Pavanchand et al. Sri Lankan Journal of Anaesthesiology 34 (3): 475-482 (2026)

doi: https://doi.org/10.4038/slja.v34i3.9915

Published date: 20th November 2026

SLJA

CASE REPORT

Fatal Anaesthetic Complication Following Sequential Spinal and General Anaesthesia in a Paediatric Patient: A Case Report

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ABSTRACT

Anaesthetic death, defined as death within 24 hours of anaesthesia administration, is rare but carries significant medicolegal implications. Fatal complications arising from sequential use of spinal and general anaesthesia in non-emergency paediatric cases are exceptionally uncommon and poorly documented.

A 16-year-old male underwent wound debridement under spinal anaesthesia with 0.5% bupivacaine (12.5 mg). Following the onset of abnormal upper limb movements, a decision was made to convert to general anaesthesia using propofol, clonidine, and atracurium. Postoperatively, the patient developed refractory convulsions requiring escalating anticonvulsant therapy (levetiracetam,

Received: 19th May 2026 **Accepted:** 12th November 2026

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lorazepam, midazolam, phenobarbitone). He subsequently developed ventricular tachycardia followed by cardiac arrest, and was declared dead at a higher referral centre. Autopsy revealed pulmonary oedema, acute kidney injury, and reactive gliosis with congested cerebral vessels. Forensic toxicology identified levetiracetam, phenobarbitone, tramadol, bupivacaine, laudanosine, and lidocaine across biological samples.

This case underscores the critical importance of LAST awareness, mandatory availability of lipid emulsion therapy, and judicious anaesthetic decision-making in non-emergency paediatric surgery.

Keywords: Spinal anaesthesia, general anaesthesia, convulsions, cardiac arrest, local anaesthetic systemic toxicity (LAST), laudanosine, medicolegal, paediatric anaesthesia

INTRODUCTION

Anaesthetic death is often defined as the death of a patient within 24 hours of administration of anaesthesia, irrespective of the contribution of anaesthesia to the cause of death.[1] Anaesthetic deaths are rare, with an occurrence rate of 1–4 deaths per 10,000 anaesthesias.[2]

Among the commonly used anaesthetic techniques, spinal anaesthesia is a regional technique known for its safety and minimal complications, which are usually easily manageable. It involves the administration of local anaesthetics such as 0.5% bupivacaine into the subarachnoid space. To enhance the quality of the block, additional medications like clonidine or opioids may be added.[3]

General anaesthesia includes the administration of neuromuscular blocking agents such as atracurium, which has been associated with anaphylactoid and anaphylactic reactions.[4]

Local Anaesthetic Systemic Toxicity (LAST) is a rare but life-threatening complication that can follow absorption of local anaesthetic agents into the systemic circulation. First-line management includes cessation of the offending agent, airway management, seizure control, and immediate administration of 20% intravenous lipid emulsion (Intralipid) as a specific antidote.

CASE PRESENTATION

A 16-year-old (55 kg) South Asian boy (paediatric patient; ASA Physical Status Classification: ASA I, no known comorbidities) presented with trauma to the right foot sustained while riding a bicycle. He was initially treated at another healthcare facility and subsequently admitted for a deep, contaminated contused-lacerated wound (3” × 3”) with muscle rupture and multiple bone fragments.

The following day, wound debridement and suturing were planned as an elective, non-emergency procedure. Pre-procedurally, the patient was sedated with midazolam 10 mg and ketamine 10 mg, following which spinal anaesthesia was administered with 12.5 mg of 0.5% hyperbaric bupivacaine.

As the patient was being positioned for the procedure, abnormal jerky movements of the upper limbs were observed. Propofol 120 mg was administered intravenously, following which the abnormal movements subsided. A decision was then made to convert from spinal to general anaesthesia. Clonidine was administered intravenously in three sequential

doses (60 mcg, 30 mcg, 30 mcg), and atracurium was given (25 mg loading, followed by 5 mg, 5 mg, 5 mg). The patient was intubated, and the procedure proceeded under general anaesthesia.

Autopsy revealed no specific gross findings. Surgical staples were removed and examined for signs of infection (none found). Histopathological examination and forensic toxicology samples were submitted as detailed in Table 2. Histopathology revealed pulmonary oedema, acute kidney injury, and reactive gliosis with congested cerebral vessels.

Table 1: Clinical timelines from administration of anaesthesia to death

Time Point	Event
T0	Spinal anaesthesia administered (bupivacaine 12.5 mg 0.5%)
T0 + ~minutes	Abnormal upper limb movements observed during positioning
Shortly after	Propofol 120 mg IV given; movements subsided. Decision to convert to general anaesthesia (GA)
Intraoperative	Clonidine (60+30+30 mcg IV), Atracurium (25+5+5+5 mg IV); intubated; procedure completed under GA
End of procedure	Attempts to extubate; abnormal jerky limb movements recurred; extubation abandoned
Post-procedure	Refractory convulsions: levetiracetam 1g IV → loraze pam 2mg IV stat → midazolam 40mg IV stat → phenobarbitone 400mg IV over 20 min; convulsion frequency/intensity decreased
T0 + ~1 hour post-op	Ventricular tachycardia; resuscitation successful
T0 + ~2 hours post-op	Bradycardia and cardiac arrest; resuscitation initiated
T0 + ~2.5 hours post-op	Patient transferred to higher centre; declared dead on arrival

Table 2: Samples submitted to the Regional Forensic Science Laboratory for analysis

No	Sample	Drugs Found
1.	Stomach and portion of small intestines	Levetiracetam, phenobarbitone
2.	Liver	Levetiracetam, phenobarbitone
3.	Kidney	Levetiracetam, phenobarbitone
4.	Blood	Levetiracetam, phenobarbitone, tramadol
5.	Lung	Levetiracetam, phenobarbitone, tramadol
6.	CSF	Levetiracetam
7.	Brain	Phenobarbitone
8.	Urine	Levetiracetam, laudanosine, lidocaine*
9.	Lumbar spinal cord segment	Levetiracetam, bupivacaine

DISCUSSION

Human error significantly impacts anaesthetic practice, often resulting in more immediate and noticeable adverse consequences than in other medical specialties. However, due to thorough pre-anaesthetic evaluations and post-operative monitoring, deaths attributable solely to anaesthesia or anaesthetic errors are exceedingly rare, occurring in approximately 0.5–0.8 cases per 100,000 anaesthetics. Intraoperative deaths, though more common at 1–30 per 100,000 cases, predominantly occur during emergency surgeries, accounting for approximately 80% of such instances.[5]

Common contributing factors include extremes of age, comorbid conditions (particularly ASA grade III and above), emergency surgery (neurosurgical or cardiac), and anaesthetic factors including drug dosage errors, hypersensitivity, incorrect labelling, accidental route-of-administration errors, insufficient experience, and intubation mishaps.[6]

ASA Classification and Paediatric Considerations

The present patient was 16 years old, classifying him as a paediatric patient by most anaesthesia standards. He was an otherwise healthy individual (ASA I), which places this case among those rare fatal anaesthetic events in low-risk patients.

Abnormal jerky limb movements occurring during or after anaesthesia may represent myoclonus, seizure-equivalent activity, or systemic drug toxicity. The term ‘seizure’ is reserved for events with electroencephalographic (EEG) confirmation; ‘convulsion’ denotes the clinical motor phenomenon.

Table 3: Proconvulsant and anticonvulsant properties of common anaesthetic agents

No	Proconvulsant	Anticonvulsant
1.	Nitrous oxide, halothane, enflurane, isoflurane	Halothane, Enflurane, Isoflurane
2.	Morphine, meperidine, fentanyl, sufentanil	—
3.	Methohexital	Thiopental
4.	Etomidate (at high doses)	Etomidate (at low doses)
5.	Diazepam (paradoxical)	Diazepam, lorazepam, midazolam
6.	Ketamine (at subanaesthetic doses)	Ketamine (at anaesthetic doses)
7.	Propofol (excitatory phenomena during induction/emergence)	Propofol (anticonvulsant at maintenance doses)
8.	Local anaesthetics e.g. bupivacaine (at toxic systemic levels)	Local anaesthetics show a biphasic effect: anticonvulsant at low plasma concentrations (e.g. IV lidocaine used in refractory seizures)

Several agents (e.g., propofol, ketamine, local anaesthetics, etomidate, diazepam) appear in both columns because their convulsant or anticonvulsant effects are dose-dependent and context-dependent. This dual classification reflects pharmacological reality and should not be interpreted as inconsistency.[7,8]

Depending on clinical context, anaesthetic drugs may act as either proconvulsants or anticonvulsants (Table 3).[7] Convulsions most commonly occur during induction or emergence from anaesthesia when drug concentrations are in a transitional range.[8]

In spinal anaesthesia, complications may arise from local anaesthetic systemic toxicity (LAST), neural injury, or block failure. Song et al. (1994) reported a case of status myoclonic seizure following spinal anaesthesia with tetracaine, attributed to systemic absorption and cephalad spread in the CSF.[9] Tonic-clonic movements following bupivacaine epidural administration are more common than following spinal anaesthesia.[10] Lee et al. observed that myoclonus is a rare neurological complication of spinal anaesthesia without lasting sequelae[11], while Kumari et al. confirmed Brown et al.'s observation that bupivacaine is 4–6 times more cardiotoxic than lignocaine, documenting cardiac arrest following seizures in an 18-year-old undergoing orchidopexy under spinal anaesthesia with bupivacaine.[12] Cardiac arrest may occur as early as 12–72 minutes after spinal anaesthesia, or be delayed by hours.[13]

Medicolegal Opinion — Cause of Death

Integrating the clinical timeline, histopathological report, and forensic toxicological data, the cause of death in this case is most consistent with refractory status epilepticus and fatal cardiac arrest precipitated by Local Anaesthetic Systemic Toxicity (LAST) from bupivacaine, compounded by probable anaphylactoid reaction to atracurium. The histopathological findings of pulmonary oedema, acute kidney injury, and reactive gliosis with congested cerebral vessels are in favour of prolonged seizure activity and cardiovascular compromise. The detection of bupivacaine in the lumbar spinal cord segment confirms CNS exposure. Laudanosine — a neurotoxic metabolite of atracurium hydrolysis — was detected in urine, suggesting that atracurium metabolism contributed to CNS excitation and the convulsive course. The contribution of antiepileptic drugs (levetiracetam, phenobarbitone) to prolonged cardiac arrest, via sodium-channel blockade, as noted by Mallick et al., cannot be excluded.[4]

Toxicological Significance of Laudanosine

Laudanosine is the principal metabolite of atracurium, which readily crosses the blood-brain barrier. Unlike atracurium itself, laudanosine lacks neuromuscular blocking activity but possesses CNS stimulant properties, with seizure-inducing potential documented in animal studies. The detection of laudanosine in urine in the context of refractory convulsions strengthens the hypothesis of a neurotoxic contribution from atracurium metabolism.

Absence of Lipid Emulsion (Intralipid) Therapy

A critical omission in the management of this case was the apparent failure to administer 20% intravenous lipid emulsion (Intralipid), the evidence-based antidote for LAST per AAGBI and ASRA guidelines. Lipid emulsion acts as a 'lipid sink,' sequestering bupivacaine from cardiac and CNS tissue. In a case of suspected bupivacaine-induced LAST with refractory seizures and subsequent cardiac arrest, early lipid emulsion administration is indicated and may have altered the outcome.

In the present case, following convulsions after spinal bupivacaine and anticonvulsant administration, the decision was made to convert to general anaesthesia with atracurium and continue surgery. Atracurium is known for anaphylactic and anaphylactoid reactions.[14] Anaphylactic reactions during general anaesthesia occur at a rate of approximately 1 in 6,000, with approximately 80% attributed to neuromuscular blocking agents.[15]

Mallick et al. have advocated for extended resuscitative efforts in cases of cardiac arrest following anaphylactic reactions to neuromuscular blockers, particularly when antiepileptic drugs have been administered, as these agents prolong sodium–potassium channel activity and thereby extend the duration of cardiac arrest.[4]

Decision to Convert Anaesthesia in a Non-Emergency Surgical Setting

The conversion from spinal to general anaesthesia following an adverse event — in a non-emergency paediatric patient — requires critical scrutiny. Published guidelines and case precedent (e.g., Antwi-Kusi et al.[16]) document such conversion primarily in emergency settings. In elective or semi-elective contexts, the appropriate response to suspected LAST is to terminate the procedure if possible, secure the airway conservatively, administer lipid emulsion, and transfer to a higher level of care.

Proceeding with general anaesthesia using a neuromuscular blocker in a patient already exhibiting CNS toxicity from bupivacaine represents a decision that warrants forensic scrutiny.

The primary recommendation for reducing anaesthetic risk is using the lowest effective dose. Data on safety ranges for local anaesthetic dosing and the frequency of LAST after peripheral nerve blocks would be highly beneficial in guiding clinical practice.[17]

CONCLUSION

Anaesthesiology remains a high-risk field with a narrow margin of safety, where rare but catastrophic complications such as refractory convulsions and cardiac arrest can arise from both spinal and general anaesthesia.

This case underscores the importance of: (1) recognising and managing LAST with lipid emulsion therapy; (2) cautious decision-making regarding anaesthetic technique conversion in non-emergency settings; (3) vigilance regarding laudanosine accumulation with repeated atracurium dosing; and (4) extended resuscitation efforts when antiepileptic agents have been administered.

Mandatory availability of 20% lipid emulsion in all anaesthesia-capable facilities, structured audit of LAST events at regional hospitals, and enhanced anaesthesia provider training in LAST recognition and management are strongly recommended.

LEARNING POINTS

The following key takeaways are highlighted for clinical and educational relevance:

- LAST (Local Anaesthetic Systemic Toxicity) should be considered immediately when convulsions follow spinal or regional anaesthesia with bupivacaine.
- 20% Intravenous Lipid Emulsion (Intralipid) must be immediately available in all anaesthesia-capable facilities per AAGBI/ASRA guidelines.
- Laudanosine, the neurotoxic metabolite of atracurium, can contribute to CNS excitation and should be considered in the differential of post-anaesthetic convulsions.
- Conversion to general anaesthesia in a non-emergency setting following suspected LAST should be critically evaluated and justified.

AUTHORS' CONTRIBUTIONS

Concept and design: PS, RA

Literature review: SM, SD

Compilation of manuscript: PS, SM

Manuscript editing and proofreading: PS, RA

DECLARATION OF CONFLICTS OF INTEREST

The authors declare no competing interests.

DECLARATION OF PATIENT'S CONSENT

Informed consent was obtained from the deceased's family for publication.

FUNDING

No external funding was received.

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