaneous infiltration of levobupivacaine 50 mg in a total volume of 20 mL, with onset of spinal block assumed
T = 5-8 min	Severe back and gluteal pain; absence of block raises suspicion of error; used ampoules reviewed and error confirmed
T = 8-10 min	Agitation, myoclonus, and generalised tonic-clonic convulsions
T = 10-12 min	Midazolam 5 mg IV; convulsions continue; surgery halted; discussion between surgeon, anaesthesiologist and neurologist
T = 15-20 min	General anaesthesia induced (propofol 100 mg, atracurium 40 mg); isoflurane maintenance; dexamethasone 16 mg IV; phenytoin 1,250 mg IV and levetiracetam 1,000 mg IV; fracture fixation proceeds (completed at approximately T = 65 min)
T = 65 min (end of surgery)	Recurrent generalised tonic-clonic convulsions; convulsive status epilepticus diagnosed clinically; diazepam (total 20 mg IV) and further propofol boluses (recorded total 400 mg before ICU); rocuronium 30 mg to facilitate ventilation; patient remained intubated
T = 65-70 min	Magnesium sulphate 4 g IV; no further convulsive activity observed (under neuromuscular blockade)
T = 90-180 min	CSF lavage (L2-L3 drainage, L4–L5 infusion; approximately 150 mL exchanged over 90 min)
Day 0, 15:30	ICU admission; mechanical ventilation with propofol infusion; phenytoin 100 mg IV 8-hourly; levetiracetam 1,000 mg IV 12-hourly; magnesium sulphate 4 g IV 12-hourly (days 0–1); dexamethasone 8 mg IV 12-hourly; methylprednisolone 250 mg IV daily; ceftriaxone, metronidazole and esomeprazole IV
Day 1	Comatose and ventilated; nasogastric feeding; fundoscopy: no papilloedema; non-contrast brain CT: no acute abnormality; enoxaparin 4,000 IU daily started
Day 2	Sedation stopped; GCS 5T; remains ventilated
Day 3	Unconscious, GCS 8 (components not recorded); phenytoin and methylprednisolone stopped; memantine, amantadine and vinpocetine started
Day 4	Unconscious, still mechanically ventilated; haemodynamically stable; no further clinically observed seizures
Day 5	Extubated by the morning of day five (exact timing not documented); conscious, alert and oriented on face-mask oxygen
Day 6	GCS 15; operated limb neurovascularly intact
Day 8	Discharged home on levetiracetam 1,000 mg twice daily, rivaroxaban 10 mg once daily and a tapering corticosteroid course over 11 days
~11 weeks	Routine EEG: normal
Three-month follow-up	Normal neurological examination; no further clinically recognised seizures on levetiracetam; levetiracetam taper begun; rivaroxaban discontinued

~3.5 months	Brain MRI: no structural abnormality identified (Figure 2)

TABLE 1: Timeline of clinical events

Intraoperative times are approximate and were reconstructed retrospectively from the clinical record and the treating team's recollection. TXA: tranexamic acid; IV: intravenous; CSF: cerebrospinal fluid; ICU: intensive care unit; CT: computed tomography; GCS: Glasgow Coma Scale (T, intubated); EEG: electroencephalogram; MRI: magnetic resonance imaging

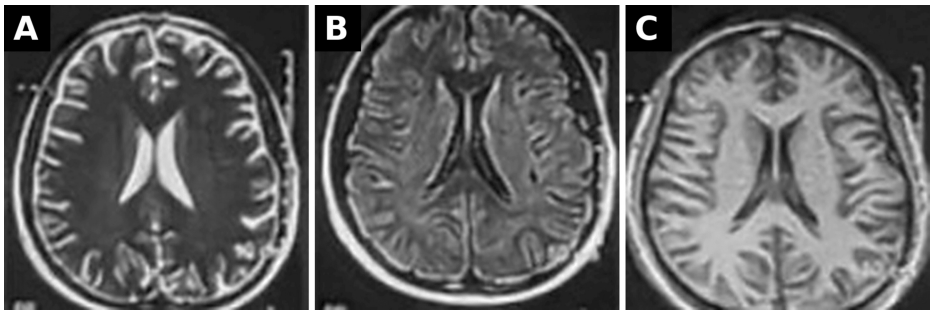


FIGURE 2: Axial brain MRI approximately three and a half months after the event, at the level of the bodies of the lateral ventricles

(A) T2-weighted image showing normal grey-white matter differentiation, normal-calibre lateral ventricles, and no cortical or subcortical signal abnormality. (B) Fluid-attenuated inversion recovery (FLAIR) image at the same level showing no parenchymal hyperintensity or oedema. (C) T1-weighted image at the same level showing no mass effect or midline shift. Representative images are shown; the remaining sequences were reported as normal. MRI: magnetic resonance imaging

Investigations

Laboratory and blood gas results are summarised in Table *2*. CSF obtained on day zero showed no pleocytosis (3 white cells/µL, no red cells), with normal glucose (64 mg/dL) and protein (31 mg/dL). Day zero blood tests showed normal electrolytes, renal function, aspartate aminotransferase, bilirubin and creatine phosphokinase, mildly raised alkaline phosphatase (200 U/L), and haemoglobin of 9.0 g/dL with normal white cell and platelet counts. The blood gas after intensive care admission on day zero during mechanical ventilation showed pH 7.399, PaCO2 31.5 mmHg, analyser-calculated standard bicarbonate 20.8 mmol/L, base excess -4.9 mmol/L and lactate 2.5 mmol/L, with hypokalaemia (2.9 mmol/L) and hyperglycaemia (134 mg/dL). On day two, during mechanical ventilation, there was transient hypercapnia and a low PaO2 in the morning (PaCO2 50.8 mmHg, PaO2 57.5 mmHg), corrected on a repeat gas that evening (pH 7.423, PaCO2 41.8 mmHg, PaO2 89.6 mmHg). Serum biochemistry on day two showed mild hypernatraemia (147 mmol/L), hyperchloraemia (108 mmol/L) and low total calcium (2.0 mmol/L), with normal renal function.

Investigation	Parameter	Day 0	Day 2	Reference range
Cerebrospinal fluid	White cell count	3 cells/μL	-	0-5 cells/μL
Cerebrospinal fluid	Red cell count	0 cells/μL	-	0 cells/μL
Cerebrospinal fluid	Glucose	64 mg/dL	-	40-70 mg/dL
Cerebrospinal fluid	Protein	31 mg/dL	-	15-45 mg/dL
Complete blood count	Haemoglobin	9.0 g/dL	-	13.5-17.5 g/dL
Complete blood count	White cell count	7.0 × 10^9/L	-	4.0-11.0 × 10^9/L
Complete blood count	Platelet count	337 × 10^9/L	-	150-400 × 10^9/L
Serum biochemistry	Sodium	141 mmol/L	147 mmol/L	135-145 mmol/L
Serum biochemistry	Potassium	3.8 mmol/L	3.6 mmol/L	3.5-5.1 mmol/L
Serum biochemistry	Chloride	-	108 mmol/L	95-105 mmol/L
Serum biochemistry	Calcium (total)	-	2.0 mmol/L	2.1-2.6 mmol/L
Serum biochemistry	Urea	38 mg/dL (6.3 mmol/L)	33 mg/dL (5.5 mmol/L)	15-45 mg/dL
Serum biochemistry	Creatinine	1.0 mg/dL (88 μmol/L)	0.8 mg/dL (70.6 μmol/L)	0.7-1.3 mg/dL
Liver function tests	Aspartate aminotransferase	29 U/L	-	10-40 U/L
Liver function tests	Alkaline phosphatase	200 U/L	-	44-147 U/L
Liver function tests	Total bilirubin	1.0 mg/dL	-	0.1-1.2 mg/dL
Muscle enzyme	Creatine phosphokinase	290 U/L	-	39-308 U/L
Blood gas*	pH	7.399	7.369	7.35-7.45
Blood gas*	PaCO2	31.5 mmHg	50.8 mmHg	35-45 mmHg
Blood gas*	PaO2	121 mmHg	57.5 mmHg	80-100 mmHg
Blood gas*	Standard bicarbonate	20.8 mmol/L	26.7 mmol/L	22-26 mmol/L
Blood gas*	Base excess	-4.9 mmol/L	+3.7 mmol/L	-2 to +2 mmol/L
Blood gas*	Lactate	2.5 mmol/L	0.9 mmol/L	0.5-1.6 mmol/L
Blood gas*	Glucose	134 mg/dL	93 mg/dL	70-104 mg/dL
Blood gas*	Potassium	2.9 mmol/L	3.5 mmol/L	3.4-4.5 mmol/L

TABLE 2: Laboratory and blood gas investigations

Day 0 values were obtained on the day of the event (CSF from the first sample taken during lavage); day 2 serum biochemistry was obtained at 10:33. *Blood gases: day 0 sample taken at 18:48 after intensive care admission; day 2 arterial sample taken at 10:59 during mechanical ventilation. Ionised calcium of 0.40 mmol/L on the day 0 blood gas was flagged by the analyser as a sample problem and is not reported. Reference ranges are those of the reporting laboratory or analyser. CSF, cerebrospinal fluid; PaCO2, arterial partial pressure of carbon dioxide; PaO2, arterial partial pressure of oxygen.

On day one, fundoscopy showed no papilloedema, and non-contrast brain computed tomography (CT) showed normal parenchymal density, a normal ventricular system, normal posterior fossa structures, no midline shift and no haemorrhage, with incidental bilateral maxillary and ethmoid sinusitis. Unfortunately, the CT images are not available for publication for technical reasons; the formal radiology report is summarised here. Brain magnetic resonance imaging (MRI) approximately three and a half months after the event showed no structural abnormality (Figure *2*).

No EEG monitoring was available during the acute phase. A clinical diagnosis of convulsive status epilepticus was made on the basis of recurrent generalised convulsions without recovery of consciousness despite first-line treatment. Because neuromuscular blocking agents were given and consciousness remained impaired for several days, ongoing nonconvulsive seizures could not be excluded during this period. A routine EEG recorded approximately 11 weeks after the event was normal but cannot clarify the acute course.

These findings did not identify a metabolic, infective, or structural cause for the seizures and were consistent with TXA neurotoxicity in this context.

Differential diagnosis

Inadvertent intrathecal TXA neurotoxicity was the primary diagnosis on the basis of the confirmed drug error, the absence of spinal block, and the rapid onset of severe back and gluteal pain, myoclonus, and generalised tonic-clonic convulsions. Local anaesthetic systemic toxicity was considered unlikely, as the only local anaesthetic given was subcutaneous infiltration of levobupivacaine 50 mg at the incision site, well below the recommended maximum dose and the presentation followed the confirmed intrathecal TXA injection. The day zero hypokalaemia, raised lactate and hyperglycaemia were recorded and had corrected by day two; their cause cannot be determined from the available data. There was no hypoglycaemia or hyponatraemia. CSF analysis did not suggest infection, and no structural abnormality was identified on the day-one CT or the follow-up MRI.

Treatment

After surgery was halted, the neurologist and anaesthesiologist coordinated management in the following sequence.

Immediate Seizure Control and Induction of General Anaesthesia

Following failure of initial benzodiazepine therapy (midazolam 5 mg IV), general anaesthesia was induced for airway protection and seizure suppression. Propofol (100 mg) and atracurium (40 mg) were used for induction and intubation, and anaesthesia was maintained with isoflurane (1.2 minimum alveolar concentration) and atracurium boluses. Phenytoin 1,250 mg IV (approximately 18 mg/kg) and levetiracetam 1,000 mg IV were given as anticonvulsant loading during the operative period. Dexamethasone 16 mg IV was also given, on the theoretical basis of limiting cerebral oedema and chemical meningeal inflammation after intrathecal exposure to a neurotoxic drug.

Management of Recurrent Seizures

At the end of surgery, recurrent generalised tonic-clonic convulsions were observed, the first lasting approximately five minutes, without recovery of consciousness between episodes and despite phenytoin and levetiracetam; the patient met clinical criteria for convulsive status epilepticus. He remained intubated throughout and was not extubated before transfer to intensive care. Diazepam (total 20 mg IV) and further propofol boluses were given; the recorded total propofol bolus dose before ICU transfer was 400 mg, and the record does not specify whether this included the 100 mg induction dose. Rocuronium 30 mg was given to facilitate ventilation. Magnesium sulphate 4 g IV was administered (infusion rate not recorded), after which no further convulsive activity was observed. Because neuromuscular blockade had been given, cessation of electrographic seizure activity could not be confirmed, and the contribution of magnesium, the other agents, or spontaneous resolution cannot be determined [7].

Cerebrospinal Fluid Lavage

After clinical stabilisation, CSF lavage was undertaken to reduce intrathecal TXA concentration [3,8,9]. Two 20-gauge Quincke spinal needles were placed at L2-L3 for drainage and L4-L5 for infusion. The first 10 mL of CSF was sent for analysis. Preservative-free normal saline was infused in 10 mL aliquots with simultaneous drainage of equal CSF volumes, exchanging approximately 150 mL over 90 minutes [3,8,9].

Intensive Care Management

The patient was admitted to the intensive care unit (ICU) at 15:30 on the day of the event, mechanically ventilated and sedated with a continuous propofol infusion, with head-of-bed elevation and nasogastric feeding. Phenytoin 100 mg IV every eight hours was continued until day three, and levetiracetam 1,000 mg IV every 12 hours was continued. Magnesium sulphate 4 g IV every 12 hours was prescribed on days zero and one; the record does not state whether the intraoperative 4 g dose formed the first of these doses. Monitoring was clinical; renal function was normal (creatinine 1.0 mg/dL on day zero and 70.6 µmol/L on day two), and serum magnesium concentrations are not available in the clinical record. Sedation was stopped on day two to allow neurological assessment.

Thromboprophylaxis

Enoxaparin 4,000 IU subcutaneously once daily was started on day one as venous thromboembolism prophylaxis following a joint multidisciplinary discussion, because the patient was considered at high thrombotic risk in the setting of lower-limb trauma, recent surgery, immobility and critical illness; this was not a specific treatment for TXA toxicity.

Corticosteroids

Dexamethasone 8 mg IV every 12 hours and methylprednisolone 250 mg IV once daily were given concurrently from the day of the event for presumed cerebral oedema and neuroinflammation, and methylprednisolone was stopped on day three; the rationale for using two corticosteroids concurrently is not recorded. There is no clinical evidence supporting corticosteroid use in TXA neurotoxicity.

Antimicrobials and Gastric Protection

intravenous ceftriaxone 1 g every 12 hours and metronidazole 500 mg every eight hours were given empirically; no infection was documented, and the day-one CT showed incidental bilateral maxillary and ethmoid sinusitis. Esomeprazole 40 mg IV daily was given as stress-ulcer prophylaxis. Intravenous paracetamol and maintenance fluids were also given.

Neuroactive Adjuncts

On day three, with consciousness still impaired after withdrawal of sedation, the treating team added memantine 10 mg twice daily, amantadine 100 mg twice daily and vinpocetine 20 mg twice daily via the nasogastric tube, with the intention of neuroprotection and promoting arousal. They were continued until at least day six and were not among the discharge medications. None has established benefit in TXA neurotoxicity.

Outcome and follow-up

Recovery was gradual. Continuous propofol sedation was maintained from ICU admission until it was stopped on day two (approximately 48 hours); sedation depth was assessed clinically, and no sedation score is documented. The patient remained in deep coma under sedation on days one and two, with daily clinical neurological assessment and neurology review; neurology planned MRI, magnetic resonance venography and angiography, and EEG once he was stable, and MRI and EEG were performed after discharge; when sedation was stopped on day two his Glasgow Coma Scale (GCS) score was 5T. He remained unconscious on day three (GCS 8; components not recorded) and was still mechanically ventilated on day four, with no further clinically observed seizures. He had been extubated by the morning of day five (exact time not documented), when he was conscious, alert and oriented on face-mask oxygen, and on day six his GCS was 15 with the operated limb neurovascularly intact. He was discharged home on day eight on oral levetiracetam 1,000 mg twice daily, rivaroxaban 10 mg once daily as extended thromboprophylaxis pending review at follow-up, and a tapering corticosteroid course over 11 days.

A routine EEG, approximately 11 weeks after the event, was normal. At three-month follow-up, the patient had recovered from surgery, had had no further clinically recognised seizures while taking levetiracetam, and had a normal neurological examination; cognitive and functional status, assessed clinically at discharge and at follow-up visits, were normal, although standardised neuropsychological testing was not undertaken. Levetiracetam was then tapered to 500 mg twice daily for a further three months, and rivaroxaban was discontinued. The patient did not attend further follow-up, so seizure recurrence after completion of the levetiracetam taper could not be assessed. Brain MRI approximately three and a half months after the event showed no structural abnormality (Figure *2*); a normal follow-up MRI does not exclude earlier transient abnormalities or subtle functional consequences.

Patient perspective

The patient provided the following account: "I was told later that a wrong medicine was injected by mistake and that this caused the seizures I experienced. I do not remember much from the first days, but I was relieved when I later learned that I would recover fully. I am grateful to the team who acted quickly and explained everything clearly to me afterwards. I hope sharing my story prevents this from happening to someone else."

Discussion

Accidental intrathecal administration of TXA is a rare but potentially catastrophic medication error in anaesthetic practice. TXA is widely used intravenously for its antifibrinolytic effect, but intrathecal exposure causes acute neurotoxicity, including severe back pain, muscle rigidity, generalised seizures, and sometimes cardiovascular instability. Fatal outcomes have been reported, particularly with refractory seizures and arrhythmias, although the published literature consists of case reports and is subject to reporting bias [1,2,5].

The biological basis of toxicity involves disruption of inhibitory neurotransmission. Experimental data suggest that TXA antagonises glycine and GABA-A receptors, resulting in neuronal disinhibition and hyperexcitability [4,6]. This mechanism is consistent with the rapid onset of convulsions and their poor response to benzodiazepines in our patient.

Visible convulsions were not controlled by midazolam, diazepam or loading doses of phenytoin and levetiracetam, and were no longer observed after additional propofol and magnesium sulphate. Because neuromuscular blockade had been given and acute EEG was unavailable, it cannot be established that seizure activity terminated at that point, and the contribution of magnesium cannot be separated from that of the other agents or from spontaneous resolution. Magnesium use for refractory seizures after intrathecal TXA has been described in a case report [7], but its efficacy has not been established, and its use here is reported as an observed intervention of uncertain benefit.

CSF lavage was performed after surgery with the intention of reducing intrathecal TXA concentration. CSF lavage has been described in a small number of case reports [3,8,9], using different techniques, including lumbar drainage with saline replacement [9] and ventriculolumbar perfusion [8]; the lumbar-lumbar exchange used here corresponds to the former. Because CSF TXA concentrations were not measured, the extent of any reduction cannot be demonstrated, and no conclusion about its effect on outcome can be drawn from this case.

Consciousness remained impaired for several days after sedation was stopped. Possible contributors include post-ictal encephalopathy, residual toxicity, the effects of sedative and anticonvulsant drugs, and ongoing nonconvulsive seizures, which could not be excluded without EEG. In this patient the delayed awakening was followed by clinical recovery, but a single case cannot establish its prognostic significance.

Treatments directed at recognised clinical problems (anticonvulsants, sedation and ventilation, thromboprophylaxis, and stress-ulcer prophylaxis) should be distinguished from those given on a theoretical or empirical basis without established benefit in TXA neurotoxicity. The latter comprised magnesium sulphate; two concurrent corticosteroids, given for presumed cerebral oedema and neuroinflammation, although the reason for combining two agents is not documented; and memantine, amantadine and vinpocetine, added on day three with the intention of neuroprotection and promoting arousal during delayed recovery of consciousness. They are reported for transparency, not as recommendations, and their concurrent use prevents attribution of recovery to any single intervention.

This case illustrates two failed safety barriers. First, TXA and bupivacaine ampoules of similar size and label appearance were stored in the same theatre tray, and the drug drawn up for spinal injection was checked only once, by an assistant. Second, the spinal block was not verified by sensory or motor testing before incision, and subcutaneous infiltration of the incision site meant that the absence of a spinal block was not apparent at incision. Either barrier, if intact, might have prevented the error or revealed it before surgery began. After the incident, TXA was withdrawn from the operating theatres at our institution. Further measures supported by previous reports include physical separation of TXA from neuraxial drugs, distinct labelling, barcode scanning, and an independent double-check before neuraxial injection [2,10]; colour coding alone should not be relied upon. Verification of block adequacy before incision should be treated as a safety check in its own right, and the absence of a block after a neuraxial injection should prompt immediate consideration of a wrong-drug error.

Completion of fracture fixation after recognition of the error was a judgement made by the operating surgeon, anaesthesiologist and neurologist, with the patient haemodynamically and respiratorily stable, the incision already made, general anaesthesia established, and the aim of avoiding a second anaesthetic and a delayed operation. The decision had potential costs: CSF lavage was delayed by approximately 45 minutes until fixation was complete, and, because neuromuscular blockade was in place, the absence of visible convulsions during surgery could not be taken as evidence of seizure control. Whether earlier abandonment of surgery and immediate lavage would have altered the course cannot be determined from this case, and it should not be taken as guidance on that question.

Strengths and limitations

This report is based on review of the available clinical, nursing and laboratory records, supplemented by the treating team's recollection for the intraoperative sequence, and reports drug doses and the course of recovery as recorded, including agents without an evidence base.

The limitations are those inherent to a single case. Causality cannot be attributed to any individual intervention. EEG was not available in the acute phase and neuromuscular blocking agents were given, so cessation of seizure activity could not be confirmed and nonconvulsive seizures during the period of impaired consciousness could not be excluded; the only EEG was a single routine recording at approximately 11 weeks. Intraoperative times are approximate and were reconstructed retrospectively. Serum magnesium concentrations, CSF opening pressure and CSF TXA concentrations were not available. Acute neuroimaging was limited to non-contrast CT, and MRI was performed only at follow-up. Cognitive and functional status were assessed clinically rather than by standardised testing, and the patient did not attend follow-up after the levetiracetam taper, so later seizure recurrence cannot be excluded. A normal follow-up MRI does not exclude earlier transient abnormalities.

Conclusions

In this case, a patient who received an estimated 280 mg of TXA intrathecally developed convulsive status epilepticus and prolonged impaired consciousness, and made a clinical neurological recovery following treatment for convulsive status epilepticus, intensive care, CSF lavage and several adjunctive treatments. The case cannot establish the efficacy of any specific rescue intervention.

The specific lessons concern the safety barriers that failed. Look-alike TXA and bupivacaine ampoules stored in the same tray and checked only once allowed the wrong drug to be drawn up, and incision without verification of the spinal block removed the last opportunity to detect the error before surgery. TXA should be stored and handled separately from neuraxial drugs, with independent pre-administration checks; block adequacy should be formally verified before incision; and a serious wrong-route error, once recognised, should prompt early neurological management. At our institution, TXA has since been withdrawn from the operating theatres.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Azhar Shabbir, Hussein Albarzanjy, AlAmeer Awad

Critical review of the manuscript for important intellectual content: Azhar Shabbir, Hussein Albarzanjy, AlAmeer Awad

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Disclosures

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