High-Dose Tranexamic Acid and Intraoperative Seizure After Cardiopulmonary Bypass

Summary of a Case Report | A&A Practice 2026;20:e02234 | Oya K, Maki J, Higashi M

Why this paper matters to Haemoview

This open-access case report in *A&A Practice* addresses a practical safety problem in cardiac surgery: tranexamic acid (TXA) is widely used in cardiovascular surgery to reduce bleeding and transfusion requirements, but it is a known dose-dependent risk factor for perioperative seizures. In this case, a 44-year-old man undergoing mitral valve repair received TXA 3000 mg immediately before cardiopulmonary bypass (CPB) and another 3000 mg four minutes after successful CPB weaning. The total 6000 mg dose, approximately 92 mg/kg, exceeded the recognised >80 mg/kg seizure-risk threshold. Continuous electroencephalography (EEG), Patient State Index (PSI), central venous oxygen saturation (ScvO2), regional oxygen saturation (rSO2) and hemodynamic monitoring captured a direct temporal association between high-dose TXA administration and seizure onset.

Study at a glance

Item	Detail
Article type	Peer-reviewed case report of TXA-associated intraoperative seizure after cardiopulmonary bypass
Authors	Oya K, Maki J, Higashi M - Kyushu University Hospital and Kyushu University Graduate School of Medical Sciences, Fukuoka, Japan
Journal	A&A Practice 20(6) (2026) e02234; DOI: 10.1213/XAA.0000000000002234; Open Access (CC BY-NC-ND)
Clinical scope	A 44-year-old man undergoing minimally invasive mitral valve repair for severe mitral regurgitation; moderate hypothermia at 33 degrees C; aortic cross-clamp time 80 minutes and CPB time 129 minutes
Core finding	Generalized convulsions occurred 13 minutes after CPB termination after a cumulative TXA dose of 6000 mg, approximately 92 mg/kg. EEG spike waves appeared one minute after the second TXA dose and increased to sustained rhythmic discharge.
ClotPro link	The primary case did not use ClotPro. In its discussion, the case report lists dose adjustment using point-of-care fibrinolysis testing among proposed TXA dosing approaches; the accompanying proof-of-concept article evaluates the ClotPro TPA-test for TXA-induced inhibition of fibrinolysis.

The clinical gap this paper closes

Perioperative seizures occur in approximately 1% of cardiac surgeries and are usually recognised postoperatively in the intensive care unit when sedation is tapered, with a median onset time of 7 hours post-surgery. The paper highlights why diagnosis is difficult: general anesthetics, sedatives and muscle relaxants can mask typical manifestations, creating a time lag that may worsen prognosis and make TXA causality harder to determine. This case closes that observational gap by documenting EEG evolution, clinical convulsion and oxygenation/hemodynamic changes continuously from the post-CPB TXA dose to treatment.

Key message: High-dose TXA can trigger seizures immediately after administration. In this case, integrated assessment of continuous EEG and ScvO2, supported by PSI, rSO2 and hemodynamic trends, enabled early detection of a masked perioperative seizure and prompt treatment.

Key findings in detail

1 | High-dose TXA exposure exceeded the recognised seizure-risk threshold

The institutional protocol used approximately 50 mg/kg of TXA before CPB and another 50 mg/kg immediately after CPB. The patient received 3000 mg before CPB and 3000 mg immediately after CPB, for a total of 6000 mg, approximately 92 mg/kg. The authors note that high cumulative TXA doses, particularly >80 mg/kg, are strong independent predictors of postoperative convulsive seizures.

Measure	Reported case value	Clinical meaning in the article
TXA exposure	3000 mg pre-CPB + 3000 mg post-CPB; total 6000 mg, approximately 92 mg/kg	Exceeded the recognised >80 mg/kg seizure-risk threshold
EEG timing	First spike wave one minute after second TXA administration; spike-wave complexes by four minutes	Provided a direct temporal association between TXA administration and electrographic seizure activity
Clinical event	Generalized convulsions with prominent muscle rigidity; train-of-four count was 1	Clinical signs occurred despite residual neuromuscular blockade
Treatment	Midazolam 5 mg terminated convulsions; propofol increased; levetiracetam 1000 mg administered	No further convulsions were reported

2 | EEG and PSI identified seizure activity before and during convulsion

Raw EEG from the SedLine monitor was normal upon weaning from CPB. One minute after the second TXA dose, the first spike wave appeared; after four minutes, spike waves transitioned into a sustained rhythmic discharge described as a status epilepticus pattern. PSI became undetectable two minutes after TXA administration and remained unmeasurable until the seizure was terminated with midazolam.

3 | ScvO2 and hemodynamic changes added an early physiologic signal

During the convulsive episode, heart rate increased from 88 to 116 bpm, arterial pressure from 76/40 to 100/54 mm Hg, cardiac output from 5.8 to 7.9 L/min, and bilateral rSO2 rose. In contrast, ScvO2 decreased from 82% to 65% while transesophageal echocardiography showed unchanged cardiac function. The authors interpret this as a global mismatch between oxygen delivery and sudden whole-body metabolic demand.

4 | Prompt treatment was followed by a favourable neurological course

Midazolam 5 mg terminated the convulsions; propofol was increased and levetiracetam 1000 mg was administered, with no further convulsions reported. In the ICU, head CT and 10 to 20 EEG showed no abnormalities. On postoperative day 1, continuous EEG confirmed absence of abnormal brain waves, the patient was extubated with no neurological deficits, and he was discharged on day 20.

5 | The authors caution that causality is multifactorial

Although the temporal relationship strongly implicated TXA, the authors caution that perioperative seizure etiology is multifactorial. Childhood febrile seizure history, anesthetic modulation of cortical excitability, and transient cerebral microembolism during deairing and CPB weaning could not be definitively excluded.

6 | TXA dosing remains unsettled

The authors state that standardised TXA regimens remain elusive and list lower-dose protocols, point-of-care fibrinolysis testing and pharmacokinetic approaches among proposed strategies to balance antifibrinolytic efficacy with neurotoxicity risk.

Relevance to ClotPro and Multiclot users

The primary case report did not evaluate ClotPro or Multiclot. Its relevance is the clinical safety scenario it documents: high-dose TXA after CPB was followed within minutes by electrographic and clinical seizure activity, while recognition can be delayed by anesthetics, sedatives and muscle relaxants. The ClotPro connection comes from the case report discussion and the referenced proof-of-concept study of the ClotPro TPA-test, which evaluated TXA-induced inhibition of fibrinolysis.

Paper finding	Relevance to users
Point-of-care TXA effect testing	Point-of-care laboratory tests for TXA concentrations are unavailable. The ClotPro TPA-test uses tPA-triggered fibrinolysis to assess TXA-induced inhibition and may allow bedside quantification of in-vivo TXA effect.
Sensitive to low TXA concentrations	In vitro, TXA inhibited tPA-induced hyperfibrinolysis at >=2 mg/L in a concentration-dependent manner, and complete inhibition of fibrinolysis occurred at >=10 mg/L.
Detects CPB patient response	In 20 cardiac surgery patients, TPA-test parameters detected antifibrinolytic effect after a 0.1 g TXA bolus at the end of CPB; complete inhibition was observed with TXA >=0.5 g.
Tracks postoperative residual effect	After 1 g TXA, the antifibrinolytic effect attenuated over 18 h in most patients, but inhibition persisted in four patients at 18 h with LT 7200 s and ML <25%.
Renal impairment signal	All four patients with residual antifibrinolytic response had grade 4 or 5 renal dysfunction; eGFR strongly correlated with TPA-test parameters at 18 h (r=0.86-0.92; P<0.0001).

Suggested conversation points

- *The case report shows why cumulative TXA exposure matters; the TPA-test study shows a point-of-care method for assessing TXA antifibrinolytic effect.*
- *The ClotPro TPA-test evaluates tPA-inducible fibrinolysis and detected low concentrations of TXA in vitro.*
- *In CPB patients, the TPA-test detected effect after 0.1 g TXA and complete fibrinolysis inhibition with TXA >=0.5 g.*
- *Persistent inhibition in severe renal impairment supports discussing renal function when considering postoperative TXA effect monitoring.*

Sources

Oya K, Maki J, Higashi M. Tranexamic Acid-Induced Seizure After Cardiopulmonary Bypass: A Case Report. *A&A Practice.* 2026;20:e02234. DOI: 10.1213/XAA.0000000000002234 | Open Access (CC BY-NC-ND)

Yoshii R, Takahashi Y, Tanaka KA, et al. Point-of-care testing for tranexamic acid efficacy: a proof-of-concept study in cardiac surgical patients. *British Journal of Anaesthesia.* 2024;132:1211-1218. DOI: 10.1016/j.bja.2024.03.023

Disclaimer

CASE REPORT

TXA-INDUCED SEIZURE AFTER CARDIOPULMONARY BYPASS

What a perioperative case report suggests about dose, monitoring, and early detection

Based on Oya K, Maki J, Higashi M. A&A Practice. 2026;20:e02234

ClotPro TPA-test evaluates tPA-triggered fibrinolysis to assess TXA-induced inhibition at point of care. In CPB patients, it detected effect after 0.1 g TXA, complete inhibition at >=0.5 g, and postoperative residual effect.

WHY THIS MATTERS

Tranexamic acid (TXA) reduces bleeding in cardiac surgery, but seizure risk rises with higher doses.

Perioperative seizures are often missed because anaesthetics and muscle relaxants mask clinical signs.

This case highlights how integrated monitoring ($ScvO_2$) may reveal evolving seizure activity earlier.

KEY CLINICAL MESSAGE

TXA is a known risk factor for perioperative seizures, and this risk is strongly dose-dependent, particularly in cardiac surgery.

High cumulative TXA doses, particularly exceeding 80 mg/kg, are strong independent predictors of postoperative convulsive seizures. Altered blood–brain barrier permeability during CPB facilitates higher cerebrospinal fluid drug concentrations, exacerbating the risk with high dosing.

Universally established standardized regimens remain elusive, with various regimens proposed based on clinical trials and dose adjustments using point-of-care fibrinolysis testing.

DOSE MATTERS

Patient weight: 65 kg

TXA administered:
- 3000 mg before CPB
- 3000 mg after CPB weaning (4 minutes post-CPB)

Each bolus ≈ 46 mg/kg

Total TXA = 6000 mg

Cumulative exposure:

≈ 92 mg/kg

Above the >80 mg/kg threshold associated with increased seizure risk in cardiac surgery.

Oya K, Maki J, Higashi M. Tranexamic Acid-Induced Seizure After Cardiopulmonary Bypass: A Case Report. A&A Practice. 2026;20:e02234.
DOI: 10.1213/XAA.0000000000002234

Haemoview Diagnostics
Redefining coagulation

DOI: 10.1213/XAA.000000000002234

Created by Haemoview Diagnostics.
Infographic design assisted by AI tools.